Journal section: review for special topic on Addictions

Uncontrolled eating:

A unifying heritable trait linked with obesity, overeating, personality, and the brain

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Abstract

Many eating-related psychological constructs have been proposed to explain obesity and over-eating. However, these constructs, including food addiction, disinhibition, hedonic hunger, emotional eating, binge eating, and the like all have similar definitions, emphasising loss of control over intake. As questionnaires measuring the constructs correlate strongly ($r>0.5$) with each other, we propose that these constructs should be reconsidered to be part of a single broad phenotype: uncontrolled eating. Such an approach enables reviewing and meta-analysing evidence obtained with each individual questionnaire. Here, we describe robust associations between uncontrolled eating, body mass index (BMI), food intake, personality traits, and brain systems. Reviewing cross-sectional and longitudinal data, we show that uncontrolled eating is phenotypically and genetically intertwined with BMI and food intake. We also review evidence on how three psychological constructs are linked with uncontrolled eating: lower cognitive control, higher negative affect and a curvilinear association with reward sensitivity. Uncontrolled eating mediates all three constructs' associations with BMI and food intake. Finally, we review and meta-analyse brain systems possibly subserving uncontrolled eating: namely, (i) the dopamine mesolimbic circuit associated with reward sensitivity, (ii) frontal cognitive networks sustaining dietary self-control, and (iii) the hypothalamus-pituitary-adrenal axis, amygdala and hippocampus supporting stress reactivity. While there are limits to the explanatory and predictive power of the uncontrolled eating phenotype, we conclude that treating different eating-related constructs as a single concept, uncontrolled eating, enables drawing robust conclusions on the relationship between food intake and BMI, psychological variables, and brain structure and function.

Introduction

Obesity is the result of a sustained excessive energy intake (Hall, 2018), and energy intake is 100% accounted by behaviour (Blundell & Finlayson, 2004). Further, evidence from genetics and brain imaging suggest that obesity's causes are rooted in the brain (Dagher, 2012; Vainik *et al.*, 2013, 2018; Locke *et al.*, 2015). Therefore, individual differences in obesity should at least partly be explained by individual differences in behaviour. Here we seek to provide an overview of a proposed unified psychological construct associated with overeating and obesity: Uncontrolled Eating. Although the current paper is intended for a special topic on addictions, we have refrained from framing current discussion from a food addiction perspective, as for reasons outlined below, many associations found with a "food addiction" questionnaire are equally likely to be found with other eating-related questionnaires.

Uncontrolled Eating as the intersection of different eating-related traits

Uncontrolled eating has been created as an umbrella term for several correlated psychological constructs. These constructs are measured by questionnaires that ask about various types of overeating (see Table 1). The name uncontrolled eating was inspired by Karlsson (2000) and chosen because it seems to most closely track the content of the questions in the commonly used eating behaviour questionnaires (Vainik, Neseliler, *et al.*, 2015). Nonetheless, the indicators of uncontrolled eating are rather diverse. As summarised in Table 1, some questionnaires are intended to measure loss of control over eating (binge eating, uncontrolled eating, loss of control over eating), whereas others target the reasons behind overeating, like negative emotions (emotional eating), exposure to appetising food (external eating, hedonic hunger, reward-based eating drive), inability to restrain food intake (disinhibition), not feeling satiated (susceptibility to hunger), craving for food, and even outright food addiction. Despite the diverse names, the

commonly used eating trait questionnaires tend to have strong correlations with each other (r>0.5) (Price *et al.*, 2015; Vainik, Neseliler, *et al.*, 2015; Mason *et al.*, 2017), suggesting that people do not differentiate well between the different reasons for overeating, at least while answering questionnaires. Intriguingly, whenever a new eating-related questionnaire is developed, the questionnaire tends to be validated by having r>0.5 correlations with existing eating-related questionnaires (Gearhardt *et al.*, 2009; Lowe *et al.*, 2009; Ruddock *et al.*, 2017), but is still proposed as measuring something new.

Table 1. Constructs that fit under the Uncontrolled Eating umbrella. Definitions are chosen from the original questionnaires or integrative reviews.

Construct	**Definition**	**Example questionnaire**
Binge eating	"Binge eating disorder is defined as recurring episodes of eating significantly more food in a short period of time than most people would eat under similar circumstances, with episodes marked by feelings of lack of control. Someone with binge eating disorder may eat too quickly, even when he or she is not hungry. The person may have feelings of guilt, embarrassment, or disgust and may binge eat alone to hide the behavior. This disorder is associated with marked distress and occurs, on average, at least once a week over three months." (American Psychiatric Association, 2013a, 2013b).	Binge Eating Scale (Gormally *et al.*, 1982)
Disinhibition Also: 'Opportunistic Eating' 'Readiness to Eat'	"A tendency towards overeating and eating opportunistically in an obesogenic environment. Examples include eating in response to negative affect, overeating when others are eating, not being able to resist temptations to eat, and overeating in response to	Three-Factor Eating Questionnaire (Stunkard & Messick, 1985)

and 'Thrifty Behaviour'	the palatability of food." (Bryant *et al.*, 2008) (p. 409)	
Emotional Eating	"Eating behavior in response to negative emotions" (Bongers & Jansen, 2016) (p. 1)	Dutch Eating Behaviour Questionnaire (Tatjana van Strien, Frijters, Bergers, & Defares, 1986). Others reviewed by: (Bongers & Jansen, 2016; Frayn & Knäuper, 2017)
External Eating	"Eating in response to food-related stimuli, regardless of the internal state of hunger or satiety" (van Strien *et al.*, 1986) (p. 296) Being affected by hedonic appeal of the food by sensory cues (e.g., palatability) (Herman & Polivy, 2008)	Dutch Eating Behaviour Questionnaire (van Strien *et al.*, 1986)
Food addiction	"…there is no consensus that [food addiction] (FA) is a clinical disorder nor is there a universally accepted definition for FA. A widely used definition for FA has emerged by mapping the DSM-IV diagnostic criteria for substance dependence to eating behaviors. These include: tolerance, withdrawal symptoms, larger amounts consumed than intended, persistent desire or unsuccessful attempts to cut down, much time spent using or recovering from substance, continual use despite knowledge of consequences, activities given up due to use of substance" (Pursey *et al.*, 2014) (p. 4553)	Yale Food Addiction Scale (Gearhardt et al., 2009)

Food craving	"An intense desire to eat a specific food item (Weingarten & Elston, 1990; Nijs *et al.*, 2007) (p. 38)	Food Cravings Questionnaire-Trait (Cepeda-Benito *et al.*, 2000; Nijs *et al.*, 2007)
Hedonic hunger. Also: appetitive drive to consume highly palatable food	2009: "Individual differences in appetite-related thoughts, feelings and motivations in environments where plentiful palatable foods are constantly available." (Lowe *et al.*, 2009) (p. 114) 2018: "hedonic hunger reflects something beyond a normative desire to eat delicious foods. It appears to identify the minority of individuals for whom such foods are particularly compelling and have cognitive (in terms of food preoccupation), affective (in terms of powerful yearnings and cravings), and behavioral (in terms of the experience of [loss of control] eating episodes) implications." (Espel-Huynh et al., 2018). (p. 8)	The Power of Food Scale (Lowe et al., 2009)
Loss of control over eating	(Loss of control over eating) "is a psychopathology construct that is uniquely associated with distress and impairment, disturbed eating behaviour, and weight-related factors in both cross-sectional and prospective studies, independent of episode size and body weight." (Goldschmidt, 2017) (p. 444)	Loss of Control over Eating Scale (Latner *et al.*, 2014)
Reward-based eating drive	"Normative but compulsive syndrome of strong eating drive", which includes a "cluster of behavioral symptoms – excessive drive to eat that results from feelings of lack of control, diminished satiety, and preoccupation with food" (Epel et al., 2014) (pp. 1-2)	The Reward-Based Eating Drive Scale (Mason *et al.*, 2017)

Susceptibility to hunger	'The extent to which hunger feelings are perceived and the extent to which such feelings evoke food intake. For example, feeling so hungry an individual eats more than three times per day, or feeling so hungry that their stomach feels like a bottomless pit." (Bryant *et al.*, 2008) (pp. 409-410)	Three-Factor Eating Questionnaire (Stunkard & Messick, 1985)
Uncontrolled Eating	"Overall difficulties in the regulation of eating" (Karlsson *et al.*, 2000) (p. 1718) "The tendency to eat more than usual because of a loss of control over intake" (Cappelleri *et al.*, 2009)(p. 612)	Three-Factor Eating Questionnaire R-18 & R-21 (Karlsson *et al.*, 2000; Cappelleri *et al.*, 2009)

Granted, there may be experimental situations where a specific questionnaire might be better at predicting food intake than others (van Strien *et al.*, 2012). In most cases, however, this is not the case (Bongers & Jansen, 2016; Frayn & Knäuper, 2017). The incremental validities of each eating questionnaire to predict an outcome, such as food intake or weight gain, should be rigorously demonstrated (Vainik, Neseliler, *et al.*, 2015; Westfall & Yarkoni, 2016; Vainik & Meule, 2018) and not just assumed to exist because the questionnaires happen to have different names. Having different names for the same underlying construct, the "jangle fallacy", creates confusion and segregation of research (Kelley, 1927). As the jangle fallacy seems common in obesity research (Vainik, Neseliler, *et al.*, 2015; Vainik & Meule, 2018) we take the approach of summarising the evidence found with any questionnaire measuring any construct in Table 1 as a quest to summarise evidence on the core concept of uncontrolled eating.

This approach is based on the common explanation of the jangle fallacy –the presumed existence of a single latent construct that causes variance in all eating-related traits (Borsboom, 2006; Vainik, Mõttus, *et al.*, 2015; Vainik, Neseliler, *et al.*, 2015). Therefore, each questionnaire in

Table 1 is an interchangeable indicator of the uncontrolled eating construct. This assumption leads to the indifference of indicator principle (Spearman, 1927) - evidence collected with each single questionnaire/indicator should also apply to other questionnaires/indicators and to general uncontrolled eating. We therefore review the literature form this perspective, while attempting to be more integrative than previous reviews that have focused only on one aspect of uncontrolled eating and did not consider the cross-talk between the measurement traditions (Bryant *et al.*, 2008; Herman & Polivy, 2008; Macht, 2008; Pursey *et al.*, 2014; Espel-Huynh *et al.*, 2018).

The main goal of mapping out the correlates of uncontrolled eating is to simply organise the literature – what are the typical behaviours, constructs, and brain features linked with the common uncontrolled eating construct? Even if the common latent construct model happens to be ultimately incorrect, there is value in documenting the associations. To draw a parallel with IQ – while the mechanism that causes similarity between indicators of IQ is debated (Van Der Maas *et al.*, 2006), measures of global IQ are still useful for predicting outcomes such as job performance and health (Gottfredson & Deary, 2004).

Further, such mapping of correlations paves the way for transforming uncontrolled eating from a statistically derived construct into an observable phenotype - a set of observable characteristics of an individual resulting from the interaction of their genotype with the environment. For instance, concurrent validity is offered by uncontrolled eating's potential associations with concrete observable physical phenotypes or behaviors, such as body mass index, weight change, amount of food eaten, and eventually brain imaging measures. To a lesser extent, there is also value in relating uncontrolled eating to other psychological constructs, as those constructs are often related to observable phenomena. For instance, dimensions of impulsivity are related to alcohol abuse, pathological gambling, and antisocial behaviour (e.g., Whiteside *et al.*, 2005).

In contrast, listing the heritability coefficients and brain correlates of uncontrolled eating should not be viewed as proof that uncontrolled eating undoubtedly has biological roots. It is well known that most observable psychological or behavioral features are heritable to some degree (Turkheimer, 2000), and many constructs have measurable brain correlates (Yarkoni *et al.*, 2011). Rather, we hope that the coefficients and associations listed in the review will be viewed as a starting point in establishing biological roots and inspire further research.

Uncontrolled eating as a continuum

Uncontrolled eating is seen as a continuum trait, as opposed to conventional binary "all or nothing" diagnostic labels. In psychiatry, considering psychopathology as dimensional traits may better depict inter-individual differences (Kozak & Cuthbert, 2016). The 5th version of Diagnostic and Statistical Manual of Mental Disorders (DSM-5) has also introduced severity levels for binge eating disorder, a clinical manifestation of uncontrolled eating (Dakanalis *et al.*, 2017). Preclinical levels of uncontrolled eating might also vary on a spectrum (Davis, 2013). At the lower end of the spectrum, eating behaviour can be regarded as "passive overeating", occurring in the absence of impulsive/compulsive symptoms. At the opposite extreme, overeating is characterised by binge eating episodes and by severe episodes of loss of control over food ingestion (Davis, 2013).

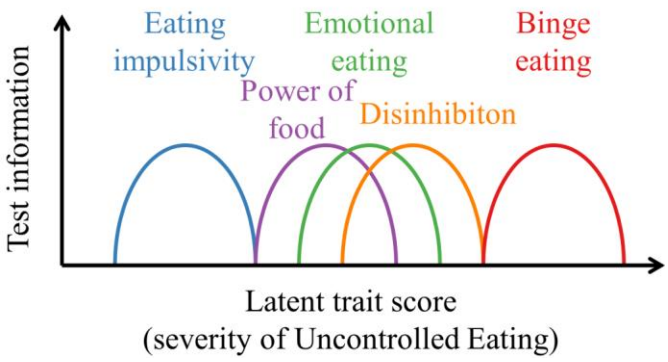

Figure 1. Idealised representation of questionnaires based on the severity of Uncontrolled Eating they measure best. Colored lines are Test Information Curves provided by Item Response Theory analysis (Baker, 2001). On the x axis, the Test Information Curves outline the region in the latent trait score they measure the best. On the y axis, Test Information reflects the factor loading to the latent trait. As this is a conceptual diagram, all questionnaires have been depicted to have equal loading to the common latent trait. The figure has been redrawn to conceptually reflect the empirical results of Vainik et al (2015).

Our group has provided preliminary empirical evidence that pre-clinical variation in uncontrolled eating is well captured by a measurement model that combines different uncontrolled eating questionnaires into a single factor (Vainik, Neseliler, *et al.*, 2015). Some questionnaire items, such as eating impulsivity, a subset of uncontrolled eating-related items from Impulsiveness subscale of NEO-PI questionnaire (e.g., "I sometimes eat too much") (Costa & McCrae, 1992; Vainik, Mõttus, *et al.*, 2015; Vainik, Neseliler, *et al.*, 2015) seem to reflect the lower end of uncontrolled eating pathology. Most questionnaires capturing emotional eating and hedonic eating might explain variance in the middle part of the spectrum, while questionnaires capturing symptoms of binge eating might reflect the severe end of uncontrolled eating (summarised in Figure 1). On their own, each questionnaire might not encompass the full spectrum of uncontrolled eating. To overcome this issue, Mason et al. constructed the Reward-related Eating Drive (RED-13) questionnaire that covers variance spanning from mild to very severe forms of uncontrolled eating (Mason *et al.*, 2017). This questionnaire has been recently been shortened (Vainik *et al.*, In press) and highlighted as a core measure in the ADOPT initiative (Accumulating Data to Optimally Predict Obesity Treatment) (Sutin *et al.*, 2018).

Cross-sectional studies link uncontrolled eating to food intake and BMI.

Uncontrolled eating measures an individual's tendency to overeat. Higher scores on various measures of uncontrolled eating relate to increased eating in different experimental settings (reviewed in Bongers & Jansen, 2016; Bryant et al., 2008; Espel-Huynh et al., 2018; Goldschmidt, 2017). People with higher uncontrolled eating scores have also higher self-reported fat intake (reviewed in Stevenson, 2017). The associations between uncontrolled eating and food intake are more consistent when uncontrolled eating is measured by Disinhibition scale (Bryant *et al.*, 2008) and loss of control over eating (Goldschmidt, 2017), whereas the associations are weaker when uncontrolled eating is measured by emotional eating (Bongers & Jansen, 2016) or

hedonic hunger (Espel-Huynh *et al.*, 2018). There may be two explanations for this. First, disinhibited eating and loss of control over eating might be "purer" measures of uncontrolled eating and therefore relate to food intake more strongly than the other traits that assess specific reasons for overeating, such as hedonic hunger or negative emotions. Second, experiments involving disinhibition and loss of control over eating typically attempt to relate the questionnaire to simple food consumption, whereas studies using emotional eating and power of food questionnaires attempt to induce or refer to a specific emotional state in participants.

