HHS Public Access

Author manuscript

Physiol Behav. Author manuscript; available in PMC 2023 December 04.

Published in final edited form as:

Physiol Behav. 2023 March 01; 260: 114049. doi:10.1016/j.physbeh.2022.114049.

Reward and inhibition in obesity and cigarette smoking: Neurobiological overlaps and clinical implications

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Abstract

Cigarette smoking and obesity are the leading causes of premature morbidity and mortality and increase the risk of all-cause mortality four-fold when comorbid. Individuals with these conditions demonstrate neurobiological and behavioral differences regarding how they respond to rewarding stimuli or engage in inhibitory control. This narrative review examines the role of reward and inhibition in cigarette smoking and obesity independently, as well as recent research demonstrating an effect of increased body mass index (BMI) on neurocognitive function in individuals who smoke. It is possible that chronic smoking and overeating of highly palatable food, contributing to obesity, dysregulates reward neurocircuitry, subsequently leading to hypofunction of brain networks associated with inhibitory control. These brain changes do not appear to be specific to food or nicotine and, as a result, can potentiate continued cross-use. Changes to reward and inhibitory function due to increased BMI may also make cessation more difficult for those comorbid for obesity and smoking.

Keywords

Obesity; Smoking; Nicotine; Reward; Inhibitory control; Dopamine; Smoking cessation

1. Introduction

Cigarette smoking and obesity are the two leading causes of premature morbidity and mortality worldwide [1–3]. Individuals who smoke cigarettes and have obesity incur four times the risk of all-cause mortality compared to lean individuals who have never smoked and eleven times the risk of cardiovascular diseases [4]. The negative health effects of cigarette smoking and obesity can shorten life by more than 10 years [5,6] and substantially impact productivity and public health costs [1,3]. Given the substantial health burden, it is critically important to characterize this comorbid population to best tailor treatments and develop interventions to prevent the co-occurrence of obesity and smoking.

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Declaration of Competing Interest

The authors declare no financial interests or conflicts of interest.

Nicotine addiction and diet-induced overweight are both conditions resulting from overconsumption of rewarding substances associated with disregard of long-term adverse outcomes, hallmarks of impulsivity, yet little is known about the overlap of these conditions. Studies have documented obesity-related differences in rate of smoking [7], quit success [8,9], and risk of negative health outcomes [4]. Obesity appears to have a complicating influence on smoking behavior and cessation. However, there is minimal speculation on why this might be so, and until recently, no demonstration of a potential brain-based mechanism. Alterations in evaluation of reward and ability to control prepotent responses are evident in both obesity and smoking; [10–13] it is possible that overlapping dysfunction in neurocircuits coding for reward and inhibitory processes may contribute to the unique presentation of the comorbid population. We hypothesize that obesity in individuals who smoke may be associated with unique alterations in response to reward, inhibitory control, and decision-making processes, thereby hindering smoking cessation in this population.

Recent reviews have comprehensively described the relationships between smoking, eating behavior, and bodyweight, as well as the similarities in neurobiology [11,12,14,15]. In particular, the literature has highlighted the impact of smoking behavior or cessation on intake and bodyweight. In contrast, this narrative review aims to examine the influence of intake and bodyweight on smoking behavior. In particular, this review will examine differences in neurobiologically-based cognitive processes (described herein as neurocognitive function) that are evident with increased body mass index (BMI) in people who smoke, emphasizing the constructs of reward and inhibition. Lastly, we hypothesize how obesity may lead to difficulty quitting smoking. Patients with higher BMIs make up 70% of those seeking treatment for smoking [16], and weight concerns increase risk of relapse [8]. Given the public health significance, understanding how BMI influences neurocognition in smoking can inform treatment to promote smoking cessation among individuals with excess body weight.

