Adverse effects of consuming high fat-sugar diets on cognition: implications for understanding obesity

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Abstract

There is increasing evidence for important roles of key cognitive processes, including attention, memory and learning, in the short-term decision making around eating. There is parallel evidence that people who are overweight or obese tend to perform worse on a variety of cognitive tasks. In this review, the evidence for these two ideas is summarised and then the idea that overconsumption of Western-style high-fat high-sugar diets may underlie the association between obesity and poorer cognitive performance is explored. In particular, evidence in animals and humans that repeated consumption of high fat or high fat and sugar diets leads to specific impairments in the functioning of the hippocampus which underpin the consequent changes in cognition is summarised. These findings lead into the vicious cycle model, which suggests that these cognitive changes have knock-on negative effects for future appetite control, and evidence that altered hippocampal function is also associated with impaired appetite control is explored. The review concludes that there is consistent evidence in the animal literature and emerging evidence from human studies that supports this vicious cycle model. It is also noted, however, that to date studies lack the nutritional specificity needed to be able to translate these basic research findings into clear nutritional effects, and concludes that there is an urgent need for additional research to clarify the precise nature of the apparent effects of consuming high fat and sugar diets on cognition.

Introduction

Historically, the study of human appetite control has been dominated by theories based around the sensing of nutritional status and the use of these sensed signals to either promote a desire to eat (hunger signals) or suppress that desire (satiety signals). There is a great deal of evidence confirming that such signals play an important role in appetite control, and the underlying peripheral and neural signals that influence eating have been well characterized (1-3). However, homeostatic models are increasingly recognised as inadequate explanations of everyday eating behaviour, particularly because of the worldwide increase in incidence of obesity driven by over-consumption. The prevailing view is that short-term appetite control involves a balance between responding to homeostatic controls arising from internal nutrient-derived signals and external signals that can over-ride homeostatic control (4-6). Eating can thus be viewed as decision making around food based on a balance between immediate rewards and homeostatic needs.

Ultimately starting or stopping eating is a decision, and these decisions are informed by a wide range of factors, certainly including physiological cues arising from detection of signals arising from sensing of nutrient status (interoceptive signals), but also by external factors including the social context of eating (7, 8), the palatability of the food on offer (9-11), the served portion size (12-15), etc.. Thus understanding the cognitive processes that underlie the decision making around meal-initiation, meal-size etc. offers the potential for new insights into human appetite control, and consequently the potential to develop more effective treatment approaches for those who struggle to regulate their appetite. But our cognitive functioning is in turn influenced by nutritional status, and in this review the focus is on one such influence, the growing evidence that repeated consumption of diets with higher levels of fat and sugar (high-fat sugar diets, HFS) alter specific functioning in key areas of the brain involved in memory, and that consequently HFS diets lead to impaired cognition which in turn leads to poor decision making around eating. To date, however, the evidence for the effects of HFS diets have largely come from animal models (see 16, 17). Here the focus is primarily on growing evidence for similar effects in studies with human volunteers.

The role of cognition in appetite control

To set the potential impact of HFS diets on cognition and appetite into context, first it is important to consider the wider role of cognition in appetite control. Many cognitive processes are involved in decision making around meals. The extent to which we are aware of food cues in the environment (attention) has long been hypothesised as a key component of our sensitivity to external drivers of ingestion (18). But more recent work (19) has focussed on our ability to recall in the short-term what we have recently eaten and to adjust our subsequent eating accordingly (memory). Memory is also key to how we identify the foods we consume (20), and even modifies our perception of flavour (21). And these memories are laid down through learning processes which integrate sensory and nutrient information through experience (22, 23). Each of these four aspects of cognition are reviewed here in brief to set the broader context for the potential way HFS diet consumption may impact on cognitive controls of ingestion.

