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Emotional eating and obesity in adults: the role of depression, sleep and genes

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Stress and other negative emotions, such as depression and anxiety, can lead to both decreased and increased food intake. The term ‘emotional eating’ has been widely used to refer to the latter response: a tendency to eat in response to negative emotions with the chosen foods being primarily energy-dense and palatable ones. Emotional eating can be caused by various mechanisms, such as using eating to cope with negative emotions or confusing internal states of hunger and satiety with physiological changes related to emotions. An increasing number of prospective studies have shown that emotional eating predicts subsequent weight gain in adults. This review discusses particularly three lines of research on emotional eating and obesity in adults. First, studies implying that emotional eating may be one behavioural mechanism linking depression and development of obesity. Secondly, studies highlighting the relevance of night sleep duration by showing that adults with a combination of shorter sleep and higher emotional eating may be especially vulnerable to weight gain. Thirdly, an emerging literature suggesting that genes may influence body weight partly through emotional eating and other eating behaviour dimensions. The review concludes by discussing what kind of implications these three avenues of research offer for obesity prevention and treatment interventions.

Emotions: Eating: Depression: Obesity: Weight gain

Emotions and eating are both inherent and recurring part of our daily lives. Research has also demonstrated that they interact with each other in multiple ways: emotional states influence the quantity and quality of foods eaten, and food intake has affective consequences that may influence subsequent food choices⁽¹⁾. Various physiological, psychological and social processes are likely to be involved in this interaction. Importantly, food intake is considered to be controlled by both homeostatic and hedonic processes with the former concerning the regulation of energy-balance and the latter the influence of pleasure, reward and palatability in food intake⁽²⁾. It might be argued that the interaction between emotions and eating has strengthened in the contemporary

food-rich environments where an increasing proportion of eating is driven by hedonic processes⁽³⁾.

This review focuses mainly on the influence of negative emotions on eating behaviour and weight changes as well as on the underlying psychological processes. Especially, three lines of literature related to emotional eating (i.e. eating triggered by negative emotions) and obesity in adults are discussed: First, studies focusing on the symptoms of depression, emotional eating and development of obesity are summarised; secondly, research on the interaction between sleep duration and emotional eating in affecting weight gain; finally, studies examining emotional eating and other eating behaviour dimensions in the context of the genetic architecture of obesity are reviewed. Before

Abbreviation: TFEQ, Three-Factor Eating Questionnaire.

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discussing these three avenues of research, some key definitions and findings of the negative emotion–eating behaviour literature are briefly overviewed.

The effect of negative emotions on eating behaviour

What are emotions and why do we experience them? Emotions arise when we evaluate a situation as being relevant to a currently active goal⁽⁴⁾. For example, if an individual learns that s/he has been selected to a highly motivating job, s/he is likely to experience positive emotions, such as excitement and joy. The opposite situation of not being selected is likely to lead to experiencing various negative emotions, such as disappointment and sadness. Another important characteristic is that emotions are multifaceted phenomena that involve changes in three domains: subjective experience, behaviour and physiology (e.g. changes in heart rate, blood pressure, respiration, sweating and gastrointestinal motility). It is thus plausible that emotions serve several important functions in our daily life: they direct our attention, influence our decision making and motivate our behaviour among other things. Compared to emotions, moods often last longer and may appear in the absence of obvious stimuli⁽⁴⁾. The term ‘stress’ is also frequently used in the negative emotion–eating behaviour literature. While both stress and emotion involve whole-body responses to significant events, stress usually refers to negative affective responses and emotion to both negative and positive affective states⁽⁵⁾.

During the past decades of research, it has become clear that stress and negative emotions may lead to both decreased and increased food intake and heightened preference for palatable energy-dense foods (for reviews, see^(6,7)). Fig. 1 summarises how the influence of negative emotions on eating depends on at least three factors: the characteristics of emotions, individuals and situations. High-intensity or high-arousal emotions (e.g. fear, tension) tend to suppress eating as they are related to physiological and behavioural responses that reduce appetite and interfere with eating⁽⁶⁾. In contrast, negative emotions with more moderate levels of arousal or intensity may decrease or increase food intake depending on the characteristics of an individual and a situation (e.g. available foods). The term ‘emotional eating’ has been widely used to refer to the latter response: a tendency to eat in response to negative emotions with the chosen foods being primarily energy-dense and highly palatable ones. While the reasons for these individual differences are not fully understood, various mechanisms are likely to be involved, such as those linked to adverse childhood experiences, learning history, chronic stress and the hypothalamic–pituitary–adrenal axis functioning and cortisol secretion⁽⁸⁾.