2. Reward and inhibition in smoking

Nicotine derives its rewarding properties through its effect on the dopaminergic (DA) system, both directly [17]and indirectly via action on nicotinic acetylcholine receptors (nAChRs) [18], leading to sharp increases in striatal DA [13]. Striatal DA receptor-expressing neurons project to a range of brain regions, including the nucleus accumbens (NAc), anterior cingulate cortex (ACC), orbitofrontal cortex (OFC), dorsolateral prefrontal cortex (DLPFC), amygdala, dorsal striatum, ventral pallidum and substantia nigra [19,20]. Smoking-related disturbance of this system has far-reaching implications for neurocognitive function, as this network plays an important role in reward identification and valuation [21–23], integration with motivational and affective drives [24–26], as well as executive function and regulation [27–29].

In human positron emission tomography (PET) studies, reductions in DA receptor availability are associated with decreased activity of the dorsolateral prefrontal cortex (DLPFC) and ACC, areas linked with inhibitory control and decision-making in substance use [30,31]. Individuals who smoke demonstrate lower striatal DA receptor availability or binding potential than nonsmokers [32–34], suggesting down-regulation and reduced

activation of prefrontal brain regions [35, 36]. In turn, this hypoactivation is associated with craving and smoking behavior [37]. Cue-reactivity studies have demonstrated that blunted activation in dopamine-rich brain regions in response to pleasant, non-smoking rewards (e.g., money, food, romantic/sexual), relative to activation in response to smoking cues, predicts abstinence in smoking cessation treatment [38–40]. As bodyweight influences brain response to reward (discussed below), it is possible that this balance may differ with BMI, subsequently shaping treatment outcomes.

Connectivity between brain regions may also be altered with smoking, specifically in and amongst the executive control networks (comprising regions of the dorsolateral prefrontal cortex and the lateral posterior parietal cortex), default mode network (including the posterior cingulate cortex, prefrontal cortex, angular gyri and parahippocampus) and salience network (comprised of the insula and anterior cingulate). Severity of nicotine dependence [41], craving [42–44] and reward response to smoking cues [45] are all associated with connectivity both within the salience network and coupled with broader brain networks. However, there are conflicting findings in the extant literature as to whether salience or default mode network resting-state connectivity is reduced [46–48] or elevated in participants who smoke as compared to nonsmokers (however see [49,50]). Given that none of these studies controlled for the potential influence of bodyweight, it is possible that differences in body mass index (BMI) may underlie the disparate findings. Similar to cue-reactivity research, studies have shown that functional connectivity may predict treatment outcomes in smoking cessation [39]. If BMI idiosyncratically changes brain network function in those who smoke, this may partially explain differences in quit success in individuals comorbid for obesity and smoking.

In the short term, nicotine improves attention and memory, and brief abstinence is associated with impairments in cognitive control and executive function [51], reinforcing smoking behavior. With chronic use, however, individuals who smoke are shown to have poorer performance on inhibitory control tasks [36,52] and hypoactivation in prefrontal areas during inhibitory control tasks [35,36,52]. Further, long-term smoking cessation may promote improvements in executive control [53,54], potentially attributable to the normalization of DA receptor availability [33,34]. Targeting these improvements in executive control may both promote and extend abstinence through neuroplastic return to function.

Through the desensitization of the nAChR and DA systems, chronic nicotine exposure dysregulates responses to reward, both smoking-related and not. Evidence suggests that diminished ability to experience reward, or hedonic capacity, is predictive of smoking uptake and progression [55], and participants who smoke report reduced reward globally than nonsmokers or those with a history of smoking [56]. With consumption, cigarettes become heavily overvalued as rewards compared to alternative reinforcers [57,58], but neuroimaging research suggests the neurobiology of smoking is linked to more global reward responsivity. For example, nicotine affects the response to secondary rewards: the anterior insula, anterior cingulate, and striatum show enhanced activation to monetary reward following acute administration [59]. Greater responsivity to monetary reward has also been linked to caudate

response to visual smoking cues [60]. As global reward response is dysregulated, cognitive processes such as decision-making may suffer as a result.

