The psychobiology of comfort eating: implications for neuropharmacological interventions

E. Leigh Gibson

Comfort eating, that is eating induced by negative affect, has been a core theme of explanations for overeating and obesity. Psychobiological explanations and processes underlying comfort eating are examined, as well as its prevalence in clinical and nonclinical populations, to consider who may be susceptible, whether certain foods are comforting, and what the implications for treatment may be. Comfort eating may occur in a substantial minority, particularly in women and the obese. Human and animal theories and models of emotional or stress-induced eating show some convergence, and may incorporate genetic predispositions such as impulsivity and reward sensitivity, associated with dopamine dysregulation underlying incentive salience. Comfort eaters show vulnerability to depression, emotional dysregulation and a need to escape negative affect and rumination. During negative affect, they preferentially consume sweet, fatty, energy-dense food, which may confer protection against stress, evidenced by suppression of the hypothalamic–pituitary–adrenal axis response, although activation of the hypothalamic–pituitary–adrenal axis may itself drive appetite for these palatable foods, and the risk of weight gain is increased. Benefits to mood may be transient, but perhaps sufficient to encourage repeated attempts to prolong mood improvement or distract from negative rumination. Cognitive behavioural treatments may be useful, but reliable drug therapy awaits further pharmacogenomic developments. Behavioural Pharmacology 23:442–460 © 2012 Wolters Kluwer Health | Lippincott Williams & Wilkins.

Keywords: depression, dopamine D2 receptors, emotional eating, glucocorticoid hormones, hypothalamic–pituitary–adrenal axis, mood, obesity, reward sensitivity, stress

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Introduction

This review considers the nature of comfort eating, and describes psychobiological explanations for it, in addition to discussing evidence for who might be susceptible and why, and what sustains comfort eating in those individuals. Although it is recognized that comfort eating may overlap with binge eating, this review is not specifically concerned with binge eating, which requires clear evidence of excessive and frequent intake; rather, comfort eating is considered a phenomenon that may be common in nonclinical populations, and may not necessarily involve episodes of excessive consumption. The relevance of comfort eating to public health is gauged by estimating its prevalence, particularly in nonclinical populations, from data available in the literature. Major theories are discussed that set comfort eating in the context of normal and abnormal eating behaviour and help to understand both how it may arise, including its relation to personality characteristics, as well as the implications for therapy. A key aspect of theories of comfort eating is understanding whether certain foods or food groups may be chosen by comfort eaters and why: theories are examined that attempt to explain why negative affect and stress may elicit eating in individuals with particular traits. The review also addresses whether comfort eating may be harmful to health, such as by increasing the risk of developing obesity or other disordered eating, or by damaging the cardio-vascular system through unhealthy food choices, or, by contrast, whether comfort eating might actually provide some benefits to health and well-being. Evidence for a role for particular neurotransmitter and neurohormonal systems is considered and thus whether neuropharmacological interventions may be feasible or even advisable.

Defining ‘comfort eating’

In the academic literature, the normal understanding of comfort eating is eating to relieve negative emotions or affect (typically depression, anxiety or anger), which is synonymous with ‘emotional eating’ (at least where the definition is restricted to negative emotions), the latter being the more commonly used term in such literature (Ganley, 1989). Another term with similar meaning is ‘stress eating’, that is eating induced by stress; indeed, consideration of ‘who eats more when stressed and why’ is at the heart of understanding comfort eating. Even so, this implies that individuals who eat more when stressed do so because of some relief that eating provides, and clearly this needs to be evidenced. This review will focus on these issues, and the evidence for psychobiological mechanisms.

An implicit assumption of stress-induced eating is that the stress results in the sort of negative affect that can elicit eating, and thus is not severe enough to suppress appetite. Whereas one can operationally define ‘comfort...
eaters’ as those who respond to stress by eating more (at least of some foods), in practice, for ethical reasons, direct evidence for this is mainly restricted to comparative studies, usually in rats (Hagan et al., 2002; Wardle and Gibson, 2002). In human studies, the tendency to ‘comfort eat’, or be an emotional eater, is typically measured by self-report questionnaires: this can be as simple as asking whether one eats more, less or the same when stressed (Oliver and Wardle, 1999), but more commonly is based on scales created from several items referring to eating more when upset, etc. There are several such instruments in common use, such as the Dutch Eating Behaviour Questionnaire (DEBQ; Van Strien et al., 1986), the revised Three Factor Eating Questionnaire using 18-items (derived from an administered 21-items); versions 1 (TFEQ-R18; Karlsson et al., 2000) and 2 (TFEQ-R18v2; Cappelleri et al., 2009), and the Emotional Eating Scale (EES; Arnow et al., 1995), the latter being developed principally for clinical populations, such as binge eaters. Recently, Nolan et al. (2010) validated the Emotional Appetite Questionnaire (EMAQ; Geliebter and Aversa, 2003), which includes assessment of the role of positive emotions and emotional situations; however, eating in response to positive emotions is beyond the definition of comfort eating. It is beyond the scope of this review to discuss these instruments in detail, but aspects of the scales will be considered where important for interpretation of findings.

Although the usual inference is that ‘comfort foods’ are those foods that, when eaten, can somehow provide relief from emotional upset, it should be noted that there is a lay concept of comfort foods that has slightly different connotations: this is the idea that comfort foods are foods highly familiar from one’s childhood, also known as ‘nursery foods’ or perhaps ‘home (or mother’s) cooking’. These foods are often associated with special occasions, and thus encoded in, and capable of recalling, happy memories. The psychological impact of such foods may be more related to security, in the sense of memories of secure attachment (or a longing for it) or at least social bonding, rather than necessarily relieving negative affect. This implies that, for some individuals, comfort eating may be related to facets of their ‘attachment style’, which also has implications for neurohormonal influences (Marazziti and Catena Dell’osso, 2008). Indeed, there is recent evidence that ‘anxious attachment’ is associated with disinhibited eating and BMI (Wilkinson et al., 2010), although an emotional eating component may not be directly involved (Phillips et al., 2012). In fact, familiarity itself might be a facet of foods that is actively sought during anxiety. As this notion of comfort ‘nursery’ or ‘home’ foods is not specifically excluded from definitions of comfort food, we cannot be certain to what extent it contributes to comfort eating. Moreover, Wansink et al. (2003) have pointed out that sex differences in comfort foods, such as men preferring hot, savoury meal-related foods, whereas women preferring sweet snack foods, might reflect differences in culinary upbringing, with women more likely to be the food provider. These and other cultural differences might underlie the observation that comfort eating in men is more likely to be motivated by positive emotions, whereas women invariably comfort eat in response to negative emotions (Dubé et al., 2005).

Prevalence of comfort eating
To gauge the importance of comfort eating, particularly if treatment is to be considered, some estimate of prevalence is required. This section will summarize the indicators of prevalence available in the literature: nonexperimental survey studies are considered with moderate to large sample sizes, primarily of noneating disordered populations, in which adult versions of standardized emotional eating questionnaires have been used (twin studies are reported in the last section). Smaller ‘naturalistic’ studies – typically in student populations – have addressed how eating is affected by stress in nonclinical populations, and show that between one-third and two-thirds of those surveyed report eating less when stressed, whereas about a quarter to a half report eating more when stressed, that is comfort eating; a small minority report no change (reviewed by Gibson, 2006). However, these surveys did not use standardized scales for measuring emotional eating; thus, estimates of prevalence are difficult to compare between study samples. Nevertheless, these findings are in notable contrast to the widely accepted view that the majority of clinical populations of disordered eating patients, obese or otherwise, show some form of emotional or comfort eating or at least affect-driven disturbance of eating (Wardle, 1987; Ganley, 1989; Vögele and Gibson, 2010).