The role of memory in appetite control

The idea that memory for recent eating events was a critical determinant of the decision to start and end a meal came to the fore with Rozin's (24) seminal study of eating in two amnesic patients. These patients failed to remember they had just eaten, and so accepted and consumed a second lunch served soon after a first lunch. An elegant series of studies lead by Higgs has demonstrated that this is not down to aberrant appetite in amnesia: healthy volunteers who are directed to recall the lunch they had earlier reliably eat less in a mid-afternoon snack intake test than do controls in other recall conditions (25-28). It is also well established that people consume more when distracted than when their attention is on eating (see 19). Notably, recall of intake is poorer for food consumed under distraction (27), while focussing on eating during lunch increases memory and reduces later intake (25). Taken together, these lines of evidence provide strong evidence for an important role for memory in short-term intake regulation.

Attention to food cues as clues to underlying eating motivation

Since humans are born with no innate attraction to any specific foods beyond a tendency to like those with sweet tastes, our preferences develop through learning about the short-term effects of consuming different items (22). Once we have acquired these associations, cues which predict the presence of foods in our environment are more likely to attract attention. Consequently, the study of the extent to which we attend to food cues can give insights into underlying eating motivation. For example, it has been repeatedly shown that healthy normal-weight participants pay greater attention to food

cues when hungry than when sated (29-32). Overweight and/or obesity individuals, however, have been shown to attend more strongly to food-related cues (typically pictures of food, 33-41) than do normal-weight controls. Moreover, studies have suggested attention to food is maintained even when sated in obese individuals (36). These effects of weight-status on attention to foods are further complicated by effects of dieting and weight dissatisfaction, with individuals with a history of dieting or weight dissatisfaction also showing greater attention to food cues (42, 43). Taken together, these data suggest that cognitive systems involved in selective and sustained attention play a key role in both normal appetite control and over-eating. They also demonstrate how our individual attention to food reflects a balance of motives for eating, partly derived from homeostatic signalling, but also influenced by both the rewarding consequences of eating and weight-related concerns about the effects of eating. Thus attention may reflect the momentary integration of eating motivation derived from the processing of multiple signals.

The role of learning in development of food preferences and the control of appetite.

Attention to food cues involves recognition of external cues that map onto our individual memories. These memories are acquired through a lifetime of exposure to foods and learning of what items are safe to eat or not, and what nutritional value we obtain from consumption. There is neither scope or relevance to fully explore the mechanisms of learning underpinning the development of our food preferences, which have been reviewed elsewhere (22, 44), but it is important to remember that these learning processes are likely to be impaired by changes to the neural systems which subserve learning and memory, including the hippocampus. In particular, animal studies strongly suggest that the hippocampus plays a key role in detection and integration of interoceptive signals into learned responses (45), an idea developed more fully later in this review. It is also notable that expression of learned preferences is sensitive to acute hunger state in healthy normal weight individuals (46, 47), but these types of learning may be disrupted by dieting (48, 49). The dependence of learning on memory for interoceptive experience post-ingestion (22) also suggests that impaired memory will interfere with these learning processes, suggesting that flavour preference acquisition may be less regulated by post-ingestive experience in obesity.

Obesity, memory and cognition

The previous section highlights, in brief, the importance of three aspects of cognition in control of appetite. Critical to the present review, however, is the idea that overconsumption of HFS diets can impact on brain function and so alter cognition. The argument then follows that these nutritionally-derived changes in brain function in turn impair short-term appetite control through the consequent changes in cognition: a vicious cycle model (16, 17) described later in more detail. For such a model to have any explanatory power for human overeating there has to be evidence that people who overeat show power memory, and there is growing evidence that this is so. Firstly, there are specific cognitive impairments associated with obesity. This has been found both in cross-sectional studies (50, 51), and more crucially in longitudinal studies (52). For example, a recent study showed that performance on a novel visual episodic memory task decreased as a function of body mass index (53), with significant impairments on all aspects of this novel memory task with increasing BMI (Figure 1). It is notable that this study only measured overall BMI, and whether the reported memory impairment was more associated with weight or overall fat content cannot be determined, and warrants further investigation. Other studies report explicit memory deficits in obese populations: for example, explicit memory deficits were found in one quarter of obese participants who were about to undergo bariatric surgery, with memory improving post-surgery (54). Recovery in this context could be due to altered diet, reduced weight or reduced body fat. Further evidence for a specific memory impairment associated with obesity came from a more recent study that contrasted a variety of measures of cognitive performance, including working memory, between obese and normal weight groups (55). The outcome suggested impaired working memory associated with obesity, but also found evidence of impairments in learning, particularly for negative outcomes. Some studies have found more general impaired cognitive function or impaired executive function associated with increased body weight, BMI or obesity (56, 57) although one study found that obesity was associated with improved overall cognitive function in older adults (58). The current consensus however is that overweight and obesity tend to be associated with poorer memory performance, consistent with the idea that overconsuming HFS diets impairs memory, but not providing direct evidence for the HFS association per se.

