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The neurobiological and behavioral overlaps of nicotine and food addiction

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ABSTRACT

Both cigarette smoking and obesity are significant public health concerns and are associated with increased risk of early mortality. It is well established that the mesolimbic dopamine pathway is an important component of the reward system within the brain and is implicated in the development of addiction. Indeed, nicotine and highly palatable foods are capable of altering dopamine release within this system, engendering addictive like responses in susceptible individuals. Although additional research is warranted, findings from animal and human literature have elucidated many of neuroadaptions that occur from exposure to nicotine and highly palatable foods, leading to a greater understanding of the underlying mechanisms contributing to these aberrant behaviors. In this review we present the findings taken from preclinical and clinical literature of the known effects of exposure to nicotine and highly palatable foods on the reward related circuitry within the brain. Further, we compare the neurobiological and behavioral overlaps between nicotine, highly palatable foods and obesity. Lastly, we examine the stigma associated with smoking, obesity and food addiction, and the consequences stigma has on the overall health and wellbeing of an individual.

1. Introduction

Both cigarette smoking and obesity are significant public health concerns, associated with a multitude of comorbities including cardiovascular disease, diabetes and cancer (Borrell and Samuel, 2014; Ezzati and Lopez, 2004; Flegal et al., 2013; Woloshin et al., 2008). Although the prevalence of smoking has declined in recent years, nearly 1 in every 5 American adults still continue to smoke cigarettes (Ahmed Jamal et al., 2015). Unlike smoking, the prevalence of overweight and obesity has sharply increased, more than doubling nationwide since 1980 and are only expected to continue to rise (Wang et al., 2011). Together, obesity related diseases and smoking are two leading causes of preventable death (Collaborators, 2015).

In an effort to reduce the rates of smoking, many states have increased the price of cigarettes, which has shown to be successful in both light (Levy et al., 2005) and heavy (Cavazos-Rehg et al., 2014) smokers and clean air restrictions have been put into place, limiting the areas in which people are legally allowed to smoke (Cochran et al., 2012; Snyder et al., 2015). Despite these public health interventions, smoking related illnesses account for more than $300 billion dollars per year in the United States alone (Ahmed Jamal et al., 2015). Although nicotine is not the only reinforcing property of cigarette smoking (Rose et al., 2010), it is the main addictive constituent within tobacco that drives the repeated behavior due to its rewarding effects within the brain (Dani and Heinemann, 1996; De Biasi and Dani, 2011).

While cigarette smoking continues to be a major public health concern, the alarmingly high rates of overweight and obesity also plague the country. While there are many causes of obesity, including genetics, environmental factors and age (Gurnani et al., 2015; Pigeyre et al., 2016), the current food environment is a significant contributor to excess weight in both children and adults (Sonntag et al., 2015). Large portion sizes, nutrient poor-energy dense foods and beverages encourages superfluous consumption of highly-palatable foods, often leading to excessive weight gain (Bucher Della Torre et al., 2015; Mattes, 2014; Rolls, 2003). Although there is currently no standard medical diagnosis for food addiction (FA), it is indisputable that certain foods are capable of eliciting addictive behaviors and associated with dysregulation of brain reward systems similar to addictive substances, like nicotine (Avena and Hoebel, 2003; Avena et al., 2005; Avena et al., 2008; Wideman et al., 2005).

Nicotine addiction and pathological eating are both maladaptive behaviors associated with serious adverse effects and share similarities on a neurobiological and behavioral level. Although the literature is replete with reviews covering obesity and nicotine addiction, and to a lesser extent, FA, to the best of our knowledge, this is the first narrative review that has summarized the overlap between these topics. All articles included were written in English and identified in PubMed. The search terms used to collect all potentially relevant articles included *reward,*

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addiction, obesity, nicotine, stigma, sugar, fat and *highly palatable*. The abstract of each article was manually checked and the full text was downloaded and read before being included in this review. This review will be a contribution to the literature as it strives to provide the reader with a greater understanding of the neurobiological underpinnings driving these chronic conditions and how they intersect with each other. In this review, we summarize the evidence taken from preclinical and clinical models of nicotine dependence and FA, as well as the overlaps between the two from a neurobiological, behavioral and social standpoint.

