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The interaction of stress and sugar consumption as a predictor of depression

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by

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Abstract of the dissertation

The interaction of stress and sugar consumption as a predictor of depression

by Adi Fish-Williamson for the partial satisfaction of the requirements for the degree of Doctor of Philosophy in Psychological Sciences University of California, Merced 2024
Dr. Jennifer Hahn-Holbrook, Chair

Depression is a leading cause of disability in the United States. Previous research has shown that added sugar consumption and stress are both risk factors for depression. Despite evidence that added sugar consumption and stress both affect the HPA axis and may thus interact to amplify the risk of depression, no research has explored this interaction. In this dissertation, I investigated the possible effects of total added sugar and sugar-sweetened beverage consumption on depression, and their potential interactions with chronic stress. In a two-study series, measures of sugar consumption, chronic stress, and depression were taken in an adult community sample at two time points (Study 1) and a sample of pregnant women followed into the postpartum period (Study 2). I hypothesized that high sugar consumption would predict more depression, and stress amplify this effect. In summary, across two studies, sugar consumption predicted depressive symptoms. However, these results were no longer statistically significant when various covariates or baseline depression levels were added into the models. No interactions between sugar and stress were found in the predicted direction. These results

suggest that, while added sugar consumption and depressive symptoms are positively correlated, their relationship is complex and further research is warranted.

Chapter 1

Depression is a serious public health concern globally. One in 10 people in the United States suffer from depression (Goodwin et al., 2022). In adolescents and young adults, the prevalence is closer to 1 in 5 (Goodwin et al., 2022), making it the most common mental health concern for this age group (James et al., 2018). Depression can be a physically and emotionally debilitating disorder, and significantly increases the risk of cardiovascular disease, stroke, diabetes, obesity, and suicide (Penninx et al., 2013; Rihmer, 2007). Like many disorders, the true cause of depression is unknown, but there is strong evidence for both genetic and environmental risk factors (Caspi et al., 2003; Lopresti et al., 2013). Research investigating lifestyle factors influencing depression has been expanding and is of particular interest due to the potential modifiability of daily habits. Alongside adequate sleep and exercise, diet has been pinpointed as a predictor of depression (Lopresti et al., 2013). Various dietary factors have been shown to increase depression risk, such as consumption of processed foods, fast food, and processed added sugar (Fish & George, 2020; Pagliai et al., 2021; Sánchez-Villegas et al., 2012). Given that added sugar is one of the largest sources of calories in the United States (*USDA*, 2019), research has investigated whether it plays a role in mental illness.

High added-sugar consumption can adversely affect physical health and lead to obesity, type II diabetes, cardiovascular disease, and mortality (Alexander Bentley et al., 2020; Collin et al., 2019; Taskinen et al., 2019). Interest in the relationship between added sugar consumption and mental health has been growing, and while the field is still relatively small, multiple studies have investigated the relationship between added-sugar consumption and depression (Fish & George, 2020; D. Hu et al., 2019a; Knüppel et al., 2017; Sanchez-Villegas et al., 2018). For example, in a cross-sectional study of 74 women, Fish and George (2020) found a positive association between sugar consumption and depressive symptoms. Moreover, in a longitudinal study of 15,546 Spanish adults in the SUN cohort, Sanchez-Villegas and colleagues (2018) found that those in the highest quartile of added sugar consumption at the 10 year follow-up were at significantly higher risk for depression (Sanchez-Villegas et al., 2018). In the Whitehall II study of 23,245 person-observations, it was found that men with the highest quartile of sugar consumption had a 25% increase in odds of having a common mental health disorder after 5 years (Knüppel et al., 2017). This effect was independent of other dietary, socioeconomic, and health factors. Together these studies suggest that high added sugar consumption may contribute to adverse mental health outcomes (Sanchez-Villegas et al., 2018).

Multiple theories have been put forth to explain the relationship between high sugar consumption and depression. One proposed mechanism is that sugar consumption causes an increase in certain gut bacteria that disrupt healthy brain function, specifically memory (Levens & Gotlib, 2010). In a rodent study that examined the effects of sugar-sweetened beverage (SSB) consumption during adolescence, juvenile rats that were given

free access to SSB had impaired hippocampal-dependent memory function and an altered gut-microbiome in adulthood (Noble et al., 2021). Existing research indicates that issues in working memory may underlie problems with regulating emotions that lead to mood disorders such as depression (Levens & Gotlib, 2010). One study in humans found that people with depression were slower to disengage from sad stimuli and faster to disengage from happy facial expressions compared to nondepressed controls (Levens & Gotlib, 2010). Another possible mechanism is through the hypothalamic-pituitary-adrenal (HPA) axis. For example, Harrell and colleagues (2015) explored the idea that diets high in fructose alter HPA function in rats, which is relevant given that dysregulated HPA function has been associated with an increased risk for depressive-like symptoms in rats and humans (Harrell et al., 2015). To test this in rodents, researchers conducted a study in which juvenile rats were either fed a high-fructose diet or chow and found that the high-fructose-fed rats showed increased anxiety-like behavior in a maze, and depressive-like behavior in a forced swim test in adulthood (Harrell et al., 2015). These findings persisted regardless of the rats' stress history (Harrell et al., 2015). Harrell and colleagues (2015) also found that the fructose-fed rats showed higher concentrations of corticosterone (i.e., cortisol in humans) than the chow-fed rats, indicating these differences can likely be attributed to HPA dysfunction (Harrell et al., 2015). As fructose is the most common sweetener in processed foods, it is plausible that similar effects could be observed in humans (Hu et al., 2019).

Recent efforts to synthesize the limited human literature previously discussed demonstrate there is an effect across studies. For example, in a meta-analysis of observational studies examining sugar-sweetened beverage consumption and depression

risk, 6 out of 10 of the articles found a statistically significant relationship (Hu et al., 2019). Overall, the meta-analysis concluded that the combined risk of depression for people consuming the highest (verses lowest consumption) of SSBs was 1.31, although there was significant heterogeneity in effect sizes across studies. One possible explanation for this heterogeneity is the methodology used in the studies. For example, cross-sectional studies may not accurately capture the effects of high added-sugar consumption over time, and consequent chronic health outcomes (Wang & Cheng, 2020). For example, over time, high added-sugar consumption can lead to inflammation, insulin dysregulation, and obesity (Tchernof & Després, 2013), all of which are linked to depression (Hryhorczuk et al., 2013; Stetler & Miller, 2011), however, cross-sectional studies are less likely to be able to capture these long-term side effects.

Here, we proposed to test a novel potential interaction in the relationship between sugar consumption and depression risk – chronic stress and sugar. Like sugar, chronic stress has been linked to inflammation (Cohen et al., 2012), as well as cardiovascular disease, type II diabetes, and, depression (Cohen et al., 2012; Stetler & Miller, 2011). Various forms of stress including inflammatory, psychological, and traumatic stress can activate the HPA axis and catalyze a chain of events in the body including hormonal changes and cortisol release (Joseph & Golden, 2017). Cortisol is a powerful glucocorticoid that is regulated by the HPA axis and plays important roles in homeostatic bodily maintenance actions such as anti-inflammation, immune system, and metabolism of adipose, carbohydrates, and protein. It can induce diseases such as Addison's disease and Cushing's syndrome (Katsu & Baker, 2021). Cortisol's most well-known role is acting as a stress hormone, which responds to both physical and emotional stress (Katsu

& Baker, 2021). In many situations, the HPA's acute cortisol response is beneficial, allowing the body to focus on potential danger, however, repeated bouts of acute stress overtime or chronic stressors can lead to oxidative stress and a cascade of physiological effects (Aschbacher et al., 2013). Oxidative stress can cause chronic inflammation and insulin resistance (Evans et al., 2005). Critically, there is empirical evidence to suggest that chronic stress may interact with sugar consumption to shape biological risk factors for depression.

Specifically, a landmark study by von Dawans and colleagues (2021) found that people who had high glucose in their system showed a greater blood sugar increase in response to stress than people who had low glucose in their system before a stressor (von Dawans et al., 2021). This effect was driven by the stress-induced decrease in insulin, which helps to capture and store excess glucose in the blood stream (von Dawans et al., 2021). In an earlier study that examined how cortisol and epinephrine affect how the body handles sugar, healthy participants received either cortisol, epinephrine, or saline infusions before and after consuming glucose. Aligning with the previous study, findings showed that blood sugar spiked more after the cortisol infusion and epinephrine infusions than the saline control group (Waldhäusl et al., 1987). The relationship between blood sugar and stress hormones has also been examined in non-human animals. In dogs, for example, glucagon, epinephrine, and cortisol were found to work together to rapidly increase blood sugar, particularly when all three were present (Eigler et al., 1979). And, in a study of 251 people with acute coronary syndromes, higher levels of cortisol were linked to more severe coronary events and elevated blood sugar (Stubbs et al., 1999). Given that insulin resistance and chronically high blood sugar are well-established risk

factor for depression (Kan et al., 2013), a reexamination of the relationship between sugar consumption and depression that explores the potential interaction effect of chronic stress is warranted. We proposed that chronic stress and sugar consumption could biologically interact to predict depressive symptoms by amplifying the effects of added sugar consumption on depression through high blood glucose and insulin resistance.

Despite the plausibility that stress could interact with sugar consumption to predict depression risk, very few studies have investigated the effects of stress as a factor in the relationship between sugar and depression. Moreover, those that have included stress in the model focused on stress as a precursor to eating behavior rather than as a biological interaction. Some previous research has found that stress leads to the consumption of comfort foods, which are often high in added sugar (Tomiyama et al., 2015; Torres & Nowson, 2007). While we do not disagree that stress is likely a predictor of how sugar can enter the body, this relationship does not preclude the possibility that chronic stress could interact with added sugar consumption. Interactions like stress and added sugar consumption are especially important to test given the fact that the results of added sugar consumption and depression studies are mixed, with some studies finding null results in some populations while other studies report significant positive associations. Therefore, the current studies aimed to investigate whether chronic stress interacts with sugar consumption in predicting depressive symptoms. To test this model, we conducted two studies—the first examined this model in an online sample of adults and the second tested the relationship in mothers followed during pregnancy into the postpartum period.