Even for studies using questionnaires to measure emotional eating, it is difficult to determine prevalence reliably, because overall means, and correlations, are typically reported, rather than the use of behaviourally validated cut-off scores or other distribution indicators. Moreover, the distribution of scale scores is likely to vary depending on, for example, whether the sample studied is a mainly normal-weight student population or an older population including a substantial proportion of overweight and obese participants. Nevertheless, there have now been sufficient moderate to large cross-sectional surveys, including some of nationally representative samples, to make a summary of prevalence, estimated from the mean scores of emotional eating questionnaires worthwhile: that is, with quite large normally distributed samples, one can draw conclusions about the distribution of scores on the scale from the means. These studies are summarized in Table 1: to allow means to be compared across different instruments, all means are given as percentage scores (i.e. scale ranges were converted into 0–100, if not already reported as such). It is clear from Table 1 that almost all of the means are less than 50%, many substantially so, except for obese groups (Karlsson et al., 2000) and the bulimic group from Wardle
Table 1 Comparison of mean % emotional eating scores from survey populations

<table>
<thead>
<tr>
<th>Reference (Country)</th>
<th>Design</th>
<th>Emotional eating measure</th>
<th>Sample (N; adult unless specified)</th>
<th>Result (mean % score)</th>
<th>Findings on EE by sex and BMI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Van Strien et al. (1986) (the Netherlands)</td>
<td>Survey, cross-sectional</td>
<td>DEBQ</td>
<td>602 women</td>
<td>26.5</td>
<td>F&gt;M</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>449 men (including 131 overweight/ obese)</td>
<td>18.0</td>
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</tr>
<tr>
<td>Wardle (1987) (UK)</td>
<td>Survey, cross-sectional</td>
<td>DEBQ</td>
<td>102 female students</td>
<td>41.3</td>
<td>F&gt;M</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>86 male students</td>
<td>31.0</td>
<td>Bulimics &gt; overweight &gt; normal weight</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>107 ‘weight watchers’</td>
<td>51.3</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>61 bulimic</td>
<td>70.8</td>
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<td></td>
<td></td>
<td></td>
<td>33 anorectic</td>
<td>31.3</td>
<td></td>
</tr>
<tr>
<td>Wardle et al. (1992) (UK)</td>
<td>Survey, cross-sectional</td>
<td>DEBQ</td>
<td>439 girls (11–18 year olds)</td>
<td>27.8</td>
<td>F&gt;M</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>402 boys (11–18 year olds)</td>
<td>23.5</td>
<td>Unrelated to BMI</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>379 men</td>
<td>32.3</td>
<td>F&gt;M (adult and child)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>290 girls (11–18 year olds)</td>
<td>31.5</td>
<td>BMI positively related in adult F only</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>270 boys (11–18 year olds)</td>
<td>23.3</td>
<td></td>
</tr>
<tr>
<td>Karlsson et al. (2000) (Sweden)</td>
<td>Survey, cross-sectional</td>
<td>TFEQ-R18</td>
<td>2603 obese women</td>
<td>63.0</td>
<td>F&gt;M</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>1774 obese men</td>
<td>_b</td>
<td>Unrelated to BMI</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>236 men (30–67 year olds)</td>
<td>43.0</td>
<td>F&gt;M (adult and child/youth)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>163 girls/young women (14–27 year olds)</td>
<td>46.0</td>
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</tr>
<tr>
<td>De Lauzon et al. (2004) (France)</td>
<td>Survey, cross-section of cohort</td>
<td>TFEQ-R18</td>
<td>284 women (90–67 year olds)</td>
<td>22.0</td>
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<td></td>
<td></td>
<td></td>
<td>1471 men</td>
<td>15.0</td>
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<td></td>
<td></td>
<td></td>
<td>781 girls (11–12 year olds)</td>
<td>15.0</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>710 boys (11–12 year olds)</td>
<td>15.0</td>
<td></td>
</tr>
<tr>
<td>Snoek et al. (2007) (the Netherlands)</td>
<td>Survey, cross-sectional</td>
<td>DEBQ</td>
<td>171 boys/young men (14–27 year olds)</td>
<td>24.3</td>
<td>F&gt;M</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>4581 girls (11–16 year olds)</td>
<td>21.3</td>
<td>BMI negatively related</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>(including 18.1% overweight, 1.7% obese)</td>
<td></td>
<td></td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>4430 boys (11–16 year olds)</td>
<td></td>
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<tr>
<td>O’Connor et al. (2008) (UK)</td>
<td>Survey, cross-sectional</td>
<td>DEBQ</td>
<td>229 women</td>
<td>35.3</td>
<td>Not reported</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>193 men (including 55 obese)</td>
<td>_b</td>
<td></td>
</tr>
<tr>
<td>Elfhag et al. (2008) (Sweden)</td>
<td>Survey, cross-sectional</td>
<td>DEBQ</td>
<td>1795 women</td>
<td>22.5</td>
<td>Not reported</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>1471 men</td>
<td>15.0</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>731 girls (11–12 year olds)</td>
<td>15.0</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>710 boys (11–12 year olds)</td>
<td>15.0</td>
<td></td>
</tr>
<tr>
<td>Cappelleri et al. (2009) (USA and Canada)</td>
<td>Clinical trial (baseline) and web-based survey</td>
<td>TFEQ-R18v2</td>
<td>284 people (1660 obese; 68 nonobese)</td>
<td>46.7</td>
<td>F&gt;M</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>1422 women</td>
<td>30.0</td>
<td>BMI positively related for web survey</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>306 men</td>
<td>34.3</td>
<td></td>
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<td></td>
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<td></td>
<td>Web survey (817 obese; 458 nonobese)</td>
<td>26.7</td>
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<td></td>
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<td></td>
<td>503 women</td>
<td></td>
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<td></td>
<td></td>
<td>722 men</td>
<td></td>
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</tr>
<tr>
<td>Anschutz et al. (2009) (the Netherlands)</td>
<td>Survey, cross-sectional</td>
<td>DEBQ</td>
<td>475 female students (including 28 underweight, 6 obese)</td>
<td>41.5</td>
<td>BMI positively related</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>845 women</td>
<td>31.5</td>
<td>F&gt;M</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>525 men (including 717 normal weight, 625 overweight/obese)</td>
<td>40.3</td>
<td>BMI related positively</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>132 boys (11–15 year olds)</td>
<td>20.0</td>
<td>20.4% F, 16.5% M are above EE cut-off</td>
</tr>
<tr>
<td>Angle et al. (2009) (Finland)</td>
<td>Clinical trial (baseline)</td>
<td>TFEQ-R18</td>
<td>2997 women (17–20 year olds)</td>
<td>22.5</td>
<td>BMI positively related from underweight to obese</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>181 underweight</td>
<td>30.3</td>
<td></td>
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<td></td>
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<td>2074 normal weight</td>
<td>37.6</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>469 overweight</td>
<td>46.6</td>
<td></td>
</tr>
<tr>
<td>Nolan et al. (2010) (USA)</td>
<td>Survey, cross-sectional</td>
<td>DEBQ</td>
<td>171 female students</td>
<td>39.3</td>
<td>EMAQ, but not DEBQ, bonded positively related to BMI</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>59 male students (including 17 obese)</td>
<td>47.0</td>
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<td></td>
<td></td>
<td></td>
<td>39.1</td>
<td></td>
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</tr>
<tr>
<td>Konttinen et al. (2010) (Finland)</td>
<td>Survey, cross-section of cohort</td>
<td>TFEQ-R18</td>
<td>2035 women</td>
<td>38.2</td>
<td>F&gt;M</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>1679 men (including 740 obese)</td>
<td>23.1</td>
<td>BMI positively related</td>
</tr>
</tbody>
</table>

DEBQ, Dutch Eating Behaviour Questionnaire; EE, emotional eating; EMAQ, Emotional Appetite Questionnaire (NE, negative emotions; NS, negative situations); F, female; M, male; TFEQ-R18, revised 18-item version 1 of Three Factor Eating Questionnaire; TFEQ-R18v2, revised 18-item version 2 of Three Factor Eating Questionnaire.

*aMeans not reported separately for men and women.

*bDerived from the distribution of scores for the entire sample (men and women).

*cSignificance of the mean difference between sexes was not reported.

*dFrom the DEBQ manual (Van Strien, 2005, cited in Nguyen-Rodriguez et al., 2009).

(1987). In the majority of studies, it is clear that the central tendency is endorsing that emotional eating is occurring less than ‘sometimes’. However, it is also apparent that the majority of studies find emotional eating to be more likely in women than men: the exception is no sex difference in the American study of adolescent school children (Nguyen-Rodriguez et al., 2009); in general, emotional eating is less likely in adolescents than in adults (Table 1). This might
also explain why there is a lack of relationship (or even an inverse relationship, Snoek et al., 2007) between emotional eating and BMI in these young samples, although a lower range of BMI may also contribute: similarly, the constrained BMI range may underlie there being no relation between emotional eating and BMI among obese Swedes (Karlsson et al., 2000). By contrast, in most of the adult samples, BMI is consistently positively related to emotional eating (Table 1).

