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Neuroscience of Reward: Implications for Food Addiction and Nutrition Policy

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Abstract

According to the World Health Organization, unhealthy diet is one key contributor to the development of non-communicable diseases. The global response to this problem has primarily involved the implementation of nutritional policies intended on raising public awareness, and providing information through nutritional guidelines and product labels. However, there is experimental evidence suggesting that certain foods may promote addictive processes and consequent unhealthy dietary choices. This review discusses neurobiological mechanisms of reward involved in the consumption of refined sugars and fats, and the aforementioned indicators of their addictive characteristics. By acknowledging that these foods can act on brain reward systems to promote excessive or addictive consumption, policy makers may need to address issues of unhealthy diets by considering approaches that target availability, regulations within the food industry, taxation and advertising.

Keywords: Reward; Dopamine; Addiction; Sugar; Conditioning

Introduction

Non-communicable diseases (NCD) are the leading cause of global mortality, accounting for 63% of deaths world-wide in 2008 alone [1]. Of these diseases, the top four causes are preventable lifestyle choices including: alcohol abuse, tobacco use, inactivity, and unhealthy diets [2]. The current review focuses specifically on the overconsumption of refined sugars and fats [3], because of links to diabetes, obesity, and other metabolic diseases [2].

The global response to this problem has primarily involved implementing nutritional policies intended on raising awareness, and providing consumers information through nutritional guidelines and product labels. However, a growing body of research suggests that these regulatory strategies may not be completely effective.

Indeed, there is evidence that certain components of processed food may activate addictive processes. For example, there are a number of neurobiological similarities between behaviours reinforced by these foods and those reinforced by addictive drugs in laboratory animals [4-6]. Moreover, in clinical studies, the Yale Food Addiction Scale (YFAS) identified common neural correlates of food and substance dependence [7,8]. Finally, conditioning has been found to play a central role in excessive seeking and taking of food and drugs in both humans and animals [6,9]. This said, the "food addiction" hypothesis is often criticized for the lack of a clear definition and generally inconsistent findings [10]. Critics also suggest that food addiction may not add significant diagnostic capability beyond conditions like Binge Eating Disorder (BED) or Bulimia, nor does it adequately explain obesity. On the basis of these arguments, it can be concluded that the evidence is not convincing enough to supersede the adverse social and economic impacts of considering food addiction as a diagnosable condition.

But, rather than dismissing this relatively young field of study, examining the validity of the constructs on which it has been proposed may be beneficial. Perhaps, food addiction does not adequately explain obesity or BED, but associated health risks are not confined to these demographics either [4]. In particular, emphasis on the overweight and obese demographics has led to the misconception that perhaps addictive-like feeding behaviour leads to obesity, but this is not a universal truth [11]. Another important consideration involves employing criteria from established models of substance dependence to examine food addiction. Significant variability in diagnostic terminology, neurobiological mechanisms, and individual vulnerability exists among drugs with known abuse potential [10,12]. These issues further complicate the discussion of the addictive potential of food.

Nevertheless, it is important to recognize that for some people, in some environments, certain foods can activate addictive processes and promote unhealthy dietary choices. Acknowledging this may help policy makers learn from other addictive substances, such as alcohol and tobacco, which have transitioned from readily available to restricted through implementation of effective policies such as limiting the prevalence of associated cues, increase taxation, and/or subsidizing on particular products, or regulating availability [13]. It is anticipated that, if applied to refined sugars, these strategies will give people a better chance to follow sound nutritional advice already being promoted.

Society and Food

Unhealthy diet is a leading risk factor for developing NCDs, but what constitutes an unhealthy diet? Highly processed and refined ingredients found in fast food and ready-made to eat food are notable examples. Processing food involves the extraction or refinement of whole foods, which then generally become energy dense and nutrient deficient [14]. As a result, these foods tend to be high in fat, sugar and salt [15].

It is clear that not all fats are bad; guidelines recommend dietary levels of omega 3 and 6 fatty acids of 1-2% daily caloric intake to avoid serious deficiency [16]. Unfortunately, processed food is essentially devoid of these essential fatty acids, while retaining high levels of saturated fats and cholesterol [15]. Various health agencies, including the United States Department of Agriculture and American Heart Association, recommend that total fat intake should be 30% or less of total caloric intake, saturated fat less than 10% and cholesterol less than 300 mg/day [16], as these fats have been linked to hypertension and cardiovascular disease [2].