Since uncontrolled eating is associated with food overconsumption, the trait should also relate to obesity and BMI. This is indeed the case – uncontrolled eating modelled as common variance across many questionnaires tends to have positive cross-sectional associations with BMI (Price *et al.*, 2015; Vainik, Neseliler, *et al.*, 2015). Moreover, almost all of the individual food behaviour questionnaires that contribute to uncontrolled eating also correlate with BMI (French *et al.*, 2012; Price *et al.*, 2015; Vainik, Neseliler, *et al.*, 2015; Goldschmidt, 2017; Mason *et al.*, 2017).

Uncontrolled eating has a genetic background

Behavioural genetic models show that uncontrolled eating is a heritable trait. Typically, behavioural genetic analysis uses twin samples and ACE models to parcellate variance of a phenotype into three components: 'A', heritable component, variance explained by genetic signal; 'C, shared environmental component, variance shaped by home and school environment that twin pairs share; and 'E' – unique environmental component, variance assumed to be explained by environmental factors that are not shared by twins, i.e. unique experiences with the world. Across twin samples from North America, Europe, and Korea, the various uncontrolled eating constructs within TFEQ and DEBQ have the following heritability: disinhibition: 40-45%, uncontrolled eating: 45-69%, emotional eating: 9-45%, external eating: 25%, and susceptibility

for hunger: 8-23% (Steinle *et al.*, 2002; Neale *et al.*, 2003; de Castro & Lilenfeld, 2005; Tholin *et al.*, 2005; Keskitalo *et al.*, 2008). Most of the estimates are close to the meta-analytic heritability of any personality trait (47%) (Vukasović & Bratko, 2015) or any behavioural trait (49%) (Polderman *et al.*, 2015). As a general trend, there is no shared environmental component ('C') for uncontrolled eating. Rather, the non-genetic variance of uncontrolled eating is explained by environmental factors that are not shared by twins ('E').

There is also a sizable genetic component in the correlations between different questionnaires measuring aspects of uncontrolled eating, as well as uncontrolled eating's correlations with BMI and food consumption. Namely, a shared genetic factor explains most of the covariance between subdimensions related to uncontrolled eating within the TFEQ (Neale *et al.*, 2003). In a similar fashion, the heritable variances ('A') of emotional eating and uncontrolled eating overlap strongly, with a genetic correlation of r_g=0.75 (as measured by TFEQ-R18, Keskitalo *et al.*, 2008). Further, heritable variance explains 58% of the phenotypic correlation between the two traits (r=0.56). Keskitalo et al. (Keskitalo et al., 2008) also found similar patterns for the associations between uncontrolled eating and BMI (r_g=0.29-0.51, 'A' explains 81%), and the correlations between uncontrolled eating and self-reported liking and consumption of sweet/salty-and-fatty foods (r_g = 0.16-0.31, 'A' explains 52-100%). The genetic overlap between uncontrolled eating and BMI calls into question whether uncontrolled eating is any different from BMI in terms of genetic signature.

A recent GWAS on food addiction has outlined that there is some meaningful genetic signal to the portion of uncontrolled eating that is independent of BMI (Cornelis *et al.*, 2016). Across 9000+ participants, the study did not find evidence for links between addiction-like eating (measured by the Yale Food Addiction Scale) and genes linked to addiction-like behaviour

through theory or previous genome-wide association studies. This argues against the view that there is shared genetic potential between other addictions and "food addiction/ uncontrolled eating" (Carlier *et al.*, 2015). Second, the study did not replicate the association between uncontrolled eating and dopamine polygenic score, a composite score of several dopamine-related alleles hypothesised to relate to addiction risk (Nikolova *et al.*, 2011; Davis *et al.*, 2013). Still, Cornelis et al (2016) found two single nucleotide polymorphisms associating with Yale Food Addiction Scale while controlling for BMI; however their role in eating behavior is not clear. This could mean that uncontrolled eating as a phenotype encompasses more than just over-eating or addictive behaviour, or that drug addiction and over-eating do not have the same genetic or neural causes. However, the results of genome-wide associations studies on behavioral traits are generally hard to explain, as the pathway from molecular mechanism to behaviour is indirect (Turkheimer & Harden, 2014).

The genetic overlap between uncontrolled eating and BMI could also suggest that the genetic risk for obesity acts via (perceived) self-control of food intake. There is indeed suggestive evidence for this: the top single nucleoid polymorphisms associated with BMI also correlate with uncontrolled eating (Cornelis *et al.*, 2014), and uncontrolled eating mediates the effect of the BMI polygenic score on BMI (Konttinen *et al.*, 2015). However, the causality in the latter model is not necessarily unidirectional. Based on longitudinal findings summarised below, uncontrolled eating can lead to higher BMI, but higher BMI can also lead to higher uncontrolled eating, that is, weight gain can change an individual's answers on uncontrolled eating questionnaires. A possible interpretation is pleiotropy – uncontrolled eating and BMI share a set of genes, which cause both variation in BMI and variation in uncontrolled eating. Once larger genome-wide studies of uncontrolled eating are available, mendelian randomisation studies become possible to

enable disentangling the genetic causality between uncontrolled eating and BMI (Lawlor *et al.*, 2008).

Despite the seemingly strong role of genetics, uncontrolled eating also appears to be susceptible to environmental influence. Non-shared environment ('E') has a role in explaining the phenotypic correlation between constructs of uncontrolled eating, the correlation between uncontrolled eating with BMI, and the associations of uncontrolled eating with liking for salty-and-fatty foods (Keskitalo *et al.*, 2008). This means, that if one twin has higher BMI or food intake due to some environmental factor, that twin is also likely to have higher uncontrolled eating. Such environment-mediated associations provide support for causal associations between traits (Turkheimer & Harden, 2014). Further, external environmental factors influence the heritability of traits: for instance, the heritability of BMI is lower in people that exercise (McCaffery *et al.*, 2009; Horn *et al.*, 2015; Graff *et al.*, 2017). Therefore, heritability only stands for genetic potential for a trait to manifest, and this potential can be altered with interventions.

In summary, the genetics-based biological background of uncontrolled eating is elusive. The hypothesised link to addiction is not well borne out by genetic studies in large samples. While family environment plays no role in uncontrolled eating, unique environmental experiences partly account for the variance in uncontrolled eating and its associations with other traits.

Longitudinal associations between uncontrolled eating and body weight

The cross-sectional covariance between uncontrolled eating and BMI (Figure 2, line1) and the possibility of genetic pleiotropy lead to the question: what are the causal mechanisms? If uncontrolled eating robustly covaries with food intake and BMI, is uncontrolled eating a mere psychological reflection of one's waistline or food intake? Does uncontrolled eating lead to

weight gain over time, or does obesity lead to increased uncontrolled eating over time? These associations can be disentangled in longitudinal designs (Figure 2, lines 4-5).

As a general feature, uncontrolled eating tends to be stable over time with a test-retest reliability r~0.8 (Vainik *et al.*, 2013) (Figure 2A, line 2). Similar stability is known for the main correlate of uncontrolled eating: BMI (Serdula *et al.*, 1993) (Figure 2A, line 3). Such evidence suggests that uncontrolled eating and BMI can be considered trait measures, as similar test-retest reliability is known for other personality traits (McCrae *et al.*, 2011).

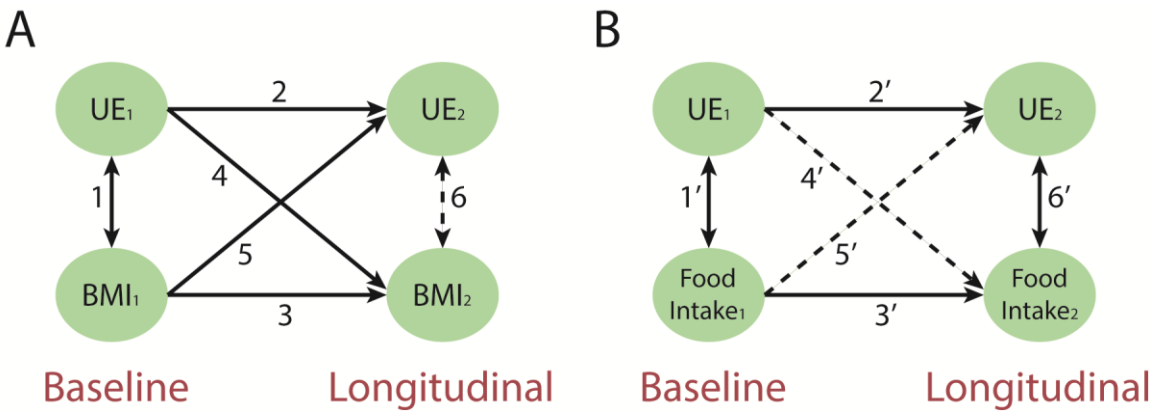

Figure 2. (A) Putative relationships between body mass index and uncontrolled eating and in longitudinal settings. (B) Independent relationships between uncontrolled eating and food intake identified from the literature. Solid lines depict that the relationship has been more consistently reported in the literature, as opposed to dashed lines. *Abbreviations:* BMI, body mass index; UE, uncontrolled eating.

Individuals who exhibit high scores in uncontrolled eating at baseline generally tend to gain weight over time (French *et al.*, 2012) (Figure 2A, line 4). Longitudinal studies have observed this trend by using questionnaires reflecting (i) emotional eating (Van Strien *et al.*, 2013;

Boggiano *et al.*, 2015), (ii) binge eating (Quick *et al.*, 2013; Micali *et al.*, 2014), (iii) disinhibited eating (Hays *et al.*, 2002; Chaput *et al.*, 2010, 2011), (iv) feelings of hunger (Hays *et al.*, 2006) and (v) food reward sensitivity (Lipsky *et al.*, 2016). Additionally, scores in uncontrolled eating seem to predict weight outcomes in clinical populations undergoing weight loss treatments. A national US registry on individuals attempting to lose weight concluded that low scores in disinhibited eating tended to be associated with greater success (Wing & Phelan, 2005). In dieters, high levels of emotional eating and overeating decreases the chances of weight loss after 4 years (Keller & Hartmann, 2016). Along similar lines, studies on obese populations have also shown that patients exhibiting low scores in impulsive eating tend to show greater longitudinal reductions in BMI after both surgical and non-surgical diet-related or lifestyle-related clinical interventions (Dalle Grave *et al.*, 2009; Legenbauer *et al.*, 2010; Teixeira *et al.*, 2010; Meany *et al.*, 2014; Ivezaj *et al.*, 2017). Uncontrolled eating questionnaires might thus prove useful to identify which patients on weight-loss treatments require additional help to achieve better outcomes.

The relationship between uncontrolled eating and body weight might not be merely unidirectional (Figure 2A, line 5). Studies suggest a more complex picture, with changes in body weight also affecting eating behaviors (Boggiano *et al.*, 2015). For instance, 6 and 12 months after bariatric surgery, scores in different uncontrolled eating scales tend to decrease, while scores on cognitive control over eating tend to increase (Dalle Grave *et al.*, 2009; Koball *et al.*, 2016; Sevinçer *et al.*, 2016; Holsen *et al.*, 2017; Ivezaj *et al.*, 2017). Scores on uncontrolled eating also show reductions after life-style weight loss interventions (Martin *et al.*, 2006).

At the same time, the rates of change in uncontrolled eating and BMI do not always correlate (Figure 2, line 6). Clinical studies suggest that certain interventions work better for weight loss, while others seem more suitable for reducing uncontrolled eating. Based on a German weight loss registry, formerly obese people still have high uncontrolled eating compared to the general population (Feller *et al.*, 2015). On the other hand, many binge-eating treatments reduce binge eating behavior but not weight (Wilson *et al.*, 2007). A recent review on various therapies' effects on weight loss and on decrease in uncontrolled eating (emotional eating or disinhibition) concluded that control-based therapies, such as cognitive-behavioural therapy, seem to succeed in reducing weight but tend not to reduce uncontrolled eating, whereas therapies more focused on accepting emotions (acceptance and commitment therapy, mindfulness) are able to reduce uncontrolled eating (Frayn & Knäuper, 2017). It seems that uncontrolled eating is then an important variable to be considered along with BMI when planning therapies, and focusing on only one of the two does not guarantee reductions of the other. Uncontrolled eating and BMI are intertwined, but still independent factors.

Longitudinal associations between food intake and uncontrolled eating

Studies disentangling the longitudinal associations between uncontrolled eating and food intake are less common. Weight loss is the main outcome of interest in most longitudinal studies, and conducting repeated food intake measurement is laborious. Still, a recent study showed that most laboratory food intake parameters have good test-retest reliability over one week ($r>0.8$; (Laessle & Geiermann, 2012)) (Figure 2B line 3'). Further, the same study found that TFEQ constructs at time 1 do not predict food intake at time 2, suggesting that natural variation in food intake is not explained by uncontrolled eating over baseline food intake (Figure 2B line 4'). Similarly, variation in food intake at time 1, i.e., by manipulating hunger levels, does not influence

uncontrolled eating scores (Witt *et al.*, 2013), with the exception of external eating (Evers *et al.*, 2011) (Figure 2B line 5').

An interesting observation is that food intake varies more from day-to-day than uncontrolled eating. An experience sampling study asked adolescents to rate uncontrolled eating and food intake daily for 20 days (Bejarano & Cushing, 2018). They found that the variability in uncontrolled eating was 2/3 between person (i.e., trait) and 1/3 within person (i.e., state) (Figure 2B line 2'), and the proportions were roughly reversed for food intake (2/3 within person and 1/3 between person). They further found that trait uncontrolled eating associated with general (trait-like) consumption of fatty foods (Figure 2B line 1'), whereas daily variation in UE associated with daily variation in starchy food consumption (Figure 2B line 6'). Taken together, current preliminary evidence seems to suggest that uncontrolled eating and food intake are more likely to be interchangeable processes – uncontrolled eating could be a broad summary of daily food intake fluctuations. However, because uncontrolled eating and food intake fluctuate at different rates, and food intake is hard to measure, proper demonstration of such covariation has not been attained.

The three psychological constructs of uncontrolled eating: reward sensitivity, cognitive control , and negative affect

Uncontrolled eating has been related to several psychological constructs, which provide clues about the psychological processes co-occuring with uncontrolled eating. The three main ones suggested by the literature are reward sensitivity, cognitive control, and negative affect. These three constructs form the core framework for impulsivity (Whiteside & Lynam, 2001; Sharma *et al.*, 2014), are linked to real-life maladaptive behaviours (Whiteside *et al.*, 2005; Sharma *et al.*, 2013), and to obesity (Vainik *et al.*, 2013). Since people have made a distinction between

domain-general and domain-specific versions of impulsivity (Tsukayama *et al.*, 2012), we also consider this distinction here.

Foremost, uncontrolled eating has been linked to *reward sensitivity* – a heightened likelihood of approach behaviour and experience of pleasant affect (Lucas *et al.*, 2000). There is some evidence that uncontrolled eating correlates positively with domain-general reward sensitivity in mostly normal-weight young adult samples (Finlayson *et al.*, 2012; Neseliler *et al.*, 2018). At the same time, reverse effects are seen in exclusively overweight and obese samples. Namely, uncontrolled eating relates negatively to Extraversion (Elfhag & Morey, 2008; Provencher *et al.*, 2008), a trait driven by reward sensitivity (Lucas *et al.*, 2000). Taken together, general reward sensitivity's association with uncontrolled eating may be moderated by weight status – overeating may be enjoyable if it does not lead to struggle with excessive weight. Similarly, a curvilinear association has also been suggested between reward sensitivity and BMI (Davis & Fox, 2008), and dopaminergic tone and BMI (Horstmann *et al.*, 2015).