Evidence for specific effects of diets high in fat and sugar on memory

Animal studies

The clearest evidence for an effect of consuming HFS diets and impaired memory comes from studies with non-human animals. A full review of this literature is beyond the scope of this paper, and has been subject to several recent critical reviews (16, 17). In brief, the basic design of studies in this area involves measuring different aspects of memory performance for rodents who are fed either a HFS or control diet. Using this approach, extended exposure to HFS diets has been shown to impair performance on tasks which have been implicated in hippocampal-dependent spatial memory, like the Radial Arm Maze or the Morris water bath (59-64). What is striking in these animal studies is that the impairment in memory is specific to spatial tasks, whereas other measures such as object recognition remain relatively unimpaired (65). This can be seen in Figure 2: performance (measured as exploration time) is impaired for rats placed on a HFS diet on a spatial (Figure 2A) but not object recognition (Figure 2B) task. These spatial memory deficits also emerge rapidly, with recent studies finding deficits emerging soon after being placed on the HFS diet (61, 65-67), as can be seen in Figure 2. Thus, the evidence that consuming Western-style HFS diets impair hippocampal-dependent memory tasks in animals is strong, and is further backed up by evidence of changes to the hippocampus itself, described in detail in several recent reviews (16, 17, 68, 69), but beyond the scope of this review.

Human studies

Most studies of effects of consuming a Western-style HFS diet on memory and broader cognitive performance have tended to rely on associations in cross-sectional studies. Studies of this kind have provided evidence consistent with the idea that consuming a HFS diet impairs memory, but because of the cross-sectional nature of the data these studies do not provide the same strong test of causation that is seen in the rodent literature. That said, the evidence is largely in line with what would be predicted from the animal findings. In terms of effects of high intake of saturated fat (SF), a cross-sectional study of middle-aged (45-60 year olds) using an extensive cognitive battery as part of a larger cohort study found an association between SF intake and increased risk of impaired cognitive functions, including memory, speed and flexibility (70). Notably, that study was conducted in the context of examining potential beneficial

effects of intake of omega 3 fatty acids, an issue discussed in greater detail later. A similar study which examined effects of intake of different types of fat on cognitive performance in an older cohort (65 years +) also reported greater cognitive decline over time in participants with higher SF intake (71). These large cohort data are further supported by a more recent but smaller study contrasting effects of fat intake on memory in younger women (72). Again, habitual fat intake was associated with specific memory impairments (poorer word recall and recognition, impaired performance on a visuo-spatial task).