We hypothesized that added sugar consumption would positively predict depressive symptoms as well as amplify the deleterious effects of chronic stress on mental health risk.

Chapter 2

1. Introduction

1.1 Stress and Depression

Decades of research has established a robust relationship between life stress and depression (Hammen, 2005; Kessler, 1997). A meta-analysis of 62 articles and 44,066 participants found that people who experienced early life stress were more likely to develop major depressive disorder before the age of 18 than those without a history of early life stress (LeMoult et al., 2020). In addition to early life stress, various types of stress such as chronic psychosocial stress, ongoing stress, and post-traumatic stress increase depression risk (Charney & Manji, 2004; Morrill et al., 2008; Siegrist, 2008). Further investigation into the mechanisms that drive this relationship uncovered several biological factors (Silva et al., 2021). For example, various genes have been identified in this as risk factors for depression such as polymorphisms in the serotonergic system, which plays a role in regulating stress-linked behaviors, emotions, cognitions and motor functions (Caspi et al., 2003). Another key mediator in the relationship between environmental stressors and depression is the HPA axis, which is a primary system that helps the body adapt and respond to stress. When a stressor is encountered, The HPA axis triggers the release of hypothalamic corticotropin-releasing hormone (CRH) which stimulates adrenocorticotropin (ACTH) release from the anterior pituitary gland which in turn triggers the release of cortisol from the adrenal cortex. Additionally, variabilities in genes are involved in the HPA axis and can mediate the impact of stress on depression risk through genes that effect stress including the Brain-Derived Neurotrophic Factor

(BDNF) promoter gene, and CRHR1, which encodes a CRH receptor (Grabe et al., 2010; Hing et al., 2018; Kundakovic et al., 2015). It is not just stress that is encountered in adulthood, but early life stress can affect the HPA axis as well, for example, adverse childhood experiences (ACEs) (Heim et al., 2008). People with ACEs have a higher risk of developing depression (Heim et al., 2008). Severe trauma essentially rewires the HPA response in a way that promotes a chronically activated stress response (Iob et al., 2021). Having a history of ACEs is a risk factor a number of chronic diseases in adulthood including diabetes, suggesting the HPA dysregulation that ACEs initiate have the potential to impair glucose metabolism (Huffhines et al., 2016). Furthermore, there is evidence to suggest that the effect of ACEs can transcend to the offspring of the impacted person. Specifically, maternal daytime cortisol fluctuations moderated the positive relationship between maternal ACEs and child behavioral issues (Thomas-Arguriuo et al., 2021). While it is evident that biological stress related mechanisms contribute to depression risk, less is known about actionable ways to mitigate the risk of these often-unavoidable stressors from developing into depression. One promising avenue that may help reduce the risk of stress increasing depression risk is diet.

1.2 Food and Depression

As the field of health psychology has grown, research has increasingly focused on how specific nutrients affect mental health. Not only has overall diet quality been linked to psychological outcomes, but different types of diets and specific nutrients are emerging as predictors as well (Lassale et al., 2018). A study by Sanchez-Villegas and colleagues (2006) found that following a Mediterranean, which diet rich in fruits and vegetables, cereals, fish, and legumes was inversely associated with depression (Sánchez-Villegas et

al., 2006). More recently, Bayes, Schloss, and Sibbritt (2020) identified that this effect may be partially attributed to the polyphenols content, which the Mediterranean diet is typically rich in (Bayes et al., 2020). Jacka and team (2011) identified that a traditional Norwegian dietary pattern, which consisted of seafood, fruits and vegetables, dairy, margarine, bread, pasta, rice, meats, legumes, and eggs, also predicted better psychological outcomes (Jacka et al., 2011). A Japanese dietary pattern high in fruit, vegetables, mushrooms, and soy products has also been associated with fewer depressive symptoms (Nanri et al., 2010). The diets studied have several commonalities including being low in added sugar, which is largely regarded as an empty calorie food devoid of essential nutrients.

As SSBs are the largest source of added sugar and overall calories in the US diet, SSB consumption is often used as an estimate for added sugar in research (Hu & Malik, 2010). In the meta-analysis by Hu and team, 6 out of the 10 examined studies found a relationship between SSB and depression (Hu et al., 2019). The earliest published study included in the meta-analysis was a cross-sectional study that included 4,741 Australians age 16 and older and used soft drinks as the SSB variable (Shi et al., 2010). They found a statistically significant positive relationship between soft drink consumption and depression, stress-related problems, suicidal idealization, psychological distress, and having a current mental health condition (Shi et al., 2010). The next two studies in the meta-analysis were cohort studies from the NIH-AARP Diet and Health Study. These studies consisted of 263,923 participants in the US and measured both soft drinks and fruit drinks as independent variables and used physician-diagnosed depression as the outcome. Interestingly, the relationship between soft drink consumption and depression

was statistically significant, but the relationship between fruit drink consumption and depression was not (Guo et al., 2014), suggesting frequent soft drink consumption may be particularly harmful to mental health. Another cross-sectional study of 13,486 Iranian children and adolescents found that soft drink consumption had a statistically significant positive relationship with self-reported psychiatric distress (Zahedi et al., 2014). Further, a cross-sectional study of 3,667 adults in China found a statistically significant positive relationship between soft drink consumption and the Self-Rating Depression Scale (SDS), a self-reported depression scale (Yu et al., 2015). In Brazil, A cross-sectional study based on 49,025 adults found that soft drink and artificial juice consumption predicted higher scores on the Patient Health Questionnaire (PHQ-9), a scale that measures depressive symptoms (Barros et al., 2017). However, a case-control study of 1,351 Chinese adults that measured sugar consumption with a composite that included sugared beverages and sugared snack consumption in adults found no relationship between the dietary variables and scores on a depression scale (Xia et al., 2017). However, the study did find that an overall "sweets pattern" was positively associated with depressive symptoms (Xia et al., 2017). Another study with null findings was a cohort study of 15,546 Spanish adults that found no relationship between sweetened drink consumption and physician-diagnosed depression (Sanchez-Villegas et al., 2018). Finally, Hu and team's meta-analysis included a two-part series in the UK adults that measured sweet food, beverages, and depression scores using the CES-D. The first was a cross-sectional study that showed positive associations between sugar consumption and depressive symptoms. The second was a cohort study that showed that after 5 years,

while the risk of depression was positively associated with sugar consumption, it was not statistically significant (Knüppel et al., 2017).

To summarize these results, the majority of studies find a significant positive correlation between SSB consumption and depression, although most diet and depression studies are cross-sectional (Wang & Cheng, 2020). The greatest weakness of cross-sectional data is the inability to establish causality, although to try and address this weakness virtually all studies on diet and depression control for potentially confounding factors relating to sex, age, body mass index, socioeconomic status, physical activity, and various health conditions such as alcoholism (Hu et al., 2019b). While these studies cannot establish causality, their cross-cultural nature, as well as their findings being dependent on the type of sweetener in foods, suggests there is reason to believe that a causal relationship may exist between frequent added-sugar consumption and depression. The current study explored stress and added sugar consumption as a novel potential interaction in this relationship longitudinally in an attempt to overcome some of the limitations that exist in many of the existing studies.

1.3 Foods and Stress

Stress is associated with altered eating behavior such as more frequent consumption of sweets and fast food, and less frequent consumption of fruits and vegetables (Mikolajczyk et al., 2009). These findings are, in part, due to comfort eating, a behavioral coping response (Finch & Tomiyama, 2015). When faced with similar levels of stress, comfort eaters experienced reduced perceived stress compared with those who did not engage in comfort eating (Finch & Tomiyama, 2015).

The relationship between stress and eating is complexified by the addictive nature of comfort foods such as those with high sugar content. Stress is a key factor in the development of addiction. Uncontrolled stress changes eating patterns by increasing hyperpalatable foods (e.g. comfort foods high in sugar and fat) which can increase allostatic load and metabolic disease over time (Yau & Potenza, 2013). This is because food exploits similar pathways in the brain to drugs by engaging the dopaminergic system (Lustig, 2010). Subsequently, a cyclical relationship emerges that begins with stress leading to comfort eating, which leads to obesity, metabolic disease, and ultimately, increasing stress (Tomiyama, 2019). Building on our understanding between stress and health outcomes, the current study aimed to explore the complex interplay between added sugar consumption, stress, and depressive symptoms.

1.4 The Current Study

The goal of the current study was to investigate the relationship between added sugar consumption and depression, and to examine an interaction relationship between added-sugar consumption, stress, and depressive symptoms. We hypothesized that 1) Added sugar consumption would be positively associated with depressive symptoms, and 2) That stress can interact with sugar consumption and depression risk in adults, amplifying the deleterious effects of sugar consumption on mood. Specifically, we predicted that people who eat diets high in sugar and experience higher levels of chronic stress would report high scores on a scale measuring depressive symptoms than those who consume high levels of sugar or experience high levels of stress alone. To test this interaction model, we recruited US adults to complete two online surveys. The survey included questionnaires for diet, stress, and depressive symptoms.