The second underlying psychological construct of uncontrolled eating is lower *cognitive control* – an individual's difficulties for flexible adaptive goal-directed action. In the case of Uncontrolled Eating, low cognitive control could mean an inability to moderate the behavioural response to heightened reward sensitivity. Several studies have associated uncontrolled eating with laboratory-based tests of cognitive control (Yeomans *et al.*, 2008; Appelhans *et al.*, 2011; Leitch *et al.*, 2013; Calvo *et al.*, 2014; Yeomans & Brace, 2015; Bartholdy *et al.*, 2016; Brace & Yeomans, 2016; Lavagnino *et al.*, 2016; Eneva *et al.*, 2017; Martin *et al.*, 2018). Examples of tests used include delay discounting, the Go-Nogo task, but also memory tests. The results are mostly inconsistent - the effects tend to be driven only by certain cognitive tests, and the same cognitive tests do not replicate from study to study. In personality literature, uncontrolled eating

correlates positively with Impulsiveness as measured by Barrat Impulsiveness Scale (e.g., Meule & Blechert, 2017), and negatively with Conscientiousness (Elfhag & Morey, 2008; Provencher *et al.*, 2008), a personality trait associated with self-management and impulse control.

A third related construct is *negative affect* – increased likelihood of experiencing negative emotions and sensitivity to negative signals in the environment. Several studies have shown that measures of uncontrolled eating positively correlate with Neuroticism (Elfhag & Morey, 2008; Provencher *et al.*, 2008), a measure of sensitivity to negative emotions. The same measures also correlate positively with depressive symptoms (Konttinen *et al.*, 2014; Paans *et al.*, 2018).

The associations between those domain-general traits and uncontrolled eating are very much expected, as these three traits seem to have eating-specific matches (Table 1). Granted, the empirical demonstration of these eating-specific subdomains of uncontrolled eating still has to be conducted. However, such a relationship is suggested by the definitions of constructs, names of questionnaires (Table 1), and item content.

Food-specific reward sensitivity – heightened sensitivity and responsiveness to food cues, is often the focus of questionnaires intending to measure constructs such as reward-related eating, hedonic eating, food craving, and external eating. A review of the evidence provides support for such "cue-reactive eating", as people with high uncontrolled eating eat more when food is on display (Bongers & Jansen, 2016). Uncontrolled eating also covaries positively with behavioural measures of wanting and liking of high-calorie foods (French *et al.*, 2014). At the same time, uncontrolled eating has weaker association with relative reinforcing value or willingness to work for food (French *et al.*, 2014; Brace & Yeomans, 2016), suggesting that uncontrolled eating may be distinct from this behavioural measure of food reward sensitivity. Similarly to uncontrolled

eating, behavioural food reward sensitivity diminishes in response to weight management interventions (Oustric *et al.*, 2018).

Poor food-related cognitive control is in the explicit focus of several constructs, such as binge eating, disinhibition, uncontrolled eating, and lack of control over eating (Table 1). In our opinion, lack of food related cognitive-control is the essence of most questionnaires discussed here, as questions on eating control are present in most questionnaires mentioned in Table 1. A conceptually related eating-related cognitive control construct is dietary restraint, an attempt to control one's food intake. Broadly, the questionnaires measuring restraint and uncontrolled eating tend not to correlate (Price *et al.*, 2015; Mason *et al.*, 2016). At the same time, different combinations of high/low uncontrolled eating and high/low restraint may produce differential behaviours (Johnson *et al.*, 2011).

Eating-related negative affect is most clearly captured in the construct of emotional eating, which attempts to measure eating as a coping mechanism when negative emotions occur (Greeno & Wing, 1994; Gibson, 2012). In their critical overview of the emotional eating construct, Bongers and Jansen (2016) suggest that emotional eating could reflect a general worry about eating and lack of perceived control. Similarly, worry about eating is present in the definition of binge eating (Table 1), and also in single items of questionnaires supposedly measuring reward sensitivity , e.g., "It's scary to think of the power that food has over me" within Power of Food Scale (Cappelleri *et al.*, 2009). Finally, uncontrolled eating correlates with having poor eating competence and eating attitudes (Lohse *et al.*, 2007; Panov, 2014).

Uncontrolled eating as a link between personality traits and eating behaviours

Reward sensitivity, cognitive control and negative affect are linked to BMI and food intake (Jokela *et al.*, 2013; Vainik *et al.*, 2013; Lunn *et al.*, 2014; Gerlach *et al.*, 2015; Emery & Levine, 2017; Stevenson, 2017; Yang *et al.*, 2018). However, the effect sizes of these associations are smaller than those between uncontrolled eating and BMI or food intake. Since uncontrolled eating questionnaires seem to host eating-specific versions of these broad traits, there have been repeated demonstrations of a hierarchical model, where uncontrolled eating mediates the effects of reward sensitivity, cognitive control and negative affect on (i) objective food intake (Kakoschke *et al.*, 2015), (ii) subjective food intake (Keller & Siegrist, 2015), and (iii) cross-sectional BMI (Meule & Blechert, 2017; Neseliler *et al.*, 2018).

Considered together, cross-sectional evidence shows that higher scores in uncontrolled eating associate with higher food intake and higher BMI. Uncontrolled eating acts as an intermediate phenotype explaining the link between broad psychological constructs and food intake/BMI (Figure 3).

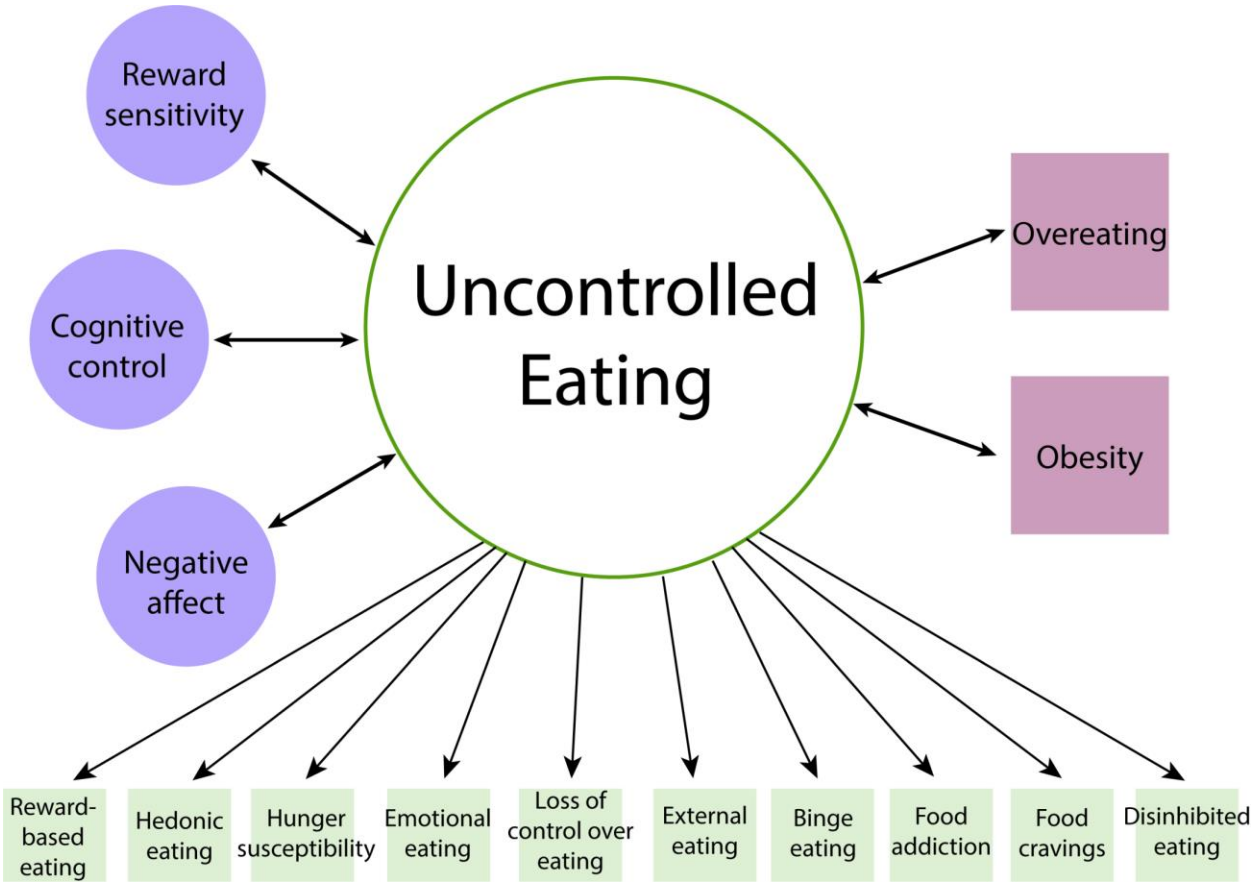

Figure 3. Uncontrolled eating as a mediator between psychological constructs (violet) and eating phenotypes (red). Bi-directional arrows allow reciprocal influences between uncontrolled eating and other variables. At the same time, uncontrolled eating is assumed to be a core latent construct influencing variance in all its indicators (eating related traits, in light green). The eating related traits could, in principle, be grouped by the three domain-specific psychological constructs mentioned in text. In the interest of brevity, this is not shown.

Neural correlates of uncontrolled eating

Uncontrolled eating is a psychometric construct with biological plausibility. In the following, we delineate a set of brain circuits and regions that are putatively relevant for uncontrolled eating. With this, we seek to provide an interdisciplinary theoretical background that will pave the road

for designing future studies and hypotheses on the relationships between uncontrolled eating, neural computations, and their associated brain circuits. These studies may have the potential to inform future clinical treatments aimed at treating extreme cases of uncontrolled eating.

We hypothesize that the neurobiological circuits governing uncontrolled eating can be broken down according to three components: reward sensitivity, cognitive control and negative emotionality.

Reward sensitivity

Reward has been traditionally linked to the function of the midbrain dopamine circuit (García-García *et al.*, 2017; Volkow *et al.*, 2017). Dopamine signals arise from the ventral tegmental area (VTA) and adjacent substantia nigra pars compacta (SNc) (Figure 4). The VTA/SNc receives energy-balance information via orexin-containing projections from the lateral hypothalamus. This connection is thought to underpin the interrelations between appetite and reward (Sweeney & Yang, 2017). The VTA/SNc, in turn, innervates the striatum, ventromedial prefrontal cortex (vmPFC), orbitofrontal cortex and amygdala and hippocampus. These areas have all been implicated in the rewarding aspects of eating, and more generally in assigning value to stimuli (Bartra *et al.*, 2013).

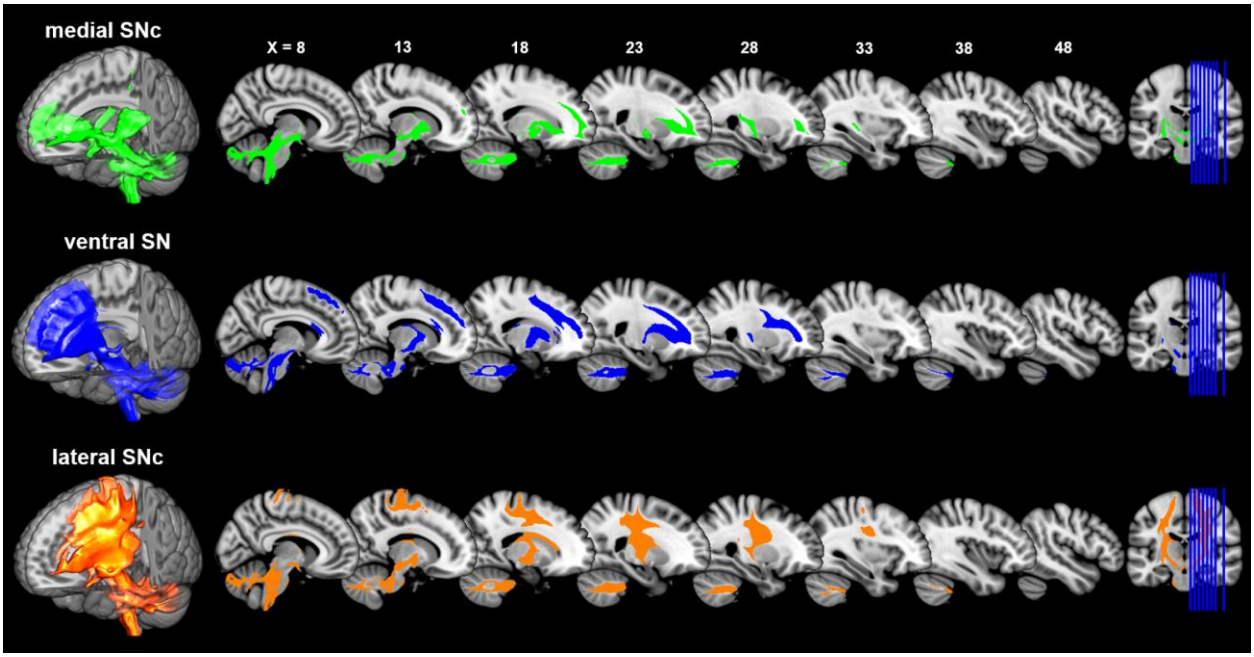

Figure 4. Whole brain white matter projections of midbrain dopamine cells. Three partitions of the substantia nigra give rise to limbic (green), associative (blue) and motor (orange) dopamine projections. Figure originally published in Zhang et al. (Zhang *et al.*, 2017) under Creative Commons Attribution License. (SN: substantia nigra; SNc: substantia nigra pars compacta).

By recruiting the striatum and the reward system, positive reinforcers (e.g. appetising foods) can potentially lead to maladaptive behaviors (e.g. uncontrolled eating) in some individuals (Koob & Le Moal, 1997; Saunders & Robinson, 2013). Greater activation of this system may reflect greater incentive salience of food cues, favouring loss of control.

In this vein, human functional magnetic resonance imaging (fMRI) studies have shown that striatal activity in response to food cues predicts chocolate intake (Lawrence *et al.*, 2012) and weight gain after a follow-up period varying between 6 months and 3 years (Stice *et al.*, 2008; Demos *et al.*, 2012; Geha *et al.*, 2013) but see (Stice & Yokum, 2018). Functional connectivity between the dorsal striatum and somatosensory cortex has also been associated with food

cravings *in situ* and it seems to predict prospective changes in BMI (Contreras-Rodríguez *et al.*, 2017).

Animal research suggests that exposure to high fat diets induces neuroplastic changes in mesolimbic dopamine synapses (Liu *et al.*, 2016) and in striatal dopamine D2 receptors (Adams *et al.*, 2015). These neuroplastic changes, in turn, might increase food reward sensitivity and explain the persistence of uncontrolled eating (Murray *et al.*, 2014).

Many investigators have attempted to link dopamine signalling to BMI (Murray *et al.*, 2014). Decreases in dopamine D2 receptor density are often reported in patients with drug addiction (Volkow *et al.*, 2013), which has been taken to imply lower dopamine signalling. With regards to uncontrolled eating, there is some controversy on whether overeating and obesity relate to *decreased* or *increased* dopamine D2 receptor density.

In an influential early positron emission tomography study, Wang et al. observed that participants with morbid obesity showed decreased D2/3 receptor binding in the striatum (Wang *et al.*, 2001). The authors argued that lower dopamine D2/3 receptor density in obesity could cause hyperphagia as a way to compensate for blunted responses in the reward circuitry (Wang *et al.*, 2001) – the so-called reward deficiency hypothesis. Others, however, have rejected the hypothesis that obesity *per se* is related to dopamine D2/3 reductions. Several studies have falsified the original Wang et al. finding: some authors suggest that D2/3 binding reductions are specific to morbid obesity (BMI > 40kg/m^2) and should not be extrapolated to milder forms of obesity. In this vein, studies recruiting obese participants with a lower BMI average (i.e., 30 < BMI < 40 kg/m^2) have observed (i) equivalent D2/3 receptor density between obese and lean participants (Eisenstein *et al.*, 2015) and also (ii) higher D2/3 striatal receptor density in obese

participants compared to lean subjects (Eisenstein *et al.*, 2013). These findings have been replicated with a different positron emission tomography D2 tracer (Dang *et al.*, 2016).

