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Link between Impulsivity and Overeating: Psychological and Neurobiological Perspectives

Dürtüsellik ve Aşırı Yeme Arasındaki İlişki: Psikolojik ve Nörobiyolojik Yaklaşımlar

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Öz

En temel tanımıyla dürtüsellik özdenetim olmadan davranma eğilimidir. Dürtüsellik eğilimi, aşırı yeme ve bununla ilgili belirtilerin ortaya çıkışı ve devamında da önemli bir rol oynamaktadır. Daha detaylı olarak; yüksek düzeydeki dürtüselliğin ve bununla ilişkili olan tepki kontrolünde zorluk, heyecana eğilim, ödül zafiyeti ve olumsuz duygulanım durumlarının, tıkınırcasına yeme bozukluğuna ve obeziteye kadar ilerleyebilen duygusal yeme üzerinde bir etkileşim etkisine sahip olduğu bulunmuştur. Bu derlemenin amacı, dürtüselliğin aşırı yeme üzerindeki olası rolüne yönelik bilişsel ve nöropsikoloji alanında yapılan çalışmalardan elde edilen kanıtları bir araya getirmek ve bunun yanında obezite ve tıkınırcasına yemek bozukluğunun tedavi yöntemlerine, dürtüsellik özelliği göz önüne alınarak yapılabilecek olası değişiklik ve eklemelere önerme yapmaktır.
Anahtar sözcükler: Dürtüsellik, aşerme, tıkınırcasına yeme, obezite, dışsal yeme örüntüsü.

Abstract

In most basic terms impulsivity is defined as a tendency to act without control. Impulsivity has been implicated in the onset, symptomatic expression, and maintenance of overeating. Specifically, high impulsivity and associated poor inhibitory control, along with sensation seeking and reward sensitivity, as well as negative affect have been found to have an interaction effect on emotional eating, which may lead to binge eating and obesity. Moreover, associations between overeating and impulsive behaviors such as difficulty in response inhibition, planning, attention, addiction, or risk taking etc. have been previously shown. The purpose of this review is to summarize the evidence from psychology regarding the potential role of the impulsivity trait in overeating, with implications on the treatments for obesity and binge eating.
Key words*:* Impulsivity, overeating, binge eating, obesity, external eating.

EATING behavior is a complex behavior associated with biological, psychological, environmental, and genetic factors (Berthoud 2004, 2011); and ultimately regulated by the brain to execute the decision to eat or not to eat. This decision can be driven by internal signals and/or external cues, with or without conscious awareness. Impulsivity, a personality trait broadly defined as acting without thinking and fully considering the consequences of the actions (Evenden 1999), is routinely associated with high-risk

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behaviors and decision making (Franken et al. 2008, Dir et al. 2013), and considered as one of the hallmarks of several psychiatric disorders (Nestor 2002) and addictions (Reynolds 2006, Gearhardt et al. 2009, Dick et al. 2010, Koob and Volkow 2010) as well as eating disorders (Waxman 2009) and 'food addiction' (Burrows et al 2017, Meule et al 2017).