2. Methods

2.1 Participants and data collection

Adults from the US, UK, Australia, and New Zealand, over the age of 18 were invited to participate in an online survey on the effects of diet and stress on wellbeing and repeated the survey after 1 month. Surveys were offered in English in February and March 2024. Participants were recruited through Prolific. Adults were eligible if they were over the age of 18, lived in the United States, the United Kingdom, Australia, or New Zealand, and spoke English. Prolific participants received a payment of $2 upon completion of the survey at baseline, and $3 upon completion of the survey at the follow up, 1 month later. The survey took approximately 10-minutes to complete at each time point. To make sure all participants were humans (not bots), the study included one short answer question asking participants to speculate what the study was about. Participants who failed to answer the question appropriately were removed from analysis. Given that this was the first study to examine the interaction of sugar consumption, stress, and depression, a conventional power analysis using effect sizes from previous findings was not possible. Therefore, in line with the commonly used recommendation by Brysbaert (2019) for detecting clinically significant results we aimed to collect a sample of 250 participants. This number was picked in anticipation that 15% of the data may not be useable due to incomplete responses or participant inattention. This would leave a final sample of 212 participants, giving us over > 80% power to detect and the recommended interaction effect size of Cohen's D of 0.40 (Brysbaert, 2019). Ultimately, a total of 250 participants completed data collection at baseline. Three participants were removed for failing the attention check question and 29 were removed for not participating in the second survey.

One participant that successfully completed the first wave failed to complete the follow-up survey, resulting in 217 participants included in the study.

2.2 Depressive symptoms

Depressive symptomology were assessed via the brief version of the Center for Epidemiologic Studies – Depression scale (CES-D), a 20-item self-report scale designed to measure depressive symptoms in the general population with higher scores indicating higher levels of depressive symptoms (Radloff, 1977). The prevalence of depressive symptoms in this sample was relatively high (M = 39.456, SD = 12.741). We planned to conduct analysis continuously to capture degree of depressive symptoms.

2.3 Measures of stress

The assessment of stress was made by administering two chronic stress scales. The Adverse Childhood Experiences (ACEs) scale was used to assess the degree to which an individual has experienced childhood trauma (Finkelhor et al., 2015). Participants responded to 14 items on a dichotomous scale, and scores were obtained by assigning each item a point if present (1) and none if absent (0). Items were then summed into an ACEs index that ranged from 0 to 14, with higher scores indicating more adverse childhood experiences (Finkelhor et al., 2015). In addition, chronic stress was measured using the 9-item Trier Inventory for Chronic Stress (TICS-EN-9), which uses a five-point scale to assess how often participants had encountered specific chronic-related stress situations or had specific experiences within the previous 3 months. Results were scored on a 4-point scale on a five-point scale (0 = never, 1 = rarely, 2 = sometimes, 3 = often, 4 = very often)(Petrowski et al., 2019).

2.4 Measures of Added Sugar Consumption

Participants' dietary consumption was measured using items from the food frequency questionnaire of the Growing Up in New Zealand study (*GUiNZ)*. Participants answered questions on how many servings of each dietary item they ate or drank over the past 4 weeks. The food items used to reflect sugar consumption were: ice cream; cakes or biscuits/cookies; soft drinks or energy drinks excluding diet drinks, fruit juices, flavored waters, and sports waters; confectionary, lollies/lollipops/suckers, sweets, and chocolates. We created a composite variable using all the items containing added sugar to measure total sugar consumption (M = 4.49, SD = 1.49) and also created a separate variable to measure SSB consumption specifically (M = 3.53, SD = 2.16).

2.5 Covariates

The following covariates were included in the model given that previous studies have shown they are related to depressive symptoms, stress, and/or sugar consumption: age, household income, education, sex, fruit consumption, vegetable consumption, and depressive symptoms at baseline.

3. Statistical analysis

We used stepwise linear regressions to assess if total sugar consumption and SSB consumption at baseline predicted depressive symptoms at the 1-month follow up in separate models. Step one included each sugar variable alone, step 2 included the TICS and ACEs and their interaction variables with sugar, step 3 included fruit and vegetable consumption, then participant demographics were added in step 4, and lastly, depressive symptoms at baseline were added in step 5. We used regression interaction terms in SPSS version 29 to test if baseline chronic stress interacts baseline sugar consumption to predict depressive symptoms at the 1-month follow up. For the total sugar model, we

included an interaction variable created by separately multiplying the TICS and ACEs scores by the total sugar composite variable. For the SSB model, the interaction variables were created by multiplying the TICS and ACEs s score variables by the SSB consumption variable. All variables were mean-centered before analysis, and before compositing when applicable, to help with model interpretation. *p* values (<.05) and 95% confidence intervals (CI) that did not overlap with zero were be used to examine whether there was a significant effect of sugar consumption at baseline on depressive symptoms after 1 month.

4. Results

4.1 Demographics

A total of 217 participants completed both data collection waves and were therefore included in this study. Of these participants, 66.8% identified as females, 84.8% resided in the UK, 84.3% were White, and the average age was 60.24 (SD = 11.53, RANGE = 40-93). See table 1 for complete demographic information on the sample. In our sample, 91 people met the CES-D cutoff score for possible depression (i.e., score of 20 (Jiang et al., 2019)), with an overall mean CES-D score of 19.456. The median frequency of total sugar consumption fell between 1-2 times per week and 3-4 times per week, and the mean frequency of SSB consumption was between 2-3 time per month and 1-2 times per week. See table 2 in the appendix for a correlation matrix showing how demographics, sugar consumption, stress and depression related to each other.

4.2 Does total sugar consumption at baseline predict depression at follow up?

Consistent with previous research, people who consumed more total added sugar at baseline reported more depressive symptoms 1 month later than those who consumed less

added sugar at baseline (see table 3 for the full stepwise regression model). This effect was no longer significant when the stress scales and the interaction variable included in the model, when fruits and vegetables consumption were included as covariates, or when demographic variables and CES-D scores at baseline were included as covariates.

4.3 Does SSB consumption at baseline predict depression at follow up?

Similar to added sugar consumption, SSB consumption at baseline predicted more depressive symptoms at the 1-month follow up, an effect that remained significant when the stress scales, interaction variable, and fruit and vegetable consumption were added into the model. This effect became a trend ($p = .068$) when demographic variables were included in the model and became non-significant when depressive symptoms at baseline were included in the model.

4.5 Interactions between sugar consumption and stress

The interaction between added sugar consumption and chronic stress at baseline did not significantly predict CES-D scores at the follow-up in any of steps that we examined. These effects remained nonsignificant when the interaction variables were entered separately.

4.6 Does depression at baseline predict sugar consumption at follow up?

We also wanted to examine the possibility of reverse causality—the idea that depression is causing increases in sugar consumption and that is why you see the positive correlation between sugar consumption and depression. There was a trend so that higher CES-D scores at baseline predicted SSB consumption at follow-up ($\beta = 0.118$, $p = .085$). However, this result became nonsignificant when TICS and ACEs scores at baseline were added to the model ($\beta = 0.060$, $p = .562$). This null result did not change when other

nutritional variables were added to the model, or when demographics variables were added to the model. Additionally, controlling for SSB at baseline did not change the results.

In the total added sugar consumption model, depression at baseline significantly predicted higher sugar consumption at follow-up (β = 0.136, *p* = .047). When stress was included in the model, this effect was no longer significant (β = -.001, *p* = .990). Including other dietary factors, and demographics did not change this pattern of results. Adding total sugar consumption at baseline to the model resulted in a trend so that higher levels of depression at baseline predicted less sugar consumption at follow up (β = -.133, *p* = .061).

4.7 Post hoc test examining if sugar mediates the relationship between stress and depression

We conducted exploratory post hoc analysis using the process macro in SPSS to see if stress predicted higher sugar consumption which, in turn, predicted more depression. In the total sugar model and the SSB model, basic conditions of mediation were not met. Specifically, although stress did predict higher total sugar consumption (β = 0.102, *p* = .018), when stress and total sugar were included in the model, total sugar was no longer a significant predictor of depression (β = 0.051, *p* = .520). In addition, in the SSB model, stress did not predict SSB consumption (β = 0.026, *p* = .704). Moreover, the indirect effects in both models were not statistically significant (SSB: 95% CI [-.0182, .0304]), total sugar: 95% CI [-.019, .0311]).

4.8 concurrent analysis

In the total added sugar model, baseline sugar consumption predicted baseline depressive symptoms (β =.186, $p < .006$). The effect was no longer statistically significant when stress and the interaction variables were included in the model (β = 0.053, $p = .668$). The interaction variables were not significant predictors of depressive symptoms. No other significant predictors emerged.

In the SSB model, baseline SSB consumption predicted baseline depressive symptoms (β = 0.170, $p < .012$). The effect no longer existed when stress and the interaction variables were included in the model (β = 0.176, $p = .114$). The interaction variables were not significant predictors of depressive symptoms. No other significant predictors emerged in this model.

5. Discussion

The goals of the current study were to examine if total sugar consumption and SSB consumption predicted depressive symptoms in the general population, and to investigate whether added sugar consumption and chronic stress interact to increase depression risk. This study found that both total sugar consumption and SSB consumption at baseline predicted depressive symptoms one month later. Although the relationship between total sugar consumption and depression seemed to be explained by stress, the relationship between SSB consumption and depressive symptoms persisted even after controlling for multiple covariates such as stress levels, other dietary factors, and demographics. However, the effect became a trend when baseline income was included in the model, and nonsignificant when CES-D scores at baseline were included in the model. It's important to note that baseline and follow-up CES-D scores were highly correlated ($r = 0.846$, $p < 0.001$) (see table 2 for a correlation matrix of our main

variables). Given that adding the CES-D scores from both timepoints shifts the outcome from reflecting overall depressive symptoms to change in depressive symptoms between the timepoints, the minimal change between baseline and follow-up depression scores makes it difficult to discern if sugar consumption has no effect on depressive symptoms over time, or if the timepoints were too close together to accurately assess an effect. Interestingly, however, we found that changes in total sugar consumption predicted changes in depressive symptoms, and this effect persisted after controlling for demographic variables and changes in stress. This suggests that there was at least some variability in depression between time points and is strong evidence that sugar and depression are related in some way, although the causal nature and the directionality of the relationship has not been established.