Another question that can be addressed from these surveys is whether there is any trend over time for the prevalence of emotional eating. Usefully, there have now been two studies of adult Dutch samples representative of the general population in the Netherlands that measured emotional eating (DEBQ) about 25 years apart (Van Strien et al., 1986, 2009). Intriguingly, for the more recent study published in 2009, average emotional eating was substantially higher (mean = 36%) than in the first study (data gathered in 1983; mean = 23%). Even so, the majority of both populations were low to moderate emotional eaters; yet, the ratings have clearly increased among the recent Dutch sample – as has obesity, in line with national trends. The two other scales of the DEBQ (Restrained and External Eating) also increased, but to lesser extents. Van Strien et al. (2009) speculated that this could reflect a disconnection from normal eating contexts and consequences brought on by the increasing dominance of screen media time among children and adolescents, promoting ‘mindless eating’. However, it also seems plausible that it could reflect an increased self-awareness of eating behaviour and attitudes, engendered by increasing media coverage of diet-related health concerns.

Thus, it seems probable that individuals who report a consistent profile of comfort eating are in the minority of the general population, even if quite a large one. Indeed, as far as the response to stress is concerned, suppression of appetite, as opposed to comfort eating, tends to be the more commonly reported outcome, depending on the degree and the nature of the stress, as well as the individual’s ability to cope with stress (Gibson, 2006); these issues are discussed later.

This tendency for emotional eating to be distributed as low to moderate in these surveys has implications for experimental tests of the predictive power of the questionnaires. Given that these questionnaires use response formats that assess the frequency of occurrence of a situation/behaviour (DEBQ; EMAQ) or the likelihood of a situation/behaviour being true (TFFEQ), median splitting of the scale scores may not represent functional comfort eaters versus noncomfort eaters, depending on how close the median is to the midpoint of the scale.

A recent study specifically addressed this issue of the predictive validity of the emotional eating measurement: to test the predictive value of scores on the DEBQ scale for emotional eating Van Strien et al. (2012) recently measured snack intake, in female students, after negative or neutral mood induction (film, between participants, data from Anschutz et al., 2008), or after stress (Trier Social Stress test; within participants) or no stress. One strength of the study is its ability to define low and high emotional eating groups using cut-off scores of the 20th and 80th percentiles of norms for that population. In both experiments, emotional eating was a significant moderator of the relationship between distress and snack intake, as expected, that is only high emotional eaters ate more when distressed, whereas low emotional eaters ate less; however, this interaction could not be found when relying only on a median split for the entire sample (Study 1, n = 124). Thus, the effect relies on having sufficiently extreme samples of both low and high emotional eaters: the lack of such variability in emotional eating may explain previous failures to show this effect. It also suggests that the DEBQ only robustly predicts overeating induced by negative affect for individuals scoring near the highest quintile.

### Theories of eating styles relevant to comfort eating

To understand current notions of comfort eating and its aetiology, it is helpful to consider briefly some relevant theoretical descriptions of eating styles and their proposed relationships with risks for overeating. Where possible, the effects of stress or negative affect on these eating styles are discussed.

#### Psychosomatic and externality theories

At a time when obesity was rare, and typically treated from a psychiatric or an analytic perspective, two theories of obesity and overeating emerged, with some similarities. Psychosomatic theory essentially proposed that the major cause of obesity was indeed hyperphagia because of emotional eating, as a means to reduce anxiety (Kaplan et al., 1957). Subsequently, Bruch (1961) suggested that a possible cause of emotional eating was a confusion between emotions, particularly negative affect, and sensations related to hunger, and that this might be acquired during childhood perhaps because of particular parenting strategies. This then begs the question why some individuals might be reinforced in their drive to eat more by emotional calming than by satiation of hunger. This issue of individual variation in susceptibility is a recurring theme of this review.

The apparent insensitivity of the obese to physiological cues underlying hunger, for which there was early experimental evidence (Stunkard, 1959), is also a key facet of the ‘externality’ theory of obesity, in which Schachter (1968) proposed that overeating, in at least some obese individuals, reflected an over-responsiveness to external cues related to food, and reviewed evidence in support of his hypothesis (e.g. eating in relation to sensory qualities, portion size, availability, time...
of day), contrasting with a tendency to ignore or be insensitive to internal cues related to physiological need. Indeed, Schachter (1968) was explicitly following up these ideas from Bruch (1961) and Stunkard (1959). In terms of comfort eating, Schachter (1968) reported the results of an experiment (very much of its time) in which fear was induced by the threat of painful electric shocks: this suppressed snack intake in a ‘taste test’ in healthy controls but not in obese individuals, which could be interpreted as early experimental evidence for comfort eating. This effect mirrored that found when comparing the amount eaten in fed and fasted states, in the same study; that is, again intake in the obese did not vary, implying insensitivity to physiological need; disappointingly, Schachter (1968) did not report the three-way ‘obesity by deprivation by fear’ interaction, which might have been informative for the theories considered here, that is by showing whether there was any dissociation for obese individuals of the influence of hunger versus fear on snack intake. Externality theory is echoed in the measures of ‘external eating’ aspects of disinhibited eating, and the renewed interest in reward sensitivity, discussed below.

**Restrained and disinhibited eating**

Current theories of emotional eating emerged from a literature examining the influence of negative affect including stress on overeating; the two key concepts in this literature are ‘restrained’ and ‘disinhibited’ eating (Van Strien et al., 1986; Wardle, 1987). Restrained eating, or dietary restraint, is the tendency to restrain (restrict) food (or energy) intake to maintain or reduce body weight, sometimes also characterized as ‘eating less than you want to’ (Van Strien et al., 2009). Although this concept overlaps with ‘dieting’, it does not necessarily imply successful weight maintenance; on the contrary, the original theory was developed to explain why ‘dieters’ are often unsuccessful in this (Herman and Mack, 1975), with the Restraint Scale consisting of items measuring weight fluctuation, dieting, bingeing and cognitions and emotions concerning amounts eaten. Thus, eating was said to be ‘restrained’ within certain limits, but events and situations, including emotional challenges, could conspire to induce eating beyond those ‘boundaries’ – an effect known as ‘disinhibition’, or ‘counter regulation’, where the event was previous food consumption. Following on from Schachter’s (1968) results showing a lack of suppression of eating by fear in obese individuals, a substantial number of experimental studies have tested the hypothesis that this effect is predicted by high restraint, whether obese or not (Greeno and Wing, 1994; Wardle and Gibson, 2002), that is restrained eaters do not suppress eating when stressed. The results are generally in support of this prediction, although, in line with comfort eating being associated with overeating, sometimes the finding was that restrained eaters and/or obese ate more under stress. Refinements to the hypothesis have included whether or not the stressor is ego threatening, and a possible role for cognitive load, rather than stress per se, in disrupting the cognitively driven restraint on eating (Ward and Mann, 2000; Wallis and Hetherington, 2004). Indeed, Wallis and Hetherington (2004) showed that eating chocolate was increased by both ego-threatening and cognitively demanding stressful tasks in restrained eaters, but only by the ego-threatening stress in emotional eaters (defined by DEBQ).

Given the nature of those Restraint Scale items, it is not surprising that obese individuals often score more highly on restrained eating than nonobese individuals (Ruderman, 1983). However, subsequent theories have proposed that normal-weight successful dieters may regularly restrain their intake by using cognitive strategies, but avoiding susceptibility to external or emotional stimuli: thus, scales were developed that explicitly sought to separate out pure cognitive restraint strategies from tendencies to be induced to overeat or at least to break the diet. These tendencies were measured together by the Disinhibition scale of the TFEQ (or Eating Inventory; Stunkard and Messick, 1985), but were separated into External and emotional eating scales in the DEBQ (Van Strien et al., 1986). Weinstein et al. (1997) reported student survey data (52 women, 49 men) that supported disinhibition (TFEQ) as a better predictor of comfort eating in women than restraint, although neither was predictive in men (possibly because of lower scores and less variance). In that study, comfort, or stress-induced, eating was defined by two questions on how stress altered their intake by using cognitive strategies, but avoiding externality theory. It was argued that such individuals may be particularly vulnerable to loss of control overeating, because of using ‘all or nothing’ rigid rules that are too easily threatened (Westenhofer et al., 1994). Using data from version 2 of the revised 18-item TFEQ (TFEQ-R18v2; emotional eating scale increased to six items) in a large clinical trial of obese patients (N=1741) and also a nonclinical web-based survey (N=1275), Cappelleri et al. (2009) found a small positive correlation between cognitive restraint and BMI in the nonobese sample, but a negative correlation in the obese sample: one might infer a tipping point such that restraint is lost beyond a certain BMI, and it may be relevant that in both samples the group with the highest restraint score did significantly more in variability in BMI than the lower restraint score groups. Nevertheless, many other studies that included overweight and/or obese participants have reported positive correlations between dietary restraint and BMI (Ruderman, 1983; Van Strien et al., 2009), so that it remains possible that being...
overweight and at least moderately obese may cause restrained eating rather than vice versa – this causality question has not been resolved (Van Strien and Koenders, 2012).

