Exploring the Bidirectional Relationship between Food Addiction and Dietary Restraint

by

Julia M. Rios

A dissertation submitted in partial fulfillment
of the requirements for the degree of
Doctor of Philosophy
(Psychology)
in The University of Michigan
2023

Doctoral Committee:

Professor Ashley N. Gearhardt, Chair
Professor Susan Gelman
Dr. Julie Lumeng
Professor Kendrin Sonneville

Julia M. Rios

jsmr@umich.edu

ORCID iD: 0000-0002-6215-8108

ACKNOWLEDGEMENTS

The studies presented in this dissertation were completed with the generous contributions of research collaborators, lab comembers, and research staff. As primary faculty advisor, Dr. Ashley Gearhardt has provided extraordinary guidance for every stage of research and manuscript preparation for all studies included in this dissertation. Dr. Julie Lumeng, Dr. Kendrin Sonneville, Dr. Susan Gelman, and Dr. Ashley Gearhardt served as the dissertation committee, providing invaluable feedback and consultation. Thanks to Dr. Martha Berg for significant contributions and Dr. Todd Chan for minor consultations on the longitudinal statistical methods for Project Media. I am additionally grateful for the opportunity to collaborate with Dr. Julie Lumeng, Dr. Alison Miller, and Dr. Kate Rosenblum, and for their immense contributions to the infant protocols and research methods for ABC Baby. Many thanks to Danielle Appugliese and Nico Kaciroti for their statistical expertise and contributions to the ABC Baby manuscripts. All studies were made possible with the exceptional assistance of research staff in the Food and Addiction Science and Treatment Laboratory and Lumeng Laboratory at the University of Michigan. And for everything else, you are my backbone: thank you, M.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

FIGURE

LIST OF APPENDICES

LIST OF ACRONYMS

ACRONYM

APA: American Psychology Association

BEBQ: Baby Eating Behavior Questionnaire

BED: Binge Eating Disorder

BMI: Body Mass Index

BN: Bulimia Nervosa

CDC: Centers for Disease Control and Prevention

CFI: Comparative Fit Index

CI: Confidence Interval

DEBQ: Dutch Eating Behavior Questionnaire

DEBQ-R: Dutch Eating Behavior Questionnaire Restraint Subscale

DSM-5: Diagnostic and Statistical Manual of Mental Disorders (5th Edition)

DSM-IV: Diagnostic and Statistical Manual of Mental Disorders (4th Edition)

dYFAS-C 2.0: Dimensional Yale Food Addiction Scale for Children Version 2.0

EF: Enjoyment of Food (Baby Eating Behavior Questionnaire subscale)

FR: Food Response (Baby Eating Behavior Questionnaire subscale)

GA: General Appetite (Baby Eating Behavior Questionnaire subscale)

HP: Highly Processed

IRB: Institutional Review Board

OR: Odds Ratio

OW: Risk for Overweight

SD: Standard Deviation

SE: Standard Error

SEM: Structural Equation Model

SR: Sucrose Response

SRMR: Standardize Root Mean Squared Residual

WLZ: Weight-for-Length Z-Score

YFAS: Yale Food Addiction Scale

YFAS 2.0: Yale Food Addiction Scale Version 2.0

ABSTRACT

The construct of food addiction, used to describe addictive-like pathological eating, has garnered considerable research attention, empirical evidence, and scholarly debate in recent years. A major point of controversy is that current models and measures of food addiction do not consider the role of dietary restraint. A small body of cross-sectional research suggests that food addiction and dietary restraint may be more closely related at some stages of development (e.g., adolescence) than others (e.g., adulthood). However, little is currently known about potential relations, directional pathways, or clinical implications of these constructs. This dissertation thereby aims to examine direct associations between food addiction and dietary restraint in adolescence and adulthood. Next, this dissertation will assess longitudinal pathways between food addiction and dietary restraint during adolescence, when the strength of the association is likely to be the strongest. Finally, this dissertation seeks to examine the construct validity of these constructs by comparing associations of maternal food addiction and maternal dietary restraint with infant eating and weight-related outcomes. Improved understanding of the nature of the relationship between food addiction and dietary restraint that results from this dissertation has important implications for public health and clinical treatment recommendations.

CHAPTER 1

Introduction and Specific Aims

Food addiction is an important emerging construct that describes "excessive overeating of high-calorie food accompanied by loss of control and intense food cravings" (Gearhardt et al., 2009; Maxwell et al., 2020). While there is currently no official diagnosis of food addiction included in the Diagnostic and Statistical Manual of Mental Disorders (DSM-5), the Yale Food Addiction Scale (YFAS) was developed to operationalize compulsive food intake to reflect the DSM-5 criteria for substance use disorders (see Appendix A; Gearhardt et al., 2011; 5th ed.; American Psychiatric Association [APA], 2013). The YFAS has proven to be a valuable diagnostic instrument for identifying a clinically significant population with food addiction that is associated with higher impulsivity, reward dysfunction, emotion dysregulation and poorer quality of life (Gearhardt et al., 2016; Kiyici et al., 2020). Since the development and validation of the YFAS, food addiction has procured a considerable research focus and a growing body of evidence supports the notion that certain foods (e.g., highly processed (HP) foods) may, for some, have addictive properties similar to addictive substances (Schulte et al., 2015).

Food addiction, as measured by the YFAS, is prevalent among individuals with binge-type eating disorders (i.e., binge eating disorder (BED) and bulimia nervosa (BN)) and is associated with a more severe presentation of disordered eating (Meule & Gearhardt, 2019; Oliveira et al., 2020). Food addiction and binge-type eating disorders both involve diminished control over eating and continued consumption despite negative consequences (Gearhardt et al., 2011). Despite this overlap, binge eating and addictive eating are nonidentical pathologies. In binge eating, an objectively large quantity of food must be consumed during a discrete period of time, accompanied by a sense of loss-of-control (APA, 2013). This does not capture the excessive or compulsive eating that occurs across the course of a day for many individuals who experience food addiction (Schulte et al., 2016). Prior studies have estimated that only 41.5% of individuals with BED in a primary care setting (Gearhardt et al., 2016) and 50-57% of individuals with BED in a specialty care setting meet criteria for food addiction (Gearhardt et al., 2012; Ivezaj et al., 2016). Additionally, over half of people who meet criteria for food addiction do not meet criteria for an existing eating disorder (Gearhardt et al., 2011). These statistics reflect both the substantial overlap and pivotal distinctions between presentations of binge eating and food addiction. As such, food addiction and binge-type eating disorders are best understood as related, but distinct, constructs.

Alongside empirical support for the concept of food addiction, remaining questions regarding its mechanisms and clinical utility have stimulated scholarly

debate. A leading criticism of the food addiction construct is that existing models do not adequately account for contributions of dietary restraint (Wiss & Avena, 2020). Dietary restraint has often been defined as a self-imposed restriction of food intake in order to lose weight or avoid weight gain (Herman & Polivy, 1975). However, empirical evidence suggests that individuals who report high levels of dietary restraint do not always appear to be actually reducing or restricting caloric intake (Lowe, 1993; Stice et al., 2004). More recently, dietary restraint has been better understood to also encompass cognitive efforts to reduce overall intake or avoid certain types of food regardless of success (Lowe et al., 2006; Polivy et al., 2020).

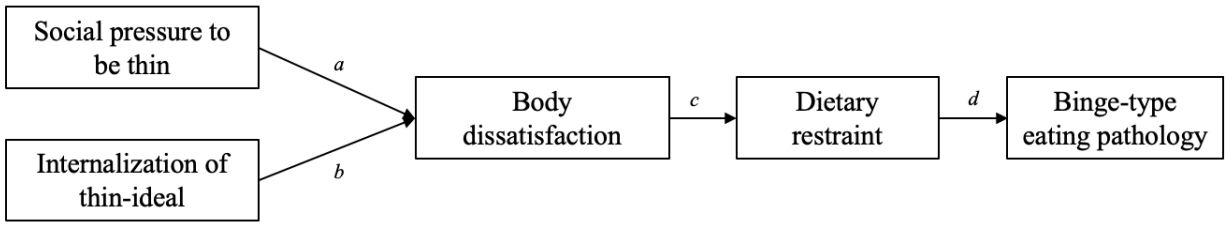
Thus, it is important for measurement instruments to include items which capture behavioral and cognitive aspects of dietary restraint. One of the most widely used scales is the Dutch Eating Behavior Questionnaire restraint subscale (DEBQ-R), which includes items that assess both actual (e.g., "Do you deliberately eat less in order to not become heavier?") and attempted (e.g., "Do you try to eat less at mealtimes than you would like to eat?") dietary restraint (see Appendix A; van Strien et al., 1986). In this dissertation, use of the term dietary restraint will therefore refer to both elements of restraint eating, as defined by the DEBQ, rather than caloric deprivation alone.

Empirical Literature on Dietary Restraint in Binge-type Eating Pathology

Eating disorder models have traditionally held that maladaptive (e.g., rigid or excessive) dietary restraint is a critical antecedent to binge eating (Herman & Polivy,

1990; Telch & Agras, 1993). According to many eating disorder models, dietary restraint creates a state of physiological and psychological deprivation which is difficult to maintain and ultimately induces pathological overeating (Kirkley et al., 1988). Introduced in 1975, the restraint theory of binge eating posits that dietary restraint relies on a shift from physiological regulation of eating behaviors to cognitive control over eating, which amplifies one's vulnerability to disinhibited eating when those cognitive processes are disrupted (Ruderman, 1986). In 2003, Fairburn published a transdiagnostic theory of eating disorders which similarly proposed that dieting precipitates binge eating observed in BED and BN (Fairburn et al., 2003). These perspectives presume dietary restraint to be a necessary precursor for loss of control type eating pathology, such as that observed in binge-type eating pathology and food addiction (Wiss & Avena, 2020; see Figure 1).

Figure 1.1. Dietary Restraint Model of Binge-type Eating Disorder Pathology



Ref. "Applicability of the dual pathway model in normal and overweight binge eaters" by Welsh & King, 2016. *Body Image*. 18, 9. (https://onlinelibrary.wiley.com/doi/pdf/10.1002/eat.20897)

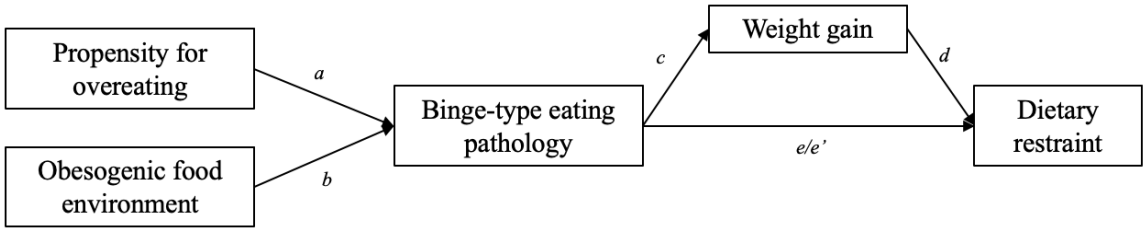
Since the introduction of early eating disorder models for restraint, numerous research studies have confirmed correlations among dietary restraint and binge eating

(Allen et al., 2011; Goldschmidt et al., 2012; Grilo et al., 2001; Johnson & Wardle, 2005). In one community study, individuals who reported dieting were two times more likely to binge eat (Andrés & Saldaña, 2014) and a large twin study found that dietary restraint reliably moderates other genetic and environmental influences on binge eating (Racine et al., 2011). Longitudinal studies using retrospective self-report data suggest that individuals with a history of dieting or weight loss attempts may be more likely to develop binge eating problems than non-dieters (Goldschmidt et al., 2012; Grilo & Masheb, 2000; Hilbert et al., 2014; Reas & Grilo, 2007; Spurrell et al., 1997). Thus, dietary restraint appears, for some, to be a relevant factor in the development or progression of binge eating pathology.

However, about half of individuals who develop BED do not report any history of dieting (Grilo & Masheb, 2000) or report that binge eating precedes dietary restraint (Brewerton et al., 2000; Tanofsky-Kraff et al., 2005). An alternative framework for understanding dietary restraint is that dieting behaviors are more likely to be implemented as a reactive strategy to an individual's vulnerability for weight gain or obesity. This perspective considers that some individuals appear to be more sensitive to the hedonic nature of rewarding foods or weight gain independent of their diet history (Lowe et al., 2009). Prior evidence indicates that some individuals may be more likely to implement dietary restraint behaviors as a reaction to overeating and binge episodes as an effort to avoid subsequent weight gain (Lowe, 2015). Individual differences in

reward response to HP foods may be particularly relevant to a tendency to develop dietary restraint in the modern obesogenic food environment characterized by accessibility to highly rewarding HP food (see Appendix B; Lowe et al., 2013; Lowe, 2015).

Figure 1.2. Vulnerability to Binge-type Eating Pathology as a Risk Factor for Dietary Restraint



Ref. "Dieting: proxy or cause of future weight gain?" by Lowe, 2015. *Obesity Reviews*, *16*(S1), 19–24. (https://doi.org/10.1111/OBR.12252); "Dieting and restrained eating as prospective predictors of weight gain." Lowe, et al., 2013. Frontiers in Psychology, 4, 577. (https://doi.org/10.3389/fpsyg.2013.00577)

Experimental treatment studies represent another challenge for binge eating models that position restraint as a causal factor. Several controlled intervention studies have demonstrated that implementation of dietary restraint (e.g., behavioral weight loss treatments where participants are instructed to restrict their dietary intake) resulted in reduced binge eating and other disordered eating pathology for individuals with BED, BN, and obesity (Goodrick et al., 1998; Grilo et al., 2011; Groesz & Stice, 2007; Presnell & Stice, 2003; Reeves et al., 2001; Striegel-Moore et al., 2010). Studies that examine effects of dietary restraint protocols for individuals with obesity also do not find increased

rates of binge eating behavior after a period of restraint (Wadden et al., 2004). In a least some individuals and circumstances, dietary restraint does not appear to elicit or worsen binge eating symptoms and may even have some beneficial effect on eating pathology. Therefore, dietary restraint may be a causal antecedent to binge eating for some, but a consequential reaction to binge eating in others.

Empirical Literature on Dietary Restraint and Food Addiction

In contrast with traditional eating disorder models, food addiction models do not currently centralize dietary restraint as a causal etiological factor (Herman & Polivy, 1990; Schulte et al., 2016; Ziauddeen & Fletcher, 2012). Because food addiction and binge eating share key features (e.g., loss of control eating and food cravings; Gearhardt et al. 2011), it stands to reason that findings on dietary restraint and related eating disorders (e.g., BED and BN) provide valuable theoretical insight regarding how dietary restraint may influence similar eating pathology in food addiction. Based on the dietary restraint literature in eating disorders, it is plausible that dietary restraint may also contribute to food addiction pathology or occur as a reaction to negative consequences of food addiction. However, restraint is not a central causal feature in known addiction pathways (Schulte et al., 2016). Thus, it is also possible that dietary restraint is not strongly associated with food addiction. Nonetheless, the empirical literature on food addiction and dietary restraint is sparse, and potential relations or directional pathways are poorly understood.

Dietary Restraint and Food Addiction in Adolescents

Adolescence is a potentially key period during which to investigate the association between food addiction and dietary restraint. Adolescence is a high-risk period for both the emergence of dieting behaviors and heightened vulnerability to addictive behaviors (Crews et al., 2007; Stice, 1998). However, few existing studies, to our knowledge, have explored food addiction and dietary restraint in adolescents. In one study, food addiction symptoms assessed by the dimensional Yale Food Addiction Scale for Children 2.0 (dYFAS-C 2.0), a version of the YFAS adapted to reflect age-appropriate symptoms (e.g., problems at school instead of work) and reading level (see Appendix A; Gearhardt et al., 2013), were positively correlated with dietary restraint scores on the DEBQ-R (r = .32; Schiestl & Gearhardt, 2018). A second study showed that YFAS scores were significantly correlated with the Three Factors Eating Questionnaire dietary restraint subscale in Turkish adolescents (OR = 1.01; Alim et al., 2021). Of note, the effect size of these associations are small. Nonetheless, these studies suggest that food addiction and dietary restraint may be related in adolescents but are limited by cross-sectional research design.