While the main added sugar consumption and depression findings of the current study generally aligned with previous findings that added sugar consumption predicts depressive symptoms, the current study did have novel findings when stress was included in the models (Hu et al., 2019). While Heim and colleagues (2008) found that higher ACEs scores predicted an increase in depression risk (Heim et al., 2008), the current study found that with sugar consumption included in the model, TICS significantly predicted depressive symptoms while ACEs did not. Perhaps this is an indication that in the context of nutrition research, current stress is a stronger predictor of depression than childhood traumas. Another explanation is that our sample had relatively low ACEs scores (M = 2.65, SE = 2.06), and it is possible ACEs scores would have been stronger predictors of depressive symptoms in this model in a population that had a higher prevalence of childhood trauma. Comparatively, the average TICS score was moderately

high (M = 15.48, SE = 6.75). Future studies that include various types of chronic stress are warranted.

Multiple studies in the field of diet and depression have found that diets rich in fruits and vegetables are protective against depression (Jacka et al., 2011; Nanri et al., 2010; Sanchez-Villegas et al., 2018). In the current study, fruits and vegetable consumption did not predict depressive symptoms with or without other variables included in the model. While these findings do not mirror all previous research, a longitudinal study by Hoare and colleagues (2018) found that fruit and vegetable consumption predicted depression cross-sectionally in adolescence, but the effect did not persist longitudinally into adulthood. Given that the current study was also longitudinal, our findings are aligned with those of Hoare et al. (2018) (Hoare et al. 2018). One explanation for the mixed findings in fruit, vegetable, and depression research may be that the type of fruit and vegetable consumed determines the effect on depression. Perhaps the fiber content, or specific vitamins in the vegetable are important predictors that are missed when vegetables and fruits are grouped together.

In a previous SSB consumption and depression meta-analysis, 6 of the 10 studies included found a positive relationship between SSB consumption and depressive symptoms (Hu et al., 2019). These findings align with results of the SSB model in the current study, which show that higher SSB consumption predicts more depressive symptoms. However, when income was added to the model, this finding is no longer statistically significant. While income was included as a covariate in 4 of the 6 studies that found a significant relationship (Shi et al., 2010, Zahedi et al., 2014, Yu et al., 2015, & Xia et al., 2017), socioeconomic factors were not included in 2 of the studies (Barros et

al., 2017, & Guo et al., 2014). Additionally, the current study found that stress is a strong predictor of depression even with sugar included in the model, however, none of the studies in the meta-analysis included stress as a covariate. Therefore, it is possible that this oversight in the studies led to an overestimation of the effect of SSB consumption on depression.

Due to this being an observational and not an experimental study, we can only infer the causal directionality based on our models, which find that added sugar consumption predicted depression. Although, while there is strong evidence to suggest that sugar may increase depression risk through biological mechanisms such as inflammation, the gut-brain axis, and disrupted HPA function (Eisenberger et al., 2010; Harrell et al., 2015; Noble et al., 2021), other research indicates that mental states may influence eating behavior and lead to an increase in ingestion of comfort foods, including those with added sugar (Finch & Tomiyama, 2015). Thus, our findings may reflect either how the body processes sugar or eating habits during emotionally challenging times. While our post hoc analyses did find that when people are stressed, they eat more sugar, and when people are stressed, they are more likely to be depressed, total added sugar consumption was not a true mediator in the relationship between stress levels and depressive symptoms. Additionally, we found that change in total added sugar consumption, but not SSB consumption, predicted change in depression. As total added sugar consumption includes foods that could be considered comfort foods, this may reflect the effect of depression on eating behavior, specifically, emotional eating during times of distress. Alternatively, these findings may also reflect the presence of a third variable in the relationship between added sugar consumption and depression that has not

yet been accounted for. This potential unknown third variable may be biological or behavioral in nature and predict how much sugar people consume and how depressed they are. Previous studies have included socioeconomic factors, health behaviors, and existing health conditions as covariates (Guo et al., 2014, Xia et al., 2016, Zahedi et al., 2014). However, that is the extent of variables that are typically considered, therefore, exploratory studies on the subject that include physiological stress and eating behaviors are warranted.

The interaction variables added in step 2 of the stepwise regression had null findings in both measures of added sugar consumption. These results were inconsistent with our hypothesis based on previous research (Von Dawans et al., 2021) that found that people who had high glucose in their system experienced the strongest physiological reaction to stress. Given these findings, we hypothesized that the long-term combination of stress and added sugar consumption would interact to predict depression. A key difference is that our study measured psychological stress, whereas Von Dawans et al. (2021) focused on physiological stress. While it is possible that sugar and chronic stress do not interact to predict depressive symptoms, it is also possible that the interaction exists, but was not captured by self-reported measures of chronic psychological stress. Additionally, the study by Von Dawans and team (2021) was designed to assess an acute stress reaction, whereas the current study is looking at chronic stress. While both acute and chronic stress involve activated the HPA response, and therefore theoretically trigger the release of stress hormones such as cortisol, the long-term activation of the HPA axis may behave differently than one that is suddenly activated by an acute stressor.

5.1 Limitations

The results of this study should be considered in the context of several limitations. As mentioned previously, the short interval between the baseline and one-month follow-up may not have allowed for meaningful variability in depression or sugar consumption levels. Longitudinal studies that include multiple measure of both sugar consumption and depressive symptoms over years are needed to untangle the directionality of this relationship. Additionally, our cohort primarily consisted of white women in the UK with above-average levels of depression (41.94% met the CES-D cutoff for possible depression), limiting the generalizability of our findings to other populations. It is possible we had such high rates of depression give that we advertised the study as a depression study. Given that diets vary widely cross-culturally, it is possible that our findings would have been different if the sample reflected a more diverse population. Furthermore, the use of a self-report screening tool to measure depressive symptoms may not accurately capture clinical levels of depression. Although the CES-D is regarded as reliable, clinical interviews are the gold standard for assessing depressive disorders (Radloff, 1977). Additionally, the current study measured added sugar consumption with a food frequency questionnaire instead of the gold standard method of collecting dietary data via multiple dietary recalls conducted by a trained interviewer. Lastly, it is possible we did not find an interaction effect because there was not enough power to detect it.

5.2 Future Directions

While the current study found that sugar consumption predicted depression one month later, the fact that the effect is no longer significant when baseline depression scores were added to the model underscores the need for a deeper understanding of the biological impact of sugar on depression and the potential role of chronic stress in this

relationship. Future research should incorporate physiological stress measures in studies examining sugar and depression and consider longitudinal designs with longer follow-up periods. Although randomized control trials are the gold standard, a trial assessing sugar, stress, and depressive symptoms in humans would be challenging. An alternative approach could be to conduct a sugar-free intervention in people with depression and include physiological stress and depressive symptoms that are measured before, during, and after the intervention. In this type of design, it would be possible to assess the directionality of the relationship and identifying potential interactions. Incorporating biological variables such as genetic information to either type of design in future studies would be valuable, given that certain genes influence the stress response and metabolism (Caspi et al., 2003, Illig et al. 2009).

6. Conclusion

This study suggests that psychological stress does not interact with sugar consumption to predict depressive symptoms. However, we did find evidence that sugar and depression are positively correlated. Understanding whether added sugar consumption is a biological risk factor for depression is crucial. If so, it could serve as a target for interventions preventing a disorder that affects millions in the US alone. Mixed findings in the literature, along with those of this study, suggest a positive overall relationship between added sugar consumption and depressive symptoms, though the exact nature of this relationship remains unclear. Further research is needed to determine the directionality of this effects and to test whether physiological stress interacts with sugar consumption in predicting depression.

Chapter 3

1. Introduction

Postpartum depression (PPD) is the most prevalent mental health condition associated with childbirth (O'hara & Swain, 1996). PPD is closely related to major depression. Not only do PPD and major depression drastically overlap in symptoms, but depressive symptoms outside the postpartum period are the strongest predictors of PPD (Agrawal et al., 2022.; Beck, 2001). In fact, in the *Diagnostic and Statistical Manual of Mental Disorders (DSM-V)*, PPD is considered when depressive symptoms onset in the postpartum period, and it is not listed as a separate disorder (American Psychiatric Association, 2013). PPD is not only detrimental to mothers' mental health, but it can also interfere with maternal-child bonding and parenting behavior, and lead to a multitude of adverse physical and emotional child developmental outcomes (Burke, 2003; Miranda & Patel, 2005; Oates, 2003). Children with depressed mothers, for example, are more likely to experience psychopathology, behavioral issues, and have lower academic achievement (Flynn et al., 2017; Goodman et al., 2011; Pearson et al., 2016). The adverse impact of maternal depression on offspring may persist into adulthood, raising lifelong risk for mental health, behavioral, and relational issues throughout the lifespan (Gilliam et al., 2015; Nonnenmacher et al., 2016; Pearson et al., 2013). In this cyclical way, mental health issues can pass down through generations, highlighting the need to understand and prevent maternal mental illness.