Various self-report questionnaires have been developed to assess the tendency to emotional eating (for a review, see⁽⁹⁾). One of the most widely used is the thirty-three-item Dutch Eating Behaviour Questionnaire⁽¹⁰⁾ containing scales for emotional eating (thirteen items, e.g. Do you have a desire to eat when you are irritated?), external eating

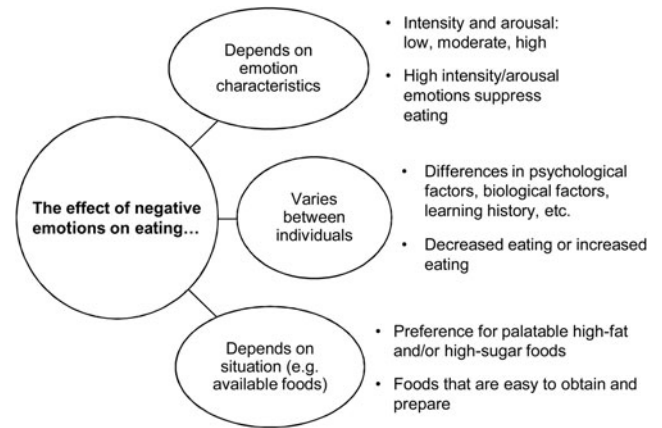


Fig. 1. Overview on how the influence of negative emotions on eating depends on the characteristics of emotions, individuals and situations (e.g. available foods).

(ten items, e.g. If food smells and looks good, do you eat more than usual?) and restrained eating (ten items, e.g. Do you try to eat less at mealtimes than you would like to eat?). The emotional eating scale of the Dutch Eating Behaviour Questionnaire measures respondents’ desire to eat under different negative emotions, including stress, depression and anxiety. The eighteen-item Three-Factor Eating Questionnaire (TFEQ)⁽¹¹⁾ with scales for emotional eating, uncontrolled eating and cognitive restraint has gained popularity in epidemiological research where shorter questionnaires are often preferred. The eighteen-item TFEQ is a revised and shortened version of the original fifty-one-item TFEQ⁽¹²⁾. The emotional eating scale of the eighteen-item TFEQ contains only three items asking respondents to evaluate how negative emotions affect their eating (When I feel anxious, I find myself eating; When I feel blue I often overeat; When I feel lonely, I console myself by eating). Although this review focuses on emotional eating, it is noteworthy that emotional eating often co-occurs with other dimensions of eating behaviour, particularly external eating characterised by overeating in response to food-related cues, such as sight and smell of attractive foods⁽⁸⁾.

Many experimental studies (for a systematic review, see⁽⁷⁾) have shown that individuals, who score high on these emotional eating scales, tend to eat more energy-dense foods after inducement of negative emotions in the laboratory (e.g. via a sad movie) compared to emotionally neutral control situations. However, not all experiments have found this effect and several factors are likely to explain the inconsistency. Some null findings might be explained by the misclassification of participants to ‘emotional eaters’ and ‘no emotional eaters’ due to the use of the median splits and/or insufficient amount of participants with extreme scores⁽¹³⁾. It is also possible that emotional eating follows only specific discrete negative emotions and these triggering emotions vary between individuals⁽¹⁴⁾. Moreover, emotional eating may depend on social context with the laboratory representing a non-natural situation where it is less likely to manifest⁽¹⁴⁾.



Several psychological mechanisms likely underlie emotional eating and a more detailed review on this topic and related theories can be found elsewhere^(6,8). In short, individuals may engage in emotional eating as an attempt to cope with stress and other negative emotions. However, in the long-term, emotional eating is often a maladaptive emotion regulation strategy. Even though experimentally induced negative mood state has found to improve immediately after eating palatable food, the effect tends to be short-term and can be followed by other negative emotions, such as feelings of guilt^(15,16). Emotional eating may also be a result of a poor interoceptive awareness characterised by deficits in sensitivity to bodily sensations. It is possible that individuals with poor interoceptive awareness confuse bodily states related to emotions with physiological internal states guiding satiety and hunger. Finally, in dieters, emotional stimuli may impair their ability to restrict food intake because processing emotions requires cognitive resources.

