Appetite, Satiety, and Food Reward in Obese Individuals: A Behavioral Phenotype Approach

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Abstract Why do some people remain lean while others are susceptible to obesity, and why do obese individuals vary in their successes in losing weight? Despite physiological processes that promote satiety and satiation, some individuals are more susceptible to overeating. While the phenomena of susceptibility to weight gain, resistance to treatment or weight loss, and individual variability are not novel, they have yet to be exploited and systematically examined to better understand how to characterise phenotypes of obesity. The identification and characterisation of distinct phenotypes not only highlight the heterogeneous nature of obesity but may also help to inform the development of more tailored strategies for the treatment and prevention of obesity. This review examines the evidence for different susceptible phenotypes of obesity that are characterised by risk factors associated with the hedonic and homeostatic systems of appetite control.

Keywords Overweight · Obesity · Susceptibility · Resistance · Phenotype food reward · Appetite · Satiety · Appetite control · Exercise response · Individual variability · Binge eating · Energy intake · Body composition

Abbreviations

BED Binge eating disorder
BES Binge Eating Scale
DLPFC Dorsolateral prefrontal cortex

HF High-fat
PET Positron emission tomography
PFC Prefrontal cortex
RMR Resting metabolic rate
SQ Satiety quotient
TFEQ Three-factor eating questionnaire

Introduction

Appetite and eating behaviour can be understood as processes of providing energy and nutrients for maintenance and growth of bodily tissues. In other words, they are partly the expression of a physiological system to meet a biological purpose. However, as with other behaviours, eating is volitional. Individuals will vary in their responses to the same stimuli, environmental cues, or signals of hunger and satiety. According to energy homeostasis models, energy need arising from the metabolism of active tissues is thought to generate a motivational drive to acquire energy from food [1]. Once food is consumed, the motivation to eat is inhibited by the processes of satiation and satiety, which involve episodic feedback from hormonal signals in the gastrointestinal tract and tonic feedback from other hormones, such as leptin and insulin, secreted in proportion to fat mass [2]. Thus, it is proposed that the net effect of these stimulatory and inhibitory signals has a determining influence on eating behaviours like meal size and meal frequency [3].

An important extension to the homeostatic model is the recognition of the role of cognitive and hedonic inputs in the regulation of food intake [4]. It is now accepted that hedonic thoughts about food and the sensory appreciation of nutrients like salt, sugar and fat are important features of the normal homeostatic response to acute and chronic energy need (Dalton and Finlayson 2013). These hedonic inputs help to account for

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eating behaviours other than meal size and frequency, such as food preference and choice. However, the modern ‘obesogenic’ environment, where highly palatable, often energy-dense foods are ubiquitous, raises the importance of cognitive and hedonic influences on the control of food intake that occur independently from, and sometimes in opposition to, an individual’s energy need or weight status. Hedonic eating behaviours can arise from opportunities for entertainment, social discourse, or relief from stress or negative mood states. Therefore, the primary act of eating for nutrition also has secondary physiological functions. Both hedonic and homeostatic processes influence the regulation and potential dysregulation of appetite control.

In this way, overconsumption in obesity may be due to either some defect in homeostatic signalling that fails to inhibit the motivation to eat, or could be due to excessive or inappropriate response to the hedonic aspects of food [5]. Furthermore, when considering the human eating response, there will rarely be one uniform response from all individuals. Instead, the response will be variable and reflect to some degree the biological variability that occurs between individuals and with individual adaptation over time. Although there appears to be no unique pattern of eating that will invariably lead to an excess of intake, there is no doubt that that, in essence, intake exceeds expenditure. Some characteristics of the expression of appetite make individuals more vulnerable to overconsumption of food—these characteristics can be regarded as risk factors. Moreover, when considering the impact of the obesogenic environment on weight gain, there is enormous variability. Some individuals gain weight whilst others remain lean. These can be termed susceptible and resistant phenotypes, respectively [6]. This typology is relevant to most environmental triggers of eating behaviour. Therefore, we should not expect forms of eating behaviour that create susceptibility to overconsumption to universally apply to everyone. The factors that connect eating behaviour to obesity cannot be uncovered from the average response of a population, as the information yielded from this approach is often restricted when the heterogeneity of obesity is ignored. Alternatively, the examination of susceptible and resistant phenotypes within the obese population, by way of an individual differences approach, offers possibilities for a deeper understanding of overeating and how it can be controlled.