However, a meta-analysis that used data from five studies did not find consistent differences in D2 receptor measures between lean and obese participants (Karlsson *et al.*, 2015). An alternative explanation is that the relationship between the severity of obesity and dopamine D2 receptors adopts an inverse-U shape (Horstmann *et al.*, 2015). Finally, previously suggested links between BMI and the Taq1A polymorphism, which confers variability of dopamine D2 receptor density, have not been borne out by a recent meta-analysis (Benton & Young, 2016).

Thus, while individual differences in reward function may affect eating control, dopamine signalling does not appear to account for much of the variance in BMI in normal to obese weight populations.

Neurobiological mechanisms of cognitive control

A recent meta-analysis of fMRI studies shows that when participants attempt to reduce their craving response to food cues, they consistently engage the anterior insula, inferior frontal gyrus/ventrolateral prefrontal cortex, supplementary motor area, dorsolateral prefrontal cortex (DLPFC), mid-cingulate cortex and temporal-parietal junction (Han *et al.*, 2018). Moreover, this response is reduced in people with higher BMI. Several studies show that activation in these regions predicts immediate food consumption: for example, participants showing higher activity in the DLPFC tended to eat a lower amount of chocolate immediately after the fMRI session (Frankort *et al.*, 2015). DLPFC activation also predicts successful weight loss in people who go on a diet (Jensen & Kirwan, 2015) or undergo bariatric surgery (Goldman *et al.*, 2013). These results point at the importance of prefrontal circuits in the regulation of body weight and hedonic eating.

Deficits in neural activity in cognitive control networks are especially prominent in individuals with binge eating disorder or bulimia nervosa. Participants with binge eating disorder exhibit lower activation of the DLPFC, inferior frontal gyrus and vmPFC when performing an fMRI inhibitory control task (Balodis *et al.*, 2015; Hege *et al.*, 2015; Lavagnino *et al.*, 2016). Patients with bulimia nervosa, similarly, show hypoconnectivity of the executive control network during an attention fMRI task (Seitz *et al.*, 2016). The identification of the brain basis of cognitive control over food intake might inform clinical strategies aimed at ameliorating uncontrolled eating in clinical populations, for instance by neuromodulation (Val-Laillet *et al.*, 2015).

Negative emotionality (stress) circuits in the brain

Neurobiological studies on negative emotionality and uncontrolled eating have mainly revolved around stress responses. Stress responses are included within the broader term of "negative emotionality". Since stress responses are easier to operationalize, they facilitate the comparisons between animal and human neurobiological studies. Stress and energy homeostasis are intimately related. Under acute stress, the hypothalamic-pituitary-adrenal (HPA) axis increases the release of cortisol, which in turn acts on metabolic pathways to maintain glucose homeostasis (Herman *et al.*, 2016).

The amygdala also has an important role in stress responses. Higher scores in UE (i.e., disinhibited eating from the TFEQ) were associated with lower functional connectivity between the amygdala and the vmPFC (Dietrich *et al.*, 2016), a key structure in the appetitive response to food cues.

The amygdala is composed of a set of nuclei that integrate homeostatic, cognitive and visceral information. Its central nucleus sends projections to dopamine midbrain structures (i.e., both the VTA and the SNc) (Watabe-Uchida *et al.*, 2012), playing a potential role in the computation of

reward. Another nucleus, the basolateral complex, sends projections to the nucleus accumbens, putatively influencing food reward processing (Janak & Tye, 2015). Outside the reward system, the basolateral amygdala presents bidirectional projections with sensory association areas (Janak & Tye, 2015). These projections are key to understand how the brain detects saliency, or emotionally relevant stimuli.

The role that the amygdala plays in stress might be partly explained by its prominent involvement in salience detection. The amygdala, in concert with other interconnected areas like the orbitofrontal cortex or anterior cingulate cortex, seems to facilitate the engagement of perception and attention towards emotionally relevant stimuli (Pessoa & Adolphs, 2010; Pourtois *et al.*, 2013). When the organism detects threat or other significant stimuli in the environment, the projections between the amygdala and the sensory cortices enhance information processing (Vuilleumier, 2005). This way, the organism can mobilise a set of resources that allow emotionally relevant information to be processed as fast as possible. In this vein, the amygdala is widely engaged during the processing of negatively valenced stimuli (e.g., fearful faces or aversive images) compared to neutral stimuli(García-García *et al.*, 2016). It is also recruited in response to both natural rewards and drug rewards (Tang *et al.*, 2012). The recruitment of salience/stress-related circuits when facing emotionally relevant stimuli is evolutionarily adaptive. An excessive overengagement of these circuits, however, might turn dysfunctional and might be a trans-diagnostic feature in addictive-like behaviors. In line with this possibility, both participants with obesity and patients with addictions seem to exhibit augmented activity in the amygdala and ventral striatum in response to reward (García-García *et al.*, 2014).

Finally, the hippocampus is another medial temporal region that participates in stress responses and food intake (Hargrave *et al.*, 2016). The hippocampus inhibits the hypothalamus and HPA

axis, adjusting cortisol levels (Stevenson & Francis, 2017). The hippocampus is additionally involved in matching food cue-reactivity to contextual information (e.g. energy balance signals), possibly via the activation of mechanisms associated with memory and habit learning (Stevenson & Francis, 2017; Higgs & Spetter, 2018).

Brain regions associated with questionnaire measures of uncontrolled eating: preliminary meta-analysis

A growing number of studies are addressing the neural correlates of self-reported uncontrolled eating. The majority, however, are hypothesis-based, and as such, they restrict their analyses to certain regions of interest (Wood *et al.*, 2016), which hampers the comparability between studies. Here we seek to provide an objective whole-brain overview of areas that exhibit consistent fMRI activity in correlation with self-reported uncontrolled eating. We conducted a preliminary meta-analysis based on coordinates and effect sizes reported using Anisotropic Effect-Size Seed-Based d Mapping (SDM) software (Radua *et al.*, 2012, 2014) and searched for peer-reviewed fMRI studies on uncontrolled eating using the following keywords: (i) neuroimaging-related: fMRI or MRI or cortex; (ii) uncontrolled eating-related: uncontrolled eating or emotional eating or hedonic eating or binge eating or food craving. We applied standard exclusion criteria, namely, (i) studies that do not include whole-brain analyses, (ii) studies on children or adolescent populations, (iii) results that are not reported in standard space, and (iv) papers on psychiatric populations. Our method of selection resulted in the inclusion of 9 fMRI studies, representing 339 participants with an age range between 18 and 60 years (Table 2). Seven studies measured a response to food cues, while two measured BOLD (blood-oxygen-level dependent) activity at rest. We set the statistical threshold at $p<0.005$ uncorrected with an additional cluster size threshold > 100 mm^3, following the standard recommendations for SDM. The model included as covariates mean age, mean BMI, percentage of females in the sample,

and specific uncontrolled eating scale used. We found that uncontrolled eating was associated with consistent enhancements of fMRI activity in the bilateral cerebellum, extending to the temporal occipital fusiform (MNI coordinates: -20,-60,-22 and 24,-56,-20) as well as with a lower fMRI recruitment of the DLPFC (bilateral middle frontal gyrus extending to the frontal pole: 38,34,22 and 34,24,40, as well as the right superior frontal gyrus: 10,26,54) (Figure 5).

The cerebellum is a functionally heterogeneous region with a role in motor performance, cognitive functions and emotion responses. The cluster obtained in the meta-analysis corresponds to a subdivision that presents dense interactions with sensorimotor regions (Buckner, 2013; Koziol *et al.*, 2014). This might be suggestive of a tendency to increased recruitment of sensory processes towards food stimuli in participants with high scores on uncontrolled eating. The results also showed that high uncontrolled eating was associated with lower recruitment of the DLPFC (middle and superior frontal gyrus). This finding is consistent with the hypothesis that uncontrolled eating is associated with a lower recruitment of self-control resources. Despite the low number of studies, this preliminary result delineates the cerebellum and the lateral prefrontal cortex as regions of interest for future studies on uncontrolled eating.

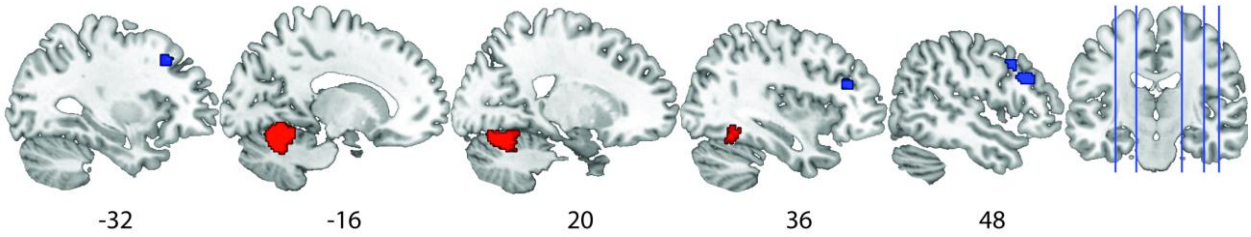

Figure 5. Associations between BOLD activity and different measures of uncontrolled eating. We conducted an SDM meta-analysis on human fMRI studies in adult participants that correlated whole-brain fMRI activity with measures of uncontrolled eating. We found that uncontrolled eating was associated with a consistent recruitment of the bilateral cerebellum (red) as well as with lower fMRI activity in the DLPFC (blue).

Table 2. Description of the papers included in our meta-analysis. Human fMRI studies showing whole-brain correlations between different measurements of uncontrolled eating and BOLD activity.

Study	***n*, mean age (*SD*), age range, % females**	**Mean BMI (SD), BMI range**	**Uncontrolled eating measurement**	**fMRI measurement**	**Statistical threshold**
(Burger *et al.*, 2016)	44, 20.8 (1.3), age range NR, 100%	23.8 (2.9), BMI range NR	Hedonic hunger (PFS)	Food reward task: response to milkshape	Uncorrected $p<0.001$, additional cluster-level correction $p<0.05$
(Chen *et al.*, 2017)	65, 21.52 (1.55), 18-27 years, 100%	20.45 (1.99) 17.16-24.56	Food Cravings Questionnaire-Trait (FCQ-T-r)	Resting-state (regional homogeneity)	Uncorrected $p<0.001$; $k>20$
(Filbey *et al.*, 2012)	26, 32.88 (11.04), age range NR, 64%	32.72 (5.98), 25.10-51.50	Binge Eating Scale	Food reward: high calorie > water taste	Cluster-level correction $p<0.05$
(Kahathuduwa *et al.*, 2018)	32, mean age (SD) NR, 18-60 years, 57%	Mean (SD) NR, 30-39.9	Food Cravings Questionnaire-Trait (FCQ-T)	Food reward: food > objects	Cluster-level correction $p<0.05$
(Passamonti et al., 2009)	21, 25.3 (SD NR), 19-38 years, 48%	24 (4.6), BMI range NR	External eating (DEBQ)	Food reward task: appetising > bland food pictures	Uncorrected $p<0.001$
(Ulrich *et al.*, 2016)	20, 24.9 (3.2), age range NR, 0%	23.9 (2.9), 20.6-32.9	Food Cravings Questionnaire-Trait (FCQ-T)	Food reward: high calorie > low calorie food	Uncorrected $p<0.001$, additional cluster-level FWE

				pictures	correction $p<0.05$
(Van Bloemendaal et al., 2015) (Study 1)	16, 57.8 (1.9), age range NR, 50%	23.2 (0.4), BMI range NR	Emotional Eating (DEBQ)	Food reward task: high calorie food > non-food stimuli	Uncorrected $p<0.001$, additional cluster-level FWE correction $p<0.05$
(Van Bloemendaal et al., 2015) (Study 2)	16, 58.0 (2.1), age range NR, 50%	32.6 (0.7), BMI range NR	Emotional Eating (DEBQ)	Food reward task: high calorie food > non-food pictures and food > non-food pictures	Uncorrected $p<0.001$, additional cluster-level FWE correction $p<0.05$
(Zhao et al., 2017)	99, 35.36 (1.15), 20-57, 37%	27.18 (0.58), 18.9-43.4	Disinhibited Eating and Hunger (TFEQ)	Resting-state (amplitude of low frequency fluctuations)	Uncorrected $p<0.001$; $k>30$

Abbreviations: BMI, body mass index; DEBQ, Dutch Eating Behavior Questionnaire; FCQ-T, Food Cravings Questionnaire-Trait; FCQ-T-r, Food Cravings Questionnaire-Trait reduced; FWE, family-wise error; k, cluster size (in voxels); NR, not reported; PFS, power of food scale; SD, standard deviation; TFEQ, Three-Factor Eating Questionnaire.

Modelling uncontrolled eating: From single concept to mutual processes

So far, we have used similarities based on definitions and covariation as justification for aggregating measures from different questionnaires and frameworks into a single uncontrolled eating factor. Such single factor frameworks have been popular in other common-trait studies, such as common intelligence (*g*) (A. R. Jensen, 1998; Spearman, 1904), common psychiatric pathology (*p*) (Caspi *et al.*, 2014; Laceulle *et al.*, 2015), or general factor of personality (Musek,

2007; Rushton & Irwing, 2008). However, covariance between questionnaires alone does not provide sufficient proof for the causal role of a single uncontrolled eating factor, as articulated in the context of psychopathology (van Bork *et al.*, 2017) and intelligence (Van Der Maas *et al.*, 2006). Bork et al and van der Maas et al have highlighted how different choices in modelling the positive covariance are near-equivalent in terms of fit statistics, but imply different causal mechanisms. Research on intelligence has proposed at least two alternative ways that a positive covariance might be explained. Such approaches should also be considered for detailed modelling of uncontrolled eating.

One is sampling theory where multiple processes depend on a shared set of independent sub-processes. Bork et al highlighted how covariation between measures of depression and anxiety disorders could occur because they have common symptoms, such as insomnia, fatigue, inattention and psychomotor agitation (American Psychiatric Association, 2013; van Bork *et al.*, 2017). In the case of uncontrolled eating, different eating-related traits could depend on the same set of subprocesses, such as increased negative affect and lack of cognitive control, while also having unique subprocesses, such as positive or negative association with reward sensitivity, possibly moderated by obesity status.

The other explanation is mutualism, where separate cognitive processes mutually co-operate and develop. In case of intelligence, improvements in reading skills tend to lead to improved reasoning skills (Van Der Maas *et al.*, 2006; Kievit *et al.*, 2017). Based on evidence summarised in the longitudinal section, one could consider uncontrolled eating and BMI as mutual variables – higher uncontrolled eating leads to weight gain, and higher weight leads to higher uncontrolled eating. In case of psychological processes behind uncontrolled eating, it seems unlikely that the mutualism could occur between eating traits in Table 1. The common construct model seems

most plausible due to explicit content similarity – overeating is the common theme in most of the questionnaires.

At the same time, mutualism is more likely at the level of underlying psychological traits. One could imagine a vicious circle between uncontrolled eating, reward sensitivity, self-control and negative affect. Namely, reward sensitivity leads to food cravings, which increases the amount of food people eat, indexed by uncontrolled eating. Such increased food cravings create challenges for cognitive-control abilities, which may sometimes fail. Such failures discourage people from attempting cognitive-control in the future and could bring in negative affect and stress. Should overeating lead to obesity, the association between uncontrolled eating and reward sensitivity could reverse. Paraphrasing Van der Maas et al (Van Der Maas et al., 2006), initially all processes of the system (reward sensitivity, cognitive control, and negative affect) could be undeveloped and uncorrelated with each other or with uncontrolled eating. However, when the cognitive system develops through interacting with the food environment, the dynamical interactions could give rise to correlations among the processes of the system.

Limitations

The current review is not without limitations. Due to space constraints, we have not discussed restrained eating (i.e., dieting) and other weight-related constructs, such as body shape concerns, which are likely to interact with uncontrolled eating.

We have also given relatively little space to studies attempting to show the incremental validity of one eating-related trait above others (van Strien *et al.*, 2012). Mostly, the field lacks convincing demonstrations, as such studies are hard to perform since they require large samples and appropriate modelling (Westfall & Yarkoni, 2016; Vainik & Meule, 2018). Therefore, we

would like to place the burden on test developers in outlining the evidence of the questionnaire's relation to outcomes above the common uncontrolled eating construct, should this be a goal.

