The Potential Role of Memory in the Development and Maintenance of Binge and Loss of Control Eating

by

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Dissertation submitted in partial fulfillment of
the requirements for the degree of
Doctor of Philosophy in the Department of
Psychology and Neuroscience in the Graduate School of
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ABSTRACT

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Abstract

Binge and loss of control eating are clinically significant symptoms that constitute syndromes characterized by a repetitive pattern of maladaptive and subjectively uncontrollable binge or loss of control eating. The hippocampal-dependent memory system appears to play an important role in the regulation of food intake in studies employing animal models. These findings may extend to humans, but research has been limited, particularly among vulnerable populations, such as those with eating pathology. Existing research suggests excessive energy intake and/or consumption of high fat, high sugar foods impairs performance on hippocampal-dependent learning and memory tasks, which in turn, is associated with excessive food intake and motivated high fat, high sugar food seeking behaviors. This vicious cycle may be particularly relevant to individuals with binge or loss of control eating, as persistent binge/LOC eating episodes are associated with several food intake patterns and individual difference factors that may strengthen the observed associations.

This study sought to examine the potential role of learning and memory in the development and maintenance of binge and LOC eating behaviors among a community sample of (N=66) young adult women, who either endorsed binge or LOC eating (n=35) or reported typical eating behaviors (n=31). Participants completed clinical diagnostic interviews, standardized neuropsychological measures of cognitive ability and hippocampal-dependent memory, self-report measures of eating behaviors, dietary

intake of highly palatable foods, depressive symptoms, psychological functioning, autobiographical memory (general and eating event memories), and current mood state and hunger. Results revealed that participants who endorsed binge or loss of control eating performed worse on several measures of hippocampal-dependent memory, including measures of visuo-spatial learning and memory, verbal learning and memory, immediate memory, and delayed memory. Findings from the autobiographical memory tasks did not support our hypothesis, but provided insight into other factors, such as the potential role of emotion, in the study of autobiographical memory and LOC eating. The current study also did not find evidence to support the predictive utility of high fat, high sugar dietary intake on differences in hippocampal functioning, at least as measured in the current sample. Future research should continue to characterize and probe hippocampal-dependent memory among those with LOC eating, and explore possible differences in the visuo-spatial learning and memory system. Future research should also seek to overcome some of the methodological challenges in measuring dietary intake of high fat, high sugar foods, to further our understanding of the vicious cycle, especially among vulnerable populations.

Dedication

To all the women in my family, who have pursued their dreams, and encouraged me to do the same. To my mother, Marilyn Keeling, my sister, Stephanie Pou, my grandmother, Esther Keeling, especially, who have each been an example of determination, grit, and perseverance. To my nieces and nephew, Truman, Keely, and Sophia, for being constant sources of joy!

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Finally, I would like to acknowledge and thank the entire Zucker Lab team for their assistance in completing this project, including the dedicated team of student volunteers who have helped me along the way: Mirai Matsurra, Julia Nicholas, George Zhang, Alyssa Smith, Dilanaz Unal, Sarah Walker, Erik Savereide, Sam Marsen, and Savannah Erwin.

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1. Introduction

Binge eating disorder (BED) is a clinically significant syndrome characterized by a repetitive pattern of maladaptive and subjectively uncontrollable binge eating, in which individuals experience a conflict between what they want to do (*stop eating*) and their behavior (*continue to eat*) (Allison & Timmerman, 2007). The affect regulation model is the most widely accepted theory of BED (Agras & Telch, 1998), which suggests bingeing regulates distressing emotions by relieving or improving the negative affective state that typically precedes binge episodes (Deaver, Miltenberger, Smyth, Meidinger, & Crosby, 2003; Johnson, Schlundt, Barclay, Carr-Nangle, & Engler, 1995). However, relief is only temporary, as a range of negative emotions (e.g., guilt, shame, disgust, depression) typically follow binges (Agras & Telch, 1998; Deaver et al., 2003; Johnson et al., 1995). It is unclear why the consequences associated with the aftermath of binge eating are not learned or used to shape or extinguish binge eating behavior. *Why do individuals persist in this maladaptive/aversive pattern of eating? Existing models on the initiation and maintenance of BED do not account for this aberrant learning process.* An obvious gap in the literature is an understanding of the contribution of other models of learning, including the explicit role of memory on the learning process.

Emerging evidence suggests an association between memory for recent eating and subsequent food intake (Higgs, 2002, 2005; Higgs & Donohoe, 2011; Higgs, Williamson, & Attwood, 2008; Higgs & Woodward, 2009). Specifically, experimentally

enhancing or impairing memory of food intake (i.e., episodic food memory), respectively decreased or increased consumption at the next snack or meal (Higgs, 2002, 2005; Higgs & Donohoe, 2011; Higgs et al., 2008; Higgs & Woodward, 2009). The hippocampus, a cortical brain structure situated deep within the medial temporal lobe, has an established role in the encoding and retrieval of declarative memories (Craig, 2006; Higgs, 2008; Morris, 2007). Numerous animal studies have found excessive energy intake, or consumption of high fat, high sugar foods impaired hippocampal functioning and performance on hippocampal-dependent learning and memory tasks (Granholm et al., 2008; Kanoski & Davidson, 2011; Kanoski, Meisel, Mullins, & Davidson, 2007; Lindqvist et al., 2006; Molteni, Barnard, Ying, Roberts, & Gómez-Pinilla, 2002). Furthermore, impaired hippocampal-dependent memory performance was associated with excessive food intake and motivated high fat, high sugar food seeking behaviors in animal models (Kanoski et al., 2007).

1.1 The "Vicious Cycle" Model

Taken together, Davidson and colleagues (2005) proposed the "vicious cycle" model of obesity (Figure 1), which posits: excessive food intake or high fat, high sugar dietary intake → impaired hippocampal functioning → hippocampal-dependent memory deficits → excessive food intake and motivated high fat, high sugar food-seeking behaviors. (Davidson, Kanoski, Walls, & Jarrard, 2005; Davidson, Kanoski, Schier, Clegg, & Benoit, 2007; Kanoski & Davidson, 2001). Davidson and colleagues have since evolved the

model over the years, including the potential role of the hippocampus in eating in the absence of hunger (Granholm et al., 2008; Kanoski & Davidson, 2011; Kanoski et al., 2007; Lindqvist et al., 2006; Molteni et al., 2002).

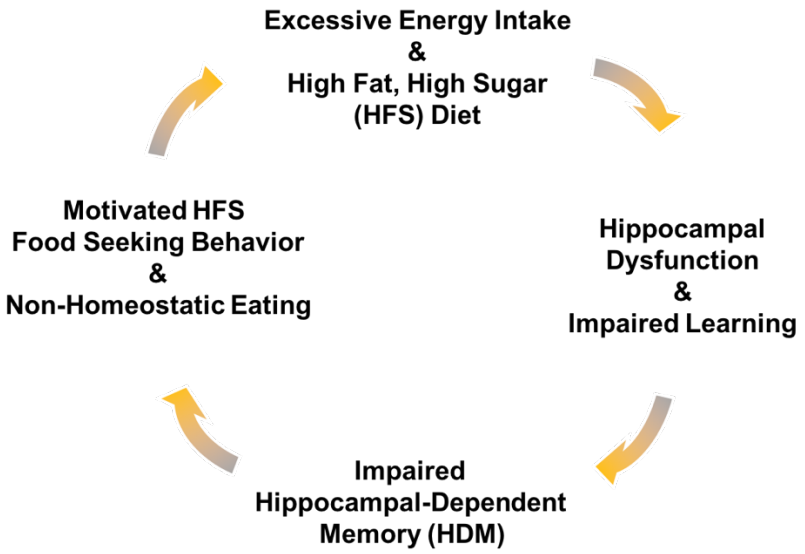


Figure 1: The Vicious Cycle Model Proposed by Davidson and Colleagues (2005). (Davidson, Kanoski, Walls, & Jarrard, 2005; Davidson, Kanoski, Schier, Clegg, & Benoit, 2007; Kanoski & Davidson, 2001)

1.2 Support for the Vicious Cycle

Preliminary evidence from normal weight college samples suggests this model may also apply to young adults (Francis & Stevenson, 2011; Higgs & Donohoe, 2011; Higgs et al., 2008). However, a notable limitation of the preliminary human studies is their exclusion of vulnerable individuals, such as those who experience binge or loss of control eating episodes. The vicious cycle may be particularly relevant to such individuals,

as binge and loss of control eating are associated with several food intake patterns and individual difference factors that may strengthen the observed associations in the model (Allison & Timmerman, 2007; Bartholome, Raymond, Lee, Peterson, & Warren, 2006; Carnell, Gibson, Benson, Ochner, & Geliebter, 2012; Davis, 2009; Davis et al., 2007; Goldfein, Walsh, Devlin, Lachaussée, & Kissileff, 1993). For example, individuals with binge and loss of control eating episodes demonstrate greater preference for and intake of highly palatable foods (i.e., high fat, high sugar foods), during and separate from binges (Allison & Timmerman, 2007; Bartholome et al., 2006; Fitzgibbon & Blackman, 2000; Goldfein et al., 1993; Goldschmidt, 2017).

While the link to binge and loss of control eating is conceptually logical, in fact, limited prior research informs the components of the model for this clinically significant group. That is, there is a dearth of literature on binge and loss of control eating and: 1) hippocampal-dependent memory functioning, generally; and 2) hippocampally-mediated food memory specifically (e.g., recalling details of a recent eating event). (Carnell et al., 2012; Davis, 2009; Davis et al., 2007; Eneva, Murray, & Chen, 2017; Manasse et al., 2016).

1.3 Binge Eating Disorder

Binge Eating Disorder (BED) is a clinically significant syndrome characterized by a repetitive pattern of maladaptive and subjectively uncontrollable binge eating. BED is the most prevalent and persistent form of eating disorder, affecting individuals across the

lifespan (Hudson, Hiripi, Pope, & Kessler, 2007; Pope et al., 2006). Point prevalence estimates for BED range from 3-5% among community samples to 19-30% among individuals seeking treatment for obesity (Hudson et al., 2007; Spitzer et al., 1992, 1993; Stunkard et al., 1996), with average lifetime duration for the disorder estimated at 8.1 to 14.4 years (Hudson et al., 2007; Pope et al., 2006). Individuals with BED who are also overweight or obese represent a particularly vulnerable group, as both conditions confer serious medical and psychiatric risks (Bulik & Reichborn-Kjennerud, 2003; Dixon, 2010; Reichborn-Kjennerud, Bulik, Sullivan, Tambs, & Harris, 2004).

1.3.1 Diagnostic Criteria for Binge Eating Disorder

To meet diagnostic criteria for BED, an individual must experience weekly objective binge eating episodes (also referred to as objective binge episodes) for three months, which are associated with three or more behavioral features (e.g., fast pace of eating, eating in the absence of hunger) and marked distress, without engaging in inappropriate compensatory behaviors (e.g., purging, excessive driven exercise) (American Psychiatric Association, 2013). Objective binge episodes are defined by consumption of an unambiguously large amount of food with a subjective sense of loss of control over eating (APA; 2013). As such, objective binge episodes represent significant energy intake, ranging from 743-2,963 calories per episode, which comprise 37-150% of daily caloric needs (i.e., based on 2000 kcal/day) (Bartholome et al., 2006; Fitzgibbon & Blackman, 2000; Goldfein et al., 1993).

Table 1 Binge Eating Disorder (BED) Diagnostic Criteria

Binge Eating Disorder (BED) Diagnostic Criteria	
All of the following must be present:	
Recurrent episodes of	Binge eating
Quantity	Unusually large amount of food
Subjective control	Sense of loss of control over eating
Frequency and duration	1 day/week for 3 months
Distress	Marked distress regarding binge eating
Compensatory behavior	No inappropriate compensatory behavior
At least three of the following must be present:	
Behavioral and psychological features	Face pace of eating; eating in the absence of hunger; eating until uncomfortably full; eating alone because of embarrassment; or feeling disgusted with oneself, depressed, or very guilty afterward

Note: Diagnostic criteria for Binge Eating Disorder (BED) were summarized from the Diagnostic and Statistical Manual-Fifth Edition (DSM-5) (American Psychiatric Association, 2013).

1.3.2 Patterns of Eating Observed in Binge Eating Disorder

Although the quality of foods consumed (or lack thereof) during binge episodes is not a diagnostic criterion for BED, several studies have attempted to characterize food intake patterns during binge eating episodes. Most studies that have examined composition of binge episodes have relied on standardized test meals (i.e., in laboratory settings, participants are instructed to binge), diary accounts/personal recordings of binge episodes at home, or clinician interviews. Taken together, findings suggest the following patterns: 1) on average, participants with BED consumed larger quantities of food during binge meals, compared to participants without BED; 2) participants with BED consumed greater quantities of food during binge meals relative to their typical or non-

binge meals; 3) participants with BED tended to consume more desserts and snack foods (e.g., potato chips, cake, ice cream) during binge meals, relative to comparison groups; and 4) no consistent differences between groups emerged regarding the relative macronutrient consumption of binge meals (i.e., percentage of energy from proteins, carbohydrates, and fats for binge meals). (Bartholome et al., 2006; Goldschmidt, 2017; Guss et al., 2002; Mitchell et al., 2012; Telch et al., 1998; Yanovski et al., 1992). The studies were limited by smaller sample sizes (e.g., most enrolled less than 25 participants) and the use of less ecologically valid laboratory test meals.

In addition to examining the composition of binge meals, several studies have sought to identify typical dietary intake patterns among participants with BED, including individuals with co-occurring overweight or obesity. Across these studies, participants with BED demonstrated greater preference for and intake of highly palatable foods, relative to those who were normal-weight or did not have BED. However, results across studies should be interpreted with caution, as a variety of definitions, terms, and measures of assessment were used for "highly palatable foods." Constructs used to capture highly palatable foods ranged from vaguely defined concepts of "Western diet" to more narrowly focused assessments of "high simple carbohydrate foods." Additionally, there was a lack of consistency across studies in the measurement of dietary intake, which ranged from abbreviated dietary intake measures, to use of 24-hour dietary recalls, to lengthier food frequency questionnaires that assessed intake patterns over the past 12 months. (Bartholome et al., 2006; England, Andrews, Jago, & Thompson, 2015;

Goldfein et al., 1993; Guss et al., 2002; Mitchell et al., 2012; Tanofsky-Kraff et al., 2009; Telch et al., 1998; Yanovski et al., 1992).

In an effort to shed light on the larger body of research in this area, Goldschmidt (2017) completed a comprehensive review of the literature on overeating, loss of control eating, and binge eating among adults and children. In the adult studies Goldschmidt (2017) reviewed, she found participants who endorsed loss of control eating of any type (i.e., objective binge episodes or subjective binge episodes) and those who reported objective overeating (i.e., overeating, without loss of control eating), reported higher overall energy intake compared to those without eating pathology (i.e., healthy adult participants). Results also showed greater consumption of highly palatable foods among adults with loss of control eating, which occurred during binge episodes and during typical meals/snacks. Findings from the studies among children and adolescents revealed youth demonstrated a preference for higher fat, higher carbohydrate/sugar foods during LOC eating episodes, but did not experience an overall increase in energy intake. (Goldscmidt, 2017).

In sum, studies examining patterns of eating behavior in BED, found positive associations between binge and loss of control eating status (e.g., diagnosis of BED) and increased consumption during both binge episodes and typical or non-binge meals. Additionally, across studies, individuals with binge and loss of control eating reported greater consumption of palatable foods, during binge and non-binge episodes, and had higher overall caloric intake.

1.3.3 Health Consequences of Binge Eating Disorder

Given the non-homeostatic energy consumption evident among individuals with loss of control eating, researchers have also sought to identify metabolic risks associated with binge and loss of control eating. BED, loss of control eating, and overeating episodes without a sense of loss of control have all been positively associated with excess weight gain, elevated body mass index (BMI), impaired insulin sensitivity, and increased risk of chronic conditions, such as diabetes and heart disease (Bulik & Reichborn-Kjennerud, 2003; Hudson et al., 2010). However, excess food consumption alone, does not explain the range of adverse health-related outcomes observed in BED. Rather, the subjective sense of loss of control over eating has emerged as a key, defining feature of binge episodes and BED. Loss of control is uniquely associated with greater levels of distress, and has been positively correlated physical and psychological impairment (Colles, Dixon, & O'Brien, 2008; Mond, Latner, Hay, Owen, & Rodgers, 2010), including psychiatric comorbidities, such as depression, anxiety, and personality disorders (Goldschmidt, 2017; Reichborn-Kjennerud, Bulik, Sullivan, Tambs, & Harris, 2004; Wilfley, Wilson, & Agras, 2003).

1.4 Loss of Control Eating

While the subjective sense of loss of control over eating is considered the most toxic feature of objective binge episodes and BED (Colles et al., 2008; Mond et al., 2010), loss of control over eating outside of a formal diagnosis of BED has not always been

considered clinically significant. However, support for the importance of loss of control over eating as a valid, stand-alone construct, irrespective of the size of an eating episode, has been growing over the past decade, as several cross-sectional and prospective studies have demonstrated the importance of LOC as an indicator of distress and impairment. (Brownstone et al., 2013; Forney et al., 2016; Goldschmidt, 2017; Jenkins et al., 2012; Palavras et al., 2013; Palavras et al.; 2018). Specifically, persistent loss of control eating episodes, in the absence of objectively large amounts of food (i.e., subjective binge episodes), have been associated with similar levels of distress, impairment, and negative health outcomes as loss of control eating in the context of objective binge episodes (Goldschmidt, 2017; Palavras et al., 2013; Palavras et al.; 2018).

Even among large community samples, loss of control has emerged as an important indicator of impairment. For example, in a community sample (*N*=549) of college-aged women, loss of control severity, irrespective of binge episode size or frequency, was associated with greater clinical impairment across a range of psychosocial domains (Vannucci et al., 2013). Additionally, subjective binge episodes have been identified as an important predictor in developmental models for partial- and full-syndrome BED, with loss of control eating in youth associated with increased risk of developing BED in adolescence and early adulthood (Sonneville et al., 2013; Hilbert et al., 2013; Tanofsky-Kraff et al., 2011; Tanofsky-Kraff et al., 2009).

Findings on loss of control eating have also prompted numerous researchers to refute the necessity of the large amount of food criterion for objective binge episodes

and a BED diagnosis (Latner & Clyne, 2008; Latner, Hildebrandt, Rosewall, Chisholm, & Hayashi, 2007; Mond et al., 2010; Wolfe et al., 2009). Instead, findings support loss of control eating as a stand-alone behavior, worthy of clinical consideration, which will be discussed next.

1.4.1 Proposed Criteria for Loss of Control Eating Disorder

The emergence of loss of control eating as an essential psychopathological construct with similarities to full-criteria BED has prompted the World Health Organization to propose expanding the diagnostic criteria for binge eating disorder in their upcoming, provisional International Classification of Diseases, 11th edition (ICD-11) guidelines (Global Clinical Practice Network WHO, 2019). Specifically, the proposed guidelines would allow for inclusion of subjective binge episodes in meeting the frequency criteria for binge eating episodes and reduce the essential features of binge eating disorder to: 1) at least once weekly binge eating episodes (subjective binge episodes and/or objective binge episodes) for three months; 2) loss of control over the eating episode; and 3) marked distress; thus eliminating the behavioral specifiers found in the DSM-5 (Palavras et al.; 2018).

1.5 Limitations of Existing Models of Binge Eating

The most widely accepted theory of BED is the "affect regulation model" (Agras & Telch, 1998), which suggests bingeing regulates distressing emotions by relieving/improving the negative affective state that typically precedes binge eating

episodes (Deaver et al., 2003; Johnson et al., 1995); Fairburn, 2008). However, relief is only temporary, as a range of negative emotions (e.g., guilt, shame, disgust, depression) typically follow binges (Agras & Telch, 1998; Deaver et al., 2003; Johnson et al., 1995). Why are the negative consequences associated with the aftermath of binge eating not learned or used to shape or extinguish binge eating behavior? Why do individuals persist in this maladaptive/aversive pattern of eating? *Existing models on the initiation and maintenance of binge and loss of control eating do not account for this aberrant learning process.* An obvious gap in the literature is an understanding of the contribution of other models of learning, including the explicit role of memory on the learning process.

To explore the possible role of memory in binge and loss of control eating, I will first review two brain structures critical for learning and memory: the hippocampus and hippocampal formation, then I will define hippocampal-dependent memory, and review the circuitry that supports the functional role of the hippocampus in forming memories, then I will explore the existing literature on each of the associations proposed in the vicious cycle model, in both animal models and human studies, as well as review clinical and behavioral features of binge and loss of control eating that may strengthen the associations found in the vicious cycle.

1.6 The Hippocampus and Hippocampal Formation

The hippocampus is a seahorse-shaped cortical brain structure situated deep within the medial temporal lobe (Stark, 2007; Hariri, 2015). In human research, the

hippocampus is often examined in the context of the hippocampal formation, which represents a larger region surrounding the hippocampus, and includes the parahippocampal cortex, entorhinal cortex, perirhinal cortex, subiculum, and dentate gyrus. The hippocampal formation functions as an "interface or relay for communication" (Hariri, 2015, p. 156) between the hippocampus and several other brain regions, including sensory association areas, the prefrontal cortex, the amygdala, and the ventral striatum (Amaral & Lavenex, 2007; Stark, 2007; Hariri, 2015).