Impulsivity is specifically implicated in the onset, symptomatic expression, and maintenance of binge eating and obesity as to explain why some people have a tendency to overeat. Numerous studies reported positive correlations between impulsivity and body mass index (BMI) in adults (Nederkoorn 2006a, Weller 2008, Jasinska et al. 2012, Davis 2013, Murphy et al. 2013, Meule and Platte 2015) and children (Fields et al. 2013). It is proposed that impulsivity may be a mediator modulating eating behavior towards obesity and increased food intake (Rydén et al. 2003, Nederkoorn 2006a, 2006b, Guerrieri et al. 2008, Weller et al. 2008, Kavakcı et al. 2011, Jasinska et al. 2012, Murphy et al. 2013), binge eating (Nasser et al. 2004, Guerrieri et al. 2007, 2009, Jansen et al. 2009, Racine et al. 2009, Manwaring et al. 2011, Pearson et al. 2014, Olsen et al. 2015), difficulty in weight maintenance (Weygandt et al. 2013, Sevinçer et al. 2014, Weygandt et al. 2015), and morbid obesity (Sarısoy et al. 2013), as well as fast food consumption (Garza et al. 2016) and tasty but unhealthy food choice in a preference task (Jasinska et al. 2012). A recent meta-analysis suggested that overweight and obese persons with binge-eating disorder (BED) exhibited impulsive behavior and greater sensitivity to reward, one of the facets of impulsivity (Stanford et al. 2009), than those without the disorder (Schag et al. 2013). BED, an eating disorder (APA 2013) characterized by frequent episodes of binge eating and loss of control, is considered an important contributor to weight gain and obesity, with approximately 42% of those with BED are indexed as obese (Kessler et al. 2013). Moreover, impulsivity trait was shown to be the best predictor of subsequent meal intake in patients with BED (Galanti et al. 2007) and impulse control problems were found to be more prevalent in patients with BED than other eating disorders (Fernandez-Aranda et al. 2008).

Several studies assessed the link between impulsivity and overeating in adolescents showing that impulsivity trait and its associated facets may contribute to the development of obesity in adulthood. Specifically, higher impulsivity has been shown in adolescents with binge eating (Nederkoorn et al. 2006b, Braet 2007, Drukker 2009, Duckworth 2010, Hartmann et al. 2010, Verdejo- Garcia et al. 2010, Fields et al. 2013) and adolescents with lack of control in eating, especially high calorie food (Nederkoorn et al. 2015). It is also reported that impulsivity assessed by high scores on two or more of its dimensions (vs. one dimension alone) predicted a greater risk of becoming obese in adulthood rather than overweight (Fields et al. 2013). Moreover, binge eating in adolescents were shown to associate with impulsivity facets (Mobbs et al. 2010, Stanford et al. 2009) such as negative urgency (Mikami et al. 2010) and tendency to engage in rash behavior when distressed (Fisher et al. 2012). Overall, results from the studies with children and adolescents appear to implicate a causal link rather than a mere correlation and a possible explanation for why a subset of adolescents are not successful in behavioral modification interventions for weight loss. Understanding the mechanisms underlying this causal link may contribute greatly for anti-obesity intervention and prevention efforts.

Obesity is a multifactorial phenomenon with various biological, psychological, and environmental factors as well as their interactions contributing to its development and maintenance. Similarly, psychiatric disorders are complex compositions of various behavioral traits and molecular mechanisms have been known to be involved in the development of these behavioral traits (Reif and Lesch 2003). It has been speculated that BED might represent a heightened neurobehavioral phenotype of obesity (Carnell et al. 2012) that are known to include changes in distinct psychological and cognitive processes that are associated with specific neural system malfunctions (Robbins et al 2012). Thus, in this review, the effects of impulsivity trait on overeating is examined from several perspectives using evidence from both psychological and neurobiological studies.

Evidence from Psychology

Physiological energy balance can be overriden by the cues (i.e. sight, smell, taste, sound, time, context, imagery etc.) from the environment triggering appetite and food intake even in the absence of a physiological energy deficit (Berthoud 2011). Eating in response to external food cues such as the sight and the smell (of food), instead of internal cues such as physiological hunger signals is termed as `external eating`. Higher external eating scores measured via Dutch Eating Behavior Questionnaire, the most commonly used self-report measuring external eating (van Strien et al. 1986), have been found to predict BMI (Jasinska et al. 2012) and positively correlate with binge eating (Popien et al. 2015, Sobik et al. 2005). In persons with obesity, external eating also positively correlated with impulsivity (Lyke and Spinella 2004, Elfhag and Morey 2008, Hou et al. 2011, Kakoschke et al. 2015). Specifically, high impulsivity was shown to associate with external eating-related constructs such as, food cue reactivity (Tetley et al. 2010, Van den Akker et al. 2013, 2014) and selective attention for food cues (Hou et al. 2011). The cue-reactivity, in this context, refers to the enhanced appetitive responding to food cues (Jansen et al. 2008), and is suggested to contribute to the prevalence of binge eating in persons with obesity (Jastreboff et al. 2013), BED (Jansen 1998), or bulimia nervosa (Jansen et al. 1989, Sysko et al 2017), an eating disorder that is associated with poor inhibitory control (Waxman 2009).