Currently, uncontrolled eating measures are all based on self-report questionnaires. As a general limitation of this technique, people can only report on phenomena they are able perceive. If people cannot differentiate the theoretical distinctions between eating-related subdomains, it will be hard to tease them apart using self-report methods. Similarly, people are somewhat inaccurate in summarising their past emotions (Thomas & Diener, 1990; Mill *et al.*, 2016) and their eating behaviours (Stubbs *et al.*, 2014), and make mistakes in attributing causes of overeating when emotions and eating intertwine (Royal & Kurtz, 2010; Adriaanse *et al.*, 2016). Therefore, future studies should ideally sample the psychological processes of interest by using either "objective", perhaps behavioural measures of uncontrolled eating (Oustric *et al.*, 2018), or use experience sampling methods, which overcome recall bias (Bejarano & Cushing, 2018).

Concluding remarks and future directions

Our review has shown that, when different eating-related questionnaires are treated as measures of the same trait (i.e., uncontrolled eating), robust patterns emerge when summarising the literature. Uncontrolled eating has phenotypic, genetic, and environmental associations with obesity status and food intake (Figure 3). While these associations seem convincing, future meta-analyses should document their true effect sizes, and whether they vary depending on the questionnaire used.

When summarising longitudinal findings, uncontrolled eating and BMI are somewhat separable phenomena mutually influencing each other. There seems to be a clear need for designing weight loss interventions in a way that they target both reduction in weight and reduction in uncontrolled eating. On the other hand, uncontrolled eating and food intake seem less separable

and appear to partly vary day-by-day. There is then great potential in designing studies utilising moment-by-moment experience sampling, to understand the dynamics between uncontrolled eating and food intake.

At the psychological trait level, uncontrolled eating seems to relate to at least three common psychological underlying traits – high reward sensitivity, low self-control, and high perceived negativity. These traits likely have different correlates at the brain systems level. Our meta-analysis highlights the correlation between uncontrolled eating and activity in brain areas related to self-control.

These brain mechanisms and psychological traits will hopefully lead to understanding the more mechanistic underpinnings of uncontrolled eating. It may well be that uncontrolled eating is not a unitary construct, but a result of complex interplay between each element. For studies seeking to understand these details, it is crucial to isolate the cognitive processes that are the "building blocks" of uncontrolled eating and see how they unfold and interact over time.

In contrast, studies interested in capturing the common uncontrolled eating factor may apply only one of the questionnaires listed in Table 1, as each of them is a reasonable indicator of uncontrolled eating. This would considerably reduce participant burden. The ADOPT initiative (Sutin *et al.*, 2018) has highlighted several short and psychometrically sound questionnaires of uncontrolled eating, which include the RED-13/RED-X5 (Vainik *et al.*, In press; Mason *et al.*, 2017).

In summary, we suggest that there is a benefit in a common uncontrolled eating construct. Granted, it might not be uncontrolled eating that *causes* variance in all questionnaires related to uncontrolled eating. However, since in practical terms the questionnaires correlate, aggregating the results across questionnaires provides solid evidence of associations of uncontrolled eating

with external variables. Given the increased statistical power gained by aggregating all the results found by any uncontrolled eating measure, there seems to be a benefit in lumping different uncontrolled eating measures together for now.

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Competing interests
The authors declare no conflicts of interest.

Author Contributions
Study conception and design (UV, IGG, AD), literature search (UV, IGG), interpretation of results (UV, IGG) and manuscript writing (UV, IGG, AD).

Data Accessibility Statement
The review is based on previously published data. Data extracted from papers for meta analysis is available as a supplementary data.

Abbreviations
ACE models: A = additive genetic effects, C = shared environment effects, E = unshared environment effects; BMI=body mass index; BOLD=blood-oxygen-level dependent; DLPFC=dorsolateral prefrontal cortex; DSM= Diagnostic and Statistical Manual of Mental Disorders; fMRI=functional magnetic resonance imaging; HPA= hypothalamic-pituitary-adrenal; MRI=magnetic resonance imaging; SDM=Seed-Based d Mapping; SN=substantia nigra; SNc= substantia nigra pars compacta; TFEQ=Three-Factor Eating Questionnaire; vmPFC= ventromedial prefrontal cortex; VTA= ventral tegmental area

References

Adams, W.K., Sussman, J.L., Kaur, S., D'souza, A.M., Kieffer, T.J., & Winstanley, C.A. (2015) Long-term, calorie-restricted intake of a high-fat diet in rats reduces impulse control and ventral striatal D2receptor signalling - two markers of addiction vulnerability. *Eur. J. Neurosci.*, **42**, 3095–3104.

Adriaanse, M.A., Prinsen, S., de Witt Huberts, J.C., de Ridder, D.T.D., & Evers, C. (2016) 'I ate too much so I must have been sad': Emotions as a confabulated reason for overeating. *Appetite*, Consumer psychology and portion size: making smaller better, **103**, 318–323.

American Psychiatric Association (2013) *Diagnostic and Statistical Manual of Mental Disorders*, Fifth Edition. edn, DSM Library. American Psychiatric Association.

Appelhans, B.M., Woolf, K., Pagoto, S.L., Schneider, K.L., Whited, M.C., & Liebman, R. (2011) Inhibiting food reward: Delay discounting, food reward sensitivity, and palatable food intake in overweight and obese women. *Obesity*, **19**, 2175–2182.

Balodis, I.M., Grilo, C.M., & Potenza, M.N. (2015) Neurobiological features of binge eating disorder. *CNS Spectr.*, **20**, 557–565.

Bartholdy, S., Dalton, B., O'Daly, O.G., Campbell, I.C., & Schmidt, U. (2016) A systematic review of the relationship between eating, weight and inhibitory control using the stop signal task. *Neurosci. Biobehav. Rev.*, **64**, 35–62.

Bartra, O., McGuire, J.T., & Kable, J.W. (2013) The valuation system: A coordinate-based meta-analysis of BOLD fMRI experiments examining neural correlates of subjective value. *NeuroImage*, **76**, 412–427.

Bejarano, C.M. & Cushing, C.C. (2018) Dietary Motivation and Hedonic Hunger Predict Palatable Food Consumption: An Intensive Longitudinal Study of Adolescents. *Ann. Behav. Med. Publ. Soc. Behav. Med.*, **52**, 773–786.

Benton, D. & Young, H.A. (2016) A meta-analysis of the relationship between brain dopamine receptors and obesity: a matter of changes in behavior rather than food addiction? *Int. J. Obes. 2005*, **40 Suppl 1**, S12-21.

Blundell, J.E. & Finlayson, G. (2004) Is susceptibility to weight gain characterized by homeostatic or hedonic risk factors for overconsumption? *Physiol. Behav.*, Festschrift in Honor of Gerard P. Smith, **82**, 21–25.

Boggiano, M.M., Wenger, L.E., Turan, B., Tatum, M.M., Morgan, P.R., & Sylvester, M.D. (2015) Eating tasty food to cope. Longitudinal association with BMI. *Appetite*, **87**, 365–370.

Bongers, P. & Jansen, A. (2016) Emotional Eating Is Not What You Think It Is and Emotional Eating Scales Do Not Measure What You Think They Measure. *Front. Psychol.*, **7**.

Borsboom, D. (2006) The attack of the psychometricians. *Psychometrika*, **71**, 425–440.

Brace, A. & Yeomans, M.R. (2016) The reinforcing value of palatable snack foods and its relationship to subtypes of behavioural and self-report impulsivity. *Eat. Behav.*, **21**, 18–23.

Bryant, E., King, N., & Blundell, J. (2008) Disinhibition: Its effects on appetite and weight regulation. *Obes. Rev.*, **9**, 409–419.

Buckner, R.L. (2013) The cerebellum and cognitive function: 25 years of insight from anatomy and neuroimaging. *Neuron*, **80**, 807–815.

Calvo, D., Galioto, R., Gunstad, J., & Spitznagel, M.B. (2014) Uncontrolled eating is associated with reduced executive functioning: Uncontrolled eating and cognitive function. *Clin. Obes.*, **4**, 172–179.

Cappelleri, J.C., Bushmakin, A.G., Gerber, R.A., Leidy, N.K., Sexton, C.C., Lowe, M.R., & Karlsson, J. (2009) Psychometric analysis of the Three-Factor Eating Questionnaire-R21: Results from a large diverse sample of obese and non-obese participants. *Int. J. Obes.*, **33**, 611–620.

Carlier, N., Marshe, V.S., Cmorejova, J., Davis, C., & Müller, D.J. (2015) Genetic Similarities between Compulsive Overeating and Addiction Phenotypes: A Case for "Food Addiction"? *Curr. Psychiatry Rep.*, **17**, 96.

Caspi, A., Houts, R.M., Belsky, D.W., Goldman-Mellor, S.J., Harrington, H., Israel, S., Meier, M.H., Ramrakha, S., Shalev, I., Poulton, R., & Moffitt, T.E. (2014) The p Factor: One General Psychopathology Factor in the Structure of Psychiatric Disorders? *Clin. Psychol. Sci. J. Assoc. Psychol. Sci.*, **2**, 119.

Chaput, J.-P., Després, J.-P., Bouchard, C., & Tremblay, A. (2011) The Association between Short Sleep Duration and Weight Gain Is Dependent on Disinhibited Eating Behavior in Adults. *Sleep*, **34**, 1291–1297.

Chaput, J.P., Sjödin, A.M., Astrup, A., Després, J.P., Bouchard, C., & Tremblay, A. (2010) Risk factors for adult overweight and obesity: The importance of looking beyond the "Big Two." *Obes. Facts*, **3**, 320–327.

Contreras-Rodríguez, O., Martín-Pérez, C., Vilar-López, R., & Verdejo-Garcia, A. (2017) Ventral and Dorsal Striatum Networks in Obesity: Link to Food Craving and Weight Gain. *Biol. Psychiatry*, **81**, 789–796.

Cornelis, M.C., Flint, A., Field, A.E., Kraft, P., Han, J., Rimm, E.B., & van Dam, R.M. (2016) A genome-wide investigation of food addiction. *Obesity*, **24**, 1336–1341.

Cornelis, M.C., Rimm, E.B., Curhan, G.C., Kraft, P., Hunter, D.J., Hu, F.B., & van Dam, R.M. (2014) Obesity susceptibility loci and uncontrolled eating, emotional eating and cognitive restraint behaviors in men and women. *Obes. Silver Spring Md*, **22**, E135-141.

Costa, P.T. & McCrae, R.R. (1992) *Revised NEO Personality Inventory (NEO-PI-R) and NEO Five-Factor Inventory (NEOFFI) – Professional Manual.* Psychological Assessment Resources, Inc., Odessa, FL.

Dagher, A. (2012) Functional brain imaging of appetite. *Trends Endocrinol. Metab. Tem*, **23**, 250–260.

Dakanalis, A., Riva, G., Serino, S., Colmegna, F., & Clerici, M. (2017) Classifying Adults with Binge Eating Disorder Based on Severity Levels. *Eur. Eat. Disord. Rev.*, **25**, 268–274.

Dalle Grave, R., Calugi, S., Corica, F., Di Domizio, S., & Marchesini, G. (2009) Psychological variables associated with weight loss in obese patients seeking treatment at medical centers. *J. Am. Diet. Assoc.*, **109**, 2010–2016.

Dang, L.C., Samanez-Larkin, G.R., Castrellon, J.J., Perkins, S.F., Cowan, R.L., & Zald, D.H. (2016) Associations between dopamine D2 receptor availability and BMI depend on age. *NeuroImage*, **138**, 176–183.

Davis, C. (2013) From Passive Overeating to "Food Addiction": A Spectrum of Compulsion and Severity. *ISRN Obes.*, **2013**, e435027.

Davis, C. & Fox, J. (2008) Sensitivity to reward and body mass index (BMI): Evidence for a non-linear relationship. *Appetite*, **50**, 43–49.

Davis, C., Loxton, N.J., Levitan, R.D., Kaplan, A.S., Carter, J.C., & Kennedy, J.L. (2013) 'Food addiction' and its association with a dopaminergic multilocus genetic profile. *Physiol. Behav.*, **118**, 63–69.

de Castro, J.M. & Lilenfeld, L.R.R. (2005) Influence of heredity on dietary restraint, disinhibition, and perceived hunger in humans. *Nutrition*, **21**, 446–455.

Demos, K.E., Heatherton, T.F., & Kelley, W.M. (2012) Individual differences in nucleus accumbens activity to food and sexual images predict weight gain and sexual behavior. *J. Neurosci. Off. J. Soc. Neurosci.*, **32**, 5549–5552.

Dietrich, A., Hollmann, M., Mathar, D., Villringer, A., & Horstmann, A. (2016) Brain regulation of food craving: relationships with weight status and eating behavior. *Int. J. Obes.*, **40**, 982–989.

Eisenstein, S.A., Antenor-Dorsey, J.A., Gredysa, D.M., Koller, J.M., Bihun, E.C., Ranck, S.A., Arbelaez, A.M., Klein, S., Perlmutter, J.S., Moerlein, S.M., Black, K.J., & Hershey, T. (2013) A comparison of D2 receptor specific binding in obese and normal-weight individuals using PET with (N-[(11) C]methyl)benperidol. *Synap. N. Y. N*, **67**, 748–756.

Eisenstein, S.A., Gredysa, D.M., Antenor-Dorsey, J.A., Green, L., Arbeláez, A.M., Koller, J.M., Black, K.J., Perlmutter, J.S., Moerlein, S.M., & Hershey, T. (2015) Insulin , central dopamine D2 receptors , and monetary reward discounting in obesity. *PloS One*, 1–20.

Elfhag, K. & Morey, L.C. (2008) Personality traits and eating behavior in the obese: poor self-control in emotional and external eating but personality assets in restrained eating. *Eat. Behav.*, **9**, 285–293.

Emery, R.L. & Levine, M.D. (2017) Questionnaire and behavioral task measures of impulsivity are differentially associated with body mass index: A comprehensive meta-analysis. *Psychol. Bull.*, **143**, 868–902.

Eneva, K.T., Murray, S.M., & Chen, E.Y. (2017) Binge-eating disorder may be distinguished by visuospatial memory deficits. *Eat. Behav.*, **26**, 159–162.

Espel-Huynh, H.M., Muratore, A.F., & Lowe, M.R. (2018) A narrative review of the construct of hedonic hunger and its measurement by the Power of Food Scale: Hedonic hunger review. *Obes. Sci. Pract.*,.

Evers, C., Stok, F.M., Danner, U.N., Salmon, S.J., de Ridder, D.T.D., & Adriaanse, M.A. (2011) The shaping role of hunger on self-reported external eating status. *Appetite*, **57**, 318–320.

Feller, S., Müller, A., Mayr, A., Engeli, S., Hilbert, A., & de Zwaan, M. (2015) What distinguishes weight loss maintainers of the German Weight Control Registry from the general population? *Obesity*, **23**, 1112–1118.

Finlayson, G., Cecil, J., Higgs, S., Hill, A., & Hetherington, M. (2012) Susceptibility to weight gain. Eating behaviour traits and physical activity as predictors of weight gain during the first year of university. *Appetite*, **58**, 1091–1098.

Frankort, A., Roefs, A., Siep, N., Roebroeck, A., Havermans, R., & Jansen, A. (2015) Neural predictors of chocolate intake following chocolate exposure. *Appetite*, **87**, 98–107.

Frayn, M. & Knäuper, B. (2017) Emotional Eating and Weight in Adults: a Review. *Curr. Psychol.*, 1–10.

French, S.A., Epstein, L.H., Jeffery, R.W., Blundell, J.E., & Wardle, J. (2012) Eating behavior dimensions. Associations with energy intake and body weight. A review. *Appetite*, **59**, 541–549.

French, S.A., Mitchell, N.R., Wolfson, J., Finlayson, G., Blundell, J.E., & Jeffery, R.W. (2014) Questionnaire and laboratory measures of eating behavior. Associations with energy intake and BMI in a community sample of working adults. *Appetite*, **72**, 50–58.