2. Nicotine

2.1. Preclinical

The mesolimbic dopamine (DA) pathway is an important component implicated in reward and addiction, and includes dopaminergic (DAergic) projections originating from the ventral tegmental area (VTA) to the nucleus accumbens (NAc) (Volkow et al., 2007; Volkow and Morales, 2015; Willuhn et al., 2010). During a resting state, DAergic neurons typically follow a tonic pattern, which is a steady, low frequency firing pattern. However, when responding to various external stimuli, DAergic neurons fire in a phasic pattern, in turn signaling reward and goal motivating behavior (Grace, 1995). Similar to other addictive substances, nicotine increases DA firing activity, exerting its reinforcing properties (Liu et al., 2012; Marti et al., 2011; Rice and Cragg, 2004). Nicotine acts on nicotinic acetylcholine receptors (nAChRs), which are comprised of numerous subtypes distributed throughout the brain and have a diverse number of neurological functions (Changeux, 2010; Gotti and Clementi, 2004; Gotti et al., 2006). Activation and antagonism of specific subtypes including β2-, α4-, α5-, α6-, and α7-containing nAChRs are implicated in the rewarding effects of nicotine (D'Souza and Markou, 2011; Kenny and Markou, 2006; Sanjakdar et al., 2015) with the β2 subtype neurons specifically essential for DA release (Picciotto et al., 1998).

Activation of nAChR and specific subtypes regulate DA release within VTA (Zhao-Shea et al., 2011) and NAc (Pontieri et al., 1996), perpetuating addictive behavior (Mameli-Engvall et al., 2006; Threlfell et al., 2012). Chronic nicotine use upregulates the expression of nAChRs (Picciotto et al., 2008) and this desensitization of nAChR within the striatum is observed after cessation in mice exposed to multiple cycles of nicotine withdrawal (Hilario et al., 2012). Nicotine also lowers tonic firing and increases phasic firing of DA neurons within the NAc shell, altering DA signaling (Zhang et al., 2009). Further, synaptic plasticity of DA neurons within the VTA is altered as a result of nicotine exposure (Mansvelder and McGehee, 2000; Mao et al., 2011), with certain subtype receptors being necessary for this effect to occur (Jin et al., 2011). In addition to neurobiological adaptations seen in nicotine exposure, there are also behavioral manifestations associated with increased risk of addiction. Both novelty seeking and heightened impulsivity are associated with increased risk for substance abuse and nicotine addiction (de Wit, 2009; Wingo et al., 2015), and chronic nicotine administration has been shown to increase impulsive behavior (Kolokotroni et al., 2014). In summary, preclinical models have allowed for the examination of long-term neuroadaptations and behavioral effects induced by nicotine and contribute to a greater understanding of the addictive process seen in humans.

2.2. Clinical

Nicotine dysregulates reward response and processing within the brain. In order to examine these changes, many studies use positron emission topography (PET) scans to assess nicotine's effect on DA release, which can be indirectly measured by a decrease in the D2 tracer [^{11}C]raclopride binding potential (BP). When comparing the effects of nicotine containing cigarettes to denicotinized cigarettes on DA release, only nicotine containing cigarettes causes a significant reduction in [-^{11}C]raclopride BP within the left and right ventral caudate, including the NAc, while also positively improving mood. However, it is important to note that both types of cigarettes reduced anxiety and cravings in nicotine dependent individuals (Brody et al., 2009). Additionally, when assessing cigarette craving in adult smokers using a multi-sensory paradigm consisting of cigarette odor and a related visual cue, there was a significant inverse relationship between craving control and generated blood oxygen level dependent (BOLD) response within the ventral and dorsal striatum, both regions associated with reward (Cortese et al., 2015). However, Barrett et al. (2004) found no significant decrease in [^{11}C]raclopride BP from acute repeated nicotine exposure compared to abstinence conditions. The authors posit that it may be due to nicotine receptor desensitization on DA neurons, as the participants had to smoke several cigarettes within 12-minute intervals, and DA release may occur only from the initial cigarette. However, subjects who found the smoking conditions pleasurable showed a significant changes [^{11}C]raclopride BP within the dorsal striatum (Barrett et al., 2004).