The aims of this review are to present some current opinions and provide an overview of the pertinent literature on the operations and underlying mechanisms of homeostatic (satiation, satiety) and hedonic (food reward: liking, wanting) processes on eating behaviour in obese phenotypes. The review illustrates how a phenotypic approach can improve our understanding of appetite control in all individuals that fall along the spectrum of resistance or susceptibility to obesity through overconsumption.

Susceptibility in an Obesogenic Environment

Even in cases where there is a strong genetic predisposition, the corresponding phenotype will only become fully expressed given an appropriate environment. It therefore follows that common phenotypes of obesity require an obesogenic environment. As the majority of the population carries at least some genes that evolved to protect against food scarcity and starvation, this means that in an obesogenic environment, the full spectrum of susceptibility within a population will be expressed [7]. Along this spectrum, it is possible to identify clusters of individuals at the extremes of susceptibility and resistance, defined according to particular markers. Characterising the ways in which these phenotypes differ can shed light on the particular biological and behavioural features that encourage overconsumption and weight gain.

Obesity is a heterogeneous disease, and there is a need to differentiate between groups of obese individuals by assessing what risk factors predisposes them to becoming obese—and in some instances, what characteristics prevent them from losing weight. How is it possible to detect a susceptible phenotype? By definition, a susceptible individual is gaining weight or has become obese. It is more difficult to detect an individual in the process of weight gain than it is to identify someone who has already attained a BMI over 30 [6]. However, the stratification of BMI that is commonly recognised (underweight <18.5 kg/m²; normal 18.5–24.9 kg/m²; overweight 25–29.9 kg/m²; obese class I >30 kg/m²; obese class II >35 kg/m²; obese class III >40 kg/m²) should not be regarded as definitive, as this classification has no grounding in aetiology and contributes little to our understanding of causation. Therefore, it is important to note that a susceptible phenotype can exist at any BMI, and susceptibility reflects the capacity of a person to persistently gain weight even after accumulating a significant amount of body fat. A useful first step in characterising susceptibility would be to study within the obese population.

The Role of Hunger, Satiation and Satiety in Obese Phenotypes

In the control of appetite, motivated behaviour can take the form of an increase in drive (hunger) in response to signals of need (e.g., metabolic turnover, or an empty gut), and an active inhibition of eating (satiation) and the maintenance of inhibition (satiety) in response to signals of repletion. The cyclical nature of this motivation to eat and subsequent food intake is conceptualised by the satiety cascade (see Fig. 1) [8], which depicts satiety as a time-dependent process: the sensation of hunger tends to oscillate before the onset of a meal and is acutely suppressed by the consumption of food; then, when the meal has ended, hunger remains low until the passage of

Fig. 1 The satiety cascade describes the various processes involved in the expression of appetite. These include the psychological events and behavioural operations, peripheral physiology and metabolic events, and neurotransmitters and metabolic interactions in the brain [8]

time and onset of the next meal. The satiety cascade demonstrates the distinction between satiation and satiety, and how episodic signals (from sensory, cognitive, post-ingestive, and post-absorptive processes) as well as tonic changes in energy balance can influence the size and frequency of meals. In a tightly controlled situation, the operation of this negative feedback system can be demonstrated in lean and obese individuals [9]. However, there are many examples of the continued urge to eat in the presence of inhibitory satiety signals, and of eating being initiated in the absence of a (hunger) drive. Therefore, although the appetite control system displays regulatory properties, the mediating processes can be readily over-ridden. Susceptibility to weight gain and obesity may therefore involve a pattern of eating that operates with a particularly strong motivational drive, or a weak inhibition from homeostatic signals that permit the onset of eating. These attributes could be expressed through enduring traits—reflecting biologically based predispositions—and through episodic states (e.g., hunger sensations, food cravings).