Because it is not possible to conduct histological analyses in human studies, it was historically difficult to isolate and characterize the various roles of the hippocampal formation. Instead, theories regarding the role of the human hippocampus often relied on data from patient populations, such as those with amnesia or other known memory deficits. Through functional neuroimaging studies and electrophysiological recordings, our understanding of the hippocampus and hippocampal formation has evolved considerably in the past 10-15 years (Morris, 2007; Stark, 2007; Hariri, 2015; Zeineh, Engel, Thompson, & Bookheimer, 2003).

Specifically, the ability to study information flow into and out of the hippocampal formation was dramatically improved when researchers at UCLA (Zeineh & Bookheimer, 2003) developed a novel data analytic algorithm to map encoding and retrieval of memories, using fMRI data. Their technique allowed the hippocampal formation to be virtually "unrolled" along its longitudinal axis (i.e., through virtual imaging), to map information flow along this pathway. Their breakthrough has contributed greatly to our

understanding of the of the human hippocampus, and served to corroborate patterns previously observed only in non-human animal studies. (Hariri, 2015; Zeineh & Bookheimer, 2003; Zeineh, Engel, Thompson, & Bookheimer, 2003).

In the next section, I will briefly review the memory terms and definitions encompassed under the large umbrella of hippocampal-dependent memory, which is the focus of the present study.

1.6.1 Hippocampal-Dependent Memory

Based on studies of the hippocampus and hippocampal formation, there is general consensus that the human hippocampus is involved in the development of long-term declarative memories. Long-term memories are divided into two categories: 1) declarative or conscious memories, and 2) implicit or non-conscious memories. For the purposes of the present study, we are primarily concerned with declarative memories. Declarative memories are the explicit, conscious recollection of events, including information about what, when, and where. Declarative memory is further broken down into: 1) episodic memory, and 2) semantic memory. Episodic memory is a specific type of declarative, long-term memory, in which a person learns, stores, and retrieves information about specific personal experiences (i.e., often denoted as episodes or events). Semantic memory on the other hand, is memory for facts and knowledge, which does not typically include information about the specific context (e.g., time, place, or event) in which the knowledge was learned. (Hariri, 2015; Goldstein, 2008).

Additionally, the hippocampal formation is critical to the encoding (i.e., creation) and recall (i.e., accessing) of long-term declarative memories (Hariri, 2015; Zeineh et al., 2003). For further understanding of this functional role of the hippocampus in forming long-term declarative memories, we will now consider the circuitry involved in this process.

1.6.2 Corticohippocampal Circuit

The corticohippocampal circuit represents the bi-directional flow of information into and out of the hippocampus (Hariri, 2015). Specifically, information from the neocortical association areas (e.g., parietal or occipital cortexes) flows into the parahippocampal region via either: a) the perirhinal cortex (i.e., ventral stream, providing 'what' information), or b) the parahippocampal cortex (i.e., dorsal stream, providing 'where' information), then flows through the entorhinal cortex, which serves as the gateway to the hippocampus. The hippocampus binds the information from the two parallel streams (i.e., ventral [what/item] and dorsal [where/context] streams) to create a memory trace in which item-in-context are bound. Because the hippocampal circuit is bi-directional, information also flows from the hippocampus, through the entorhinal cortex, to the parahippocampal region, and to the neocortical association areas (see Figure 2). Findings from neuroimaging studies emphasize the critical role of the hippocampus in binding 'what' and 'where' information (i.e., item-in-context). Importantly, this ancient circuitry is the same in primates and rodents, and is present in all vertebrates, which is

helpful in the translation of hippocampus-based research in non-human models to human models. (Stark, 2007; Morris, 2007; Dickerson & Eichenbaum, 2010; Hariri, 2015).

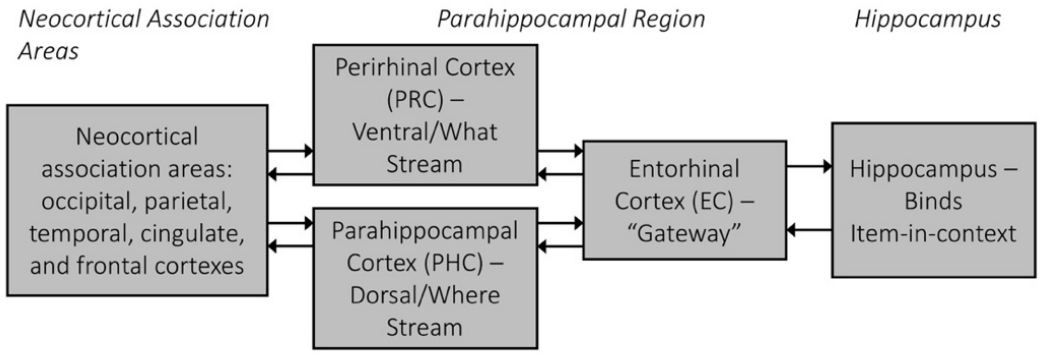


Figure 2: Functional Organization of Declarative Memory within the Corticohippocampal Circuit, as Proposed by Dickerson & Eichenbaum (2010) and Depicted by Hariri (2015).

Building on this understanding of the hippocampal formation and hippocampal circuitry, I will next provide a context for considering research based on animal models, then I will examine the contribution of energy intake on impaired brain functioning, explore food types that alter cognitive functioning more generally and hippocampal functioning more specifically, and review novel human studies on the potential role of memory deficits and excessive food intake.

1.6.3 Animal Models of Hippocampal-Dependent Memory

Many theories on human hippocampal function are inferred from animal studies of hippocampal function using rodent, primate, and/or bird models. Animal models can

be helpful in understanding the human hippocampus because of the similarities in anatomy, physiology, and cell biology between the human and animal hippocampus. The major limitation is that animals are not able to use language to communicate learning or awareness of their past or future. The techniques and methods used in animal studies include pharmacological and genetic interventions (e.g., neurotoxic lesions) and surgical (i.e., permanent) lesions, which allow for more specific tests of hippocampal function compared to methods used in human studies. These techniques suggest the hippocampus is essential for both episodic memory (i.e., recollections about events, which are unique to personal experiences) and relational learning (i.e., encoding of relationships between multiple items and events). (Morris, 2007; Hariri, 2015)

1.6.4 Energy Intake and Brain Functioning

Animal and human studies suggest an inverse relationship between energy intake and brain functioning. Excessive energy intake impairs brain functioning, while moderate caloric deficits (i.e., that produce weight loss within a normal weight range) improve brain functioning (Stranahan & Mattson, 2008). Over time, chronic excessive energy intake (e.g., which often leads to obesity and/or diabetes) may confer additional risk by accelerating "brain ageing" (Stranahan & Mattson, 2008, p. 210). As defined by these authors, accelerated brain ageing results from neurons becoming less able to adapt to and rebound from stress, which results in deficits in cognitive function beyond typical age-related impairment (Stranahan & Mattson, 2008, 2012).

Brain ageing may be observed at the structural, synaptic, behavioral, and biochemical levels. Specifically, parameters that help index brain growth and development, with implications for determining the rate of brain maturation include: 1) adult neurogenesis (i.e., generation of new neurons), 2) synaptic plasticity, and 3) neurotrophic factor expression. Adult neurogenesis is the generation of new neurons throughout adulthood/life. In humans, other mammals, and rodents, adult neurogenesis occurs within the hippocampus proper as well as the dentate gyrus (Gould, 2007).

Long-term potentiation (LTP) is the strengthening of the signal transmission between two neurons through stimulation of the perforant path (Bliss, Collingridge, & Morris, 2003). LTP is considered an index of synaptic efficiency (i.e., strength and plasticity) and is proposed as a substrate and mechanism whereby learning and memory occur at the cellular level (Stranahan & Mattson, 2008). Brain derived neurotrophic factor (BDNF) is a nerve growth factor which promotes neurogenesis, LTP, and synaptic plasticity (Noble, Billington, Kotz, & Wang, 2011; Stranahan & Mattson, 2008). Excessive energy intake (i.e., that produces weight gain) is proposed to impact each of these neural growth parameters, which in turn affect hippocampal-dependent learning and memory. Specifically, excessive energy intake is associated with decreased neurogenesis, decreased synaptic plasticity and LTP, and decreased levels of BDNF (Farr et al., 2008; Lindqvist et al., 2006; Molteni, Barnard, Ying, Roberts, & Gomez-Pinilla, 2002; Stranahan & Mattson, 2008, 2012; Stranahan et al., 2008). While these studies demonstrate the importance of examining the effects of excessive caloric intake on cognitive impairment,

it is also important to consider the types of foods consumed, as different macronutrient compositions may differentially impact cognitive function in individuals who are at risk of loss of control or binge eating.

1.6.5 High Fat, High Sugar Dietary Intake, Cognitive Functioning, and Memory Systems

Studies on the effects of high fat, high sugar diets provide evidence of a positive association between high fat, high sugar dietary intake and cognitive impairment, above and beyond the negative effects of excessive caloric intake (Eskelinen et al., 2008; Farr et al., 2008; Kanoski, 2012; Kanoski & Davidson, 2011; Molteni et al., 2002; Stranahan & Mattson, 2012). The high fat, high sugar diet is characterized by consumption of highly-palatable foods, such as those high in saturated fats and refined carbohydrates (also called simple sugars) (Cordain et al., 2005). Although high fat, high sugar diets contain a variety of macro- and micronutrients, most research has focused on the effects of saturated fats and simple sugars. It is well known that high fat, high sugar diets produce excess energy intake and weight gain (Hu, Manson, et al., 2001; Hu, van Dam, & Liu, 2001; Schulze, Fung, Manson, Willett, & Hu, 2006), but findings also suggest high fat, high sugar diets independently impair adult neurogenesis, decrease synaptic and structural/dendritic plasticity, and decrease BDNF expression (Farr et al., 2008; Kanoski & Davidson, 2011; Molteni et al., 2002; Stranahan & Mattson, 2012). Individuals with binge or loss of control eating may experience a 'double whammy' in terms of cognitive

impairment, as both excessive calories and high fat, high sugar dietary intake independently affect cognitive function.

In addition to impaired cognitive functioning and brain ageing in general, high fat, high sugar diets also specifically impair hippocampal functioning (Kanoski & Davidson, 2011; Kanoski, Zhang, Zheng, & Davidson, 2010; Lindqvist et al., 2006; Molteni et al., 2002; Stranahan, Cutler, Button, Telljohann, & Mattson, 2011; Stranahan et al., 2008). Numerous animal studies have examined the impact of high fat, high sugar diets (and other components of the Western diet) on hippocampal-dependent learning and memory. Hippocampal-dependent spatial learning and memory is most commonly assessed using the Morris Water Maze task, which typically involves fear/aversive conditioning. Rats were randomized to a HFS diet or a low-fat complex carbohydrate (LFCC) diet for 2 months, 6 months or 2 years (N=5-8 per group). Rats on the high fat, high sugar diet showed statistically significant reductions in hippocampal BDNF levels (i.e., measured via mRNA and protein) compared to rats on the LFCC diet. Hippocampal, but not cerebral cortex, BDNF mRNA values were lower at each time point in the HFS group, with the lowest levels found at 2 years (i.e., mRNA -42%). In terms of behavior, rats on the high fat, high sugar diet showed decreased performance on the spatial learning task (i.e., water maze), which was significantly correlated with reduced hippocampal BDNF levels. These performance deficits were found after only 2 months on the high fat, high sugar diet. (Molteni et al., 2002)

Subsequently, studies have largely upheld these findings (Jurdak, Lichtenstein, & Kanarek, 2008; Kanoski & Davidson, 2011; Stranahan & Mattson, 2012) and shown high fat, high sugar diets increase oxidative stress and alter lipid metabolism (Stranahan et al., 2011; Farr et al., 2008). In summary, animal studies examining the effects of high fat, high sugar diets show rodents demonstrate impaired hippocampal-dependent spatial and non-spatial learning and memory, even after only 72 hours or 9 days on the high fat, high sugar diets (Granholm et al., 2008; Jurdak et al., 2008; Kanoski & Davidson, 2010; Kanoski, Meisel, Mullins, & Davidson, 2007; Kanoski et al., 2010; Molteni et al., 2002; Pistell et al., 2010; Yu, Wang, & Huang, 2009).

1.6.6 Human Studies on the Hippocampus, Memory, and Energy Intake

Early observations of H.M. and other patients with amnesia suggested a relation between the hippocampus, learning, memory, and food intake (Hebben, Corkin, Eichenbaum, & Shedlack, 1985; Rozin, Dow, Moscovitch, & Rajaram, 1998). Specifically, these cases highlighted the tendency of patients with hippocampal removal/damage to eat meal after meal, in short succession, without reporting changes in hunger or satiety. While several of these patients also had extra-hippocampal damage/dysfunction, their pattern of overeating and hyperphagia was not well characterized by extra-hippocampal deficits (Higgs, 2002). In addition, rodent models of amnesia support the direct contribution of the hippocampus in overeating. Namely, rodents with amnesia (induced via hippocampal lesions) show significantly more appetitive food behaviors, increased

food intake, and greater weight gains compared to controls (Clifton, Vickers, & Somerville, 1998; Davidson & Jarrard, 1993; Davidson, Kanoski, Walls, & Jarrard, 2005). Since these early case studies, numerous researchers have sought to examine the relation between the hippocampus, learning, memory, and food intake among non-clinical human samples.

Within the last decade, human studies among community samples have suggested an inverse association between episodic food memories and subsequent food intake (Higgs, 2002, 2005, 2008; Higgs & Donohoe, 2011; Higgs, Williamson, & Attwood, 2008; Higgs & Woodward, 2009). An episodic food memory is a declarative memory of a specific eating episode, which includes food attributes, time, and place where food was consumed (Higgs & Donohoe, 2011). Across numerous studies, impaired episodic memory for the last meal is associated with increased subsequent snack consumption. These findings are specific to the last food episode, as recall of either lunch from the previous day or non-food memories had no effect on snack intake (Higgs, 2002; Higgs, Williamson, & Attwood, 2008). Several factors have been identified that either enhance or impair episodic food memories (and impact subsequent food intake). For example, disrupting food memory encoding (e.g., via television distraction) reduced vividness of the episodic memory and increased subsequent snack intake (Higgs, 2008; Higgs & Woodward, 2009). On the other hand, enhancing recall of the most recently eaten meal decreased subsequent food intake (Higgs, 2002). Taken together, accurate episodic memories of foods eaten in the recent past are associated with reduced food

consumption (Higgs, 2002, 2005, 2008; Higgs & Donohoe, 2011; Higgs et al., 2008; Higgs & Woodward, 2009). Given the hippocampus has an established role in episodic memory, it is hypothesized that hippocampal dysfunction may compromise the accuracy of episodic food memories and contribute to increased food consumption. Over time, the hippocampal dysfunction and increased consumption could interact together to promote non-homeostatic eating, weight gain, and loss of control eating.

In summary, energy intake (i.e., amount and type) directly influences brain functioning, the hippocampus, and learning and memory, which in turn influences energy intake in both human and animal models of obesity and binge or loss of control eating. Specifically, individuals with binge or loss of control eating, with or without co-occurring obesity, demonstrate both excessive caloric intake and greater preference for and intake of highly palatable foods (i.e., high fat, high sugar dietary intake), relative to normal-weight individuals or individuals without binge or loss of control eating. High fat, high sugar foods are also easily available and noticed, which contribute to motivated high fat, high sugar food seeking behavior in both human and animal studies of obesity, binge, or loss of control eating. Both excessive energy intake and high fat, high sugar dietary intake contribute to cognitive impairment in the form of decreased neurogenesis, decreased synaptic plasticity and LTP, and decreased levels of BDNF. High fat, high sugar dietary intake also directly impairs hippocampal-dependent spatial and non-spatial learning and memory. Finally, hippocampal damage/dysfunction may lead to a pattern of overeating as the accuracy of episodic food memories are compromised. Over time, the

hippocampal dysfunction and increased consumption could promote non-homeostatic eating, loss of control or binge eating, and/or weight gain, further contributing to the "vicious cycle."

These findings provide support for examining the vicious cycle model among individuals with of binge and loss of control eating, who are particularly vulnerable to the associations suggested by the model, given the constellation of symptoms, behavioral patterns, and impairment associated with this clinical population. Next, I will briefly consider two well-validated measures of hippocampal-dependent memory, as potential methods for characterizing and probing possible impairments in hippocampal-dependent memory among those with loss of control eating.

1.7 Measures of Hippocampal-Dependent Memory

1.7.1 Performance-Based Measures of Hippocampal Dependent Memory

The Wechsler Memory Scale – Fourth Edition (WMS-IV) (Wechsler, Holdnack, & Drozdick, 2009), provides a standardized neuropsychological measure of hippocampal-dependent memory (i.e., declarative memory) and visual working memory. Specifically, WMS-IV is comprised of six subtests (Figure 3), with an ability to derive composite scores for auditory memory, visual memory, visual working memory, immediate memory, and delayed memory. Four of the six subtests are repeated (i.e., logical memory, verbal paired associates, visual reproduction, and designs), which contribute an immediate memory composite score and delayed memory composite score. For the delayed

conditions of the subtests, information is recalled 20-30 minutes after the immediate conditions (Wechsler, Holdnack, & Drozdick, 2009).

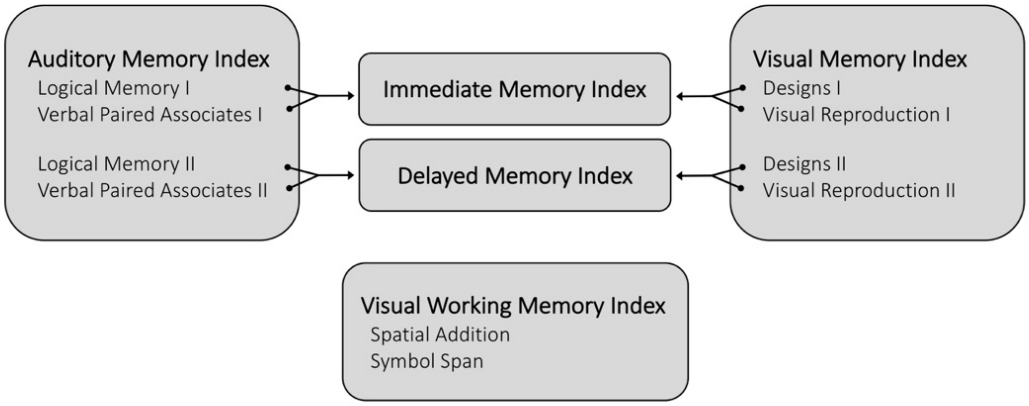


Figure 3: Test Framework of the Wechsler Memory Scale - Fourth Edition

In a series of validation studies for the current edition, the WMS-IV demonstrated high concurrent, discriminant, and predictive validity (Wechsler, Holdnack, & Drozdick, 2009). Specifically, the WMS-IV effectively discriminated between a sample of patients with mild Alzheimer's disease (*N*=48) and non-clinical, matched controls. Patients with Alzheimer's disease scored significantly lower across all subtests and index scores, compared to the matched control group. Given the clinical features of Alzheimer's disease (i.e., diagnostic criteria include established episodic memory deficits and temporal lobe atrophy, confirmed via neuroimaging), the observed group differences in memory performance were anticipated. However, they also held after controlling for

cognitive ability. (Lange & Chelune, 2006; Wechsler, Holdnack, & Drozdick, 2009). In addition, the WMS-IV demonstrated discriminant and predictive validity among a sample of patients (*N*=50) with lower levels of impairment (i.e., patients with Mild Cognitive Impairment). Patients with MCI scored significantly lower than matched controls on all subtest scores and index. Furthermore, patients with MCI who later received a diagnosis of Alzheimer's disease showed greater memory impairment on initial assessment, than patients with MCI who did not convert to Alzheimer's disease. (Perri, Serra, Carlesimo, & Caltagirone, 2007; Wechsler, Holdnack, & Drozdick, 2009).

In addition to the validation studies, numerous researchers have employed the WMS and similar measures (e.g., CVLT, Rey's Complex Figure Task) in conjunction with neuroimaging studies, to more precisely identify areas of congruence between performance measures of hippocampal-dependent functioning and those identified or mapped through fMRI. These studies have been conducted among healthy adults, those with mild cognitive complaints, and healthy aging populations, in addition to those with more advanced stages of memory concerns (e.g., Alzheimer's disease).

The body of research in this area suggests a central role of the hippocampus in memory, which maps onto WMS performance measures for both verbal and spatial memory tasks. More specifically, hippocampal total volume, and subfield volumes (especially subiculum and C1), have been positively associated with performance on the visual and verbal episodic memory tasks of the WMS, including across immediate and delayed conditions. Taken together, these findings suggest an important role of the WMS

in identifying hippocampal dysfunction. (Eldridge et al., 2005; Hayashi et al., 2018; Ezzati et al., 2016; Nauer et al., 2015; Suthana et al, 2011; Zeineh et al., 2003; Zammit et al., 2016).