Although nicotine is the main component driving cigarette addiction, other chemicals present in cigarettes, as well as the sensorimotor aspect associated with smoking, mediate a reward-related response (Rose et al., 2000; Rose et al., 2010). Electronic cigarettes (EC) deliver nicotine in vapor form, which allows for the continued behavior of smoking, but in theory, with less hazardous compounds and therefore less associated health risk (Benowitz and Burbank, 2016). However the long- term safety of EC continues to be challenged (Bean and Smith, 2016; Callahan-Lyon, 2014). Importantly, the delivery rate of nicotine into the bloodstream likely impacts the addictiveness of ECs. While newer models are proving to be more efficient when compared to older models in rate of delivery, not all are as efficient as traditional cigarettes (Farsalinos et al., 2014). These differences may partially explain why ECs are often utilized as a harm reduction method instead of complete substitution, and sometimes rejected by individuals who currently smoke (Pechacek et al., 2016). Although it appears as though ECs are capable of eliciting a reward response, more research examining the variables that impact response as well as differences between ECs and traditional cigarettes should be undertaken in the future.

3. Food addiction

3.1. Preclinical

Animal studies utilizing bingeing models of sugar and fat have provided insight on their abuse potential when given separately and in combination (Avena and Hoebel, 2003; Avena et al., 2005; Avena et al., 2009; Rada et al., 2010). Sugar bingeing behavior is evoked when rats are granted intermittent access to sucrose solution. On this paradigm, rats are maintained on a daily schedule of 12-hour food restriction followed by 12-hours access to standard chow and 10% sucrose solution offered 4 h into the dark period. Mirroring what is observed in the drug abuse literature, on this feeding schedule, rats show signs of bingeing, cross-sensitization (Avena and Hoebel, 2003), tolerance and craving (Avena et al., 2005). Rats also exhibit behavioral and neurochemical signs of withdrawal after being given the opioid antagonist naloxone, and when food deprived (Avena et al., 2008; Colantuoni et al., 2002). Notably, whereas the immensity of food-induced DA release diminishes after repeated access to a specific food, DA release in response to sugar in rats on this schedule remain increased relative to baseline measurements over 21 days (Rada et al., 2005). Extended access to highly palatable or "cafeteria-style" diets also induce hyperphagia. The cafeteria-style diet causes weight gain and obesity, as well as an increase in reward threshold (Johnson and Kenny, 2010). Moreover, increased craving during abstinence and insensitivity to punishment ensues (Johnson and Kenny, 2010), with rats being more resistant to

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foot shock when lever pressing for palatable food than when lever pressing for methamphetamine (Krasnova et al., 2014).

To date, there is no definitive answer to whether functional changes within the reward related pathways precede obesity, or if these neural adaptations are a response to excess weight gain and poor diet. Similar to what is seen in humans, when provided with the choice of highly palatable diet, some rodents are obesity resistant (OR) while others are obesity prone (OP) (Rada et al., 2010) and marked differences in response to reward as well as genetic variations affecting DA circuitry have been observed (Valenza et al., 2015). When basal levels of DA were measured via microdialysis, extracellular DA levels in the NAc shell were significantly lower in OP rats relative to OR rats. Further, when given a high fat meal, although DA release occurred in OP and OR rats, levels of NAc DA remained lower over time in the OP rats. This may contribute to increased responsiveness to highly palatable food seen in OP animals compared to OR rats (Rada et al., 2010). However, Davis et al. (2008) found a high fat diet (HFD) independent of obesity contributes to diminished responding for sucrose reward and lessens the rewarding value of amphetamine cue. Further, HFD alone was capable of decreasing DA turnover within the NAc. In addition to differences in DA transmission and behavior, D2R expression is also altered. Indeed, lower striatal expression of D2R has been found in rats who overeat sugar (Colantuoni et al., 2001), are exposed to a HFD (Narayanaswami et al., 2013), and are allowed extended access to cafeteria style diet (Johnson and Kenny, 2010). Reduced levels of D2R have also been inversely associated with body weight (Johnson and Kenny, 2010) and DIO (Narayanaswami et al., 2013), similar to what had been seen in human obesity (de Weijer et al., 2011) and drug addiction (Murray et al., 2014). Although future research is needed, the above data demonstrate the addictive like responses engendered by highly palatable foods and obesity in preclinical models, which is similar to drugs of abuse.