Of particular relevance to comfort eating, Haynes et al. (2003) showed, using experimentally induced stress (task failure), that stress-induced eating, from a buffet lunch, was seen reliably in women scoring high on both the restraint and the disinhibition scales of the TFEQ, and to a lesser extent in those scoring low on both scales, whereas high disinhibition combined with low restraint was associated with eating the most under no stress but less under stress. These latter women were presumably easily disinhibited to eat more by the buffet meal; however, it would appear that a history of restrained eating is necessary to permit disinhibition under stress.

Although appetite ratings did not differ by restraint or disinhibition, it is possible that a tendency to undereat when not stressed might encourage comfort eating when stressed, in easily disinhibited women, as the comfort foods would likely be harder to resist if any negative energy balance were present – a prediction supported by animal models of ‘comfort eating’ discussed below. It may be relevant that mood data suggested that high disinhibited eaters may also be more affected by stress.

Similar findings were reported in a more recent study examining the effects of positive, negative and neutral mood manipulations (using films) on snack intake in women (Yeomans and Coughlan, 2009). That is, women who were both disinhibited and restrained eaters ate more after the positive mood manipulation. Interestingly, disinhibited women who were also low in restraint ate most after the positive mood film; any relation to adiposity was not addressed in that study, but in a questionnaire survey of emotional eating, underweight respondents claimed to eat more during positive mood states (Geliebter and Aversa, 2003). Furthermore, negative mood induction was recently shown to increase the urge to eat only in ‘high disinhibition’ women who had been exposed to (but not actually tasted) desired foods (Laxton et al., 2011). These and other studies (Lowe and Kral, 2006) suggest that restrained eating per se is not a very reliable predictor of eating behaviour during negative affect.

Using a progressive ratio reinforcement task to measure the relative reinforcing value of energy-dense snack foods (vs. that for reading interesting media) in 273 obese and nonobese adults, Epstein et al. (2012) showed recently that disinhibition alone predicted snack energy intake and BMI, but it also enhanced the association of food reinforcement with both intake of such snacks and BMI. There was also a weaker moderating effect of restrained eating (TFEQ), in that restrained eaters who also found snack food more reinforcing were more likely to have a higher BMI. Together with the findings discussed earlier, this would fit with restraint both resulting in enhanced food reinforcement through eating during negative energy balance and allowing overeating when the cognitive control is broken by overpowering temptation.

These eating styles of restraint and disinhibition have begun to be considered in the context of broader personality characteristics such as impulsivity, (poor) delay discounting (ability to wait for a larger delayed reward) and reward sensitivity (Davis and Fox, 2008; Yeomans et al., 2008; Appelhans et al., 2011; Volkow et al., 2011). However, whereas disinhibition may include an emotional as well as an external eating component, when these constructs were considered separately, and their relationships with depression scores were examined by structural equation modelling, in a sample of 549 mainly obese or overweight women, depression was directly predictive of emotional but not external eating (Ouwens et al., 2009). Moreover, the final model showed that impulsivity was predictive of both emotional and external eating, but mainly contributed to emotional, but not external, eating by interacting with a measure of sensitivity to one’s feelings. These findings may help to explain the results from an experimental manipulation of negative affect (task failure) in a younger nonobese student sample, whereby the ability of negative affect to increase self-reported emotional eating tendencies was slightly enhanced by impulsivity (Bekker et al., 2004). These intermediate traits between depression and emotional eating are also in line with understanding comfort eating as a component of broader emotional dysregulation (Tice et al., 2001).

External or uncontrolled eating may combine facets of impulsivity, weak delay discounting and reward sensitivity: it is therefore quite surprising that, in a nationally representative sample of 1342 Dutch adults, restrained and emotional eating both moderated the relationship between reported overeating tendencies and being overweight, whereas external eating did not (Van Strien et al., 2009). Restrained and emotional eating were both higher in overweight than in normal-weight participants, but external eating was not. Moreover, when the effects of restrained and emotional eating were partialled out in a regression analysis, external eating actually became associated with a lower risk of being overweight.

In summary, this section has shown how a need to measure emotional eating as a separate construct emerged from earlier theories, and that emotional eating scores are often stronger predictors of overeating than other eating styles such as restraint or disinhibition. Emotional eating as a risk factor for obesity is considered further in the final section.

Are some foods more comforting than others?
Intuitively, foods eaten to provide relief from negative affect may have particular qualities, such as sensory or nutritional attributes, that allow them to have an
emotional impact on the eater. Thus, in contrast to the effects of restrained eating, for which the total energy intake may be the key goal, the prediction for emotional eating is that any increase in intake should be for particular food groups that could induce a reinforcing shift in emotional state. This section briefly reviews animal and human evidence to support this hypothesis. It should be acknowledged that the animal work typically involves the use of apparently quite strong stressors, such as physical restraint in rats, whereas comfort eating in human beings occurs to negative affect where the level of stress (if present) is evidently not severe enough to suppress appetite, at least in susceptible individuals. Nevertheless, there is often a striking similarity in the effects on eating behaviour and food choice between studies in the different species, and the purpose of this section is to summarize evidence that comfort eating is associated with consumption of particular foods. Furthermore, in humans, stimulation of the hypothalamic–pituitary–adrenal (HPA) axis and the release of cortisol can occur in response to relatively mild eating-related events including protein-rich meals (Gibson et al., 1999) and food anticipation or withdrawal (Ott et al., 2011), not just severe stressors. The extent of HPA axis activity can also be predicted by quite subtle variations in affective predisposition (Steptoe et al., 2007).

Evidence from animal studies

Results of earlier work using animal models to study the effects of stress on food choice were rather inconsistent, perhaps because of the varied approaches used (Wardle and Gibson, 2002). However, an interesting observation was that stress could reduce rats’ tolerance of unpalatable adulteration of food, for example with bitter quinine, implying one mechanism by which comfort eating might focus on palatable rather than on bland or unpalatable foods. By comparison, more recent models suggest that stress-induced ‘binge eating’ can be found in rats, provided that they experience both stress and food restriction, and that they have a choice of highly palatable food (e.g., chocolate) as well as bland laboratory chow (Hagan et al., 2002; Boggiano et al., 2005) (cf. the interaction of restraint and disinhibition in women, above).

Compelling evidence also began to emerge from animal models that eating certain foods might counteract some physiological effects of stress, for example an energy-dense diet can prevent the loss of appetite caused by inescapable stress in rats (Dess et al., 1998). Indeed, there is now substantial support for suppression of the HPA axis activation in rats allowed to eat sweet and/or fatty foods (Buwalda et al., 2001; Dallman et al., 2003; Pecoraro et al., 2006). A particularly powerful finding was that when adrenalectomized rats voluntarily consumed sucrose (but not saccharin), the metabolic deficits that normally follow this procedure, in part because of the lack of corticosterone, seemed to be prevented (Bell et al., 2000); in other words, sucrose intake appeared to substitute for the missing glucocorticoid stress hormone. Another related finding of potential relevance to comfort eating was that rats allowed a free choice of solid fat (e.g., beef, pork or vegetable shortening) or chow had reduced HPA axis responses to stress (physical restraint), including both lower adrenocorticotropic hormone (ACTH) and corticosterone responses, compared with rats fed a 50% solid fat/chow mix (similar in ratio and amount to that eaten by the choice group) (La Fleur et al., 2005). Thus, choice rather than energy per se may be important, or conversely, having one’s preferred food forcibly adulterated may prevent the stress reduction that free consumption of solid fat allows. That study also suggested that the adipocytokine leptin was not a critical signal for dampening down the HPA axis responses (ACTH, corticosterone) as the levels increased equally for both diet conditions.

In a more recent study, in which rats were fed for 7 days with sucrose or solid fat or both (separately) before restraint stress, all diets inhibited the HPA axis responses to stress, and were particularly effective in reducing the initial peak in ACTH release (Foster et al., 2009). However, the least effective condition was the choice of both sucrose and solid fat, despite this group eating the most energy and depositing the most fat. Foster et al. (2009) also showed that eating sucrose compared with solid fat can exert separate effects on brain limbic pathways and corticotrophin releasing hormone synthesis and distribution of activation. In addition, after the stressor, rats ate some sucrose (where available), but not much solid fat, indicating that sucrose may be more reinforcing to eat after stress than fat, and possibly that the rats had learned this, although consuming sweet foods when stressed may also be driven innately (Booth et al., 2010). Another study showed that 2-h restricted daily (or 3 days/week) access to a sugar/solid fat mix, which induced binge-type consumption, was more effective in suppressing HPA axis activation to restraint stress than unrestricted access to this mix, and also maintained poststress intake of the mix (Kinzig et al., 2008).