Fat-Free Mass, Resting Metabolic Rate and the Hunger Drive in Obesity

The idea of energy expenditure as a metabolic driver of eating is a longstanding concept in the study of energy balance and

appetite control. Total daily energy expenditure as a determinant of energy intake, for example, has previously been proposed by Edholm [10]. However, due to the large daily fluctuation in physical activity levels, Edholm (1955) [10] found no within-day association between energy expenditure and energy intake. However, this proposition has recently been reconsidered with the suggestion that resting metabolic rate (RMR)—the largest and most relatively stable component of daily energy expenditure—may be a functionally relevant biological signal for energy need, and may therefore act as a regulator of appetite control and food intake [11, 12••]. Blundell and colleagues used a novel energy balance framework with controlled measures of body composition, resting metabolism, objectively measured within-day food intake, and appetite sensations to study this proposition in a sample of overweight and obese individuals [11, 13]. They found that fat-free mass—the largest contributor to resting metabolic rate—was closely associated with self-determined test meal intake, and overall daily energy intake, while BMI or fat mass were not [11]. Subsequent studies further demonstrated that RMR was a strong predictor of daily food intake and that individuals with the highest levels of RMR (independent of age and gender) also reported the highest levels of hunger in the periods between laboratory test meals [14].

A relationship between fat-free mass/RMR and the motivation to eat has implications for understanding appetite

control in obese individuals. For example, most theories do not explain why obese individuals, who carry large amounts of stored energy, should experience strong feelings of hunger and an increased drive to eat. The recognition that obese individuals possess not only a large amount of adipose tissue, but also increased fat-free mass, with a proportionately raised RMR, is in keeping with an increased drive to eat. Moreover, this drive to eat is quite compatible with, and independent of, the accumulation of fat stores and development of leptin resistance, as fat mass makes a relatively small contribution to RMR in comparison to fat-free mass. The independent effects of fat and fat-free mass on ad libitum food intake have since been confirmed in other studies involving obese adults [15].

Satiety Quotient and the Low Satiety Phenotype

Impaired appetite control in obese individuals may also be attributed to a weakened satiety response to food. Indeed, some obese individuals report that their own eating patterns bear no relation to feelings of hunger or fullness, suggesting an altered or weakened recognition and response to these internal sensations [16•]. Barkeling et al. [17] conducted an investigation of appetite sensations and psychometric eating behaviour scores in obese patients, who either reported a ‘good’ relationship between their appetite and eating (i.e., start eating when hungry; stop eating when full) or ‘no relationship’ between appetite and eating (i.e., never hungry before meals; never full after meals). The authors found no difference in sensations of desire to eat, hunger, or fullness in relation to fixed-energy test meals consumed in the laboratory. However, those who reported no relationship between appetite and eating showed a relatively weaker suppression of prospective consumption (i.e., how much additional food could be eaten) immediately after the meals.

Building on these observations, Drapeau and colleagues have examined the prevalence and psychobiological characteristics of the ‘low-satiety phenotype’ in lean and obese individuals [18]. Using a standardised methodology, the authors calculated an individual’s ‘Satiety Quotient’ (SQ) from the change in recorded appetite sensations in response to a fixed portion of food. Therefore, individuals with a ‘low-satiety phenotype’ (SQ: <8 mm/100 kcal) were those who showed an impaired capacity to detect appetite sensations, and reduced intensity and duration of post-ingestive activity after consuming a standard food. Drapeau and colleagues report that approximately 10 % of obese patients referred for weight loss treatment are characterised by the low-satiety phenotype [19]. In a detailed behavioural and metabolic examination of obese individuals with or without this phenotype, a low SQ was associated with increased state anxiety, night eating symptoms, and external locus of hunger. There were no

differences in fasting levels of insulin, leptin, or total ghrelin, but the low-satiety phenotype did show a blunted cortisol response to a standard test meal, suggesting some level of HPA axis activity dysregulation [16•]. These results show that a weak-satiety response to a meal can be objectively measured and is observed in some, but not all, obese individuals.

Susceptibility and Resistance to a High-Fat Diet

Using the appropriate methods, dietary fat intake is a characteristic that can be used to differentiate between habitual high-fat and low-fat consumers [20–22]. Not surprisingly, given the energy density of fat, habitual high-fat consumers (termed HF-phenotypes) are more likely to be overweight or obese, but a large degree of variability in the distribution of BMI in this phenotype has been observed [22]. Therefore, while a diet high in fat can be linked to obesity, some of these individuals appear to be resistant to fat-induced hyperphagia. Characterisation of these phenotypes has revealed that HF-phenotypes report higher baseline hunger levels, and quicker recovery of hunger following a meal compared to LF-phenotypes. Further to this, when provided with ad libitum access to either high-fat or high-carbohydrate foods, HF-phenotypes exhibited a greater preference for high-fat foods compared to LF-phenotypes [20]. Furthermore, it has been shown that susceptible individuals are distinguishable from resistant individuals across a number of characteristics. Firstly, susceptible individuals showed a weaker suppression of hunger following the consumption of high-fat foods, an effect that was not observed following the consumption of low-fat foods, or in resistant individuals. Secondly, susceptible individuals retained a strong hedonic response to high-fat foods following satiation compared to resistant individuals, who exhibited a preference for low-fat foods. Thirdly, susceptible individuals scored higher on the trait Disinhibition and trait Hunger factors of the Three-Factor Eating Questionnaire (TFEQ; [23]), which suggested that they might have been more prone to opportunistic eating compared to resistant individuals. Finally, susceptible individuals reported eating more in response to a negative mood, whereas the resistant individuals reported eating less [24]. These findings suggest that susceptibility to weight gain in habitual high-fat consumers is characterised by a broad range of homeostatic and hedonic risk factors that contribute to poor appetite control and overconsumption.