As well, high-fructose corn syrup, a refined sugar produced by chemical processing of corn, is one of the most commonly used food additives today. Although it is employed in many processed foods, its use in soft drinks has been suggested to have the biggest impact on health [17]. There is a significant relationship between increased consumption of sugar-sweetened beverages and weight gain [18,19], metabolic syndrome, and hypertension [20]. As illustrated below, current trends in consumption patterns make a compelling argument for the risks associated with excessive consumption of these foods.

Consumption trends and health implications

Over the past 50 years, there has been a significant global increased consumption of processed food accompanied by a decrease in the use of raw/ unprocessed culinary ingredients. Using six separate household food budget surveys, Moubarac and colleagues documented consumption trends in Canada by examining the caloric contribution of various foods (unprocessed or minimally processed foods, processed culinary ingredients, and ready-to-serve processed or ultra-processed foods) to the total dietary energy availability (kcal per capita) [15]. Their results appear staggering. Between 1938 and 2011, the proportion contributed by unprocessed or minimally processed foods dropped from 37% to 13%, while ultra-processed ready-to-serve food increased from 29% to 62%. A report by Popkin indicated that this transition is in fact a global issue and suggests that fast food and soft drink industries as primary contributors to this phenomenon [17].

Consumption rates of refined sugar are also cause for concern. The WHO has recently lowered the recommended limit of added sugar from 10% to 5% of daily caloric intake [21]. But, a report by Langlois and Garriguet revealed that Canadians are eating four times this amount, consuming enough sugar to account for 21% of daily energy intake [22]. A major contributor in this regard is the soft drink industry. Per capita consumption of soft drinks in the US has increased by 500% in the past 50 years according to the USDA [23].

Overall, the relationship between recent dietary and health trends supports the consequences of unhealthy eating behaviour. Both adult and childhood obesity have been increasing worldwide. The Center for Disease Control reported that prevalence of obesity prior to 1990 did not exceed 15% in any state, but by 2010 over a dozen states exceed 30% with several others reaching 25% [24]. This has drawn considerable attention due to the myriad of health risks exacerbated by obesity [25]. Thus, prevalence of type 2 diabetes has been rising in the general population [26], as well as in children, a condition that was relatively uncommon in youth until the early 1990's [27]. Moreover, according to the public health agency of Canada, between 1998 and 2008 the prevalence of diabetes rose from 3.3% to 5.6 % [28]. Overall, healthcare costs associated with obesity have risen by almost $60 billion between 1998 and 2008 [29]. These numbers prompted the prediction of an unprecedented decline in life expectancy in the US over the 21st century [30].

Current policy targets

Current trends of poor diet related health consequences have gained significant attention on the global stage and steps have been taken to address the issue. Examples of current strategies include: the global strategy on diet, physical activity and health (2004) and the population-based prevention strategies for childhood obesity (2009), among others. These initiatives raise awareness and mandate that producers to provide nutritional information required for people to make informed dietary decisions. Most notable are those promoting dietary guidelines and nutritional labelling, which informs the public about the contents of food and what constitutes a healthy diet.

There is some evidence that these policies are successfully raising awareness. Over the past 25 years, more shoppers reported their purchases were strongly influenced by health concerns [31]. As well, market research reported a 57% increase of annual spending from 1996 to 2006 on weight-loss programs and related products in the US [32]. Unfortunately, public awareness does not ensure that people will make healthy choices. Current nutritional policies rely on the individual's ability to self-regulate their intake of processed food but fail to consider biological and environmental influences that may compromise self-control.