According to restraint-based theories for eating pathology, these prior findings could be interpreted as evidence that dietary restraint increases risk for addictive eating behaviors. However, these results could also signify that adolescents with higher propensity for addictive eating may be more likely to engage in dietary restraint as a

reaction to patterns of overconsumption and possible weight gain. If dietary restraint is a stronger predictor of future food addiction, this may support restraint-based theories for a causal role of dietary restraint. Alternatively, if food addiction is a stronger predictor of future dietary restraint, this may support a model of food addiction in which risk is primarily underlied by exposure to HP foods, and dietary restraint occurs as a consequence, rather than a cause, of addictive eating (see Appendix 2). If neither dietary restraint nor food addiction predicts longitudinal change in either construct, this may indicate that dietary restraint is a less relevant construct in food addiction than binge-type eating disorders.

Limitations of cross-sectional prior studies makes it difficult to speculate on the nature of the relationship between food addiction and dietary restraint over time. Thus, part of the first aim of this dissertation is to determine whether food and dietary restraint are correlated in the present sample of adolescents. Moreover, longitudinal research is needed to better understand temporal associations and possible directionality between food addiction and dietary restraint that appear during adolescence. Therefore, the second aim of this dissertation is to assess competing theoretical pathways by evaluating the emergence of food addiction symptoms and dietary restraint in a longitudinal study of adolescents. If empirical evidence indicates that dietary restraint is a prominent risk factor for future food addiction, interventions should aim to reduce dietary restraint to prevent food addiction in adolescents.

However, if longitudinal data indicates that food addiction predicts future increases in dietary restraint, reducing food addiction may be effective in preventing dietary restraint. It is also plausible that food addiction and dietary restraint each influence the other over time in a cyclic fashion, although one may be more temporally primary. Alternatively, if food addiction and dietary restraint are unrelated to one another over time in adolescents, this would suggest that restraint may not be central factor in food addiction at this developmental stage and would not be a key concept to consider in clinical intervention efforts.

Exploring Food Addiction and Dietary Restraint in Adults

In contrast to adolescents, food addiction and dietary restraint appear to be uncorrelated in adults in the United States. Validation of the YFAS 2.0 demonstrated discriminate validity between food addiction and dietary restraint in a two large community samples by detecting a non-significant association between the constructs (Gearhardt et al., 2016). Later development of the modified YFAS (mYFAS), which utilizes a briefer scale, replicated these findings (Schulte & Gearhardt, 2017). Food addiction and dietary restraint have also been uncorrelated in clinical samples of individuals with BED and/or obesity (Carter et al., 2019; Gearhardt et al., 2012, 2013). One international study found a small association between YFAS 2.0 food addiction scores and DEBQ-R restraint scores ($\eta^2 = .035$, $p < .001$) among young adults (aged 18-30) in Russia (Borisenkov et al., 2020). Another study in Turkey showed a small association

between scores on the YFAS scale and the Eating Disorder Examination Questionnaire Restraint subscale (z = -2.28, d = 0.18, p < .01) among adults (aged 20-54). Other international studies have failed to find a similar link (Imperatori et al., 2019; Legendre & Bégin, 2020). Therefore, in addition to adolescents, the first aim of this dissertation will also examine whether food and dietary restraint are correlated in the present sample of adults.

Though prior findings indicate that food addiction and dietary restraint are likely more distinct constructs in adults, the reasons underlying differential associations between food addiction and dietary restraint in adolescence and adulthood are uncertain. In adults, food addiction is associated with a history of weight cycling (Gearhardt et al., 2014; Gearhardt et al., 2016), which suggests that repeated dieting failures may reduce the motivation to continue restraining food intake over time (Schiestl & Gearhardt, 2018). By adulthood, individuals may no longer perceive dietary restraint as a viable or effective option to combat undesirable patterns of eating or weight gain. This is supported by evidence that drive for thinness and weight-related dietary restraint strategies are reduced in women between adolescence to adulthood, which may reflect an overall reduction in cognitive and behavioral dietary restraint despite weight gain (Keel et al., 2007; Neumark-Sztainer et al., 2018). Thus, dietary restraint may be more closely associated with food addiction during adolescence, when

younger individuals have experienced fewer failed attempts to restrain intake and are still motivated to engage in dietary restraint behaviors.

Another potential way of further understanding the food addiction and dietary restraint constructs in adults is to investigate whether they are associated with similar or distinct correlates and outcomes. If food addiction and dietary restraint exhibit similar associations with theoretically relevant correlates and outcomes (e.g., eating and weight-related characteristics), this would support the notion that food addiction and dietary restraint may be closely related constructs. However, if food addiction and dietary restraint exhibit dissimilar correlates and outcomes, this would instead indicate that food addiction and dietary restraint are more divergent constructs with distinct etiological pathways and clinical implications.

Maternal-Child Predictors for Eating and Weight-related Outcomes. Directly comparing how maternal food addiction and maternal dietary restraint are associated with similar or distinct outcomes in children may be a clinically relevant domain in which to investigate these constructs. Maternal food addiction and maternal dietary restraint have both been associated with adverse child outcomes. Parental food addiction is linked to child addictive eating, higher child BMI z-scores, and child risk for obesity in children 5-12 years of age (Burrows et al., 2017). Moreover, when compared to parental BMI, parental addictive eating proves to be a better predictor of these eating behavior and weight outcomes (Burrows et al., 2017). However, no prior

study has examined how maternal addictive eating predicts eating behavior and weight during the infant stage.

Prior studies also indicate that maternal dietary restraint influences child eating behaviors and weight outcomes in older children (i.e., ages 5-9), although some findings are mixed. In one study, maternal dietary restraint was associated with greater BMI z-score change for children between 1.5 and 2.5 years (Rodgers et al., 2013). Another study found a link between maternal dietary restraint and reduced capacity to regulate caloric intake in a free-access experimental setting at age 5 (Birch & Fisher, 2000). Maternal dietary restraint has also been shown to predict child dietary restraint and weight preoccupation in children aged 8-15 (Rüther & Richman, 2013; Snoek et al., 2013), though other studies have reported null findings in similar age groups (Hill & Franklin, 1998; Ogden & Steward, 2000; Sanftner et al., 2007). Some researchers have proposed that links between maternal dietary restraint and negative child outcomes are the result of increased maternal monitoring or restriction of the child's diet, which is thought to foster self-restrictive attitudes or behaviors towards food in the child (e.g., Birch & Fisher, 2000; Tiggemann & Lowes, 2002). These findings highlight the theoretical overlap of maternal food addiction and maternal dietary restraint on child eating and weight-related outcomes. However, no prior study has assessed associations with either maternal food addiction or maternal dietary restraint during the infant stage.

In addition to maternal eating behavior, maternal weight status has been identified as an important predictor of child eating and weight outcomes. Maternal BMI has been found to reliably predict child BMI from birth through adolescence (Heude et al., 2005) and odds of child obesity at ages 1-14 increase by 264% when maternal pre-pregnancy BMI is in the obese range (Heslehurst et al., 2019). Furthermore, pre-pregnancy BMI is associated with important prenatal and fetal outcomes including fetal growth and neonatal anthropometrics (e.g., birth weight; Santos et al., 2009). Longitudinal data also suggest that maternal overweight or obese weight status increases risk of disordered eating behavior (i.e., binge eating, preoccupation with body weight and shape, and food restriction) in children across the first 13 years of life (Micali et al., 2018). However, no study, to our knowledge, has directly investigated associations between maternal pre-pregnancy BMI and infant eating behaviors (e.g., enjoyment of food, food responsiveness, or general appetite) before 12 months of age. In light of the known associations between maternal BMI and child BMI during gestation and later childhood, it will be important to also consider any effects of maternal pre-pregnancy BMI on early infant eating behaviors and risk for overweight.

Limited prior research in infants may be due, in part, to unique challenges of measuring infant feeding and weight characteristics. Infants cannot report on their own behavior, many behavioral assays have not been validated for this developmental stage and taking anthropometric measures in infants requires special training. However,

infant eating behaviors can be phenotyped through both maternal report and objective measurements of behavior. The Baby Eating Behavior Questionnaire (BEBQ) provides a parent-report psychometric measure of infant appetite in the context of exclusive milk-feeding (i.e., breast milk or formula) including the infant's enjoyment of food, satiety responsiveness, general appetite, and food responsiveness (Llewellyn et al., 2011). To provide a quantitative assessment of eating behavior, hedonic reward response to sweet flavors (i.e. sucrose) can be captured in infants using a validated index of behavioral measures including tongue protrusions, lip smacking, and bringing fingers to the mouth (Rios et al., 2020). If appropriately measured, infant's risk for overweight can be calculated using weight-for-length z-scores (WFL-z) and percentiles (≥85th percentile) defined by the World Health Organization (WHO, 2014). Thus, the combined use of subjective report and objective behavioral methodologies provides a comprehensive assay of infant eating behavior and weight outcomes at this early stage of life.

Infancy is a critical period of development marked by rapid growth and the establishment of certain feeding (e.g., appetitive traits) and weight-related characteristics (e.g., risk for overweight or obesity; Birch et al., 2007). Moreover, infancy is a period of time where many dietary factors are held constant. Early infancy (prior to 6 months of age) is associated with the consumption of a more homogenous diet (i.e., breast milk and formula; Clayton et al., 2013) and infants have not yet been introduced or weaned to solids (Khakoo & Lack, 2004). This may limit variation in dietary intake,

as well as variation in maternal feeding styles. Infants also have a reduced cognitive capacity to internalize thin ideals or restrictive eating attitudes which often accompany maternal dietary restraint and may influence eating behavior even by early childhood (Goldschmidt et al., 2008). Thus, infancy represents an ideal yet understudied developmental stage to investigate the transmission of maternal risk factors on child feeding and weight-related outcomes.

Thus, the third aim of this dissertation is to test the impact of maternal food addiction and maternal dietary restraint on subjective and objectively measured early infant eating behavior and risk for overweight (i.e., maternal report of infant feeding style, hedonic response to sucrose, and high risk WFL-z for overweight) in infants at four months of age. This study will simultaneously examine effects of maternal food addiction and dietary restraint to determine how maternal risk profiles may be associated with distinct (or similar) early infant feeding and weight characteristics allowing for more specificity in clinical interventions and future research targets. If maternal food addiction and maternal dietary restraint are predictive of similar infant feeding and weight-related characteristics, this would highlight similarities between the constructs and clinical interventions should offer similar recommendations for mothers with both eating presentations. However, distinct associations with infant feeding and weight-related outcomes would support the conceptualization of these as distinct

constructs and highlight the need to tailor early interventions for infants based on maternal eating style (i.e., food addiction or dietary restraint).

Summary of Specific Aims

Lack of explicit research on food addiction and dietary restraint represents a critical gap in the existing literature on this topic. Presently, very little is known about the association between food addiction and dietary restraint including potential directionality, clinical outcomes, and changes in association across the lifespan. This dissertation will consist of two studies to advance our empirical understanding of food addiction and dietary restraint across key developmental periods. These studies will utilize data that has been previously collected through larger studies conducted by our lab and collaborating researchers (i.e., Project Media and ABC Baby).

Aim 1. Assess zero-order correlations between food addiction and dietary restraint in two unique samples during adolescence and adulthood. Based on prior findings, we hypothesized that food addiction and dietary restraint will be significantly correlated in the sample of adolescents (n = 127, M = 14.8 years, SD = 3.1), but will not be significantly correlated in the sample of adults (n = 204, M = 31.5 years, SD = 4.9).

Aim 2. Examine longitudinal associations between food addiction and dietary restraint during adolescence, a critical period of development in which eating pathology often emerges, in order to better elucidate directional pathways between these constructs. Given the lack of extant longitudinal research, we did not have *apriori*

hypotheses regarding the directionality of temporal pathways between food addiction and dietary restraint.

Aim 3. Compare the associations of maternal food addiction and maternal dietary restraint with subjective and objectively measured early infant eating behaviors (i.e., maternal report of infant eating behavior, appetite, and hedonic response to sucrose) and risk for overweight in dyads of post-partum mothers and infants at four months of age. Given the lack of extant research, we did not have *apriori* hypotheses regarding the specific pattern of associations that would emerge between the maternal eating and weight factors and the infant eating behaviors and risk for overweight.

CHAPTER 2

Evaluating Bidirectional Predictive Pathways between Dietary Restraint and Food Addiction in Adolescents

Methods and materials

Participants

Recruitment. As part of a larger longitudinal study examining adolescent eating behavior and responsivity to food advertisements (Project Media), adolescent participants (i.e., 13-16 years of age) were recruited from southeast Michigan using print and online advertisements. A parent or guardian provided written informed consent and adolescents provided written informed assent prior to enrollment. Adolescents (N = 193), ranging from 13 to 16 years of age (M = 14.28, SD = 1.03), were recruited for the full study. The dimensional YFAS-C 2.0 (dYFAS-C 2.0) was added to the questionnaire battery later in data collection, and 127 participants completed the measure at the initial wave of data collection (Time 1). Participant descriptives are summarized in Table 2.1. This study was approved by the University of Michigan Institutional Review Board (IRB) and complied with the ethical standards of the APA (APA, 2013).

Inclusion and Exclusion Criteria. Due to the aims of the larger study investigating eating behavior and reward response to food marketing in adolescents, participant exclusion criteria included the following factors known to influence reward functioning: (1) a history of or a current eating disorder diagnosis, (2) current mood, anxiety, trauma, or psychotic disorders, (3) current prescription for a psychotropic medication, and (4) underweight BMI status.

Table 2.1 Adolescent Participant Demographics, Descriptives, and Data Missingness (n = 127)

	Total (n)	Percent (%)
Gender		
Male	61	48.0
Female	66	52.0
Race		
American Indian/Alaska Native	3	2.4
Black/African American	19	15.0
White	91	71.7
Other	1	0.8
Mixed	8	6.3
Unknown	5	3.9
Parental Education		
Less than High School	15	11.8
High School Degree	5	3.9
Some College	19	15
Associates Degree	11	8.7
Bachelor's Degree	35	27.6
Advanced Degree	42	33.1
	Mean (SD)	Range (min, max)
Age (months)	177.3 (12.4)	(156.0, 202.5)
BMI z-score	0.95 (0.9)	(-1.2, 2.7)

Note. SD = standard deviation; BMI = body mass index. Data missingness was determined by availability of data for primary variables included in the cross-lagged panel analysis (i.e., food addiction (YFAS) and dietary restraint (DEBQ-R) at each time point.

Measures

dYFAS-C 2.0. The dYFAS-C 2.0 is a 35-item self-report measure that operationalizes food addiction characteristics in children and adolescents based on the same DSM-5 criteria for substance use disorders as the YFAS (Schiestl & Gearhardt, 2018; Gearhardt et al., 2016). When completing the dYFAS-C 2.0, participants are instructed to think about foods high in refined carbohydrates and/or fats, as these foods have been most evidenced in food addiction (Schulte et al., 2015). All items are reported on a 5-point Likert scale (from 0 = never to 4 = always). Prior research suggests that problematic substance use in adolescence is more accurately conceptualized as a continuous rather than a categorical syndrome (Liu, 2017). Thus, the dYFAS-C 2.0 utilizes a dimensional scoring approaching. Item scores are summed with higher scores indicating more severe food addiction. The dYFAS-C 2.0 demonstrates good convergent and incremental validity as well as internal consistency (Schiestl & Gearhardt, 2018). In the current sample, dYFAS-C 2.0 scores demonstrated good internal consistency (α = .90).

Dutch Eating Behaviors Questionnaire Restraint Subscale. The Dutch Eating Behaviors Questionnaire Restraint Subscale **(**DEBQ) is a 33-item self-report survey designed to capture various aspects of eating style including external eating, emotional eating, and restrained eating. The 10-item restrained eating subscale (DEBQ-R) measures intentions and attempts to reduce food intake or to avoid certain food types.

All items were reported on a 5-point Likert scale (from 1 = never to 5 = very often). Scores on the DEBQ-R reflect the average of all items, with higher scores indicating greater degree of dietary restraint. The DEBQ demonstrates good predictive validity and internal consistency in adults (Strien et al., 2012; van Strien et al., 1986) as well as adolescents (Banasiak et al., 2001; Laessle et al., 1989). In the current sample, DEBQ-R scores demonstrated excellent internal consistency (α = 0.93).