Individuals who smoke have more difficulty delaying monetary and consumable rewards than never-smokers and those who have successfully quit [61–65], and greater difficulty delaying is correlated with cigarette self-administration [66] though not with dependence [67]. Compared to those who do not, individuals who smoke show reduced ventral striatal activation in response to delayed reward, suggesting delayed reinforcers are more devalued in people who smoke [68]. Delay discounting, or the extent to which delayed reinforcers are devalued, is associated with lower activation in middle and superior frontal regions in those who smoke when presented with immediate rewards [64]. Nonsmokers will show a negative correlation between delay discounting and frontal connectivity with reward-related regions [69,70] – in other words, reduced connectivity in fronto-striatal networks, or disinhibition of reward responsivity, is linked to greater delay discounting. However, in individuals who smoke, greater frontal connectivity with the insula is linked with greater discounting [71]. Insular connectivity is heavily implicated in cue-induced craving in cigarette smoking [43,45,72,73] and nicotine dependence [72]suggesting that dysfunction in this circuit may elevate vulnerability to immediate rewards and subsequent motivation to smoke, without the inhibitory influence of the frontal executive control network. Additional research has demonstrated that fronto-insular connectivity, highlighting the influence of reward valuation and subsequent motivation on smoking-associated neurobiology.

3. Reward and inhibition in obesity

Caloric intake sets off a complicated cascade of neurotransmitter release, both central and peripheral, and is modulated by hedonic and homeostatic systems. Diet-induced obesity, by definition, is related to chronic consumption beyond nutritional needs and may be due as much to a failure of the homeostatic system as to hedonic over-control. Largely, however, dopamine (DA) is the primary transmitter indicated in eating behavior. Food intake potentiates the release of DA in the dorsal striatum, with greater release associated with both higher palatability [74] and higher fat content, independent of flavor [75]. Bello and colleagues [76] determined that chronic highly palatable food consumption leads to downregulation and reduced sensitivity in D2 receptors. Neuroimaging in humans has shown that obese individuals have lower striatopallidal receptor availability of DA than individuals with normal weight [77]. There is somewhat conflicting evidence of a relationship between D2 and BMI [78], which may be linked to a parabolic pattern of reward sensitivity as BMI increases, with those at very low or very high weights exhibiting reduced reward sensitivity while levels in those in the overweight or mildly obese range are elevated [79,80]. Increased striatal DA release seen in mild or early obesity may lead to decreased D2 receptor availability when an individual is more severely obese but not at earlier stages of weight gain [81]. BMI may have a dynamic relationship with reward such that responses to rewards and the relationship of that response with behavior change as bodyweight increases.

Greater sensitivity to rewards, in general, may be a preexisting characteristic that drives overeating and, thereby, weight gain [82]. However, some studies suggest that while greater reward sensitivity predicts consumption, it is not necessarily correlated with overweight

[83]. Individuals with obesity are willing to work harder for food reward, demonstrating a higher relative reinforcing value of food [84], and additionally find alternatives less reinforcing [84,85]. High food reinforcement is also associated with future weight gain [86]. Notably, regular consumption may lead to reduced rewarding value of food over time [87,88], and this reduction is also associated with future weight gain [87]. Beyond food, individuals with overweight [89] and obesity [90,91] show increased neural response to monetary reward in the ventral striatum, amygdala, and medial frontal cortex. This research suggests bodyweight, similar to smoking influences neural responses to non-natural rewards, pointing to a more global effect of chronic overeating on reward processing.