An indirect evidence for the role of impulsivity in overeating may be deduced from the studies showing that conscientiousness, a personality trait that is opposite to impulsivity, promotes healthy eating behavior and decreases unhealthy eating behavior (Goldberg and Strycker 2002, Mottus et al. 2012, Lunn et al. 2014, Gerlach et al. 2015, Olsen et al. 2015). Olsen and colleagues (2015) reported that people who scored high on conscientiousness displayed a lower tendency to eat on impulse and tended to plan their following meal, in comparison to people who scored lower in conscientiousness ratings. Moreover, conscientiousness was also shown to have negative correlations with binge eating episodes (DSM-5) (Koren et al. 2014) and external eating (Elfhag and Morey 2008). Thus, it seems plausible that external eating contributes greatly to the etiology of overeating in today`s food environment and impulsivity trait may be a susceptibility factor for the effects of external eating.

Numerous studies, including the ones failing to find a strong relationship between external eating and overweight status (Wardle 1987, van Strien et al. 2009) suggest that high external eating may be a characteristic of a subset of overweight people who are also high on impulsivity (van Strien et al. 2009). It is possible that impulsivity may be triggering a facilitated acquisition of a conditioned response of eating when the right cue is present (Van den Akker et al. 2013). In other words, impulsivity may increase cue-reactivity which results in, what is defined as, the external eating. In Pavlov's classical conditioning, the learning of an association between a neutral cue and a biologically relevant cue–results in a biologically relevant response to the neutral cue which then becomes the conditioned stimulus and the biologically relevant response to the conditioned stimulus is called the conditioned response (Pavlov 1927). In external eating, it is assumed that the learned associations between the food and the external cues may trigger a conditioned response: the appetite. Thus, any among the abundance of external food cues in today's world (Wardle 1990, Harris et al. 2009, Hermans et al. 2012,) may function as a conditioned stimulus, triggering a conditioned response (external eating), especially in persons who are attentive and sensitive to these cues and/or impulsive (Wardle 1990, Jansen 1994).

The food cues were also shown to trigger eating-related physiological mechanisms (e.g. insulin and glucose responses, salivation etc.) along with the conditioned response of external eating (Berthoud 2011) and to modulate the sensitivity of the circuitry of the brain towards food rewards, ultimately affecting appetite (Volkow et al. 2011). Hunger driven by starvation defends a physiological energy state for survival and occurs mainly without awareness, thus is referred to as 'homeostatic hunger' (Berridge et al. 2010). Food cues can override homeostatic hunger and may trigger eating even in the absence of a physiological energy deficit (Berthoud 2011) which is referred to as hedonic hunger; a non-homeostatic system governed by a different neural circuitry than the network underlying homeostatic hunger (Levitsky 2005, Lowe and Butryn 2007). Today, we live in cue-abundant, obesogenic food environment in which palatable and calorie-dense foods are easily and readily available (Rolls 2003, Drewnowski 2004, Nestle 2006, Carnell and Wardle 2008, Andreyeva et al. 2011). This may cause the cue-triggered hedonic eating to override the hunger-driven homeostatic eating and inevitably contribute overeating. Indeed, obesity has been associated with more hedonic eating -than homeostatic, and with hypersensitivity to these everyday cues (Blundell and Finlayson 2004, Giesen et al. 2010). Thus, although the reasons for being more vulnerable to the effects of these environmental cues are still being investigated, the hypothesis of impulsivity predisposing one to external eating and overeating appears highly plausible.