Demographics and Anthropometry. Participants were asked to complete a demographics questionnaire at the first study visit (Time 1). Participants were asked to self-report their date of birth (which was used to calculate age in months), race, gender (as male, female, other gender identity, or prefer not to identify), and parental education level. Participant height and weight measurements were taken at the first study visit (Time 1). Participants were asked to remove any shoes, hats, and outerwear. Participant heights and weights were taken twice to confirm accuracy. Participants were weighed to the nearest 0.1 kg using a Detecto Portable Scale. If weights differed by more than 0.1 kg, the measurements were repeated. Participant height was measured using an O'Leary Acrylic Stadiometer to the nearest 1 cm. If height measurements differed by more than 1 cm, the measurements were repeated. Participant BMI z-scores were calculated using percentiles determined by the CDC for children and teens (Kuczmarski et al., 2002). Participant demographics and anthropometry measures are summarized in Table 2.1.

Data Analytic Plan

Statistical tests were completed using R version 4.1.2 and the *lavaan* package (Rosseel, 2012). Preliminary analyses were completed to verify that these data did not violate assumptions for cross-lagged panel analysis including normality, stationarity, and synchronicity (Kenny & Harackiewicz, 1979). To reduce the impact of missingness, data was imputed for dYFAS-C 2.0 and BMI z-scores at Time 2 (n = 115) and Time 3 (n = 91). Individuals who completed all measures at all time points (n = 91) did not significantly differ from individuals who did not complete Time 2 and/or Time 3 of the study (n = 36) on any of our variables of interest (i.e., food addiction or dietary restraint, all $p > .05$), covariates (i.e., age, gender, or BMI z-score, all $p > .05$), or demographics (i.e., race or parental education; all $p > .05$). No differences in patterns of significance were observed in the correlations or cross-lagged panel analyses between the unadjusted and imputed data sets, so results from the imputed sample were used for all analyses presented in this chapter. Full information maximum likelihood estimation was also used to maximize sample size, given an assumption that all missing data was missing at random (Tabachnick & Fidell, 2019). Prior to analysis, outlier values from both primary variables (food addiction and dietary restraint) were winsorized (Wilcox, 2005) and both variables were standardized. To control for possible confounding effects of differences in baseline age, gender, or BMI z-score (Snoek et al., 2008; Wiedemann et al.,

2021), these were considered as covariates in the model. Age and BMI z-score variables were mean-centered, and gender variable was contrast-coded.

To address Aim 1, zero-order correlations were tested among variables of interest and covariates at Time 1, Time 2, and Time 3. We hypothesized that food addiction and dietary restraint will be significantly correlated at each time point. To address Aim 2, temporal associations between food addiction and dietary restraint were examined using a cross-lagged panel design across three waves (Time 1, Time 2, and Time 3). The model simultaneously estimated the cross-lagged relationships between the two variables, as well as auto-regressive paths for each variable across time. All effects were assumed to be constant across the three time points; therefore, a single estimate was computed for each cross-lagged and auto-regressive relationship, independent of time (see Figure 1.1 for labeled path diagram). We did not have *apriori* hypotheses regarding the directionality of temporal pathways between food addiction and dietary restraint.

Results

Food addiction and dietary restraint were correlated at each time point ($r = 0.34$, $p = <.001$; $r = 0.34$, $p = < .001$; $r = 0.43$, $p = < .001$; Aim 1). Zero-order correlations among variables of interest and covariates at each time point are detailed in Table 2.2. Inclusion of covariates (i.e., gender, age, and BMI z-score) did not change the patterns of significance for any findings in the cross-lagged panel analysis but did result in poorer

model fit (respectively CFI = 0.939, SRMR = .051, CFI = .838, SRMR = .134;). Thus, for ease of interpretation and improved model fit, results and figures reported here reflect the unadjusted structural equation models (SEM). Figures and results for the adjusted SEM including covariates are provided in Appendix C.

The cross-lagged panel analysis revealed that food addiction significantly predicted future dietary restraint (b = 0.22, SE = 0.06, p < .001). Dietary restraint did not significantly predict future food addiction (b = 0.09, SE = 0.05, p > .05; see Table 2.3 for all model estimates). In comparing the difference between coefficients for each of the cross-lagged paths, Path 1 (food addiction predicting dietary restraint) was significantly stronger than Path 2 (dietary restraint predicting food addiction; b = 0.162, SE = 0.078, p < .05). Auto-regressive paths were significant for both food addiction (b = 0.613, SE = 0.052, p < .001) and dietary restraint (b = 0.585, SE = 0.055, p < .001) over time.

Table 2.2. Summary of Bivariate Correlations Among Adolescent Food Addiction, Dietary Restraint, and Associated Covariates

	Gender	Age	BMI Z-score
Food Addiction (dYFAS-C 2.0)			
Time 1	0.29***	0.16	0.33***
Time 2	0.28**	0.25**	0.28**
Time 3	0.22*	0.15	0.18*
Dietary Restraint (DEBQ-R)			
Time 1	0.24**	0.11	0.43***
Time 2	0.24**	0.18	0.33***
Time 3	0.15	0.18	0.39***

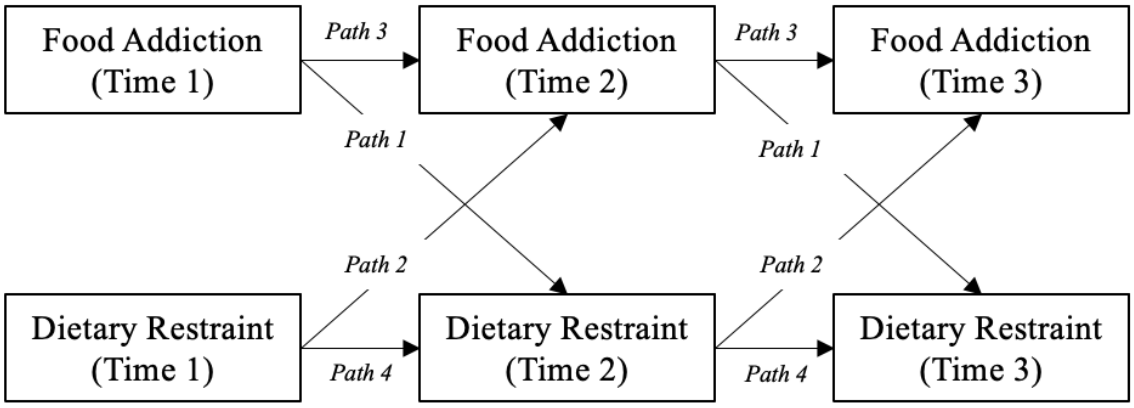
Note. dYFAS-C 2.0 = Dimensional Yale Food Addiction Scale for Children 2.0. DEBQ-R = Dutch Eating Behavior Questionnaire Restraint Subscale. Asterisks denote significance: * $p < .05$, ** $p < .05$, and *** $p < .001$. Gender (coded 1=male, 2=female).

Table 2.3. Standardized Regression Coefficients for Food Addiction and Dietary Restraint from Unadjusted Structural Equation Models

							CI (95%)	
Path	Predictor	Outcome	*b*	SE	*z*	*p*	lower	upper
1	Food addiction	Dietary restraint	0.25	0.06	4.39	< .001	0.14	0.36
2	Dietary restraint	Food addiction	0.09	0.05	1.69	0.09	-0.01	0.19
3	Food addiction	Food addiction	0.61	0.05	11.87	< .001	0.51	0.71
4	Dietary restraint	Dietary restraint	0.59	0.06	10.60	< .001	0.48	0.69

Note. $X^2(8) = 25.68$; SRMR = 0.05; CFI = 0.94. CI = confidence interval. A post-hoc sensitivity analysis using pwrSEM ((Wang & Rhemtulla, 2021) with 10,000 simulations and a seed of 23 indicated that our model had 100% power to detect an effect of this size for Path 1, and 41% power to detect an effect of this size for Path 2. Predictor variables were measured at T1, outcome variables were measured at times T2 and T3.

Figure 2.1. Path Diagram for Unadjusted Cross-lagged Panel Analysis between Food Addiction and Dietary Restraint



Discussion

In a longitudinal study of 127 adolescents, we assessed and compared the strength of predictive pathways between food addiction symptoms and dietary restraint across two years. Cross-lagged panel analyses showed that food addiction significantly predicted future dietary restraint over time. In contrast, dietary restraint did not predict future food addiction. To address our primary research question, we computed the difference between the coefficients for each of the cross-lagged paths. The path for food addiction predicting dietary restraint (Path 1) was stronger than the path for dietary restraint predicting food addiction (Path 2). Auto-regressive paths for food addiction (Path 3) and dietary restraint (Path 4) were both significant, indicating the stability of these predictors over time.

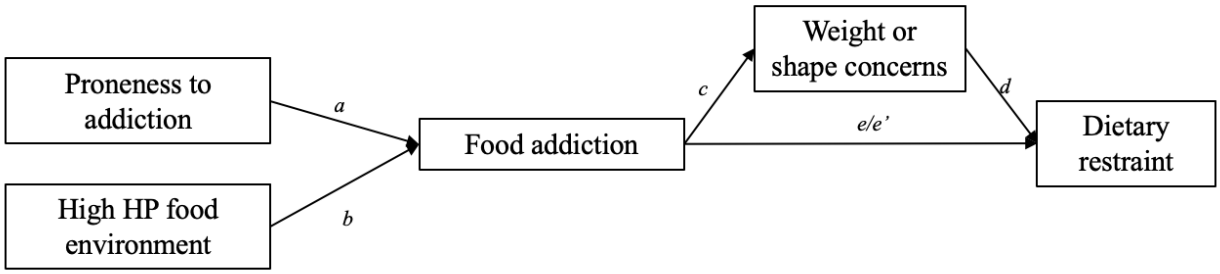
The present findings provide additional support to the existing literature that food addiction and dietary restraint demonstrate some association in adolescents (Alim et al., 2021; Gearhardt et al., 2013; Schiestl & Gearhardt, 2018). While some researchers have thereby speculated that dietary restraint plays a causal role in the development or progression of food addiction (Wiss & Avena, 2020), these longitudinal findings found that food addiction is a stronger predictor of future dietary restraint than dietary restraint is of future food addiction. This suggests that food addiction may be more likely to emerge prior to dietary restraint, and that dietary restraint occurs as a consequence rather than a cause of food addiction (see Figure 2.2, path *e*). This is consistent with other models of addiction (e.g., substance use disorders) in which

individuals exhibit restraint in an effort to control addictive behaviors or substance use (Schulte et al., 2016; Ziauddeen & Fletcher, 2012).

While no prior studies have directly examined temporal pathways between food addiction and dietary restraint, some longitudinal research has previously explored the role of dietary restraint in related constructs of binge-type eating pathology. While dietary restraint appears to be a relevant factor in binge-type eating pathology for some individuals (e.g., about half of individual who develop BED; Grilo & Masheb, 2000; Hilbert et al., 2014; Reas & Grilo, 2007), binge-type eating behaviors are reported to precede dietary restraint for many others (Grilo & Masheb, 2000; Brewerton et al., 2000; Tanofsky-Kraff et al., 2005). The present findings suggest that the relationship between food addiction and dietary restraint may be more consistent with the subgroup of individuals for whom dietary restraint appears to occur as a reaction to binge- type eating pathology (e.g., in an effort to avoid weight gain; Lowe, 2015; Lowe et al., 2013). While these constructs do share important features (e.g., loss of control over eating), prior studies have reported that only 42-57% of individuals with BED also endorse food addiction (Gearhardt et al., 2012; Gearhardt et al., 2016; Ivezaj et al., 2016). Future research could test whether individuals with co-occurring food addiction may represent a possible subtype of BED who are more likely to report binge eating prior to dieting behaviors than individuals without food addiction.

In sum, while these constructs do appear to be related during this stage of development, longitudinal analyses do not support a causal role of dietary restraint in mechanistic models of food addiction. Rather, the present findings provide stronger empirical support for a model of food addiction in which risk may be underlied by alternative factors, such as exposure to HP foods (Lowe et al., 2013; Lowe, 2015), clinical co-morbidities and psychological risk factors (e.g., addiction proneness; Davis et al., 2011), stronger reward sensitivity (Loxton & Tipman, 2017), or vulnerability for weight gain (Lowe, 2015; Lowe at al., 2013). Therefore, evidenced associations between food addiction and dietary restraint in adolescents may reflect attempts to manage an addictive response to HP foods (Figure 2.2). The present study provides empirical evidence for the direct temporal pathway between food addiction and dietary restraint (illustrated in Figure 2.2, path *e*). It will be important for future research to investigate theoretical risk factors (e.g., proneness to addiction or exposure to HP foods; Figure 2.2, paths *a* and *b*) or mediators (e.g., weight or shape concerns; Figure 2.2, path *e'*) in the model.

Figure 2.2. Proposed Model of Vulnerability to Food Addiction as a Risk Factor for Dietary Restraint



Clinical implications

If food addiction is a stronger predictor of future dietary restraint, strategies aimed specifically at ameliorating food addiction symptoms may be most effective for reducing future dietary restraint and any amplifying effects on future food addiction. The implementation of prevention (e.g., health-promoting school and home settings; Lee et al., 2010), treatment (e.g., adapted addiction treatment programs, treatment of comorbid psychopathology; Cassin & Sockalingam, 2021), and policy (e.g., restrictions on HP food marketing that targets teens; Harris et al., 2021) interventions during this crucial stage of development may have substantial benefits for reducing both food addiction and dietary restraint behaviors in adolescents. Currently, the majority of prior research has been dedicated to evaluating the food addiction construct, and much less research has explored or tested intervention approaches. This will be an important future direction for food addiction research.

There is a critical need for research-guided public health recommendations for dietary restraint that address the impairment and distress related to symptoms of food

addiction (Gearhardt, 2009) and living in a social environment that stigmatizes weight gain and fatness (Brown et al., 2022). Importantly, existing research suggests that not all forms of dietary restraint are associated with equally poor eating outcomes. For example, rigid dietary restraint (e.g., all-or-nothing dieting approach) compared with flexible dietary restraint (e.g., graduated dieting approach) appears to be more strongly associated with binge-type eating when dieting rules are violated (Westenhoefer et al., 1999). Prior research also shows improvements in eating outcomes (e.g., reduced external eating and emotional eating) when dietary restraint is implemented regularly and proactively (e.g., routine restraint) compared to irregularly and retroactively (e.g., compensatory restraint following diet noncompliance; Schembre et al., 2009). It is therefore possible that the type of dietary restraint which occurs in reaction to food addiction may have differential impacts on health and well-being. Intervention strategies that promote more flexible and proactive dietary restraining behaviors may have some pro-health utility. However, additional research is needed to better understand which forms of dietary restraint may be most harmful, neutral, or beneficial for promotion of healthy eating behaviors and how this may interact with a propensity for food addiction. It will be critical for future research to explore and develop dietary recommendations for individuals who endorse food addiction symptoms and struggle to control their eating.

Strengths, limitations, and future directions

This study offers number of important strengths and contributions to the empirical literature. This is the first longitudinal study design exploring temporal pathways between food addiction and dietary restraint allowing for inferences regarding the nature and directionality of the relationship between these constructs. Furthermore, this study involved adolescent participants and utilized developmentally appropriate psychometrics, which provided an assessment of food addiction and dietary restraint during a key developmental period in which eating pathology and dieting behaviors often emerge (Mussell et al., 1995; Stice et al., 1998).

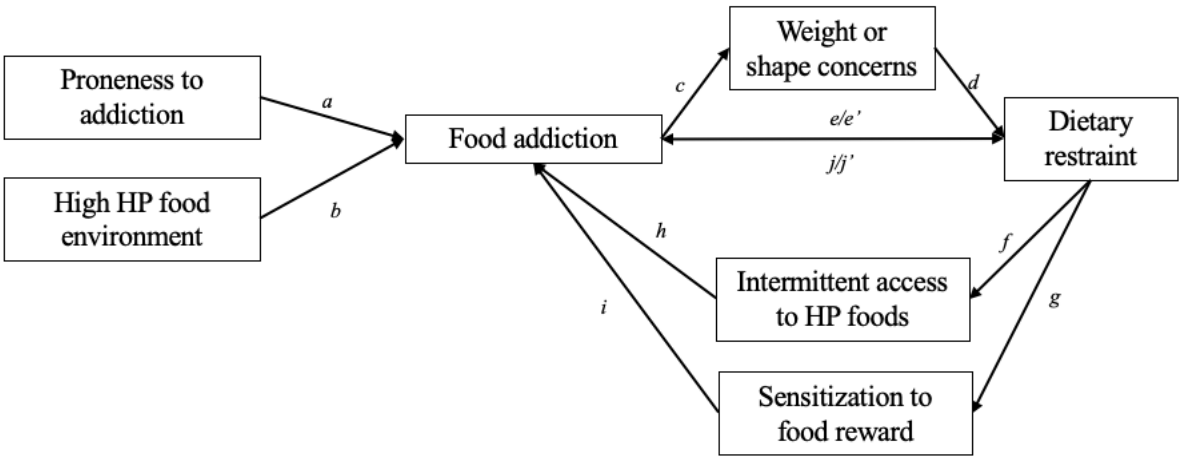
However, because this study was limited to a two-year span during adolescence, much remains unknown about early life risk factors and long-term progression of both food addiction and dietary restraint. Existing research indicates that by adulthood, food addiction and dietary restraint have a weaker association (Legendre & Bégin, 2020) or may no longer be associated (Gearhardt et al., 2016; Imperatori et al., 2019; Schulte & Gearhardt, 2017). Without longitudinal data spanning adolescence and adulthood, it is difficult to pinpoint why this association seems to diminish over time. Ongoing research is needed to better understand additional factors which may contribute to risk for both food addiction and dietary restraint, as well as how associations between these constructs change throughout various stages of development. The present study was also limited to a relatively small and well-resourced sample. Additional research is needed to test how these findings may generalize to larger and more diverse

populations. Use of a larger sample size would also provide more power to examine possible moderating effects of key covariates (i.e., age, gender, and BMI z-score).