More in line with the animal approach was a study which contrasted cognitive performance between individuals classified as having "whole food" or "processed food" diets based on analysis of 4,693 participants from the Whitehall cohort study (73). The processed food group had increased risk of cognitive deficit, including poorer memory, whereas the whole food group had reduced risk, although controlling for potential confounders (socioeconomic status, education etc.) reduced the significance. In a different approach, Francis and Stevenson (74) developed a modified food frequency questionnaire to estimate HFS diet intake, and then examined how performance on standardised memory tasks (verbal paired associates, VPA, and the logical memory subtests from the Wechsler Memory Scale, LMWMS) varied as a function of HFS. The Dietary Fat and Free Sugar Questionnaire (DFSQ) assesses frequency of consumption of 26 foods and drinks that are high in saturated fat and/or added sugar, and has been shown to be a fairly reliable measure of actual intake of high-fat and sweetened products (75). In line with animal data, higher HFS intake was associated with poorer performance on these memory tasks, but not on other tasks (the Trail Making Test and the Wisconsin Card Sort Test). Since both VPA and LMWMS tasks have been shown to be impaired in patients with hippocampal damage, whereas the other tasks measure frontal executive function, these associations provide support for the idea that HFS intake is associated with impaired memory through an impact on the hippocampus, although they are again based on an association rather than a clear causal test. These findings were replicated in a follow-up study (76), again with higher HFS intake correlating with poorer performance on the LMWMS. A more recent study by the same group provided further evidence of effects of HFS on memory (77). Here, healthy volunteers who varied in their habitual HFS consumption measured using the DFSQ completed the verbal paired associate (VPA) test, which is well established to

involve the hippocampus. In line with previous findings, performance on the VPA decreased as a function of HFS score. Thus there is growing body of evidence from cross-sectional studies relating HFS diets to impaired memory. The idea that HFS diets directly impact on hippocampal function was further supported by the finding that a standardized measure of consumption of a Western-style diet was associated with decreased overall volume of the left hippocampus in a sample of 255 middle-aged men (78). However, all of the studies discussed so far report associations between HFS or Western-style diets and memory or hippocampal function: thus these studies are consistent with the broader idea that these diets impair hippocampal function but cannot establish causation.

The most convincing test for the theory that intake of HFS diets impairs hippocampal function would be to examine changes in hippocampal sensitive memory tasks before and after an intervention where the proportion of HFS in the diet was artificially increased or decreased. Such tests raise obvious ethical issues, and there have been few studies which have attempted to do so to date. One study that used this approach (79) did find impaired cognitive function (increased reaction time and impaired attention) after placing 20 healthy volunteers on a high fat diet for seven days, although they did not include any specific hippocampal-dependent memory tests. Placing healthy young men on a high-fat, low-carbohydrate diet for five days also impaired attention, but also showed a deterioration in speed of retrieval from short-term memory (80). Thus both studies suggest similarly acute effects of consuming high fat diets on cognitive performance that has been reported in animal studies, although some caution is needed in interpretation since both studies were small.

Overall current human data is consistent with the broader idea that repeated consumption of Western-style HFS diets may impair cognitive function, possibly through a particular action on the hippocampus, but more studies are needed to fully characterise these effects and establish greater specificity and causation.

The vicious cycle model

The argument that changes in function of the hippocampus brought about by habitual over-consumption of Western-style HFS diets promotes unhealthy diet choice has been encapsulated in the Vicious Cycle mode (VCM) developed by Davidson and colleagues

(16, 81-83). The essential argument underlying the VCM (Figure 3) is that the hippocampus plays a key role in the decisions to start and end meals. Feeding on HFS diets is then hypothesised to promote overconsumption and consequent weight gain, and also leads to alterations in hippocampal function. It is then argued that these changes in the hippocampus disrupt normal controls of ingestion leading, so promoting continued over-consumption, leading to a vicious cycle of further overeating. The implication is that once an individual has overconsumed sufficient HFS diet to impair hippocampal functioning, the resulting damage to the hippocampus and consequent alterations in cognitive functions make it harder to make the necessary changes in behaviour to reduce intake and adopt a healthier diet. Critical to the VCM is the idea that the hippocampus plays a key role in control of normal food intake (82, 84-88) as well as its more widely known role in memory (89-91). The evidence for a role of the hippocampus in control of feeding comes largely from animal studies. For example, rats with lesions to the hippocampus took smaller more frequent meals than do normal rats (92), while temporary inactivation of the hippocampus also reduced the intermeal interval (93). But the evidence reviewed earlier of a key role of short-term memory in normal control of appetite in humans is consistent with this. What is now needed is a critical evaluation of the extent to which the key changes in behaviour proposed by the VCM, and evidenced in animals, is also seen in studies with humans.