Previous research suggests that dietary factors can impact postpartum depression risk, and high sugar consumption has been found to be particularly problematic (Fish-

Williamson & Hahn-Holbrook, 2023; Ker et al., 2021). For example, in Taiwan, mothers who scored high for SSB dependence on a substance abuse scale were more likely to score higher on the EPDS, the gold standard for screening for PPD, compared to mothers who showed less dependence on SSB (Ker et al., 2021). Similar findings persisted on a global level such that in a meta-regression of 412 studies in 46 countries, more country level SSB consumption predicted higher national postpartum depression prevalence (Fish-Williamson & Hahn-Holbrook, 2023). Moreover, in years when countries consumed more SSB, those same countries had higher rates of PPD (Fish-Williamson & Hahn-Holbrook, 2023). The results of the studies on PPD and sugar consumption mirror conclusions drawn from research done outside of the postpartum period, which has also found that sugar consumption increases depression risk generally (Sanchez-Villegas et al., 2018). Several possible mechanisms have been postulated to explain the relationship between sugar consumption and postpartum depression risk. First, research has shown that sugar can have adverse effects on the hypothalamic-pituitary-adrenal (HPA) axis and increase IL-6 and proinflammatory cytokines, which can cross the blood barrier and have been shown to increase depressive symptoms (Eisenberger et al., 2010). Pregnancy drastically alters the HPA axis such that cortisol levels are three times higher than normal levels by the third trimester (Duthie & Reynolds, 2013), with stress exacerbating these HPA alterations in pregnancy (Duthie & Reynolds, 2013). Moreover, given that pregnancy puts incredible strain on the body's blood glucose and HPA axis regulation systems, the effect of high sugar consumption on the HPA axis could potentially be intensified during pregnancy. Furthermore, it is probable that the effect of HPA

dysregulation in pregnancy persists into the postpartum period, raising the risk for postpartum depression (Glynn et al., 2013).

Similarly, research shows that stress is a major risk factor for PPD (Beck, 1996). This relationship is true for many types of chronic stress, including life stress, psychosocial stress, child-care stress, pregnancy-specific stress, and the stress of acculturation (Bashiri & Spielvogel, 1999; Beck, 1996; Caparros-Gonzalez et al., 2017; Katon et al., 2014). Biological processes of stress, including HPA dysregulation, cortisol levels, and inflammatory processes, have all been linked in previous research to predict PPD (Caparros-Gonzalez et al., 2017; Yim et al., 2015). For example, in a study of 44 pregnant women, hair cortisol levels predicted 21.7% of the variance of postpartum depression symptoms (Caparros-Gonzalez et al., 2017).

No studies to our knowledge have looked at how stress and sugar may work in tandem as risk factors for PPD. Despite this, biologically, stress can lead to high blood sugar and promote inflammation, both of which in combination with high sugar consumption could lead to prolonged elevated blood sugar, a risk factor for depression (Lustman et al., 2000). A study of 151 males found that people who have sugar in their system show an increase in cortisol release when exposed to stressors compared to people who do not have sugar in their system (von Dawans et al., 2021). Given these findings, it is plausible that the combination of stress and high sugar consumption would increase the risk of depression compared to each predictor alone (von Dawans et al., 2021).

The goal of the current study was to investigate whether added sugar consumption predicts PPD levels, and if there was an interaction relationship between added sugar consumption, stress, and PPD symptoms. We hypothesized that chronic stress would

amplify the deleterious effect of high added sugar consumption on postpartum depression symptoms such that the positive relationship between high added sugar consumption and depression would be stronger under conditions of high chronic stress relative to low chronic stress. To test this interaction model, we used a large nationally representative dataset from New Zealand to test whether diet and stress during pregnancy work together to predict mothers' PPD risk at 9 months postpartum.

2. Methods

2.1 Participants

This study utilized data from the Growing Up in New Zealand (GUiNZ, *Growing Up in New Zealand*) longitudinal cohort study. A cohort of 6,853 pregnant mothers were enrolled between 2009 and 2010. The GUiNZ team collected data in the last trimester of pregnancy, and postpartum at 9 months, 2 years, and 4.5 years of child age. Pregnant women were recruited by GUiNZ recruiters or health professionals in medical and community environments (Morton et al., 2014). The sample enrolled was representative of the New Zealand population on key sociodemographic and ethnic characteristics. As such, this sample is ethnically diverse, with the top five represented ethnicities being 60.9% European, 12.8% Māori, 11.6% Asian, 11.4% Pacific, and 1.6% Middle Eastern, Latin American, and African. Full details about the GUiNZ study are reported in Morton et al. (Morton et al., 2014).

All GUiNZ participants who had prenatal and 9-month data available were be included in the analyses for the current study. Prenatal data were not available for 2.2% of the GUiNZ sample and complete 9-month data were not available for 6%. While study retention at 4.5 years was 90% of the baseline, the missing data can be largely attributed

to time limitations during interviews, participants skipping one of the waves of data collection, and the participants' ability to opt out of answering certain questions (S. Morton et al., 2017; Peterson et al., 2019). The current study uses data from mothers who completed assessments of prenatal stress and diet during pregnancy and PPD data at the 9-month follow-up.

The GUiNZ study was approved by the Ministry of Health Northern Y Regional Ethics Committee (NTY/08/06/055), and informed consent was obtained from all participants.

2.2 Measures of added sugar consumption

Added sugar consumption was calculated as a composite using items from the food frequency questionnaire completed in the antenatal wave of data collection. Participants answered questions on how many servings of each dietary item they ate or drank over the past 4 weeks. The food items used were ice cream; cakes or biscuits; soft drinks or energy drinks excluding diet drinks, fruit juices, flavored waters, and sports waters; confectionary, lollies, sweets, and chocolates. We created a composite variable for total added sugar consumption (M = 4.11, SD = 1.08) using all the survey items containing sugar to measure total sugar consumption and created a separate variable to measure SSBs (M = 3.52, SD = 1.78).

2.3 Measures of chronic stress

Information on maternal chronic stress was collected at both the antenatal and 9-month data collection wave. Maternal stress during pregnancy was measured using several tools including the 10-item Perceived Stress Scale (PSS) (Cohen et al., 1983), the 9-item Warmth and Hostility Scale (Melby et al., 1993), the 6-item Conflict scale (Pryor, 2004)), and a 6-item Family Stress scale specifically designed for the GUiNZ study.

Environmental stress levels were collected from the items on external support from the Family Support Scale (Dunst, 1984). Higher scores on all scales except for the PSS indicate a lower-stress environment. We investigated if the scales hang together using principal component analysis (PCA). Scales that fit together were z-scored and averaged to create a chronic stress composite.

2.4 Measures of Postpartum depression

PPD was measured using the Edinburgh Postnatal Depression Scale (EPDS), a 10-item self-report, widely used tool specially designed to measure PPD (M = 5.17, SD = 4.53). EPDS scores can range from 0-30 with higher values representing more PPD symptoms (Cox et al., 1987). The EPDS was validated to detect depressive symptoms in the postpartum period, making it more reliable and accurate in detecting PPD than traditional self-report depression tools (Cox et al., 1987).

3. Statistical Analysis

We used stepwise linear regressions to assess if total sugar consumption and SSB consumption during pregnancy, predicted PPD symptoms at the 9-months postpartum. Step one included each sugar variable alone, step 2 included the chronic stress composite and its interaction variable with sugar, fruit and vegetable consumption were added in step 3, then participant demographics were added in step 4, and lastly, EPDS scores at baseline were added in step 5. All analysis was conducted in SPSS version 28 in an online workspace portal provided by the GUiNZ study.

We used regression interaction terms to test if baseline chronic stress interacted with baseline total sugar consumption and baseline SSB consumption to predict

depressive symptoms at the 9-month follow-up in separate models. For the total sugar model, we included an interaction variable created by multiplying the chronic stress score by the total added sugar consumption composite variable. For the SSB model, the interaction variables were created by multiplying the chronic stress score by the SSB consumption variable. All variables were mean-centered before analysis, and before compositing when applicable, to help with model interpretation. Effect sizes and *p* values (<.05) were be used to examine whether there was an effect of added sugar consumption at baseline on depressive symptoms after the 9 month postpartum follow-up. The following covariates were included in the model given that previous studies have shown they are related to depressive symptoms, stress, and/or sugar consumption: maternal education, citrus fruit consumption, non-citrus fruit consumption, leafy green vegetable consumption, non-leafy green vegetable consumption. Given that NZ deprivation Quantile is a measure of socioeconomic status and is included in the chronic stress composite, and 60.1% of participants were missing income data, we chose to exclude income as a covariate.

4. Results

4.1 Distinction of relevant chronic stress scales by PCA

We conducted PCA to determine which of the chronic stress scales could be combined to create a chronic stress composite. The correlational matrix showed that no variables were correlated over .80, suggesting that each scale measured a unique aspect of chronic stress. The scree plot suggested that a one factor structure was the best fit. The factor loading of the Family Support Scale was poor, and so therefore was removed. After removing the Family Support Scale, the remaining seven components were retained for rotation,

accounting for 71% of the total variance in our dataset. The chronic stress composite variable was therefore made up of seven components: Warmth and Hostility Scale, Perceived Stress Scale, Verbal Conflict Scale, Physical Conflict Scale, Family Stress Scale, NZ Deprivation Quantile, and the variable "It is safe to walk around the [neighbourhood] at night?".

4.2 Preliminary analysis

Mothers were included in this study if they had stress, diet, and depression data. Of the 6832 women who participated in the original GUiNZ study, 4866 were included in the current study. A total of 1966 participants were lost due to missing data on the following: diet (n = 1346), stress (n = 1018), or PPD (n = 75). The majority of participants that were missing dietary data were also missing stress data. Table 4 presents the demographic information of this cohort. See table 6 for a correlation matrix showing how demographics, stress, sugar and depression data correlated in this study. The mean EPDS score at 9 months postpartum was 6.04 (SE = 4.857), and 16.67% of the sample scored above the depression cutoff of 10. The average SSB consumption was between 1-2 times per day, and the average total sugar consumption was between 5-6 times per week and 1 time per day. The mean chronic stress composite score was 9.9 with a standard deviation of 2.409.