It is important to note that these studies were primarily investigating how energy-rich diets eaten for at least a week before the stress, and associated body fat gained, were able to suppress the neuroendocrine response to that stress. However, most of the studies were not set up to test the hypothesis that rats (or human beings) may learn to like foods that alleviate stress. Nevertheless, a recent study in rhesus monkeys examining the effects of psychosocial stress from subordination reported that, after an acute stressor, energy eaten specifically from a high-fat and sugar diet was positively predicted by the increase in cortisol caused by the stressor (Michopoulos et al., 2012). Furthermore, there was evidence that these stressed monkeys acquired a habit of greater energy intake, even when access to the high-fat and sugar diet was withdrawn. It has been pointed out that it makes
adaptive sense for an animal to eat energy-dense foods when their safety, and therefore the time available to choose and eat food, may be threatened (Pecoraro et al., 2006). Indeed, Pecoraro et al. (2004) showed that chronic stress caused rats to select more sugar and fat instead of chow, that is a more energy-dense diet, even though they did not eat more energy overall. This shift in diet in turn was associated with inhibition of the HPA axis. There is increasing evidence from animal studies that activation of the HPA axis and the impact of the released glucocorticoid hormones lead to amplification of the incentive salience of cues to reward and perhaps also to punishment (Pecoraro et al., 2006). For example, injection of corticotrophin releasing hormone into the nucleus accumbens, a key site processing incentive salience, enhanced rats’ lever pressing for sucrose in response to a predictive cue for that reward (Pecina et al., 2006). Finally, perhaps echoing the observation in humans that comfort eaters are in the minority and reflect individual variation, it should be noted that Pecoraro et al. (2006) discovered that some of the observations in rats described above could not be reproduced consistently in other breeding lines of rats, even within the Sprague-Dawley strain.

Evidence in human beings

Several human studies have also linked the HPA axis response to stress to changes in food choice and intake: these will now be reviewed, together with other human studies linking comfort eating with changes in food choice. Although the experimental studies largely address the impact of stress on eating and food choice, rather than specifically emotional eating, such studies are thus comparable with the animal studies reviewed above; furthermore, observational studies that include measures of emotional eating are also considered here. Research into comfort eating, or stress-induced eating, and food choice (or dietary composition) in human beings has involved surveys, observational (or quasi-experimental), experimental laboratory, or epidemiological studies. The observational (or naturalistic) studies typically make use of known stressful episodes in participants’ lives, such as exams for students, or very busy work periods, or else ask participants to keep a diary of mood and stressful events. For example, during high workload, female office workers reported eating more fat and energy compared with a normal workload period (McCann et al., 1990). Similarly, in department store workers, busy periods were associated with an increased intake of fat, sugar and energy, but only in restrained eaters (Wardle et al., 2000).

Exam periods among adolescent or young adult students have been associated with higher energy intake (Michaud et al., 1990) or, when negative mood is also present, with a less healthy diet (Weidner et al., 1996). Similarly, a national cross-sectional survey of Turkish students found that stress was strongly linked to less likelihood of eating fruit and vegetables (Unusan, 2006). However, not all studies have detected changes in diet during exam periods (O’Donnell et al., 1987; Pollard et al., 1995); moreover, students have financial constraints (themselves stressful) on the healthiness of their diets, and also frequently experience stress at other times of the year.

In the UK study of the DEBQ measures in adolescents (Wardle et al., 1992), school children completed 24-h dietary recalls; whereas restraint was associated with less energy intake and external eating with more, there was no consistent relationship with emotional eating for either energy or macronutrient intake. However, this could in part be because of the relatively low levels of emotional eating in that sample, as well as the likelihood that emotional eating may not have been elicited in many of those 24-h periods, that is the impact of emotional eating on what or how much is eaten may be more ephemeral than that for either restraint or externality.

In a sample of 47 female American college students asked to record their food intake and mood just before each eating episode, over 12 days, eating in the presence of negative mood was only associated with snack intake, not meal consumption, and greater snack intake under these conditions was only observed for overweight students (Lowe and Fisher, 1983).

In a diary study comparing high and low stress weeks (hours worked), adult male and female nurses and teachers (N = 69) reported eating more ‘fast food’ during high stress weeks (Steptoe et al., 1998). Moreover, comfort eaters (defined by the mood scale of the Food Choice Questionnaire) ate more sweet foods (excluding chocolate) than noncomfort eaters, irrespective of workload. The men, but not women, reported greater intake of red meat during stress; however, the male sample was quite small (N = 24), and thus requires caution in interpretation. Fruit consumption was not affected by stress, although consumption of vegetables was not reported.

In a larger study of adult workers (N = 422) recording daily stressors and diet, stressful periods were associated with eating more high fat/sugar snacks and less main meals and vegetables (O’Connor et al., 2008). Moreover, the associations for stress and high-fat and sugar snacks were moderated specifically by emotional eating (DEBQ), supporting the prediction that high emotional eaters eat more such snacks when stressed. This same group also examined a possible role for the HPA axis in linking stress (daily hassles) to snacking (defined as between-meal food intake) by measuring cortisol response to a stressor involving public speaking and a mental subtraction task in 50 women (Newman et al., 2007). On the basis of this cortisol stress response, women were grouped either as high or as low reactors for analysis. The intensity and frequency of daily hassles both predicted snack intake, and this relationship was stronger for the high reactors. However, the relations
between positive and negative mood and snacking (less and more, respectively) were only significant for the low reactors. This interesting finding suggests there could be a dissociation between cortisol-driven snacking and mood-driven snacking: in other words, it questions whether high stress reactors are in fact primarily comfort eaters. It should be noted that the ‘high reactors’ were defined on their response to an acute stressor only. It is also possible that such ‘high reactors’ are not in fact dysfunctional; on the contrary, an unresponsive HPA axis has been associated with atypical depression (Gold and Chrousos, 2002), which is the form associated with hyperphagia, as distinct from the loss of appetite associated with more typical melancholic depression, and a recent report showed that a history of chronic stress in women was associated with a lower cortisol response to an acute stressor (while at the same time they felt more stressed), and that these low-reactor but higher stressed women reported higher levels of emotional eating (Tomiyama et al., 2011). In addition, the evidence that these women tended to have more abdominal adiposity, which in turn was correlated with lower cortisol, is in line with Dallman’s ‘chronic stress response network’ theory (Dallman et al., 2003) that eating an energy-dense diet and accumulating more central fat can suppress the responsiveness of the HPA axis, possibly mediated by an increased sensitivity of the axis to negative feedback inhibition from the circulating hormone. Nevertheless, it is at odds with earlier research showing that abdominal adiposity in women was associated with higher cortisol release after stress (Moyer et al., 1994; Epel et al., 2000), although this finding was not replicated in men (Epel et al., 1999). A key difference might be the level of chronic stress experienced in the highly stressed women in the study of Tomiyama et al. (2011). In addition, daily cortisol secretion, indexed by urinary free cortisol, is positively correlated with waist circumference and energy intake in women, independent of BMI (Vicennati et al., 2011): this appears to conflict with the finding, in nonobese young women, that free fatty acid infusion inhibits the HPA axis (Lanfranco et al., 2004), although in obese individuals, insulin resistance as well as stress can lead to an increase in free fatty acid levels in association with higher cortisol levels (Bjorntorp, 1999). Furthermore, elevated glucocorticoid release is known to enhance motivation for appetitive behaviours (Pecoraro et al., 2006), and thus, it is difficult to dissociate cause and effect in such findings.

In another laboratory stress study, in 59 women, that similarly divided participants into high and low stress reactors on the basis of cortisol response, and incidentally offered snacks to eat, high stress reactors were seen to eat more of sweet snack foods, and more energy when stressed (Epel et al., 2001). By comparison, when the effects of a laboratory stressor were examined on food choice over lunch, in 68 men and women, there was no effect of stress on intake or choice overall, but emotional eaters specifically ate more sweet fatty foods when stressed (Oliver et al., 2000). By contrast, cognitive restraint (DEBQ) did not significantly interact with stress and eating. In an experimental study in women using anticipated public speaking as a stressor and a computer-based task to assess the relative reinforcing value of snacks versus fruit and vegetables, binge eating tendency (associated with comfort eating) interacted with self-reported stress sensitivity (Goldfield et al., 2008): stress-reactive binge eaters found snacks more reinforcing when stressed, whereas the opposite was the case for nonbinge eaters. Stress made no difference to food value in those less sensitive to the stress.