Additional studies with stronger power may also help shed light on predictive pathways between dietary restraint and future food addiction. Although the pathway between dietary restraint predicting future food addiction did not reach significance, cross-lagged panel analysis revealed some association between dietary restraint and future food addiction that the current study may have been under-powered to detect. This may be indicative of a cyclic effect in which dietary restraint also amplifies food addiction symptoms to some degree (e.g., via mechanisms described in the dietary restraint model or dual pathway model for binge-type eating pathology; Welsh & King, 2016). It is also possible that alternating between periods of restricted eating (characteristic of dietary restraint; Johnson et al., 2012) and excessive eating (characteristic of food addiction; Gearhardt et al., 2011; Gearhardt et al., 2016) may contribute to an intermittent pattern of access. Intermittent access has been found to sensitize the mesolimbic dopaminergic system to substance-related cues and enhance the development of addictive response to psychoactive drugs and substances common in HP foods (i.e., sugar; Avena et al., 2008). Thus, if individuals are experiencing intermittent access to HP foods due to dietary restraint, this could be a pathway that enhances a propensity to develop food addiction (see Figure 2.3; Kosheleff et al., 2018). Despite this possibility, the current study supports the hypothesis that Path 1 (food

addiction leading to future dietary restraint) is the stronger pathway. It will be important for future research to better understand the bidirectional mechanisms that drive the association between food addiction and dietary restraint in adolescents.

Figure 2.3. Proposed Model of Vulnerability to Food Addiction as a Risk Factor for Dietary Restraint and Bidirectional Amplification of Addictive Response



Conclusions

This study aimed to investigate temporal pathways between food addiction and dietary restraint longitudinally in adolescents to increase knowledge on the nature and directionality of the relationship between these constructs. In this sample of 127 adolescents, food addiction significantly predicted future dietary restraint, but dietary restraint did not predict future food addiction, over time. Moreover, the temporal pathway between food addiction as a predictor for future dietary restraint was stronger than the longitudinal pathway for dietary restraint as a predictor for future food

addiction. While it has been theorized that dietary restraint plays a causal role in the development of addictive eating (Wiss & Avena, 2020), no prior studies have tested this hypothesis with a longitudinal study design. These results utilizing longitudinal data indicate that dietary restraint may be more likely to occur as a reaction or consequence to food addiction symptoms. These findings highlight the potential for intervention or prevention efforts that target reducing food addiction as a more effective strategy to promote overall healthy eating. Additional longitudinal research is needed to identify mechanistic pathways that underlie the bidirectional relationship between food addiction and dietary restraint across the course of development.

CHAPTER 3

Role of Maternal Risk Factors in Early Emergence of Infant Feeding and Weight Characteristics

Methods and materials

Participants

Recruitment. The ABC Baby study is an observational longitudinal cohort study of 284 infant-mother dyads recruited from the community. To facilitate recruitment, infant-mother dyads could enter the study at infant age 2 weeks, 2 months, or 4 months. The data collected at study entry is referred to as "baseline." The current analysis is limited to the 204 mother-infant dyads who provided data for maternal factors and infant eating behavior at age 4 months. Characteristics of the sample are summarized in Table 3.1. The sample of 204 dyads included in the current analyses did not differ on infant or race/ethnicity from the participants excluded due to missing data on the variables of interest (all $p > .05$). However, dyads included in the analyses did have higher maternal education than those who were not included ($\chi^2 = 14.1$, $p < .05$). This study was approved by the University of Michigan IRB. Mothers provided written informed consent for themselves and their infants.

Inclusion and Exclusion Criteria. Inclusion criteria at the time of enrollment were: (1) infant was born between 37–42 weeks gestation, (2) birth weight was within the 3rd–97th percentiles based on gestational age and sex, and (3) no significant perinatal or neonatal complications occurred. Exclusions were: (1) mother non-fluency in English; (2) foster care; (3) the mother was < 18 years old; (4) medical problems or known diagnosis affecting infant's current or future eating, growth or development; (5) child protective services involvement in the neonatal period; (6) infant had not consumed at least 2 ounces in one feeding from an artificial nipple and bottle at least once per week, or mother did not plan for the infant to do so by 2 months of age. Newly identified diagnoses or conditions reported during scheduling or study visits were assessed by practicing pediatrician PI Dr. Julie Lumeng to determine eligibility status. This study was approved by the University of Michigan IRB and complied with the ethical standards of the American Psychological Association (APA, 2013).

Table 3.1. Infant and Mother Sample Demographics and Descriptives (n = 204)

Mother Characteristics	Total (n)	Percent (%)
Maternal race/ethnicity		
Black, non-Hispanic	25	12.3
Hispanic, any race	8	3.9
White, non-Hispanic	142	69.6
Other, non-Hispanic	27	13.2
Missing	2	1.0
Maternal education		
High School Diploma or Less	16	7.8

Some College Credit	47	23.0
College Degree	71	34.8
Post-graduate Degree	70	34.3
	Mean (SD)	Range (min, max)
Maternal age at baseline visit (years)	31.5 (4.9)	(18.5, 46.4)

Infant Characteristics

	Total (n)	Percent (%)
Infant sex		
Male	103	50.5
Female	101	49.5
Infant race/ethnicity		
Black, non-Hispanic	26	12.7
Hispanic, any race	12	5.9
White, non-Hispanic	128	62.7
Other, non-Hispanic	37	18.1
Missing	1	0.5
Infant age at enrollment (baseline visit)		
2 weeks	127	62.3
2 months	77	3.7
Infant dietary intake at 4 months		
Breast milk exclusively	103	50.5
Formula exclusively	48	23.5
Combination breast milk and Formula	41	20.1
Missing	12	5.9
	Mean (SD)	Range (min, max)
Infant gestational age (weeks)	39.6 (1.1)	(37.0, 41.4)
Infant age at 4-month visit (days)	133.8 (13.5)	(109, 178)
Infant birth weight (kg)	3.5 (0.4)	(2.4, 4.5)
Birth weight z-score for gestational age and sex	-.01 (0.9)	(-1.8, 1.8)
Infant WLZ at 4-month visit	0.05 (0.9)	(-2.8, 2.3)

Note. SD = standard deviation; kg = kilograms; WLZ = weight-for-length z-score. If missing data occurred for any variable, it was included in the table.

Measures

Maternal Factors

YFAS. The YFAS is a 25-item self-report measure that operationalizes food addiction characteristics based on DSM-IV criteria for substance use disorders (Gearhardt et al., 2009). When completing the YFAS items, participants are instructed to think about foods high in refined carbohydrates and/or fats. Mothers completed the YFAS scale at baseline and were instructed to think about their eating in the past 12 months. YFAS questionnaire items prompted participants to endorse the frequency in which they experienced various addictive-like eating behaviors. The YFAS demonstrates good convergent, discriminant, and incremental validity as well as internal consistency (Gearhardt et al., 2009). In the current sample, YFAS scores demonstrated acceptable internal consistency (α = .67).

Dutch Eating Behaviors Questionnaire – Restraint Subscale (DEBQ-R). The Dutch Eating Behavior Questionnaire (DEBQ) is a 33-item self-report survey designed to capture various aspects of eating style including external eating, emotional eating, and restrained eating. Mothers completed the DEBQ at the 2-month study visit. This analysis focused on the 10-item restrained eating subscale (DEBQ-R),which measures intentions and attempts to reduce food intake or to avoid certain food types. All items were reported on a 5-point Likert scale (from 1 = "never" to 5 = "very often"). Scores on the DEBQ-R reflect the average of all items, with higher scores indicating more dietary

restraint. The DEBQ demonstrates good predictive validity and internal consistency (Van Strien et al., 1986; Van Strien et al., 2012). In the current sample, DEBQ-R scores demonstrated good internal consistency (α = .88).

Pre-Pregnancy BMI. Mothers' pre-pregnancy weight was obtained by self-report at baseline. Mothers were asked "Just before you got pregnant with [child's name], how much did you weigh?" and were asked to provide their pre-pregnancy weight in pounds. Maternal height was measured using a Seca 217 portable stadiometer at the baseline visit. Pre-pregnancy BMI was calculated as weight in kilograms divided by height in meters squared (see Table 3.2).

Infant Factors

Baby Eating Behaviors Questionnaire (BEBQ). The BEBQ is an 18-item parent-report questionnaire developed to capture variation in eating and appetitive traits during the first six months of infancy. The four subscales of the BEBQ are designed to assess general appetite, enjoyment of food, food responsiveness, slowness in eating, and satiety responsiveness. One additional item measures general appetite. The BEBQ instructs caregivers to respond according to their baby's feeding style for a "typical daytime feed." All items are reported on a 5-point Likert scale (from 1 = "never" to 5 = "always"). Scores are summed and averaged for each subscale with higher scores indicating increased reported expression of the feeding behavior or general appetite. The BEBQ has demonstrated good validity and good internal consistency (Llewellyn et

al., 2011; Oyama et al., 2021). In the current study, subscales for enjoyment of food (α = .70) and food responsiveness (α = .78) demonstrated acceptable validity. Subscales for satiety responsiveness (α = .44) and slowness in eating (α = .57) demonstrated poor validity and were excluded from the study.

Infant Hedonic Response to Sucrose. Infant hedonic response to sucrose was assessed using standardized methods validated for infants at 4 months of age (Rios et al., 2020; Steiner et al., 2001). Alternating doses of sterile water (0.2 mL) and sucrose solutions (24% and 50% sucrose; 0.2 mL) were administered to the central dorsal area of the infant's tongue via needleless syringe. Order of delivery of 24% and 50% sucrose solutions was counterbalanced across participants. Following administration of each solution (water, 24% sucrose, and 50% sucrose), infants were videotaped for a 30- or 60-second response phase to code for infant behavioral reactions evidenced to signal hedonic liking (i.e., tongue protrusion, lip smacking, and bringing fingers or hands to the mouth; Steiner et al., 2001). Response phases were divided into 10-second intervals that were coded for whether a behavioral reaction occurred during that time period. Next, the proportion of intervals in which the behavior occurred was calculated for each behavioral reaction, ranging from 0 (no behavior in any interval) to 1 (behavior occurred in every interval). A composite sucrose reactivity score was calculated by averaging the proportion of intervals that behavioral reactions occurred for all behavioral reactions that differentiated between sucrose and water (i.e., bringing fingers

to the mouth and lip smacking at 24% sucrose concentration and bringing fingers to the mouth, lip smacking, and tongue protrusion at 50% sucrose concentration; Rios et al., 2020). This sucrose paradigm was only administered to a subset of infants (n = 96) enrolled in the larger study.

Anthropometry. Infants were weighed without clothing or diaper to the nearest 0.1 kg with a Tanita BD-585 Digital Pediatric Baby Scale. Infant weights were duplicated and averaged to maximize accuracy. If weights differed by more than 0.1 kg, the measurements were repeated. Infant length was measured with an Ellard Instruments Acrylic Pediatric Stadiometer to the nearest 0.1 cm. The infant was aligned in the Frankfort horizontal plane with legs positioned according to standards by Shorr (1984). Infant length was measured twice and averaged. If length measurements differed by more than 0.2 cm, the infant was measured one more time. Infant measurements were taken at the 4-month study visit (see Table 3.2). All research staff were certified and recertified annually in infant and adult measurement technique. Infant WLZ was calculated based on age and sex-specific growth charts (WHO, 2006). Infant risk for overweight or obesity is defined as a weight-for-length≥85th percentile based on the WHO standards (2014; see Table 3.2).

Milk Consumption. To assess infant dietary intake, mothers responded to select questions from age-appropriate questionnaires developed by the Centers for Disease Control and Prevention (CDC, 2015; Grummer-Strawn et al., 2010). At the 4-month visit,

mothers were asked to report how many times in the past seven days their infant was fed breast milk, formula, or cow/soy/other milk types. Infants were categorized as exclusively fed breast milk, exclusively fed formula, or combination breast milk and formula (see Table 3.2).

Table 3.2. Maternal and Infant Variable Descriptives

	n	Mean (SD)/n (percent)	Range (min, max)
Maternal Food Addiction (YFAS)	204	1.7 (1.3)	(0, 7)
Maternal Dietary Restraint (DEBQ)	204	2.3 (0.7)	(1, 4)
Maternal Pre-pregnancy BMI	204	27.8 (7.2)	(17.0, 62.2)
Infant Enjoyment of Food (BEBQ)	192	4.5 (0.4)	(3, 5)
Infant Food Responsiveness (BEBQ)	189	2.2 (0.7)	(1, 5)
Infant General Appetite (BEBQ)	188	4.0 (0.9)	(1, 5)
Infant Sucrose Response	79	0.4 (0.1)	(0.1, 0.9)
Infant Risk for Overweight	198	44 (15.4%)*	(0, 1)**

Note. SD = standard deviation, *n = infants at risk for overweight according to WHO guidelines, **infant risk for overweight variable was dummy coded (0 = infant WLZ not at risk for infant overweight, 1 = WLZ at risk for infant overweight).

Data Analytic Plan

Statistical tests were completed using SAS 9.4 software and IBM SPSS Statistics 27.0 software. Missing data was removed with list-wise deletion. To address Aim 1, zero-order correlations were tested among maternal independent variables of interest (i.e., food addiction and dietary restraint). We hypothesized that food addiction and dietary restraint would not be significantly correlated at each time point. To address

Aim 3, all maternal risk factors (i.e., food addiction, dietary restraint, and pre-pregnancy BMI) were simultaneously included as predictors in four separate linear regression models predicting infant sucrose response, enjoyment of food, food responsiveness, and general appetite. This allowed us to assess associations with each maternal risk factor while controlling for the variance of the other maternal risk factors. Infant risk for overweight was dichotomized and dummy coded (0 = "no risk for infant overweight" and 1 = "risk for infant overweight") and logistic regression was used to assess associations of the maternal risk factors, included simultaneously in the model, with infant risk for overweight. Potential covariates that have been implicated in the prior literature as relevant to infant eating behaviors and risk for overweight were considered (Nisbett & Gurwitz, 1970; Rogers & Blissett, 2017). Covariates which demonstrated a significant association with the dependent variable of interest were included for each linear and logistic regression model (specific covariates included in each model are indicated in Table 3.3).

Results

Preliminary analyses were completed to verify that these data did not violate assumptions for linear regression including linear relationship, multivariate normality, multicollinearity, auto-correlation, and homoscedasticity (Poole & O'Farrell, 1971). Correlations between the maternal risk variables were calculated to assess for multicollinearity. Maternal food addiction and dietary restraint were each positively

correlated with pre-pregnancy BMI, respectively (r = .21, p < .01; r = .26, p < .001), but were not correlated with each other (r = .04; p = .57). Correlations (Pearson's r) between potential covariates and infant outcome variables were calculated to determine which covariates would be included in which models (p < .05; see Table 3.3).

Linear and logistic regression models revealed that maternal food addiction was associated with greater infant risk for overweight (β = .28, p = .04). Maternal dietary restraint was associated with lower infant appetite (β = -0.20, p = .005), but greater objectively measured infant hedonic response to sucrose (β = 0.24, p = .03). Maternal pre-pregnancy BMI was associated with maternal report of greater infant appetite (β = 0.27, p < .001). Findings are reported in Table 3.3.

