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Review article

Eating behavior as a new frontier in memory research

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ABSTRACT

The study of memory is commonly associated with neuroscience, aging, education, and eyewitness testimony. Here we discuss how eating behavior is also heavily intertwined—and yet considerably understudied in its relation to memory processes. Both are influenced by similar neuroendocrine signals (e.g., leptin and ghrelin) and are dependent on hippocampal functions. While learning processes have long been implicated in influencing eating behavior, recent research has shown how memory of recent eating modulates future consumption. In humans, obesity is associated with impaired memory performance, and in rodents, dietary-induced obesity causes rapid decrements to memory. Lesions to the hippocampus disrupt memory but also induce obesity, highlighting a cyclic relationship between obesity and memory impairment. Enhancing memory of eating has been shown to reduce future eating and yet, little is known about what influences memory of eating or how memory of eating differs from memory for other behaviors. We discuss recent advancements in these areas and highlight fruitful research pursuits afforded by combining the study of memory with the study of eating behavior.

1. Introduction

The scientific study of memory is close to 150 years old and has evolved greatly since the days of Ebbinghaus, Bartlett, and James (Bower, 2000). At the level of basic science, significant strides have been made in describing mnemonic processes (Baddeley, 2000; Bjork and Bjork, 1992), uncovering neurological underpinnings of memory formation (Bird and Burgess, 2008; Kandel et al., 2014; Squire, 2004; Squire and Wixted, 2011), and computationally modeling memory systems (Burgess and Hitch, 2005; Kahana, 2020). The study of memory has also extended into applied settings. Elizabeth Loftus, for example, has done tremendous work detailing the role of false memories in the criminal justice system (Loftus, 1975; Loftus and Hoffman, 1989). Others have detailed the relation of memory and aging (Castel et al., 2007; Hess, 2005; Park and Festini, 2017), and of course, an ongoing quest continues to search for behavioral or pharmaceutical interventions that can improve people's mnemonic capabilities. The themes outlined above are often found in basic textbooks of memory (e.g. Baddeley et al., 2014) and are what many would commonly associate with the scientific discipline of memory. However, memory researchers would be well-served to consider eating behavior as an emerging frontier in the study of memory. Not only do these two processes rely on similar neural architecture, namely the hippocampus (Stevenson and Francis, 2017), but recent work has demonstrated that memory processes affect eating behavior and eating behavior can similarly affect memory processes (Higgs and Spetter, 2018). The purpose of this review, therefore, is to shed light on the recent intertwining of these (to many) seemingly distant areas of psychological science and illustrate to those interested in memory processes that there is much to glean by studying eating behavior.

2. A lesson from the study of learning

Memory's sister discipline, learning, has been tied to eating behavior since Pavlov and his pioneering work on digestive processes. Pavlov famously discovered that neutral cues (Conditional Stimuli [CS]) that preceded appetitive outcomes such as food or food odor (Unconditional Stimuli [US]) could elicit metabolic responses, such as the release of digestive enzymes, so long as the animal had properly learned the CS-US association. Moreover, he demonstrated that different food stimuli (e.g., bread or meat) influenced the amount and viscosity of saliva produced, suggesting metabolic responses are tailored to better digest previously encountered foods (Pavlov, 1910; Smith, 1995). Others have continued to demonstrate the influence of learning processes on eating behavior. Associations between flavors and their postingestive consequences create conditioned taste aversions and preferences. For example, Bolles et al. (1981) paired two different flavors with either flour (CS+) or chalk (CS-) and gave rats prolonged access to both mixtures. In a subsequent

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test, both flavors were paired with a flour/chalk mixture, but rats overwhelmingly preferred the flavor previously paired with the caloric flour outcome (CS+). Similar studies have shown evidence of conditioned taste and flavor preferences in children (Birch, 2009; Johnson et al., 1991) and adults (Kern et al., 1993; Zellner et al., 1983). Much of our recent understanding of conditioned taste preferences comes from the work of Anthony Sclafani and his colleagues who have, among other things, demonstrated various aspects of the neurobiology of this learning (for reviews see Myers, 2018; Sclafani, 2018).

Similarly, flavors paired with illness on a single occasion can result in taste aversions to those flavors, even with extended temporal delays between experiencing the flavor and illness (Garcia et al., 1955). Evidence of these conditioned taste aversions exists even in fetal rats who then retain those aversions later in periadolescence (Gruest et al., 2004; Stickrod et al., 1982). Conditioned taste aversion in humans is also well documented (Bernstein and Webster, 1980; Rozin and Zellner, 1985) and has important implications for cancer treatment, as food eaten prior to radiation can quickly become disliked (Redd and Andrykowski, 1982). Learning typically requires multiple trials of the CS and US being paired in close temporal proximity and evidence of previous learning tends to wane over time. That conditioned taste aversion violates these norms suggests that learning processes differ when learning about food and postingestive consequences than when learning about other types of paired events. Indeed, though fear conditioning can occur following a single CS-US pairing, learning will not occur if the CS and US are not paired in close temporal proximity and even post-natal rats can acquire but not retain fear learning for prolonged periods of time (Sanders et al., 2020). These reviewed findings also demonstrate how simple Pavlovian relationships can have significant impacts on eating behavior.

2.1. Learning and disordered eating

Learning processes have more recently been invoked to understand disordered eating and its effects, such as overeating, anorexia nervosa, bulimia nervosa, and obesity. Berridge et al. (2010) outline a dissociation between food "liking" and "wanting" and suggest that alterations in reward learning contribute to overeating (for a recent review, see Morales and Berridge, 2020). Cues such as restaurant logos and scents gain incentive value as a consequence of becoming associated with food outcomes and then motivate eating behavior when later experienced. These motivated eating bouts represent increases in reward "wanting" but not necessarily hedonic reward "liking." Such a dissociation is a hallmark feature of addiction phenotypes (Finlayson et al., 2007; Robinson et al., 2015; Robinson and Berridge, 2008). For instance, Watson et al. (2014) taught human participants to press buttons for either chocolate or popcorn rewards. Satiating participants on one reward resulted in biased responding for the other. Nevertheless, even when satiated on chocolate for example, presenting a neutral cue that had previously been associated with chocolate increased the chocolate button key response, indicating an increased desire for the chocolate (i.e. Pavlovian to Instrumental Transfer). This type of habitual responding, in which responses are made simply because of an association with a stimulus, likely contributes to a significant amount of overeating, and can be contrasted to goal-directed behavior in which a response is made with the intention to receive a specific outcome (van't Riet et al., 2011).