Evidence from Neurobiology

The neurotransmitter dopamine (DA) plays a critical role in reward and pleasure (Kringelbach and Berridge 2010). Alterations in DA signaling have been implicated in the vulnerability for various psychiatric disorders including eating disorders (Frieling et al. 2010), as well as compulsive overeating (Doehring et al. 2009), and obesity (Stoeckel et al 2008). Functional magnetic resonance imaging studies revealed that in response to high calorie food cues, greater activity in dopaminergic neural pathways was found in obese (vs. normal-weight) individuals (Beaver et al. 2006, Rothemund et al. 2007, Stoeckel et al. 2008, Batterink et al. 2010) and obese binge eaters (vs. obese non-binge eaters) (Geliebter et al. 2006), implicating a higher reward value of food for overweight and obese individuals. Moreover, the incentive value of the food has been shown to increase independently of the weight status: self-reported hunger ratings are positively correlated with DA activity in the striatum -one of the neural sites related to processing reward and pleasure (Berridge and Kringelbach 2008), during food-cue exposure (Volkow et al. 2002) suggesting that in a state of hunger, food may be rendered as more rewarding (Lowe and Butryn 2007). Overall, it is well documented that stimuli resulting in increased striatal DA are experienced as rewarding and cause motivation to attain the reward-pertaining stimulus, potentially leading addicted behavior (Volkow et al. 2002, 2014).

Interestingly, although higher dopaminergic activity in striatum has been associated with overeating and obesity, recent evidence also shows lower dopaminergic signaling in relation to overeating. Positron emission tomography scan studies, measuring DA signaling in humans in vivo, showed decreases in striatal DA receptor (D2) availability and binding in persons with obesity (Wang et al. 2001, 2004, Haltia et al. 2007, Volkow et al. 2008). It is possible that the overactivity of the striatal dopaminergic system by chronic exposure to a reward (i.e. food) in obese individuals may result in reduced density and sensitivity of DA receptors, possibly as an adaptation to high DA, ultimately resulting in low dopaminergic activity. This adaptation process, when considering the high incentive value of reward (i.e. food) in obesity especially when combined with the state of hunger regardless of the weight status, may promote further compulsive food intake (Ifland et al. 2009). Thus, it is hypothesized that overeating may also occur as an individual's attempt to compensate for dopaminergic hypoactivity (Tomasi and Volkow 2013).

Although the dopaminergic involvement in eating behavior is far more complicated and how adaptive changes in the DA circuitry associate with obesity and overeating remains to be elucidated and is beyond the scope of this review, the discrepancy between the studies showing increased and decreased DA activity in overeating may be partially explained by the differential roles of DA (O'Doherty et al. 2004). DA is the key modulator within the meso-limbo-cortical system that is originating in the ventral tegmental area project to the ventral striatum, specifically to the nucleus accumbens (mesolimbic pathway), and to the dorsal striatum and prefrontal cortex (mesocortical pathway). The mesolimbic pathway is implicated in the actual reward processing and immediate reward value (Baldo and Kelley 2007, Jimura et al 2013) and increased ventral striatum activity was also reported in impulsive individuals (Balodis et al. 2012, Jimura et al. 2013). The mesocortical pathway on the other hand, has been shown to have a role in motor control (Toni and Passingham 1999), modulation of stimulus-response or stimulus-response-reward associations (Robinson et al. 2009, Ghahremani 2012), reward prediction error (Schultz, 2012), delayed discounting -defined by reward devaluation with delay to its receipt (Volkow and Baler 2015) and cognitive flexibility (Robbins et al. 2012). In persons with BED, hypoactivity in the prefrontal cortex was shown to be related to increased impulsiveness and decreased response inhibition (Hege et al. 2014). Moreover, lower white matter integrity within mesocortical system is associated with higher choice impulsivity (delayed discounting task) in normal-weight individuals (Peper et al. 2012). The hypoactivity in dopaminergic activity in mesocortical system may be a neurobiological marker of impulsivity, increasing the vulnerability to engaging in the compulsive behavior and thus to overeat by failing to inhibit the overactive mesolimbic DA system that is signaling high reward value (Ongur and Price 2000, Kelley 2004, Fudge et al. 2005, Kelley et al. 2005, Geliebter et al. 2006, Gainetdinov 2007, Wang et al. 2009). Thus, together, the mesolimbic and the mesocortical pathways are involved in the integration of complex appetitive and aversive predictions to coordinate the behavior towards gratification.

