Physiology & Behavior 136 (2014) 185–193

Contents lists available at ScienceDirect

Physiology & Behavior

journal homepage: www.elsevier.com/locate/phb

Human cognitive function and the obesogenic environment

Ashley A. Martin [a,*], Terry L. Davidson [b]

[a] School of Experimental Psychology, Nutrition and Behaviour Unit, University of Bristol, Bristol, UK
[b] Department of Psychology and Center for Behavioral Neuroscience, American University, Washington, DC, United States

HIGHLIGHTS

- Obesity is associated with cognitive impairment both in early and late life.
- Certain cognitive impairments may reduce the inhibitory control of food intake.
- These cognitive impairments can develop as a result of eating a Western diet (WD).
- The "obesogenic" environment may be a product of WD-induced cognitive impairment.

ARTICLE INFO

Article history:
Received 14 December 2013
Received in revised form 17 February 2014
Accepted 21 February 2014
Available online 11 March 2014

Keywords:
Obesity
Western diet
Memory
Hippocampus
Inhibition

ABSTRACT

Evidence is accumulating which suggests that, in addition to leading to unprecedented rates of obesity, the current food environment is contributing to the development of cognitive impairment and dementia. Recent experimental research indicates that many of the cognitive deficits associated with obesity involve fundamental inhibitory processes that have important roles in the control of food intake, implicating these cognitive impairments as a risk factor for weight gain. Here, we review experiments that link obesity with deficits in memory, attentional, and behavioral control and contemplate how these deficits may predispose individuals to overeat. Specifically, we discuss how deficits in inhibitory control may reduce one's ability to resist eating when confronted with the variety of foods and food cues that are ubiquitous in today's environment. Special attention is given to the importance of memory inhibition to the control of eating and appetitive behavior, and the role of the hippocampus in this process. We also discuss the potential etiology of both obesity and obesity-related cognitive impairment, highlighting non-human animal research which links both of these effects to the consumption of the modern "Western" diet that is high in saturated fats and simple carbohydrates. We conclude that part of what makes the current food environment "obesogenic" is the increased presence of food cues and the increased consumption of a diet which compromises our ability to resist those cues. Improving control over food-related cognitive processing may be useful not only for combating the obesity epidemic but also for minimizing the risk of serious cognitive disorder later in life.

1. Introduction

The current food environment in Western and westernized societies is characterized by the widespread availability of low cost, energy-dense, highly-palatable, foods and beverages, and an abundance of external cues that keep thoughts of these foods and beverages almost constantly in mind. It has often been claimed that this combination of factors has helped to create an "obesogenic" environment that overwhelms the physiological controls that normally maintain energy balance and body weight (e.g., [1,2]). Along with excess energy intake and body weight gain, this environment is associated with increased incidence of Type II diabetes (T2DM), cardiovascular disease, hypertension, depression, and certain types of cancer (e.g., [3]).

Accumulating evidence suggests that cognitive dysfunction should also be prominent on the list of the adverse health consequences of living in an obesogenic environment. A number of recent epidemiological studies have reported links between obesity and cognitive dysfunction [4,5]. In addition, well-controlled non-human animal studies indicate that consuming an energy-rich "Western"-style diet (WD) that is high in sugar and saturated fat can promote not only obesity but also impairments in specific types of cognitive functions. Such WD-induced impairments are accompanied by signs of pathology in the brain substrates for those functions. Furthermore, animals that are most sensitive to this diet-induced obesity are also most sensitive to the effects of that diet on brain pathophysiology and learning and memory function. These findings are discussed in recent reviews [7–9].

* Corresponding author at: School of Experimental Psychology, University of Bristol, 12a Priory Road, Bristol BS8 1TU, UK.
E-mail address: ashley.martin@bristol.ac.uk (A.A. Martin).

http://dx.doi.org/10.1016/j.physbeh.2014.02.062
0031-9384/

The purpose of the present paper is to (a) summarize findings that link excess energy intake and body weight to cognitive dysfunction in humans; (b) describe the types of cognitive processes that might be involved with energy and body weight regulation; and (c) discuss how impairments in these cognitive processes could contribute to the capacity of the obesogenic environment to promote overeating and weight gain.

2. Links between excess intake, body weight, and cognitive dysfunction across the lifespan

There is concern that the obesity "epidemic" heralds a coming epidemic of Alzheimer's Disease (AD) and other dementias. Being obese or overweight in mid-life is associated with higher incidence of cognitive dementia in old age, independent of T2DM and cardiovascular-related co-morbidities [10]. Furthermore, it appears that people with high levels of central adiposity (i.e., excess accumulation of abdominal body fat) in middle-age are at increased risk of dementia in late-life, even if they possess an otherwise normal body weight [11].

There is also evidence that links being overweight or obese to cognitive decline not only during old-age but across the lifespan. For example, BMI and body adiposity were found to be negatively related to academic achievement and response inhibition in children 7–9 years old [12,13]. In another study [14] which compared obese and lean 12-year-old boys, obesity was associated with reduced attention endurance and increased perseverative errors in a test of set-shifting abilities, the Wisconsin Card Sorting Task. Similar results were reported by Verdejo-Garcia et al. [15], who found that obese adolescents aged 13–16 years performed worse than normal weight adolescents on indexes of inhibition, cognitive flexibility, and decision-making. Based on the results of these and other studies, it appears that excessive body weight is associated with deficits in some types of cognitive capabilities and that these deficits are not restricted to late-life but are present even in children and adolescents.