On the opposite side of the spectrum, anorexia nervosa (Foerde and Steinglass, 2017; Keating, 2010; Wagner et al., 2007) and bulimia nervosa (Grob et al., 2012; Wagner et al., 2010) are thought to be associated with impairments in reward learning, namely the ability to experience and learn from past rewarding events. This is likely due in part to the fact that anorexia nervosa is associated with abnormal dopaminergic responding—typically low responding during eating (O'Hara et al., 2015; Södersten et al., 2016; Wagner et al., 2007). Related, some models posit the restricted eating and excessive exercise becomes rewarding in anorexia, thus reinforcing these behaviors and driving further weight loss (Fladung et al., 2010; Keating, 2010).

To conclude, learning theory and eating behavior have a long and rich history. It should be noted that the majority of the research combining these two areas has come at the hand of learning theorists and behavioral neuroscientists (for a review, see Boutelle and Bouton, 2015). Meanwhile, the recent linkages between memory and eating processes have been primarily—but not exclusively—conducted by researchers who specialize in eating behavior. Their work has been instrumental in demonstrating the mnemonic control of eating but suggests that cognitive psychologists well-versed in mnemonic processes might offer insights to even further advance this new and growing arena.

3. Quantifying the overlap between memory processes and eating behavior

The aim of this review is to highlight the interconnected nature of research on memory processes and eating behavior. To 'quantify' the extent of this overlap, we conducted a literature review in December 2020 using the database PubMed. Drawn from the overlapping topics to be discussed throughout this review, we created search terms (see Supplementary Material) to "measure" the relatedness of these *topics* of both our *bases*: memory processes and eating behavior. We used the number of returned articles as our metric of overlap size between each base with each topic. Note, the overlap between our bases and some topics (e.g., memory processes and hippocampal functions; eating behavior and obesity) is massive, and so it is not possible to stringently select articles based on any formal set of criteria. That said, the pure number of returned articles is still telling of the relative size of the overlap between each base and topic and can be used to make judgements of the relative size from one overlap to another. Fig. 1 illustrates the existing overlap between memory processes and eating behavior and the various topics we have identified in this review. The mnemonic control of eating, determinants of memory of eating, hippocampal contributions to eating, neuroendocrine influence on memory, and relationship between obesity and memory performance are all areas currently understudied and ripe for future research.

4. Memory's effect on eating

The first demonstration between declarative memory and eating behavior was a report by Hebben et al. (1985) regarding the famous amnesiac patient H.M. It was observed that his reports of hunger were not influenced by recent eating events. Amazingly, he was once documented to have eaten an entire meal just one minute after he had previously consumed the exact same full meal—although it was not initially recognized as being caused by deficits to memory of eating. This finding has since been shown in other amnesic patients, some of whom have been willing to consume up to 3 full meals in under 90 min (over 1000 total calories) and points to the importance of memory of past eating events influencing current eating behavior (Rozin et al., 1998). Higgs et al. (2008b) followed up on this work and demonstrated that this effect is not due to impairments in sensory specific satiety.

That memory affects eating behavior has also been demonstrated in healthy populations. Higgs (2002) cued participants to think about their most recent meal immediately before consuming a subsequent snack. In Experiment 1, participants in the control group received no cue, and in Experiment 2, participants were cued to remember their lunch from the previous day or they received no cue. In both experiments, only recalling one's most recent meal reduced eating at the subsequent snack test. This effect has now been replicated several times across a number of different labs (Collins and Stafford, 2015; Higgs et al., 2008a,b; Szypula et al., 2020), although it can be affected by factors such as mood (Collins and Stafford, 2015) and depth of recall (Szypula et al., 2020). Moreover, Vartanian et al. (2016) replicated the effect using the traditional retrospective approach but also by having participants imagine eating a future meal, which suggests that similar neural and cognitive processes underlie both retrospective memory and episodic future thinking

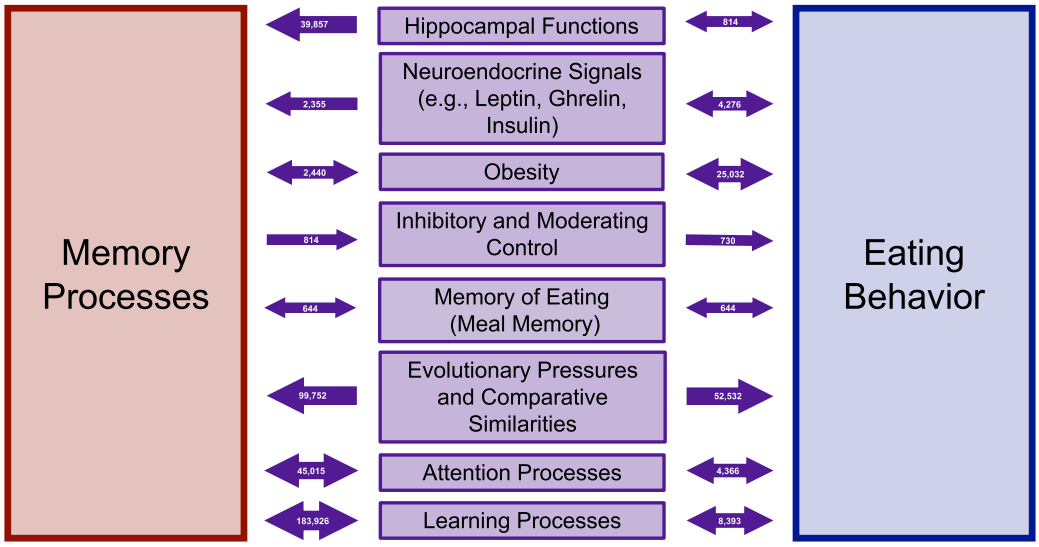

Fig. 1. Possible connections between memory processes and eating behavior supported by existing research. Arrow directionality indicates hypothesized causal relationship and arrow weight (log transformed) and value inside, indicates amount of existing research that supports each relationship (see Supplementary Material for details). Intervening variables are not mutually exclusive.

(Schacter et al., 2007, 2017) and that both contribute to the mnemonic control of eating. These data suggest that retrieval of a recent meal memory can modulate future eating.