3.2. Clinical

Since there are no strong evolutionary mechanisms able to override the temptations of the modern food environment, weight gain and obesity often result in susceptible individuals. Sugar and fat activate different regions within the brain; whereas fat appears to activate regions responsible for associative learning, sugar appears to activate areas of DAergic pathways (Stice et al., 2013). Differences in reward pathways are seen between lean and obese (Tuominen et al., 2015; Wang et al., 2014; Wang et al., 2001), giving credence to why certain individuals consume excess amounts of food despite the myriad of health risks associated with obesity. Findings from the neuroimaging literature support differential brain activation based on weight status in response to highly palatable foods, particularly an inverse association between body mass index (BMI) and BOLD activation within the dorsal striatum (DS) (Cosgrove et al., 2015) when using functional magnetic resonance imaging (fMRI). In addition, a recent PET study using [^{11}C]raclopride found an inverse relationship between DA release within the ventral striatum and BMI in response to consumption of a 75 g glucose drink (Wang et al., 2014). In a prospective fMRI study, overweight or obese women who gained weight over 6 months showed a decrease in activation within the right caudate in response to a HFHS milkshake when compared to baseline. This effect was not observed in women who were weight stable. These results support the notion that weight gain in of itself may contribute to a reduced striatal response to highly palatable food, which would increase the risk of overconsumption of these foods in order to receive the same pleasurable effects (Stice et al., 2010). However, genetic variation also plays a role on reward response to anticipation and receiving highly palatable food and weight gain. A recent large 3-year prospective study by Stice et al. (2015) conducted in normal weight adolescents found an interaction between those with the Taq1A allele (which is associated with altered DA signaling), BOLD response within the caudate upon receiving a milkshake, and future body fat gain. Collectively, these data show that weight status as well as genetic polymorphisms causing an excess or deficit of DA signaling may increase risk of overconsumption and body fat gain leading to obesity.

Another avenue of interest is the availability of striatal D2R/D3R in obesity compared to lean individuals. Discrepancies within the literature exist, with some data reporting lower baseline availability in obesity compared to lean individuals (van de Giessen et al., 2014; Wang et al., 2001) and other studies finding no difference (Eisenstein et al., 2013; Karlsson et al., 2015). Conversely, higher availability of striatal D2R/D3R in obesity has also been found. Recently, Cosgrove et al. (2015) found a positive association between BMI and D2R/D3R availability. However, since the relationship between BOLD response and D2R/D3R availability was unrelated, the authors posit the perturbation of reward response within the DS of individuals with an elevated BMI may not be resultant of D2R/D3R availability. Corroborating these findings was a positive association between D2- like receptor binding potential (D2BP) and BMI in the DS and the lateral striatum (LS). After correcting for BMI, D2BP in the LS was positively associated with opportunistic eating, which is associated with eating in the absence of hunger (Fay et al., 2015), suggesting a relationship between eating behavior, and not weight status on altered DA signaling (Guo et al., 2014). Inconsistencies within the literature may partially be due to different BMI distributions between studies. Further, different radiotracers used may also account for differential findings, as certain radiotracers compete with endogenous DA. In addition, many of these studies are cross-sectional, and therefore cannot infer causation. Yet, despite these limitations, a relationship between reward dysregulation and highly palatable foods is evident.