Sometimes, an effect of stress on food choice can be easy to demonstrate: Zellner et al. (2006) simply gave 34 young female students either solvable (control) or unsolvable (stressed group) anagrams and allowed them to eat freely from servings of snacks, that is crisps, peanuts, chocolate M&Ms and grapes. The stressed group ate more M&Ms and fewer grapes than the unstressed group; the other foods were less popular and unaffected by stress condition. However, eating attitudes were not measured in this sample, and thus we cannot know the contribution of emotional eating tendencies. Zellner et al. (2007) repeated this experiment in men, but found quite the opposite result, in that it was the unstressed group who ate more of the unhealthy snacks (crisps and M&Ms). Again, we do not know whether any of the men were in fact emotional eaters, although it seems unlikely that many were: Oliver et al. (2000) found that emotional eating predicted food choice changes induced by stress in both men and women. However, there are many reports that stress suppresses food intake in individuals low in restraint, as men often are (Greeno and Wing, 1994; O’Connor et al., 2008). By contrast, there is additional evidence that some men may indeed be susceptible to comfort eating: using food frequency and questionnaire data, we reported recently that men who were both poor at stress coping and suffering from stress at work reported eating more sweet fatty foods than other men (Gibson et al., 2008). Two recent experimental studies of stress and eating in female students (Van Strien et al., 2012) make it clear that to show reliably that emotional eaters are susceptible to increased energy intake during stress, it is necessary to have a wide distribution of emotional eating tendencies in the sample (see above). In agreement with other findings, where foods varying in energy density and sensory properties have been available, stressed emotional eaters specifically ate more of the sweet fatty ‘butter cake’ versus the healthier less energy-dense foods.

There is also survey-based and epidemiological evidence to suggest that stress and/or negative mood may be associated with changes in food choice, not just intake. For example, in US adolescents, depressive symptoms were associated with increased intake of sweet soft drinks, as well as more disordered eating including meal
skipping (Fulkerson et al., 2004). Similarly, in 11–13-year-old children in London, those with greater self-reported stress ate more fatty foods and snacks but less fruit and vegetables, and tended to skip breakfast (Cartwright et al., 2003). In Finnish adults, ‘stress-driven eaters’ ate more high-fat foods and had higher BMI (Laitinen et al., 2003). In a more recent Finnish survey of 1679 men and 2035 women, emotional eating (TFEQ-R18) was predictive of higher intake of sweet foods for both men and women and for nonsweet fatty foods in men (Kontrinien et al., 2010). In addition, the positive correlation between depression and sweet food intake was largely explained by emotional eating. In a survey of 3266 Swedish parents and 1441 12-year-old children, emotional eating (albeit averaging <2 on the DEBQ scale) was correlated with greater frequency of eating confectionery and soft drinks (except for boys); external eating was also similarly related, whereas restrained eating was associated with eating confectionery less often (Elfhag et al., 2008). In 520 French adults, emotional eating was associated with more snacking on cakes, pastries, biscuits and oily fruit such as olives, as well as sweet beverages, whereas eating more energy-dense food was predicted by uncontrolled (disinhibited) eating (De Lauzon et al., 2004). However, in another French sample, of 1320 parents and adolescent children, emotional eating (DEBQ) was unrelated to macronutrient intake (from 3-day diet diaries) (Lluch et al., 2000). Similarly, in a sample of 475 young Dutch women, emotional eating was not related to macronutrient or energy intake (Anschutz et al., 2009), although such nutrient-based data can obscure subtle changes in food choice.

In an online survey of nearly 3000 Australian adults with clinical depression, chocolate was craved by half the respondents and believed by them to provide relief from the negative affect (Parker and Crawford, 2007); these chocolate cravers also had more stress-prone personalities. A similar pattern was observed in 931 men and women from San Diego, California, who were free from antidepressant medication: Rose et al. (2010) examined the relationship between depression scores (Center for Epidemiological Studies Depression Scale) and the rate of chocolate consumption. Using three cut-points in the distribution of depression scores, chocolate consumption was shown to increase markedly with increasing severity of depression: thus, the group scoring below the cut-point for any depression reported eating 5.4 servings per month on average, whereas those with scores indicating moderate depression ate 8.4 servings per month, and the highest scoring group (likely major depression) ate a mean of 11.8 servings per month. This trend was clearly statistically significant, and could not be accounted for by greater energy, fat or carbohydrate intake. The trend was present for both men and women, although the effect was somewhat stronger in women. Obviously, in these cross-sectional data, causality cannot be determined. Furthermore, a prospective study in 3486 UK civil servants, which allows some assessment of causality, or at least sequence, examined the risk for depression (using the same instrument) 5 years after the assessment of diet and other confounders (Akbaraly et al., 2009): a healthy ‘whole food’ diet was found to reduce the risk of depression by 26%, whereas a high-fat ‘processed food’ diet, including sweet desserts, predicted 58% greater risk of depression 5 years later. Still, the lack of effect of energy and fat intake on the depression–chocolate link makes this reverse-causality explanation less likely for the findings of Rose et al. (2010). A harder finding to interpret, but nevertheless intriguing, and related, is that per capita sugar consumption across six nations was very highly correlated ($r = 0.95$) with the annual rates of depression for those countries (Westover and Marangell, 2002).

In smaller surveys, some quite consistent findings emerge. Among 212 London students asked about how stress affects their eating, including the intake of a variety of main food groups, sweet fatty foods including chocolate were chosen when stressed, even among those who claimed they usually ate less overall when stressed. By contrast, fruit, vegetables, meat and fish were clearly eaten less when stressed. This effect was replicated in another London student population, in whom it was shown that both emotional and uncontrolled eating were associated with self-reported stress eating (Vogele and Gibson, 2010). Among US male and female students, preferred comfort foods were sweet, especially chocolate (Zellner et al., 2006); comfort (stress) eating was predicted by higher scores on the Restraint Scale, but as discussed earlier, this could reflect emotional and uncontrolled eating components as well. In an online survey of female US students, stress resulted in less healthy food choices, and among those eating more when stressed, sweet and/or high-fat foods were typically chosen (Kandiah et al., 2006). In 60 obese Swedes, higher neuroticism was associated with a preference for sweet and fatty foods (Elfhag and Erlanson-Albertsson, 2006): disinhibited eating (TFEQ) showed a similar but nonsignificant trend; emotional eating was not measured but may have been subsumed under the disinhibition measure.

In two surveys of comfort food preferences in randomly sampled North American adults, a slightly different picture emerged (Wansink et al., 2003). In the first survey asking about preferred comfort foods, with 411 complete responses out of 1000 sampled, potato crisps (US ‘chips’) were the most frequently endorsed comfort food; other fatty and sweet snack foods were popular, including chocolate and ice cream. However, 40% of the sample preferred meal-type foods as ‘comfort foods’ including pasta and pizza. Unfortunately, we do not know how many of the participants would actually score highly on an emotional eating questionnaire, and the interpretation of comfort foods here could include the nursery food connotation discussed earlier. Indeed, the finding
Can comfort foods improve mood and/or relieve stress?

Although the question of whether comfort foods can actually comfort the eater is clearly a key one, there is relatively limited direct evidence available, and the temporal characteristics of such an effect are even less understood. Thus, both direct and indirect evidence is discussed here. Mood and food can be related in several ways, including influences of positive mood on sensory appeal and appetite, as reviewed elsewhere (Gibson, 2006; Macht, 2008), but this review focuses on the effects of foods on negative affect. Furthermore, Ganley (1989) reviewed some early studies of the impact of eating on negative affect; these suggested that eating was most likely to reduce negative affect that was diffuse and difficult both to explain or label and to control. This is a theme that is reflected in the experimental manipulations of affect described in the following studies. Another theme that emerges from those earlier studies is that stress-induced eating depended on the food available being palatable and energy dense. This may be important to the extent that comfort eating can be distinguished from binge eating: although both may be elicited by some form of negative affect, the very large intakes associated with binge eating may suppress emotions such as anxiety and anger in part by the huge demands on the digestive system shifting autonomic balance in favour of parasympathetic outflow for an extended time: thus, bingeing is typically followed by lower energy levels, sleepiness, etc. (Ganley, 1989; Waters et al., 2001). By comparison, comfort eating, which might include relatively moderate snack food intake, may rely more on subtle sensory or neurophysiological consequences of particular foods. Nevertheless, they may both share an important outcome that the foods eaten provide some alleviation of negative affect, even if primarily by distraction from the thoughts and threats that may be engendering the negative affect (Heatherton and Baumeister, 1991; Waters et al., 2001). Interestingly, in a study of emotional antecedents of binge eating, bingeing was predicted best by tense arousal (anxiety) and yet the binge itself did not reduce that emotional state (Waters et al., 2001): the inference is that bingeing may be reinforced more by escape from negative rumination than by affect reduction. However, bingeing may have its own negative consequences on mood that more moderate comfort eating may not.