4.1. Reduced memory of eating increases future eating

Efforts and manipulations that target the encoding of meal memories impact future eating. These studies typically involve an initial meal that is consumed while distracted or non-distracted, a follow up snack test, and finally, participants are asked to recall elements of the initial meal. For instance, Higgs and Woodward (2009) distracted participants while eating by having them watch television. At a later snack test, the distracted participants ate more than participants who did not watch television during the initial eating phase. The distracted participants also rated their memory of the initial meal as being less "vivid" than did the control participants. With that said, it is not necessarily clear that self-perceived memory vividness accurately relates to memory strength or accessibility. Mittal et al. (2011) conducted a similar procedure but critically asked participants to recall how much food they consumed during the initial eating phase. They found that participants in the distracted eating group significantly underestimated the amount of food they had previously consumed compared to the non-distracted group. However, this was confounded in that the distracted eating manipulation also caused those participants to consume more food in the initial eating task, therefore increasing the likelihood for underestimation (Francis et al., 2017). Thus, future research should investigate how distracted eating affects recall of eating while holding the initial meal constant. Oldham-Cooper et al. (2011) conducted such a study by having some participants play a video game to distract them. However, their memory test asked participants to remember the serial order of the foods they consumed during the initial eating event and not specifically how much of each food they had consumed. Nevertheless, distracted eating impaired serial-order memory relative to non-distracted eating, and all of these studies found distracted eating led to increased future snacking (for a review and meta-analysis, see Robinson et al., 2013).

Brunstrom et al. (2012) utilized a clever disappearing soup bowl mechanism to study the effect of memory on hunger. Participants were seated in front of a large or small portion of tomato soup. While some participants seated in front of the small portion consumed that small portion, others unknowingly consumed the large portion as it was covertly pumped into the bowl. Similarly, those seated in front of the large portion either ate the large portion or unknowingly ate the small portion as the soup was covertly siphoned out. Immediately after eating and one hour later, the amount of food actually consumed predicted self-reported hunger levels. However, two and three hours after eating the soup, self-reported hunger was predicted by memory of the portion size consumed (i.e., participants who were seated at the small portion were hungry regardless of how much they ate, and participants seated at the large portion were less hungry regardless of how much they actually ate). Thus, immediately after eating, self-reported hunger levels appear most influenced by putative physiological mechanisms, but as more time passes, memory of what and how much was consumed most influences hunger. This is particularly interesting in light of the fact that while distracted eating increases consumption of the current meal, distracted eating actually has a larger effect on increasing consumption at a subsequent meal (Robinson et al., 2013).

4.2. Enhanced memory of eating (sometimes) reduces future eating

Manipulations to enhance memory of eating have had mixed results in reducing future snacking. Several studies have found that instructing some participants to focus on sensory aspects of eating or to eat mindfully reduces later snacking compared to relevant controls (Allirot et al., 2018; Higgs, 2015; Higgs and Donohoe, 2011; Robinson et al., 2014b; Seguias and Tapper, 2018a,b). Within these studies, there is also mixed evidence as to whether or not attentive eating manipulations enhances memory of eating (Higgs and Spetter, 2018). Additionally, a number of more recent studies with attentive or mindful eating interventions have not reduced future snacking (Tapper and Seguias, 2020; Whitelock et al., 2019a, 2018; Whitelock et al., 2019b). Further research is needed to clarify the existence of this effect. One possible explanation of these inconsistencies is that memory for eating in the control conditions is already exceptionally strong, which would explain why memory for eating is not always enhanced by these manipulations, nor is future eating always reduced. Evidence in favor of this interpretation comes from a recent study that showed even distracted eating is better remembered than other similar behaviors (Seitz et al., 2021). Thus, while it may be possible to reduce memory of eating through distraction, all things equal, eating events are likely to be well remembered (likely because of their evolutionary significance; see below).

4.3. Replication in an animal model and concluding framework

Advances in technological equipment in neuroscience have also demonstrated the role of memory in eating behavior. Using rats, Hannapel et al. (2019) optogenetically inhibited the dorsal and ventral hippocampus (dHC & vHC) before, during, or after an eating event, and then measured amount of future food consumed and latency to initiate subsequent eating. Only when either the dHC or vHC was inactivated after the meal was consumed, thus disrupting memory consolidation, did the rats increase their amount of future eating and also show a reduced duration between eating bouts. These results were found using lab chow, sucrose solution, and saccharin solution as the main food variable. That rats who had their memory of eating the saccharin solution disrupted were quicker to initiate their next meal and consume more during that meal (relative to rats with an intact memory of eating the saccharin), suggests that it was not a lack of nutrients motivating the animal to eat (because saccharin contains no calories) but rather, their memory of their last meal. Thus, these results suggest a strong mediating relationship between memory of recent eating and future eating. In sum, there is considerable evidence that reducing meal memories, either through amnesia, distraction, deception, or optogenetics, increases future eating behavior and mixed evidence that attentive and/or mindful eating techniques can reduce future eating.

5. Eating ailments and memory

Given the global rise of eating related ailments (Hoek, 2016), and specifically obesity (Bentham et al., 2017), many have explored their effects on cognitive processes including memory. A number of correlational studies have established a negative correlation between Body Mass Index (BMI) and a variety of mnemonic capabilities. Cheke et al. (2016) designed a novel "Treasure-Hunt Task" designed specifically to test definitive features of episodic memory. Their results show a negative correlation between BMI and episodic memory (but see Cole and Pauly-Takacs, 2017). Other studies with less sophisticated memory measures (e.g. wordlist recall, verbal list learning) have largely found deficits in memory associated with higher BMI (Cournot et al., 2006; Gunstad et al., 2006; Prickett et al., 2018), although not all studies have found this relationship (for reviews see Higgs and Spetter, 2018; Loprinzi and Frith, 2018; Prickett et al., 2015). Neuroimaging studies also point to structural deficits and damage to memory associated areas being associated with overweight and obesity. For instance, numerous studies have shown reduced grey matter volume in hippocampus and prefrontal cortex in individuals with overweight and obesity (García-García et al., 2019; Herrmann et al., 2019; Kharabian Masouleh et al., 2016; Laurent et al., 2020; Medic et al., 2016; Raji et al., 2010; Willette and Kapogiannis, 2015).