Emotional eating, food intake and obesity in observational studies

Epidemiological studies utilising different types of FFQ to assess food intake have demonstrated that higher self-reported emotional eating is related to greater intakes of sweet and non-sweet energy-dense foods and snacks, such as sweets and chocolate, cakes and biscuits, sweet and savoury pastries, pizza, hamburgers, French fries and sausages^(17–20). A relatively large (with more than 400 participants) 4-week diary study provided interesting insights by investigating day-to-day within-person effects of daily hassles on food intake⁽²¹⁾. Participants were asked to list each food eaten between meals and to report each stressor or hassle experienced and its intensity at the end of each day. The study found that daily hassles were related to increased consumption of high-fat or sugar snacks, particularly in emotional eaters. As emotional eating is related to personality, correlating particularly with neuroticism characterised by the propensity to experience negative emotions⁽²²⁾, these findings might also partly reflect the effects of underlying personality traits on linking stress and eating behaviour.

Moreover, observations from the UK National Diet and Nutrition Survey suggest that emotional eating relates to a distinct pattern of eating architecture⁽²³⁾. In other words, individuals with higher emotional eating scores on the Dutch Eating Behaviour Questionnaire tended to have smaller meals and more frequent snacking over a longer period every day (with an earlier start and a later finish time) than those scoring lower on emotional eating. These observations are potentially important by suggesting that interventions promoting healthy food intake in emotional eaters might benefit from targeting the number, timing and energetic content of snacks. However, further research is needed to identify what are the most effective behaviour change strategies to encourage such changes in emotional eaters.

In line with the afore-mentioned results, self-reported emotional eating has been associated with higher BMI, waist circumference and body fat percentage in cross-sectional studies^(24–26). Although emotional eating was unrelated to 2-year change in BMI in a French community-based cohort⁽²⁷⁾, other prospective cohort studies conducted in the Netherlands, Finland, France, USA and Korea have shown that adults (both genders or only women) with higher emotional eating at baseline tend to gain more weight over time^(28–33). Taken together, epidemiological evidence rather consistently supports the idea that self-reported emotional eaters consume energy-dense foods more frequently and are at increased risk for developing obesity.

Symptoms of depression and emotional eating

Depression represents one type of negative emotion or mood that may trigger food intake in emotional eaters, and research has shown that individuals with elevated depressive symptoms report higher levels of emotional eating^(25,34,35). However, depression is a heterogeneous syndrome with atypical and melancholic depression increasingly being differentiated in depression studies⁽³⁶⁾. While melancholic depression is typically characterised by loss of appetite and subsequent weight loss, atypical depression is characterised by vegetative symptoms of increased appetite and weight gain. The prevalence of the latter depression subtype is not as low as the name suggests. Epidemiological studies using the Diagnostic and Statistical Manual of Mental Disorders criteria indicated that 15–29% of depressed patients have atypical depression, and the corresponding prevalence rate from the clinical studies was 18–36%⁽³⁷⁾. Emotional eating has been proposed to represent a marker of atypical depression, because it shares with this depression subtype the atypical feature of increased appetite in response to distress. In line with this, Paans *et al.* found in the Netherlands Study of Depression and Anxiety that vegetative symptoms characteristic of atypical depression (increase in appetite, weight gain) had relatively stronger positive associations with emotional eating than other individual depressive symptoms⁽³⁵⁾.

Accumulating evidence from cross-sectional and prospective studies further suggests that emotional eating is one pathway linking depression with weight gain and obesity^(25,30,32,33,38–40). The results from the few prospective studies analysing weight change over time as an outcome are particularly interesting. van Strien *et al.* observed that emotional eating acted as a mediator between depression and 5-year weight gain in Dutch mothers (although not in Dutch fathers)⁽⁴⁰⁾. More specifically, depressive symptoms were related to higher emotional eating and emotional eating predicted a greater increase in BMI independently of depressive symptoms. Depression was also indirectly related to BMI change via emotional eating. Kontinen *et al.* extended these results by showing in a large population-based cohort of Finnish 25–74-year-olds that the effects of depression on a greater increase in BMI and waist

circumference over 7 years were mediated by emotional eating⁽³³⁾.

In addition, it has become clear that the relationship between depression and obesity is bidirectional: besides that depression can lead to subsequent weight gain and development of obesity, it is also possible that obesity leads to later development of depression^(41,42). The findings from a national sample of mid-life US adults with 18-year follow-up suggest that emotional eating plays a role in this bidirectional depression–obesity association⁽³²⁾. Path analyses conducted in women revealed that physical impairment, social dysfunction and emotional eating mediated the development of obesity from depression, and that physical impairment and emotional eating mediated the development of depression from obesity.