Table 3.3. Associations between Maternal Risk Factors and Infant Eating Behaviors and Risk for Overweight at 4-months (n = 204)

							CI (95%), OR	
Model	Maternal IV	Infant DV	n	β	SE	Sig.	Lower	Upper
1	Food Addiction (YFAS)	Enjoyment of Food[1] (BEBQ)	192	-0.04	0.03	.62	0.01	0.11
	Dietary Restraint (DEBQ-R)			-0.08	0.05	.27	-0.14	0.04
	Pre-pregnancy BMI			0.14	0.01	.08	0.00	0.02
2	Food Addiction (YFAS)	Food Responsiveness[1, 2, 3] (BEBQ)	189	0.06	0.04	.42	0.00	0.01
	Dietary Restraint (DEBQ-R)			-0.12	0.06	.10	-0.23	0.02
	Pre-pregnancy BMI			-0.05	0.01	.47	-0.02	0.01
3	Food Addiction (YFAS)	General Appetite[1,2] (BEBQ)	188	-0.12	0.05	.10	-0.18	0.02
	Dietary Restraint (DEBQ-R)			-0.20	0.09	<.01	-0.41	-0.07
	Pre-pregnancy BMI			0.27	0.01	<.001	0.02	0.05
4	Food Addiction (YFAS)	Sucrose Response[4]	79	-0.06	0.01	.59	-0.03	0.02
	Dietary Restraint (DEBQ-R)			0.24	0.03	.03	0.01	0.11
	Pre-pregnancy BMI			0.06	0.00	.58	0.00	0.01
	Food Addiction (YFAS)			0.28	0.13	.04	1.0	1.7

5	Dietary Restraint (DEBQ-R)	Risk for overweight	198	-0.02	0.28	.93	0.6	1.7
	Pre-pregnancy BMI			0.05	0.03	.06	1.0	1.1

Note. CI = confidence interval, OR = odds ratio, YFAS = Yale Food Addiction Scale 2.0; DEBQ = Dutch Eating Behaviors Questionnaire, Restraint subscale; BMI = body mass index. Standardized beta coefficients (β) are reported to allow for comparison across models. to allow for comparison across models. Superscript numbers indicate covariates included in each model based on significant correlation with infant variables of interest: 1. Maternal education (coded 1=high school diploma or less, 2=some college credit, 3=college degree, 4=post-graduate degree), 2. Maternal age 3. Milk consumption (coded 1=exclusively fed breast milk, 2=exclusively fed formula, 3= breast milk and formula in combination). No covariates were associated with infant risk for overweight (all $p > .05$), 4. Child sex (coded 0=female, 1=male); no covariates were included in this model.

Discussion

In a sample of 204 infant-mother dyads, we simultaneously investigated the association of maternal risk factors (i.e., food addiction, dietary restraint, and pre-pregnancy BMI) with infant eating behaviors and risk for overweight at 4 months. Maternal food addiction was associated with greater infant risk for overweight. Maternal dietary restraint was associated with lower infant appetite, but greater infant hedonic response to sucrose. Higher maternal pre-pregnancy BMI was associated with greater infant appetite. Each of the present findings are discussed in more detail below.

Notably, maternal food addiction was the only significant predictor of infant risk for overweight. This finding is consistent with the existing literature that parental food addiction predicts higher child BMI z-scores and risk for obesity in children 5-12 years of age (Burrows et al., 2017). Given the significant association of maternal food addiction with infant risk for overweight, it is possible that related differences in maternal eating behaviors during prenatal development influence future weight status

of the offspring. Prior research has found that maternal food addiction is associated with greater eating in the absence of hunger during pregnancy and greater consumption of both minimally and highly processed foods during pregnancy (Lipsky et al., 2021). Thus, differences in prenatal dietary exposure could represent one plausible factor implicated in associations between maternal food addiction and infant risk for overweight. However, additional research is needed to shed light on the complex interaction among multiple possible mechanisms (e.g., genetics, individual differences in infant reward drive) which may underlie intergenerational transmission of maternal food addiction on infant risk for overweight.

Despite greater risk for overweight, these infants did not exhibit differences in their own responses to food according to maternal report of general appetite and objective measures of sucrose response. In a model that did not include covariates, maternal food addiction was associated with maternal report of infant food responsiveness (see Appendix F), though this effect became nonsignificant after accounting for covariates. In older children, maternal food addiction has been linked to constructs related to sensitivity to food reward and greater cravings in children (Burrows et al., 2017; Santos-Flores et al., 2021). Thus, it is also plausible that differences in infant appetite and eating behavior related to maternal food addiction may emerge later in development. At four months, most infants are still exclusively consuming milk (i.e., breast milk and/or formula; Clayton et al., 2013), and the inclusion of solid foods

later in childhood represents a dietary shift to foods with a wider range of rewarding properties, including foods thought to be more addictive (e.g., HP foods, high-fat and high-sugar foods; Schulte et al., 2015). Therefore, significant differences in food responsiveness or appetite may be more demonstrable later in childhood, after the addition of diverse solid foods to the diet. Future longitudinal research is needed to better understand the relationship between maternal food addiction and child eating behavior and overweight risk across prenatal, infant, and child stages of development.

Maternal dietary restraint was associated with greater sucrose response but maternal report of lower general appetite in the infants. There are several possible interpretations for these combined findings. One hypothesis is that because these infants have a greater hedonic response to sugars (e.g., those in breast milk and formula), they require less food to satiate their food drive and mothers are accurately assessing lower infant appetite (Rios et al., 2020). It is also possible that mothers' restraint of their own diets is associated with differences in interpretation of their infant's appetite (Powell et al., 2011). Research in older children has linked maternal dietary restraint to greater maternal monitoring or restriction of the child's diet (Birch & Fisher, 2000; Tiggemann & Lowes, 2002). Such lines of research indicate the possibility that maternal dietary restraint may be linked to a greater tendency to underestimate the infant's appetite. Therefore, it is also possible that these infants do not experience lower appetite due to greater sucrose responsiveness, but mothers with more restraint are

interpreting their children as having a lower appetite. Inclusion of more objective behavioral measures for infant appetite (e.g., milk sucking rate, volume of consumption; Carnell & Wardle, 2007) in future studies could help shed light on this topic. Overall, more research is needed to determine exactly how maternal dietary restraint, infant appetite, and infant hedonic response to sucrose are interrelated during this period of early infancy.

The present study also detected a significant association between pre-pregnancy BMI and maternal report of greater infant general appetite. In older children, maternal pre-pregnancy BMI is associated with some differences in appetitive traits, although findings have been mixed. For example, maternal pre-pregnancy weight status has been linked to poorer appetite regulation in girls but not boys aged 3-5 (Boone-Heinonen et al., 2019). The current study demonstrates that maternal pre-pregnancy BMI appears to be associated with greater appetite in early infancy as well. When accounting for contributions of maternal food addition and dietary restraint, the association between pre-pregnancy BMI and infant risk for overweight did not meet significance, although it was trend-level. This lack of significance is notable given the known heritability of body size, particularly during childhood (Elks et al., 2012). However, the underpinnings of BMI are complex and multifaceted (Hill et al., 2000), and research efforts have emphasized the importance of recognizing the heterogenous behavioral phenotypes that may underlie lifelong weight status (Brownell & Wadden, 1991). Notably, maternal

food addiction was associated with both infant risk for overweight and maternal pre-pregnancy BMI. These findings suggest that the behavioral phenotype of maternal food addiction may be more strongly related to infant risk for overweight than maternal pre-pregnancy BMI alone.

Empirical and Clinical Implications

This study sheds additional light on the direct association between maternal food addiction and dietary restraint. Some critiques of the food addiction construct have suggested that models of food addiction do not adequately account for the impacts of dietary restraint on addictive eating behaviors (Wiss & Avena, 2020). However, the present results are consistent with prior research findings that food addiction and dietary restraint are both correlated with BMI (Gearhardt et al., 2014; Van Strien et al., 2014), but are not correlated with each other (Gearhardt et al., 2016; Schulte & Gearhardt, 2017; Carter et al., 2019; Gearhardt et al., 2012, 2013). Moreover, this study suggests that maternal food addiction and dietary restraint are also correlated with unique outcomes in infant eating behaviors and risk for overweight. In sum, these findings support an empirical conceptualization of food addiction and dietary restraint as unique high-risk phenotypes which are both associated with higher BMI but are ultimately distinct constructs.

This divergence in outcomes between maternal food addiction and dietary restraint further highlights the need to tailor unique intervention recommendations for

each of these presentations. Given the lack of association between food addiction and dietary restraint in this sample, it is also likely that mothers who struggle with food addictions symptoms may be experiencing an opposite profile of behaviors (e.g., excessive eating) than mothers who report dietary restraint (e.g., restricted eating). Therefore, ideal intervention strategies and dietary recommendations to address food addiction and dietary restraint may also differ. For example, interventions for the food addiction phenotype might focus on skills to cope with food cravings and increase inhibitory control. In contrast, interventions for dietary restraint may focus of reducing rigid dietary rules and increasing flexibility. It is also plausible that the preventative interventions designed to reduce the potential for problematic eating behaviors in the offspring of these mothers may be more effective if tailored based on maternal factors. However, future research is needed to explore potential strategies and their efficacy.

Strengths, Limitations, and Future Directions

There are some important strengths to highlight in the current study. This was the first study to investigate maternal characteristics and infant behavior in dyads with infants as early as four months old. This allowed us to examine infant behaviors during a developmental period in which most infants have not been introduced to solid foods, which limits the impact of dietary variability in our findings. Relative to earlier developmental periods, by 4 months mothers have had more time to understand and observe their infants, which may increase their ability to accurately report on their

infants' eating behavior. Further, this study implemented best-practice measures for phenotyping infant anthropometry and behavior, which utilized both subjective (maternal report) and more objective behavioral (sucrose facial response coding) measures to assess infant outcomes.

There are also several limitations to consider. The cross-sectional sample does not allow us to determine how these associations may change over the course of early development, particularly as infants are introduced to solid foods. This is an important future direction. There are also some limitations to administering measures for maternal risk factors during the early post-partum period (i.e., infant age 2 weeks). Retrospective self-report of pre-pregnancy weight may increase error in the measure of maternal pre-pregnancy BMI. Additionally, the YFAS and DEBQ-R scales do not specify for participants to report on eating behaviors exclusively during the post-partum period. Mothers may have considered pre-pregnancy or prenatal eating behavior in their responses, and additional research is needed to better understand how food addiction and dietary restraint may differ across pre-pregnancy, prenatal, and post-partum stages. Finally, this study utilized a sample with limited geographical and sociocultural diversity. Moreover, the sucrose protocol was only administered to a smaller subset of infants (n = 96) and thus, may have been underpowered to detect significant effects. Future research is needed to assess how associations between these maternal and infant characteristics may generalize across larger and more diverse populations.

Conclusions

This study aimed to investigate how maternal characteristics including food addiction, dietary restraint, and pre-pregnancy body mass index (BMI) are associated with eating behaviors and risk for overweight in infancy. In this sample of 204 infant-mother dyads, maternal food addiction, dietary restraint, and pre-pregnancy BMI were each associated with distinct infant eating behaviors and risk for overweight at four months of age. Maternal food addiction was the only maternal variable associated with greater risk for infant overweight. Maternal dietary restraint was associated with maternal report of lower infant appetite, but objective measurement of greater infant hedonic liking of sugar. Higher maternal pre-pregnancy BMI was associated with maternal report of greater infant appetite. Findings highlight the potential for targeted prevention efforts early in development based on these distinct maternal risk factors. Additional research is needed to identify the mechanistic pathways driving the associations between maternal risk factors and early infancy eating- and weight-related outcomes. Finally, it will be important to investigate whether these early infant patterns predict the development of future high-risk eating behaviors or excessive weight gain across the course of development.

CHAPTER 4

Integration and Conclusions

Exploring the Bidirectional Relationship Between Food Addiction and Dietary Restraint

This dissertation aimed to advance our empirical understanding of the bidirectional relationship between food addiction and dietary restraint. Study 1 (Evaluating Bidirectional Predictive Pathways between Food Addiction and Dietary Restraint in Adolescents) utilized a longitudinal study design to evaluate directional pathways between food addiction and dietary restraint during adolescence, a critical period of development in which eating pathology often emerges (Mussel et al., 1995; Stice et al., 1998). In response to Aim 1, this study showed that food addiction and dietary restraint were correlated at a small to medium effect size at three distinct time points during adolescence. In response to Aim 2, longitudinal analyses revealed that food addiction predicted future dietary restraint, but dietary restraint did not predict future food addiction.

Study 2 (High-risk Maternal Predictors of Early Infancy Eating Behaviors and Risk for Overweight) sought to compare associations of maternal food addiction and

dietary restraint with early infant eating behaviors and risk for overweight in dyads of post-partum mothers and infants at four months of age. In response to Aim 1, this study revealed that food addiction and dietary restraint were not significantly associated with each other in a population of adult post-partum mothers. In response to Aim 3, this study also illustrated that maternal food addiction and dietary restraint were each associated with distinct infant eating behaviors and risk for overweight at four months of age.

Combined findings from Study 1 and Study 2 are consistent with prior studies which evidence an association between food addiction and dietary restraint in adolescence ((Alim et al., 2021; Gearhardt et al., 2013; Schiestl & Gearhardt, 2018) but the diminishment or disappearance of that link by adulthood (Gearhardt et al., 2016; Imperatori et al., 2019; Legendre & Bégin, 2020; Schulte & Gearhardt, 2017). It currently remains unclear why food addiction and dietary restraint appear to be more tightly linked in adolescents, but unrelated in adults. One possibility is that individuals are more likely to react to the early emergence of food addiction symptoms with attempts to restrict or reduce their dietary intake (Keel et al., 2007; Neumark-Sztainer et al., 2018). However, after multiple failed attempts to control eating or reduce body weight, individuals may become less likely to implement dietary restraint over time (Schiestl & Gearhardt, 2018). More extensive longitudinal research spanning adolescence and adulthood is needed in order to better understand the developmental course of the

relationship between food addiction and dietary restraint as well as the mechanisms which drive their association.

Given the evidence for associations between food addiction and dietary restraint in prior cross-sectional samples of adolescents (Alim et al., 2021; Gearhardt et al., 2013; Schiestl & Gearhardt, 2018), some researchers have theorized that dietary restraint may play a causal role in the development of food addiction symptoms (Wiss & Avena, 2020). However, longitudinal findings from Study 1 illustrate that food addiction appears to predict future dietary restraint, while dietary restraint did not significantly predict future food addiction. Thus, the present longitudinal analyses do not support a causal or etiological role of dietary restraint in food addiction models (see Figure 2.2). Rather, our findings support a model of food addiction in which dietary restraint is more likely to occur as a reaction to addictive eating symptoms and related outcomes (e.g., weight or shape concerns; see Figure 2.3; Lowe, 2015). Additional research is needed to investigate whether specific aspects of dietary restraint may further reinforce or exacerbate addictive eating qualities via intermittent patterns of access to HP foods or neural sensitization to the rewarding properties of HP foods (Avena et al., 2008).

Results from Study 2 additionally highlight the distinction between food addiction and dietary restraint constructs. Maternal food addiction and dietary restraint were each uniquely associated with different infant eating and weight characteristics, supporting the conceptualization of food addiction and dietary restraint as distinct

clinical constructs and the need to tailor intervention recommendations based on individual maternal eating phenotypes. An important direction for future research will be the development of effective intervention and prevention strategies specific to food addiction and dietary restraint for pre-natal and post-partum mothers.

Overall Limitations and Future Directions

In sum, the findings of the current dissertation have provided key insight into the bidirectional relationship between food addiction and dietary restraint from several key perspectives. Food addiction and dietary restraint appear to be more closely related during adolescents, an important developmental period when the onset of eating pathology and dieting behaviors frequently emerge, but less closely by adulthood. For those adolescents, food addiction appears to precede and predict dietary restraint, highlighting the need for interventions that target ways to reduce risk of addictive eating in teens. Additionally, food addiction and dietary restraint each map onto distinct infant eating and weight outcomes, reinforcing the conceptualization of food addiction and dietary restraint as distinct constructs that require tailored intervention strategies for prenatal and post-partum mothers.