1.7.2 Autobiographical Memory for Events

What facilitates detailed remembering of personally experienced events? The rich history of autobiographical memory research provides model a for probing such questions. According to the basic-systems model of episodic memory, developed by Rubin (2006), the hippocampus is also essential for autobiographical memories of specific, single, personally-experienced events. Specifically, the hippocampus and surrounding neural regions contribute to encoding of event-specific details at the time of an event, and facilitate the construction of detailed visual imagery at retrieval, through complex coordination between the explicit memory system, the emotion system, and the search-and-retrieval systems, which includes coordination between the memory systems, motivational states, and executive functioning systems (Rubin, 2006; Rubin & Umanath, 2014).

Research findings to date in the field of autobiographical memory research suggest a sense of reliving, visual imagery, an ability to construct a scene, and an ability to view that scene from the first-person perspective at memory retrieval, all help produce memories that are rated more vividly, which in turn, are positively associated with HDM functioning (Butler, Rice, Wooldridge, & Rubin, 2016; Rubin, 2006; Rubin &

Umanath, 2015; Rice & Rubin, 2009). Other factors that facilitate memory for personally experienced events include emotionally evocative experiences and motivational states that enhance and even narrow the focus of what is encoded and recalled, such as a focus threat-related details in the case of traumatic events (Berntsen, 2002; Rubin & Umanath, 2015; Rubin, Labar, et al., 2008). Several factors have also been shown to impair retrieval of autobiographical events, such as clinical states of PTSD, depression, dementia. Among these populations, autobiographical memories tend to be overgeneralized, less specific, or tend to involve the use of more general knowledge to "reconstruct" aspects of the memory that have been forgotten or were not encoded at the time of the event (Rubin, 2003; Rubin & Umanath, 2015; Butler et al., 2016).

No known studies have examined vividness of autobiographical memories among samples with binge or loss of control eating disorder, or sought to assess impaired retrieval of autobiographical memories in the context of these clinical samples. Further, no known studies have used the AMQ to examine autobiographical memory for specific eating-related events. Previous studies have used cue words to intentionally evoke food, weight, body image related themes or memories, but none have asked participants to recall an eating event memory to examine what is recalled and how vividly the event is remembered. For one example, a study by Johannessen & Bernsten (2009), examined the role of motivation for weight loss on autobiographical memory recall among a sample of dieters and non-dieters. They found memories recalled in response to dieting-related cue words were rated as more central to personal identify than non-dieting related cue

words, but found no differences on ratings of vividness between the groups for neutral cue words. (Johannessen & Bernsten, 2009).

1.7.3 Autobiographical Memory for Eating Events

Given the above limitations, the current study sought to develop a novel eating event memory probe for the Autobiographical Memory Questionnaire (AMQ-Eating), to characterize and probe general and eating-specific event memories. (Rubin et al., 2003). The (AMQ-Eating) was designed to elicit memories of eating events, which would then be rated across eight memory characteristics (i.e., rehearsal, visual imagery, auditory imagery, recollection, strength of emotion, positive emotion, negative emotion, and intensity of emotion), identical to the rating scales used in the standard AMQ (Rubin et al., 2003).

Given the complex interplay between emotion, motivation, and memory encoding, consolidation, and retrieval (Murty & Adcock; Murty & Dickerson, 2017; Murty et al., 2011), and research studies demonstrating LOC eating is associated with higher levels of negative emotions during *both* LOC eating episodes (e.g., distress, disgust, depressed mood), and non-LOC eating episodes (e.g., higher proportion of day-to-day eating experiences associated with guilt or fear of losing control among those with LOC eating), the AMQ-Eating measure was designed to be neutral. That is, the prompt for remembering an eating event was explicitly designed to be neutral (i.e., no imposed emotional valence for the eating event was stated or implied), and participants were

asked to voluntarily select a personally-experienced eating event to remember (see Appendix for instrument).

1.8 Current Study

The proposed study seeks to fill the existing gaps in the literature by characterizing hippocampal-dependent memory among *N*=66 young adult women who experience clinically significant loss of control (LOC) eating, and women with typical eating behaviors (i.e., women who do not experience clinically significant binge or LOC eating), by investigating the following specific aims:

1.8.1 Specific Aim 1: Characterize Memory Performance

Specific Aim 1. Characterize hippocampal-dependent memory (HDM) among *N*=66 young adult women (i.e., aged 18-30), with and without LOC eating, by examining performance on the Wechsler Memory Scale-Fourth Edition (WMS-IV), a standardized neuropsychological measure of declarative memory, including subtest scores for logical memory, verbal paired associates, visual reproduction, designs, and composite scores for auditory memory, visual memory, immediate memory, and delayed memory.

Hypothesis Aim 1. Research findings support of the vicious cycle (Davidson, Kanoski, Walls, & Jarrard, 2005; Davidson, Kanoski, Schier, Clegg, & Benoit, 2007; Kanoski & Davidson, 2001) and provide evidence that the WMS-IV differentiates clinical samples with known hippocampal-dependent memory deficits (e.g., patients with Mild Cognitive Impairment or Alzheimer's disease) from healthy participants (Wechsler, Holdnack, &

Drozdick, 2009). Therefore, it was predicted that participants with LOC eating would demonstrate lower scores across the range of WMS-IV subtests and indices that measure declarative memory, compared to participants in the typical eating group.

1.8.2 Specific Aim 2: Characterize Autobiographical Memory

Specific Aim 2a. Characterize memory for general autobiographical events among women, with and without LOC eating, using the standardized Autobiographical Memory Questionnaire (AMQ), which includes vividness ratings across eight memory characteristics (i.e., rehearsal, visual imagery, auditory imagery, recollection, strength of emotion, positive emotion, negative emotion, and intensity of emotion) (Rubin et al., 2003; 2004).

Hypothesis Aim 2a. Findings from autobiographical memory studies support an established link between dysfunction in hippocampally-dependent memory systems and less vivid ratings for visual imagery, auditory imagery, and a sense of reliving when remembering autobiographical events (Rubin & Umanath, 2015). Therefore, it was hypothesized that participants in the LOC eating group would rate autobiographical memories for general events with less vivid visual imagery, less vivid auditory imagery, and with a weaker sense of reliving, compared to participants in the typical eating group.

Specific Aim 2b. Characterize memory for autobiographical *eating* events among women, with and without LOC eating, using a *novel*, modified version of the Autobiographical Memory Questionnaire (AMQ-Eating), which includes rating eight

memory characteristics (i.e., rehearsal, visual imagery, auditory imagery, recollection, strength of emotion, positive emotion, negative emotion, and intensity of emotion), using standardized items from the general AMQ. (Rubin et al., 2003; 2004).

Hypotheses Aim 2b. This specific research aim was intended to be exploratory in nature, given the AMQ-Eating was a novel task established for the present study. Furthermore, the prompt for AMQ-Eating was explicitly designed to be neutral (i.e., no imposed emotional valence for the eating event was stated or implied), and participants voluntarily selected a personally-experienced eating event to remember (see Appendix for instrument). Given the complex interplay between emotion, motivation, and memory encoding, consolidation, and retrieval (Murty & Adcock; Murty & Dickerson, 2017; Murty et al., 2011), and research studies demonstrating LOC eating is associated with higher levels of negative emotions during *both* LOC eating episodes (e.g., distress, disgust, depressed mood), and non-LOC eating episodes (e.g., higher proportion of day-to-day eating experiences associated with guilt or fear of losing control among those with LOC eating), it was hypothesized that participants in the LOC eating group would endorse higher ratings of emotional experience (e.g., emotional intensity, strength of emotion) during recall of eating events, compared to typical eating group participants. Moreover, depending on the content and context of remembered eating events, memory vividness ratings could either be enhanced (e.g., if attending to the rewarding elements of cooking a favorite meal with a friend) or blunted (e.g., if recalling a LOC or otherwise distressing eating event).

1.8.3 Specific Aim 3: Contribution of High Fat, High Sugar and BMI

Specific Aim 3a. Examine whether self-reported dietary intake of high fat, high sugar (HFS) foods, and measured body mass index (BMI), emerge as significant predictors of hippocampal-dependent memory functioning, as assessed via performance on the WMS-IV, and memory vividness ratings on the autobiographical memory questionnaire (AMQ).

Hypothesis Aim 3a. Based on prior studies supporting the vicious cycle (Davidson, Kanoski, Walls, & Jarrard, 2005; Davidson, Kanoski, Schier, Clegg, & Benoit, 2007; Kanoski & Davidson, 2001), it was hypothesized that greater HFS dietary intake, and higher BMI, will significantly predict lower performance on the WMS and predict less vivid ratings on the AMQ across memory characteristics. Further, it was predicted that the magnitude of the effect would be larger for hippocampally-mediated tasks, such as the WMS-IV Design subtest, and the visual imagery, auditory imagery, and subjective sense of reliving ratings on the AMQ.

2. Methods

2.1 Recruitment and Procedures

Participants were recruited from the Research Triangle community and Duke University undergraduate student subject pool (SONA) for a study on thinking styles and eating behaviors. Participants were recruited via flyers posted and distributed in the community, on the Duke community listserve (i.e., Duke List), Dr. Zucker's Eating Disorder Research lab website and lab twitter feed, and through the Duke SONA web portal. Recruitment materials included a brief description of the eligibility criteria for each group (e.g., females aged 18-30, occasionally experience binge or loss of control eating, or healthy volunteers who do not experience binge or LOC eating). Interested participants were directed to contact the researchers via email and then asked to complete an online pre-screening survey using the secure platform Qualtrics, with the option to conduct the pre-screening by phone. The prescreening survey included an online consent form, a brief assessment of binge eating behaviors, and other screening questions related to the inclusion/exclusion criteria.

Eligible volunteers were invited to participate in a 3.5-hour in-person laboratory assessment visit. During the laboratory visit, participants were consented in-person by the graduate student researcher, and completed self-report measures of current hunger, mood state, psychological functioning, dietary intake/composition, eating-related behaviors, and two autobiographical memory questionnaires (general and food-specific

memories). Participants then completed standardized neuropsychological measures of hippocampal-dependent memory and cognitive ability, which were conducted by the a graduate student researcher (studying clinical psychology), and a trained undergraduate research assistant.

The graduate student researcher conducted the clinical diagnostic interview to assess eating behaviors and eating disorder symptoms, and took anthropometric measurements. Participants were instructed to eat a meal or a snack before their scheduled appointment time, ideally within one-hour prior to the study visit. Snacks were made readily available and offered as an option, if participants did not eat before their visit, or as needed, anytime during the visit or scheduled breaks. Participants were compensated up to $35.00 for completion of the in-person assessment visit. Duke undergraduates participating through the SONA portal received 0.5 credit hours for completing the prescreening survey and up to an additional 3.5 credit hours for completing the in-person laboratory assessment visit.

2.2 Participants

Participants were 66 young adult women (aged 18-30), recruited from the Research Triangle area of North Carolina and the Duke University undergraduate subject pool (SONA) between 2017-2019. The sample comprised 2 groups: 1) n=35 women *with* persistent binge or loss of control eating (including BED), across the weight spectrum; and 2) n=31 women *without* binge or LOC eating, across the weight spectrum. Exclusion

criteria for the group *without* binge or LOC eating included: current pregnancy; current diagnosis or history of an eating disorder (e.g., anorexia nervosa, bulimia nervosa, or BED); current mental health condition (i.e., history of mental health condition is acceptable); and current BDI score above 19 (i.e., scores <=19 indicate minimal to mild symptoms of depression). Exclusion criteria for the group *with* binge or LOC eating included: current pregnancy, current diagnosis of anorexia nervosa. Past history of eating disorder(s), current use of compensatory behaviors, elevated depressive symptoms, or current use of psychotropic medications to manage depression and/or anxiety were acceptable among the group with binge or LOC eating.

For inclusion into the study, participants were recruited via flyers and web announcements for a study on "thinking styles and eating behaviors." Specifically, two groups of adult females (age 18-30) were recruited. For the participants with binge or loss of control eating, they responded to a call for volunteers who were "interested in how your attention and thinking style might influence your eating?," with eligibility criteria listed as "you occasionally engage in binge eating or lose control of your eating." For those with typical eating behaviors, participants responded to a call for *healthy* female volunteers, who "do not occasionally engage in binge eating or lose control of your eating."

Despite these two recruitment strategies, researchers initially had difficulty recruiting participants who neatly fit into the binge eating disorder (BED) or no-BED classifications originally proposed for study inclusion. As such, the study team expanded

the inclusion criteria to include individuals with BED who experienced more severe symptom profiles (e.g., no upper limit to BDI, use of inappropriate compensatory behaviors allowed). This greatly decreased the number of screen fails for the study and allowed for a broader range of experiences with binge and loss of control eating.

2.3 Group Classification Scheme

Given the aforementioned conundrum of group classification, and emerging research on LOC eating and the proposed changes to the BED criteria for the upcoming (ICD-11) diagnostic criteria, as well as the formal definition of BED provided in the DSM-IV, we developed the following classification scheme to group included participants into those with clinically significant loss of control eating and those with "typical eating." For the loss of control (LOC) eating group, participants needed to experience weekly episodes of either objective or subjective binge eating, during the past 3 months, and with marked distress. This was assessed with the EDE Interview modules designed to diagnose BED (i.e., binge eating, dietary restriction, typical pattern of eating, use of inappropriate compensatory behaviors). Notably, and consistent with the new ICD-11 criteria, we did not require at least three behavioral features for inclusion into the LOC eating group, but we did retain the marked distressed criterion established for the DSM-IV diagnosis of BED.

Our sample included n=31 participants with typical eating (47%) and n=35 participants with loss of control (LOC) eating (53%). Among the LOC eating group, 11

(16.7%) met full diagnostic criteria for BED, 5 (7.6%) met full diagnostic criteria for BN, 10 (15.2%) met full diagnostic criteria for the newly proposed ICD-11 binge or LOC eating disorder, and 9 (25.7%) met diagnostic criteria for eating disorder not otherwise specified (EDNOS), which included subclinical binge or loss of control eating disorders (i.e., as defined by DSM-5 and ICD-11 criteria). Importantly, those with EDNOS endorsed marked distress regarding their binge or loss of control eating. Among the typical eating group, 17 (54.8%) endorsed some binge or loss of control eating (less than weekly), or overeating episodes, but did not meet marked distress criteria for inclusion in the LOC eating group, and 14 (45.2%) engaged in typical eating patterns, including some mild overeating or loss of control eating, but which was infrequent, and did not cause distress (e.g., often described as planned/budgeted for in typical pattern of eating). (see Table 2).

Table 2 Participant Classification Scheme, by Group

n (%)	Participants with LOC Eating (n=35)	n (%)	Participants with Typical Eating (n=31)
11 (31.4%)	Met full diagnostic criteria for BED (DSM-5)	17 (54.8%)	Endorsed some binge or loss of control eating; denied distress regarding objective and/or subjective binge eating episodes
5 (14.3%)	Met full diagnostic criteria for BN (DSM-5)		
10 (28.6%)	Met full diagnostic criteria for the ICD-11 LOC eating disorder	14 (45.2%)	Typical eating patterns, with some mild overeating episodes; denied distress regarding any eating experiences.
9 (25.7%)	Met diagnostic criteria for eating disorder not otherwise specified (EDNOS), (i.e., subclinical DSM-5 or subclinical ICD-11 criteria), including marked distress regarding binge or loss of control eating		

Note: LOC=loss of control; ICD-11 is the International Congress on Disorders, 11th edition, which include proposed changed to diagnosis of binge and LOC eating; DSM-5 is the American Psychiatric Association Diagnostic Manual (APA, 2013).

2.4 Study Prescreening Measures

2.4.1 Mental Health Screener

The DSM-5 Self-Rated Level 1 Cross-Cutting Symptom Measure—Adult (APA, 2013) was used to screen for possible mental health disorder(s). Specifically, the measure includes 1-2 screening questions per disorder/symptom cluster of interest (e.g., depression, anxiety, memory problems). Participants are asked to rate how much (or how often) they were bothered by a list of problems during the past two weeks (e.g., "Feeling more irritated, grouchy, or angry than usual?"). Each item was rated using a 5-point Likert scale from "0=none or not at all" to "4=severe or nearly every day." Eligibility cut-

points were pre-determined for each item (see Appendix for items and cut points). If indicated, additional follow-up information was gathered during the clinical interview that was conducted at the laboratory assessment visit (e.g., to characterize the participant's history of mental health concerns). (APA, 2013).

2.4.2 Depressive Symptoms

Depressive symptoms were assessed using the Beck Depression Inventory, Second Edition (BDI-II). The BDI-II is a 21-item measure designed to assess presence and severity of symptoms of depression over the past two weeks (Beck, Steer, & Brown, 1996). Each item includes 4 responses, ranging in value from 0 to 3. Item scores are summed to create an overall score, with higher scores indicating more severe depressive symptoms (range 0 to 63). The following cut-points have been established for the BDI-II: 0-13=minimal depression; 14-19=mild depression; 20-28=moderate depression; 29-63=severe depression. The BDI-II has demonstrated high internal consistency (i.e., α=.93 among a large college sample, and α=.92 among outpatients) (Beck et al., 1996). In the present sample, internal consistency was adequate (α = .79).

2.4.3 Eating Pathology

For the prescreen survey, symptoms of eating pathology were assessed using the Eating Pathology Symptom Inventory (EPSI) (Forbush et al., 2013). The EPSI was developed as a comprehensive and multidimensional measure of pathological eating behaviors. The EPSI contains 45-items, in which participants were asked to respond how

frequently various statements applied to them on a 5-point Likert scale ranging from "never" to "very often." The measure contains eight subscales: Body Dissatisfaction, Binge Eating, Cognitive Restraint, Purging, Restricting, Excessive Exercise, Negative Attitudes toward Obesity, and Muscle Building. Scores for each subscale were calculated by summing the responses for each item in the subscale. A validation study of the EPSI in a sample of female college students (*N*=625) showed adequate internal consistency for most subscales, with a median coefficient alpha across subscales of 0.87 (Forbush, Wildes, & Hunt, 2013). The Binge Eating subscale was used as a preliminary assessment of group eligibility, to determine if participants would likely be classified into the binge or LOC eating clinical group or the healthy control group. The Binge Eating subscale was also used as a measure of binge eating severity in covariate analyses. The Binge Eating subscale has demonstrated good internal consistency (α=.86) among a sample of undergraduate students and excellent internal consistency (α=.93) among a sample of eating disorder patients (Forbush et al., 2013; Forbush, Wildes, & Hunt, 2014). In the current study, the binge eating subscale demonstrated excellent internal consistency (α=.92) among our sample (*N*=66).

2.5 Study Measures – Clinician Administered

All of the clinician-administered assessments cognitive ability and memory functioning/performance were conducted using standardized tests and administration procedures. The assessments were administered by the graduate student researcher

(i.e., doctoral student in clinical psychology) and a trained undergraduate research assistant, and were completed in a non-medical, low-stimulation room in our laboratory research offices in Durham, NC.

2.5.1 Eating Disorder Behaviors

Eating disorder behaviors were assessed using the standardized Eating Disorder Examination (EDE) interview. The EDE (Cooper & Fairburn, 1987) is the most well-established and validated structured interview used to assess eating disorder psychopathology, and has demonstrated strong reliability among patients with BED (Grilo, Masheb, Lozano-Blanco, & Barry, 2004). The EDE, version 17.0D (April 2014) (Fairburn, Cooper, & O'Connor, 2014). The EDE was used in the present study to assess participants' pattern of eating, episodes of objective binge eating, loss of control eating (i.e., subjective binge eating) and overeating (OO), behavioral features of objective binge episodes (i.e., part of the diagnostic criteria for BED), restraint over eating, eating concern (e.g., fear of losing control over eating), and compensatory behaviors (i.e., self-induced vomiting, laxative misuse, diuretic misuse, driven exercise, dietary restriction, and other extreme weight control behaviors).

In addition to the standard modules used to diagnose binge eating disorder, a few supplemental questions were added to more fully assess LOC eating, based on the ICD-11 proposed revisions to the diagnosis of binge eating disorder, which diminish the importance of "objectively large amount" of food consumed during a binge episode, in

favor of either subjective or objective binge eating episodes. The supplemental questions were used to assess distress and the behavioral features associated with subjective binge eating episodes. Please see Appendix E. for the supplemental questions. (Palavras et al., 2013; Goldschmidt, 2017; Palavras et al., 2018).

2.5.2 Cognitive Ability

The two-subtest form of the Wechsler Abbreviated Scale of Intelligence, Second Edition (WASI-II) (Wechsler, 2011) was used to assess general cognitive ability. The two-subtest form includes the vocabulary and matrix reasoning subtests, provides a full scale IQ score (FSIQ-2), and can be administered in about 15 minutes (compared to the 4 subtest form, which takes 30 minutes). The WASI-II is considered a brief, reliable measure of cognitive ability that produces FSIQ scores that are comparable with the more comprehensive and lengthy (i.e., 60-90 minute) Wechsler Adult Intelligence Scale, Fourth Edition (WAIS-IV) (Wechsler, 2011).