Chocolate is probably the archetypal comfort food: survey data suggest that chocolate is one of the most likely foods to be eaten during stress or negative mood (Gibson, 2006), as well as being the most craved food (Rozin et al., 1991; Gibson and Desmond, 1999). There are several experimental studies that lend some support for the popular idea that chocolate can lift mood. Chocolate, particularly milk chocolate, is most likely popular as a snack in part because of the energy content (Gibson and Desmond, 1999), and the sensory properties predictive of that (Michener and Rozin, 1994), but also because of the psychoactive effects of the methylxanthine components, caffeine and theobromine: thus, Smit et al. (2004) showed, in a well-controlled study, that these compounds, in levels normally found in a small bar of chocolate, can improve mood as well as cognition, and led to increased liking of a drink flavour paired with them (Smit and Blackburn, 2005). Indeed, the beneficial effects of these compounds in chocolate might underlie its long history of use apparently for psychoactive reasons, even in forms lacking any innate sensory appeal. It is possible that chocolate, eaten on an empty stomach, could provide a sufficient stimulus to insulin release, such that brain serotonin synthesis is increased by increased uptake of the precursor amino acid tryptophan across the blood–brain barrier; yet, we are not aware of any attempt to test this directly (Gibson, 2011). However, the sweetness of some chocolate might be enough to alleviate stress through stimulation of endogenous opioid release, as sweet and perhaps fatty taste is known to calm distraught human and rat infants, and in the latter at least, this effect is blocked by opiate antagonists (Blass et al., 1989); moreover, chocolate chip cookies can alleviate pain in female students when other nonsweet foods are ineffective (Mercer and Holder, 1997).

In female students in whom negative mood was induced by an unsolvable task, those with binge eating or bulimic tendencies (i.e. with at least moderate scores on the Bulimia Test-Revised questionnaire) ate more chocolate chip cookies during a bogus taste test and reported improved mood, compared with controls (low scorers) (Kisler and Corcoran, 1997). The authors pointed out that their apparently ‘comfort eating’ group scored below the usual clinical cut-off for bulimia nervosa (BN) on that questionnaire, but clearly had greater bingeing tendencies than the control group: the point is that eating chocolate chip cookies alleviated negative mood in this susceptible group. Similarly, Macht and Mueller (2007) showed that chocolate could reverse the negative mood induced by a sad film, but more strongly in comfort (emotional; DEBQ) eaters. This effect occurred within a minute but lasted for just three minutes. The additional benefit to comfort eaters could reflect either a disposition or being more practised in using chocolate in that way.

Willner et al. (1998) examined the effects of negative versus positive mood induction, using music, on motivation to eat sweet milk chocolate ‘buttons’, compared with
that for nonsweet carob buttons (a form of chocolate substitute that participants reported disliking, perhaps in part because of unfamiliarity), in a progressive ratio task. This task involved the young women being rewarded with chocolate for pressing the spacebar a required number of times, which increased geometrically by doubling each time. The main measure was the breakpoint, that is the maximum number of presses after which the participant no longer responded for 2 min. Negative mood substantially increased the breakpoint, and number of buttons achieved, specifically for the chocolate reinforcer. It was found that the concurrent craving for chocolate (measured by questionnaire), which was increased by negative mood induction, correlated in this group with the breakpoint for chocolate: as Willner et al. (1998) cite other evidence that negative mood is associated with a reduction in sensory pleasure from foods such as chocolate, it may have been the increased craving, rather than the sensory reinforcer value of the chocolate per se, that was driving the responding. By comparison, despite participants’ self-report about dislike for carob, following the positive mood induction, both the breakpoints and the number of buttons consumed did not differ between the chocolate and the carob conditions. There was also a slight indication that contented mood, which declined after the negative mood music, improved on completion of the task, whereas contentedness remained stable throughout in the positive mood condition. Of course, it is not known to what extent the women were comfort eaters, but it would be expected that comfort eaters would show increased chocolate craving during negative mood, reflecting the pattern observed in this study. Moreover, we discussed above evidence that comfort eaters who are stressed work harder for energy-dense snacks relative to fruits and vegetables (Goldfield et al., 2008), which contrasts with the reduction of this relative reinforcing value of snacks by state anxiety, although only in unrestrained eaters (Goldfield and Legg, 2006) (dissinhibited eating was not measured). In other words, feeling stressed or anxious is likely to distract from wanting energy-dense food, unless one is a comfort eater, when the opposite is the case, probably because the comfort foods distract from the perceived stress (Heatherton and Baumeister, 1991; see below).

Some indirect evidence that comfort foods may be comforting comes from the observation that patients with winter depression (Seasonal Affective Disorder) typically eat more sweet (and perhaps starchy) foods during winter (and less after light therapy) (Krauchi et al., 1997); moreover, seasonal affective disorder sufferers score highly on the DEBQ emotional eating scale. These facets are predictors of weight gain during the winter. This is reminiscent of the increased frequency of chocolate eating associated with greater depression scores in a nonclinical sample discussed earlier (Rose et al., 2010); however, one might therefore infer that chocolate is not very effective in improving mood over the long term, and this conclusion was reached by Parker et al. (2006) in a review of chocolate and mood. It is also in agreement with the conclusion of Willner et al. (1998) that negative affect drives a desire for chocolate (in some individuals at least) that may be independent of the reinforcing value of chocolate. Instead, comfort eating may be one aspect of a broader problem of emotional dysregulation; for example, rumination of negative thoughts that is typical of depression predicts binge eating tendencies as well as other unhealthy behaviours, all of which may serve to distract from the negative thoughts (Selby et al., 2008). Indeed, the apparent transient improvement in mood after eating comfort foods might simply follow from a break from such rumination.

Overall, it is clear that comfort eating is likely to be associated with greater intake of highly palatable, especially sweet and/or fatty foods (with variation between sexes). These foods may have a short-term effect in reducing negative affect, but the habitual use of such foods to improve mood may be more dependent on their ability to distract from negative rumination, in susceptible individuals.

**Is comfort eating harmful and can it be treated?**

The most obvious concern for comfort eating and health is that it will lead to overconsumption of energy and ultimately to obesity. Indeed, emotional or comfort eating is one of the earliest theoretical accounts of obesity (Kaplan et al., 1957; Stunkard, 1959; Bruch, 1961); however, those early accounts included aspects in their theories that would now be regarded as related to restrained or external eating (Schachter, 1968), which are now more widely regarded as separable from emotional eating (Van Strien et al., 2009).

Ganley (1989) reviewed three decades of mainly clinical studies since those early theories were published, to address the contribution of comfort eating to obesity. Although clinical reports of episodic eating induced by stress or negative affect (sometimes termed binge eating, although unless excessive intake is confirmed, comfort or emotional eating is more accurate) were commonplace for obese patients, nevertheless, the actual proportion of patients (undergoing weight loss programmes) showing this eating behaviour was almost invariably no more than half, that is often a minority of patients. This is in agreement with the findings on prevalence discussed earlier. However, where nonobese controls were studied, evidence of emotional eating tended to be less than that in obese individuals, and in surveys of obese individuals not in weight loss programmes, reports of emotional eating were much higher, and also higher than that in control groups. Even so, there is a risk that at least some of the association of negative affect and eating commonly reported by obese patients could be an epiphenomenon,
that is the obese overeat regularly and suffer negative affect and stressful lives, but there may not necessarily be a causal link. However, experimental studies have largely found that obese individuals were more likely to show comfort eating (of energy-dense foods) in response to stress than nonobese individuals, and a meta-analysis of relevant prospective studies concluded that stress made a small but significant contribution towards weight gain (Wardle et al., 2011). Moreover, most modern conceptualizations of the aetiology of obesity incorporate the impact of stress on eating in some form (Butland et al., 2007).