It is, of course, important to note that this seemingly strong correlation between obesity and worsened memory performance poses a causal conundrum. Are individuals gaining weight because of their poorer mnemonic abilities or is weight gain causing deficits in memory? The relationship between weight gain and mnemonic deficits could also be cyclic. For instance, a one year longitudinal study with children (age 6–11) found that differences in some cognitive abilities (e.g., attention shifting, affective decision making) at the beginning of the study could predict BMI at the study's conclusion, but that initial BMI measures could also predict some cognitive abilities (e.g., working memory) at the study's conclusion (Groppe and Elsner, 2017). Non-human animal studies have therefore become important in understanding the causal and cyclic relationship between weight gain and memory impairments.

5.1. Evidence from non-human animal studies

The results from non-human animal studies tell a similar, albeit better controlled, story as those discussed above. A large number of studies in rats and mice have shown dietary induced obesity—or consuming diets known to cause obesity—results in rapid impairment on memory tasks, with the strongest deficits in spatial memory (Abbott et al., 2019; Cordner and Tamashiro, 2015). As an example, Kanoski and Davidson (2010) put rats on a diet high in fat and sugar (hereafter "Western diet") and showed impairments in a spatial memory task after only 72 h and stable deficits to working memory were observed after 30 days on the diet. Others have found similar rapid impairments to spatial memory as a result of Western diets (Beilharz et al., 2014; Tran and Westbrook, 2015). McLean et al. (2018) more recently demonstrated impaired episodic and contextual memory performance after just one day of exposure to a high fat diet, and Tran and Westbrook (2018) showed impaired familiarity judgments following acute exposure to a Western diet. This suggests Western diet decreases the quality of "what" memories in episodic memory. In fact, even exposing lactating mothers to a Western diet impaired elements of episodic memory (object location and recency learning) in their weaning pups when tested in adolescence (Wait et al., 2021).

Mechanistically, Western diets might impair mnemonic performance via neuroinflammation (Beilharz et al., 2015; Freeman et al., 2014; Veniaminova et al., 2020), reduced neuroplasticity (Abbott et al., 2019; Morin et al., 2017; Spinelli et al., 2017), decreased blood brain barrier function (Davidson et al., 2012; Hargrave et al., 2016; Kanoski et al., 2010), and altered neuroendocrine (e.g., leptin, ghrelin, insulin) signaling (Kanoski and Grill, 2017; Suarez et al., 2019). These diets might also impair memory performance through their effects on other cognitive processes, such as motivation (Blaisdell et al., 2014) and sustained attention (Blaisdell et al., 2017). While animal models provide ideal conditions for studying the effects of high fat and high sugar diets on memory, recent well controlled experiments in humans found sizable deficits in a number of memory tasks following just four days of eating a high fat and high sugar breakfast (Attuquayefio et al., 2017) and seven days of a high fat and high sugar diet (Stevenson et al., 2020). These data from studies in rodents, and now humans, make clear that dietary induced obesity, or simply consuming obesogenic diets, can cause deficits in memory processes.

5.2. The bidirectional relationship between obesity/poor diet and memory impairment

Despite mounting data that dietary induced obesity impairs memory, these data cannot entirely explain the correlation between human BMI and memory deficits. Just as inducing obesity begets memory impairments, studies in rodents similarly show that inducing memory impairments begets obesity. Forloni et al. (1986) and King et al. (1993) were among the first to demonstrate that lesions to the hippocampus result not only in memory deficits, but also hyperphagia. Davidson et al. (2009) provided a more precise and better controlled replication of this effect, demonstrating that destruction of the hippocampus results in increased food intake, body weight gain, and decreased general behavioral and metabolic activity. Damage to the hippocampus also results in impairments in detecting interoceptive cues related to hunger and satiety (Berriman et al., 2016; Davidson et al., 2010; Hebben et al., 1985; Kennedy and Shapiro, 2004).

This bidirectional relationship has been described by Terry Davidson and his colleagues as a "vicious cycle" of Western diet and cognitive decline (Davidson et al., 2005, 2014; Davidson et al., 2019a,b; Kanoski and Davidson, 2011). According to this model, there are both excitatory and inhibitory associations between food cues and their postingestive consequences. The notion of competing excitatory and inhibitory associations is well documented in learning theory (Bouton, 2004; Rescorla, 1993). A unique component of Davidson's model is that the excitatory association is thought to be hippocampal-independent while the inhibitory association is thought to rely on hippocampal-dependent processes such as interoceptive cues and memory of recent eating. Competing activation strengths of both associations dictates eating behavior. This model is particularly illuminating in light of the fact that intake of a

Western Diet leads to hippocampal dysfunction (reviewed above). Hippocampal dysfunction then results in an impaired ability to retrieve meal memories, detect interoceptive cues of satiety and hunger, and use other hippocampal dependent cognitive processes to appropriately inhibit eating behavior resulting in further intake of the Western diet, thus perpetuating the vicious cycle. This model adds nuance to the association between human BMI and memory deficits, and warns against the interpretation that poor diet simply causes obesity and cognitive impairments. Further, it suggests the need to develop separate intervention strategies aimed at targeting the hippocampal-independent excitatory and hippocampal-dependent inhibitory associations between food cues and their postingestive outcomes.

6. Determinants of memory of eating

The reviewed findings suggest memory of recent eating plays an important role in moderating future food consumption. Despite this, little is known about the factors that influence memory of eating. This should be the focus of future research because even small reductions in calorie consumption (e.g., 100 calories per day) could prevent weight gain in most of the population (Hill et al., 2003, and see Hall et al., 2011, for more sophisticated estimates). For instance, when Higgs (2002) asked participants to recall their most recent meal prior to consuming a snack, they observed a 21 % (Experiment 1, 61–85.5 kCal) and 49 % (Experiment 2, 93.5–131.1 kCal) reduction in snacking compared to participants asked to think about anything (Experiment 1) or recall a meal from the day before (Experiment 2). Using a similar manipulation and better powered study, Szypula et al. (2020) observed a 14 % reduction, roughly equivalent to 70 fewer calories. Importantly, while Higgs (2002) showed this effect in younger unrestrained females, Szypula et al. recruited a more diverse sample of both young and old female participants and those with a wider range of BMI. This suggests this method is broadly effective in reducing future eating, although it remains untested how such a manipulation influences eating in males. Intriguingly, when Seguias and Tapper (2018a,b) had some male and female participants mindfully eat their lunch by focusing on the sensory properties of food, they observed a 118 calorie (~58 %) reduction in subsequent snacking by females but also a 69 calorie (~36 %) reduction in subsequent snacking in males relative to a control group who focused on their heartbeat during the initial lunch. Thus, understanding the determinants of memory of eating may be insightful in designing interventions that enhance memory of eating and thus reduce unnecessary consumption and cue-induced eating in a wide range of individuals.