Dangerous Combination

Industry response to nutrition policy

One fundamental difficulty lies with the apparent variability of industry response to nutrition policies. Private food companies probably face government pressure to transition to healthier options, and they may therefore implement policies of self-regulation such as reducing marketing to children, reducing unhealthy products in schools, and implementing responsible package labelling [33]. But, it cannot be overlooked that food has become a commodity, which means the industry must be profitable [14]. Therefore, private companies continue the sale of highly processed foods and simply offer healthier options [14]. These companies claim that there is nothing inherently wrong with the sale of processed foods in moderate quantities [14]. The core of this argument thus places the responsibility on each individual to be mindful of nutritional advices and limit consumption of unhealthy food through self-control.

While the onus rests firmly on the individual, it appears that major corporations continue to generate profit by promoting unhealthy products. Coca Cola sponsored the 5th International Congress on Physical Activity and Public Health, and they are also the official drink of the Olympics [34]. Another prominent example is McDonalds, who also sponsors the Olympics. McDonalds has started offering healthier options, such as salads and veggie wraps; however, a survey conducted by Dumanovsky et al. reported that the most popular meal purchased at McDonalds in the US is a Big Mac, fries and a medium regular soda, which contains 95 g of sugar and 1360 calories [27,35]. Other companies that are attempting a transition to healthy products are experiencing some difficulties. For example, a report on vendors in public recreation facilities in British Columbia found that those selling healthier foods were losing customers to vendors selling more palatable foods [36].

Food addiction: classic substance dependence?

Promoting nutrition awareness and relying on individual self-control may not be adequate strategies for addressing unhealthy eating behaviour [13,37]. This is because there are striking behavioural and neurobiological similarities between chronic exposure to components of processed food and to drugs of abuse, staging the suggestion that some of these foods may be addictive [3-5]. A common characteristic of addictive substances is their ability to promote consumption despite known negative consequences [12]. As such, the intent of current nutrition policy is likely to fall short of addressing health trends discussed above.

A review of the current understanding of substance dependence is beneficial before examining the validity of the food addiction hypothesis. Koob and Volkow describe substance dependence as compulsive seeking and taking of a substance leading to disruption of functional living and health [12]. Addiction is often characterized by a recurrent pattern of compulsive drug seeking, loss of control during drug intake (binging), and intense drug craving [38]. These behaviours have been categorized into 11 diagnostic criteria for substance-use disorders (SUD) in the DSM-V, and have also been adapted to animal models for the investigation of underlying biological mechanisms of addiction [12].

Addictive behaviours involve drug-induced neurobiological changes in neural systems that naturally control responses to rewards [39]. More specifically, addiction has been conceived as a learning and memory disorder, and brain reward systems adapt to repeated drug exposure and the organism develops habitual responses to environmental stimuli predictive of drugs [9]. This said, excessive intake of drugs also affects neural systems involved in executive functions and reactivity to stressful stimuli. Compromised inhibitory function and excessive reactivity to stress interact to promote the transition from drug use to addiction and dependence [12,40,41]. Although several neurochemical systems are involved in these processes [12,42], the mesolimbic (ventral tegmental area to nucleus accumbens) and mesocortical (ventral tegmental area to various cortical regions) dopamine systems are considered central [41]. Dopamine activity is involved in reward learning [43], reward sensitivity [41], as well as sensitization to drug effects [44], and cue learning by which drug predictors become salient [45].

Food is a nutritional reward required for life and, therefore, not inherently harmful. However, hyper-processing creates energy dense foods which act on dopaminergic systems producing effects similar to drugs of abuse [3]. Four lines of evidence from studies in laboratory animals support the hypothesis that food, and more specifically sugars and fats, activate addictive processes. First, there is evidence of cross-sensitization between sugar and drugs of abuse. In an elegant set of studies, Avena and Hoebel demonstrated the bi-directionality of this effect using sucrose and amphetamine [46,47]: both motor hyperactivity and hyper-consummatory behaviours were observed upon exposure to an otherwise ineffective dose of the novel substance (sucrose or amphetamine) after pre-exposure to the other. Interestingly, Foley and colleagues revealed a role of dopamine in this effect, as rats exhibited motor hyperactivity to quinpirole (a dopamine D2/D3 receptor agonist) after pre-exposure to sucrose [48].