There are many scales within the literature used to measure different facets of maladaptive eating behaviors including the Emotional Eating Scale (van Strien et al., 2013), Power of Food Scale (Lowe et al., 2009), Eysenck Personality Questionnaire-Revised Addiction Scale (Lent and Swencionis, 2012), Dutch Eating Behavior Questionnaire, Eating Inventory and Food Craving Acceptance and Action Questionnaire (Juarascio et al., 2011; Williamson et al., 2007). Since its development in 2009, the Yale Food Addiction Scale (YFAS) is used to measure addictive-like behaviors toward specific types of foods. Although FA is not currently recognized as a clinical diagnosis, the YFAS applies the DSM-IV criteria for substance dependence over the previous 12 months (Gearhardt et al., 2009) and has recently been suggested as an effective tool to measure FA changes over time (Pursey et al., 2016). The YFAS has been utilized to assess behavioral addiction to food in clinical (Pepino et al., 2014) and non-clinical samples (Davis et al., 2011), as well as across different cultures (Brunault et al., 2014; Chen et al., 2015) and children (Gearhardt et al., 2013). FA appears to have a positive association with body weight (Murphy et al., 2013; Pedram et al., 2013; Raymond and Lovell, 2015), however the relationship may not be so simplistic (Meule, 2012), as other studies have found no such association (Berenson et al., 2015; Gearhardt et al., 2011). However, FA diagnosis seems to have higher prevalence in certain populations including women and individuals with eating disorder diagnosis (Gearhardt et al., 2014; Pursey et al., 2014). Recent evidence also supports a genetic basis for FA. By creating a multilocus genetic profile score (MLGP) comprised of 6 polymorphisms known to enhance DA signaling and reactivity within the ventral striatum, Davis et al. (2013) found individuals diagnosed with FA had higher MLGP scores compared to controls. These genetic alterations are also positively associated with binge eating, increased cravings and emotional eating (Davis et al., 2013). Notably, not all foods trigger addictive like consumption. Similar to drugs of abuse, which are rarely found in their natural state, foods that are highly processed and refined seem to be the most problematic for people. Foods high in sugar and fat, and typically low in fiber are quickly absorbed into the bloodstream and this increased "dose" likely contributes to the problematic eating behaviors associated with highly processed foods (Schulte et al., 2015). Overall, research in laboratory

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animals and humans alike support the FA construct given the evidence that certain food ingredients have the capacity to engender neural adaptations and addictive like behaviors.

4. Overlap among obesity, food addiction and nicotine

Like other drugs of abuse, both nicotine and highly palatable foods usurp the pathways responsible for reward, which contribute to the development of addiction. Indeed, the rates of smoking are higher for people in treatment for addiction (Guydish et al., 2015) and with morbid obesity (Levine et al., 2007) when compared to the general population, and is prevalent among people with eating disorders (Kelly-Weeder et al., 2014) supporting shared behavioral and biological correlates between obesity, food and nicotine addiction. However, nicotine is also known for its anorectic action and underlying influence on homeostatic mechanisms, making the relationship between smoking and weight an area of great interest.

Usually, people who currently smoke are leaner and have higher resting energy expenditure compared to people who never smoked (Al-Riyami and Afifi, 2003; Blauw et al., 2015; Plurphanswat and Rodu, 2014). Nonetheless, it is important to note that this inverse relationship is not linear, as higher odds of smoking have been found in individuals with morbid obesity compared to those who fall within normal weight range (Chatkin et al., 2010). In addition, long-term smokers are more likely to be overweight (Mackay et al., 2013), and heavy smokers are more likely to be obese than moderate and light smokers (Dare et al., 2015), Despite these findings, postcessation weight gain and increased appetite are common concerns and an unfortunate reality for many, as well as a deterrent for quitting (Aubin et al., 2012; Pisinger and Jorgensen, 2007; Scherr et al., 2015; Tuovinen et al., 2015). Further, people use smoking as an unhealthy way to control weight; with frequency of smoking being associated with binge eating as well as dieting (White, 2012).

Currently there has been no research specifically examining the relationship between FA construct and smoking. More of the focus has been on the effect nicotine has on neural and behavioral mechanisms implicated on hedonic feeding, since both affect the same reward related pathways within the brain. Interestingly, nicotine decreases hedonic hunger in non-smokers as well; when nicotine replacement gum was given during a fasted state to never- smokers, reduced functional coupling from the hypothalamus cluster and the NAc during palatable food presentations was seen (Kroemer et al., 2013). Further, non-smokers are more likely to respond to visual cues of palatable foods, including pizza and ice-cream, compared to individuals who smoke (Machulska et al., 2015), supporting the ability of nicotine to reduce responsiveness to the pleasurable aspects of palatable foods.