García-García, I., Horstmann, A., Jurado, M.A., Garolera, M., Chaudhry, S.J., Margulies, D.S., Villringer, A., & Neumann, J. (2014) Reward processing in obesity, substance addiction and non-substance addiction. *Obes. Rev.*, **15**, 853–869.

García-García, I., Kube, J., Gaebler, M., Horstmann, A., Villringer, A., & Neumann, J. (2016) Neural processing of negative emotional stimuli and the influence of age, sex and task-related characteristics. *Neurosci. Biobehav. Rev.*, **68**, 773–793.

García-García, I., Zeighami, Y., & Dagher, A. (2017) Reward prediction errors in drug addiction and Parkinson's disease: from neurophysiology to neuroimaging. *Curr. Neurol. Neurosci. Rep.*, **17**, 46.

Gearhardt, A.N., Corbin, W.R., & Brownell, K.D. (2009) Preliminary validation of the Yale food addiction scale. *Appetite*, **52**, 430–436.

Geha, P.Y., Aschenbrenner, K., Felsted, J., O'Malley, S.S., & Small, D.M. (2013) Altered hypothalamic response to food in smokers. *Am. J. Clin. Nutr.*, **97**, 15–22.

Gerlach, G., Herpertz, S., & Loeber, S. (2015) Personality traits and obesity: a systematic review. *Obes. Rev. Off. J. Int. Assoc. Study Obes.*, **16**, 32–63.

Gibson, E.L. (2012) The psychobiology of comfort eating: implications for neuropharmacological interventions. *Behav. Pharmacol.*, **23**, 442.

Goldman, R.L., Canterberry, M., Borckardt, J.J., Madan, A., Byrne, T.K., George, M.S., O'Neil, P.M., & Hanlon, C.A. (2013) Executive control circuitry differentiates degree of success in weight loss following gastric-bypass surgery. *Obesity*, **21**, 2189–2196.

Goldschmidt, A.B. (2017) Are loss of control while eating and overeating valid constructs? A critical review of the literature. *Obes. Rev.*, **18**, 412–449.

Gottfredson, L.S. & Deary, I.J. (2004) Intelligence Predicts Health and Longevity, but Why? *Curr. Dir. Psychol. Sci.*, **13**, 1–4.

Graff, M., Scott, R.A., Justice, A.E., Young, K.L., Feitosa, M.F., Barata, L., Winkler, T.W., Chu, A.Y., Mahajan, A., Hadley, D., Xue, L., Workalemahu, T., Heard-Costa, N.L., den Hoed, M., Ahluwalia, T.S., Qi, Q., Ngwa, J.S., Renström, F., Quaye, L., Eicher, J.D., Hayes, J.E., Cornelis, M., Kutalik, Z., Lim, E., Luan, J., Huffman, J.E., Zhang, W., Zhao, W., Griffin, P.J., Haller, T., Ahmad, S., Marques-Vidal, P.M., Bien, S., Yengo, L., Teumer, A., Smith, A.V., Kumari, M., Harder, M.N., Justesen, J.M., Kleber, M.E., Hollensted, M., Lohman, K., Rivera, N. V., Whitfield, J.B., Zhao, J.H., Stringham, H.M., Lyytikäinen, L.-P., Huppertz, C., Willemsen, G., Peyrot, W.J., Wu, Y., Kristiansson, K., Demirkan, A., Fornage, M., Hassinen, M., Bielak, L.F., Cadby, G., Tanaka, T., Mägi, R., van der Most, P.J., Jackson, A.U., Bragg-Gresham, J.L., Vitart, V., Marten, J., Navarro, P., Bellis, C., Pasko, D., Johansson, Å., Snitker, S., Cheng, Y.-C., Eriksson, J., Lim, U., Aadahl, M., Adair, L.S., Amin, N., Balkau, B., Auvinen, J., Beilby, J., Bergman, R.N., Bergmann, S., Bertoni, A.G., Blangero, J., Bonnefond, A., Bonnycastle, L.L., Borja, J.B., Brage, S., Busonero, F., Buyske, S., Campbell, H., Chines, P.S., Collins, F.S., Corre, T., Smith, G.D., Delgado, G.E., Dueker, N., Dörr, M., Ebeling, T., Eiriksdottir, G., Esko, T., Faul, J.D., Fu, M., Færch, K., Gieger, C., Gläser, S., Gong, J., Gordon-Larsen, P., Grallert, H., Grammer, T.B., Grarup, N., van Grootheest, G., Harald, K., Hastie, N.D., Havulinna, A.S., Hernandez, D., Hindorff, L., Hocking, L.J., Holmens, O.L., Holzapfel, C., Hottenga, J.J., Huang, J., Huang, T., Hui, J., Huth, C., Hutri-Kähönen, N., James, A.L., Jansson, J.-O., Jhun, M.A., Juonala, M., Kinnunen, L., Koistinen, H.A., Kolcic, I., Komulainen, P., Kuusisto, J., Kvaløy, K., Kähönen, M., Lakka, T.A., Launer, L.J., Lehne, B., Lindgren, C.M., Lorentzon, M., Luben, R., Marre, M., Milaneschi, Y., Monda,

K.L., Montgomery, G.W., De Moor, M.H.M., Mulas, A., Müller-Nurasyid, M., Musk, A.W., Männikkö, R., Männistö, S., Narisu, N., Nauck, M., Nettleton, J.A., Nolte, I.M., Oldehinkel, A.J., Olden, M., Ong, K.K., Padmanabhan, S., Paternoster, L., Perez, J., Perola, M., Peters, A., Peters, U., Peyser, P.A., Prokopenko, I., Puolijoki, H., Raitakari, O.T., Rankinen, T., Rasmussen-Torvik, L.J., Rawal, R., Ridker, P.M., Rose, L.M., Rudan, I., Sarti, C., Sarzynski, M.A., Savonen, K., Scott, W.R., Sanna, S., Shuldiner, A.R., Sidney, S., Silbernagel, G., Smith, B.H., Smith, J.A., Snieder, H., Stančáková, A., Sternfeld, B., Swift, A.J., Tammelin, T., Tan, S.-T., Thorand, B., Thuillier, D., Vandenput, L., Vestergaard, H., van Vliet-Ostaptchouk, J. V., Vohl, M.-C., Völker, U., Waeber, G., Walker, M., Wild, S., Wong, A., Wright, A.F., Zillikens, M.C., Zubair, N., Haiman, C.A., Lemarchand, L., Gyllensten, U., Ohlsson, C., Hofman, A., Rivadeneira, F., Uitterlinden, A.G., Pérusse, L., Wilson, J.F., Hayward, C., Polasek, O., Cucca, F., Hveem, K., Hartman, C.A., Tönjes, A., Bandinelli, S., Palmer, L.J., Kardia, S.L.R., Rauramaa, R., Sørensen, T.I.A., Tuomilehto, J., Salomaa, V., Penninx, B.W.J.H., de Geus, E.J.C., Boomsma, D.I., Lehtimäki, T., Mangino, M., Laakso, M., Bouchard, C., Martin, N.G., Kuh, D., Liu, Y., Linneberg, A., März, W., Strauch, K., Kivimäki, M., Harris, T.B., Gudnason, V., Völzke, H., Qi, L., Järvelin, M.-R., Chambers, J.C., Kooner, J.S., Froguel, P., Kooperberg, C., Vollenweider, P., Hallmans, G., Hansen, T., Pedersen, O., Metspalu, A., Wareham, N.J., Langenberg, C., Weir, D.R., Porteous, D.J., Boerwinkle, E., Chasman, D.I., Abecasis, G.R., Barroso, I., McCarthy, M.I., Frayling, T.M., O'Connell, J.R., van Duijn, C.M., Boehnke, M., Heid, I.M., Mohlke, K.L., Strachan, D.P., Fox, C.S., Liu, C.-T., Hirschhorn, J.N., Klein, R.J., Johnson, A.D., Borecki, I.B., Franks, P.W., North, K.E., Cupples, L.A., Loos, R.J.F., & Kilpeläinen, T.O. (2017) Genome-wide physical activity interactions in adiposity - A meta-analysis of 200,452 adults. *PLoS Genet.*, **13**, e1006528.

Greeno, C.G. & Wing, R.R. (1994) Stress-induced eating. *Psychol. Bull.*, **115**, 444–464.

Hall, K.D. (2018) Did the Food Environment Cause the Obesity Epidemic? *Obesity*, **26**, 11–13.

Han, J.E., Boachie, N., Garcia-Garcia, I., Michaud, A., & Dagher, A. (2018) Neural correlates of dietary self-control in healthy adults: A meta-analysis of functional brain imaging studies. *Physiol. Behav.*, Proceedings of the SSIB 2017 Annual Meeting, **192**, 98–108.

Hargrave, S.L., Jones, S., & Davidson, T.L. (2016) The outward spiral: a vicious cycle model of obesity and cognitive dysfunction. *Curr Opin Behav Sci*, **9**, 40–46.

Hays, N.P., Bathalon, G.P., McCrory, M. a, Roubenoff, R., Lipman, R., & Roberts, S.B. (2002) Eating behavior correlates of adult weight gain and obesity in healthy women aged 55 – 65 y. *Am. J. Clin. Nutr.*, **75**, 476–483.

Hays, N.P., Bathalon, G.P., Roubenoff, R., McCrory, M.A., & Roberts, S.B. (2006) Eating behavior and weight change in healthy postmenopausal women: Results of a 4-year longitudinal study. *J. Gerontol. - Ser. Biol. Sci. Med. Sci.*, **61**, 608–615.

Hege, M.A., Stingl, K.T., Kullmann, S., Schag, K., Giel, K.E., Zipfel, S., & Preissl, H. (2015) Attentional impulsivity in binge eating disorder modulates response inhibition performance and frontal brain networks. *Int. J. Obes. 2005*, **39**, 353–360.

Herman, C.P. & Polivy, J. (2008) External cues in the control of food intake in humans: the sensory-normative distinction. *Physiol. Behav.*, **94**, 722–728.

Herman, J.P., McKlveen, J.M., Ghosal, S., Kopp, B., Wulsin, A., Makinson, R., Scheimann, J., & Myers, B. (2016) Regulation of the hypothalamic-pituitary- adrenocortical stress response. *Compr. Physiol.*, **6**, 603–621.

Higgs, S. & Spetter, M.S. (2018) Cognitive Control of Eating: the Role of Memory in Appetite and Weight Gain. *Curr. Obes. Rep.*, **7**, 50–59.

Holsen, L.M., Davidson, P., Cerit, H., Hye, T., Moondra, P., Haimovici, F., Sogg, S., Shikora, S., Goldstein, J.M., Evins, A.E., & Stoeckel, L.E. (2017) Neural predictors of 12-month weight loss outcomes following bariatric surgery. *Int. J. Obes.*, 1–9.

Horn, E.E., Turkheimer, E., Strachan, E., & Duncan, G.E. (2015) Behavioral and Environmental Modification of the Genetic Influence on Body Mass Index: A Twin Study. *Behav. Genet.*, **45**, 409–426.

Horstmann, A., Fenske, W.K., & Hankir, M.K. (2015) Argument for a non-linear relationship between severity of human obesity and dopaminergic tone. *Obes. Rev.*, **16**, 821–830.

Ivezaj, V., Wiedemann, A.A., & Grilo, C.M. (2017) Food addiction and bariatric surgery: a systematic review of the literature. *Obes. Rev.*, **18**, 1386–1397.

Janak, P.H. & Tye, K.M. (2015) From circuits to behaviour in the amygdala. *Nature*, **517**, 284–292.

Jensen, A.R. (1998) *The g Factor: The Science of Mental Ability*, Human evolution, behavior, and intelligence. Praeger, Westport, Conn.

Jensen, C.D. & Kirwan, C.B. (2015) Functional brain response to food images in successful adolescent weight losers compared with normal-weight and overweight controls. *Obesity*, **23**, 630–636.

Johnson, F., Pratt, M., & Wardle, J. (2011) Dietary restraint and self-regulation in eating behavior. *Int. J. Obes.*, **36**, 665–674.

Jokela, M., Hintsanen, M., Hakulinen, C., Batty, G.D., Nabi, H., Singh-Manoux, A., & Kivimäki, M. (2013) Association of personality with the development and persistence of obesity: a meta-analysis based on individual–participant data. *Obes. Rev.*, **14**, 315–323.

Kakoschke, N., Kemps, E., & Tiggemann, M. (2015) External eating mediates the relationship between impulsivity and unhealthy food intake. *Physiol. Behav.*, **147**, 117–121.

Karlsson, H.K., Tuominen, L., Tuulari, J.J., Hirvonen, J., Parkkola, R., Helin, S., Salminen, P., Nuutila, P., & Nummenmaa, L. (2015) Obesity is associated with decreased -opioid but unaltered dopamine D2 receptor availability in the brain. *J. Neurosci.*, **35**, 3959–3965.

Karlsson, J., Persson, L.O., Sjöström, L., & Sullivan, M. (2000) Psychometric properties and factor structure of the Three-Factor Eating Questionnaire (TFEQ) in obese men and women. Results from the Swedish Obese Subjects (SOS) study. *Int. J. Obes. Relat. Metab. Disord. J. Int. Assoc. Study Obes.*, **24**, 1715–1725.

Keller, C. & Hartmann, C. (2016) Not merely a question of self-control: The longitudinal effects of overeating behaviors, diet quality and physical activity on dieters' perceived diet success. *Appetite*, **107**, 213–221.

Keller, C. & Siegrist, M. (2015) Does personality influence eating styles and food choices? Direct and indirect effects. *Appetite*, **84**, 128–138.

Kelley, T.L. (1927) *Interpretation of Educational Measurements*. World Book Co., Oxford, England.

Keskitalo, K., Tuorila, H., Spector, T.D., Cherkas, L.F., Knaapila, A., Kaprio, J., Silventoinen, K., & Perola, M. (2008) The Three-Factor Eating Questionnaire, body mass index, and responses to sweet and salty fatty foods: a twin study of genetic and environmental associations. *Am. J. Clin. Nutr.*, **88**, 263–271.

Kievit, R.A., Lindenberger, U., Goodyer, I.M., Jones, P.B., Fonagy, P., Bullmore, E.T., Network, the N. in P., & Dolan, R.J. (2017) Mutualistic Coupling Between Vocabulary and

Reasoning Supports Cognitive Development During Late Adolescence and Early Adulthood. *Psychol. Sci.*, **28**, 1419–1431.

Koball, A.M., Clark, M.M., Collazo-Clavell, M., Kellogg, T., Ames, G., Ebbert, J., & Grothe, K.B. (2016) The relationship among food addiction, negative mood, and eating-disordered behaviors in patients seeking to have bariatric surgery. *Surg. Obes. Relat. Dis.*, **12**, 165–170.

Konttinen, H., Kronholm, E., Partonen, T., Kanerva, N., Männistö, S., & Haukkala, A. (2014) Morningness–eveningness, depressive symptoms, and emotional eating: A population-based study. *Chronobiol. Int.*, **31**, 554–563.

Konttinen, H., Llewellyn, C., Wardle, J., Silventoinen, K., Joensuu, A., Männistö, S., Salomaa, V., Jousilahti, P., Kaprio, J., Perola, M., & Haukkala, A. (2015) Appetitive traits as behavioural pathways in genetic susceptibility to obesity: a population-based cross-sectional study. *Sci. Rep.*, **5**, 14726.

Koob, G.F. & Le Moal, M. (1997) Drug abuse: hedonic homeostatic dysregulation. *Science*, **278**, 52–58.

Kozak, M.J. & Cuthbert, B.N. (2016) The NIMH Research Domain Criteria Initiative: Background, Issues, and Pragmatics. *Psychophysiology*, **53**, 286–297.

Koziol, L.F., Budding, D., Andreasen, N., D'Arrigo, S., Bulgheroni, S., Imamizu, H., Ito, M., Manto, M., Marvel, C., Parker, K., Pezzulo, G., Ramnani, N., Riva, D., Schmahmann, J., Vandervert, L., & Yamazaki, T. (2014) Consensus paper: The cerebellum's role in movement and cognition. *Cerebellum*, **13**, 151–177.