The vicious cycle model: evidence from studies of human eating behaviour

The evidence of impaired memory associated with HFS diet consumption reviewed earlier implicates the hippocampus in short-term memory impairment. However, the VCM suggests that these apparent changes to hippocampal function then alters key aspects of appetite control. Of these, two aspects have been examined in recent studies: decreased sensitivity to interoceptive cues and reduced inhibitory control. While current evidence is still limited, these recent studies provide some support for what is proposed in the VCM.

The first aspect of the VCM that has been looked at is the extent to which the effects of HFS on memory also impacts on awareness of interoceptive cues associated with eating. Studies in animals have shown a critical role for the hippocampus in integrating the use of interoceptive cues in control of feeding (94, 95). In humans, high scores on the DFS in a normal-weight population were associated with reduced sensitivity to

internal hunger/satiety cues, evidenced by altered effects of ingestion on the experience of appetite in those with high HFS intake (74). Similarly, DFS scores were associated with smaller reductions in ratings of incentive but not hedonic motivations to eat in a state of satiety (77). These measures were derived from ratings of food liking and desire to eat at the start and end of lunch: both ratings declined overall, but the rate of decrease for the desire to eat measure was markedly slower for those with high HFS intake. These results are further supported in a study which tested the extent to which habitual HFS intake in a healthy normal-weight population were related to sensitivity to thirst-related interoceptive cues (76). Here, changes in thirst sensations after a thirst-inducing challenge (eating salty crisps) decreased as a function of increased HFS intake, interpreted as further evidence of poorer interoceptive awareness in HFS consumers.

A second key prediction from the VCM is that people who habitually consume excessive amounts of HFS diets will also display poorer impulse control. In parallel to the development of the VCM here has been a large increase in interest in the role of impulsivity as a risk factor for development of obesity, based on the idea that the risky decision making and emphasis on short-term rewards which are key features of impulsivity could contribute to the poor dietary choices that underlie overeating and HFS consumption. There is now a wealth of studies reporting that higher measures of impulsivity are associated with obesity (96-100)and uncontrolled eating (101). None of those studies specifically assessed habitual HFS intake, however a recent study reported an association between HFS intake measured using the DFSQ (102). They reported that HFS intake was associated with higher scores on two standardised measures of impulsivity: overall scores on the Barratt Impulsivity Scale and the Urgency measure from the Urgency, Premeditation, Perseverance and Sensation Seeking questionnaire.

The VCM also has other testable implications for human ingestion which remain largely unexplored. There is now considerable scope for new research to clarify the full extent to which habitual dietary patterns around HFS consumption do cause long-term changes in cognitive function which in turn makes it harder to self-regulate appetite and food choice.

A brief word on mechanism

How then might excessive intake of HFS diets alter hippocampal function? Based on extensive research in animals, evidence of three potential mechanisms has emerged and these have been reviewed in detail elsewhere (16, 17, 68). A number of potential mechanisms have been identified, and here the three where there appears to strongest evidence is briefly summarised: neuroinflammation, effects on the blood-brain barrier and altered levels of Brain Derived Neurotrophic Factor (BDNF).

The idea that specific responses to diet can lead to an inflammatory response is well established: inflammatory responses, particularly chronic low-grade inflammation, have been implicated in many diet-related diseases, including heart disease (e.g. 103, 104, 105), diabetes (see 106, 107) and depression (e.g. 108). Many studies have found higher levels of inflammatory markers in obese than normal weight participants (see 109, 110, 111). It has also been argued, with considerable evidence, that inflammation may be a key link between obesity and diabetes (112), and that this damage in turn alters subsequent food intake regulation (16, 113). In the present context, the key findings come principally from studies with animals that show increased levels of inflammatory markers for rats placed on HFS diets (see 63), and particularly evidence of specific neuroinflammatory responses in the hippocampus (114-116). Thus diet-induced inflammation provides a potential explanation for the specific impact of HF and HFS diets on hippocampal-dependent cognitive processes.