2.5.3 Memory Performance

The Wechsler Memory Scale – Fourth Edition (WMS-IV) (Wechsler, Holdnack, & Drozdick, 2009), provides a standardized neuropsychological measure of hippocampal-dependent memory (i.e., declarative memory) and visual working memory. Specifically, WMS-IV is comprised of six subtests, with an ability to derive composite scores for auditory memory, visual memory, visual working memory, immediate memory, and delayed memory. Four of the six subtests are repeated (i.e., logical memory, verbal

paired associates, visual reproduction, and designs), which contribute an immediate memory composite score and delayed memory composite score. For the delayed conditions of the subtests, information is recalled 20-30 minutes after the immediate conditions (Wechsler, Holdnack, & Drozdick, 2009).

The auditory memory domain is comprised of two subtests: logical memory and verbal paired associates. The logical memory subtest includes two stories which are read to the examinee once (i.e., single trial learning). The examinee recalls each story, which are recorded verbatim and scored for number of details accurately recalled. For the verbal paired associates task, there are four learning trials of 14 word pairs, including 4 pairs that are semantically related (e.g., sky and cloud) and 10 pairs that are not semantically related (e.g., bed and lost). During prompted recall (i.e., examinees asked "which word goes with bed"), the examinee receives feedback on each answer, across all four trials (e.g., "that's correct" or "bed goes with lost") (Wechsler, Holdnack, & Drozdick, 2009).

Within the visual memory domain, two subtests are included: visual reproduction and designs. For the visual reproduction task, five (increasingly complex) figures are presented for 10 seconds each, and examinees are asked to draw the figure(s) after each presentation (immediate free recall), and again after a 20-30 minute delay. Items are scored based on key visual features and recall of the relationship between design elements (e.g., the half circle is to the left of the triangle, and the vertex of the triangle touches the middle third of the arc of the semi-circle). For the designs subtest, abstract

designs are presented in the formation of a 4 x 4 grid. For each of the four stimuli, an increasing number of designs are to be recalled (ranging from 4 to 8 design cards in the grid). After presentation of each stimulus, the examinee must select the correct cards from a group cards (comprised of 1:1 ratio of target cards to distractor cards). Designs are scored for content (i.e., target card selected) and correct spatial location in the grid, with partial credit given (e.g., distractor card selected) and bonus points awarded (i.e., for placing the target card in the correct location in the grid). (Wechsler, Holdnack, & Drozdick, 2009).

2.5.4 Height, Weight, BMI, and Waist Measurements

Height, weight, and waist circumference were assessed in our laboratory following National Health and Nutrition Examination Survey (NHANES) guidelines and procedures (NHANES, 2013). Participants were weighed in one layer of light clothing, without shoes and pockets emptied, using a calibrated, high capacity digital flat scale (i.e., Seca 813) that measured to the nearest 0.01 kg. Height was measured to the nearest 0.1 cm using a portable stadiometer (Tanita BWB-800). Waist circumference was measured to the nearest 0.1 cm using a retractable tape measure. BMI (kg/m^2) was calculated for each participant using measured height and weight.

2.6 Study Measures – Self-Report

2.6.1 Autobiographical Memory – General and Eating Events

The Autobiographical Memory Questionnaire (AMQ) prompts participants to remember a specific event from their own life and then answer a series of questions about the properties associated with remembering the event (Rubin, Schrauf, & Greenberg, 2003). Participants first recorded a general memory by responding to the standard AMQ prompt to remember a specific event from their own life. Then, participants recorded an eating-related memory by responding to a modified AMQ prompt (developed for the present study), to recall a specific eating event from their own life, such as eating a meal, food, or snack (see Appendix D). Then participants answered a series of questions from the AMQ regarding characteristics of the memory, including ratings of: 1) rehearsal, visual imagery, auditory memory, recollection/reliving, strength of emotions, positivity of emotions, negativity of emotions, and intensity of emotions, which were each rated on a 7-point scale; 2) recency of the event (i.e., with response options ranging from within the past day to more than 10 years ago); and 3) event age estimated in years (see Appendix D. for specific questions and anchors from the AMQ) (Rubin, Schrauf, & Greenberg, 2003).

2.6.2 Dietary Intake of Fat and Sugar

The Dietary Fat and Sugar – Short Questionnaire (DFS-SQ) was modified and used to assess intake of highly palatable foods in the present study, as an estimate of saturated fat and refined sugar intake. The original DFS-SQ was developed in Australia

(Francis & Stevenson, 2013), and included 26 items, each rated on a 5-point Likert scale for frequency of consumption during the past 12 months (i.e., "once a month or less" to "5 or more times per week"). Scores on the DFS-SQ were calculated by adding scores for each item to create a total sum score, with higher scores indicating greater frequency of consuming HFS foods (range of scores 24-120). In their initial validation study among a sample of adults (*N*=40), internal consistency for the DFS-SQ was adequate at ($\alpha = 0.76$) (Francis & Stevenson, 2013).

For the present study, the following modifications were made to the DSF-SQ: 1) the wording of some food items were changed to reflect culturally-specific American terms; 2) the response options were expanded from a 5-point scale to an 8-point scale; and 3) several food items were added to reflect common contributions to saturated fat and added sugar intake in the American diet (i.e., 36 total items). To illustrate these modifications, a few examples are highlighted here, with the full set of items and modifications detailed in the Appendix (including a side by side comparison with the original measure). For example, the original measure's item for non-chocolate type candy was reworded from "lollies" to "other candy, like hard candy, caramels, lollipops, jelly beans." The response option changes included adding anchors to reflect frequency ratings for "never" and "everyday." The modified response options were based on other food frequency questionnaires commonly used in epidemiological studies in the United States, including the well-validated Block Food Frequency Questionnaire (Block, Woods, Potosky, & Clifford, 1990). For a final example, items such as "sweetened breakfast

cereals," were added to assess other significant sources of dietary fat and sugar, common in the American diet. The reliability of the modified DSF-SQ used in the present study (36 items) was high, i.e., it demonstrated good internal consistency (α=.83) among our sample of young women (*N*=66).

2.6.3 Hunger and Satiety

An appetite visual analogue scale (VAS) was used to measure hunger, satiety, and fullness (Flint, Raben, Blundell, & Astrup, 2000) at the beginning of the laboratory assessment visit. The VAS included three 100mm bars with free range sliders, anchored by two points. For example, to assess hunger, the hunger VAS anchors were "not at all hungry" at one end of the bar, and "extremely hungry" at the other end of the bar. Participants were asked to place the free range slider into the position on the bar that best represented their current level of hunger, satiety, and fullness. The bar position selected was given a value (range 0 to 100) on the backend (i.e., participant was not shown the number on the screen). Participants were also asked to estimate the amount of time since their last meal or snack, which was used to confirm they had eaten within approximately 1 hour of their scheduled study visit.

2.6.4 Affective State

The Positive and Negative Affect Schedule (PANAS) was used to measure current mood state (Watson, Clark, & Tellegen, 1988). The PANAS includes 20 items containing emotion words (e.g., interested, scared, nervous, determined), which are rated using a 5-

point scale (i.e., 1=very slightly or not at all; 2=a little; 3=moderately; 4=quite a bit; 5=extremely) for discrete time periods, such as during the past week. The present study used the time period, "right now, that is at the present moment," (Watson et al., 1988), administered at the beginning of the laboratory assessment visit. The PANAS has demonstrated strong psychometric properties, including high internal consistency (i.e., α range .84 to .90) and low inter-correlation between the positive and negative subscales (i.e., sharing only 1-5% of their variance) (Watson et al., 1988). The measure was reliable in the present sample as well (*N*=66), with good internal consistency demonstrated for both the positive subscale (i.e., α=.86) and the negative subscale (i.e., α=.85).

2.6.5 Food Reward Responsiveness

Responsiveness to food cues in the environment was assessed using the revised version of the Power of Food Scale (PFS) (Lowe et al., 2009), and revised (Cappelleri et al., 2009). The PFS is a 15-item self-report questionnaire that measures individual differences in psychological responsiveness to the availability, physical presence, and taste (but not ingestion) of food. The questionnaire consists of statements, to which participants rate their agreement on a 5-point Likert scale, from "1 = do not agree at all" to "5 = strongly agree" (Lowe et al., 2009). The scale contains three subscales: food available (6 items), food present (4 items), and food tasted (5 items). Each subscale is computed as the sum total of the items in the subscale, with scores ranging from 5 to 30 for each subscale, and from 15 to 75 for the overall measure, with higher scores representing higher

responsiveness to food cues. In the original validation article, the PFS demonstrated high internal consistency (α = .91) for the full scale, among a sample of healthy college students (*N*=466) (Lowe et al., 2009). Test-retest reliability was also adequate when the scale was administered again 4 months later (r = .77, $p < 0.01$) to a smaller subsample of healthy college students (*N*=72) (Lowe at al., 2009). The revised PFS has also demonstrated high reliability among a clinical sample of adults with obesity enrolled in a weight loss treatment study (i.e., range α = 0.81 to α = 0.91) (Cappelleri et al., 2009). In the present study (*N*=66), the overall scale (15 items) demonstrated high reliability (α = .92). The food available and food present subscales had good internal consistency (α = .85 and α = .84, respectively), while the food tasted subscale (α = .75) demonstrated adequate reliability in the present sample.

2.7 Data Analytic Strategy

2.7.1 Sample Size Determination

The sample size of 66 participants (i.e., with 2 groups of approximately 33 participants each) was calculated a priori using G*Power 3.1 (Faul, Erdfelder, Buchner, & Lang, 2009; Faul, Erdfelder, Lang, & Buchner, 2007), commonly used in behavioral research. The compromise power analysis was used, which is useful when balancing available resources and Type I and Type II error risks (Faul et al., 2007). The analysis was based on statistical tests for ANOVA (i.e., fixed effects, omnibus, one-way), with *N*=66 participants, two groups, and a medium population effect size of *f*=0.35, which yielded a

significance level of α=0.05 and power of β=0.80. The medium effect size was selected based on the (limited) research examining working memory deficits in BED. For example, in a study examining cognitive functioning among *N*=76 obese subjects, with and without BED, (who were matched on BMI, age, years of schooling, and general intelligence), individuals with BED showed relatively greater deficits on a measure of working memory (i.e., digit span backward) (M_{BED}=4.29, $M_{Non\text{-}BED}$=5.13, OR=2.30, *p*=.02) (Duchesne et al., 2010). In general, ORs close to 1.00 represent a weak relationship between variables, whereas ORs over 3.0 (or less than .33) represent a strong relationship (Haddock, Rindskopf, & Shadish, 1998). Given the aforementioned OR was 2.3 (Duchesne et al., 2010), it was predicted that a medium effect size would likely be observed in the present study.

2.7.2 Data Analyses

Data analyses were performed using SPSS Statistic, version 25 (IBM Statistic, 2018). Tests of hypotheses were based on the general linear model (GLM), to investigate the relationship between the dependent variables and the predictor variables. To ensure GLM would appropriately accommodate the analyses for the present sample, the data were visually inspected and statistically examined to verify a reasonable approximation of the normal distribution and homogeneity of variance, for the entire sample (*N*=66) and within each group (i.e., binge/LOC group, n=35; and typical eating group, n=31) (Field, 2012). When data were examined across groups, BMI values were significantly not

normal, $D(66) = 0.11$, $p < .05$. The assumptions of normality were then tested within groups, and BMI values remained significantly different from the normal distribution within the binge/LOC eating group (i.e., $D(35) = 0.17$, $p < .05$, skewness=2.52, kurtosis=11.08), but not within the typical eating group ($D(31) = 0.09$, $p = .20$, skewness=.69, kurtosis=.21). As such, BMI data were transformed using three different models: inverse, log, and square root of the BMI values, and the log transformed BMI values were selected, as they best met the assumptions of normality, while retaining the rank order of the variable.

All other outcome variables for demonstrated skewness <|2| and kurtosis values <|3.29|within acceptable limits. (Field, 2012). Based on these diagnostics, it was deemed appropriate to continue the analyses using parametric tests to examine the study aims and hypotheses. Table 3 provides normality indicators for the continuous model covariates examined in Aim 3. It was also determined at the preliminary analyses stage for Aim 3 that the BMI log transformed variable violated another assumption (i.e., homoscedasticity). Therefore, to retain the BMI log transformed variable in the analyses, a more robust form of regression was selected for all analyses in Aim 3, which does not rely on assumptions of normality and homoscedasticity (Field, 2012, p. 350-354). Specifically, robust regression analyses were utilized, which generate 95% bias corrected and accelerated confidence intervals and standard errors, based on 1000 bootstrap samples. These data are demarcated as such and presented in the data summary tables and narratives below. (Field, 2012, p. 350-354).

Table 3 Normality Indicators for the Continuous Model Covariates

Variable	Group	Skewness	Kurtosis
Body Mass Index	0	.69	.21
	1	2.52	11.08
Inverse of BMI	0	-.12	-.40
	1	-.26	2.91
Beck Depression Index (BDI)	0	.94	.47
	1	1.29	1.51
DFS Intake	0	-.60	.96
	1	.09	-.52
EPSI Binge Eating Subscale	0	.48	-.25
	1	.62	-.50
Total LOC Eating Episodes	0	.61	1.39
	1	-1.00	2.76
WASI-II Composite Score	0	.46	-.50
	1	.62	-.18
WASI-II Percentile Rank	0	-.72	.02
	1	-.74	-.69

Note: EPSI=Eating Pathology Symptoms Inventory (Forbush et al., 2013); DFS=Dietary Intake of Fat and Sugar, modified from (Francis & Stevenson, 2013); BDI=Beck Depression Index (Beck, 1996); LOC=Loss of Control; WASI=Wechsler Abbreviated Scale of Intelligence – 2nd Edition

3. Results

3.1 Sample Demographics

Descriptive statistics were used to summarize the sample, clinical, and eating characteristics, stratified by group. Participant demographic variables are presented in Table 4. The mean age was 21.9 years old for the typical eating group, and was 23.1 years old for the loss of control (LOC) eating group. In terms of race and ethnicity, 59% of participants in the overall sample identified as White; 27.2% identified as Asian; 7.6% identified as Multi-racial/Multi-ethnic; 4% identified as Black or African American; and 4.5% identified as Hispanic/Latino(a/x). The sample also included 27.2% of participants who endorsed English as a Second Language.

Table 4 Sample Demographics, by Group

	Sample with Typical Eating (n=31)	Sample with Loss of Control Eating (n=35)
Age: Mean (SD), Range	21.86 (2.99), 18.58-29.5	23.05 (2.97), 18.28-29.21
Race/Ethnicity: Count (%)		
Asian	10 (32.3)	8 (22.9)
Black	2 (6.5)	1 (2.9)
Hispanic/Latino	1 (3.2)	2 (5.7)
Multi-Racial	0 (0)	5 (14.3)
White	18 (58.1)	21 (54.3)
Level of Education: Count (%)		
High School	3 (9.7)	0 (0)
College Student	17 (54.8)	15 (42.9)
College Degree	7 (22.6)	13 (34.3)
Graduate Student	3 (9.7)	7 (20.0)
Masters Degree	0 (0)	1 (2.7)
Professional/Doctoral Degree	1 (3.2)	0 (0)

Body Mass Index: Mean (SD), Range	22.28 (2.79), 17.92-29.43	24.04 (3.97), 17.05-41.71
English as a Second Language	7 (22.6)	11 (31.4)

Note: SD=Standard Deviation; Participants could endorse more than one race/ethnicity, and the numbers here sum to more than 100%.

3.2 Clinical Characteristics

For clinical variables assessed, descriptive statistics are reported in Table 5. The sample data are stratified by group, for clarity. Independent samples t-tests (two-tailed, $\alpha = .05$) were used to compare group means for the following measures: all Eating Pathology Symptoms Inventory (EPSI) subscales (Forbush et al., 2013), all Power of Food (PFS) subscales (Lowe et al., 2009; Cappelleri et al., 2009), modified Dietary Intake of Fat and Sugar (DFS) (Francis & Stevenson, 2013), Beck Depression Index (BDI) (Beck, 1996), and Positive and Negative Affect Schedule (PANAS) (Watson et al., 1988).

Statistically significant differences were found between observed group scores for all of the EPSI subscales, except Muscle Building, with participants in the LOC eating group scoring significantly higher on all of the subscales, compared to the group with typical eating. Significant group differences were also found on the Food Present subscale of the Power of Food Scale (PFS) and depression score for the Beck Depression Index (BDI), with the clinical sample scoring significantly higher on both measures. There were no statistically significant group differences on the modified Dietary Fat and Sugar

Intake (DFS), or on either the positive or negative subscales of the Positive and Negative Affect Schedule (PANAS).

Table 5 Sample Clinical Characteristics, by Group

Clinical Variable, Mean (SD)	Sample with Typical Eating (n=31)	Sample with Loss of Control Eating (n=35)	t	*p*	(df)
EPSI subscale					
Body Dissatisfaction	6.94 (4.71)	14.60 (6.55)	-5.50	<.001^{++}	(64)
Binge Eating	3.45 (2.29)	10.82 (7.33)	-5.65	<.001^{++}	(64)
Cognitive Restraint	4.48 (2.20)	5.86 (2.81)	-2.22	.03*	(64)
Purging	0.10 (0.30)	1.49 (3.64)	-2.25	.03*	(64)
Restricting	2.13 (2.95)	4.31 (5.07)	-2.16	.04*	(64)
Excessive Exercise	4.39 (4.24)	7.69 (5.60)	-2.71	.01*	(64)
Negative Attitudes Towards Obesity	1.74 (2.16)	4.74 (5.86)	-2.82	<.01^{+}	(64)
Muscle Building	1.32 (1.72)	2.29 (3.03)	-1.61	.12	(64)
Power of Food Scale					
PFS Food Available Subscale	15.77 (4.17)	16.74 (4.11)	-0.89	.38	(55)
PFS Food Present Subscale	8.79 (3.14)	12.21 (4.04)	-3.56	<.01^{+}	(55)
PFS Food Tasted Subscale	15.87 (4.24)	16.00 (3.55)	-0.13	.90	(55)
Dietary Intake of Fat and Sugar					
DFS – Short Form, Modified	98.33 (22.60)	96.75 (21.26)	0.28	.78	(60)
Beck Depression Index					
BDI Depression score	3.65 (3.30)	10.29 (9.23)	-3.98	<.001^{++}	(64)
PANAS					
Positive Subscale Negative Subscale	27.34 (7.26) 11.14 (1.33)	24.74 (6.83) 12.85 (4.98)	1.47 -1.93	.15 .06	(61) (61)

Note: EPSI=Eating Pathology Symptoms Inventory (Forbush et al., 2013); PFS=Power of Food Scale (Lowe et al., 2009; Cappelleri et al., 2009); DFS=Dietary Intake of Fat and Sugar, modified from (Francis & Stevenson, 2013); BDI=Beck Depression Index (Beck, 1996); PANAS= Positive and Negative Affect Schedule (Watson et al., 1988); SD=Standard Deviation; t=t value; df=degrees of freedom. Independent samples t-tests (two-tailed, $\alpha = .05$) were used to compare group means. $^{*}p < .05$, $^{+}p < .01$, $^{++}p < .001$

3.3 Eating Characteristics

Additionally, several eating patterns were rated during the Eating Disorder Examination (EDE) Interview (Fairburn et al., 2014), and data from a few clinically meaningful variables are presented in Table 6. Independent samples t-tests (two-tailed, $\alpha = .05$) were used to compare group means. Data for typical pattern of eating during the past month are presented for breakfast, lunch, and dinner, with responses rated on a seven-point scale (i.e., 0=meal not eaten, 1=meal eaten 1-5 days, 2=meal eaten 6-12 days, 3=meal eaten 13-15 days, 4=meal eaten 16-22 days, 5=meal eaten 23-27 days, and 6=meal eaten every day). Total number of objective binge episodes, subjective binge eating episodes, and loss of control (LOC) eating episodes (i.e., sum of objective and subjective binge episodes) are presented for the past three months (Fairburn et al., 2014).

There were no statistically significant differences found between average group ratings on the pattern of eating variables: breakfast, lunch, or dinner. For the loss of control (LOC) eating behaviors, statistically significant group differences were found for total number of LOC eating episodes, total objective binge episodes, and total subjective binge eating episodes. These differences were all in the expected direction, with the sample of participants in the LOC eating group endorsing significantly more loss of control eating behaviors compared to participants in the typical eating group.