While there are robust findings of worse performance on inhibitory motor tasks in individuals with obesity (see reviews [92,93]), others only show a difference on food-related inhibitory tasks [94–96], or fail to show a link to BMI at all [97–99]. Over time, inhibition does seem to predict weight change, however. In obesity treatment, those who demonstrate worse inhibitory control have been shown to lose less weight, both among children [100] and adults [101]. Further, brain response related to inhibitory control is altered with obesity. Women with obesity demonstrate reduced activation when inhibiting behavioral responses in the insula, inferior parietal cortex, cuneus, and supplementary motor area [97] and when practicing ‘appetite control’ in the medial frontal cortex, caudate, and ACC [102]. Poorer performance during inhibitory tasks has been associated with hypoactivation and reduced glucose metabolism of the prefrontal cortex in obesity as well [36,93,103,104]. Reduced response in frontal inhibitory regions has also been linked to greater delay discounting in individuals with obesity [104,105], as well as inhibitory control dysfunction, and is shown to predict weight change [106,107]. Like individuals who smoke, those with higher BMIs or obesity tend to have higher delay discounting rates than their lean counterparts [98,105,108,109]. Delay discounting may also predict diet success and weight change over time [106,110], as well as intake in both lean and overweight samples [111,112]. The particular effects of increasing BMI on inhibitory control are not restricted to eating behavior but may impair responding more generally in such a way as to dysregulate response to other rewards.

4. Influence of eating and bodyweight on reward & inhibition in people who smoke

Dysregulation of reward-related networks has been posited to impact inhibition, such that prolonged consumption of appetitive rewards will lead to dysfunction in the prefrontal neurocircuitry involved in executive control and decision-making [113]. It follows that regular overconsumption of highly-palatable food or nicotine would lead to more general difficulty in inhibitory control. Subsequently, it is hypothesized that neurobiological dysfunction in inhibitory control-related regions would be compounded by effects from regular nicotine use and highly palatable food consumption, creating exponentially greater disturbance to executive function in individuals with obesity who smoke. However, despite the considerable overlap in the pathophysiology of smoking and obesity and the high health risks associated with comorbidity, we know comparatively little about how comorbid obesity and smoking influence neurocognitive function.

Behaviorally, only one study has examined delay discounting in the comorbid population, showing that adolescents with comorbid obesity and smoking have significantly greater delay discounting than those without, suggesting that obesity may confer risk of dysfunctional reward-related decision-making above and beyond smoking alone [114]. Blendy and colleagues demonstrated reduced nicotine reward from smoking in humans and mice with obesity, suggesting that the drivers of smoking behavior in individuals with obesity may be related to behavioral rather than chemical reinforcement [115]. These studies suggest that conditioned cues that predict rewards like smoking or eating may be particularly salient in this population and make cessation more difficult.

To examine this, we explored the influence of BMI on brain response to evocative smoking cues and found that increasing BMI was negatively associated with activation of the right dlPFC during smoking cue exposure [116]. Further, individuals with overweight and obesity who smoke showed significantly reduced response in the dlPFC compared to lean. Notably, greater commission errors on a Go/NoGo inhibitory control task were correlated with blunted right dlPFC response to smoking cues amongst smoking participants with obesity but not those who were overweight or lean. It is possible that reduced activation in the right dlPFC with obesity may thus lead to difficulty regulating cue-potentiated craving and smoking motivation. Extending this research to functional connectivity also showed an influence of BMI in chronic cigarette smoking, particularly in the salience and default mode networks [117]. Differences between weight groups in the relationship between connectivity in the salience network and smoking behavior were also evident. Sutherland and colleagues hypothesize that dysregulated connectivity amongst these networks may underlie the maintenance of nicotine addiction through hypersensitivity to drug cues, somatic symptoms of withdrawal, and the cognitive impairments associated with acute abstinence [44]. That this varies with BMI suggests that vulnerability to relapse may also differ depending on bodyweight.

Self-reported motivation to smoke may also be differentially related to smoking behavior depending on BMI. While adults seeking treatment for smoking endorsed similar automatic or addictive motivation to smoke regardless of weight status, automatic and addictive motivations did not uniformly predict past or current smoking behavior [117]. Collectively, these findings suggest that BMI may influence reward valuation, motivation, and inhibitory control in those who smoke in such a way that could result in less success quitting smoking in individuals with obesity or greater need for supplemental therapies [118, 119]. However, future research is necessary to determine if this is above and beyond the effects of BMI in nonsmokers.