Second, binging and withdrawal have been reported during and after prolonged sugar consumption. Thus, given intermittent daily access to glucose and food for 30 days, rats develop binging along with increased dopamine D1 receptor binding in the nucleus accumbens and decreased D2 receptor binding in the dorsal striatum [49,50]. Furthermore, somatic (tremors, chattering teeth and head shakes) [51], and emotional signs of withdrawal [51,52], as well as increased consumption upon re-exposure to sucrose [53], have also been reported. Whether withdrawal from sugar is identical to withdrawal from drugs, however, is not completely clear because it is very difficult to equate sugar to drug exposure, and it may not be ecologically valid to employ levels of sugar exposure that are necessary to observe a withdrawal response.

Third, animals that self-administer sucrose or high-fructose corn syrup in operant chambers respond in ways that closely resemble the self-administration of cocaine, and display alterations of mu-opioid and D2 dopamine receptors in the striatum that are also observed in cocaine-exposed rats [54-56]. Finally, there is evidence that unlimited exposure to palatable food can promote compulsive seeking and excessive consummatory behaviour. Teegarden and Bale reported that rats trained to consume a diet high in fat and sugars were more willing to endure an aversive environment in order to regain access to their preferred diet [57]. Johnson and Kenny explored a highly palatable, calorie dense cafeteria diet, and found that over time rats consistently preferred the cafeteria diet over the standard chow, became obese, and persisted consumption of the palatable diet despite the risk of an aversive stimulus [58]. Importantly, these behaviours were accompanied by increased brain reward threshold and a reduction of striatal dopamine receptors [58].

The YFAS scale [7,8] was developed to assess whether an individual's eating behaviour would meet the DSM criteria for substance dependence. Although scores on the YFAS do not correlate with body mass index [59], the use of this scale in overweight or obese subjects has generated interesting findings. For example, Bégin compared a sample of obese individuals seeking help controlling their eating behaviour to individuals with other substance use disorders [60]. It was found that obese individuals who scored high on the YFAS better matched individuals with other SUDs on measures of impulsivity. As well, high YFAS scores predicted the frequency of snacking, emotional eating, and associated comorbid disorders such as BED, depression, and attention deficit hyperactive disorder in childhood [59]. Interestingly, several neural correlates of addictive eating have been identified using the YFAS. In a study by Gearhardt and colleagues, fasted obese and lean subjects were shown cues of palatable food followed by the delivery of a palatable solution [61]. They found that individuals who scored higher on the YFAS demonstrated increased activation in areas of the brain involved in encoding the incentive-salience of food cues (dorso-lateral prefrontal cortex, medial orbito-frontal cortex [62], and caudate nucleus [63]). Also, high scores were associated with reduced activity in the lateral orbito-frontal cortex, a region involved in behavioural inhibition during consumption of rewards [64]. Interestingly, these brain regions are a part of the mesocortical dopamine system, which plays a key role in SUDs, as discussed above.

Another interesting finding relevant to the current discussion is evidence of cue reactivity in the context of addictive-like eating behaviour. In fact, people who struggle with substance use disorders are very familiar with the physiological responses experienced when presented with cues associated with their drug of choice [65]. Cue reactivity is well documented in animals and humans in the context of many substances including cocaine, tobacco, heroin and alcohol [65]. Meule and colleagues documented a similar effect in adults meeting criteria for food addiction using the YFAS [66]. They found that

participants fitting four or more criteria demonstrated significantly greater reactivity to food cues than those fitting one or no criteria.

Because it is known that only a small fraction of individuals who use drugs eventually progress to dependence [12,41], it is expected that only vulnerable individuals will be predisposed to addictive-like eating behaviours [67]. Indeed, a genetic profile involving several alleles has been implicated in mediating reward-responsive overeating [68]. Of particular interest is the Taq1 A1 allele, which is associated with increased reward sensitivity [69], palatable food preference [70], diminished striatal dopamine activity [71], and it is expressed in many individuals with SUDs and eating disorders [72]. It has been hypothesized that individuals expressing the Taq1 A1 allele are more susceptible to seek and consume palatable food as a form of compensatory behaviour [71]. Individual vulnerabilities have also been observed in rodents: the motivation to consume palatable food is more pronounced in obesity-prone rats [73], and rats more sensitive to the incentive properties of palatable food are significantly more sensitive to the reinforcing effect of cocaine [74].