The transition from voluntary to habitual and progressively compulsive addictive behavior has been associated with the transition of the dopaminergic projections from the ventral to dorsal striatum, with a possible reduction in inhibitory cortical control (Everitt and Robbins 2013). Supporting this, decreased availability of a specific group of dopamine receptors (D2R) in the dorsal striatum (i.e. putamen) in morbidly obese participants (Wang et al 2001) and reduced activity in dorsal striatum (i.e. caudate nucleus) in response to palatable food consumption (Stice et al. 2008) have been reported. Moreover, persons with putamen lesions were reported to have compulsive and repetitive stereotypic behavior (Uher and Treasure 2005, Slama et al. 2012,). Activity in the dorsolateral prefrontal cortex –a brain area has been known to be related to the impulse control and cognitive inhibition (Diamond 1990, Volkow and Baler 2015), predominantly receives projections from dorsal striatum (i.e. putamen), was found to be negatively correlated with body weight (Volkow et al. 2009, Kishinevsky et al. 2012). Higher activity in this area was associated with healthy food choices (Killgore and Yurgelun-Todd 2005, Hare et al. 2009, Batterink et al. 2010), and also shown to predict real-world diet success in obese patients at a 12-week intervention (Weygandt et al. 2013) and at 1-year follow-up (Weygandt et al. 2015).

Evidence from Therapies for Obesity

Cognitive and Behavioral Therapies

Impulsivity has been speculated to be a predictor of anti-obesity treatment outcomes; i.e. lower weight loss in obese children with higher impulsivity was reported following cognitive behavior therapy (CBT) (Nederkoorn et al. 2007, Koren et al. 2014). Moreover, some patients, following weight loss achieved by bariatric surgeries, were reported to develop problems in impulse control (Sevincer 2016). A wide range of successful CBTs (Cooper et al. 2010) targeting subconscious processes leading to 'mindless eating' has been developed to improve human decision making towards healthy food choices. These methods employ several strategies to enhance the cognitive and behavioral control of the eating behavior (Oğuz et al. 2016), and decrease the high reward signaling in the brain by reframing of a reward, or both, with the ultimate aim of replacing 'mindless eating' with 'mindful eating' (Dickenson et al. 2013, Wansink and Chandon 2014).

Cue reactivity and reward sensitivity may be reduced by a method called cue exposure with response prevention (CERP), in which the association between the food-cues and the binges are extinguished (Jansen et al. 1989, Toro et al. 2003 Martinez-Mallén et al. 2007, Frankort et al. 2014). During CERP treatment for binge eating, Pavlovian conditioning is broken down: the association between the food and the food cue (i.e. smell, context etc) is eliminated with a repeated food cue exposure without access to the food, and thus a conditioned stimulus no longer predicts the unconditioned stimulus, resulting in the extinction of the conditioned response; the appetite. Current findings from studies, in which elimination of cue reactivity is shown to decrease external eating, imply that CERP could be more beneficial for persons high in impulsivity. Decremental effects of CERP on chocolate craving (Van Gucht et al. 2008, Frankort et al. 2014) and bulimia nervosa (Jansen et al. 1992) as well as other addiction disorders (i.e. smoking, alcohol dependency) (Choi et al. 2011, Vollstädt-Klein et al. 2011) have been previously shown, however its effectiveness is yet to be validated further to consider CERP as a behavioral modification supplement for weight loss programs for a subset of individuals with high impulsivity.