The long-term reward neuroadaptations induced by nicotine (Kenny and Markou, 2006) as well as biological susceptibility may contribute to the rewarding value of highly palatable food in the absence of nicotine. This in turn may increase the likelihood of subsequent weight gain that occurs in many people when attempting to quit smoking. One explanation may be due to altered expression of D2Rs in the striatum, which is seen in obesity as well as substance abuse (Blum et al., 1996; Kenny et al., 2013). The presence of the DRD2-A1 allele (which is associated with decreased D2R density (Pohjalainen et al., 1998)) is associated with decreased success of quitting smoking (Cinciripini et al., 2004) and have a greater response to food reward postcessation compared to carriers of the DRD2-A2/A2 allele. Further, after 6 months of abstinence from nicotine, increase in food reward was a positive predictor of weight gain, but this effect was attenuated in participants treated with bupropion, a DA transporter inhibitor (Fava et al., 2005; Lerman et al., 2004). In addition to genetic variation, evidence supports the relationship between nicotine deprivation and heightened responding for palatable food. During periods of nicotine deprivation, female smokers worked significantly harder for carbohydrate rich snacks over money, when compared to when they were not nicotine deprived (Spring et al., 2003). Moreover, when subjects who were attempting to quit smoking were randomized to a low carbohydrate diet or a moderately reduced fat diet, those who were in the low carbohydrate group reported higher withdrawal scores at week 4 and 12, suggesting carbohydrates, often found in highly palatable food, may play a role in lessening withdrawal symptoms (Heggen et al., 2015). However, another study found little behavioral evidence supporting the overlapping relationship between food cravings and cigarette smoking during periods of abstinence (Alsene et al., 2003) and therefore more research would be beneficial to better elucidate the relationship between nicotine and highly palatable foods.

Lastly, nicotine replacement therapy such as the nicotine patch or EC, have been used as a way to curtail appetite and prevent weight gain, although the results are conflicting and more research is needed (Farley et al., 2012). This effect may be dependent on rate of absorption and metabolism; just as foods reported to be more addictive are rapidly digested and absorbed (Schulte et al., 2015), the rate of nicotine absorption likely impacts appetite and food cravings during attempts of smoking cessation. It is well established that nicotine metabolism varies between individuals (Nakajima et al., 2006), and the type of nicotine replacement may also account for difference in study results. Since newer models of ECs have a faster rate of nicotine absorption compared to older models (Farsalinos et al., 2014), it can be speculated that newer models may have a more profound impact on appetite. However it is also important to consider the effect EC flavors may have on appetite and weight. While a wide variety of EC flavors are available, sweet flavors appear to be the most popular among consumers (Berg, 2016; Wang et al., 2015). Theoretically, sweet flavors may elicit a heightened reward response compared to flavors that are not sweet, which may partially explain why this flavor profile is reported as more enjoyable. Although this hypothesis has not yet been tested, examining whether sweet flavors of ECs can help alleviate sweet cravings would be an interesting avenue of study. However, since sweet flavors from an EC lack the post-ingestive consequences of caloric sweeteners, similar to artificial sweeteners that provide a sweet taste without calories (Low et al., 2014), the reward response may differ. Interestingly, there has been a significant increase in experimentation with EC among individuals who are overweight and obese, but it is important to note that it was not as a means of weight control (Strong et al., 2015).

Overall, future work specifically examining the overlap between the addictive nature of highly palatable foods and nicotine is needed in order to mitigate the negative consequences associated with both addictions. Not only will this provide a stronger foundation for favorable treatments to prevent postcessation weight gain that discourages quitting, but may also parlay into improved treatment for other addictions.

5. Social stigma between obesity and smoking

The word “stigma” dates back to the late 1500′s and is described as “as mark of disgrace (Anon, 2015a),” and often results in discrimination (Angermeyer and Matschinger, 2005). It is well established that many people have prejudice against individuals who are obese, and more recently, the same is observed against individuals who smoke. While smoking was once considered a normal behavior and socially acceptable, over the recent years smoking has become denormalized (Castaldelli-Maia et al., 2015). This public health effort is not only targeted at the tobacco industry, but also against the act of smoking itself. Indeed, quitting tobacco is associated with multiple health benefits (Anon, 2015b) however the denormalization of smoking affect people differently, causing some people to attempt quitting, while others feel frustration and punished (Betzner et al., 2012). Although the rates of smoking have decreased, some argue the stigmatization of tobacco may be detrimental to susceptible groups and negatively impact access to healthcare (Bell et al., 2010; Colgrove et al., 2011). For example, the stigma associated with the relationship between smoking and lung cancer is reported as a source of guilt and distress in oncology patients