One of the potential explanations for how neuroinflammation alters hippocampal function might be through changes in levels of BDNF. BDNF acts to support the differentiation of neurons, and crucially is abundant in the hippocampus. BDNF plays a key role in long-term memory (see 117, 118), and so any changes in BDNF are likely to also affect memory and so appear a prime candidate as a link between the diet-induced changes in hippocampal function and memory highlighted in this review. There is now considerable evidence for alterations in hippocampal BDNF in animals fed HF or HFS diets (see 16). For example, rats consuming a HFS diet had reduced BDNF in the ventral hippocampus, alongside hippocampal-dependent changes in cognitive performance (119). Other studies also found reduced hippocampal BDNF in animals consuming HF or HFS diets (59, 120). Together, these studies clearly implicate changes in BDNF in diet-induced hippocampal dysfunction, although what causes the changes in BDNF needs to be clarified.

The selectivity of the semipermeable blood-brain barrier separates the brain from circulating blood, so protecting the brain from circulating pathogens. It is then surprising that the effectiveness of the blood-brain barrier can be affected by diet. For example, mid-life BMI predicted the integrity of the blood-brain barrier 24 years later, with higher BMI associated with less effective blood-brain barriers in these women (121). That study does not directly implicate diet, although higher BMI would be expected to relate to higher intake. Animal studies have however reported specific deficits in the blood-brain barrier for rats maintained on HFS (122) or HF high-cholesterol (123) diets. Thus repeated consumption of these types of diet do appear to impair the effectiveness of the blood-brain barrier.

So far there is evidence for three potential explanations for the effects of Western-style HF or HFS diets on hippocampal function: whether these act independently or are related (for example reduced effectiveness of the blood brain barrier contributing to local inflammation which might reduce BDNF production) remains unknown. But they do offer plausible and testable mechanisms that could underpin the cognitive deficits.

The cause of hippocampal changes: diets, nutrients or adiposity?

A weakness in this research area to date is the poor specification of what dietary changes are involved. Animal studies use diets with high levels of added (typically saturated) fat and/or sugar: whether it is the effects of overconsuming the enhanced elements of these diets or consequent dilution of other features of the diet that are key to the observed effects needs to be clarified. Of particular relevance is the nature of the fats in the diet, since there is evidence that omega 3 fats in particular may act to reduce inflammation (e.g. 124, 125), and increasing saturated fat content of a diet may act to alter the proportion of omega 3 indirectly, for example. Thus there is urgent need to examine the specificity of the dietary changes observed in the animal studies that largely underpin this research area. A further issue in interpretation of the animal studies is that these diets also lead to increased body-weight, and determining whether the inflammatory responses etc. are then a direct consequence of the diet or adiposity cannot be easily determined.

Human studies to date are essentially based on the relative proportion of HFS foods in an individual's diet estimated from self-report food frequency measures. That replicable behavioural findings emerge from this simple measure implies that more highly specified dietary analyses may be able to identify more specifically what dietary features are critical. A key feature of the main human studies (74, 76, 77), however, is that they are able to dissociate the effects of diet from the potential confound of adiposity, since all of these studies were in normal-weight volunteers and the critical relationships between behavioural measures and HFS intake were still evident once BMI had been controlled for.

Future directions

This short review highlights the current state of knowledge of the potential impact of Western diets on cognition and consequently on appetite. But it also highlights major shortcomings in our current understanding. Most critical is the lack of dietary specificity in the studies underpinning research findings in this area to date. There is now a real opportunity to data-mine existing longitudinal studies which hold nutritional and cognitive measures to test the hypotheses generated from the current findings in a more representative large population. Such analyses could potentially dissociate more fully effects of diet per se from weight gain, and identify whether it is excess of certain foodtypes or nutrients that are the key risks for cognitive impairment. There is also a need to test in humans more of the predictions from the VCM: what is the impact of HFS consumption on food-related reward and attention, for example. The ideas and hypotheses in this area are intriguing, and have the potential to make major advances in our understanding of causes and consequences of overeating, and warrant urgent further investigation.