4.3 Total added sugar consumption in pregnancy predicting PPD

Consistent with the results of chapter 2, women who consumed more total added sugar during pregnancy reported more depressive symptoms at 9 months postpartum compared to women who consumed less added sugar during pregnancy (See table 7 for the results of the full stepwise model). This effect remained significant when the chronic stress

composite and the interaction between stress and sugar consumption were included in the model. Likewise, the effect persisted when fruits and vegetables consumption and maternal education were included as covariates. However, when EPDS scores during pregnancy were included in the model, total sugar consumption no longer predicted depressive symptoms at 9 months postpartum.

4.4 SSB consumption in pregnancy predicting PPD

Mirroring added sugar consumption, SSB consumption in pregnancy predicted more depressive symptoms at 9 months postpartum, an effect that remained significant when the chronic stress composite and interaction variable, fruit and vegetable consumption, and maternal education were added into the model. This effect did not remain significant when EPDS scores during pregnancy were included in the model.

4.5 Interactions between sugar consumption and stress in pregnancy

The interaction of total sugar and chronic stress predicted depressive symptoms, but the interaction of SSB and chronic stress did not. See figure 1 for the interaction between total sugar and chronic stress on PPD. In the opposite direction than we predicted, mothers who ate *less* sugar in pregnancy had a stronger relationship between stress and PPD compared to women who ate *more* sugar.

5. Discussion

The goals of the current study were to examine if total sugar consumption and SSB consumption in pregnancy predicted depressive symptoms in a population of postpartum women, and to investigate whether added sugar consumption and chronic stress in pregnancy interact to increase PPD risk. Similar to Study 1, the current study found that both total sugar consumption and SSB consumption at baseline predicted

depressive symptoms at follow up, but the effect was no longer significant after adding depression scores at baseline to the model, indicating added sugar consumption does not predict change in depressive symptoms. Study 2 demonstrated that this predictive relationship extends beyond one month and includes postpartum depression.

Our findings in the GUiNZ cohort were consistent with previous findings in Taiwan that showed SSB consumption predicted EPDS scores (Ker et al., 2021). While the current study is a diverse reflection of the New Zealand population, it is limited to residents of New Zealand and therefore is not necessarily generalizable to other global populations. Given that other research has found similar findings in other cultures gives credence to the possibility that this relationship may be true in other populations as well. Additionally, both studies are consistent with the cross-national meta-analysis by Fish-Williamson & Hahn-Holbrook (2023) which found that added national sugar consumption predicts national rates of PPD across countries (Fish-Williamson & Hahn-Holbrook. 2023).

In the current study, while the relationship between SSB consumption and PPD in Step 1 was stronger than that between total sugar consumption and PPD, both types of sugar consumption remained a significant predictor of PPD until depressive symptoms during pregnancy was entered. Unfortunately, the 9-month data did not include diet, and therefore we could not assess reverse causality by examining if depressive symptoms during pregnancy predicted sugar consumption postpartum. Given that EPDS scores in pregnancy and EPDS scores at 9 months were moderately correlated (r = .452, p <.001), it is possible the scores between the timepoints were too similar for an effect to reflect change in depressive symptoms. Having a history of depression is one of the stronger

predictor of PPD (Agrawal et al., 2022), which may have drowned out any possible diet-related effects. Although other longitudinal studies that examined added sugar consumption and depressive symptoms exist, to our knowledge, no previous studies have controlled for depression at baseline.

While both total sugar and SSB consumption predicted depressive symptoms, the effect size decreases considerably after controlling for chronic stress (from $\beta = .097$, $p < .001$ to $\beta = .04$, $p = .008$ in the total added sugar consumption model and $\beta = .129$, $p < .001$ to $\beta = .05$, $p < .012$ in the SSB model). Given that the NZ Deprivation Quantile is included in the chronic stress composite, it is possible that the substantial decrease of the effect size in SSB is reflective of socioeconomic status. Specifically, low socioeconomic status is a predictor of higher SSB consumption for reasons such as affordability, and lack of access to safe drinking water (Bolt-Evensen et al., 2018; Vercammen et al., 2018). Given that our chronic stress composite included psychological stress as well as socioeconomic status related stress, it is hard to know which one had a stronger effect.

Previous research has found that chronic stress is a major risk factor for PPD, and that this relationship is consistent across many types of stress such as pregnancy, family, and life stress (Bashiri & Spielvogel, 1999; Beck, 1996; Caparros-Gonzalez et al., 2017; Katon et al., 2014). This is consistent with our findings that the stress composite, which included family, life, and various other stressors, is a strong predictor of PPD. study also adds to the literature that stress remains a strong predictor with dietary factors added into the model. In addition to predicting PPD, stress also predicts consumption of comfort foods, which is typically sweet and high in added sugar (Tomiyama, Dallman, & Epel, 2011). In fact, consuming comfort foods may offer some protection against chronic

stress. An animal study found that rodents that ate "comfort" foods developed more mesenteric fat, which reduces HPA axis activity, therefore reducing the rodents' stress response (Pecoraro et al., 2004). This effect was examined cross-sectionally in humans, when 59 women were exposed to an acute stressor in a lab and underwent cortisol and dexamethasone assessment. The researchers found that the women with highest level of stress had greater BMI and sagittal diameter, reported greater emotional eating, and had a blunted physiological stress response compared to women with lower stress, mirroring the findings of the rodent research (Tomiyama, Dallman, & Epel, 2011). Given that added sugar consumption may alter HPA functions both in the short-term through added sugar consumption causing high blood sugar, leading to a stronger HPA axis response, and in the long-term through comfort food consumption leading to increased body fat that can reduce HPA axis response, assessing depressive symptoms alongside physiological stress and sugar consumption is key when attempting to understand the effects of added sugar consumption and stress on PPD. Furthermore, while increased body fat may be beneficial in reducing HPA axis response, obesity increases the risk of developing PPD, suggesting that although added sugar consumption may indirectly reduce stress, the subsequent weight gain may negate protection against PPD (LaCoursiere et al., 2010).

Relatedly, the current study found that added sugar consumption was less predictive of PPD when chronic stress was included in the model than without it. While most studies examining sugar and depressive symptoms account for covariates such as socioeconomic status and health conditions, to our knowledge, no studies include stress levels in the analysis, which may lead to an overestimation of the direct effect. Our findings suggest that to accurately capture the relationship between sugar and PPD, stress

should be statistically adjusted for, especially given the fact that stress predicts sugar consumption and could act as a third variable. Notably, sugar consumption is still a statistically significant predictor of PPD when controlling for stress, indicating that while the effect is stronger for chronic stress, added sugar consumption is a predictor independently from stress.

In the current study, the combination of total sugar and stress interacted to predict PPD with a very small effect size. Interestingly, this interaction was in the opposite direction of our hypothesis, suggesting that consuming more (compared to less) total sugar during times of stress in pregnancy predicted lower rates of PPD. Due to the large sample size, such a small effect may not be meaningful, particularly because it was not found in the SSB consumption counterpart. If such an interaction existed, it would be expected to remain consistent across both total sugar consumption levels and SSB consumption. However, if this finding is a true interaction, one potential explanation could involve coping. It is possible that using sugar consumption as a coping mechanism during stressful times could be effective in the short term in elevating mood and reducing subjective symptoms of depression. Given that this interaction was found in the total sugar measure but not in SSB, it is possible that the finding is reflective of emotional eating behavior. Stereotypically, comfort foods are an indulgent or nostalgic food such as cake or cookies, which is better aligned with the total sugar measure than SSB. Another post hoc interpretation is that given that pregnancy is a metabolically taxing period, some pregnant women may require more sugar than others. Therefore, a currently untested possibility is that there may be a potential biological mechanism behind pregnant women

with higher blood sugar being more physiologically resilient to stress, but this explanation should be tested cautiously.

5.1 Limitations

Due to the observational nature of this study, we were unable to determine the directionality of the effect of added sugar consumption on PPD. This limitation means we cannot confidently determine whether sugar consumption biologically promotes the development of PPD or whether women prone to PPD consume more sugar during pregnancy. Similar to Study 1, the current study's stress composite measured psychological stress rather than physiological stress. Since the foundation of our hypothesis regarding added sugar consumption and chronic stress interacting was based on the body's biological response to stress, an ideal stress measure would more effectively capture physiological stress. Another limitation is that EPDS scores in pregnancy and at 9 months postpartum were moderately correlated (see table 6 for correlation matrix), making it difficult to assess change in depression levels. Additionally, we used a self-report screener to assess PPD instead of conducting clinical diagnosis interviews, and a self-report food frequency questionnaire instead of the gold standard of conducting multiple dietary recalls lead by a trained interviewer.

5.2 Future Directions

Future research on added sugar consumption and PPD is warranted, particularly studies that consider physiological stress measures as covariates, given the evidence that biological stress may predict PPD levels. Longitudinal studies examining the interaction between total sugar consumption and chronic stress predicting long-term depressive symptoms would provide a clearer picture of how to interpret the statistically significant

interaction found in the current study. Given the unique metabolic needs of pregnancy, research on how these may influence added sugar consumption is recommended.

6. Conclusion

This study adds to the literature on sugar and depression by examining PPD. Findings suggest that added sugar consumption in pregnancy predicts PPD levels at 9 months postpartum. The current study found a small interaction between added sugar consumption and chronic stress in pregnancy predicting PPD rates at follow-up, suggesting the possibility of sugar consumption as a coping behavior or a pregnancy-related stress protective mechanism. Further, research that includes data points on depression levels and added sugar consumption collected prior to pregnancy is essential for a deeper understanding of the relationship between sugar and depression relationship, as well as the interaction effects. Specifically, it is recommended that studies include longitudinal physiological stress measures such as cortisol, given these may be closer approximations of the biological processes that underly depression, as well as pre-pregnancy dietary and depression data.