Table 6 Sample Eating Characteristics, by Group

EDE Interview Ratings, Mean (SD)	Sample with Typical Eating (n=31)	Sample with Loss of Control Eating (n=35)	t	df
Pattern of eating (past month)				
Average rating breakfast	4.61 (1.43)	3.97 (1.77)	1.60	(64)
Average rating for lunch	4.84 (1.39)	5.31 (1.08)	-1.56	(64)
Average rating for dinner	5.77 (0.50)	5.66 (0.68)	0.79	(64)
LOC eating (past 3 months)				
Total LOC eating episodes	6.45 (7.17)	36.26 (3.03)	-9.05^{++}	(46)
Objective binge episodes	3.29 (5.09)	20.63 (16.12)	-6.03^{++}	(42)
Subjective binge episodes	3.16 (5.29)	15.63 (10.10)	-6.38^{++}	(53)

Note: EDE=Eating Disorder Examination Interview (Fairburn et al., 2014); LOC=Loss of Control; SD=Standard Deviation; t=t value; df=degrees of freedom. Independent samples t-tests (two-tailed, α = .05) were used to compare group means. For the LOC eating ratings, robust statistics for t and df are reported, which do not rely on assumptions of homoscedasticity. *$p < .05$, $^{+}p < .01$, $^{++}p < .001$

3.4 Cognitive Characteristics

Results from the Wechsler Abbreviated Scale of Intelligence-Second Edition (WASI-II) (Wechsler & Zhou, 2011) indicated there were no statistically significant differences between groups on the composite scores for the full-scale IQ, $F(1,63) = 3.00$, $p = .09$, $d = 0.42$. On average, participants in the typical eating group had a full-scale IQ slightly below participants in the LOC eating group ($M_{Typical}$=115.16, SD=11.59; M_{LOC}=119.88, SD=10.40). Although this difference was not statistically significant, it did represent a small to medium effect, $d = 0.42$. There were no statistically significant differences on the two subtest scores, i.e., vocabulary and matrix reasoning (Table 7).

Table 7 Sample Cognitive Characteristics, by Group

WASI-II, Mean (SD)	**Sample with Typical Eating (n=31)**	**Sample with LOC Eating (n=35)**	*F*(1,63)	*p*	Cohens *d*
Full-Scale IQ (2-subtests)	115.16 (11.59)	119.88 (10.40)	3.00	.09	.42
Vocabulary Raw Score	43.48 (3.87)	45.06 (3.63)	2.86	.10	.42
Matrix Reasoning Raw Score	23.13 (2.32)	23.91 (2.02)	2.11	.15	.36

Note: WASI-II=Wechsler Abbreviated Scale of Intelligence-Second Edition (Wechsler & Zhou, 2011); LOC=Loss of Control; SD=Standard Deviation; Full-Scale IQ was estimated using the two-subtest form of the test; *F* statistics presented are for tests of between group differences, based on (1,63) degrees of freedom. Effect sizes were estimated using Cohens *d*; $^{*}p < .05$, $^{+}p < .01$, $^{++}p < .001$

3.5 Results Aim 1

For this Aim, one-way between-subjects ANOVAs were conducted to assess group differences between the typical and LOC eating groups, on subtests and composite scores that comprise the Wechsler Memory Scale-Fourth Edition (WMS-IV) (Wechsler, 2009). The results are summarized in Table 8, and are described below for each of the following domains: visual memory, auditory memory, immediate memory, and delayed memory.

Table 8 Standardized Memory Performance, by Group

WMS-IV Index Score: Mean (SD) Subtest Raw Score: Mean (SD)	Typical Eating Group (n=31)	LOC Eating Group (n=35)	*F*(1,63)	*p*	Cohens *d*
Visual Memory Index	**113.00 (10.56)**	**106.74 (11.26)**	**5.13**	**.03***	**.55**
Designs I	88.38 (11.13)	82.56 (10.70)	4.47	.04*	.52
Designs II	79.41 (13.68)	70.56 (12.73)	7.07	.01*	.64
Visual Reproduction I	40.72 (2.22)	40.06 (2.63)	1.16	.29	.27
Visual Reproduction II	37.31 (5.70)	36.29 (5.52)	0.52	.48	.18
Auditory Memory Index	**113.93 (10.71)**	**109.12 (10.98)**	**3.08**	**.08**	**.44**
Logical Memory I	31.38 (5.70)	29.76 (4.71)	1.52	.22	.23
Logical Memory II	29.62 (5.99)	28.09 (5.97)	1.03	.31	.29
Verbal Paired Associates I	46.48 (6.29)	41.91 (9.90)	4.60	.04*	.53
Verbal Paired Associates II	13.28 (1.22)	12.38 (2.22)	3.74	.06	.53
Visual Working Memory Index					
Symbol Span	28.81 (5.12)	28.36 (5.02)	0.11	.75	.09
Immediate Memory Index	**113.17 (10.46)**	**107.44 (9.99)**	**4.94**	**.03***	**.54**
Delayed Memory Index	**117.34 (9.63)**	**110.82 (10.80)**	**6.30**	**.02***	**.61**

Note: WMS-IV=Wechsler Memory Scale-Fourth Edition (Wechsler, 2009); LOC=Loss of Control; SD=Standard Deviation. *F* statistics presented are for tests of between group differences, based on (1,63) degrees of freedom; *$p < .05$, $^{+}p < .01$, $^{++}p < .001$

3.5.1 Visual Memory

Within the visual memory domain, there were two statistically significant group differences at the subtest level, and one at the index level. On average, participants in the typical eating group had higher scores on the Designs I subtest, (*M*=88.38, SD=11.13),

than those in the LOC eating group (*M*=82.38, SD=10.70), which was statistically significant, $F(1,63) = 4.47$, $p = .04$, and represented a medium-sized effect, $d = 0.52$. Participants in the typical eating group also had higher scores on the Designs II subtest, (*M*=79.41, SD=13.68), compared to those in the LOC eating group (*M*=70.56, SD=12.73), which was statistically significant, $F(1,63) = 7.07$, $p = .01$, and also represented a medium-sized effect, $d = 0.64$. At the index level, significant group differences were found between the average Visual Memory Index scores $F(1,63) = 5.13$, $p = .03$, with the typical eating group scoring significantly higher (*M*=113.00, SD=10.56), than the LOC eating group (*M*=106.74, SD=11.26), which represented a medium-sized effect, $d = 0.55$.

3.5.2 Auditory Memory

Within the auditory memory domain, there was one statistically significant group difference at the subtest level, and a medium-sized effect at the index level. On average, participants in the typical eating group had higher scores on the Verbal Paired Associates I subtest, (*M*=46.48, SD=6.29), compared to those in the LOC eating group (*M*=41.91, SD=9.90), which was statistically significant, $F(1,63) = 4.60$, $p = .04$, and represented a medium-sized effect, $d = 0.53$. Participants in the typical eating group had higher scores on Auditory Memory Index, (*M*=113.93, SD=10.71), compared to those in the LOC eating group (*M*=109.12, SD=10.98). While this difference was not statistically significant, $F(1,63) = 3.08$, $p = .08$, it did represent a medium-sized effect, $d = 0.44$.

3.5.3 Immediate and Delayed Memory

The Immediate Memory Index is derived from all of the immediate recall scaled scores from Logical Memory, Verbal Paired Associates, Designs, and Visual Reproduction, while the Delayed Memory Index is derived from all the delayed recall scaled scores across those same domains. Participants in the typical eating group had higher scores on the Immediate Memory Index, (*M*=113.17, SD=10.46), compared to those in the LOC eating group (*M*=107.44, SD=9.99). This difference was statistically significant, $F(1,63) = 4.94$, $p = .03$, and represented a medium-sized effect, $d = 0.54$. In addition, participants in the typical eating group had higher scores on the Delayed Memory Index, (*M*=117.34, SD=9.63), compared to those in the LOC eating group (*M*=110.82, SD=10.80). This difference was statistically significant, $F(1,63) = 6.30$, $p = .02$, and represented a medium-sized effect, $d = 0.61$.

3.6 Results Aim 2

One-way between-subjects ANOVAs were conducted to assess group differences between the typical and loss of control (LOC) eating groups, on mean ratings of vividness for autobiographical memories, as characterized by Rubin, Schrauf, and Greenberg (2003). Key findings from the statistical inference tests, effect size calculations, and descriptive direction of mean group differences are described herein, organized by memory characteristic. Under each memory domain, both types of events recalled (i.e., general and eating events) are included. Given the exploratory nature of the AMQ-Eating,

no specific hypotheses were explored regarding potential within-subject effects for the present study, but findings are organized below, and summarized in Table 9, to facilitate consideration of such hypotheses, for the interested reader.

Overall, results indicated a significant main effect of group on positive emotion ratings during recall of both general and eating events, with participants in the typical eating group rating both types of autobiographical memories as significantly more positive than participants in the LOC eating group. Additionally, there was a significant main effect of group on ratings of emotional intensity while remembering the general event. That is, participants in the typical eating group rated higher levels of emotional intensely during recall of general events, compared to participants in the LOC eating group. Tests of interaction between group (i.e., typical eating vs. LOC eating) and event memory type (i.e., general vs. eating) were probed, and all interaction effects were non-significant (p values > 0.05).

Positive Emotions. On average, participants in the typical eating group rated their emotions more positively when remembering general events (M=5.33, SD=1.86), compared to those in the LOC eating group (M=4.27, SD=2.00). The difference between groups was statistically significant $F(1,58) = 4.43$, $p = 0.04$, and the magnitude of this difference represented a medium-sized effect, $d = 0.55$. Additionally, participants in the typical eating group also rated their emotions more positively when remembering eating events (M=6.15, SD=1.10), compared to those in the LOC eating group (M=5.15, SD=1.92). The group differences in positive emotion ratings during remembering of

eating events was statistically significant $F(1,58) = 5.72$, $p = 0.02$, and represented a medium-sized effect, $d = 0.62$.

Intensity of Emotions. Participants in the typical eating group rated experiencing their emotions more intensely during recall of general events (M=4.96, SD=1.22), compared to those in the LOC eating group (M=4.12, SD=1.76). This difference was statistically significant, $F(1,58) = 4.41$, $p = 0.04$, and the magnitude of the difference represented a medium-sized effect, $d = 0.54$. On average, participants in the typical eating group also rated their emotions more intensely when remembering eating events (M=3.96, SD=1.79), compared to those in the LOC eating group (M=3.64, SD=1.67). However, this difference was not statistically significant $F(1,58) = 0.53$, $p = 0.47$, and the magnitude of the difference represented a small effect, $d = 0.19$.

Strength of Emotions. On average, participants in the typical eating group rated their emotions more strongly when remembering general events (M=5.04, SD=1.22), compared to participants in the LOC eating group (M=4.61, SD=1.66). Although this difference was not statistically significant, $F(1,58) = 1.26$, $p = 0.27$, it did represent a small effect size, $d = 0.29$. For the eating events recalled, group differences on strength of emotion ratings were not observed. That is, participants in the typical eating group rated their strength of emotions experienced during eating event recall (M=4.22, SD=1.67), similarly to participants in the LOC eating group (M=4.27, SD=1.65), which was not significantly different, $F(1,58) = 0.01$, $p = 0.91$, $d = 0.03$.

Negative Emotions. On average, participants in the typical eating group rated their emotions less negatively when remembering general events (*M*=2.52, SD=1.91), compared to those in the LOC eating group (*M*=3.09, SD=2.11). This difference was not statistically significant $F(1,58) = 1.19$, $p = 0.28$, but, it did represent a small effect, $d = 0.28$. Participants in the typical eating group also rated their emotions less negatively when remembering eating events (*M*=1.41, SD=1.08), compared to those in the LOC eating group (*M*=2.06, SD=1.97). While this difference was not statistically significant $F(1,58) = 2.38$, $p = 0.13$, it did represent a small to medium-sized effect, $d = 0.40$.

Reliving. Participants in the typical eating group rated a greater sense of reliving during recall of general events (*M*=4.96, SD=1.68), compared to those in the LOC eating group (*M*=4.67, SD=1.88), but this difference was not statistically significant, $F(1,58) = 0.41$, $p = 0.53$, $d = 0.16$. During remembering of eating events, participants in the typical eating group also endorsed a greater sense of reliving (*M*=4.67, SD=1.78), compared to participants in the LOC eating group (*M*=4.21, SD=1.80), which was not statistically significant $F(1,58) = 0.96$, $p = 0.33$, but it did represent a small-sized effect, $d = 0.26$.

Visual Imagery. On average, participants in the typical eating group rated similar levels of visual imagery when recalling general events (*M*=6.07, SD=0.87), compared to LOC eating participants (*M*=6.03, SD=0.95), and these group differences were not statistically significantly, $F(1,58) = 0.03$, $p = 0.86$, $d = 0.04$. Similar patterns were observed for eating events, i.e., average visual imagery ratings during recall of eating events were comparable for participants in the typical eating group (*M*=5.41, SD=1.58) and

participants in the LOC eating group (*M*=5.42, SD=1.20), and were not significantly different, $F(1,58) = 0.002$, $p = 0.96$, $d = 0.01$.

Auditory Imagery. On average, participants in the typical eating group rated more vivid auditory imagery when remembering general events (*M*=4.89, SD=1.50), compared to those in the LOC eating group (*M*=4.64, SD=1.64), but this difference was not statistically significant $F(1,58) = 0.38$, $p = 0.54$, $d = 0.16$. Participants in the typical eating group also rated auditory imagery more vividly during recall for eating events (*M*=4.11, SD=1.81), compared to those in the LOC eating group (*M*=3.97, SD=1.78), but again, the difference between group ratings was not statistically significant, $F(1,58) = 0.09$, $p = 0.76$, $d = 0.08$.

Rehearsal. On average, participants in the typical eating group rated less frequent rehearsal for general events remembered (*M*=4.22, SD=1.42), compared to those in the LOC eating group (*M*=4.48, SD=1.00). While this difference was not statistically significant $F(1,58) = 0.70$, $p = 0.41$, it did represent a small effect, $d = 0.21$. Participants in the typical eating group rated more frequent rehearsal for eating events (*M*=3.70, SD=1.14), compared to those in the LOC eating group (*M*=3.58, SD=1.20), but these differences were not statistically significant $F(1,58) = 0.18$, $p = 0.68$, $d = 0.10$.

Table 9 Characteristics of Autobiographical Memories Recalled for General and Eating Events, by Group

Event Type	General Event Memory						Eating Event Memory						Group by Event
Group	Typical Eating Group (n=27)		LOC Eating Group (n=33)		Tests of Between Group Differences		Typical Eating Group (n=27)		LOC Eating Group (n=33)		Tests of Between Group Differences		Tests of Inter-action
Memory Characteristic Item from the AMQ	M	SD	M	SD	*F ratio*	*p*	M	SD	M	SD	*F ratio*	*p*	*p*
Rehearsal I have thought or talked about this event	4.22	1.42	4.48	1.00	0.70	.41	3.70	1.14	3.58	1.20	0.18	.68	.36
Visual Imagery I can see it in my mind	6.07	0.87	6.03	0.95	0.03	.86	5.41	1.58	5.42	1.20	.002	.96	.87
Auditory Imagery I can hear it in my mind	4.89	1.50	4.64	1.64	0.38	.54	4.11	1.81	3.97	1.78	0.09	.76	.83
Sense of Reliving I feel as though I am reliving it again	4.96	1.68	4.67	1.88	0.41	.53	4.67	1.78	4.21	1.80	0.96	.33	.74
Emotions I feel the emotions as strongly as I did then	5.04	1.22	4.61	1.66	1.26	.27	4.22	1.67	4.27	1.65	0.01	.91	.34
The emotions are extremely positive	5.33	1.86	4.27	2.00	4.43	.04*	6.15	1.10	5.15	1.92	5.72	.02*	.92
The emotions are extremely negative	2.52	1.91	3.09	2.11	1.19	.28	1.41	1.08	2.06	1.97	2.38	.13	.90
The emotions I feel are intense	4.96	1.22	4.12	1.76	4.41	.04*	3.96	1.79	3.64	1.67	0.53	.47	.25

Note: AMQ=Autobiographical Memory Questionnaire (Rubin, Schrauf, Greenberg, 2003); LOC=Loss of Control; M=Mean; SD=Standard Deviation. $^{*}p < .05$, $^{+}p < .01$, $^{++}p < .001$. *F ratio* = value of *F* ratio, based on (1,58) degrees of freedom. One-way between-subjects ANOVAs were conducted to assess group differences between the typical and LOC eating groups, on mean ratings of memory vividness, across eight domains, using the AMQ measure developed by Rubin, Schrauf, & Greenberg (2003). Group mean differences were assessed for the general event remembered and the *novel* eating event remembered.

3.7 Results Aim 3

Hierarchical regression models were used to estimate the potential contribution of high fat, high sugar dietary intake, and body mass index (BMI), in the prediction of visual memory and auditory memory on the WMS-IV. Hierarchical regression models were also used to estimate the potential contribution of high fat, high sugar dietary intake, and body mass index (BMI), in the prediction of autobiographical memory ratings for visual imagery, auditory imagery, and reliving on the AMQ.

In the hierarchical regression models described below, the dummy-coded grouping variable (i.e., 0=typical eating group; 1=LOC eating group) was entered at Step 1, dietary fat and sugar intake (DFS) was entered at Step 2, and body mass index was entered at Step 3. Recall that earlier sample BMI data violated the assumptions of normality necessary for use with standard parametric tests of hypotheses (Field, 2012). As such, sample BMI data were log transformed during the diagnostic data cleaning process. In addition, a more robust form of regression was selected for all analyses in Aim 3, because it does not rely on assumptions of normality and homoscedasticity (Field, 2012, p. 350-354). Specifically, robust regression analyses generate 95% bias corrected and accelerated confidence intervals and standard errors, based on 1000 bootstrap samples, which are presented in the data summary tables and narratives below. (Field, 2012, p. 350-354).

Overall, results indicated neither high fat, high sugar dietary intake nor BMI improved prediction of visual or auditory memory performance, as measured by the WMS-IV. Additionally, neither high fat, high sugar dietary intake nor BMI improved prediction of visual imagery, auditory imagery, or reliving ratings for general events remembered, as measured by the AMQ. Results from the regression analyses for memory performance are described first, by domain, followed by results from the regression analyses for autobiographical memory ratings, organized by memory characteristic.

3.7.1 Memory Performance

Visual memory performance. At Step 1, there was a significant effect of group membership on visual memory performance [$F(1,58) = 6.77$, $p = .01$, $R^2 = .11$], with 11% of the variance in visual memory performance explained by group membership. At Step 2, the inclusion of dietary fat and sugar intake did not significantly improve model predictions for visual memory performance, [$F(2,57) = 3.99$, $p = .02$, $R^2 = .12$, $\Delta R^2 = .02$, $p = .28$]. At Step 3, the inclusion of the body mass index variable, also did not significantly improve model predictions for visual memory performance, [$F(3,56) = 2.71$, $p = .05$, $R^2 = .13$, $\Delta R^2 = .004$, $p = .62$]. These findings are summarized in Table 10.

Table 10 Linear Model of Predictors of Visual Memory Performance, with Robust Estimates Based on 1000 Bootstrap Samples

Outcome →	Visual Memory Performance (Index Score)				
	b (95% bias corrected and accelerated CI)	SE	β	*t*	*p*
Step 1					
Constant	113.37 (109.22; 117.52)	2.07		54.65	<.001^{++}
Grouping variable	-7.28 (-12.88, -1.68)	2.80	-.32	-2.60	.01*
Step 2					
Constant	106.56 (93.38; 119.74)	6.58		16.19	<.001^{++}
Grouping variable	-7.22 (-12.81; -1.63)	2.79	-.32	-2.58	.01*
Dietary Fat & Sugar Intake	0.07 (-0.06, 0.20)	0.06	.14	1.09	.28
Step 3					
Constant	90.45 (25.26, 155.63)	32.54		2.78	<.01^{+}
Grouping variable	-7.55 (-13.34, -1.77)	2.89	-.34	-2.62	.01*
Dietary Fat & Sugar Intake	0.07 (-0.06, 0.20)	0.06	.14	1.08	.28
BMI (log transformed)	-11.95 (-35.39, 59.30)	23.64	-.07	0.51	.62
R^2 = .11 for Step 1; ΔR^2 = .02, (p = .28) for Step 2; ΔR^2 < .01 (p=.62) for Step 3					

Note: Robust regression analyses were used to produce 95% bias corrected and accelerated confidence intervals, which are reported in parentheses. Confidence intervals and standard errors were based on 1000 bootstrap samples. The dependent variable is Visual Memory Index, an index score derived from four subtests of the Wechsler Memory Scale-IV. Grouping variable: 0=typical eating group; 1=loss of control (LOC) eating group; BMI=body mass index; *b*=unstandardized beta coefficients, SE=standard error of coefficients, β=standardized beta coefficients, *t*=t-value, *p*=p-value; $^{*}p < .05$, $^{+}p < .01$, $^{++}p < .001$.

Auditory memory performance. At Step 1, there was no significant effect of group membership on auditory memory performance [$F(1,58) = 2.56$, p =.12, R^2 = .04]. At Step 2, the inclusion of dietary fat and sugar intake did not significantly improve model

predictions for auditory memory performance, [$F(2,57) = 2.36$, $p = .10$, $R^2 = .08$, $\Delta R^2 = .03$, $p = .15$]. At Step 3, the inclusion of body mass index (log transformed), also did not significantly improve model predictions for auditory memory performance, [$F(3,56) = 1.65$, $p = .19$, $R^2 = .08$, $\Delta R^2 = .005$, $p = .59$]. These findings are summarized in Table 11.