Finally, similarly to drug addiction, there is significant evidence that childhood exposure to stress and a family history of substance use can increase the risk of developing eating disorders and obesity later in life. For example, a longitudinal study found that childhood maltreatment is linked to an increased risk of developing eating disorders [75]. Interestingly, a family history of alcohol abuse is also associated with an inherent preference for sweet tastes [76] and predicts the risk of obesity in young individuals [77]. Similarly, children of mothers who smoke during pregnancy are higher risk for developing eating disorders and weight problems [78].

Implications

Given the neurobiological and behavioural evidence reviewed above, could the food industry take advantage of the addictive potential of sugars and fats to create an environment that promotes the consumption of their products? Because food is commodity, it is in companies' best interest to bombard consumers with logos and advertisements to ensure maximum profitability [14]. These can act as conditioned stimuli, much the same way a picture of a cigarette pack can act as a cue for smokers [9], hindering attempts to alter lifestyle choices and consume healthier food. Overall, the weight of the evidence above suggests that regulating marketing strategies may be effective in curbing consumption of unhealthy foods.

Adjusting the Current Mind Set

Food addiction did not appear in the recently updated DSM-V because of inconsistencies in the literature and lack of clear definition [10]. It is possible to argue that the evidence is not convincing enough to supersede potential adverse social and economic impacts of considering food addiction as a diagnosable condition. For example, it can be argued that framing food and drugs of abuse within a similar model of mental health disease can impede motivation required to make appropriate lifestyle adjustments necessary for good health such as exercise. Moreover, it has been suggested that food addiction does not adequately explain existing conditions, such as binge eating disorder, and that its inclusion in the DSM would not add significant diagnostic capability for treating these conditions. Although these are valid arguments, the food addiction hypothesis is still relatively new and rather than discarding it all together, it is more useful to examine the validity of the constructs on which it has been proposed.

One potential issue has been the focus on the obese population. Weight gain results from the overconsumption of food and consequent imbalance of energy intake, and this has led to the suggestion that addictive-like feeding behaviour is the cause of obesity. This, however, is not necessarily the case [11] because obesity is an exceptionally multifaceted issue with a complex etiology that makes it impossible to understand using a framework focused on one cause. In addition, it is possible to over consume processed food, not become overweight, and still be at an increased risk of heart disease and diabetes. There is also reasonable indication that one does not need to be obese to be addicted to food either [27]. Thus, although obesity is an important demographic variable to investigate the neurobiology of overconsumption of food, it should not be used as the prototype for food addiction [79].

Another consideration is the use of other addictive substances to model food addiction. While this approach is informative, there is a very important fundamental difference that must be taken into consideration. Food is ubiquitous throughout society and a necessity for life, which makes defining exactly when food is being abused difficult. In this regard, it is critical for research in this field to determine specifically what makes some food addictive and what sets these foods aside from those which we are required to eat [37]. Also, considerable variability exists among addictive substances regarding the underlying mechanism that initially promotes consumption. Opiate dependence is driven by a pattern of withdrawal and negative affect, psychomotor stimulants users follow a pattern of binging and intoxication, and smokers of nicotine and cannabis exhibit a preoccupation and anticipation pattern [12].

As such, there are problems with using the DSM criteria for substance dependence to define food addiction because of inappropriate terminology. This is especially evident when examining withdrawal, tolerance, and seeking characteristics. With regard to withdrawal, are the symptoms expressed while experiencing abstinence from these foods an effect of withdrawal or simply energy deficits? A recent study in laboratory animals indicated that although naltrexone precipitates robust signs of affective withdrawal in heroin pre-treated animals, the effect in animals pre-treated with high fructose corn syrup are much less pronounced [52]. In the description of tolerance, the DSM-IV highlights "intoxication," which is a term that may not be suitable for consumption of food. The DSM-V added "desired effect" but this is also ambiguous and could be difficult to interpret in the context of food consumption. Ultimately, the initial motivation to consume food is nutrition, which is not comparable to intoxication [79]. Seeking criteria are also ambiguous in the context of food consumption for two reasons: first, food seeking is a basic biological necessity; and second, food is legal, unlike many drugs, and harmful effects are not as readily apparent [10]. Essentially, you cannot go to jail for eating or overdose on food. Thus, determining when healthy food seeking becomes harmful can be difficult.