Chapter 4

1.1 Final discussion

We conducted two studies to examine if added sugar consumption predicted depressive symptoms, and whether added sugar consumption and chronic stress interact to heighten the effect of this relationship. Study 1 revealed that, in a general population, people who consumed more added sugar, measured as total sugar consumption and SSB consumption, had an increased risk for depressive symptoms. Study 2 revealed the same finding was true for expectant mothers using a longer follow-up period, total sugar consumption in pregnancy predicted PPD and remained significant when controlling for all covariates except depressive symptoms during pregnancy. In addition, the total sugar consumption and chronic stress interaction variable in pregnancy (Study 2) was statistically significant. This suggests that sugar consumption might offer psychological protection against PPD, at least in the short term, in contexts of high stress.

Both studies 1 and 2 found that SSB has a bigger effect size in predicting depressive symptoms than that of total sugar consumption. SSB's often have low nutrient density and are almost entirely sugar. On the other hand, total sugar consumption could include a wider range of nutrient density, particularly if the foods consumed are of high nutrient quality. For example, a homemade banana loaf would be counted as cake on the food frequency questionnaire, and it would likely have added sugar, but that amount of sugar compared to a serving of soda would possibly be lower, and the banana loaf would also have protein from the egg, and fiber and potassium from the banana. An additional consideration is that foods high in fiber take longer to metabolize, therefore slowing

down the release of sugar in the system and potentially altering its effects on the body (Capuano, 2017). Overall, the total sugar consumption measure may be less predictive of depressive symptoms because it captures foods that can be nutritionally dense enough to offset some of the negative effects of the sugar.

Study 1 examined two stress scales separately and found that with sugar consumption included in the models, higher scores on the Trier Inventory for Chronic Stress were strong predictors of depressive symptoms, while the Adverse Childhood Experiences scale scores were not. Perhaps this discrepancy indicates that certain stressors are more highly associated with depression in the context of nutrition research. Without sugar variables in the model, both scales in study 1 were statistically significant predictors of depression. Although the chronic stress composite used in study 2 could not show the effects of each type of stressor individually, the overall effect of the stress composite was substantial in both the total sugar consumption model ($\beta = 0.382$, $p < .001$) and the SSB model ($\beta = 0.375$, $p < .001$). All the scales used in the chronic stress composite measure current stressors, and therefore align closer with the TICS used in study 2, while the ACEs measures past traumas. These findings indicate that when assessing the effects of diet and stress on depression, current psychological stressors may be stronger predictors of depression than past traumas.

Among the covariates used in both studies, the only variable that consistently predicted depressive symptoms at the follow up was depressive symptoms at baseline. In study 1, the month between baseline and follow up was not long enough to see drastic changes in depression levels, leading to a high correlation between CES-D scores at baseline and at follow up ($r = 0.846$, $p < 0.001$). In study 2, depressive symptoms during

pregnancy were moderately correlated with PPD at 9 months ($r = 0.452$, $p < 0.001$), indicating that findings for study 1 may have been different if the data collection timepoints had been farther apart. Additional longitudinal and lifespan studies in the field that include baseline depressive symptoms as a covariate would allow for better understanding of the relationship between added sugar consumption and change in depression.

Given that, across both studies, stress was a strong predictor of depression even with added sugar consumption included in the model, this study highlights the importance of including stress as a covariate in all added sugar consumption and depression studies. While many of the aforementioned studies controlled for demographic and socioeconomic variables, none of the previous studies included a stress scale. Due to this discrepancy, it is possible that the relationship between added sugar consumption and depression is largely overestimated in the literature.

Although the interaction finding between total added sugar consumption and chronic stress was statistically significant in study 2, we believe it should be interpreted with caution. Given that the interaction found was small, particularly for a relatively large sample, the finding may not be meaningful ($\beta = -0.052$, $p < .001$ for total sugar consumption compared to $\beta = -0.035$, $p = .079$ in SSB consumption). In addition, the SSB model found a trend but not a find significant interaction, which would be expected considering that SSB is a slightly stronger predictor of PPD than total sugar consumption on its own. Additionally, the interaction findings were not consistent across study 1 and 2, suggesting that if it is a true finding, it is not homogenous across populations.

1.2 Limitations

While both study 1 and 2 found that added sugar consumption predicted depressive symptoms, neither study was able to assess the directionality of the relationship. The hypothesis that the current studies were designed based on suggests that the biological effects of sugar consumption promote depressive symptoms, however it is possible that our findings reflect depression changing eating behavior. Given that higher depressive symptoms predicted a trend of less sugar consumption over time in the total added sugar consumption model and in the SSB consumption model when baseline sugar consumption was statistically controlled (total added sugar consumption: $\beta = -1.884$, $p < .061$, SSB: $\beta = -1.836$, $p < .068$), results suggest that depression at time 1 predicts *less change* in sugar consumption over time, and that the direction of the relationship seems to be more nuanced than depressed people are simply eating more sugar. Instead, results indicate a complex relationship in which added sugar consumption predicts more depression overall, however, people that are already depressed ate less sugar over time than those with fewer depressive symptoms.

A second limitation in both studies is the homogeneity of the samples. Study 1 largely consisted of white women from the UK, while study 2 was a sample of postpartum women in New Zealand. Given that sugar consumption and stress levels vary widely cross-culturally, it is possible that a similar design with a sample in another country would have different findings. A third limitation in both studies is the self-report screeners used to assess depressive symptoms and diet. While both the CES-D and EPDS are widely used to assess depression and PPD, respectively, they are both self-report screeners by nature and therefore not as strong as a clinical diagnosis interview of depression. Additionally, the gold standard dietary data collection is through conducting

multiple dietary recalls lead by a trained interviewer, making it a preferred method over the food frequency questionnaires used in the current studies.

1.3 Future directions

We recommend two main takeaways for future research in the field - the first is that sugar and depression studies should include a measure of stress as a covariate alongside the widely used socioeconomic and demographic covariates. The current studies highlight that failing to do so may inflate the direct effect of added sugar consumption on depressive symptoms in both the general population and in the postpartum period. The second is that psychological stress may not be able to accurately capture a potential interaction between sugar consumption and stress, therefore, future studies should consider including both psychological and physiological measures as they may be closer approximations of the biological processes hypothesized to underly depression risk.

Lastly, given that there is a gap in the literature establishing a clear direction in the relationship between added sugar consumption and depressive symptoms in humans, future studies designed to fill that gap would be beneficial. Such studies could include a longer span of data collection, with dietary and depression data collected at multiple timepoints, showing the longitudinal effects of high sugar consumption on new cases of depression, and assessing whether changes in diet or depression are driving the effect. Randomized controlled trials that reduce sugar consumption then measure depression could also help us assess causality. Such an intervention would ideally give us insight into participant mental health before, during, and after eliminating sugar, and assess whether depressive symptoms were reduced when added sugar was removed from the diet.

Conclusion

Total added sugar consumption and SSB consumption predicted depressive symptoms in the general population and in postpartum. However, these results were no longer statistically significant in the general population when income was added into the model, and in the postpartum population when depression in pregnancy was added into the model. Additionally, chronic stress was consistently a stronger predictor of depressive symptoms than added sugar consumption. There was limited evidence in study 2 that sugar may interact with stress to protect women against postpartum depression. While we encourage this finding to be interpreted with caution due to the inconsistency between studies, one possible explanation we bring forth is that sugar consumption may be used as a successful coping mechanism in the stressful pregnancy period. These results suggest that, while added sugar consumption and depressive symptoms are positively correlated, further research including chronic psychological and physiological stress is warranted.

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APPENDIX

Table 1. *Participant demographics, chapter 2.*

Demographic variables	*N*	M(SD)/%
Age	215	60.24 (11.531)
Sex	217	
Male		33.20%
Female		66.80%
Race/ethnicity	217	
White		84.30%
Black or African		4.60%
Indigenous or Native		5.10%
Asian		0.50%
Pacific Islander		1.40%
Hispanic or Latino/a		3.20%
Other		0.90%
Previous chronic condition	217	
Yes		71.00%
No		29.00%
Country of residence	217	
USA		9.20%
UK		84.80%
New Zealand		0.90%
Australia		5.10%
Education	217	
No formal education		0.50%
Some primary education		2.30%
Graduated high school		17.10%
Some post-secondary education		17.10%
Completed post-secondary education		5.50%
Bachelor's degree		39.20%
Master's degree		15.20%
Doctorate		3.20%
Number of people in household	217	3.03(1.475)
Combined household income	217	
USA	20	$40,001 - $60,000
UK	184	£45,001 - £60,000
New Zealand	11	AUD 75,001 - 95,000
Australia	2	NZD 110,001 - 130,000
CES-D score	217	19.456 (12.741)
TICS score	217	15.475 (6.745)
ACEs score	217	2.65 (2.054)
Total sugar consumption	217	4.487(1.491)
SSB consumption	217	13.461(4.474)

Table 2. *Correlation matrix of PPD, stress, and sugar consumption over time, chapter 2*.