6.1. Psychological and environmental determinants of memory of eating

Few studies have directly addressed what influences memory of eating in humans. New et al. (2007) had participants walk around a farmer's market and sample items from each of the vendors. Participants then entered an opaque tent where they were asked to point to where each vendor was. The pointing error was linearly related with the caloric density of the food items, suggesting enhanced spatial memory for consuming high calorie foods. It should be noted that this is a somewhat crude measure of spatial memory, however the external validity of this measure and the study in general is impressive. Allan and Allan (2013) created a computer-based version of this task, where various food items were placed along a campus map. They found not only a spatial memory bias for high calorie foods but also that this bias was positively correlated with participant BMI, such that individuals with higher BMI showed a stronger bias towards remembering the spatial location of the high calorie images. That individuals with higher BMI showed enhanced memory for the location of foods is peculiar given higher BMI is typically associated with spatial and episodic memory deficits. Indeed in our own studies (see discussion below), we have found evidence of weak positive correlations between memory of eating accuracy and BMI (Seitz et al., 2021) and between memory of high calorie food stimuli and BMI (Seitz et al., 2020). Thus, individuals with overweight and obesity might show some memory biases towards eating and food relevant cues but this remains poorly understood. While speculative, this may be similar to how older adults show general deficits in memory performance but compensate for this by prioritizing memory for high value information (Castel, 2005; Castel et al., 2002). Recent work has replicated the finding of enhanced spatial memory for high calorie food images and shown this effect is independent of personal experience with the food, duration of encoding, or hedonic evaluation of the food (de Vries et al., 2020). While these studies suggest the caloric density of a food item might enhance spatial memory, they are correlational in nature, two have simply used food images, and they do not speak to whether caloric density influences memory for how much food was consumed—an episodic component of the memory separate from its spatial location.

Seitz et al. (2021) provided an experimental test of the influence of caloric density on memory for how much food was consumed. Participants completed the Memory of Eating Task (MEaT) whereby they watched a video while being cued to eat every time a tone was sounded. This allowed the experimenters to manipulate exactly when and how often participants ate. Participants consumed the same amount (30 pieces) of either M&Ms, salted peanuts, or plain popcorn. When asked to recall how many pieces of food they consumed, participants who ate the two high calorie foods (around 5 calories per piece) were more accurate (less overestimation and underestimation) than those who ate the low-calorie popcorn (less than one calorie per piece) who systematically underestimated the number of food items consumed. The results from these studies suggest one factor that influences memory of eating is the caloric density of the food item consumed. However, whether it is specifically the caloric density that is influencing memory of eating or some other component of the food that correlates with caloric density (e.g., nutrient content, texture, flavor) remains untested.

The speed/rate at which food is consumed might also be reasonably expected to influence memory for how much food was consumed. Distributing learning trials, by increasing the inter-trial-interval for example, yields better retention (Cepeda et al., 2006; Underwood, 1961). Thus, while eating a meal, slower or more distributed eating (i.e., more time between each bite) should result in better memory of that meal than eating at a faster pace. This could potentially serve as one mechanism underlying the findings that slower pace of eating is associated with lower rates of obesity (Robinson et al., 2014a), because of memory's moderating role on consumption. It could also explain why slower eating has been experimentally shown to reduce the amount of calories consumed during a meal (Bolhuis et al., 2011; Martin et al., 2007; Scisco et al., 2011) and reduces post meal hunger levels (Andrade et al., 2008, 2012). Ferriday et al. (2015) fed participants tomato soup via a modified feeding tube. Three hours later, participants were asked to pour into a bowl the amount of soup they remembered consuming, and those who consumed the soup slowly were more accurate at this task than those who consumed the soup quickly. A limitation of this study was that consuming soup via a pump was a contrived and likely salient eating scenario which may have influenced memory performance and had limited applicability to actual eating behavior. Hawton et al. (2018) had participants consume a pasta dish either quickly ($n = 11$) or slowly ($n = 10$) and they controlled eating pace using an auditory cue. Two hours later, participants who ate slowly were more accurate in recognizing the correct portion size of their pasta dish in an array of images. One thing to note about this design and the design used by Ferriday et al. was that the memory test occurred several hours after consuming the food. This is important for understanding how memory of recent eating moderates future eating (which is expected to occur several hours after the initial eating event), but in terms of evaluating the strength of the initial encoded memory, it is possible that participant hunger levels may have influenced their responses. That is, just as memory of eating influences subsequent hunger levels (Brunstrom et al., 2012), hunger levels might also influence reported memory of eating.

To test immediately after encoding and speak specifically to the

retrieval strength of the encoded memory of eating (Bjork and Bjork, 1992), Seitz et al. (2021) used the MEaT task (described above) to investigate how eating rate influences memory of eating immediately following the initial eating event. Participants picked up the food item and placed it in their mouths—in contrast to food being pumped into their mouths and the memory test involved recall of how many M&Ms were consumed. As hypothesized, participants who completed a slow version of the MEaT, consuming 30 M&Ms on average once every 45 s, were significantly more accurate in remembering how many M&Ms they consumed compared to participants who consumed 30 M&Ms quickly (on average once every 15 s) (Seitz et al., 2021). This occurred even though the retrieval interval was identical in both conditions (i.e., the fast group started eating later into the video so both groups ended at the same time). One potential mechanism underlying this effect may be enhanced rehearsal in the slower eating group, although we find this explanation unlikely given participants were not aware they were being tested on their memory of eating. Nevertheless, slower and more distributed eating appears to effectively enhance memory of eating, although its downstream effects on later food consumption are less known. This also demonstrates how simple manipulations known to enhance memory (e.g., distributed vs massed encoding) can be applied to enhance memory of eating. Perhaps other techniques that strengthen encoding processes or aid in enhanced retrieval (e.g., matching encoding and retrieval environments) may be used to similarly enhance memory of eating.