Compensatory Responses to Exercise: Individual Variability

Anecdotal and empirical evidence shows that people vary in their response to exercise interventions. Similar to energy restriction, it is intuitive that the body might offset exercise-

induced increases in energy expenditure with an orexigenic drive to eat. Of course, there are several compensatory responses (e.g., resting metabolic rate (RMR), non-exercise activity thermogenesis) that could potentially explain the individual variability in weight loss [25]. However, given the relative potent caloric contribution of energy intake to energy balance, it could be argued that appetite and energy intake responses are more important. Therefore, susceptibility to resisting weight loss and to re-gaining weight after exercise-induced weight loss could be explained by a predisposition to an orexigenic drive that compensates for an imposed energy deficit.

The phenomena of individual variability, responders/non-responders, and weight gainers/maintainers are not new [26–28]. However, they have yet to be exploited, especially for models that could characterise susceptibility to weight gain and resistance to weight loss. For example, when exercise is supervised and calibrated to the individual, there is a large individual variability in weight loss [29]. Obese individuals participating in a supervised 12-week program of exercise experienced an average weight loss of 3.3 kg. However, reporting the mean weight loss disguised the large range of weight loss that occurred (–14.0 kg to +2.0 kg). Individuals who were ‘resistant’ to the theoretical weight loss (non-responders) were characterised by experiencing an orexigenic drive reflected in higher levels of fasting hunger sensations [30]. Evidence also indicated that the susceptibility to compensate for an exercise-induced increase in energy expenditure was associated with particular eating behaviour traits. Using the same data from King et al.’s 2007 12-week intervention study, those individuals who experienced a decrease in TFEQ trait Disinhibition and an increase in trait Dietary Restraint showed higher levels of weight loss [31]. Collectively, this evidence supports the concept that some people are less responsive to exercise-induced weight loss due to a susceptibility to safeguarding the energy deficit via increasing energy intake. The elevated motivation to eat to compensate for the exercise-induced energy expenditure could be physiologically or psychologically driven, or a combination of both. Physiological drivers include substrate oxidation [32], and appetite peptides (e.g., [33]). However, these physiological drivers need to be manifested in behaviour for any changes in energy intake to occur. The decision to act out the behaviour is partly volitional and will depend on factors such as individual goals and dietary restraint. The prefrontal cortex (PFC), or the limbic region, is responsible for decision-making and executive control and is influenced by several regions in relation to food intake [34–36]. The OFC and dorsolateral prefrontal cortex (DLPFC) are activated by food intake receipt in relation to expectation of reward and behavioural control. This activation is positively associated with dietary restraint [35] and decreases with satiation [37]. Physiological changes induced by acute exercise and any significant accumulated increase in

energy expenditure from chronic exercise could interact with these processes. Elevated restraint scores may be indicative of individuals with over-responsive reward circuitry and a desire to overcome this [38]. Furthermore, the OFC and the amygdala jointly evaluate hedonic value of food. However, the OFC is responsible for generating the incentive for action in both instances and is a reliable neurological measure of appetite and food reward [37, 39, 40]. Achieving energy balance requires an extensive range of neural regions, recruited in the promotion of eating to meet perceived energy needs [41]. Indeed, acute exercise has been shown to attenuate the neural response to food imagery [36, 42]. While the physiological susceptibility to compensate in response to exercise has been explored (e.g. [43]), the phenomenon of reward-related responses has not. Some people could be more resistant to exercise-induced weight loss because they perceive food as a reward for exercise, and/or underestimate the energy value of food relative to the energy cost of exercise.