Laceulle, O.M., Vollebergh, W.A.M., & Ormel, J. (2015) The Structure of Psychopathology in Adolescence: Replication of a General Psychopathology Factor in the TRAILS Study. *Clin. Psychol. Sci.*, **3**, 850–860.

Laessle, R. & Geiermann, L. (2012) Reliability of laboratory measurement of human food intake. *Appetite*, **58**, 249–251.

Lavagnino, L., Arnone, D., Cao, B., Soares, J.C., & Selvaraj, S. (2016) Inhibitory control in obesity and binge eating disorder: A systematic review and meta-analysis of neurocognitive and neuroimaging studies. *Neurosci. Biobehav. Rev.*, **68**, 714–726.

Lawlor, D.A., Harbord, R.M., Sterne, J.A.C., Timpson, N., & Davey Smith, G. (2008) Mendelian randomization: using genes as instruments for making causal inferences in epidemiology. *Stat. Med.*, **27**, 1133–1163.

Lawrence, N.S., Hinton, E.C., Parkinson, J.A., & Lawrence, A.D. (2012) Nucleus accumbens response to food cues predicts subsequent snack consumption in women and increased body mass index in those with reduced self-control. *NeuroImage*, **63**, 415–422.

Legenbauer, T.M., de Zwaan, M., Mühlhans, B., Petrak, F., & Herpertz, S. (2010) Do mental disorders and eating patterns affect long-term weight loss maintenance? *Gen. Hosp. Psychiatry*, **32**, 132–140.

Leitch, M.A., Morgan, M.J., & Yeomans, M.R. (2013) Different subtypes of impulsivity differentiate uncontrolled eating and dietary restraint. *Appetite*, **69**, 54–63.

Lipsky, L.M., Nansel, T.R., Haynie, D.L., Liu, D., Eisenberg, M.H., & Simons-Morton, B. (2016) Power of Food Scale in association with weight outcomes and dieting in a nationally representative cohort of U.S. young adults. *Appetite*, **105**, 385–391.

Liu, S., Globa, A.K., Mills, F., Naef, L., Qiao, M., Bamji, S.X., & Borgland, S.L. (2016) Consumption of palatable food primes food approach behavior by rapidly increasing synaptic density in the VTA. *Proc. Natl. Acad. Sci.*, **113**, 2520–2525.

Locke, A.E., Kahali, B., Berndt, S.I., Justice, A.E., Pers, T.H., Day, F.R., Powell, C., Vedantam, S., Buchkovich, M.L., Yang, J., Croteau-Chonka, D.C., Esko, T., Fall, T., Ferreira, T., Gustafsson, S., Kutalik, Z., Luan, J., Mägi, R., Randall, J.C., Winkler, T.W., Wood, A.R., Workalemahu, T., Faul, J.D., Smith, J.A., Zhao, J.H., Zhao, W., Chen, J., Fehrmann, R., Hedman, Å.K., Karjalainen, J., Schmidt, E.M., Absher, D., Amin, N., Anderson, D., Beekman, M., Bolton, J.L., Bragg-Gresham, J.L., Buyske, S., Demirkan, A., Deng, G., Ehret, G.B., Feenstra, B., Feitosa, M.F., Fischer, K., Goel, A., Gong, J., Jackson, A.U., Kanoni, S., Kleber, M.E., Kristiansson, K., Lim, U., Lotay, V., Mangino, M., Leach, I.M., Medina-Gomez, C., Medland, S.E., Nalls, M.A., Palmer, C.D., Pasko, D., Pechlivanis, S., Peters, M.J., Prokopenko, I., Shungin, D., Stančáková, A., Strawbridge, R.J., Sung, Y.J., Tanaka, T., Teumer, A., Trompet, S., van der Laan, S.W., van Setten, J., Van Vliet-Ostaptchouk, J. V., Wang, Z., Yengo, L., Zhang, W., Isaacs, A., Albrecht, E., Ärnlöv, J., Arscott, G.M., Attwood, A.P., Bandinelli, S., Barrett, A., Bas, I.N., Bellis, C., Bennett, A.J., Berne, C., Blagieva, R., Blüher, M., Böhringer, S., Bonnycastle, L.L., Böttcher, Y., Boyd, H.A., Bruinenberg, M., Caspersen, I.H., Chen, Y.-D.I., Clarke, R., Daw, E.W., de Craen, A.J.M., Delgado, G., Dimitriou, M., Doney, A.S.F., Eklund, N., Estrada, K., Eury, E., Folkersen, L., Fraser, R.M., Garcia, M.E., Geller, F., Giedraitis, V., Gigante, B., Go, A.S., Golay, A., Goodall, A.H., Gordon, S.D., Gorski, M., Grabe, H.-J., Grallert, H., Grammer, T.B., Gräßler, J., Grönberg, H., Groves, C.J., Gusto, G., Haessler, J., Hall, P., Haller, T., Hallmans, G., Hartman, C.A., Hassinen, M., Hayward, C., Heard-Costa, N.L., Helmer, Q., Hengstenberg, C., Holmen, O., Hottenga, J.-J., James, A.L., Jeff, J.M., Johansson, Å., Jolley, J., Juliusdottir, T., Kinnunen, L., Koenig, W., Koskenvuo, M., Kratzer, W., Laitinen, J., Lamina, C., Leander, K., Lee, N.R., Lichtner, P., Lind, L., Lindström, J., Lo, K.S., Lobbens, S., Lorbeer, R., Lu, Y., Mach, F., Magnusson, P.K.E., Mahajan, A., McArdle, W.L., McLachlan, S., Menni, C., Merger, S., Mihailov, E., Milani, L., Moayyeri, A., Monda, K.L., Morken, M.A., Mulas, A., Müller, G., Müller-Nurasyid, M., Musk, A.W., Nagaraja, R., Nöthen, M.M., Nolte, I.M., Pilz, S., Rayner, N.W., Renstrom, F., Rettig, R., Ried, J.S., Ripke, S., Robertson, N.R., Rose, L.M., Sanna, S., Scharnagl, H., Scholtens, S., Schumacher, F.R., Scott, W.R., Seufferlein, T., Shi, J., Smith, A.V., Smolonska, J., Stanton, A. V., Steinthorsdottir, V., Stirrups, K., Stringham, H.M., Sundström, J., Swertz, M.A., Swift, A.J., Syvänen, A.-C., Tan, S.-T., Tayo, B.O., Thorand, B., Thorleifsson, G., Tyrer, J.P., Uh, H.-W., Vandenput, L., Verhulst, F.C., Vermeulen, S.H., Verweij, N., Vonk, J.M., Waite, L.L., Warren, H.R., Waterworth, D., Weedon, M.N., Wilkens, L.R., Willenborg, C., Wilsgaard, T., Wojczynski, M.K., Wong, A., Wright, A.F., Zhang, Q., Brennan, E.P., Choi, M., Dastani, Z., Drong, A.W., Eriksson, P., Franco-Cereceda, A., Gådin, J.R., Gharavi, A.G., Goddard, M.E., Handsaker, R.E., Huang, J., Karpe, F., Kathiresan, S., Keildson, S., Kiryluk, K., Kubo, M., Lee, J.-Y., Liang, L., Lifton, R.P., Ma, B., McCarroll, S.A., McKnight, A.J., Min, J.L., Moffatt, M.F., Montgomery, G.W., Murabito, J.M., Nicholson, G., Nyholt, D.R., Okada, Y., Perry, J.R.B., Dorajoo, R., Reinmaa, E., Salem, R.M., Sandholm, N., Scott, R.A., Stolk, L., Takahashi, A., Tanaka, T., van 't Hooft, F.M., Vinkhuyzen, A.A.E., Westra, H.-J., Zheng, W., Zondervan, K.T., Heath, A.C., Arveiler, D., Bakker, S.J.L., Beilby, J., Bergman, R.N., Blangero, J., Bovet, P., Campbell, H., Caulfield, M.J., Cesana, G., Chakravarti, A., Chasman, D.I., Chines, P.S., Collins, F.S., Crawford, D.C., Cupples, L.A., Cusi, D., Danesh, J., de Faire, U., den Ruijter, H.M., Dominiczak, A.F., Erbel, R., Erdmann, J., Eriksson, J.G., Farrall, M., Felix, S.B.,

Ferrannini, E., Ferrières, J., Ford, I., Forouhi, N.G., Forrester, T., Franco, O.H., Gansevoort, R.T., Gejman, P. V., Gieger, C., Gottesman, O., Gudnason, V., Gyllensten, U., Hall, A.S., Harris, T.B., Hattersley, A.T., Hicks, A.A., Hindorff, L.A., Hingorani, A.D., Hofman, A., Homuth, G., Hovingh, G.K., Humphries, S.E., Hunt, S.C., Hyppönen, E., Illig, T., Jacobs, K.B., Jarvelin, M.-R., Jöckel, K.-H., Johansen, B., Jousilahti, P., Jukema, J.W., Jula, A.M., Kaprio, J., Kastelein, J.J.P., Keinanen-Kiukaanniemi, S.M., Kiemeney, L.A., Knekt, P., Kooner, J.S., Kooperberg, C., Kovacs, P., Kraja, A.T., Kumari, M., Kuusisto, J., Lakka, T.A., Langenberg, C., Marchand, L. Le, Lehtimäki, T., Lyssenko, V., Männistö, S., Marette, A., Matise, T.C., McKenzie, C.A., McKnight, B., Moll, F.L., Morris, A.D., Morris, A.P., Murray, J.C., Nelis, M., Ohlsson, C., Oldehinkel, A.J., Ong, K.K., Madden, P.A.F., Pasterkamp, G., Peden, J.F., Peters, A., Postma, D.S., Pramstaller, P.P., Price, J.F., Qi, L., Raitakari, O.T., Rankinen, T., Rao, D.C., Rice, T.K., Ridker, P.M., Rioux, J.D., Ritchie, M.D., Rudan, I., Salomaa, V., Samani, N.J., Saramies, J., Sarzynski, M.A., Schunkert, H., Schwarz, P.E.H., Sever, P., Shuldiner, A.R., Sinisalo, J., Stolk, R.P., Strauch, K., Tönjes, A., Trégouët, D.-A., Tremblay, A., Tremoli, E., Virtamo, J., Vohl, M.-C., Völker, U., Waeber, G., Willemsen, G., Witteman, J.C., Zillikens, M.C., Adair, L.S., Amouyel, P., Asselbergs, F.W., Assimes, T.L., Bochud, M., Boehm, B.O., Boerwinkle, E., Bornstein, S.R., Bottinger, E.P., Bouchard, C., Cauchi, S., Chambers, J.C., Chanock, S.J., Cooper, R.S., de Bakker, P.I.W., Dedoussis, G., Ferrucci, L., Franks, P.W., Froguel, P., Groop, L.C., Haiman, C.A., Hamsten, A., Hui, J., Hunter, D.J., Hveem, K., Kaplan, R.C., Kivimaki, M., Kuh, D., Laakso, M., Liu, Y., Martin, N.G., März, W., Melbye, M., Metspalu, A., Moebus, S., Munroe, P.B., Njølstad, I., Oostra, B.A., Palmer, C.N.A., Pedersen, N.L., Perola, M., Pérusse, L., Peters, U., Power, C., Quertermous, T., Rauramaa, R., Rivadeneira, F., Saaristo, T.E., Saleheen, D., Sattar, N., Schadt, E.E., Schlessinger, D., Slagboom, P.E., Snieder, H., Spector, T.D., Thorsteinsdottir, U., Stumvoll, M., Tuomilehto, J., Uitterlinden, A.G., Uusitupa, M., van der Harst, P., Walker, M., Wallaschofski, H., Wareham, N.J., Watkins, H., Weir, D.R., Wichmann, H.-E., Wilson, J.F., Zanen, P., Borecki, I.B., Deloukas, P., Fox, C.S., Heid, I.M., O'Connell, J.R., Strachan, D.P., Stefansson, K., van Duijn, C.M., Abecasis, G.R., Franke, L., Frayling, T.M., McCarthy, M.I., Visscher, P.M., Scherag, A., Willer, C.J., Boehnke, M., Mohlke, K.L., Lindgren, C.M., Beckmann, J.S., Barroso, I., North, K.E., Ingelsson, E., Hirschhorn, J.N., Loos, R.J.F., & Speliotes, E.K. (2015) Genetic studies of body mass index yield new insights for obesity biology. *Nature*, **518**, 197–206.

Lohse, B., Satter, E., Horacek, T., Gebreselassie, T., & Oakland, M.J. (2007) Measuring eating competence: Psychometric properties and validity of the ecSatter inventory. *J. Nutr. Educ. Behav.*, **39**, S154–S166.

Lowe, M.R., Butryn, M.L., Didie, E.R., Annunziato, R.A., Thomas, J.G., Crerand, C.E., Ochner, C.N., Coletta, M.C., Bellace, D., & Wallaert, M. (2009) The Power of Food Scale. A new measure of the psychological influence of the food environment. *Appetite*, **53**, 114–118.

Lucas, R.E., Diener, E., Grob, A., Suh, E.M., & Shao, L. (2000) Cross-cultural evidence for the fundamental features of extraversion. *J. Pers. Soc. Psychol.*, **79**, 452–468.

Lunn, T.E., Nowson, C.A., Worsley, A., & Torres, S.J. (2014) Does personality affect dietary intake? *Nutrition*, **30**, 403–409.

Macht, M. (2008) How emotions affect eating: a five-way model. *Appetite*, **50**, 1–11.

Martin, A.A., Davidson, T.L., & McCrory, M.A. (2018) Deficits in episodic memory are related to uncontrolled eating in a sample of healthy adults. *Appetite*, Executive function training & eating behavior, **124**, 33–42.

Martin, C.K., O/'Neil, P.M., & Pawlow, L. (2006) Changes in Food Cravings during Low-Calorie and Very-Low-Calorie Diets[ast]. *Obesity*, **14**, 115–121.

Mason, A.E., Epel, E.S., Aschbacher, K., Lustig, R.H., Acree, M., Kristeller, J., Cohn, M., Dallman, M., Moran, P.J., Bacchetti, P., Laraia, B., Hecht, F.M., & Daubenmier, J. (2016) Reduced reward-driven eating accounts for the impact of a mindfulness-based diet and exercise intervention on weight loss: Data from the SHINE randomized controlled trial. *Appetite*, **100**, 86–93.

Mason, A.E., Vainik, U., Acree, M., Tomiyama, A.J., Dagher, A., Epel, E.S., & Hecht, F.M. (2017) Improving Assessment of the Spectrum of Reward-Related Eating: The RED-13. *Front. Psychol.*, **8**.

McCaffery, J.M., Papandonatos, G.D., Bond, D.S., Lyons, M.J., & Wing, R.R. (2009) Gene X environment interaction of vigorous exercise and body mass index among male Vietnam-era twins. *Am. J. Clin. Nutr.*, **89**, 1011–1018.

McCrae, R.R., Kurtz, J.E., Yamagata, S., & Terracciano, A. (2011) Internal Consistency, Retest Reliability, and their Implications For Personality Scale Validity. *Personal. Soc. Psychol. Rev. Off. J. Soc. Personal. Soc. Psychol. Inc*, **15**, 28–50.

Meany, G., Conceição, E., & Mitchell, J.E. (2014) Binge eating, binge eating disorder and loss of control eating: Effects on weight outcomes after bariatric surgery. *Eur. Eat. Disord. Rev.*, **22**, 87–91.

Meule, A. & Blechert, J. (2017) Indirect effects of trait impulsivity on body mass. *Eat. Behav.*, **26**, 66–69.

Micali, N., Ploubidis, G., De Stavola, B., Simonoff, E., & Treasure, J. (2014) Frequency and patterns of eating disorder symptoms in early adolescence. *J. Adolesc. Health*, **54**, 574–581.

Mill, A., Realo, A., & Allik, J. (2016) Retrospective Ratings of Emotions: the Effects of Age, Daily Tiredness, and Personality. *Front. Psychol.*, **6**.

Murray, S., Tulloch, A., Gold, M.S., & Avena, N.M. (2014) Hormonal and neural mechanisms of food reward, eating behaviour and obesity. *Nat. Rev. Endocrinol.*, **10**, 540–552.