Table 11 Linear Model of Predictors of Auditory Memory Performance, with Robust Estimates Based on 1000 Bootstrap Samples

Outcome →	Auditory Memory Performance (Index Score)				
	b (95% bias corrected and accelerated CI)	SE	β	*t*	*p*
Step 1					
Constant	113.82 (109.60; 118.03)	2.11		54.07	<.001++
Grouping variable	-4.54 (-10.22, 1.14)	2.84	-.21	-1.60	.12
Step 2					
Constant	104.68 (91.41; 117.95)	6.63		15.80	<.001++
Grouping variable	-4.46 (-10.09; 1.17)	2.81	-.20	-1.59	.12
Dietary Fat & Sugar Intake	0.09 (-0.04, 0.22)	0.06	.19	1.45	.15
Step 3					
Constant	87.32 (21.71, 152.93)	32.75		2.67	.01*
Grouping variable	-4.82 (-10.64, 1.00)	2.91	-.22	-1.66	.10
Dietary Fat & Sugar Intake	0.09 (-0.04, 0.22)	0.07	.19	1.44	.16
BMI (log transformed)	12.88 (-34.77, 60.53)	23.79	.07	0.54	.59
$R^2 = .04$ for Step 1; $\Delta R^2 = .03$ ($p = .15$) for Step 2; $\Delta R^2 < .01$ ($p = .59$) for Step 3					

Note: Robust regression analyses were used to produce 95% bias corrected and accelerated confidence intervals, which are reported in parentheses. Confidence intervals and standard errors were based on 1000 bootstrap samples. The dependent variable is Auditory Memory Index, an index score derived from four subtests of the Wechsler Memory Scale-IV. Grouping variable: 0=typical eating group; 1=loss of

control (LOC) eating group; BMI=body mass index; *b*=unstandardized beta coefficients, SE=standard error of coefficients, β=standardized beta coefficients, *t*=t-value, *p*=p-value; $^{*}p < .05$, $^{+}p < .01$, $^{++}p < .001$.

3.7.2 Autobiographical Memory Ratings

Finally, hierarchical regression models were used to estimate three memory vividness ratings from the general AMQ (i.e., visual imagery, auditory imagery, and a sense of reliving), which were are all hypothesized to be hippocampally-mediated. The same procedures described above were used to fit each of the three models, i.e., dummy-coded grouping variable (i.e., 0=typical eating group; 1=LOC eating group) was entered at Step 1, dietary fat and sugar intake (DFS) was entered at Step 2, and body mass index (log) was entered at Step 3, using the robust regression analyses.

Visual Imagery. At Step 1, there was no significant effect of group membership on visual imagery ratings [$F(1,57) = 0.06$, $p = .82$, $R^2 = .001$]. At Step 2, the inclusion of dietary fat and sugar intake did not significantly improve model predictions for visual imagery ratings, [$F(1,56) = 0.91$, $p = .41$, $R^2 = .03$, $\Delta R^2 = .03$, $p = .19$]. At Step 3, the inclusion of body mass index (log transformed), also did not significantly improve model predictions for visual imagery, [$F(1,55) = 1.59$, $p = .20$, $R^2 = .08$, $\Delta R^2 = .05$, $p = .09$]. These results are summarized in Table 12.

Table 12 Linear Model of Predictors of Visual Imagery Ratings, with Robust Estimates Based on 1000 Bootstrap Samples

Outcome →	Visual Imagery Ratings				
	b (95% bias corrected and accelerated CI)	SE	β	*t*	*p*
Step 1					
Constant	6.07 (5.75, 6.39)	0.16		37.99	<.001^{++}
Grouping variable	0.05 (-.38, .49)	0.22	.03	.24	.82
Step 2					
Constant	5.43 (4.41; 6.45)	0.51		10.67	<.001^{++}
Grouping variable	0.05 (-.38, .48)	0.22	.03	.24	.81
Dietary Fat & Sugar Intake	0.01 (-0.01, 0.02)	0.01	.17	1.33	.19
Step 3					
Constant	1.30 (-3.66, 6.26)	2.48		.53	.60
Grouping variable	-0.04 (-.48, .40)	0.22	-.02	-.18	.86
Dietary Fat & Sugar Intake	0.01 (-.01, .02)	0.01	.18	1.38	.17
BMI (log transformed)	3.05 (-.54, 6.64)	1.79	.23	1.71	.09
R^2 = .001 for Step 1; ΔR^2 = .03 (p = .15) for Step 2; ΔR^2 =.05 (p=.09) for Step 3					

Note: Robust regression analyses were used to produce 95% bias corrected and accelerated confidence intervals, which are reported in parentheses. Confidence intervals and standard errors were based on 1000 bootstrap samples. Dependent variable is visual imagery rating during recall of a general event on the Autobiographical Memory Questionnaire (AMQ) (Rubin, Schrauf, Greenberg, 2003); Grouping variable: 0=typical eating group; 1=loss of control (LOC) eating group; BMI=body mass index; *b*=unstandardized beta coefficients, SE=standard error of coefficients, β=standardized beta coefficients, *t*=t-value, *p*=p-value; *p < .05, ^{+}p < .01, ^{++}p < .001.

Auditory Imagery. At Step 1, there was no significant effect of group membership on auditory imagery ratings [$F(1,57) = 0.24$, p =.63, R^2 = .004]. At Step 2, the inclusion of dietary fat and sugar intake did not significantly improve model predictions for auditory

imagery ratings, [$F(1,56) = 1.17$, $p = .32$, $R^2 = .04$, $\Delta R^2 = .04$, $p = .15$]. At Step 3, the inclusion of body mass index (log transformed), also did not significantly improve model predictions for auditory imagery, [$F(1,55) = 1.52$, $p = .21$, $R^2 = .08$, $\Delta R^2 = .04$, $p = .13$]. These results are summarized in Table 13.

Table 13 Linear Model of Predictors of Auditory Imagery Ratings, with Robust Estimates Based on 1000 Bootstrap Samples

Outcome →	Auditory Imagery Ratings				
	b (95% bias corrected and accelerated CI)	SE	β	*t*	*p*
Step 1					
Constant	4.89 (4.28, 5.50)	0.30		16.12	<.001++
Grouping variable	-.20 (-1.03, .62)	0.41	-.07	-.49	.63
Step 2					
Constant	3.56 (1.63; 5.49)	0.96		3.70	<.001++
Grouping variable	-.20 (-1.02, .62)	0.41	-.06	-.49	.63
Dietary Fat & Sugar Intake	-0.01 (-0.01, 0.03)	0.01	.19	1.45	.15
Step 3					
Constant	-3.45 (-12.87, 5.98)	4.71		-.73	.47
Grouping variable	-.35 (-1.19, .48)	0.42	-.11	-.85	.40
Dietary Fat & Sugar Intake	-0.01 (-.01, .03)	0.01	.19	1.50	.14
BMI (log transformed)	5.18 (-1.64, 12.00)	3.40	.20	1.52	.13
$R^2 = .004$ for Step 1; $\Delta R^2 = .04$ ($p = .15$) for Step 2; $\Delta R^2 = .04$ ($p = .13$) for Step 3					

Note: Robust regression analyses were used to produce 95% bias corrected and accelerated confidence intervals, which are reported in parentheses. Confidence intervals and standard errors were based on 1000 bootstrap samples; Dependent variable is auditory imagery rating during recall of a general event on the Autobiographical Memory Questionnaire (AMQ) (Rubin, Schrauf, Greenberg, 2003); Grouping variable: 0=typical eating group; 1=loss of control (LOC) eating group; BMI=body mass index; *b*=unstandardized

beta coefficients, SE=standard error of coefficients, β=standardized beta coefficients, *t*=t-value, *p*=p-value; *$p < .05$, $^{+}p < .01$, $^{++}p < .001$.

Reliving. At Step 1, there was no significant effect of group membership on reliving ratings [$F(1,57) = 0.16$, $p = .69$, $R^2 = .003$]. At Step 2, the inclusion of dietary fat and sugar intake did not significantly improve model predictions for reliving ratings, [$F(1,56) = 1.39$, $p = .26$, $R^2 = .05$, $\Delta R^2 = .05$, $p = .11$]. At Step 3, the inclusion of body mass index (log transformed), also did not significantly improve model predictions for reliving ratings, [$F(1,55) = 1.18$, $p = .33$, $R^2 = .06$, $\Delta R^2 = .01$, $p = .39$]. These results are summarized in Table 14.

Table 14 Linear Model of Predictors of Reliving Ratings, with Robust Estimates Based on 1000 Bootstrap Samples

Outcome →	Reliving Ratings				
	b (95% bias corrected and accelerated CI)	SE	β	*t*	*p*
Step 1					
Constant	4.96 (4.29, 5.63)	0.34		14.83	<.001^{++}
Grouping variable	-.18 (-1.09, .73)	0.46	-.05	-.40	.69
Step 2					
Constant	3.34 (1.22, 5.46)	1.06		3.15	<.01^{+}
Grouping variable	-.18 (-1.08, .72)	0.45	-.05	-.40	.69
Dietary Fat & Sugar Intake	0.01 (-0.00, 0.04)	0.01	.21	1.62	.11
Step 3					
Constant	-1.13 (-11.64, 9.37)	5.24		-.22	.83
Grouping variable	-.28 (-1.21, .65)	0.46	-.08	-.60	.55
Dietary Fat & Sugar Intake	0.02 (-.00, .04)	0.01	.21	1.63	.11
BMI (log transformed)	3.30 (-4.30, 10.90)	3.79	.12	.87	.39
R^2 = .003 for Step 1; ΔR^2 = .05 (p = .11) for Step 2; ΔR^2 < .01 (p=.39) for Step 3					

Note: Robust regression analyses were used to produce 95% bias corrected and accelerated confidence intervals, which are reported in parentheses. Confidence intervals and standard errors were based on 1000 bootstrap samples; Dependent variable is reliving rating during recall of a general event on the Autobiographical Memory Questionnaire (AMQ) (Rubin, Schrauf, Greenberg, 2003),Grouping variable: 0=typical eating group; 1=loss of control (LOC) eating group; BMI=body mass index; *b*=unstandardized beta coefficients, SE=standard error of coefficients, β=standardized beta coefficients, *t*=t-value, *p*=p-value; *p < .05, ^{+}p < .01, ^{++}p < .001.

4. Discussion

This study sought to characterize memory functioning among a sample of young adult women with loss of control (LOC) and typical eating, by examining two aspects of hippocampal-dependent memory functioning, as captured via performance on a standardized neuropsychological measure of memory, the Wechsler Memory Scale-IV, and through use of a well-validated autobiographical memory task, the Autobiographical Memory Questionnaire (AMQ). Additionally, this study sought to develop and test a novel autobiographical memory probe designed to elicit vivid recall of an eating event, through a modified AMQ (i.e., AMQ-Eating). Finally, this study aimed to capture additional psychological and behavioral features associated with loss of control eating, in an effort to identify relevant co-variates hypothesized to feed into the "vicious cycle" model.

Results of the present study revealed a significant difference between groups in the visual memory domain. Specifically, participants in the LOC eating group performed significantly below those in the typical eating group on the visual memory index and the designs subtest. The designs subtest task requires learning (encoding) items-in-context and recalling this visio-spatial information both immediately and after a 20-30 minute delay. Notably, participants in the LOC eating group functioned significantly below the typical eating group on both the immediate and delayed conditions of this task. Existing literature on hippocampal-mediated memory functioning suggests a central role of the hippocampus and hippocampal formation in encoding items-in-context (Dickerson &

Eichenbaum, 2010), as well as a central role of the hippocampus in spatial processing and memory (Ezzati et al., 2016). These results provide preliminary support for our hypotheses that those in the LOC eating group would demonstrate lower scores on hippocampal-dependent memory tasks, relative to those in the typical eating group.

Results of the present study also revealed a significant difference within the auditory memory domain, as demonstrated by significantly lower scores on the word pairs tasks. For this task, participants learned 14-word pairs over the course of four learning trials, in which four pairs were semantically related and ten pairs were not. In the present study, participants in the LOC eating group had significantly lower scores on the immediate recall condition of this task, but not on the delayed condition. Existing research provides support for the role of the hippocampus in verbal learning, especially for tasks that involve associative learning (Aslaksen et al., 2018). Given the word pair tasks' unique structure of four learning trials (with feedback), it also provides a unique opportunity to understand participant's associative learning over time, and to potentially differentiate between difficulties in memory functioning at the levels of encoding, consolidation, storage, and retrieval. Because participants in the LOC eating group in our study did not significantly differ from participants in the typical eating group on the delayed recall condition of the word pairs task, it may be that the LOC participants learned the word pairs at a slower rate, but eventually caught up to the participants in the typical eating group, as evidenced by the similar delayed recall scores across groups. Future studies should examine the trial-by-trial performance to better understand the

subprocesses of learning and memory involved. A recent study by Tayim and colleagues (2016) provides a model for such an examination. Specifically, the researchers examined neurocognitive processes that contributed to memory improvement following traumatic brain injury by employing an item-specific analytic approach to data collected from the California Verbal Learning Test – 2nd edition (CVLT-II; Delis, Kramer, Kaplan, & Ober, 2000), which was administered longitudinally. By utilizing this approach, they were able to identify deficits (and improvements) at the memory subsystem level (i.e., encoding, consolidation, and retrieval) and characterize where the breakdown in memory (skills) was occurring (Tayim et al., 2016). Notably, the CVLT-II is a parallel task to the word pairs subtest on the WMS-IV, and thus provides a roadmap for such future analyses.

Future studies should also examine potential deficits in the executive functioning system on contribution to deficits in memory encoding, storage, and retrieval, as this may help illuminate potential mechanisms to aberrant eating behavior. Previous research has established a link between adults with obesity, binge, and/or LOC eating and greater difficulties with executive functioning (Duchesne et al., 2010; Gettens & Gorin, 2017; Rouel et al., 2016). In the present study, we used the WASI-II to measure general cognitive ability, which is frequently employed by researchers as an efficient estimate of general intelligence. This measure, while useful in perhaps characterizing an individual's capacity to learn within a Westernized educational system, does little to help illuminate the potential impact of differences in sustained attention, processing speed, and working memory, which are known to impact scores on more comprehensive measures of IQ, as

well as the WMS. In the present study, we found no significant differences between groups on the full-scale IQ, with similar mean scores on the two-subtest estimate of intelligence (i.e., *M*=119.88 in LOC; *M*=115.16 in typical eating). As such, the differences observed in the present study between groups on performance on the designs subtest, visual memory index, and word pairs subtest may reflect actual memory deficits, or may reflect underlying deficits in visual-spatial processing, visual scanning, and sustained attention, which we did not measure. Future studies should employ longer versions of IQ testing, or add supplemental subtests, such as D-KEFS Trails, to help characterize any underlying differences in these cognitive skills/abilities.

While the findings from the WMS-IV provided preliminary support for our hypotheses, results from the AMQ were counter to our predictions. Specifically, no significant differences were found between groups on ratings of visual imagery, auditory imagery, or a sense of reliving during either the general or eating event autobiographical memory tasks. An interesting other finding was the significant group differences on ratings of emotion during recall. Specifically, participants in the typical eating group rated both types of autobiographical memories as significantly more positive than participants in the LOC eating group. In addition, participants in the typical eating group rated feeling their emotions more intensely during recall of general events, compared to participants in the LOC eating group. While most of the observed group differences were non-significant, the emotion-based ratings do provide some potential trends to examine in future studies. For example, for the general memory, those in the LOC eating group rated

less strength of emotion, less positive emotion, more negative emotion, and less intensity of emotion, compared to the typical eating group. However, for the eating event memory, participants in the LOC group rated strength of emotion more similarly to the typical eating group, suggesting a possible interaction for this construct. Future studies would be strengthened by inclusion of additional conditions, randomized presentation of event type, and inclusion of several cue words to illicit numerous general and eating-specific memories. Qualitative responses from the present study (i.e., memories recorded for the general and eating conditions) could also be analyzed qualitatively to identify themes and inform potential future cue words, which may be salient to those with LOC eating. In light of the significant effects observed in the present study on emotional ratings, future research should consider probing how emotions among those with LOC eating may be affecting memory generally and memory for eating-specific episodes. Given the established role of emotion on episodic memories (Talarico et al., 2004), and the complex interplay between emotion, motivation, and memory encoding, consolidation, and retrieval (Murty & Adcock; Murty & Dickerson, 2017; Murty et al., 2011), future studies should expand upon the results here by examining these constructs among those with LOC eating.

Findings from the current study revealed there were no group differences in dietary intake of foods high in fat and sugar, as measured by the DFS-SQ modified. This was despite demonstrated high reliability of the DFS-SQ modified (i.e., internal consistency: α=.83) among the current sample (*N*=66). Additionally, the dietary intake of

high fat, high sugar foods did not emerge as a significant predictor of hippocampal-dependent memory functioning, as assess via the WMS-IV and memory vividness ratings on the autobiographical memory questionnaire (AMQ). While it is possible that finding in human studies do not replicate findings reported using animal models, it is also conceivable that our choice of methodology was not sensitive enough to capture this relationship. We employed a modified DFS, which is a brief measure of dietary intake focusing solely on frequency – not quantity – of intake. As such, we may have failed to capture a key parameter of high fat, high sugar dietary consumption. Our results may reflect actual similarities in DFS intake across the groups, reflect measurement error (e.g., measure not sensitive enough to capture differences), or reflect some other variable that was not captured in the present study. Our study was limited by the fact that we did not measure the overall dietary intake of our sample. Thus, future studies should include more comprehensive measures of dietary intake, such as the Block Food Frequency Questionnaire (Block, Woods, Potosky, & Clifford, 1990), to explore the specific and relative contribution of dietary fat, sugar, protein, fruits, vegetables, and carbohydrates on memory outcomes associated with LOC eating. Although the Block FFQ is much longer than the DFS, it has been shown to accurately approximate the actual intake of participants enrolled in 8-24 week behavioral weight loss interventions (i.e., adults with obesity). The study found the FFQ to be an accurate estimate of actual food intake, as verified by daily food logs and modeling caloric intake with observed weight losses over

the intervention period, demonstrating the utility of the FFQ measure (Middleton-Ross et al., 2011).

In terms of clinical implications, our findings did support the current emphasis on subjective binge eating episodes as being critical to understanding the phenomenology of binge eating disorder as, according to these findings, the groups did not objectively differ in the amount of high fat, high sugar foods consumed. The clinical group may be distinguished, in part, by greater distress and impairment following the consumption of a similar type and quantity of food as typical eaters. Further, because our questionnaire solely focused on dietary intake of high fat, high sugar foods, and not the full range of eating habits (e.g., fruit and veggie intake), demand characteristics may have also played a role in our findings. Given higher levels of weight stigma, shame, and guilt associated with eating (i.e., excess guilt associated with both binge and non-binge episodes) reported among binge eating disorder and LOC participants generally, individuals in our LOC sample may have been less inclined to endorse higher levels of DFS intake. One solution to overcome these issues in future research would be to employ more comprehensive measures of eating behaviors, instead of measures solely focused on DFS intake, such as the Block Food Frequency Questionnaire (Block, Woods, Potosky, & Clifford, 1990).

Given the central role of dietary intake in disorders of eating, future studies should also consider developing measures specific to the behavioral features and dietary intake patterns among vulnerable populations, such as those with LOC eating. For

example, Kalantar-Zadeh and colleagues (2011) modified a food frequency questionnaire (FFQ) for patients with chronic conditions that require dialysis, to screen for intake patterns that increased risk for medical complications. For binge or LOC eating, a measure could potentially be developed to identify types of foods or patterns of eating that are most closely associated with distress, which could be used to monitor treatment goals, screen for potential clinical cases, or identify different levels of distress within clinical samples.

In addition to the limitations mentioned herein, this study was also limited by the heterogenous nature of our LOC group, the unequal sample sizes between groups, and a relatively small sample size. This study was strengthened by the use of several measures of hippocampal-dependent memory, including standardized behavioral performance measures and the autobiographical memory questionnaire. The study was also strengthened by the use of the clinical diagnostic interview (i.e., Eating Disorder Examination [EDE]), which was administered by a skilled doctoral-level clinician researcher. The EDE interview enabled us to measure frequency, amount, distress, impairment, and behavioral features associated with both objective and subjective binge eating episodes, and allowed us to fully characterize the LOC eating experiences within and between groups.

5. Conclusion

Binge and loss of control eating are clinically significant symptoms that constitute syndromes characterized by a repetitive pattern of maladaptive and subjectively uncontrollable binge or loss of control eating. The hippocampal-dependent memory system appears to play an important role in the regulation of food intake in studies employing animal models. These findings may extend to humans, but research has been limited, particularly among vulnerable populations, such as those with eating pathology. Existing research suggests excessive energy intake and/or consumption of high fat, high sugar foods impairs performance on hippocampal-dependent learning and memory tasks, which in turn, is associated with excessive food intake and motivated high fat, high sugar food seeking behaviors. This vicious cycle may be particularly relevant to individuals with binge or loss of control eating, as persistent binge/LOC eating episodes are associated with several food intake patterns and individual difference factors that may strengthen the observed associations.