Limitations aside, the latest edition of the DSM has employed an updated framework to describe addictions that may be useful to accommodate foods. The understanding of substance dependence continues to develop and influence the diagnostic framework of SUDs. The DSM-IV used a strict categorical method, applying an indiscriminate seven item list to all substances with an all-or-nothing approach, where meeting three or more criteria indicated substance dependence. In contrast, the DSM-V employs a dimensional approach with distinct diagnostic criteria for several classes of substances, and a separate set of criteria for behavioural addictions such as gambling

[11]. Whether food addiction would best fit within substance or behavioural addictions remains unclear.

Policy Implications

If one assumes that the food addiction hypothesis is viable, then it becomes questionable whether the self-regulation approach currently employed by industry is sufficient to address the adverse health trends discussed above [37]. But, if regulatory policies were to be implemented, what would they look like? Also, there are significant hurdles to overcome in order to implement effective policy changes. For example, one would expect that regulations could have damaging implications to corporations such as McDonald's or Coca-Cola, which have billions of dollars at stake [13]. Perhaps, considering legislative progress involved in regulating other addictive substances such as alcohol and tobacco, may offer some insight for effective policy approaches regarding unhealthy foods [37].

Several addictive substances have undergone notable transitions from freely available to restricted. Examples include opium, cocaine (formerly a primary ingredient in Coca-Cola), and most recently tobacco [80]. Tobacco has become more difficult to obtain due to a series of restrictions implemented over the past 40 years [37]. Of particular relevance to the current discussion is the elimination of public advertisement and restrictions on the displays of cigarette packaging in stores. A survey conducted in Norway, found that eliminating tobacco displays in point-of-sale settings was perceived as particularly beneficial and significantly reduced the temptation to purchase cigarettes by youth [81]. Should the advertisement of products high in sugars and fats be similarly restricted?

Ultimately, policy makers should employ a substance-focused approach to strengthen current nutrition policy [13,37]. This could include restricting advertisements that promote processed food, removing sweets from point-of-sale settings, subsidizing the production of whole foods, and taxing products containing added sugar. These policies would provide incentive for the food industry to endorse a bona fide transition toward healthier food, promote an environment that supports healthy lifestyle choices, and reinforce sound nutritional advice already being promoted. Interestingly, a few of these approaches are already being implemented. For example, several countries, including Great Brittan, Mexico, Hungary, and Finland have implemented a sugar tax [82], and PepsiCo has recently announced that they will cut all sugar content from their drinks by 2025 [83]. It is important to continue monitoring the impact of these approaches to determine their effectiveness over time.

Conclusion

It appears that current regulatory strategies inadequately address the prevalence of unhealthy diets and the related health consequences. Effective government intervention must implement nutrition policies that consider both the biological and environmental influences on negative eating behaviours presented throughout this review. The global response currently addressing this issue is effectively increasing public concern; however, unhealthy food remains a popular choice and adverse health trends continue to present difficulties for public health.

Current nutrition policy allows companies to continue the sale of unhealthy food under industry-derived self-regulation policies that place the onus entirely on the public to practice self-control. Evidence presented in this review indicates that prevalent food cues combined with addictive potential of certain foods creates an environment that ultimately compromises this strategy. Therefore, introducing policies that limit the use of refined sugar or restrict advertisement on products containing sugar may be an effective and pragmatic first step in reducing the prevalence of unhealthy diets.

Finally, the study of food addiction is in its infancy and there is much to discover. Indeed, only a small proportion of obesity is linked to food addiction; but, individual vulnerability to the health consequences and addictive processes associated with the consumption of processed food are not confined to this demographic. Ultimately, the long-term consequences of the effect of sugar on the brain are not yet known. Is it possible that excessive exposure to sugar in children may predispose them to reward related pathologies in adulthood? How can neuroscience address this question and how would this impact nutrition policies?

Author Contributions

T.H. and F.L. contributed to the conception and designed of this review; T.H. and F.L. wrote the paper.

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