Concluding remarks

This brief review summarised the importance of cognitive factors in human appetite control and then speculated how changes in a core brain area associated with cognition, and particularly memory, may be disrupted by habitual overconsumption of energy-dense HFS diets. The animal research in this area is persuasive in terms of the specificity of the effects on the hippocampus, but is less impressive in considering the nutritional specification of the diets that have these effects. Human research in this area

is emerging, and is consistent with the broad findings from the animal models, however. The overall conclusion would be that excessive intake of readily metabolized nutrients may lead to a sub-clinical inflammatory response which in turn may impair performance of key areas of the brain. The consequent changes in cognition in turn impair the ability to engage with the healthy lifestyle needed to reverse the effects. Research is now needed to test these ideas more broadly, and consider how these ideas may be valuable in novel approaches to countering the obesity crisis.

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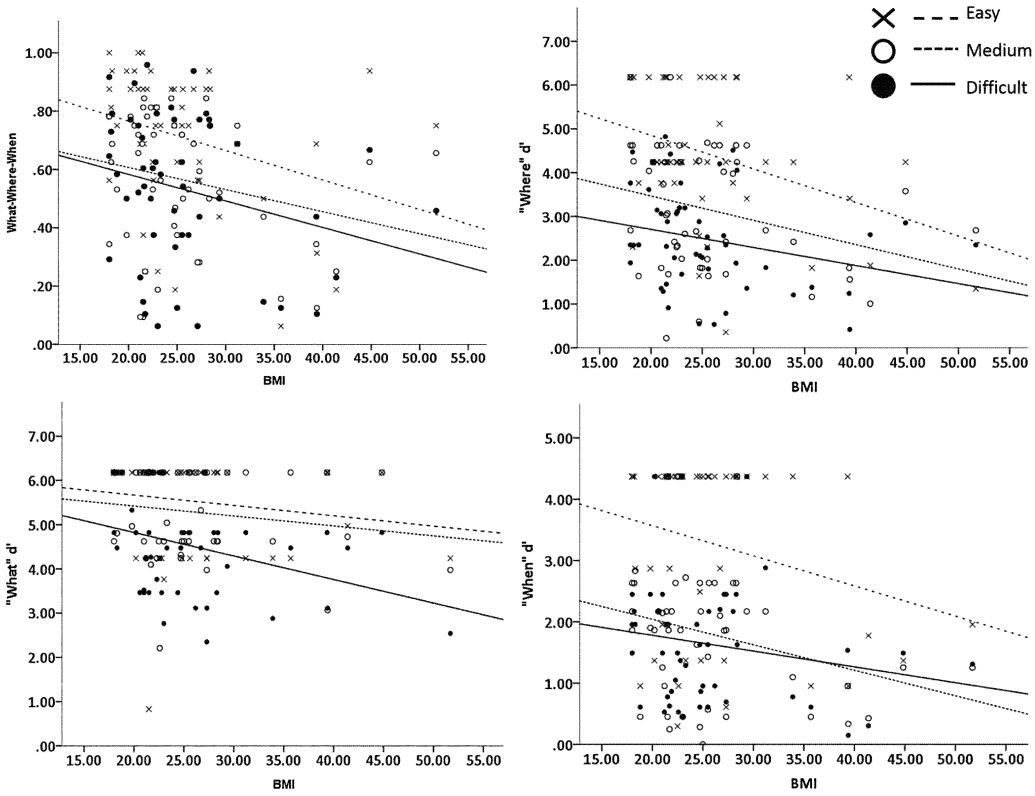
Figure legends

Figure 1. Associations between BMI and performance on a novel spatial test of episodic memory (the what where when task), redrawn from Cheke, Simons (53), with permission.

Figure 2. Performance on (A) a spatial (place recognition) and (B) non-spatial (object recognition) task for rats fed a chow () or high-fat-high-sugar diet (). Higher exploration proportion values reflect better memory. Figure adapted from data in (65) with permission.