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(Lehto, 2014). The discrimination felt by people who smoke often leads to change in smoking related behaviors (Ritchie et al., 2010), regret (Fong et al., 2004), as well as feelings of self-stigma (Evans-Polce et al., 2015). Although this may lead to reductions in smoking (Evans-Polce et al., 2015), failed attempts to quit may translate into internalization of negative feelings about oneself, and therefore warrants additional exploration (Castaldelli-Maia et al., 2015). In addition, due to the rapid increase in popularity of ECs, it is necessary to consider the stigma associated with its use. Users of ECs face higher stigma when compared to non-smokers (Brown-Johnson and Popova, 2016) and report similar ignominy as individuals who smoke traditional cigarettes, since they are often still viewed as a "smokers" (Soule et al., 2016). It will be interesting to see what affect the newer EC safety data has on discrimination and stigma associated with its use, as well as possible intra-group stigma among individuals who smoke electronic versus traditional cigarettes.

While the stigma associated with smoking has recently increased, efforts have been made to decrease the stigma associated with obesity (Poustchi et al., 2013; Puhl et al., 2014). Unfortunately, prejudice against individuals with obesity still exists. Individuals who are obese are often viewed as lazy and unintelligent, and lack self-control or willpower (Puhl and Heuer, 2009). Furthermore, women who are obese may be scrutinized to a greater extent than men (De Brun et al., 2014). A recent meta-analysis found that children who are overweight or obese are more likely to be victims of bullying compared to their children who are within the normal weight range (van Geel et al., 2014), and children who are severely obese are especially ostracized (Harrist et al., 2016). Excess weight and the associated stigma incontrovertibly negatively impacts physical and psychological health (Hunger and Major, 2015), however research shows that when there is stronger emphasis on the biological contribution of obesity, stigma appears to be decreased (Ebneter et al., 2011) and the negative connotation typically associated with obesity produces less blame when understanding the FA construct (Latner et al., 2014). However differences in perception exist, as individuals within the normal weight range are more likely to believe obesity is caused by personal choice, where as individuals who are obese are more likely to believe genetics and biology are to blame (Lee et al., 2014). Among overweight and obese weight loss seeking participants, those with higher FA symptom count exhibited greater internalized weight stigma and anti-fat attitudes toward themselves, however they were also more likely to dislike other individuals who are obese (Burmeister et al., 2013). Indeed, internalization of weight stigma likely exacerbates the negative consequences of excess weight on both physical and mental health. Individuals who are overweight or obese who internalize weight bias have greater fat phobia, but have less motivation to exercise and less self- efficacy to lose weight (Pearl et al., 2015). Given the effects of internalized weight bias, weight loss efforts are likely attenuated, further exacerbating the problem. Interestingly, some argue that by using FA as a cause of obesity, treatment strategies may change, with more emphasis placed on pharmaceutical approaches instead of lifestyle modifications. It has recently been shown that individuals who support the FA construct as a cause for obesity, support treatments that include education and therapy, and not be coercive (Lee et al., 2014). In conclusion, the stigmas associated with obesity and smoking have consequences on both an individual and treatment level and examining which groups are most susceptible to stigma warrant additional research. Nevertheless, regardless of the condition being stigmatized, internalizing bias dampens self-efficacy, likely leading to poorer health outcomes.

6. Conclusion

Although the short and long-term health ramifications resulting from overindulgence of highly palatable foods and cigarette smoking are well established, it is not enough to deter susceptible individuals from becoming addicted. Preclinical and clinical research has started to unravel the neuronal and behavioral overlaps seen between nicotine and highly palatable food on reward related regions within the brain. However, further insights into the underlying mechanisms contributing to nicotine and FA are still needed to fully elucidate the driving force behind these aberrant behaviors. A greater understanding of the shared neuropsychological parallels between obesity and addiction may lead to a more personalized treatment, leading to improved outcomes, ultimately improving quality of life.

Conflict of interest statement

The authors declare that there are no conflicts of interest.

Transparency document

The Transparency document related to this article can be found, in the online version.

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