This study sought to examine the potential role of learning and memory in the development and maintenance of binge and LOC eating behaviors among a community sample of (*N*=66) young adult women, who either endorsed binge or LOC eating (*n*=35) or reported typical eating behaviors (n=31). Participants completed clinical diagnostic interviews, standardized neuropsychological measures of cognitive ability and hippocampal-dependent memory, self-report measures of eating behaviors, dietary intake of highly palatable foods, depressive symptoms, psychological functioning,

autobiographical memory (general and eating event memories), and current mood state and hunger. Results revealed that participants who endorsed binge or loss of control eating performed worse on several measures of hippocampal-dependent memory, including measures of visuo-spatial learning and memory, verbal learning and memory, immediate memory, and delayed memory. Findings from the autobiographical memory tasks did not support our hypothesis, but provided insight into other factors, such as the potential role of emotion, in the study of autobiographical memory and LOC eating. The current study also did not find evidence to support the predictive utility of high fat, high sugar dietary intake on differences in hippocampal functioning, at least as measured in the current sample. Future research should continue to characterize and probe hippocampal-dependent memory among those with LOC eating, and explore possible differences in the visuo-spatial learning and memory system. Future research should also seek to overcome some of the methodological challenges in measuring dietary intake of high fat, high sugar foods, to further our understanding of the vicious cycle, especially among vulnerable populations.

Appendix A: Prescreening Consent

Thank you for your interest in the thinking styles and eating behavior research study being conducted at Duke University under the supervision of Dr. Nancy Zucker. I would like to ask you some questions that will help us determine if you qualify to be in this research study. After we talk on the phone, I will also ask you to participate in a screening survey online. The total time this will take is about 5 minutes on the phone and about 10-15 minutes to complete the online survey. The questions I will ask you on the phone and in the online survey will involve you giving information about your weight, your psychological health, and your eating habits. Answering these questions is voluntary. You are under no obligation to answer them, and not answering them will have no effect on your health care at Duke or your grades if you are a student at Duke. Not answering the questions, however, means that you will not be eligible to participate in this research study. If at any time during this prescreening you would like to stop and not participate, or if you have any questions, just let me know.

Here's some information about the confidentiality of the information you provide us today. After we talk on the phone, you will complete an online survey. The information you provide us through the online survey will be attained through Qualtrics.com, a secure database system used by researchers at Duke. Qualtrics uses many levels of security to ensure that the data remain private and secure. They employ a third-party firm to conduct daily audits of security and the data are behind the latest firewall and intrusion

prevention technology. After transmission of the data, only Dr. Zucker and her research team will be able to access the data, as the database is password protected. Only Dr. Zucker and her research team will have access to this password. Once the data are obtained, they will be downloaded and stored in a secure main database on a password-protected hard drive. This database will only be accessible by individuals directly involved in the execution of the research. No unauthorized person will have access to the database or to the names of participants. If you have any questions or concerns, please feel free to contact Dr. Zucker at (919) 668-2281 or nancy.zucker@dm.duke.edu . Do you agree to proceed with the screening questions?

- o I agree
- o I do not agree

Appendix B: Online Screening Measures

Eating Pathology Symptoms Inventory (EPSI)
Administered via Qualtrics

Instructions: Below is a list of experiences and problems that people sometimes have. Read each item to determine how well it describes your recent experiences. Then select the option that best describes how frequently each statement applied to you during the past four weeks, including today. Use this scale when answering:

Never	Rarely	Sometimes	Often	Very often
0	1	2	3	4

1. I did not like how clothes fit the shape of my body
2. I tried to exclude "unhealthy" foods from my diet
3. I ate when I was not hungry
4. People told me that I do not eat very much
5. I felt that I needed to exercise nearly every day
6. People would be surprised if they knew how little I ate
7. I used muscle building supplements
8. I pushed myself extremely hard when I exercised
9. I snacked throughout the evening without realizing it
10. I got full more easily than most people
11. I considered taking diuretics to lose weight
12. I tried on different outfits, because I did not like how I looked
13. I thought laxatives are a good way to lose weight

14. I thought that obese people lack self-control

15. I thought about taking steroids as a way to get more muscular

16. I used diet teas or cleansing teas to lose weight

17. I used diet pills

18. I did not like how my body looked

19. I ate until I was uncomfortably full

20. I felt that overweight people are lazy

21. I counted the calories of foods I ate

22. I planned my days around exercising

23. I thought my butt was too big

24. I did not like the size of my thighs

25. I wished the shape of my body was different

26. I was disgusted by the sight of an overweight person wearing tight clothes

27. I made myself vomit in order to lose weight

28. I did not notice how much I ate until after I had finished eating

29. I considered taking a muscle building supplement

30. I felt that overweight people are unattractive

31. I engaged in strenuous exercise at least five days per week

32. I thought my muscles were too small

33. I got full after eating what most people would consider a small amount of food

34. I was not satisfied with the size of my hips

35. I used protein supplements

36. People encouraged me to eat more

37. If someone offered me food, I felt that I could not resist eating it

38. I was disgusted by the sight of obese people

39. I stuffed myself with food to the point of feeling sick

40. I tried to avoid foods with high calorie content

41. I exercised to the point of exhaustion

42. I used diuretics in order to lose weight

43. I skipped two meals in a row

44. I ate as if I was on autopilot

45. I ate a very large amount of food in a short period of time (e.g., within 2 hours)

DSM-5 Self-Rated Level 1 Cross-Cutting Symptoms Measure – Adult

Instructions: The questions below ask about things that might have bothered you. For each question, circle the number that best described how much (or how often) you have been bothered by each problem during the **past TWO (2) WEEKS.**

During the **past TWO (2) WEEKS,** how much (or how often) have you been bothered by the following problems?	None Not at all	Slight Rare, less than a day or two	Mild Several days	Moderate More than half the days	Severe Nearly every day
Little interest or pleasure in doing things?	0	1	2	3	4
Feeling down, depressed, or hopeless?	0	1	2	3	4
Feeling more irritated, grouchy, or angry than usual?	0	1	2	3	4
Sleeping less than usual, but still have a lot of energy?	0	1	2	3	4
Starting lots more projects than usual or doing more risky things than usual?	0	1	2	3	4
Feeling nervous, anxious, frightened, worried, or on edge?	0	1	2	3	4
Feeling panic or being frightened?	0	1	2	3	4
Avoiding situations that make you anxious?	0	1	2	3	4
Hearing things other people couldn't hear, such as voices, even when no one was around?	0	1	2	3	4
Feeling that someone could hear your thoughts, or that you could hear what another person was thinking?	0	1	2	3	4
Problems with memory (e.g., learning new information) or with location (e.g., finding your way home)?	0	1	2	3	4
Unpleasant thoughts, urges, or images that repeatedly enter your mind?	0	1	2	3	4
Feeling driven to perform certain behaviors or mental acts over and over again?	0	1	2	3	4
Drinking at least 4 drinks of any kind of alcohol in a single day?	0	1	2	3	4
Smoking any cigarettes, a cigar, or pipe, or using snuff or chewing tobacco?	0	1	2	3	4
Using any of the following medicines ON YOUR OWN (that is, without a doctor's prescription), OR in greater amounts, OR longer than prescribed [e.g., painkillers (like Vicodin), stimulants (like Ritalin or Adderall), sedatives or tranquilizers (like sleeping pills or Valium), or drugs like marijuana, cocaine or crack, club drugs (like ecstasy), hallucinogens (like LSD), heroin, inhalants or solvents (like glue), or methamphetamine (like speed)]?	0	1	2	3	4

Items, by domain:	Scoring and inclusion criteria for each item:
Depression domain:	
Little interest or pleasure in doing things?	This item needs to have a score of 3 or less
Feeling down, depressed, or hopeless?	This item needs to have a score of 3 or less
Anger domain:	
Feeling more irritated, grouchy, or angry than usual?	This item needs to have a score of 3 or less
Mania domain:	
Sleeping less than usual, but still have a lot of energy?	This item needs to have a score of 3 or less
Starting lots more projects than usual or doing more risky things than usual?	This item needs to have a score of 3 or less
Anxiety domain:	
Feeling nervous, anxious, frightened, worried, or on edge?	This item needs to have a score of 3 or less
Feeling panic or being frightened?	This item needs to have a score of 3 or less
Avoiding situations that make you anxious?	This item needs to have a score of 3 or less
Psychosis domain:	
Hearing things other people couldn't hear, such as voices even when no one was around?	This item needs to have a score of 0
Feeling that someone could hear your thoughts, or that you could hear what another person was thinking?	This item needs to have a score of 0
Memory domain:	
Problems with memory (e.g., learning new information) or with location (e.g., finding your way home)?	This item needs to have a score of 0
Repetitive thoughts and behaviors domain:	
Unpleasant thoughts, urges, or images that repeatedly enter your mind?	This item needs to have a score of 1 or less
Feeling driven to perform certain behaviors or mental acts over and over again?	This item needs to have a score of 1 or less
Substance use domain:	
Drinking at least 4 drinks of any kind of alcohol in a single day?	This item needs to have a score of 2 or less
Smoking any cigarettes, a cigar, or pipe, or using snuff or chewing tobacco?	This item needs to have a score of 1 or less
Using any of the following medicines ON YOUR OWN (that is, without a doctor's prescription), OR in greater amounts, OR longer than prescribed [e.g., painkillers (like Vicodin), stimulants (like Ritalin or Adderall), sedatives or tranquilizers (like sleeping pills or Valium), or drugs like marijuana, cocaine or crack, club drugs (like ecstasy), hallucinogens (like LSD), heroin, inhalants or solvents (like glue), or methamphetamine (like speed)]?	This item needs to have a score of 0

Beck Depression Inventory-II

Administered via Qualtrics

Instructions: This questionnaire consists of 21 groups of statements. Please read each group of statements carefully, and then pick out the statement in each group that best describes the way you have been feeling during the past two weeks, including today. Select the number beside the statement you have picked. If several statements in the group seem to apply equally well, select the highest number for that group.

1. Sadness
    - 0 I do not feel sad.
    - 1 I feel sad much of the time.
    - 2 I am sad all the time.
    - 3 I am so sad or unhappy that I can't stand it.

2. Pessimism
    - 0 I am not discouraged about my future.
    - 1 I feel more discouraged about my future than I used to be.
    - 2 I do not expect things to work out for me.
    - 3 I feel my future is hopeless and will only get worse.

3. Past Failure
    - 0 I do not feel like a failure.
    - 1 I have failed more than I should have.
    - 2 As I look back, I see a lot of failures.
    - 3 I feel like I am a total failure as a person.

4. Loss of Pleasure
    - 0 I get as much pleasure as I ever did from the things I enjoy.
    - 1 I don't enjoy things as much as I used to.
    - 2 I get very little pleasure from the things I used to enjoy.
    - 3 I can't get any pleasure from the things I used to enjoy.

5. Guilty Feelings
    - 0 I don't feel particularly guilty.
    - 1 I feel guilty over many things I have done or should have done.
    - 2 I feel quite guilty most of the time.
    - 3 I feel guilty all of the time.

6. Punishment Feelings
    - 0 I don't feel I am being punished.
    - 1 I feel I may be punished.
    - 2 I expected to be punished.
    - 3 I feel I am being punished.

7. Self-Dislike
    0 I feel the same about myself as ever.
    1 I have lost confidence in myself.
    2 I am disappointed in myself.
    3 I dislike myself.

8. Self-Criticalness
    0 I don't criticize or blame myself more than usual.
    1 I am more critical of myself than I used to be.
    2 I criticize myself for all of my faults.
    3 I blame myself for everything bad that happens.

9. Suicidal Thoughts or Wishes
    0 I don't have any thoughts of killing myself.
    1 I have thoughts of killing myself, but I would not carry them out.
    2 I would like to kill myself.
    3 I would kill myself if I had the chance.

10. Crying
    0 I don't cry any more than I used to.
    1 I cry more than I used to.
    2 I cry over every little thing.
    3 I feel like crying, but I can't.

11. Agitation
    0 I am no more restless or wound up than usual.
    1 I feel more restless or wound up than usual.
    2 I am so restless or agitated that it's hard to stay still.
    3 I am so restless or agitated that I have to keep moving or doing something.

12. Loss of Interest
    0 I have not lost interest in other people or activities.
    1 I am less interested in other people or things than before.
    2 I have lost most of my interest in other people or things.
    3 It's hard to get interested in anything.

13. Indecisiveness
    0 I make decisions about as well as ever.
    1 I find it more difficult to make decisions than usual.
    2 I have much greater difficulty in making decisions than I used to.
    3 I have trouble making any decisions.

14. Worthlessness
    - 0 I do not feel I am worthless.
    - 1 I don't consider myself as worthwhile and useful as I used to.
    - 2 I feel more worthless as compared to other people.
    - 3 I feel utterly worthless.

15. Loss of Energy
    - 0 I have as much energy as ever.
    - 1 I have less energy than I used to have.
    - 2 I don't have enough energy to do very much.
    - 3 I don't have enough energy to do anything.

16. Changes in Sleeping Pattern
    - 0 I have not experienced any change in my sleeping pattern.
    - 1a I sleep somewhat more than usual.
    - 1b I sleep somewhat less than usual.
    - 2a I sleep a lot more than usual.
    - 2b I sleep a lot less than usual.
    - 3a I sleep most of the day.
    - 3b I wake up 1-2 hours early and can't get back to sleep.

17. Irritability
    - 0 I am no more irritable than usual.
    - 1 I am more irritable than usual.
    - 2 I am much more irritable than usual.
    - 3 I am irritable all the time.

18. Changes in Appetite
    - 0 I have not experienced any change in my appetite.
    - 1a My appetite is somewhat less than usual.
    - 1b My appetite is somewhat greater than usual.
    - 2a My appetite is much less than before.
    - 2b My appetite is much greater than usual.
    - 3a I have no appetite at all.
    - 3b I crave food all the time.

19. Concentration Difficulty
    - 0 I can concentrate as well as ever.
    - 1 I can't concentrate as well as usual.
    - 2 It's hard to keep my mind on anything for very long.
    - 3 I find I can't concentrate on anything.

20. Tiredness or Fatigue
    - 0 I am no more tired or fatigued than usual.

1 I get more tired or fatigued more easily than usual.
2 I am too tired or fatigued to do a lot of the things I used to do.
3 I am too tired or fatigued to do most of the things I used to do.

21. Loss of Interest in Sex
0 I have not noticed any recent change in my interest in sex.
1 I am less interested in sex than I used to be.
2 I am much less interested in sex now.
3 I have lost interest in sex completely.

Demographic and Eligibility Questions

Administered via Qualtrics

1. Please describe your ethnicity and/or race.

2. To which ethnic or racial group(s) do you identify? (select **all** that apply)
   - o American Indian or Alaska Native (1)
   - o Asian (2)
   - o Black or African American (3)
   - o Hispanic or Latino or Spanish origin (4)
   - o Multiracial (5)
   - o Native Hawaiian or Other Pacific Islander (6)
   - o White (7)
   - o Other, please specify (8) _____
   - o I prefer not to categorize my ethnicity and/or race (9)

3. What is your height? (Please give your best estimate in feet and inches, e.g., 5ft, 4in)

4. What is your weight at present? (Please give your best estimate in pounds).

5. Do you have a history of an eating disorder? Yes No

   a. *Display This Question if YES*: Please describe your history with an eating disorder (e.g., when did the problem first begin, what type of disorder did/do you have, what treatment (if any) did/do you receive, describe your recovery, etc.)

6. Do you have a color vision deficiency or color blindness? Yes No

7. Are you currently pregnant? Yes Maybe or Not Sure No

8. Are you currently taking any prescription medications for anxiety, mood, depression, attention, or any other psychological concerns? Yes No

   a. *Display This Question if YES*: Please describe the prescription medications you are taking (i.e., for psychological concerns).

Appendix C: Consent Form

INTRODUCTION

You are being asked to take part in a research study at the Duke University Medical Center (DUMC). Research studies are voluntary and include only people who choose to take part. Please read this consent form carefully and take your time making your decision. In order to decide whether you wish to be a part of this research study, you should know enough about its risks and benefits to make an informed judgment. This consent form gives you detailed information about the research study that a member of the research team will discuss with you. This discussion should go over all aspects of this research: its purpose, the procedures that will be performed, any risks of the procedures, and possible benefits. As the study doctor or study staff discusses this consent form with you, please ask him/her to explain any words or information that you do not clearly understand. You can choose not to participate, and you can choose to end your participation at any time during the study.

Please tell the study doctor or study staff if you are taking part in another research study.

WHO WILL BE MY DOCTOR ON THIS STUDY?

If you decide to participate, Dr. Nancy Zucker will be your doctor for the study and will be in contact with your regular health care provider throughout the time that you are in the study and afterwards, if needed.

WHY IS THIS STUDY BEING DONE?

The purpose of this study is to understand how individuals differ in their attention and other thinking styles, and how such differences are associated with eating behaviors.

HOW MANY PEOPLE WILL PARTICIPATE IN THIS STUDY?

Approximately 300 women will take part in this study at Duke.

WHAT IS INVOLVED IN THIS STUDY?

If you agree to be in this study, you will be asked to sign and date this consent form. You will participate in a 3-hour laboratory assessment visit, which will include the following:

1. You will participate in a structured interview conducted by a member of the study team. The interview will include questions about your medical history, mental health concerns, and eating behaviors, and will last approximately 30-60 minutes.
2. Your height, weight, and waist circumference (girth) will be measured by a study team member.
3. You will participate in behavioral testing for approximately 60-75 minutes. This testing will be administered by a study team member and will include tasks and activities to understand how you learn, solve problems, pay attention, and remember information.
4. Finally, you will complete several online self-report survey questionnaires, using a desktop computer, which will take approximately 30-60 minutes to complete. The surveys will ask about your health, eating behaviors, and current hunger and

mood. If needed, the online surveys may be completed outside of the 3-hour laboratory assessment visit (e.g., at home on a personal computer).

Your participation in this study is completely voluntary. You can choose to stop participating at any time without penalty or loss of any benefits to which you are entitled. You will be offered a scheduled break about half way through your assessment visit. You may also take any additional breaks that you need during the visit.

HOW LONG WILL I BE IN THIS STUDY?

If all survey questionnaires are completed, your participation in the study will end at the completion of your assessment visit. Total time for your participation in the study is expected to be 3-4 hours. We may need to contact you by phone or email following the assessment visit to ask for clarification about some of your responses.

You can choose to stop participating at any time, and we encourage you to talk to the study doctor or study staff if you would like to stop participating in the study.

WHAT ARE THE RISKS OF PARTICIPATING IN THIS STUDY?

There are no physical risks associated with this study. As with any study, however, there is the potential risk of loss of confidentiality. We will take every precaution to minimize this risk and keep your information confidential (such as using code numbers rather than names and keeping all data on a password protected database); but this cannot be guaranteed.

In addition, some of the questions we will ask you as part of this study may make you feel uncomfortable or some of the behavioral tasks may cause you to feel tired (e.g., after long periods of concentrating) or frustrated (e.g., if you are not able to complete all

of the items). You may take a break at anytime during the assessment visit. You may also refuse to answer any of the questions or you may stop your participation in this study at any time.

WHAT ARE THE BENEFITS OF PARTICIPATING IN THIS STUDY?

This research study is not a diagnostic medical or psychological test and it is not likely that the procedures included in this study will be of direct benefit to you. However, the information obtained from this study will be used to improve our knowledge of the learning processes involved in healthy and unhealthy eating behaviors among women. Knowledge from this study may help us develop more effective treatments for individuals with eating disorders.

If you wish, the study researcher or staff member can share the results of your behavioral tests and self-report survey questionnaires by providing a one-page summary of your results. This may help you to better understand your learning style and cognitive strengths, although this cannot be guaranteed.

WHAT ARE THE COSTS OF PARTICIPATING IN THIS STUDY?

There are no costs associated with participation in this study.

WHAT ABOUT PAYMENT FOR PARTICIPATION?

You will be paid approximately $10/hour, up to $35, or you will earn course credit for completing the in-person assessment visit and online survey. The entire study should last about 3-4 hours. You will receive your compensation at the end of the in-person assessment visit.

WILL MY INFORMATION BE KEPT CONFIDENTIAL?

As part of this study, Dr. Zucker and her research team will maintain study records that identify you. Study records that identify you will be kept confidential as required by law. Federal Privacy Regulations provide safeguards for privacy, security, and authorized access. Except when required by law or for your care, you will not be identified by name, social security number, address, telephone number, or any other direct personal identifier in study records disclosed outside of Duke University Health System (DUHS). For records disclosed outside of DUHS, you will be assigned a unique code number. The key to the code will be kept securely at DUHS, accessible only to Dr. Zucker and her research team.

Certain journals require that the data for their publication be entered into a larger database accessible to other researchers. No personal identifiers will be associated with this data. While information and data resulting from this study may be published in a scientific journal or presented at scientific meetings, your identity will not be revealed or used in any scientific reports of this study.