Figure 3. A schematic summary of the Vicious Cycle model proposed by Davidson and colleagues, adapted from (17).


Easy
Medium
Difficult
What-Where-When
"Where" d'
"What" d'
"When" d'
BMI

766

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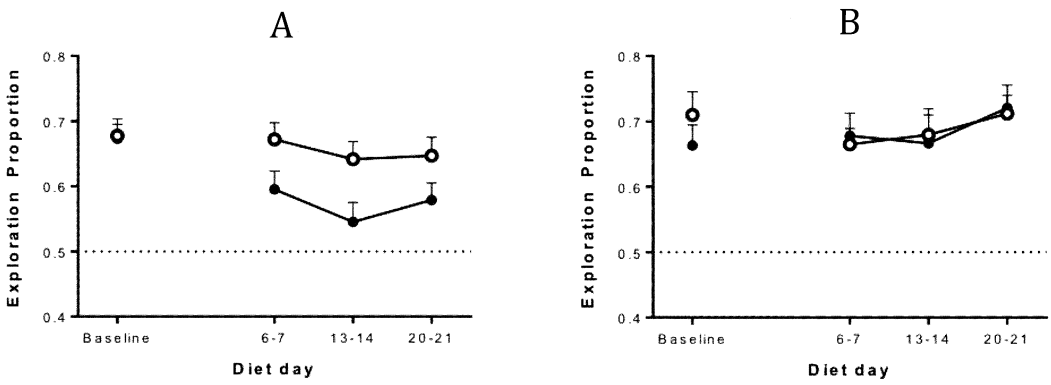
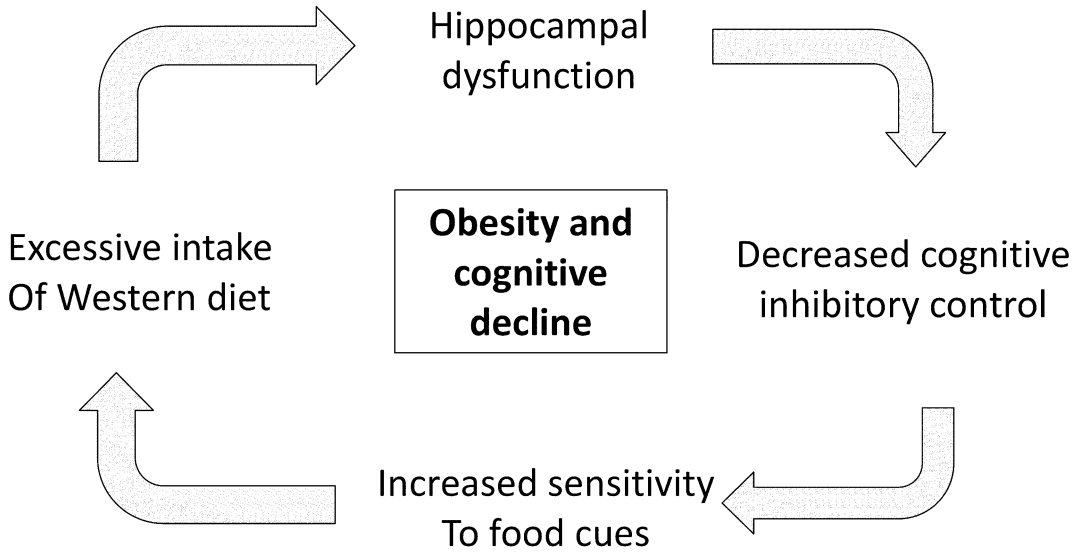

A
B
Exploration Proportion
0.8
0.7
0.6
0.5
0.4
Baseline
6-7
13-14
20-21
Diet day

768

769


Hippocampal dysfunction
Decreased cognitive inhibitory control
Increased sensitivity To food cues
Excessive intake Of Western diet
Obesity and cognitive decline

770