The extant studies suggest that the caloric density of the food item consumed and rate at which it is eaten affects later recall. Still though, there remain a host of additional factors related to the food items themselves (e.g., nutrient density, flavors, novelty, etc.), and nature of the eating experience (e.g., alone vs with others, time of day, meal size, etc.) that may also influence memory of eating. Future research is needed to uncover additional determinants of memory of eating and how such changes in memory of eating influence its regulatory control of future eating.

Source monitoring and reality monitoring also likely influence memory of eating. Source monitoring involves determining the origin of memories and may be particularly difficult for eating behavior given its frequent and ritualistic occurrence (Bradburn et al., 1987). Children—who typically exhibit more errors in source monitoring, were found to report a high number of intrusions (i.e., memory for things they did not eat) when asked to report their breakfast from 24 -hs prior (Baxter et al., 2008). Reality monitoring involves determining whether memories are based on external or internal sources and could be a challenge for individuals who often think about food and eating events and those with so called "food addiction" (Gearhardt et al., 2009). Both processes are relevant to individuals trying to remember the content and quantity of their recent meals and yet, to our knowledge, have not been specifically studied in relation to eating behavior.

6.2. Physiological and neuroendocrine determinants of memory of eating

While the psychological determinants of memory for eating are still largely unknown, much work has demonstrated the physiological and neuroendocrine signals that influence memory of eating. Leptin is a gut-derived hormone that communicates with the hypothalamus to effectively induce feelings of fullness and cease eating (Farooqi et al., 1999). Receptors for leptin are also found in the hippocampus (Lathe, 2001) and leptin administration to the hippocampus generally enhances memory function (Malekizadeh et al., 2017; Oomura et al., 2006). Paradoxically however, leptin administration to the hippocampus decreases learning about food relevant information. For instance, in rats, leptin administration to the ventral (but not dorsal) hippocampus impairs memory consolidation for the spatial location of food (Kanoski et al., 2011) and systemic leptin administration attenuates conditioned place preference for sucrose (Figlewicz et al., 2004; Shimizu et al., 2017). Leptin, therefore, may aid in encoding the reward value of food, with high volumes of leptin in the vHC resulting in the attenuation of food's value and decreased leptin resulting in enhanced value assigned to food (Davis et al., 2011; Domingos et al., 2011; Hommel et al., 2006). Alternatively, it is possible that high volumes of leptin in the vHC may promote the association of food relevant information with feelings of satiety. This association then competes with and can suppress the excitatory association between food relevant information and rewarding food outcomes (Davidson et al., 2019a,b; and described earlier). Thus, animals might not demonstrate conditioned place preference because the "place" has been associated with feelings of fullness or nonrewarding food intake which prevents expression of any excitatory associations between the "place" and food (Kanoski et al., 2011). By either account, because leptin serves as a satiety signal, high levels of leptin in the brain might indicate to the animal that it is not necessary to remember eating related information (perhaps to prioritize learning about other information) (Kanoski and Grill, 2017) or indicate that a food cue will no longer be followed by a reinforcing outcome—either of which would reduce certain aspects of memory of eating.

Less is known how leptin influences memory in humans. While leptin serves as a signal of fullness, a paradoxical finding is that individuals with obesity reliably exhibit higher concentrations of serum leptin (Francisco et al., 2018; Zimmet et al., 1996). This is thought to be the result of impaired transport of leptin across the blood-brain barrier (BBB) (Münzberg et al., 2005) and/or weakened leptin receptor sensitivity, and is why obesity is said to be associated with *leptin resistance* (Myers et al., 2010; Scarpace and Zhang, 2009). Because leptin administration to the vHC is thought to devalue food reward, the lack of leptin reaching the vHC and perhaps other critical regions may inflate the rewarding value of food outcomes. Suggestive of this, exogenous leptin concentrations (high concentrations being indicative of insulin insensitivity) were correlated with greater activation of striatal-limbic regions when viewing food images (Grosshans et al., 2012; Jastreboff et al., 2014). Despite these intriguing results, little research in humans has directly addressed the role of leptin in learning about and remembering food versus nonfood information.

Ghrelin is another gut-derived hormone implicated in both homeostatic regulation of eating as well as having contributions to learning and memory. Ghrelin is often referred to as the hunger hormone because its signaling to the hypothalamus is believed to induce hunger (Müller et al., 2015). Following training on a passive avoidance assay, rats given ghrelin administration to the cerebral ventricles (Carlini et al., 2002) or hippocampus (Carlini et al., 2004) improved memory performance in a dose-dependent fashion. Ghrelin knockout mice demonstrate impairments in a novel object recognition task but this deficit is attenuated following subcutaneous ghrelin replacement (Diano et al., 2006). Ghrelin also appears to play a role in spatial and contextual memory as ghrelin antagonists disrupt conditioned place preferences with food rewards (Chuang et al., 2011; Perello et al., 2010). Related, ghrelin administration to vHC enhances cue-potentiated feeding (i.e., initiating a meal following a food predictive cue even when sated) (Kanoski et al., 2013) and disrupting ghrelin activity reduces cue-potentiated feeding (Walker et al., 2012). At a neurobiological level, leptin knockout mice show reductions in hippocampal spinal density (Cahill et al., 2014) but peripheral ghrelin administration increases hippocampal spinal density in ghrelin deficient mice (Diano et al., 2006).

Recent work has begun to implicate ghrelin in human memory formation. Intravenous ghrelin administration increases cerebral blood flow in the hippocampus, amygdala, orbito-frontal cortex, and striatum when viewing food stimuli but not nonfood stimuli (Malik et al., 2008). Similarly, intravenous ghrelin enhances cue-food reward learning by increasing connectivity between the hippocampus and ventral striatum (Han et al., 2018). This suggests ghrelin may enhance the rewarding value of food cues in both humans and rodents or that ghrelin enhances the memorability of food relevant information. That said, these studies in humans have limitations due to their procedural indices of enhanced learning. As an example, the reported finding of intravenous ghrelin

enhancing the formation for cue-food reward learning was demonstrated by pairing an image with a food odor and then finding faster reaction time in answering a descriptive question about the image paired with food vs non food odors (e.g., whether the image is composed of straight or curvy lines). Thus, there is need to demonstrate the effects of ghrelin on the formation of food relevant memories using additional procedures and measures of memory.

While we have focused on just leptin and ghrelin as neuroendocrine determinants of memory of eating, others may be implicated as well (e.g., insulin, CCK, Glucagon-Like Peptide 1, Neuropeptide Y). There is growing evidence of this in rodents (for an exhaustive review, see Suarez et al., 2019), but little work has addressed these mechanisms in humans. Collaborations between human memory researchers and neuroendocrine specialists would be particularly fruitful in moving forward.