Variables	1	2	3	4	5	6	7	8	9	10	11	12	13
1. CES-D scores at baseline	1	.846**	.747**	.635**	.383**	.191**	.142*	.174*	0.119	-0.152	0.008	-0.070	-0.196
2. CES-D scores at follow-up	-	1	.687**	.756**	.359**	.142*	.167*	.169*	.140*	-.157*	-.015	-.018	-.191**
3. TICS scores at baseline	-	-	1	.763**	.429**	.161*	.197**	.026	.130	-.170*	.079	.060	-.117
4. TICS scores at follow up	-	-	-	1	.322**	.176**	.242**	.101	.174*	-.126	.079	.093	-.142*
5. ACEs scores	-	-	-	-	1	.001	-.019	-.008	-.001	-.047	.080	.007	-.164*
6. Total sugar consumption at baseline	-	-	-	-	-	1	.753**	.623**	.447**	-.181*	-.063	.001	.077
7. Total sugar consumption at follow up	-	-	-	-	-	-	1	.390**	.616**	-.160*	-.054	.009	.059
8. SSB consumption at baseline	-	-	-	-	-	-	-	1	.541**	-.227**	-.185**	-.083	.048
9. SSB consumption at follow-up	-	-	-	-	-	-	-	-	1	-.241**	-.118**	-.123	.001
10. Age	-	-	-	-	-	-	-	-	-	1	.126	.034	-.058
11. Sex	-	-	-	-	-	-	-	-	-	-	1	.114	-.025
12. Education	-	-	-	-	-	-	-	-	-	-	-	1	.146*
13. Income	-	-	-	-	-	-	-	-	-	-	-	-	1

Text in red shows correlations with behavioral measures of maternal sensitivity or infant mood during the play session

** Correlation is significant at the .01 level (2-tailed)

* Correlation is significant at the .05 level (2-tailed)

Table 3. *Stepwise regressions of sugar consumption at baseline as a predictor of depressive symptoms at 1 month follow-up, chapter 2.*

Total Sugar Consumption			**SSB Consumption**		
Step 1: Sugar Predicting Depressive Symptoms					
	Stand. B	*p value*		Stand. B	*p value*
Total Sugar Consumption	0.142	0.038	SSB Consumption	0.169	0.013
Step 2: Time 1 Stress and Interactions					
Total Sugar Consumption	0.124	0.356	SSB Consumption	0.26	0.033
TICS Score	0.647	<.001	TICS Score	0.649	<.001
ACEs Score	-0.082	0.141	ACEs Score	-0.089	0.103
Total Sugar x TICS Interaction	-0.105	0.459	SSB x TICS Interaction	-0.089	0.103
Total Sugar x ACEs Interaction	-0.065	0.275	SSB x ACEs Interaction	-0.115	0.373
Step 3: Dietary Factors Added to the Model					
	Stand. B	*p value*		Stand. B	*p value*
Total Sugar	0.128	0.347	SSB Consumption	0.264	0.032
TICS Score	0.645	<.001	TICS Score	0.648	<.001
ACEs Score	-0.079	0.155	ACEs Score	-0.087	0.113
Total Sugar x TICS Interaction	-0.109	0.447	SSB x TICS Interaction	-0.123	0.345
Total Sugar x ACEs Interaction	-0.068	0.26	SSB x ACEs Interaction	0.015	0.802
Total Fruit	0.032	0.578	Total Fruit	0.032	0.568
Total Vegetables	-0.056	0.333	Total Vegetables	-0.041	0.47
Step 4: Demographics Added to the Model					
	Stand. B	*p value*		Stand. B	*p value*
Total Sugar	0.106	0.436	SSB Consumption	0.228	0.068
TICS Score	0.646	<.001	TICS Score	0.648	<.001
ACEs Score	-0.069	0.217	ACEs Score	-0.073	0.182
Total Sugar x TICS Interaction	-0.083	0.563	SSB x TICS Interaction	-0.087	0.51
Total Sugar x ACEs Interaction	-0.063	0.304	SSB x ACEs Interaction	0.021	0.727
Total Fruit	0.036	0.532	Total Fruit	0.034	0.551
Total Vegetables	-0.019	0.749	Total Vegetables	-0.006	0.915
Age	-0.012	0.819	Age	-0.004	0.933
Sex	-0.068	0.182	Sex	-0.84	0.402
Education	-0.034	0.53	Education	-0.574	0.567
Income	-0.104	0.049	Income	-2.036	0.043
Step 5: Depression at baseline Added to the Model					
	Stand. B	*p value*		Stand. B	*p value*
Total Sugar	0.055	0.584	SSB Consumption	0.114	0.225
TICS Score	0.12	0.04	TICS Score	0.124	0.036
ACEs Score	-0.017	0.681	ACEs Score	-0.023	0.577
Total Sugar x TICS Interaction	-0.026	0.571	SSB x TICS Interaction	-0.011	0.803
Total Sugar x ACEs Interaction	-0.092	0.385	SSB x ACEs Interaction	-0.091	0.36
Total Fruit	0.026	0.535	Total Fruit	0.027	0.528
Total Vegetables	0.021	0.637	Total Vegetables	0.02	0.655
Age	-0.017	0.652	Age	-0.012	0.754
Sex	-0.039	0.308	Sex	-0.03	0.438
Education	0.024	0.545	Education	0.028	0.481
Income	-0.035	0.377	Income	-0.04	0.304
CES-D Score at Time 1	0.753	<.001	CES-D Score at Time 1	0.736	<.001

Table 4. *Participant demographics, chapter 3.*

Demographic Information	*N*	M(SD)/%
Age	4866	30.23 (5.734)
Race/ethnicity (including multiple identities)	4866	
NZ European		58.73%
Pacific Islander		35.16%
Other		6.11%
Current health status	4866	
Poor		1.6%
Fair		7.7%
Good		25.40%
Very good		41.90%
Excellent		23.40%
Education	4856	
No secondary school		5.70%
Secondary school		22.30%
Diploma/Trade Certification		30.10%
Bachelor's degree		24.60%
Higher degree		17.40%
Number of people in household	4859	2.62 (1.801)
NZ deprivation quantile	4866	3.11 (1.435)
Combined household income	1934	NZD 82712.96 (74196.108)
EPDS score at 9 months	4866	5.17 (4.53)
Total sugar consumption	4866	4.112(1.084)
SSB consumption	2856	3.52 (1.783)
Chronic stress	4866	9.9 (2.409)

Table 5. *Correlation Matrix between EPDS scores in pregnancy, EPDS scores at 9 months, chronic stress, and sugar consumption, chapter 3.*

	1	2	3	4	5	6
1. EPDS scores in postpartum	1	.452**	.389**	.109**	.129**	-.123**
2. EPDS scores in pregnancy	-	1	.528**	.143**	.160**	-.140**
3. Chronic stress	-	-	1	.190**	.236**	-.280**
4. Total sugar consumption	-	-	-	1	.662**	-.181**
5. SSB consumption	-	-	-	-	1	-.268**
6. Maternal Education	-	-	-	-	-	1

$^{**}p < .001$

Table 6. *Stepwise Regressions of Sugar Consumption in Pregnancy as a Predictor of Depressive Symptoms at 9 Months Postpartum, chapter 3.*

Total Sugar Consumption			**SSB Consumption**		
Step 1: Sugar Predicting Depressive Symptoms					
	Stand. B	*p value*		Stand. B	*p value*
Total Sugar Consumption	0.097	<.001	SSB Consumption	0.129	<.001
Step 2: Time 1 Stress and Interactions					
Total Sugar Consumption	0.04	0.008	SSB Consumption	0.05	0.012
Chronic Stress Composite	0.382	<.001	Chronic Stress Composite	0.375	<.001
Total Sugar and Stress Interaction	-0.052	<.001	SSB and Stress Interaction	-0.035	0.079
Step 3: Dietary Factors Added to the Model					
	Stand. B	*p value*		Stand. B	*p value*
Total Sugar Consumption	0.044	0.004	Total Sugar Consumption	0.048	0.016
Chronic Stress Composite	0.375	<.001	Chronic Stress Composite	0.369	<.001
Sugar and Stress Interaction	-0.051	<.001	SSB and Stress Interaction	-0.034	0.091
Green Leafy Vegetables	0	0.978	Green Leafy Vegetables	-0.002	0.931
Other Vegetables	-0.034	0.033	Other Vegetables	-0.032	0.126
Citrus Fruits	0.006	0.673	Citrus Fruits	0.007	0.716
Non-Citrus Fruits	-0.022	0.162	Non-Citrus Fruits	-0.019	0.339
Step 4: Demographics Added to the Model					
	Stand. B	*p value*		Stand. B	*p value*
Total Sugar Consumption	0.024	0.095	SSB Consumption	0.046	0.025
Chronic Stress Composite	0.189	<.001	Chronic Stress Composite	0.367	<.001
Sugar and Stress Interaction	-0.04	0.005	SSB and Stress Interaction	-0.033	0.095
Green Leafy Vegetables	-0.001	0.921	Green Leafy Vegetables	-0.002	0.925
Other Vegetables	-0.023	0.132	Other Vegetables	-0.03	0.141
Citrus Fruits	0.008	0.594	Citrus Fruits	-0.006	0.764
Non-Citrus Fruits	-0.021	0.161	Non-Citrus Fruits	-0.019	0.357
Maternal Education	-0.018	0.214	Maternal Education	-0.011	0.592
Step 5: Depression at Time 1 Added to the Model					
	Stand. B	*p value*		Stand. B	*p value*
Total Sugar Consumption	0.024	0.095	SSB Consumption	0.031	0.11
Chronic Stress Composite	0.189	<.001	Chronic Stress Composite	0.185	<.001
Sugar and Stress Interaction	-0.04	0.005	SSB and Stress Interaction	-0.027	0.153
Green Leafy Vegetables	-0.001	0.921	Green Leafy Vegetables	-0.003	0.895
Other Vegetables	-0.023	0.132	Other Vegetables	-0.022	0.36
Citrus Fruits	0.008	0.594	Citrus Fruits	0.009	0.528
Non-Citrus Fruits	-0.021	0.161	Non-Citrus Fruits	-0.02	0.655
Maternal Education	-0.018	0.214	Maternal Education	-0.014	0.459
EPDS in Pregnancy	0.346	<.001	EPDS in Pregnancy	0.347	<.001

Figure 1. *Scatterplot of Interaction Between Added Sugar and Chronic Stress in Pregnancy Predicting PPD, Chapter 3.*