5. Obesity may lead to difficulty quitting smoking

Comorbid smoking and obesity likely share neurobehavioral underpinnings, identifying which could inform interventions for comorbid behavior change. Individuals with overweight or obesity comprise 70% of treatment-seeking smoking patients, gain the most weight after quitting, are the least accepting of that weight gain [16,120–122] and are more likely to relapse back to smoking as a result [8,9]. Interventions targeting smoking and overweight have modest success at best [123–125]. Behavior change interventions treat each

behavior independently, ignoring potential reward-related mechanisms shared by smoking and excess food intake [16,120–124]. Individuals comorbid for smoking and obesity may require different treatments for smoking cessation than those who are lean or overweight or may find it more difficult to quit smoking. Evidence suggests that individuals with obesity who smoke are less likely to achieve smoking abstinence using transdermal nicotine [126] and may be more likely to use medication [119]. People comorbid for smoking and obesity are more likely to smoke heavily as well [7], which may contribute to difficulty quitting, but it is unclear why they are more likely to smoke more.

Alterations in the neurobiology of reward and inhibition may contribute to the development of diet-induced obesity and smoking uptake. At the same time, overconsumption of food and nicotine may interactively perpetuate the neurobehavioral dysfunction that drives both issues. Regular reward stimulation through overconsumption may dysregulate this circuitry both through a combination of overvaluation or sensitization of reinforcers [55,57,127–129] and habituation to reward receipt [87,85]. This dysregulation leads to disturbed modulation of prefrontal inhibitory regions [30], resulting in difficulty inhibiting prepotent behavioral responses and continued compulsive intake. Given this feedback loop, chronic consumption of multiple rewarding substances (e.g., nicotine and highly-palatable food) would arguably interact to impair neurobiological inhibition to a greater extent than overconsumption of just one. Difficulty of quitting smoking among patients with obesity is thus unsurprising. Treatments specifically focused on improving inhibitory control ((e.g., mindfulness [130]) or modulating frontal activation ((e.g., transcranial magnetic stimulation [131, 132]) could target the unique neurocognitive presentation of individuals with obesity with greater efficacy. In addition, Episodic Future Thinking (EFT) treatment targets delay discounting and has shown promise in reducing demand and craving for both nicotine and fast food when combined with health goal-setting [133]. Specifically designing treatment to elevate reward response to non-cigarette and non-food cues, improve function in frontal inhibitory control regions, and reduce the influence of time delay on reward valuation may provide supplemental support for obese patients making a quit attempt.

6. Conclusions

Between 30% - 40% of people with obesity in the U.S. smoke cigarettes [134], compared with less than 15% of the general population [135]. Individuals comorbid for obesity and smoking also have a higher daily smoking rate and smoke for more years [7]. Those who both smoke cigarettes and have obesity incur four times the risk of all-cause mortality compared to lean individuals who have never smoked and eleven times the risk of cardiovascular diseases [4]. The negative health effects of cigarette smoking and obesity can shorten life expectancy [5,6] and substantially impact productivity and public health costs [1]. Despite public health impact, little research has focused on understanding this comorbidity, particularly the neurobehavioral function of those who smoke and have obesity. Advances in our understanding of neurocognition in smoking and obesity independently and extensive behavioral and epidemiological literature on the comorbidity highlight the possibility that chronic administration of one reward may directly influence the brain response to the other, thereby maintaining both behaviors. However, less is known about whether obesity and smoking are additive, interactive, or neither in their influence on

reward-related decision-making. Exploring neurobiological function as it relates to food and bodyweight in individuals who smoke may allow for a greater understanding of nicotine use [136]. If comorbidity is associated with unique patterns of brain function and behavior, as we hypothesize, it is vital to tailor treatments to this population to increase effectiveness and improve outcomes.

Funding

There is no funding to report for this submission.

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