The Role of Food Reward in Obese Phenotypes

The psychological components of food reward have been proposed to operate at implicit (automatic, unconscious) and explicit (subjective, conscious) levels [44]. The experience of reward typically involves a combination of liking and wanting, however obesity and susceptibility to reward-driven overeating may be characterised by instances where the processes of food reward become enhanced, attenuated or even dissociated, and thus contribute to certain forms of overeating and eating pathology [45]. This has led to the emergence of seemingly incompatible theories with regards to the role of reward as a risk factor for overconsumption, weight gain and the development of obesity. The first, termed the Reward Surfeit Model, proposes that obese individuals experience a greater level of reward from food as a result of a hyper-functioning reward system [46–48], while the second, termed the Reward Deficit Model, proposes that obese individuals have an innate, low-reward response and therefore increase their use of rewarding substances in order to obtain an ‘optimal’ level of reward [49–51].

The reward deficit model posits that rewarding substances (like addictive drugs and palatable foods), which increase the level of dopamine in the brain, are used as a form of self-medication to boost relatively low levels of dopamine [49–51]. Consistent with this model, Wang et al. [50], using positron emission tomography (PET), demonstrated that a small group of extremely obese individuals had reduced striatal dopamine D2 receptor binding compared to lean individuals, with the lowest binding observed in those with the highest BMI. More recently, however, research regarding dopamine availability and obesity has been less consistent, with some research supporting this initial finding [52] and

some not [53]. The discrepancies in findings may, in part, be attributable to the differences in the severity of obesity in the samples studied. In two fMRI studies, Stice and colleagues have demonstrated that compared to their lean counterparts, obese adolescents showed less activation in the dorsal striatum in response to the consumption of a palatable milkshake versus a tasteless control solution [54, 55]; however, more recently this finding was not replicated [56]. Felsted et al. [57] examined whether the association between striatal response and the consumption of a palatable milkshake was moderated by the presence of the Taq1A A1 allele, which has previously been associated with a 30–40 % reduction in the number of dopamine D2 receptors in the striatum, and with weaker dopamine signalling [58–60]. They demonstrated that decreased activation in response to the consumption of a palatable milkshake was only evident in individuals with at least one copy of the Taq1A A1 allele.

The reward surfeit model posits that hyper-sensitivity to rewarding substances, specifically with regards to the current review, to the rewarding aspects of food, and to their associated cues in the environment, constitutes a risk factor for overeating due to the increased motivation to approach and consume palatable foods [46–48]. Research consistent with this model has shown that obese individuals exhibit enhanced activation in brain regions associated with reward in response to images of palatable foods compared to lean controls, with greater activation observed in the amygdala, striatum, insula and orbitofrontal cortex [61–64]. Further to this, Felsted et al. (2010) demonstrated that increased activation to food images in overweight and obese individuals may be moderated by the Taq1A A2 allele, as individuals with the A2/A2 genotype had greater activation in the orbitofrontal cortex and the midbrain in response to consuming a palatable milkshake [57]. In another study, Yokum et al. [65•] demonstrated that greater activation in the orbitofrontal cortex during the orientation of attention to palatable food images was associated with a significant increase in BMI at 1-year follow-up in a sample of adolescent females. Behavioural evidence also suggests that obese individuals are hyper-responsive to reward-related cues.

In an attempt to resolve the seemingly opposing models, some authors have suggested that a hypo-functioning reward system may be a consequence rather than a cause of obesity, in which the dopamine receptors have been downregulated in response to excessive activation by the increased consumption of palatable foods [66–68]. In support of this suggestion, an fMRI study demonstrated that females who gained weight over a period of 6 months showed a marked decline in striatal response to the consumption of palatable food compared to baseline, and compared to females who had remained weight-stable [69]. Furthermore, Burger and Stice (2012) demonstrated that adolescents who reported frequent consumption of ice cream exhibited specific, attenuated reward-region activation in response to the consumption of that food [70].