Habits, unlike goals, are initiated effortlessly and automatically, with minimal or no liaison with the effort conscious cognitive control and attention, and so the behavior may easily be triggered by the environmental cues at all times. Thus, the automation of the associations between cues and responses may be extinguished by a strategy of goal setting (Kivetz et al. 2006) which aims to bring in more conscious effort on the choice process and make implicit processes explicit (Paulus et al. 2013, Koningsbruggen et al. 2014). It also increases introception of which dysfunction have been suggested to play a role in cue-triggered drug use in addiction (Paulus et al. 2009). A no-go training, in which participants learn to withhold the impulse of eating behavior towards the palatable food by a bottom-up process also resulted in significantly lower consumption (Houben and Jansen 2011, Koningsbruggen et al. 2014). Moreover, eliminating the impulsive response before it is triggered by the stimulus, via a training on inhibiting the selective and narrowed attention towards the stimulus (i.e. food and/or food cue) rather than inhibiting the impulsive response upon capturing the stimulus, has been shown to be effective (Brooks et al. 2011, Houben and Jansen 2011, Giel et al. 2013, Fields et al 2017). A precommitment strategy for a future course of action without the need for reliance on willpower has also been proposed (Camerer et al. 2004) in which a decision for a future choice is made now when in a "cold state" rather than deciding in the face of a present choice when in a "hot state" and an impulsive choice is more prevalent (Metcalfe and Mischel 1999, Loewenstein 2000, Hanks et al. 2013). Overall, reductions in the indulgence of the reward and food-related impulsive choice may be achieved by various cognitive strategies such as, eliminating the established association between the attention-grabbing cues and the impulsive go response, inducing cognitive control, and strengthening the influence of deliberate processes such as long-term health goals on behavior. Thus, cognitive and behavioral strategies targeting impulsivity appears to contribute to the effectiveness of the weight loss regimes, which also implies an important role for impulsivity in overeating.

Neural Stimulation Therapies

Non-invasive brain stimulation enables targeted manipulation of cortical excitability, and is proposed to reverse the 'assumed' altered neural circuitry in various types of psychiatric disorders as well as overeating (Schlaepfer et al. 2010, Berlim et al. 2011). The most common modalities are transcranial direct current stimulation (tDCS) and repetitive transcranial magnetic stimulation (rTMS), a variant of TMS involving repetitive pulses to induce longer-lasting changes in cortical activity in experimental and clinical contexts. Although moderate in magnitude, recent meta-analysis (Lowe et al. 2017) supported a causal effect of neuromodulation of the dorsolateral prefrontal cortex on food cravings via rTMS and not tDCS. rTMS of dorsolateral prefrontal cortex has been shown to reduce subjective ratings (e.g. urge to restrict, feeling full etc.) during the exposure to visual and real food stimuli (van den Eynde et al. 2010) and values assigned to food (Camus et al. 2009). Opposite results reported (Figner et al. 2010) may be explained by the differences in pulse strength as high frequency (>1 Hz) stimulation tends to increase activity while low frequency stimulation tends to depress activity (di Lazzaro et al. 2005). Moreover, improvements in delay discounting, an effective marker for intervention success for obesity and other addiction disorders (Koffarnus et al. 2013), have shown following TMS applied to the dorsolateral prefrontal cortex (Cho et al. 2010).

Decreases in sweet cravings, total and preferred food intake, desire to binge eat following tDCS in persons with BED have been reported (Burgess et al. 2016) however, impulsive tendencies in choice behavior were shown to be less susceptible to the ameliorating effects of tDCS (Kekic et al. 2014). Although in the same study, single-session tDCS has not been effective in lowering food cravings, same group, in a recent study (Kekic et al. 2017), also reported transient improvements on the wanting/liking of food and on bulimic behaviors during the 24-hour post-stimulation following three sessions of bilateral tDCS in patients with bulimia nervosa. However, in healthy-weight women, single-session unilateral tDCS has not been effective to reduce food cravings, high caloric food choice and calorie intake (Georgii et al. 2017).