Your records may be reviewed in order to meet federal or state regulations. Reviewers may include representatives of the National Institutes of Health, the Office of Human Research Protection, and the Duke University Health System Institutional Review Board. If any of these groups review your research record, they may also need to review your entire medical record. If information about you is disclosed to outside reviewers for audit purposes, it may be further disclosed by them and may not be covered by the federal privacy regulations.

The study results will be retained in your research record for at least six years after the study is completed. At that time either the research information not already in your medical record will be destroyed or information identifying you will be removed from such study results at DUHS. Any research information in your medical record will be kept indefinitely.

WHAT ABOUT RESEARCH RELATED INJURIES?

Immediate necessary medical care is available at Duke University Medical Center in the event that you are injured as a result of your participation in this research study. However, there is no commitment by Duke University, Duke University Health System, Inc., or your Duke physicians to provide monetary compensation or free medical care to you in the event of a study-related injury.

For questions about the study or research-related injury, contact Dr. Nancy Zucker at (919) 668-2281 during regular business hours and at (919) 308-9140 after hours and on weekends and holidays.

WHAT ABOUT MY RIGHTS TO DECLINE PARTICIPATION OR WITHDRAW FROM THE STUDY?

You may choose not to be in the study, or, if you agree to be in the study, you may withdraw from the study at any time. If you withdraw from the study, no new data about you will be collected for study purposes other than data needed to keep track of your withdrawal.

Your decision not to participate or to withdraw from the study will not involve any penalty or loss of benefits to which you are entitled, and will not affect your access to

health care at Duke or your grades (if you are a student at Duke). If you do decide to withdraw, we ask that you contact Dr. Nancy Zucker in writing and let her know that you are withdrawing from the study. Her mailing address is Box 3842, Duke University Medical Center, Durham, NC 27710.

WHOM DO I CALL IF I HAVE QUESTIONS OR PROBLEMS?

For questions about the study or a research-related injury, or if you have problems, concerns, questions, or suggestions about the research, contact Dr. Nancy Zucker at (919) 668-2281 during regular business hours and at (919) 308-9140 after hours and on weekends and holidays.

For questions about your rights as a research participant, or to discuss problems, concerns, questions or suggestions related to the research, or to obtain information or offer input about the research, contact the Duke University Health System Institutional Review Board (IRB) Office at (919) 668-5111.

STATEMENT OF CONSENT

"The purpose of this study, procedures to be followed, risks and benefits have been explained to me. I have been allowed to ask questions, and my questions have been answered to my satisfaction. I have been told whom to contact if I have questions, to discuss problems, concerns, or suggestions related to the research, or to obtain information or offer input about the research. I have read this consent form and agree to be in this study, with the understanding that I may withdraw at any time. I have been told that I will be given a signed and dated copy of this consent form."

_____	_____	_____
Signature of Subject	Date	Time
_____	_____	_____
Signature of Person Obtaining Consent	Date	Time

you would like to stop and not participate, or if you have any questions, just let me know.

Appendix D: Self-Report Measures for Study Visit

Dietary Fat and Sugar Intake (DFS) Short Questionnaire

Australian and Investigator-Modified Versions

	Original Australian Measure		Investigator-Modified DFS Measure
1	Mince, beef or lamb, for example, in hamburgers, nachos or bolognaise	1	Hamburgers, cheeseburgers, meat loaf, or other ground beef dish
		2	Tacos, burritos, burrito bowls, nachos, enchiladas, etc. with meat or chicken
2	Beef or pork such as steak, ribs, roasts or in sandwiches	3	Beef or pork, such as steak, ribs, roasts, or in sandwiches or frozen dinners
3	Fried chicken or chicken burgers	4	Fried chicken, chicken burgers, turkey burgers
		5	Fried fish, fish sandwich, fish sticks, fish nuggets
4	Sausages, frankfurts, or salami	6	Hot dogs, sausages (like Italian or chorizo), boloney, salami
5	Bacon	7	Bacon, breakfast sausage
6	Salad dressings (not low fat)	8	Salad dressings (not low fat)
7	Margarine, butter or oil in cooking	9	Butter, margarine or oil in cooking, or on bread, potatoes, vegetables, etc.
8	Eggs (not egg whites alone)	10	Eggs (not egg whites alone)
9	Pizza	11	Pizza, including frozen, take-out, restaurant, homemade
10	Cheese or cheese spread (not low fat)	12	Cheese, sliced cheese, or cheese spread, including on sandwiches (not low fat)
11	French fries, fried potatoes	13	French fries, fried potatoes, hash browns
12	Corn chips, potato chips, popcorn with butter	14	Snacks like potato chips, corn/tortilla chips, popcorn with butter or oil (not low fat pretzels)

		15	Peanuts, cashews, almonds, or other nuts or seeds
13	Doughnuts, pastries, croissants	16	Doughnuts, pastries, croissants, sweet rolls, coffee cake
14	Cakes, cookies	17	Cake, cupcakes, cookies, pie, cobbler
		18	Breakfast bars, granola bars, protein bars, Power bars
15	Ice cream (not sorbet or low fat)	19	Ice cream, ice cream bars, gelato, milkshakes (not low fat)
16	Chocolate	20	Chocolate, candy bars
17	Lollies	21	Other candy, like hard candy, caramels, lollipops, jelly beans (not chocolate)
18	Spreads incl. peanut butter, jam, honey	22	Peanut butter, mayonnaise, sandwich spreads, aioli
		23	Jelly, jam, honey, syrup, agave syrup, cane juice
19	Pancakes or French toast	24	Pancakes, French toast, waffles, biscuits, muffins, Pop Tarts
		25	Sweetened breakfast cereals, like Frosted Flakes, Corn Pops, Fruit Loops, Cocoa Puffs
		26	Smoothies (e.g., fruit smoothies), meal replacement drinks or supplements (e.g., Slimfast, Ensure), protein shakes
20	Sports drinks (e.g., Gatorade) or energy drinks (e.g. Red Bull)	27	Sports drinks (e.g., Gatorade) or energy drinks (e.g., Red Bull, Monster) (not diet drinks)
21	Soft drink (not including diet)	28	Regular soft drinks (e.g, Coke), or bottled drinks like Snapple (not diet drinks)
		29	100% fruit juice, such as orange, apple, grapefruit, including fresh, frozen, or bottled
23	Other sweetened beverages (e.g., juice with added sugar, cordial, sweetened teas)	30	Other sweetened beverages (e.g., lemonade, sweet tea, juice made with high fructose corn syrup or sugar)
22	Milk (full fat only). Include milk drunk by itself or in	31	Milk (whole milk or 2% milk only). Include milk drunk by itself or in

	cappuccinos, milkshakes, hot chocolates etc.		coffee/espresso drinks, cereal, milkshakes, hot chocolate, etc.
		32	Beer, wine, liquor, mixed drinks, or other alcoholic beverages
24	White bread (white bread only)	33	White bread, including French bread, rolls, hamburger buns, bagels, English muffins, or sandwich bread (white bread only)
25	In the past year, how many times have you eaten food from a takeaway or fast food restaurant for example McDonalds, KFC, Mexican, Chinese, Thai, Italian (pizza or pasta)?	34	In the past year, how many times have you eaten food from a fast food restaurant or pizza place (either dine-in or take-out), such as McDonalds, Burger King, Biscuitville, Taco Bell, Dominos?
		35	In the past year, how many times have you eaten food from another type of restaurant (either dine-in or take-out), such as Chipotle, TGIF, Chinese, Thai, Italian, Mexican?
26	In the past week, how many teaspoons of sugar have you added to your beverages, cereal or food?	36	In the past week, how many teaspoons (tsp) of sugar, in total, have you added to your beverages, cereal or food?

Note: The original Australian DFS-SQ measure was developed by Francis & Stevenson (2013).

Response Options

The original Australian measure (Francis & Stevenson, 2013) uses the following five-point scale:

- 1=Less than 1 per month
- 2=2-3 per month
- 3=1-2 per week
- 4=3-4 per week
- 5=5+ per week

Response Options (Investigator-Modified)

The investigator-modified version uses the following eight-point scale:

- 0=Never
- 1=Less than monthly

- 2=Once a month
- 3=2-3 times a month
- 4=Once a week
- 5=2-3 times a week
- 6=4-6 times a week
- 7=Every Day

Positive and Negative Affect Schedule (PANAS)

Instructions: This scale consists of a number of words that describe different feelings and emotions. Please read each item and indicate to what extent you feel this way right now, that is, at the present moment. Use the following scale from 1 to 5 for your responses.

	very slightly or not at all (1)	a little (2)	moderately (3)	quite a bit (4)	extremely (5)
interested	(1)	(2)	(3)	(4)	(5)
distressed	(1)	(2)	(3)	(4)	(5)
excited	(1)	(2)	(3)	(4)	(5)
upset	(1)	(2)	(3)	(4)	(5)
strong	(1)	(2)	(3)	(4)	(5)
guilty	(1)	(2)	(3)	(4)	(5)
scared	(1)	(2)	(3)	(4)	(5)
hostile	(1)	(2)	(3)	(4)	(5)
enthusiastic	(1)	(2)	(3)	(4)	(5)
proud	(1)	(2)	(3)	(4)	(5)
irritable	(1)	(2)	(3)	(4)	(5)
alert	(1)	(2)	(3)	(4)	(5)
ashamed	(1)	(2)	(3)	(4)	(5)
inspired	(1)	(2)	(3)	(4)	(5)
nervous	(1)	(2)	(3)	(4)	(5)
determined	(1)	(2)	(3)	(4)	(5)
attentive	(1)	(2)	(3)	(4)	(5)
jittery	(1)	(2)	(3)	(4)	(5)
active	(1)	(2)	(3)	(4)	(5)
afraid	(1)	(2)	(3)	(4)	(5)

Power of Food Scale (PFS)

Instructions: Please indicate the extent to which you agree that the following items describe you. Use the following scale from 1 to 5 for your responses.

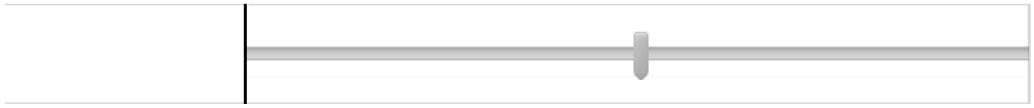
	I don't agree (1)	I agree a little (2)	I agree somewhat (3)	I agree quite a bit (4)	I strongly agree (5)
I find myself thinking about food even when I'm not physically hungry.	(1)	(2)	(3)	(4)	(5)
I get more pleasure from eating than I do from almost anything else.	(1)	(2)	(3)	(4)	(5)
If I see or smell a food I like, I get a powerful urge to have some.	(1)	(2)	(3)	(4)	(5)
When I'm around a fattening food I love, it's hard to stop myself from at least tasting it.	(1)	(2)	(3)	(4)	(5)
It's scary to think of the power that food has over me.	(1)	(2)	(3)	(4)	(5)
When I know a delicious food is available, I can't help myself from thinking about having some.	(1)	(2)	(3)	(4)	(5)
I love the taste of certain foods so much that I can't avoid eating them even if they're bad for me.	(1)	(2)	(3)	(4)	(5)
Just before I taste a favorite food, I feel intense anticipation.	(1)	(2)	(3)	(4)	(5)
When I eat delicious food I focus a lot on how good it tastes.	(1)	(2)	(3)	(4)	(5)
Sometimes, when I'm doing everyday activities, I get an urge to eat 'out of the blue' (for no apparent reason).	(1)	(2)	(3)	(4)	(5)
I think I enjoy eating a lot more than most other people.	(1)	(2)	(3)	(4)	(5)
Hearing someone describe a great meal makes me really want to have something to eat.	(1)	(2)	(3)	(4)	(5)
It seems like I have food on my mind a lot.	(1)	(2)	(3)	(4)	(5)
It's very important to me that the foods I eat are as delicious as possible.	(1)	(2)	(3)	(4)	(5)
Before I eat a favorite food, my mouth tends to flood with saliva.	(1)	(2)	(3)	(4)	(5)

Hunger Visual Analog Scale

Administered via Qualtrics

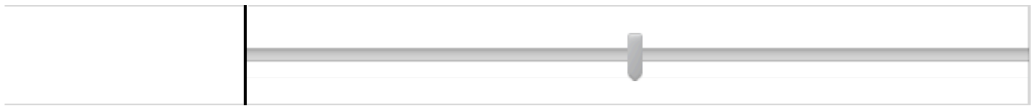
Please rate your current level of hunger.

Not at all hungry — Extremely hungry



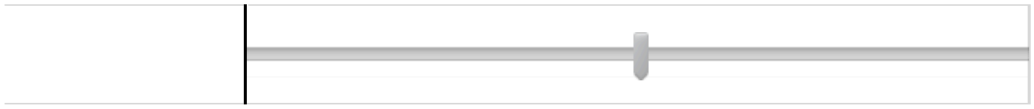
Please rate your current level of satiety.

Not at all satiated — Extremely satiated



Please rate your current level of fullness.

Not at all full — Extremely full



What time did you eat your last meal/snack?

_____

Autobiographical Memory Questionnaire (AMQ)
Standard Prompt for General Event Memory
Administered via Qualtrics

People can remember events from their own life, like what they ate for breakfast or the first time they rode a bicycle. For the next task, we are interested in these types of memories.

Instructions: Think of an event that...

(1) Is from your own life. Do not choose an event that you heard about from someone else.
(2) Is a specific event. For example, do not write, "eating breakfast" but try to think of a specific scene like, "I was eating breakfast with my boss and I spilled maple syrup into her lap."
(3) You have thought or talked about often.

The event can be from any time in your life. On the next page, you will be asked to describe the event you thought of that fits criteria 1-3 above. Now, take some time to think of an event. It may take a few minutes, and that is normal.

Press the button only when you have the event in mind. Take your time.

Please describe the event you thought of in as much detail as you can. Remember that it should be a specific scene that you have thought or talked about often.

When did the event you are remembering originally occur? (Choose the most accurate range.)

- Within the past day
- Within the past week
- Within the past month
- Within the past 3 months
- Within the past year
- Within the past 5 years
- Within the past 10 years
- More than 10 years ago

Please estimate how old you were when this event occurred: _____

Autobiographical Memory Questionnaire (AMQ)
Investigator-Developed Prompt for Eating Event Memory
Administered via Qualtrics

People can remember eating episodes and events, such as a meal or snack they ate recently. People can also remember special occasion meals, such as dining at a favorite restaurant for a birthday, or the first time they tried a new food, or eating their favorite home-cooked meal. For the next task, we are interested in these memories for either typical or special occasion eating events or episodes.

Instructions: Think of an event that...

(1) Involves a specific eating episode, such as eating a specific meal, food, or snack. It should be an eating event/episode in which you personally ate the food.
(2) Is a specific event. For example, do not write "yogurt, granola, and coffee for breakfast on weekdays" but try to think of a *particular* eating episode, meal, or event that involved eating food.
(3) You have thought or talked about often.

This could be something you ate a long time ago or very recently. On the next page, you will be asked to describe an eating episode/event that fits criteria 1-3 above. Now, take some time to think of an event. It may take a few minutes, and that is normal.

Press the button only when you have the event in mind. Take your time.

Please describe the event you thought of in as much detail as you can. Remember that it should be a specific eating event/episode that you have thought or talked about often.

When did the event you are remembering originally occur? (Choose the most accurate range.)

- Within the past day
- Within the past week
- Within the past month
- Within the past 3 months
- Within the past year
- Within the past 5 years
- Within the past 10 years
- More than 10 years ago

Please estimate how old you were when this event occurred: _____

Autobiographical Memory Questionnaire (AMQ)

Memory Characteristic Ratings for both General and Eating Event Memories

Administered via Qualtrics

Construct Variable	Question	Anchors
Rehearsal of Memory		
Thought or Talked About Event	Since it happened, I have thought or talked about this event.	1 = not at all, 3 = sometimes, 5 = many times, 7 = more than for any other (memory) / (event)
Sensory		
Visual Imagery	When remembering the event, I can see it in my mind.	1 = not at all, 3 = vaguely, 5 = distinctly, 7 = (as clearly as it were happening now) / (as clearly as an event happening in front me)
Auditory Imagery	When remembering the event, I can hear it in my mind.	1 = not at all, 3 = vaguely, 5 = distinctly, 7 = as clearly as it were happening now / (as clearly as an event happening in front me)
Recollection		
Reliving	When remembering the event, I feel as though I am reliving it again.	1 = not at all, 3 = vaguely, 5 = distinctly, 7 = as clearly as if it were happening now / as clearly as if it were happening to me now
Emotions		
Strength of Emotions	When remembering the event, I feel the emotions as strongly as I did then.	1 = not at all, 3 = vaguely, 5 = distinctly, 7 = as clearly as if it were happening now
Positivity of Emotions	When remembering the event, the emotions are extremely positive.	1 = not at all, 3 = hardly, 5 = somewhat, 7 = entirely
Negativity of Emotions	When remembering the event, the emotions are extremely negative.	1 = not at all, 3 = hardly, 5 = somewhat, 7 = entirely
Intensity of Emotions	The emotions I feel are intense	1 = not at all, 3 = hardly, 5 = somewhat, 7 = entirely
Perspective		

First or Third Person Perspective	When you remember the event, do you see it:	Through your own eyes, like a first-person perspective? As an outside observer, like a third-person perspective? As a mixture of the two perspectives above Neither (no visualization)
Flexible Perspective	If you try, can you switch perspectives?	Yes, No, Does not apply
Availability/Memory Age		
Recency of Event	When did the event you are remembering originally occur?	Within the past day, Within the past week, Within the past month, Within the past 3 months, Within the past year, Within the past 5 years, Within the past 10 years, More than 10 years ago
Event Age	Please estimate how old you were when this event occurred.	Age estimated in years

Note: Items included were from the AMQ developed by (Greenberg & Rubin, 2003; Rubin, 2006; Rubin, Schrauf, & Greenberg, 2003) and the perspective measure was developed by (Butler, Rice, Wooldridge, & Rubin, 2016)).

Appendix E: Eating Disorder Examination Addition

Eating Disorder Examination 17.0D
Loss of Control Eating Behavioral Features

This investigator-developed addition to the Eating Disorder Examination (EDE), Version 17.0D Interview Guide (Fairburn, Cooper, & O'Connor, 2014), was designed to assess behavioral features and distress associated with loss of control (LOC) eating episodes (i.e., subjective binge episodes [SBEs]). This module was asked after the Binge Eating Disorder Module (pg. 15) of the EDE, 17.0D Interview Guide.

LOSS OF CONTROL (LOC) EATING DISORDER MODULE

[Enter this module if participant endorses subjective bulimic episodes SBEs (no minimum number, but at least 3 in the preceding 12 weeks will facilitate completion of the features of SBE). Otherwise rate 9. Use a respondent-based interviewing style, rather than the investigator-based style of the EDE.]

Features Associated with Loss of Control Eating Episodes

During these episodes (refer to subjective bulimic episodes that are representative of those over the past three months)**, have you *typically* .....**

... Eaten much more rapidly than normal?
... Eaten until you have felt uncomfortably full?
... Eaten large amounts of food when you haven't felt physically hungry?
... Eaten alone because you have felt embarrassed about how much *or what (e.g., type of foods)* you were eating?
... Felt disgusted with yourself, depressed, or very guilty?

[Rate each feature individually using the binary scheme below.]
0 - Feature not present
1 - Feature present

Distress about Loss of Control Eating

In general, over the past three months how distressed or upset have you felt about these episodes (refer to subjective bulimic episodes that are representative of those over the past three months)**?**

[Rate the presence of marked distress about the loss of control (SBE) eating. This may stem from the actual behaviour itself or its potential effect on body shape and weight.]

0 – No marked distress
1 – Marked

RETURN TO EDE STYLE OF QUESTIONING

Note: Investigator-Developed Addition to the Eating Disorder Examination, Version 17.0D Interview Guide (Fairburn, Cooper, & O'Connor, 2014).

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Biography

Lori Keeling attended American University, in Washington, DC, graduating from the School of Public Affairs in 1997 with a Bachelor of Arts Degree in Interdisciplinary Studies. Before attending Duke, she spent 10 years working in the field of public health and health promotion, where she focused on developing health interventions for adults and children to help them maintain a healthy weight and manage diabetes and heart disease. Ms. Keeling completed a Master of Public Health Degree in International Health from Emory University's School of Public Health in 2001, where she completed a Master's thesis on "Working together in development and health: Lessons learned from a qualitative study in Uganda." She completed her Master of Arts degree in Psychology at Duke in 2013, with her Major Area Paper titled, "The potential role of memory in the development and maintenance of binge eating disorder," under the mentorship of Nancy Zucker, PhD. In July 2018, she completed her American Psychological Association (APA)-accredited clinical psychology internship at Dartmouth-Hitchcock Medical Center, Department of Psychiatry in Lebanon, New Hampshire. Her graduate fellowships and honors include a Bass Instructional Fellowship: Instructor of Record, to teach an undergraduate course in the Psychology of Mindfulness Meditation, and the Charles LaFitte Graduate Research Award for her dissertation research study.