Sleep duration and emotional eating

Insufficient sleep has been postulated to be one of the key non-traditional (i.e. not directly related to energy-balance) determinants of excess body weight with different lines of evidence linking short sleep duration and positive energy-balance⁽⁴³⁾. But what is insufficient sleep or short sleep duration? Grandner *et al.* have outlined working definitions for these and related terms⁽⁴⁴⁾. They define sleep insufficiency as a reduction in sleep time of a magnitude associated with negative health outcomes, and point out that many epidemiological studies use the term short sleep to refer to a habitual sleep time of 6 h or less. The hectic pace of contemporary lifestyle in prosperous societies is prone to interfere with sleeping, and the occurrence of insomnia-related symptoms has risen in several countries during the past decades. For example, epidemiological data from Finland demonstrated that the prevalence of working-age adults reporting occasional insomnia-related symptoms increased from 30 to 45% between 1972 and 2012⁽⁴⁵⁾. It is however noteworthy that findings on secular trends in adult sleep duration have been more inconsistent⁽⁴⁶⁾.

In general, the present literature points to the importance of studying whether short sleep duration and poor sleep quality interact with emotional eating in affecting weight gain. First, it has become clear that insufficient sleep and stress are closely intertwined: the former can be both a marker of perceived stress and a source of stress itself⁽⁴⁶⁾. Secondly, evidence is emerging that insufficient sleep interferes with emotion regulation via a number of neurobiological, behavioural and cognitive processes⁽⁴⁷⁾. For example, insufficient sleep produces more negative and less positive emotions, and can affect the ability to choose an appropriate emotion regulation strategy and implement that strategy successfully. Thirdly, epidemiological studies in adults have consistently associated short sleep duration with higher total energy intake and higher total fat intake⁽⁴⁸⁾. Short sleep may also associate with irregular eating behaviour; short sleepers tend to eat fewer main meals and more frequent, smaller, energy-dense and highly palatable snacks throughout the day (and particularly at night) than longer sleepers.

To date, at least three studies have provided evidence that short sleep duration might strengthen the association between emotional eating and subsequent weight gain. In a sample of Dutch employees, women with a combination of short sleep duration and high emotional eating experienced the greatest increase in BMI over 2 years⁽⁴⁹⁾. Chaput *et al.* assessed a broader construct of disinhibition (i.e. a tendency to overeat in response to food cues or emotional cues) in a sample of French Canadian adults with 6-year follow-up⁽⁵⁰⁾. They found that individuals having both short sleep duration and high disinhibition were more likely to gain weight and increase their waist circumference over time, while short-duration sleepers having low disinhibition were not more likely to increase their adiposity indicators than were average-duration sleepers. Moreover, a similar pattern was observed in relation to energy intake assessed using a 3-day food record. In the large Finnish adult cohort discussed in the previous section, emotional eating predicted higher increase in BMI and waist circumference over 7 years in shorter sleepers (7 h or less), but not in longer sleepers (9 h or more)⁽³³⁾.

It is important to note that short sleep duration represents only a proxy measure for insufficient sleep; future research should therefore explore these associations using a diverse set of sleep measures and in specific population groups, including shift workers. Short sleep is likely to be intertwined with perceived stress in individuals who reduce their sleep time to meet other demands of daily life and in those experiencing sleep problems, but not in those for whom short sleep schedule represents their natural way of functioning⁽⁴⁴⁾. The role of chronotype (i.e. individual's inherent preference for mornings or evenings) in this context also deserves attention because eveningness has shown to correlate positively with both depression and emotional eating⁽⁵¹⁾.

Genetic susceptibility to obesity and emotional eating

An increasing number of studies have investigated emotional eating and other eating behaviour dimensions in the context of the genetic architecture of obesity. Genetic factors have a substantial influence on within-population variation in BMI: in a large pooled study of 140 000 twin pairs aged 20–80 years, the heritability estimates for BMI (i.e. the proportion of total BMI variation explained by genetic variation) varied between 60 and 80% depending on age⁽⁵²⁾. Albeit learning and environmental factors are important determinants of eating behaviours, observations from twin studies imply that also genetic factors influence these behaviours (assessed using the eighteen-item TFEQ or the Dutch Eating Behaviour Questionnaire). In Swedish, Finnish and British adult twins, the size of the heritability estimates for restrained eating (26–63%), uncontrolled eating (45–69%) and emotional eating (9–60%) varied between small and moderate^(53,54). Similarly, a study in Korean adults (twins and their family members) reported heritability estimates of 31% for restrained eating, 25% for emotional eating and 25% for external eating⁽⁵⁵⁾.