7. Special status of memory of eating

Are meal memories uniquely different from other memories? There are several reasons to suspect memory for eating may be particularly strong and well encoded, stored, and/or retrieved relative to memory for other non-eating behaviors.

7.1. Evolutionary arguments

Comparative analyses in non-human animals demonstrate the important role that foraging and eating behavior has had on shaping memory processes. For instance, some species of birds (e.g., Clark's Nutcrackers and Black-Capped Chickadees) have evolved remarkable mnemonic capabilities (via hippocampal enlargement and specialization) allowing them to remember the location of cached food over several months (Balda and Kamil, 1992; Feeney et al., 2009; Sherry et al., 1992; Shettleworth, 1990). Scrub jays distinguish between the location of perishable (worms) and non-perishable (peanuts) food items depending on the time between caching and retrieval (Clayton and Dickinson, 1998) which is suggestive of episodic memory (Crystal, 2010; Tulving, 2002). Evidence of episodic memory in rodents is also found when rats are tasked with remembering the location of distinct food flavors, some of which are experimentally devalued (Babb and Crystal, 2006). While comparative studies often use appetitive food outcomes to motivate animal behavior, the fact that nearly all evidence of episodic-like memory comes from animals remembering specific details about food (e.g., Babb and Crystal, 2006; Clayton and Dickinson, 1998; de Kort et al., 2005; Feeney et al., 2009; Roberts et al., 2008; Zhou and Crystal, 2009), as opposed to an aversive outcome like shock (but see, Iordanova et al., 2008), raises the possibility that episodic memory evolved to facilitate learning about how to obtain food. Under this assumption, memory is expected to be best for eating behaviors, because it is precisely what the memory system was "designed" to do.

While we can certainly use our learning and memory capabilities for a whole host of tasks (e.g., list-learning, remembering where one left their keys, etc.), we feel there is a case to be made that these are exaptations—that is, tasks that have shifted from their original evolutionary function and may or may not be currently relevant to evolutionary fitness (Buss et al., 1998; Gould and Vrba, 1982). There are several design features that suggest this. As mentioned earlier, though conditioned taste aversion and fear conditioning can both occur with a single pairing of the CS and US, conditioned taste aversion can occur even with extended gaps between the CS and US, is more resistant to extinction, and occurs earlier in development than fear conditioning. Similarly, under states of hunger and resource scarcity, drosophila were shown to down regulate specific dopaminergic neurons responsible for fear conditioning—rending this learning severely reduced while leaving appetitive conditioning intact. When these neurons were artificially activated, fear learning resumed but at a cost to the overall survival of these flies (Plaçais and Preat, 2013). Similar patterns have been found in mice who, when briefly fasted before learning, show impaired fear conditioning, and when briefly fasted before extinction, exhibit facilitated extinction (Verma et al., 2016). This suggests the resource-heavy process of fear conditioning can be temporarily "shut off" in times of starvation and that this is an evolutionarily conserved trait. The facilitated extinction by hunger suggests a trade-off between expressing fear states as well as being concerned by fearful stimuli and searching for food. Finally the types of animals that serve to benefit the most from fear conditioning, prey animals, paradoxically consume significantly more food than predators (who presumably gain less from fear conditioning) (Raubenheimer and Simpson, 1997). Thus, prey animals are likely to be highly reliant on using learning and memory mechanisms to obtain food and may even prioritize this learning over learning to avoid prey in times of hunger. Taken together, these patterns are suggestive of learning and memory capabilities having evolved, at least primarily, to aid animals in foraging.

Related to this point, recent studies show biases to the human memory system that appear to be reflective of evolutionary pressures. As an example, simply imagining oneself performing fitness relevant tasks, such as surviving in the grasslands or parenting a child, while encoding information, can result in increased retention of that information (Nairne et al., 2007; Seitz et al., 2018). There are also reported biases towards remembering potential sources of contamination (Bonin et al., 2019; Fernandes et al., 2017), future mates (Pandeirada et al., 2017), and potentially untrustworthy individuals (Hou and Liu, 2019; Kroneisen, 2018). Thus, it appears the evolutionary significance of encoded information potentiates its ability to be later recalled (Seitz et al., 2019). If true, memory for eating should be particularly well-remembered, because of its obvious relevance to survival.

7.2. Functionality arguments

As reviewed earlier, memory of recent eating plays an important role in moderating future food consumption. A popular stance among memory researchers is that the key adaptive feature of memory is its ability to generate predictions about future events (Josselyn and Tonegawa, 2020; Mullally and Maguire, 2014; Schacter et al., 2012). And yet, memory for everyday behaviors and events is generally poor (Misra et al., 2018). This may be because at the time of encoding, it is difficult to gauge the importance or future relevance of any given event/behavior. As an example, when standing in line next to an individual, one may not strongly encode aspects of their physical characteristics. As a result, if that person is later accused of committing a crime, it may be difficult to accurately report details of that person to the authorities. Memory of eating, however, is different, assuming there is some recognition (conscious or not) that encoding this eating event is of particular importance given a recollection of its details will later be used to moderate future food intake. While only speculative, this reasoning makes the same prediction as the evolutionary argument—that memory of eating should be better remembered than similar noneating behaviors. With that said, the effect of memory of recent eating on regulating future eating has been reported to wane over 3 -hs (Higgs, 2002). On one hand, this might be taken as evidence that the meal memory is not particularly strong, but on the other hand, may be evidence that temporal information regarding when the meal took place is also strongly integrated in the memory. That is, in "deciding" whether to consume a meal one might integrate information about the content, quantity, and timing of their last meal. Thus, even if one consumed a large portion of a high-quality food, if this meal occurred 5 h ago, this temporal information should be used to no longer inhibit future eating.

7.3. Evidence of superior memory for eating behavior

Seitz et al. (2021) directly tested how memory differs for eating compared to a similar non-eating behavior. Participants watched a film in front of a bowl of M&Ms and an opaque container. As they watched, a tone was randomly sounded 30 times, which cued some participants to

eat an M&M and others to move an M&M from the bowl to the container. Participants who ate the M&Ms were significantly better at remembering how many times they performed this task (reduced task memory error), despite all participants performing nearly identical procedural behaviors under identical conditions. A follow up experiment ruled out glucose provided by consuming the M&Ms as a potential physiological explanation behind this effect (c.f. Glenn et al., 2014; Smith et al., 2011). These results support the prediction that memory of eating is particularly strong, although it remains unclear if this is due to their importance in moderating future eating behavior, their evolutionary significance, or some combination of the two.