However, the studies included in this dissertation were limited to relatively small, nondiverse, and well-resourced samples (see Table 2.1 and Table 3.2). It will be important for future research to assess whether the present findings are replicable in larger, more diverse participant samples. Such studies could also explore the potential

differences in effects across gender and sociocultural identities. Further, we recognize the need for more extensive longitudinal study designs. For example, it remains unknown how behaviors related to food addiction and dietary restraint emerge and develop from infancy into adolescence. Longitudinal data spanning these developmental stages could provide more information regarding phenotypes and mechanisms that may increase risk for both food addiction and dietary restraint. Such findings are critical for the development of more effective identification, prevention, or intervention strategies that could implemented earlier in the lifespan. Additionally, ongoing longitudinal data could help elucidate why the association between food addiction and dietary restraint appears to emerge in adolescence but diminish or disappear by adulthood.

Taken together, the combined findings of this dissertation aim to advance our empirical understanding of food addiction and dietary restraint as related, but distinct, constructs with a potentially bidirectional relationship which appears to evolve over time. A deeper understanding of the nature of the relationship between food addiction and dietary restraint that results from this dissertation has important implications for public health and clinical treatment recommendations.

APPENDICES

APPENDIX A

DSM-5 Substance Use Disorder and YFAS 2.0 Food Addiction Diagnostic Indicators

Table A.1. DSM-5 Substance Use Disorder and YFAS 2.0 Food Addiction Diagnostic Indicators

DSM-5 substance use disorder diagnostic criteria
Substance taken in larger amount and for longer period than intended
Persistent desire or repeated unsuccessful attempts to quit
Much time/activity to obtain, use, recover
Craving or strong urges to use the substance
Recurrent use resulting in failure to fulfill major obligations at work, school, or home
Continued use despite social or interpersonal problems
Important social, occupational, or recreational activities given up or reduced
Recurrent substance use in physically hazardous situations
Continued use despite physical or psychological problems
Tolerance to pleasurable or intoxicating effects of the substance
Characteristic withdrawal symptoms; substance taken to relieve withdrawal

YFAS 2.0 food addiction clinical indicators
Food consumed in larger quantities or over long period of time than intended
Persistent desire or unsuccessful efforts to reduce consumption of certain foods
Considerable time spent to obtain, consume, or recover from effects of food
Craving or strong urges to consume certain foods
Continued consumption despite failure to fulfill major role obligation
Continued consumption despite social or interpersonal problems
Giving up important social, occupational, or recreational activities because of food consumption

Recurrent consumption of food in physically hazardous situations
Continuing to eat certain foods despite physical or psychological problems
Tolerance to pleasurable effects of food
Characteristic withdrawal symptoms; certain foods consumed to relieve withdrawal

APPENDIX B

Questionnaire Items

Table B.1 YFAS Questionnaire Items

Yale Food Addiction Scale (YFAS)

1. I find that when I start eating certain foods, I end up eating much more than planned
2. I find myself continuing to consume certain foods even though I am no longer hungry
3. I eat to the point where I feel physically ill
4. Not eating certain types of food or cutting down on certain types of food is something I worry about
5. I spend a lot of time feeling sluggish or fatigued from overeating
6. I find myself constantly eating certain foods throughout the day
7. I find that when certain foods are not available, I will go out of my way to obtain them. For example, I will drive to the store to purchase certain foods even though I have other options available to me at home.
8. There have been times when I consumed certain foods so often or in such large quantities that I started to eat food instead of working, spending time with my family or friends, or engaging in other important activities or recreational activities I enjoy.
9. There have been times when I consumed certain foods so often or in such large quantities that I spent time dealing with negative feelings from overeating instead of working, spending time with my family or friends, or engaging in other important activities or recreational activities I enjoy.
10. There have been times when I avoided professional or social situations where certain foods were available because I was afraid I would overeat.
11. There have been times when I avoided professional or social situations because I was not able to consume certain foods there.
12. I have had withdrawal symptoms such as agitation, anxiety, or other physical symptoms when I cut down or stopped eating certain foods. (Please do NOT

13. include withdrawal symptoms caused by cutting down on caffeinated beverages such as soda pop, coffee, tea, energy drinks, etc.)
14. I have consumed certain foods to prevent feelings of anxiety, agitation, or other physical symptoms that were developing. (Please do NOT include consumption of caffeinated beverages such as soda pop, coffee, tea, energy drinks, etc.)
15. I have found that I have elevated desire for or urges to consume certain foods when I cut down or stop eating them.
16. My behavior with respect to food and eating causes significant distress.
17. I experience significant problems in my ability to function effectively (daily routine, job/school, social activities, family activities, health difficulties) because of food and eating.
18. My food consumption has caused significant psychological problems such as depression, anxiety, self-loathing, or guilt.
19. My food consumption has caused significant physical problems or made a physical problem worse.
20. I kept consuming the same types of food or the same amount of food even though I was having emotional and/or physical problems.
21. Over time, I have found that I need to eat more and more to get the feeling I want, such as reduced negative emotions or increased pleasure.
22. I have found that eating the same amount of food does not reduce my negative emotions or increase pleasurable feelings the way it used to.
23. I want to cut down or stop eating certain kinds of food.
24. I have tried to cut down or stop eating certain kinds of food.
25. I have been successful at cutting down or not eating these kinds of food
26. How many times in the past year did you try to cut down or stop eating certain foods altogether?

Note. Participants are instructed to respond to items based on the last 12 months and to consider foods with highest addictive potential (i.e., HP foods). Items 1-16 are reported on a 5-point Likert scale (0 = never; 1 = once a month; 2 = 2-4 times a month, 3 = 3 times a week, 4 = 4 or more times a week). Items 17-25 are coded yes/no (0 = no, 1 = yes). Item 25 was reported based on number of times in past 21 days (1 = 1 time, 2 = 2 times, 3 = 3times, 4 = 4 times, 5 = 5 or more times).

Table B.2. dYFAS-C 2.0 Questionnaire Items

Yale Food Addiction Scale for Children 2.0 (dYFAS-C 2.0)

---

1. When I started to eat certain foods, I found it hard to stop.
2. I kept eating certain foods even though I was not hungry.

3. I ate until my stomach hurt or I felt sick.
4. I worried about cutting down on certain foods but ate them anyway.
5. I spent a lot of time feeling tired from eating too much.
6. I ate certain foods all day long.
7. If I could not find a food I wanted, I tried hard to get it. (examples: asked a friend to get it for me, found a vending machine, snuck food when people weren't looking)
8. When I cut down on or stopped eating certain foods, I felt angry, upset or sad.
9. When I cut down or stopped eating certain foods, I craved them a lot more.
10. I kept eating too much even though it made me feel sad, nervous, or guilty.
11. I kept eating too much even though it made me unhealthy.
12. When I ate the same amount of food, it didn't make me feel as good as it used to. (examples: feel happy, calm, relaxed)
13. I really wanted to cut down on or stop eating certain kinds of foods, but I just couldn't.
14. I was craving certain foods so much that I felt like I had to eat them right away.
15. I tried to cut down on certain foods, but it didn't work.

Note. Participants are instructed to respond to items based on the last 12 months and to consider foods with highest addictive potential (i.e., HP foods). All items are reported on a 5-point Likert scale (from 0 = never to 4 = always).

Table B.3. DEBQ-R Questionnaire Items

Dutch Eating Behaviors Questionnaire Restraint Subscale

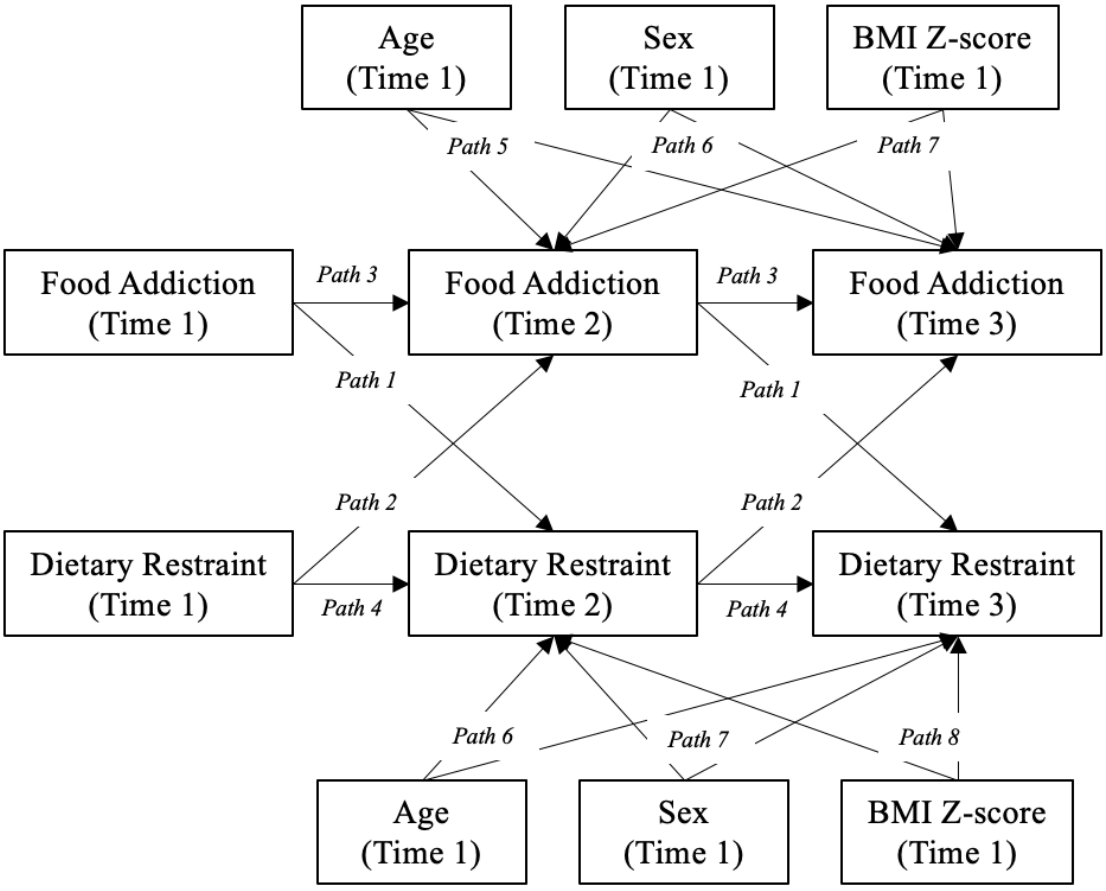
1. If you have put on weight, do you eat less than you usually do?
2. Do you try to eat less at mealtimes than you would like to eat?
3. How often do you refuse food or drink offered because you are concerned about your weight? *
4. Do you watch exactly what you eat?
5. Do you deliberately eat foods that are slimming?
6. When you have eaten too much, do you eat less than usual the following days?
7. Do you deliberately eat less in order to not become heavier?
8. How often do you try not to eat between meals because you are watching your weight?
9. How often in the evening do you try not to eat because you are watching you weight?
10. Do you take into account your weight with what you eat?

Note. Participants are instructed to consider if each item is true about themselves.. All items are reported on a 5-point Likert scale from 1 (“never”) to 5 (“very often).

APPENDIX C

Supplementary Visual Materials for Adjusted Structural Equation Models with Covariates

Figure C.1. Path Diagram for Adjusted Cross-lagged Panel Analysis among Food Addiction, Dietary Restraint, and Covariates (Age, Gender, and BMI z-score)



Note. Path 1: food addiction as a predictor for future dietary restraint; Path 2: dietary restraint as a predictor for future food addiction; Path 3: auto-regressive path for food addiction over time; Path 4:

auto-regressive path for dietary restraint over time; Paths 5, 6, and 7: confounding effects of covariates (age, gender, and BMI z-Score).

Table C.1. Standardized Regression Coefficients from Adjusted Structural Equation Models with Covariates

Path	Predictor	Outcome	*b*	SE	*Z*	*p*	95% CI lower	95% CI upper
1	Food addiction	Dietary restraint	0.22	0.06	3.76	< .001	0.11	0.34
2	Dietary restraint	Food addiction	0.07	0.06	1.22	0.22	-0.04	0.17
3	Food addiction	Food addiction	0.58	0.05	10.84	< .001	0.48	0.68
4	Dietary restraint	Dietary restraint	0.55	0.06	9.20	< .001	0.43	0.66
5	Age	Dietary restraint	0.01	0.05	0.25	.80	-0.09	0.12
6	Gender	Dietary restraint	0.05	0.11	0.44	.66	-0.16	0.26
7	BMI	Dietary restraint	0.10	0.06	1.62	.10	-0.02	0.23
8	Age	Food addiction	0.09	0.05	1.92	.06	-0.002	0.19
9	Gender	Food addiction	0.15	0.10	1.47	.14	-0.05	0.34
10	BMI	Food addiction	0.01	0.06	0.18	.86	-0.11	0.13

Note. X2(22) = 79.74; SRMR = 0.14; CFI = 0.83. A post-hoc sensitivity analysis using pwrSEM (Wang & Rhemtulla, in press) with 10,000 simulations and a seed of 23 indicated that our model had 99% power to detect an effect of this size for Path 1, and 28% power to detect an effect of this size for Path 2. Predictor variables were measured at T1, outcome variables were measured at times T2 and T3.

APPENDIX D

Associations between Maternal Risk Factors and Infant Eating Behaviors and Risk for Overweight at 4-months without Covariates

Table D.1. Associations between Maternal Risk Factors and Infant Eating Behaviors and Risk for Overweight at 4-months without Covariates (n = 204)

							CI (95%), OR	
Model	Maternal IV	Infant DV	n	β	SE	Sig.	Lower	Upper
1	Food Addiction (YFAS)	EF	192	-0.03	0.03	.97	0.05	0.05
	Dietary Restraint (DEBQ-R)			-0.10	0.05	.16	-0.15	0.03
	Pre-pregnancy BMI			0.17	0.01	.03	0.00	0.02
2	Food Addiction (YFAS)	FR	189	0.18	0.04	.02	0.00	0.01
	Dietary Restraint (DEBQ-R)			-0.13	0.07	.08	-0.23	0.02
	Pre-pregnancy BMI			-0.02	0.01	.78	-0.02	0.01
3	Food Addiction (YFAS)	GA	188	-0.07	0.05	.36	-0.14	0.05
	Dietary Restraint (DEBQ-R)			-0.22	0.09	<.01	-0.44	-0.10
	Pre-pregnancy BMI			0.29	0.01	<.001	0.02	0.05
4	Food Addiction (YFAS)	SR	79	-0.11	0.01	.34	-0.04	0.01
	Dietary Restraint (DEBQ-R)			0.24	0.03	.03	0.01	0.11
	Pre-pregnancy BMI			0.11	0.00	.33	0.00	0.01
5	Food Addiction (YFAS)	OW	198	0.28	0.13	.04	1.0	1.7
	Dietary Restraint (DEBQ-R)			-0.02	0.28	.93	0.6	1.7
	Pre-pregnancy BMI			0.05	0.03	.06	1.0	1.1

Note. CI = confidence interval, OR = odds ratio, YFAS = Yale Food Addiction Scale 2.0; DEBQ = Dutch Eating Behaviors Questionnaire, Restraint subscale; BMI = body mass index, DV = dependent variable, EF = Enjoyment of Food (BEBQ subscale), FR = Food Responsiveness (BEBQ subscale), GA = General Appetite (BEBQ subscale), SR = Sucrose Response (Rios et al., 2020), OW = Risk for Overweight (WHO, 2014). Standardized beta coefficients (β) are reported to allow for comparison across models.