Other evidence suggests that both cognitive dementia and milder cognitive deficits are also associated with intake of Western diet. For example, Gustaw-Rothenberg [16] reported that the dietary pattern of Polish patients with AD was characterized by a high intake of meat, butter, high-fat dairy products, eggs, and refined sugar, whereas the dietary pattern of non-demented age-matched controls was characterized by a high intake of grains and vegetables. Furthermore, according to Eskelinen et al. [17] consuming a diet containing a high compared to a low level of saturated fat (a main component of the Western diet) at midlife was associated with an increased risk of mild cognitive impairment in late adulthood.

Similar links between WD consumption and cognitive impairment have been observed in younger adults and children. Cohen et al. [18] reported that adults aged 50–69 exhibited fewer perseverative errors and better attention/concentration and processing speed on tests of cognitive function if they were lean than obese. Notably, the lean group also self-reported consistently consuming more "high quality" food (defined as farm produce, fish, whole grains, and nuts) and less "low quality" food (defined as meats, refined carbohydrates, fried food, fast food, junk food, and alcohol). Similarly, Jasinska et al. [19] found that weaker control over attention and motor responses was associated with higher preference for "junk foods" (potato chips, nachos, candy bars) and lower preference for "healthy" snacks (apples, bananas, carrots) in a cohort of college undergraduates. In a study of 4th graders (ages ~9–10 years), Riggs et al. [20] found that greater self-reported snack food intake and less consumption of fruits and vegetables were associated with poorer performance on an index of cognitive functioning that was comprised of measures of inhibitory control, emotional control, working memory, and planning/organizational ability (separate scores for each subscale were not presented).

Riggs et al. suggested that the relationship between increased intake of snack food and reduced cognitive functioning might be bi-directional. The possibility of a bi-directional relationship was also suggested by Guxens et al. [21], who reported that for children 4 years of age, higher scores on a test of general cognitive function were associated with less risk for becoming overweight by age 6. Thus, deficits in certain types of cognitive functions may not only be a consequence of obesity and consumption of the Western diet, but they may also *promote* excessive weight gain and caloric intake, potentially creating a detrimental developmental cascade. This possibility is discussed in detail later on in this paper, when we identify the types of cognitive impairments that are most likely to influence food intake control and review the evidence linking each of these deficits to overeating and obesity.

3. The obesogenic environment and progressive cognitive decline

Our hypothesis is that the same dietary factors that lead to obesity (e.g., consumption of a high carbohydrate/high fat diet) also impair cognitive functioning, and that these cognitive deficits have the potential to compromise energy and weight regulation. Viewed this way, the same environmental risk factors for obesity (e.g., increased availability of low cost, high-calorie food) can also be viewed as risk factors for cognitive decline in that they encourage consumption of a Western diet. One of the implications of this view is that childhood obesity and/or consumption of obesogenic diets may have lasting consequences on cognitive functioning in adulthood. While these impairments are likely to be subtle early in life, there are reasons to suspect that when combined with the normal aging process these deficits, and the pathologies that underlie them, may become increasingly serious. Consistent with this possibility are recent attempts to trace the progression over time of brain pathologies that are thought to lead first to mild cognitive impairment, then to full-blown AD. Several analyses of this progression agree that the first signs of brain disease can occur at least 50 years prior to the emergence of serious cognitive dysfunction [22]. These signs originate in the hippocampal formation, an area in the medial temporal lobe of the brain, which is comprised of four subregions: the dentate gyrus, the hippocampus proper (i.e., the CA1, CA2, and CA3 subfields), the subicular complex (i.e., the subiculum, presubiculum, and parasubiculum), and the entorhinal cortex [23,24]. Over time, these pathological changes spread into the orbitofrontal cortex, striatum, lateral hypothalamus and other areas that are interconnected with the hippocampus [25,26].

Consistent with the possibility that early life, diet-induced brain pathologies may have detrimental effects on cognition later in life, consumption of a WD has been shown to have adverse effects on the hippocampus and has also been identified as a potential contributor to the pathogenesis of AD. Specifically, research from several laboratories shows that intake of WD in nonhuman animals reduced hippocampal neurogenesis, increased hippocampal inflammation, and increased permeability of the blood–brain barrier (BBB) leading to the accumulation of exogenous substances in the hippocampus [27,28,140]. At the same time, independent research has provided evidence that reduced hippocampal neurogenesis (e.g., [29,30]), increased brain inflammation [31,32], and increased BBB permeability [33–35] are at least as important as amyloidosis in the pathogenesis of AD (e.g., [36,37]). In the following section, we describe the types of cognitive impairments that might occur as a result of these pathologies and describe how these deficits could contribute to energy and body weight dysregulation.

4. Cognitive and behavioral inhibitory processes and energy regulation

It is well known that cues such as the sight, smell, and even thought of palatable food can induce appetite and promote eating (for recent reviews, see [38,39]). Thus, in order to lose weight or prevent weight gain, one must resist the urge to eat when confronted with these food cues. This ability to resist involves both behavioral and cognitive control. That is, in addition to preventing oneself from engaging in the physical act of eating, one must also refrain from thinking about the positive