More specific mechanisms by which eating a meal becomes so well remembered is similarly, at this point, unknown. For instance, it is possible that eating is more strongly encoded than other actions—potentially via enhanced attention. It is also possible that memory of eating is more easily retrieved or less prone to interference. At present, we simply know that eating a meal is especially well remembered, and what accounts for this special status has yet to be identified. The neurological underpinnings responsible for this enhanced memory might inform on this matter and is in its own right an interesting research pursuit. The neural underpinnings responsible for calorically dense food items being better remembered than consuming the same number of a low-calorie food items is similarly intriguing (Seitz et al., 2021). These questions are especially compelling in light of the various sensory inputs that could moderate the enhanced memory of eating—because eating involves input from all five senses (Delwiche, 2012; Fantino, 1984; Havermans et al., 2010; Spence, 2015). Additionally, many foods are associated with rich memory networks (Allen, 2012). Even the smell of certain foods can bring back memories of childhood and special events. This richness in associations may enhance memory from a connectionist perspective. The diversity and complexity of different flavor and food combinations also makes memory of eating some foods less susceptible to retroactive interference. Alternatively, from an evolutionary perspective, there might be pressures to enhance the memory of eating novel compared to previously consumed food items, because novel foods could serve as pathogen vectors and cause other bodily harms (c.f., Seligman, 1970). Sensory knockouts, whole-brain imaging, and controlled behavioral studies are needed to elucidate what leads to enhanced memory of eating and enhanced memory of eating high calorie foods.

7.4. Memory of eating: Superior but still imperfect

Although the literature reviewed above indicates that meal memories are more accurate relative to non-meal memories, there is also evidence of systematic underestimation of the amount of food consumed. Studies have shown a similar bias towards underestimating the amount of food consumed immediately (~30 % in Seitz et al., 2021) and 24 h after consumption (Armstrong et al., 2000; Baxter et al., 2002; Fries et al., 1995). These data should inform an ongoing debate within nutritional and medical communities regarding the validity of self-reported dietary assessment techniques. That there is a discrepancy between self-reported and actual eating, particularly among individuals with higher BMI, has long been a concern in nutritional research (Dao et al., 2019; Lichtman et al., 1992; Macdiarmid and Blundell, 1998; Schoeller et al., 2013) but some have recently argued that self-reported energy intakes are entirely inadequate measures that should not be used in scientific studies (Archer et al., 2018; Schoeller et al., 2013). If participants are so inaccurate in recalling how much food they consumed just minutes earlier (Seitz et al., 2021), relying on memory-based measures of dietary intake is likely to result in highly unreliable findings. As memory researchers have been instrumental in advising detectives and police officers on proper techniques for interviewing witnesses and victims (Geiselman et al., 1986), we suggest they might also be useful in informing more reliable measures of reporting dietary intake by dietitians and in the study of human nutrition (e.g., Martin et al., 2012).

8. Conclusion

The study of memory is at the heart of cognitive science. While many might associate the study of memory as having connections with aging, education, neuroscience, and/or eyewitness testimony reliability, this review has shown that memory and eating are also highly intertwined. Both memory processes and eating behavior appear heavily reliant on hippocampal functions (Stevenson and Francis, 2017; Swithers et al., 2009) and are also influenced by similar neuroendocrine signals (e.g., leptin and ghrelin) (Hsu et al., 2016; Kanoski and Grill, 2017; Suarez et al., 2019). Interestingly, whereas the hippocampus has predominantly been implicated in memory and only recently implicated in regulating eating behavior and being impacted by obesity, the lateral hypothalamus has long been implicated in eating behavior and only very recently been found to be critical in learning cue-food associations (i.e., Pavlovian Conditioning) (Sharpe et al., 2017) and altered by obesity (Rossi et al., 2019). Associative learning processes have long been implicated in influencing eating behavior, particularly as it relates to taste preference and avoidance (Sclafani, 2001). More recent studies now show episodic memory processes influence eating behavior, in that episodic memory of recent eating moderates future intake (Higgs and Spetter, 2018). In animal models, dietary-induced obesity causes memory impairments (Beilharz et al., 2015) and conversely, inducing memory impairments in rodents causes obesity (Davidson et al., 2009). Similar patterns are shown in humans (Attuquayefio et al., 2017; Cheke et al., 2016; Prickett et al., 2015). While enhancing memory of eating may be a potential intervention to reduce overconsumption, little is known about the factors that influence memory of eating. It may also be the case, that memory for eating is particularly strong relative to other behaviors (Seitz et al., 2021) and yet, still an unreliable source for nutritional studies measuring dietary intake.

Throughout this review, we have speculated on areas of future research we believe to be fruitful. For instance, though much work has shown memory deficits are associated with obesity, and that memory for recent eating moderates future eating, to our knowledge, no studies have examined how participant BMI interacts with this latter pattern. Similarly, while attentional biases to food and food cues are observed in participants with obesity (Hagan et al., 2020; Werthmann et al., 2015), it remains untested whether *mnemonic* biases for these items are also observed and more or less pronounced in those with obesity. How memory of eating differs in populations with normal versus overweight and obese BMI may be particularly interesting, given serum levels of leptin and ghrelin differ in these populations (Klok et al., 2007) and are also implicated in mnemonic processes (Suarez et al., 2019). Related, while both leptin and ghrelin influence physiological states of hunger and interact with the hippocampus to improve memory formation, there has been a considerable dearth of research on how hunger states influence general memory performance and memory of eating/food stimuli. The neural underpinnings of memory of eating as well as the factors that influence these memories are still largely unknown, as are methods to improve memory of eating. Increasing memory of eating might reduce future overconsumption and also increase the reliability of self-reported dietary intake measures. As diseases of overconsumption continue to rise and as methods to study and understand mnemonic processes advance, the combination of these two seemingly distant areas should result in exciting research pursuits with relevance to both clinical and basic science.

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Appendix A. Supplementary data

Supplementary material related to this article can be found, in the online version, at doi:https://doi.org/10.1016/j.neubiorev.2021.05.024.

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