Deep brain stimulation (DBS), an invasive brain stimulation method and a reversible neurosurgical procedure that drives continuous and high-frequency stimulation of the targeted brain areas (Schlaepfer et al. 2010), has been proposed for treatment of obesity and Prader Willi syndrome (Ho et al. 2015) –a genetic disorder causing compulsive eating and obese phenotype (Bittel et al. 2005). Moreover, DBS has also shown to be effective in treating alcoholism (Müller et al. 2009) and obsessive-compulsive disorder (de Koning et al. 2011, Figee et al. 2013) in which impulsivity is known to be a common trait. Overall, although future research needs to specify which appetitive behaviors can be modulated by brain stimulation and which populations might profit from it the most, current results appear to confirm the link between the impulsivity trait and overeating.

Evidence from Genetics

Impulsivity has been shown to be partially heritable (Schachar et al. 2011, Anokhin et al. 2015). Studies showed a common genetic etiology between overeating and impulsivity (and related aspects) (Kuntsi et al. 2006, Wood et al. 2010, Schachar et al. 2011, Kamijo et al. 2012, Nederkoorn et al. 2012, Bevilacqua and Goldman 2013, Crosbie et al. 2013, Frazier-Wood et al. 2014, Anokhin et al. 2015, Filbey and Yezhuvath 2017) suggesting impulsivity to be a heritable and an intermediary trait predisposing individuals to overeating (Gottesman and Gould 2003, Doyle et al. 2005, Flint and Munafo 2006). Specifically, several mutations on the expression of the encoding genes that are involved in monoamine neurotransmission in the brain such as, dopamine transporter (Forbes et al. 2009, Paloyelis et al. 2010), catechol-O-methyl transferase (COMT) -an enzyme responsible from DA degradation in the prefrontal cortex (Bottiger et al. 2007, Soeiro-De-Souza et al. 2013), DA receptors (D2, D4, D2/D3) (Eisenberg et al. 2007, Forbes et al. 2009, White et al. 2009, de Weijer et al. 2011, Trifilieff and Martinez 2014,), as well as serotonin transporter (Lesch et al. 1996, Steiger 2005,) and receptors (Bevilacqua and Goldman, 2013), have been commonly implicated in the impulsivity trait. Supporting this, several of these neural sites have been targeted by the pharmaceutical therapies for obesity and binging (Smith et al. 2010, Bai and Wang 2010, Halford

2010, Bello and Liang 2011, Fidler et al. 2011, O'Neil et al. 2012, Balodis et al. 2014, Vickers et al. 2017).

Conclusion

In the light of these findings, impulsivity trait seems to appear as an endophenotype predisposing a person overeating and possibly obesity (Robins et al. 2012). Understanding the degree of mediation of the eating behavior by the impulsivity trait and elucidating whether impulsivity is an endophenotype for overeating, may allow researchers and health care professionals to better explain the resistance to lifestyle interventions (Gottesman and Gould, 2003) and target impulsivity for more effective obesity interventions (Winkler et al. 2012, Roth et al. 2013). Overall, in the absence of robust long-term changes in our cue-laden environment, in which individuals on a daily basis have to make immediate decisions on an optimal choice among a vast number of tempting palatable foods, anti-obesity interventions that target impulsivity trait seem to be not only effective but also necessary, at least in a subgroup of obese individuals or BED patients with high impulsivity.

Specifically, the interplay between the genetic and neurobiological impulsivity markers and the neuropeptides and gut hormones involved in body weight regulation, must be addressed in future studies with the goal of tracking shared genetic factors and their contribution to the neurobiological bases. Single photon emission computed tomography studies of regional kinetic uptake using radioligands for dopamine and serotonin transporters in combination with functional neuroimaging and neural connectivity approaches, as well as methods for investigating the biomarkers and genetic substrates such as metabolomics, proteomics, genomics, epigenetics, optogenetics, will help elucidating the possible role of other neurotransmitters and neuropeptides besides the dopamine and serotonin systems.

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