The Dynamic Vulnerability Model of obesity posits that individuals at risk for weight gain are initially hyper-responsive to the rewarding aspects of food, which drives overconsumption [67] and leads to a reduction in striatal dopamine activation during palatable food intake. Concurrent with the emergence of a hyposensitive state in response to the consumption of palatable foods, it is proposed that the regions that encode the motivational value of food become hyper-responsive, leading to increased activation in the anticipation of, but not the consumption of food [67, 71]. Therefore, during the development of obesity, the hedonic value obtained from consuming palatable foods is suggested to decrease, whereas the motivational value of palatable foods (and their associated cues) are suggested to increase [72]. Evidence for this model in humans is still preliminary. In a partial test of the model, Stice et al. (2011) demonstrated that adolescents categorised as being at high-risk for the development of obesity—defined as having two obese parents—showed greater activation in the somatosensory region in response to the consumption of palatable food compared to adolescents categorised as low-risk, while there were no differences in activation in response to a cue that predicted food intake [71]. From these findings, Stice et al. (2011) have predicted that hyper-responsivity to food cues would develop over time if the individuals over-consumed and gained weight [71].

Trait Binge Eating as a Phenotype of Obesity

One of the changes made in the recent publication of the fifth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) was the reassignment of Binge Eating Disorder (BED), from a provisional to a formal eating disorder. In the general population, the prevalence of BED is estimated to be between 0.7 and 3.0 % [73, 74]. However, the emotions, cognitions and behaviours associated with binge eating are estimated to occur in 10–20 % of the general obese population [75, 76], and collectively constitute a trait that can be psychometrically assessed along a continuum and applied to the general population. Research has demonstrated that the trait or disposition to binge eat, assessed using the Binge Eating Scale (BES; [77]), is functional at low to moderate levels in lean [78] and overweight or obese individuals [79, 80]. Therefore, the tendency to binge eat has been proposed as a psychobiological marker for susceptibility to reward-driven overeating that may constitute a risk factor for obesity and weight gain [81, 82, 83]. Consistent with this, Finlayson et al. [84] demonstrated that greater levels of trait binge eating were positively associated with increases in fat mass over a period of 1 year in first-year undergraduate students.

In an fMRI study, Filbey et al. (2012) examined the neural response to a cue that signalled the delivery of a liked high-

calorie drink in a sample of overweight or obese individuals with moderate levels of binge eating severity. They found that exposure to the reward-related cue elicited activation in reward-related regions, including the amygdala, insula and putamen. They also demonstrated that this activation was moderated by the level of binge eating severity, with the greatest activation being observed in individuals with higher binge eating scores [80]. However, the findings from this study should be interpreted with caution due to the lack of a non-binge-eating control group. Dalton et al. (2013) examined the influence of trait binge eating on food reward and energy intake, in age-matched lean and overweight or obese females. It was shown that individuals with high scores on the BES were characterised by an increased intake of, and craving for, high-fat sweet foods and by enhanced wanting for these foods compared to those with low scores. Moreover, high scorers had a greater liking for food overall and greater levels of central adiposity compared to low scorers [79•]. In another study, Dalton, Blundell, and Finlayson (under review) extended these findings to the free-living environment by measuring energy intake over two 24-hour periods; one using laboratory-based test meal methodology and one using 24-hour dietary recall procedures. In line with previous research, they found that obese individuals with high BES scores had greater levels of adiposity, greater liking for food overall, and enhanced desire and cravings for high-fat sweet foods compared to obese individuals with low BES scores. Furthermore, the preference for, and greater intake of, high-fat sweet foods was observed under both laboratory and free-living conditions in high scorers. Therefore, these findings suggest that trait binge eating appears to identify an ecologically valid, behavioural phenotype of obesity that is characterised by reliable psychological and anthropometric characteristics.

Conclusion

Within a population there is a spectrum of vulnerability to weight gain and obesity—with some individuals being more susceptible or resistant to overeating than others. Vulnerability to overeating may be influenced by risk factors in either the homeostatic or hedonic systems of appetite control, or may reflect a combination of both. The research presented in this review highlights the value of examining the heterogeneous nature of obesity, and going beyond the average response to consider important individual differences within the obese population. By doing this, it is possible to gain a deeper understanding of the processes and factors that characterise phenotypes resistant or susceptible to overconsumption. The identification and characterisation of distinct phenotypes may help to inform the development of more tailored strategies for the prevention and treatment of obesity.

Compliance with Ethics Guidelines

Conflict of Interest Michelle Dalton, Graham Finlayson, Emma Esdaile, and Neil King declare that they have no conflict of interest.

Human and Animal Rights and Informed Consent This article does not contain any studies with animal subjects performed by any of the authors. With regard to the authors' research cited in this paper, all procedures were followed in accordance with the ethical standards of the responsible committee on human experimentation and with the Helsinki Declaration of 1975, as revised in 2000 and 2008.

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