BIBLIOGRAPHY

REFERENCES

Alim, N., Gokustun, K., Caliskan, G., & Besler, Z. (2021). Do Disordered Eating Behaviors Have an Effect on Food Addiction? *Health Behavior and Policy Review*, *8*(4). https://doi.org/10.14485/HBPR.8.4.4

Allen, K. L., Byrne, S. M., & McLean, N. J. (2011). The dual-pathway and cognitive-behavioural models of binge eating: prospective evaluation and comparison. *European Child & Adolescent Psychiatry 2011 21:1*, *21*(1), 51–62. https://doi.org/10.1007/S00787-011-0231-Z

American Psychiatric Association. (2013). *Diagnostic and Statistical Manual of Mental Disorders*. American Psychiatric Association. https://doi.org/10.1176/appi.books.9780890425596

Andrés, A., & Saldaña, C. (2014). Body dissatisfaction and dietary restraint influence binge eating behavior. *Nutrition Research*, *34*(11), 944–950. https://doi.org/10.1016/J.NUTRES.2014.09.003

Avena, N. M., Rada, P., & Hoebel, B. G. (2008). Evidence for sugar addiction: Behavioral and neurochemical effects of intermittent, excessive sugar intake. In *Neuroscience and Biobehavioral Reviews* (Vol. 32, Issue 1, pp. 20–39). NIH Public Access.

https://doi.org/10.1016/j.neubiorev.2007.04.019

Banasiak, S. J., Wertheim, E. H., Koerner, J., & Voudouris, N. J. (2001). Test-retest reliability and internal consistency of a variety of measures of dietary restraint and body concerns in a sample of adolescent girls. *International Journal of Eating Disorders*, *29*(1), 85–89. https://doi.org/10.1002/1098-108X(200101)29:1<85::AID-EAT14>3.0.CO;2-G

Birch, L. L., & Fisher, J. O. (2000). Mothers' child-feeding practices influence daughters' eating and weight. *American Journal of Clinical Nutrition*, *71*(5), 1054–1061. https://doi.org/10.1093/ajcn/71.5.1054

Birch, L., Savage, J. S., & Ventura, A. (2007). Influences on the Development of Children's Eating Behaviours: From Infancy to Adolescence. *Canadian Journal of Dietetic Practice and Research*, *68*(1), s1–s56. /pmc/articles/PMC2678872/

Boone-Heinonen, J., Weeks, H. M., Sturza, J., Miller, A. L., Lumeng, J. C., & Bauer, K. W. (2019). Prenatal predictors of objectively measured appetite regulation in low-income toddlers and preschool-age children. *Pediatric Obesity*, *14*(11). https://doi.org/10.1111/ijpo.12554

Borisenkov, M. F., Popov, S. V., Pecherkina, A. A., Dorogina, O. I., Martinson, E. A., Vetosheva, V. I., Gubin, D. G., Solovieva, S. V., Turovinina, E. F., & Symaniuk, E. E. (2020). Food addiction in young adult residents of Russia: Associations with emotional and anthropometric characteristics. *European Eating Disorders Review*,

28(4), 465–472. https://doi.org/10.1002/ERV.2731

Brewerton, T., Brewerton, T. D., Dansky, B. S., Kilpatrick, D. G., & O'neil, P. M. (2000). Which comes first in the pathogenesis of bulimia nervosa: Dieting or bingeing? Cite this paper Which Comes First in the Pathogenesis of Bulimia Nervosa: Dieting or Bingeing? *Int J Eat Disord*, *28*, 259–264.

Brown, A., Flint, S. W., & Batterham, R. L. (2022). Pervasiveness, impact and implications of weight stigma. *EClinicalMedicine*, *47*, 101408. https://doi.org/10.1016/J.ECLINM.2022.101408

Brownell, K. D., & Wadden, T. A. (1991). The heterogeneity of obesity: fitting treatments to individuals. *Behavior Therapy*, *22*(2), 153–177. https://doi.org/10.1016/S0005-7894(05)80174-1

Burrows, T., Skinner, J., Joyner, M. A., Palmieri, J., Vaughan, K., & Gearhardt, A. N. (2017). Food addiction in children: Associations with obesity, parental food addiction and feeding practices. *Eating Behaviors*, *26*, 114–120. https://doi.org/10.1016/j.eatbeh.2017.02.004

Carnell, S., & Wardle, J. (2007). Measuring behavioural susceptibility to obesity: Validation of the child eating behaviour questionnaire. *Appetite*, *48*(1), 104–113. https://doi.org/10.1016/J.APPET.2006.07.075

Carter, J. C., Van Wijk, M., & Rowsell, M. (2019). Symptoms of 'food addiction' in binge eating disorder using the Yale Food Addiction Scale version 2.0. *Appetite*, *133*, 362–

369. https://doi.org/10.1016/J.APPET.2018.11.032

Cassin, S., & Sockalingam, S. (2021). The Clinical Utility of Food Addiction and Eating Addiction. In *The Clinical Utility of Food Addiction and Eating Addiction*. https://doi.org/10.3390/books978-3-0365-1109-2

Centers for Disease Control and Prevention. (2015). *About Child & Teen BMI | Healthy Weight | CDC*. https://www.cdc.gov/healthyweight/assessing/bmi/childrens_bmi/about_childrens_bmi.html

Clayton, H. B., Li, R., Perrine, C. G., & Scanlon, K. S. (2013). Prevalence and reasons for introducing infants early to solid foods: Variations by milk feeding type. *Pediatrics*, *131*(4). https://doi.org/10.1542/peds.2012-2265

Crews, F., He, J., & Hodge, C. (2007). Adolescent cortical development: A critical period of vulnerability for addiction. *Pharmacology Biochemistry and Behavior*, *86*(2), 189–199. https://doi.org/10.1016/J.PBB.2006.12.001

Elks, C. E., Hoed, M. den, Zhao, J. H., Sharp, S. J., Wareham, N. J., Loos, R. J. F., & Ong, K. K. (2012). Variability in the heritability of body mass index: A systematic review and meta-regression. *Frontiers in Endocrinology*, *3*(FEB), 29. https://doi.org/10.3389/FENDO.2012.00029/BIBTEX

Fairburn, C. G., Cooper, Z., & Shafran, R. (2003). Cognitive behaviour therapy for eating disorders: a "transdiagnostic" theory and treatment. *Behaviour Research and Therapy*,

41, 509–528. https://doi.org/10.1016/S0005-7967(02)00088-8

Gearhardt, A. N., Boswell, R. G., & White, M. A. (2014). The association of "food addiction" with disordered eating and body mass index. *Eating Behaviors*, *15*(3), 427–433. https://doi.org/10.1016/J.EATBEH.2014.05.001

Gearhardt, A. N., Corbin, W. R., & Brownell, K. D. (2009). Preliminary validation of the Yale Food Addiction Scale. *Appetite*, *52*(2), 430–436. https://doi.org/10.1016/J.APPET.2008.12.003

Gearhardt, A. N., Corbin, W. R., & Brownell, K. D. (2016). Development of the Yale Food Addiction Scale Version 2.0. *Psychology of Addictive Behaviors*, *30*(1), 113–121. https://doi.org/10.1037/adb0000136

Gearhardt, A. N., White, M. A., Masheb, R. M., & Grilo, C. M. (2013). An examination of food addiction in a racially diverse sample of obese patients with binge eating disorder in primary care settings. *Comprehensive Psychiatry*, *54*(5), 500–505. https://doi.org/10.1016/J.COMPPSYCH.2012.12.009

Gearhardt, A. N., White, M. A., Masheb, R. M., Morgan, P. T., Crosby, R. D., & Grilo, C. M. (2012). An Examination of the Food Addiction Construct in Obese Patients with Binge Eating Disorder. *International Journal of Eating Disorders*, *45*(5), 657–663. https://doi.org/10.1002/eat.20957

Gearhardt, A. N., White, M. A., & Potenza, M. N. (2011). Binge Eating Disorder and Food Addiction. *Current Drug Abuse Reviews*, *4*(3), 201. /pmc/articles/PMC3671377/

Goldschmidt, A. B., Aspen, V. P., Sinton, M. M., Tanofsky-Kraff, M., & Wilfley, D. E. (2008). Disordered Eating Attitudes and Behaviors in Overweight Youth. *Obesity*, *16*(2), 257–264. https://doi.org/10.1038/OBY.2007.48

Goldschmidt, A. B., Wall, M., Loth, K. A., Le Grange, D., & Neumark-Sztainer, D. (2012). Which Dieters Are at Risk for the Onset of Binge Eating? A Prospective Study of Adolescents and Young Adults. *Journal of Adolescent Health*, *51*(1), 86–92. https://doi.org/10.1016/J.JADOHEALTH.2011.11.001

Goodrick, G. K., Poston, W. S. C., Kimball, K. T., Reeves, R. S., & Foreyt, J. P. (1998). Nondieting versus dieting treatment for overweight binge-eating women. *Journal of Consulting and Clinical Psychology*, *66*(2), 363–368. https://doi.org/10.1037/0022-006X.66.2.363

Grilo, C. M., & Masheb, R. M. (2000). Onset of dieting vs binge eating in outpatients with binge eating disorder. *International Journal of Obesity*, *24*(4), 404–409. https://doi.org/10.1038/sj.ijo.0801171

Grilo, Carlos M., Masheb, R. M., & Wilson, G. T. (2001). A comparison of different methods for assessing the features of eating disorders in patients with binge eating disorder. *Journal of Consulting and Clinical Psychology*, *69*(2), 317–322. https://doi.org/10.1037/0022-006X.69.2.317

Grilo, Carlos M., Masheb, R. M., Wilson, G. T., Gueorguieva, R., & White, M. A. (2011). Cognitive-behavioral therapy, behavioral weight loss, and sequential treatment for

obese patients with binge-eating disorder: A randomized controlled trial. *Journal of Consulting and Clinical Psychology*, *79*(5), 675–685. https://doi.org/10.1037/a0025049

Groesz, L. M., & Stice, E. (2007). An experimental test of the effects of dieting on bulimic symptoms: The impact of eating episode frequency. *Behaviour Research and Therapy*, *45*(1), 49–62. https://doi.org/10.1016/J.BRAT.2006.01.010

Grummer-Strawn, L. M., Reinold, C., & Krebs, N. F. (2010). Use of world health organization and CDC growth charts for children aged 0-59 months in the United States. *Morbidity and Mortality Weekly Report*, *59*(RR-9), 1–14. http://www.ncbi.nlm.nih.gov/pubmed/20829749

Harris, J. L., Yokum, S., & Fleming-Milici, F. (2021). Hooked on Junk: Emerging Evidence on How Food Marketing Affects Adolescents' Diets and Long-Term Health. In *Current Addiction Reports* (Vol. 8, Issue 1, pp. 19–27). https://doi.org/10.1007/s40429-020-00346-4

Herman, C. P., & Polivy, J. (1975). Anxiety, restraint, and eating behavior. *Journal of Abnormal Psychology*, *84*(6), 666–672. https://doi.org/10.1037/0021-843X.84.6.666

Herman, C. P., & Polivy, J. (1990). From dietary restraint to binge eating: Attaching causes to effects. *Appetite*, *14*(2), 123–125. https://doi.org/10.1016/0195-6663(90)90009-W

Heslehurst, N., Vieira, R., Akhter, Z., Bailey, H., Slack, E., Ngongalah, L., Pemu, A., & Rankin, J. (2019). The association between maternal body mass index and child

obesity: A systematic review and meta-analysis. *PLOS Medicine*, *16*(6), e1002817. https://doi.org/10.1371/JOURNAL.PMED.1002817

Heude, B., Kettaneh, A., Rakotovao, R., Bresson, J. L., Borys, J. M., Ducimetière, P., & Charles, M. A. (2005). Anthropometric relationships between parents and children throughout childhood: the Fleurbaix–Laventie Ville Santé Study. *International Journal of Obesity 2005 29:10*, *29*(10), 1222–1229. https://doi.org/10.1038/sj.ijo.0802920

Hilbert, A., Pike, K. M., Goldschmidt, A. B., Wilfley, D. E., Fairburn, C. G., Dohm, F.-A., Timothy Walsh, B., & Striegel Weissman, R. (2014). Risk factors across the eating disorders. *Psychiatry Research*, *220*(1–2), 500–506. http://dx.doi.org/10.1016/j.psychres.2014.05.054

Hill, A. J., & Franklin, J. A. (1998). Mothers, daughters and dieting: Investigating the transmission of weight control. *British Journal of Clinical Psychology*, *37*(1), 3–13. https://doi.org/10.1111/j.2044-8260.1998.tb01275.x

Imperatori, C., Fabbricatore, M., Lester, D., Manzoni, G. M., Gianluca Castelnuovo, ·, Raimondi, G., & Innamorati, · Marco. (2019). Psychometric properties of the modified Yale Food Addiction Scale Version 2.0 in an Italian non-clinical sample. *Eating and Weight Disorders - Studies on Anorexia, Bulimia and Obesity*, *24*, 37–45. https://doi.org/10.1007/s40519-018-0607-x

Ivezaj, V., White, M. A., & Grilo, C. M. (2016). Examining binge-eating disorder and food addiction in adults with overweight and obesity. *Obesity*, *24*(10), 2064–2069.

https://doi.org/10.1002/OBY.21607

Johnson, F, Pratt, M., & Wardle, J. (2012). Dietary restraint and self-regulation in eating behavior. In *International Journal of Obesity* (Vol. 36, Issue 5, pp. 665–674). https://doi.org/10.1038/ijo.2011.156

Johnson, Fiona, & Wardle, J. (2005). Dietary restraint, body dissatisfaction, and psychological distress: A prospective analysis. *Journal of Abnormal Psychology*, *114*(1), 119–125. https://doi.org/10.1037/0021-843X.114.1.119

Keel, P. K., Baxter, M. G., Heatherton, T. F., & Joiner, T. E. (2007). A 20-year longitudinal study of body weight, dieting, and eating disorder symptoms. *Journal of Abnormal Psychology*, *116*(2), 422–432. https://doi.org/10.1037/0021-843X.116.2.422

Kenny, D. A., & Harackiewicz, J. M. (1979). Cross-lagged panel correlation: Practice and promise. *Journal of Applied Psychology*, *64*(4), 372–379. https://doi.org/10.1037/0021-9010.64.4.372

Khakoo, G. A., & Lack, G. (2004). Introduction of solids to the infant diet. *Archives of Disease in Childhood*, *89*(4), 295–295. https://doi.org/10.1136/ADC.2003.039016

Kirkley, B. G., Burge, J. C., & Ammerman, A. (1988). Dietary restraint, binge eating, and dietary behavior patterns. *International Journal of Eating Disorders*, *7*(6), 771–778. https://doi.org/10.1002/1098-108X(198811)7:6<771::AID-EAT2260070606>3.0.CO;2-F

Kiyici, S., Koca, N., Sigirli, D., Aslan, B. B., Guclu, M., & Kisakol, G. (2020). Food Addiction Correlates with Psychosocial Functioning More Than Metabolic

Parameters in Patients with Obesity. *Metabolic Syndrome and Related Disorders*, *18*(3), 161–167. https://doi.org/10.1089/met.2019.0108

Kosheleff, A. R., Araki, J., Hsueh, J., Le, A., Quizon, K., Ostlund, S. B., Maidment, N. T., & Murphy, N. P. (2018). Pattern of access determines influence of junk food diet on cue sensitivity and palatability. *Appetite*, *123*, 135–145. https://doi.org/10.1016/J.APPET.2017.12.009

Laessle, R. G., Tuschl, R. J., Kotthaus, B. C., & Prike, K. M. (1989). A comparison of the validity of three scales for the assessment of dietary restraint. *Journal of Abnormal Psychology*, *98*(4), 504–507. https://doi.org/10.1037/0021-843X.98.4.504

Lee, A., Ho, M., & Keung, V. (2010). Healthy School as an Ecological Model for Prevention of Childhood Obesity. *Research in Sports Medicine*, *18*(1), 49–61. https://doi.org/10.1080/15438620903423825

Legendre, M., & Bégin, C. (2020). French validation of the addiction-like eating behavior scale and its clinical implication. *Eating and Weight Disorders - Studies on Anorexia, Bulimia and Obesity 2020 26:6*, *26*(6), 1893–1902. https://doi.org/10.1007/S40519-020-01039-7

Lipsky, L. M., Burger, K. S., Faith, M. S., Siega-Riz, A. M., Liu, A., Shearrer, G. E., & Nansel, T. R. (2021). Pregnant Women Consume a Similar Proportion of Highly vs Minimally Processed Foods in the Absence of Hunger, Leading to Large Differences in Energy Intake. *Journal of the Academy of Nutrition and Dietetics*, *121*(3),

446–457. https://doi.org/10.1016/J.JAND.2020.09.036

Llewellyn, C. H., van Jaarsveld, C. H. M., Johnson, L., Carnell, S., & Wardle, J. (2011). Development and factor structure of the Baby Eating Behaviour Questionnaire in the Gemini birth cohort. *Appetite*, *57*(2), 388–396. https://doi.org/10.1016/J.APPET.2011.05.324

Lowe, M. R. (2015). Dieting: proxy or cause of future weight gain? *Obesity Reviews*, *16*(S1), 19–24. https://doi.org/10.1111/OBR.12252

Lowe, Michael R. (1993). The effects of dieting on eating behavior: A three-factor model. *Psychological Bulletin*, *114*(1), 100–121. https://doi.org/10.1037/0033-2909.114.1.100