Musek, J. (2007) A general factor of personality: Evidence for the Big One in the five-factor model. *J. Res. Personal.*, **41**, 1213–1233.

Neale, B.M., Mazzeo, S.E., & Bulik, C.M. (2003) A twin study of dietary restraint, disinhibition and hunger: an examination of the eating inventory (three factor eating questionnaire). *Twin Res. Off. J. Int. Soc. Twin Stud.*, **6**, 471–478.

Neseliler, S., Vainik, U., Dadar, M., Yau, Y.H., García-García, I., Scala, S.G., Zeighami, Y., Collins, D.L., & Dagher, A. (2018) Neural and behavioral endophenotypes of obesity. *bioRxiv*, 348821.

Nikolova, Y.S., Ferrell, R.E., Manuck, S.B., & Hariri, A.R. (2011) Multilocus Genetic Profile for Dopamine Signaling Predicts Ventral Striatum Reactivity. *Neuropsychopharmacology*, **36**, 1940–1947.

Oustric, P., Gibbons, C., Beaulieu, K., Blundell, J., & Finlayson, G. (2018) Changes in food reward during weight management interventions – a systematic review. *Obes. Rev.*, **19**, 1642–1658.

Paans, N.P.G., Bot, M., Brouwer, I.A., Visser, M., Roca, M., Kohls, E., Watkins, E., & Penninx, B.W.J.H. (2018) The association between depression and eating styles in four European countries: The MooDFOOD prevention study. *J. Psychosom. Res.*, **108**, 85–92.

Panov, L. (2014) Toitumiskompetentsust mõõtva küsimustiku ecSatter Inventory adapteerimine eesti keelde [Adaptation of the eating competence measurement scale ecSatter Inventory into Estonian] (Thesis).

Pessoa, L. & Adolphs, R. (2010) Emotion processing and the amygdala: from a 'low road' to 'many roads' of evaluating biological significance. *Nat. Rev. Neurosci.*, **11**, 773–783.

Polderman, T.J.C., Benyamin, B., de Leeuw, C.A., Sullivan, P.F., van Bochoven, A., Visscher, P.M., & Posthuma, D. (2015) Meta-analysis of the heritability of human traits based on fifty years of twin studies. *Nat. Genet.*, **47**, 702–709.

Pourtois, G., Schettino, A., & Vuilleumier, P. (2013) Brain mechanisms for emotional influences on perception and attention: What is magic and what is not. *Biol. Psychol.*, **92**, 492–512.

Price, M., Higgs, S., & Lee, M. (2015) Self-reported eating traits: Underlying components of food responsivity and dietary restriction are positively related to BMI. *Appetite*, **95**, 203–210.

Provencher, V., Bégin, C., Gagnon-Girouard, M.P., Tremblay, A., Boivin, S., & Lemieux, S. (2008) Personality traits in overweight and obese women: Associations with BMI and eating behaviors. *Eat. Behav.*, **9**, 294–302.

Pursey, K.M., Stanwell, P., Gearhardt, A.N., Collins, C.E., & Burrows, T.L. (2014) The prevalence of food addiction as assessed by the yale food addiction scale: A systematic review. *Nutrients*, **6**, 4552–4590.

Quick, V., Wall, M., Larson, N., Haines, J., & Neumark-Sztainer, D. (2013) Personal, behavioral and socio-environmental predictors of overweight incidence in young adults: 10-yr longitudinal findings. *Int. J. Behav. Nutr. Phys. Act.*, **10**, 37.

Radua, J., Mataix-Cols, D., Phillips, M.L., El-Hage, W., Kronhaus, D.M., Cardoner, N., & Surguladze, S. (2012) A new meta-analytic method for neuroimaging studies that combines reported peak coordinates and statistical parametric maps. *Eur. Psychiatry*, **27**, 605–611.

Radua, J., Rubia, K., Canales-Rodríguez, E.J., Pomarol-Clotet, E., Fusar-Poli, P., & Mataix-Cols, D. (2014) Anisotropic kernels for coordinate-based meta-analyses of neuroimaging studies. *Front. Psychiatry*, **5**, 1–8.

Royal, J.D. & Kurtz, J.L. (2010) I ate what?! The effect of stress and dispositional eating style on food intake and behavioral awareness. *Personal. Individ. Differ.*, **49**, 565–569.

Ruddock, H.K., Christiansen, P., Halford, J.C.G., & Hardman, C.A. (2017) The development and validation of the Addiction-like Eating Behaviour Scale. *Int. J. Obes.*, **41**, 1710–1717.

Rushton, J.P. & Irwing, P. (2008) A General Factor of Personality (GFP) from two meta-analyses of the Big Five: and. *Personal. Individ. Differ.*, **45**, 679–683.

Saunders, B.T. & Robinson, T.E. (2013) Individual variation in resisting temptation: implications for addiction. *Neurosci. Biobehav. Rev.*, **37**, 1955–1975.

Seitz, J., Hueck, M., Dahmen, B., Schulte-Rüther, M., Legenbauer, T., Herpertz-Dahlmann, B., & Konrad, K. (2016) Attention network dysfunction in bulimia nervosa - An fMRI study. *PLoS ONE*, **11**, 1–18.

Serdula, M.K., Ivery, D., Coates, R.J., Freedman, D.S., Williamson, D.F., & Byers, T. (1993) Do Obese Children Become Obese Adults? A Review of the Literature. *Prev. Med.*, **22**, 167–177.

Sevinçer, G.M., Konuk, N., Bozkurt, S., & Coşkun, H. (2016) Food addiction and the outcome of bariatric surgery at 1-year: Prospective observational study. *Psychiatry Res.*, **244**, 159–164.

Sharma, L., Kohl, K., Morgan, T.A., & Clark, L.A. (2013) "Impulsivity": Relations between self-report and behavior. *J. Pers. Soc. Psychol.*, **104**, 559–575.

Sharma, L., Markon, K.E., & Clark, L.A. (2014) Toward a theory of distinct types of "impulsive" behaviors: A meta-analysis of self-report and behavioral measures. *Psychol. Bull.*, **140**, 374–408.

Spearman, C. (1904) "General Intelligence," Objectively Determined and Measured. *Am. J. Psychol.*, **15**, 201–292.

Spearman, C. (1927) *The Abilities Of Man*. Macmillan And Company., Limited.

Steinle, N.I., Hsueh, W.-C., Snitker, S., Pollin, T.I., Sakul, H., St Jean, P.L., Bell, C.J., Mitchell, B.D., & Shuldiner, A.R. (2002) Eating behavior in the Old Order Amish: heritability analysis and a genome-wide linkage analysis. *Am. J. Clin. Nutr.*, **75**, 1098–1106.

Stevenson, R.J. (2017) Psychological correlates of habitual diet in healthy adults. *Psychol. Bull.*, **143**, 53–90.

Stevenson, R.J. & Francis, H.M. (2017) The hippocampus and the regulation of human food intake **143**, 1011–1032.

Stice, E., Spoor, S., Bohon, C., & Small, D.M. (2008) Blunted striatal response to food is moderated by TaqIA A1 Allele. *Science*, **322**, 449–452.

Stice, E. & Yokum, S. (2018) Relation of neural response to palatable food tastes and images to future weight gain: Using bootstrap sampling to examine replicability of neuroimaging findings. *NeuroImage*, **183**, 522–531.

Stubbs, R.J., O'Reilly, L.M., Whybrow, S., Fuller, Z., Johnstone, A.M., Livingstone, M.B.E., Ritz, P., & Horgan, G.W. (2014) Measuring the difference between actual and reported food intakes in the context of energy balance under laboratory conditions. *Br. J. Nutr.*, **111**, 2032–2043.

Sutin, A.R., Boutelle, K., Czajkowski, S.M., Epel, E.S., Green, P.A., Hunter, C.M., Rice, E.L., Williams, D.M., Young-Hyman, D., & Rothman, A.J. (2018) Accumulating Data to Optimally Predict Obesity Treatment (ADOPT) Core Measures: Psychosocial Domain. *Obesity*, **26**, S45–S54.

Sweeney, P. & Yang, Y. (2017) Neural circuit mechanisms underlying emotional regulation of homeostatic feeding. *Trends Endocrinol. Metab.*, **28**, 437–448.

Tang, D.W., Fellows, L.K., Small, D.M., & Dagher, A. (2012) Food and drug cues activate similar brain regions: a meta-analysis of functional MRI studies. *Physiol. Behav.*, **106**, 317–324.

Teixeira, P.J., Silva, M.N., Coutinho, S.R., Palmeira, A.L., Mata, J., Vieira, P.N., Carraça, E. V., Santos, T.C., & Sardinha, L.B. (2010) Mediators of weight loss and weight loss maintenance in middle-aged women. *Obesity*, **18**, 725–735.

Tholin, S., Rasmussen, F., Tynelius, P., & Karlsson, J. (2005) Genetic and environmental influences on eating behavior: the Swedish Young Male Twins Study. *Am. J. Clin. Nutr.*, **81**, 564–569.

Thomas, D.L. & Diener, E. (1990) Memory accuracy in the recall of emotions. *J. Pers. Soc. Psychol.*, **59**, 291–297.

Tsukayama, E., Duckworth, A.L., & Kim, B. (2012) Resisting everything except temptation: Evidence and an explanation for domain-specific impulsivity. *Eur. J. Personal.*, **26**, 318–334.

Turkheimer, E. (2000) Three Laws of Behavior Genetics and What They Mean. *Curr. Dir. Psychol. Sci.*, **9**, 160–164.

Turkheimer, E. & Harden, K.P. (2014) Behavior genetic research methods: Testing quasi-causal hypotheses using multivariate twin data. In *Handbook of Research Methods in Social and Personality Psychology, 2nd Ed*. Cambridge University Press, New York, NY, US, pp. 159–187.

Vainik, U., Baker, T.E., Dadar, M., Zeighami, Y., Michaud, A., Zhang, Y., Alanis, J.C.G., Misic, B., Collins, D.L., & Dagher, A. (2018) Neurobehavioral correlates of obesity are largely heritable. *Proc. Natl. Acad. Sci.*, **115**, 9312–9317.

Vainik, U., Dagher, A., Dubé, L., Fellows, L.K., Dube, L., & Fellows, L.K. (2013) Neurobehavioural correlates of body mass index and eating behaviours in adults: A systematic review. *Neurosci. Biobehav. Rev.*, **37**, 279–299.

Vainik, U., Han, J.E., Epel, E., Tomiyama, A.J., Dagher, A., & Mason, A.E. (In press) Rapid Assessment of Reward-Related Eating: The RED-X5. *Obesity*,.

Vainik, U. & Meule, A. (2018) Jangle fallacy epidemic in obesity research: a comment on Ruddock et al. (2017). *Int. J. Obes.*, **42**, 585–586.

Vainik, U., Mõttus, R., Allik, J., Esko, T., & Realo, A. (2015) Are Trait–Outcome Associations Caused by Scales or Particular Items? Example Analysis of Personality Facets and BMI. *Eur. J. Personal.*, **29**, 688–634.

Vainik, U., Neseliler, S., Konstabel, K., Fellows, L.K., & Dagher, A. (2015) Eating traits questionnaires as a continuum of a single concept. Uncontrolled eating. *Appetite*, **90**, 229–239.

Val-Laillet, D., Aarts, E., Weber, B., Ferrari, M., Quaresima, V., Stoeckel, L.E., Alonso-Alonso, M., Audette, M., Malbert, C.H., & Stice, E. (2015) Neuroimaging and neuromodulation approaches to study eating behavior and prevent and treat eating disorders and obesity. *NeuroImage Clin.*, **8**, 1–31.

van Bork, R., Epskamp, S., Rhemtulla, M., Borsboom, D., & van der Maas, H.L.J. (2017) What is the p-factor of psychopathology? Some risks of general factor modeling. *Theory Psychol.*, **27**, 759–773.

Van Der Maas, H.L.J., Dolan, C.V., Grasman, R.P.P.P., Wicherts, J.M., Huizenga, H.M., & Raijmakers, M.E.J. (2006) A dynamical model of general intelligence: The positive manifold of intelligence by mutualism. *Psychol. Rev.*, **113**, 842–861.

Van Strien, T., Cebolla, A., Etchemendy, E., Gutiérrez-Maldonado, J., Ferrer-García, M., Botella, C., & Baños, R. (2013) Emotional eating and food intake after sadness and joy. *Appetite*, **66**, 20–25.

van Strien, T., Herman, C.P., & Anschutz, D. (2012) The predictive validity of the DEBQ-external eating scale for eating in response to food commercials while watching television. *Int. J. Eat. Disord.*, **45**, 257–262.

Volkow, N.D., Wang, G.-J., Tomasi, D., & Baler, R.D. (2013) Obesity and addiction: neurobiological overlaps. *Obes. Rev. Off. J. Int. Assoc. Study Obes.*, **14**, 2–18.

Volkow, N.D., Wise, R.A., & Baler, R. (2017) The dopamine motive system: implications for drug and food addiction. *Nat. Rev. Neurosci.*, **18**, 741–752.

Vuilleumier, P. (2005) How brains beware: neural mechanisms of emotional attention. *Trends Cogn. Sci.*, **9**, 585–594.

Vukasović, T. & Bratko, D. (2015) Heritability of personality: A meta-analysis of behavior genetic studies. *Psychol. Bull.*, **141**, 769–785.

Wang, G.J., Volkow, N.D., Logan, J., Pappas, N.R., Wong, C.T., Zhu, W., Netusil, N., & Fowler, J.S.S. (2001) Brain dopamine and obesity. *The Lancet*, **357**, 354–357.

Watabe-Uchida, M., Zhu, L., Ogawa, S.K., Vamanrao, A., & Uchida, N. (2012) Whole-brain mapping of direct inputs to midbrain dopamine neurons. *Neuron*, **74**, 858–873.

Westfall, J. & Yarkoni, T. (2016) Statistically Controlling for Confounding Constructs Is Harder than You Think. *PLOS ONE*, **11**, e0152719.

Whiteside, S.P. & Lynam, D.R. (2001) The Five Factor Model and impulsivity: Using a structural model of personality to understand impulsivity. *Personal. Individ. Differ.*, **30**, 669–689.

Whiteside, S.P., Lynam, D.R., Miller, J.D., & Reynolds, S.K. (2005) Validation of the UPPS impulsive behaviour scale: A four-factor model of impulsivity. *Eur. J. Personal.*, **19**, 559–574.

Wilson, G.T., Grilo, C.M., & Vitousek, K.M. (2007) Psychological treatment of eating disorders. *Am. Psychol.*, **62**, 199–216.

Wing, R.R. & Phelan, S. (2005) Long-term weight loss maintenance. *Am. J. Clin. Nutr.*, **82**, 222S-225S.

Witt, A.A., Raggio, G.A., Butryn, M.L., & Lowe, M.R. (2013) Do hunger and exposure to food affect scores on a measure of hedonic hunger? An experimental study. *Appetite*,.

Wood, S.M.W., Schembre, S.M., He, Q., Engelmann, J.M., Ames, S.L., & Bechara, A. (2016) Emotional eating and routine restraint scores are associated with activity in brain regions involved in urge and self-control. *Physiol. Behav.*, **165**, 405–412.

Yang, Y., Shields, G.S., Guo, C., & Liu, Y. (2018) Executive function performance in obesity and overweight individuals: A meta-analysis and review. *Neurosci. Biobehav. Rev.*, **84**, 225–244.

Yarkoni, T., Poldrack, R.A., Nichols, T.E., Van Essen, D.C., & Wager, T.D. (2011) Large-scale automated synthesis of human functional neuroimaging data. *Nat. Methods*, **8**, 665–670.

Yeomans, M.R. & Brace, A. (2015) Cued to Act on Impulse: More Impulsive Choice and Risky Decision Making by Women Susceptible to Overeating after Exposure to Food Stimuli. *PLOS ONE*, **10**, e0137626.

Yeomans, M.R., Leitch, M., & Mobini, S. (2008) Impulsivity is associated with the disinhibition but not restraint factor from the Three Factor Eating Questionnaire. *Appetite*, **50**, 469–476.