The emergence of genome-wide association studies has made it possible to identify common genetic variants that have only a small effect on the trait, such as BMI. Meta-analyses combining the results of genome-wide association studies of European-descent individuals have identified hundreds of SNP associated with higher BMI^(56,57). Although the identified SNP explain only a small amount of total genetic variation in BMI, these findings have opened the opportunity to extend understanding of the mechanisms that mediate the effects of common genetic variants on obesity. Appetite-related eating behaviours (including emotional eating) have postulated to represent one such pathway. This idea is supported by the data that many of the BMI-related SNP are expressed particularly in the hypothalamus and pituitary gland, which have a key role in regulating food intake and energy expenditure⁽⁵⁷⁾.

In two Finnish adult cohorts⁽⁵⁸⁾ and in French and UK adult cohorts⁽⁵⁹⁾, participants with a higher polygenic risk score of obesity had also a slightly higher tendency to emotional and uncontrolled eating. Moreover, the positive effects of the polygenic risk score on BMI and waist circumference were partly mediated by these eating behaviours. Comparable results were obtained in a Canadian study in which perceived hunger, habitual disinhibition and situational disinhibition (although not emotional disinhibition) acted as mediators⁽⁶⁰⁾. Mediation was not formally tested in two US cohorts of older adults, but the effect of the polygenic risk score on BMI weakened slightly after controlling for restrained, uncontrolled and emotional eating⁽⁶¹⁾. In line with these molecular genetic findings, the twin study mentioned earlier found that phenotypic correlations between BMI and emotional eating or uncontrolled eating were largely due to shared genetic factors (which may also include gene–environment interactions)⁽⁵⁴⁾.

Although this cross-sectional research offers tentative evidence that genes influence obesity via appetite-related eating behaviours, important questions remain⁽⁶²⁾. Other processes are likely to be involved; for instance, studies have consistently found that genetic susceptibility to obesity is more pronounced in physically inactive individuals than in active individuals^(63,64). It is also possible that the causal pathways are more complex than those implied by the tested mediation models. While the examined eating behaviours were postulated to affect the development of obesity in these models, reverse causation might operate via different mechanisms, such as body fat gain impairing the control of food intake, and increased body size leading to greater experiences of weight stigma and stress^(2,65).

Conclusions

What kind of practical implications do these three lines of research provide for obesity prevention and treatment programmes? Essentially, they suggest that individuals with a high susceptibility to emotional eating might benefit from interventions that teach emotion regulation skills and distress tolerance strategies and aim to improve

emotional well-being. A recent review concluded that several such approaches have shown promising results in reducing emotional eating and facilitating weight loss, including mindfulness, acceptance and commitment therapy, cognitive behaviour therapy and dialectical behaviour therapy⁽⁹⁾. However, further high-quality studies on this topic are still needed.

The literature also suggests that interventions that improve other lifestyle behaviours, such as sleep and physical activity, might offer a novel avenue to promote healthy food intake patterns in emotional eaters. A pilot study performed in habitually short sleepers indicated that sleep extension was feasible and led to decreased intake of free sugars⁽⁶⁶⁾. While the study did not contain information on emotional eating or other eating behaviour dimensions, it encourages continuing this type of intervention research. Moreover, physical exercise is used as a treatment for depression disorders⁽⁶⁷⁾, and some data exist that exercise can alleviate unhealthy food intake in response to stress⁽⁶⁸⁾.

Perhaps paradoxically, research into the genetic architecture of obesity has reinforced the view that there is an urgent need to modify the present food-rich environment⁽⁶²⁾. Evidence that genetic factors play a role in individual differences in eating behaviours emphasises that people are not equal in terms of their inherent vulnerability to the environment in which palatable energy-dense foods are constantly available, cheap and intensively promoted. In fact, this is a part of a wider and highly topical discussion that a radical transformation of the global food system is urgently needed in order to improve human health and well-being and environmental sustainability on Earth⁽⁶⁹⁾.

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Conflict of Interest

None.

Authorship

The author had sole responsibility for all aspects of preparation of this paper.

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