Lowe, Michael R., Annunziato, R. A., Markowitz, J. T., Didie, E., Bellace, D. L., Riddell, L., Maille, C., McKinney, S., & Stice, E. (2006). Multiple types of dieting prospectively predict weight gain during the freshman year of college. *Appetite*, *47*(1), 83–90. https://doi.org/10.1016/J.APPET.2006.03.160

Lowe, Michael R., Butryn, M. L., Didie, E. R., Annunziato, R. A., Thomas, J. G., Crerand, C. E., Ochner, C. N., Coletta, M. C., Bellace, D., Wallaert, M., & Halford, J. (2009). The Power of Food Scale. A new measure of the psychological influence of the food environment. *Appetite*, *53*(1), 114–118. https://doi.org/10.1016/J.APPET.2009.05.016

Lowe, Michael R., Doshi, S. D., Katterman, S. N., & Feig, E. H. (2013). Dieting and restrained eating as prospective predictors of weight gain. *Frontiers in Psychology*, *4*, 577. https://doi.org/10.3389/fpsyg.2013.00577

Maxwell, A. L., Gardiner, E., & Loxton, N. J. (2020). Investigating the relationship between reward sensitivity, impulsivity, and food addiction: A systematic review. *European Eating Disorders Review*, *28*(4), 368–384. https://doi.org/10.1002/ERV.2732

Meule, A., & Gearhardt, A. N. (2019). Ten Years of the Yale Food Addiction Scale: a Review of Version 2.0. *Current Addiction Reports 2019 6:3*, *6*(3), 218–228. https://doi.org/10.1007/S40429-019-00261-3

Micali, N., Daniel, R. M., Ploubidis, G. B., & De Stavola, B. L. (2018). Maternal prepregnancy weight status and adolescent eating disorder behaviors. *Epidemiology*, *29*(4), 579–589. https://doi.org/10.1097/EDE.0000000000000850

Mussell, M. P., Mitchell, J. E., Weller, C. L., Raymond, N. C., Crow, S. J., & Crosby, R. D. (1995). Onset of binge eating, dieting, obesity, and mood disorders among subjects seeking treatment for binge eating disorder. *International Journal of Eating Disorders*, *17*(4), 395–401. https://doi.org/10.1002/1098-108X(199505)17:4<395::AID-EAT2260170412>3.0.CO;2-I

Neumark-Sztainer, D., Wall, M. M., Chen, C., Larson, N. I., Christoph, M. J., & Sherwood, N. E. (2018). Eating, Activity, and Weight-related Problems From Adolescence to Adulthood. *American Journal of Preventive Medicine*, *55*(2), 133–141. https://doi.org/10.1016/J.AMEPRE.2018.04.032

Nisbett, R. E., & Gurwitz, S. B. (1970). Weight, sex, and the eating behavior of human newborns. *Journal of Comparative and Physiological Psychology*, *73*(2), 245–253.

https://doi.org/10.1037/H0030250

Ogden, J., & Steward, J. (2000). The role of the mother-daughter relationship in explaining weight concern. *International Journal of Eating Disorders*, *28*(1), 78–83. https://doi.org/10.1002/(SICI)1098-108X(200007)28:1<78::AID-EAT9>3.0.CO;2-N

Oliveira, E., Kim, H. S., Lacroix, E., Vasques, M. de F., Durante, C. R., Pereira, D., Cabral, J. R., Bernstein, P. S., Garcia, X., Ritchie, E. V., & Tavares, H. (2020). The Clinical Utility of Food Addiction: Characteristics and Psychosocial Impairments in a Treatment-Seeking Sample. *Nutrients 2020, Vol. 12, Page 3388*, *12*(11), 3388. https://doi.org/10.3390/NU12113388

Oyama, S., Arslanian, K. J., Fidow, U. T., Naseri, T., Soti-Ulberg, C., & Hawley, N. L. (2021). Factorial validation analysis of the Baby and Children's Eating Behavior Questionnaires in Samoa. *Eating Behaviors*, *42*, 101530. https://doi.org/10.1016/J.EATBEH.2021.101530

Polivy, J., Herman, C. P., & Mills, J. S. (2020). What is restrained eating and how do we identify it? *Appetite*, *155*, 104820. https://doi.org/10.1016/J.APPET.2020.104820

Poole, M. A., & O'Farrell, P. N. (1971). The Assumptions of the Linear Regression Model. *Transactions of the Institute of British Geographers*, *52*, 145. https://doi.org/10.2307/621706

Powell, F. C., Farrow, C. V., & Meyer, C. (2011). Food avoidance in children. The influence of maternal feeding practices and behaviours. *Appetite*, *57*(3), 683–692.

https://doi.org/10.1016/J.APPET.2011.08.011

Presnell, K., & Stice, E. (2003). An experimental test of the effect of weight-loss dieting on bulimic pathology: Tipping the scales in a different direction. *Journal of Abnormal Psychology*, *112*(1), 166–170. https://doi.org/10.1037/0021-843X.112.1.166

Racine, S. E., Burt, S. A., Iacono, W. G., McGue, M., & Klump, K. L. (2011). Dietary Restraint Moderates Genetic Risk for Binge Eating. *Journal of Abnormal Psychology*, *120*(1), 119. https://doi.org/10.1037/A0020895

Reas, D. L., & Grilo, C. M. (2007). Timing and sequence of the onset of overweight, dieting, and binge eating in overweight patients with binge eating disorder. *International Journal of Eating Disorders*, *40*(2), 165–170. https://doi.org/10.1002/EAT.20353

Reeves, R. S., McPherson, R. S., Nichaman, M. Z., Harrist, R. B., Foreyt, J. P., & Goodrick, G. K. (2001). Nutrient Intake of Obese Female Binge Eaters. *Journal of the American Dietetic Association*, *101*(2), 209–215. https://doi.org/10.1016/S0002-8223(01)00055-4

Rios, J. M., Miller, A. L., Lumeng, J. C., Rosenblum, K., Appugliese, D. P., Kaciroti, N., & Gearhardt, A. N. (2020). Behavioral Responses to Sucrose as an Indicator of Positive Hedonic Response Across the First Six Months of Infancy. *Physiology & Behavior*, *223*, 112914. https://doi.org/10.1016/J.PHYSBEH.2020.112914

Rodgers, R. F., Paxton, S. J., McLean, S. A., Campbell, K. J., Wertheim, E. H., Skouteris,

H., & Gibbons, K. (2013). Do maternal body dissatisfaction and dietary restraint predict weight gain in young pre-school children? A 1-year follow-up study. *Appetite*, *67*, 30–36. https://doi.org/10.1016/J.APPET.2013.03.009

Rogers, S. L., & Blissett, J. (2017). Breastfeeding duration and its relation to weight gain, eating behaviours and positive maternal feeding practices in infancy. *Appetite*, *108*, 399–406. https://doi.org/10.1016/J.APPET.2016.10.020

Rosseel, Y. (2012). lavaan: An R Package for Structural Equation Modeling. *Journal of Statistical Software*, *48*, 1–36. https://doi.org/10.18637/JSS.V048.I02

Ruderman, A. J. (1986). Dietary restraint: A theoretical and empirical review. *Psychological Bulletin*, *99*(2), 247–262. https://doi.org/10.1037/0033-2909.99.2.247

Rüther, N. M., & Richman, C. L. (2013). The relationship between mothers' eating restraint and their children's attitudes and behaviors. *Bulletin of the Psychonomic Society 1993 31:3*, *31*(3), 217–220. https://doi.org/10.3758/BF03337328

Sanftner, J. L., Crowther, J. H., Crawford, P. A., & Watts, D. D. (2007). *Eating Disorders Maternal influences (or lack thereof) on daughters' eating attitudes and behaviors*. https://doi.org/10.1080/10640269608249182

Santos-Flores, J. M., Cárdenas-Villarreal, V. M., Gutierrez-Valverde, J. M., Pacheco-Pérez, L. A., Paz-Morales, M. de los Á., Guevara-Valtier, M. C., Santos-Flores, J. M., Cárdenas-Villarreal, V. M., Gutierrez-Valverde, J. M., Pacheco-Pérez, L. A., Paz-Morales, M. de los Á., & Guevara-Valtier, M. C. (2021). Sensitivity to reward, food

addiction and obesity in mothers and children. *Sanus*, *6*, e187. https://doi.org/10.36789/REVSANUS.VI1.187

Santos, J. L., Kain, J., Dominguez-Vásquez, P., Lera, L., Galván, M., Corvalán, C., & Uauy, R. (2009). Maternal anthropometry and feeding behavior toward preschool children: Association with childhood body mass index in an observational study of Chilean families. *International Journal of Behavioral Nutrition and Physical Activity*, *6*(1), 1–8. https://doi.org/10.1186/1479-5868-6-93

Schembre, S., Greene, G., & Melanson, K. (2009). Development and validation of a weight-related eating questionnaire. *Eating Behaviors*, *10*(2), 119–124. https://doi.org/10.1016/J.EATBEH.2009.03.006

Schiestl, E. T., & Gearhardt, A. N. (2018). Preliminary validation of the Yale Food Addiction Scale for Children 2.0: A dimensional approach to scoring. *European Eating Disorders Review*, *26*(6), 605–617. https://doi.org/10.1002/ERV.2648

Schulte, E. M., Avena, N. M., & Gearhardt, A. N. (2015). Which Foods May Be Addictive? The Roles of Processing, Fat Content, and Glycemic Load. *PLOS ONE*, *10*(2), e0117959. https://doi.org/10.1371/JOURNAL.PONE.0117959

Schulte, E. M., & Gearhardt, A. N. (2017). Development of the Modified Yale Food Addiction Scale Version 2.0. *European Eating Disorders Review*, *25*(4), 302–308. https://doi.org/10.1002/erv.2515

Schulte, E. M., Grilo, C. M., & Gearhardt, A. N. (2016). Shared and unique mechanisms

underlying binge eating disorder and addictive disorders. In *Clinical Psychology Review* (Vol. 44, pp. 125–139). Elsevier Inc. https://doi.org/10.1016/j.cpr.2016.02.001

Shorr, I. (1984). *How to Weigh and Measure Children.* Hunger Watch. https://www.worldcat.org/title/how-to-weigh-and-measure-children/oclc/656595576

Snoek, H. M., Engels, R. C. M. E., van Strien, T., & Otten, R. (2013). Emotional, external and restrained eating behaviour and BMI trajectories in adolescence. *Appetite*, *67*, 81–87. https://doi.org/10.1016/J.APPET.2013.03.014

Snoek, H. M., van Strien, T., Janssens, J. M. A. M., & Engels, R. C. M. E. (2008). Restrained Eating and BMI: A Longitudinal Study Among Adolescents. *Health Psychology*, *27*(6), 753–759. https://doi.org/10.1037/0278-6133.27.6.753

Spurrell, E. B., Wilfley, D. E., Tanofsky, M. B., & Brownell, K. D. (1997). Age of onset for binge eating: Are there different pathways to binge eating? *International Journal of Eating Disorders*, *21*(1), 55–65. https://doi.org/10.1002/(SICI)1098-108X(199701)21:1<55::AID-EAT7>3.0.CO;2-2

Steiner, J. E., Glaser, D., Hawilo, M. E., & Berridge, K. C. (2001). Comparative expression of hedonic impact: Affective reactions to taste by human infants and other primates. *Neuroscience & Biobehavioral Reviews*, *25*(1), 53–74. https://doi.org/10.1016/S0149-7634(00)00051-8

Stice, E. (1998). Prospective relation of dieting behaviors to weight change in a

community sample of adolescents. *Behavior Therapy*, *29*(2), 277–297. https://doi.org/10.1016/S0005-7894(98)80007-5

Stice, E., Fisher, M., & Lowe, M. R. (2004). Are Dietary Restraint Scales Valid Measures of Acute Dietary Restriction? Unobtrusive Observational Data Suggest Not. *Psychological Assessment*, *16*(1), 51–59. https://doi.org/10.1037/1040-3590.16.1.51

Stice, E., Mazotti, L., Krebs, M., & Martin, S. (1998). Predictors of adolescent dieting behaviors: A longitudinal study. *Psychology of Addictive Behaviors*, *12*(3), 195–205. https://doi.org/10.1037/0893-164X.12.3.195

Striegel-Moore, R. H., Wilson, G. T., DeBar, L., Perrin, N., Lynch, F., Rosselli, F., & Kraemer, H. C. (2010). Cognitive Behavioral Guided Self-Help for the Treatment of Recurrent Binge Eating. *Journal of Consulting and Clinical Psychology*, *78*(3), 312–321. https://doi.org/10.1037/a0018915

Strien, T. van, Herman, C. P., & Anschutz, D. (2012). The predictive validity of the DEBQ-external eating scale for eating in response to food commercials while watching television. *International Journal of Eating Disorders*, *45*(2), 257–262. https://doi.org/10.1002/EAT.20940

Tabachnick, B. G., & Fidell, L. S. (2019). *Using Multivariate Statistics Title: Using multivariate statistics*. https://lccn.loc.gov/2017040173

Tanofsky-Kraff, M., Faden, D., Yanovski, S. Z., Wilfley, D. E., & Yanovski, J. A. (2005). The Perceived Onset of Dieting and Loss of Control Eating Behaviors in

Overweight Children. *The International Journal of Eating Disorders*, *38*(2), 112. https://doi.org/10.1002/EAT.20158

Telch, C. F., & Agras, W. S. (1993). The effects of a very low calorie diet on binge eating. *Behavior Therapy*, *24*(2), 177–193. https://doi.org/10.1016/S0005-7894(05)80262-X

Tiggemann, M., & Lowes, J. (2002). Predictors of maternal control over children's eating behaviour. *Appetite*, *39*(1), 1–7. https://doi.org/10.1006/APPE.2002.0487

van Strien, T., Frijters, J. E., Bergers, G. P., & Defares, P. B. (1986). The Dutch Eating Behavior Questionnaire (DEBQ) for Assessment of Restrained, Emotional, and External Eating Behavior. In *Eating Disorders* (Vol. 5, Issue 2). http://www.seven-health.com/wp-content/uploads/2018/08/The-Dutch-Eating-Behavior-Questionnaire-DEBQ.pdf

Van Strien, T., Herman, C. P., & Verheijden, M. W. (2014). Dietary restraint and body mass change. A 3-year follow up study in a representative Dutch sample. *Appetite*, *76*, 44–49. https://doi.org/10.1016/J.APPET.2014.01.015

Wadden, T. A., Foster, G. D., Sarwer, D. B., Anderson, D. A., Gladis, M., Sanderson, R. S., Letchak, R., Berkowitz, R. I., & Phelan, S. (2004). Dieting and the development of eating disorders in obese women: results of a randomized controlled trial. *The American Journal of Clinical Nutrition*, *80*(3), 560–568. https://doi.org/10.1093/AJCN/80.3.560

Wang, Y. A., & Rhemtulla, M. (2021). Power Analysis for Parameter Estimation in

Structural Equation Modeling: A Discussion and Tutorial. *Advances in Methods and Practices in Psychological Science*, *4*(1). https://doi.org/10.1177/2515245920918253

Welsh, D. M., & King, R. M. (2016). Applicability of the dual pathway model in normal and overweight binge eaters. *Body Image*, *18*, 162–167. https://doi.org/10.1016/J.BODYIM.2016.06.007

Westenhoefer, J., Stunkard, A. J., & Pudel, V. (1999). Validation of the Flexible and Rigid Control Dimensions of Dietary Restraint. *Int J Eat Disord*, *26*, 53–64. https://doi.org/10.1002/(SICI)1098-108X(199907)26:1

Wiedemann, A. A., Carr, M. M., Ivezaj, V., & Barnes, R. D. (2021). Examining the construct validity of food addiction severity specifiers. *Eating and Weight Disorders*, *26*(5), 1503–1509. https://doi.org/10.1007/S40519-020-00957-W/TABLES/2

Wilcox, R. (2005). Trimming and Winsorization. *Encyclopedia of Biostatistics*. https://doi.org/10.1002/0470011815.B2A15165

Wiss, D. A., & Avena, N. M. (2020). Food Addiction, Binge Eating, and the Role of Dietary Restraint: Converging Evidence from Animal and Human Studies. *Binge Eating: A Transdiagnostic Psychopathology*, 193–209. https://doi.org/10.1007/978-3-030-43562-2_14

Ziauddeen, H., & Fletcher, P. C. (2012). *Is food addiction a valid and useful concept?* https://doi.org/10.1111/j.1467-789X.2012.01046.x