Earlier studies, of at least a moderate sample size, that included measures of both emotional eating and obesity typically reported higher emotional eating in obese or overweight groups (Van Strien et al., 1985; Wardle, 1987) (Table 1). However, since the 1980s, the prevalence of obesity has increased at least three-fold in many countries; thus, one might expect a similar increase in emotional eating. There are insufficient data to address this directly, but the studies carried out 25 years apart on two representative samples of Dutch adults discussed earlier did show that emotional eating had on average increased significantly, as had obesity (Van Strien et al., 1986, 2009). In the recent study, both low dietary restraint and high emotional eating were predictive of overeating and weight gain (cross-sectionally), whereas external eating was not, and the authors conclude that ‘we may be well advised to seek an “emotional” explanation for the current obesity epidemic’ (Van Strien et al., 2009, p. 386).

In their study of a French population, De Lauzon et al. (2004) did not report relationships between BMI and emotional eating; however, they found that middle-aged men, but not women, who scored high on emotional eating consumed significantly more energy. By contrast, in the slimmer young adults and teenagers, emotional eating was unrelated to energy intake, whereas cognitive restraint in the women was predictive of less intake. In a follow-up study in this population, emotional eating was not related to adiposity measures (De Lauzon-Guillain et al., 2006), unlike cognitive restraint, although it should be borne in mind that the emotional eating scale of that version of the TFEQ-R18 is limited to three items.

One quite impressive finding is from a study of 2997 young Finnish women (aged 17–20) (Anglé et al., 2009): the sample included women classified by BMI as underweight, normal weight, overweight and obese, and emotional eating (TFEQ-R18) increased significantly with each category of increasing BMI (Table 1).

In a recent large online survey of lifestyle in Dutch bank employees (N = 3272), emotional eating was the strongest predictor, among lifestyle factors, of behaviours related to weight gain, in both men and women (Van Strien and Koenders, 2012), although the perceived or the actual role of stress and emotions in these workers might be somewhat inflated, as a zeitgeist of the current economic situation.

In a prospective study of 4065 UK teenagers over 5 years, perceived stress did not predict greater weight gain; instead, indices of higher adiposity were associated over all 5 years with higher stress, after adjusting for likely confounding variables (Van Jaarsveld et al., 2009).

It seems probable that comfort eating often, although not invariably, leads to weight gain: but even if obesity is not a result, health could still be compromised by the increased consumption of high-fat energy-rich foods. A short-term prospective study looked at stress (comfort) eating in medical students as a possible predictor of health outcomes related to the metabolic syndrome. Epel et al. (2004) followed these students from a baseline measurement through two exam periods: stress eaters gained more weight, and had higher nocturnal insulin, cortisol levels and blood total/high-density lipoprotein-cholesterol ratio; thus, comfort eating can predict poorer health outcomes, even in a young educated sample. This effect might be driven by poor diet, but if comfort eaters are also more stress reactive (see above), the physiological consequences could be contributing towards worse health and greater adiposity (Steptoe and Wardle, 2005). In older male caregivers of spouses with Alzheimer’s disease, the chronic stress of caring was predictive of psychological distress, which predicted poorer health behaviour, including a high-fat diet, and that in turn predicted metabolic syndrome and coronary heart disease (Vitaliano et al., 2002); the women in the study were largely protected by hormone replacement therapy.

In students in the UK, both self-reported weight gain and weight loss after the first year at university were predicted by stress levels, particularly in women (Serlachius et al., 2007); a plausible explanation for the weight gain could be comfort eating. In a study of stress and weight change in 71 student nurses, with measures 12 weeks apart, before and during exams, the independent predictor was dietary restraint (measured by the Eating Disorders Examination Questionnaire), which was associated with weight gain, after adjusting for other eating disorder tendencies, including bingeing, and changes in cortisol (Roberts et al., 2007). Emotional eating was not explicitly measured, although it is possible that the restraint measure captured some aspect of that behaviour; also, as discussed above, restrained eating can interact with and moderate the impact of emotional eating on diet (Yeomans and Coughlan, 2009). The reduction in restraint might have permitted emotional eating to enhance energy intake. Furthermore, Van Strien and Van de Laar (2008) have shown prospectively that, whereas dietary restraint and external eating can change after 4 years, and predict changes in fat and energy consumption.
intake, respectively, emotional eating tendencies seem to be more resistant to change.

In summary, the balance of evidence links emotional eating both to overeating, particularly of energy-dense foods, and to increased adiposity, with a likely increased risk to cardiovascular health. However, there is some variability that may be related to predisposing factors. The next section considers examples of these in terms of genetic and neurochemical associations.

**Genetic and neuropharmacological aspects**

This stability of emotional eating over the years has also been reported in children (Ashcroft et al., 2008), among other eating phenotypes, and suggests a trait-like quality that likely has a strong genetic component. Indeed, a study of Swedish male twins concluded that emotional eating (TFEQ-R18) showed high heritability of 60% (Tholín et al., 2005); however, other twin studies of both sexes have reported somewhat less heritability, for example 9–45% for UK and Finnish populations (TFEQ-R18) (Keskitalo et al., 2008), and 25% for a Korean population (DEBQ) (Sung et al., 2010). Interestingly, in the UK and Finnish twin samples, emotional eating also showed quite a strong genetic link to liking for sweet fatty foods (Keskitalo et al., 2008).

Although developmental stability of emotional eating could also be attributable to parenting style, one study that compared 214 families with obese or overweight parents with 114 families with normal-weight parents did not find any difference among mothers’ use of an emotional feeding style, although the obese mothers did show less control of their children’s eating (Wardle et al., 2002). Conversely, and intriguingly, excessive parental control over a child’s behaviour in general has been found to increase the risk of emotional eating, but only if the child has some genetic vulnerability related to their dopamine D2 receptor binding or expression. Thus, Van Strien et al. (2010) genotyped 428 adolescents for alleles of the Taq1 polymorphism (A1A1, A1A2 or A2A2), as well as measuring, at baseline and 4 years later, emotional eating (DEBQ) and the adolescents’ ratings of their parents control over their children’s behaviour. The Taq1 A1 allele may be associated with reduced dopamine-binding affinity at the D2 receptor (Thompson et al., 1997) or possibly reduced expression of D2 receptors (Stice et al., 2008a); as the D2 receptor can be either a postsynaptic receptor on nondopaminergic target cells or a presynaptic autoreceptor on dopamine releasing neurons (Le Foll et al., 2009), predictions for the behaviour of the A1 variants are not clear cut, and postsynaptic D2 receptors may also be downregulated over time, perhaps as a result of chronic overeating (Stice et al., 2008a; Johnson and Kenny, 2010). For instance, if this polymorphism compromises autoreceptor function, it would be associated with greater release of dopamine, for example in the ventral tegmental pathways, than in the case of the A2 homozygous variant. Thus, in D2 receptor knockout mice, psycho-stimulant drugs release more dopamine than in their wild-type littermates (reviewed by Le Foll et al., 2009); moreover, reward sensitivity, for both cocaine and food, is enhanced in mice lacking specifically the D2 autoreceptor, and that therefore release more dopamine in response to cocaine (Bello et al., 2011). In any event, Van Strien et al. (2010) did not find direct associations between A1 allele variants and either emotional eating or parental control, although emotional eating was significantly positively correlated with parental control at baseline. Instead, they found a significant interaction, such that the A1 genotype was associated with greater emotional eating after a 4-year follow-up only if the adolescents also reported high parental control, that is a gene–environment interaction. One could speculate that the restrictive parenting interacted with the poorly regulated dopamine output to exaggerate the incentive salience of energy-rich foods on the rare occasions they were accessed. Also, because analyses controlled for baseline levels of emotional, external and restrained eating and BMI, a reverse-causality explanation, that is that parents were more controlling because of their child’s overeating tendencies, is less likely.

These results also need to be considered in the context of emerging findings on dopamine polymorphisms, the risk of obesity and activation of reward and sensory pathways in response to presentation of visual or gustatory food stimuli using brain imaging, particularly functional MRI. For example, Stice et al. (2008a) found that young women with higher BMI showed less activation of reward pathways such as the caudate nucleus in response to expecting or tasting milkshake versus tasteless water, and furthermore, this inverse relationship was only true for women with the Taq1A A1 allele. In a second prospective study of weight change in female adolescents, the presence of the A1 allele again exaggerated the association at baseline between adiposity and blunted caudate responses to milkshake. At the 1-year follow-up, the increase in BMI was positively related to caudate activation only in girls without the A1 allele (Stice et al., 2008a). These associations in motivational pathways contrast with the finding that, at the same time, obese adolescent females show greater activation of gustatory cortex to milkshake, compared with lean adolescents (Stice et al., 2008b), implying a dissociation between perceptual and motivational responding. Notwithstanding any functional differences in dopamine activation (assuming that the regional activations are closely related to dopamine function), this might suggest cognitive inhibition of motivational pathways such as might result from greater restraint. However, a role for the distinct genotypes in restraint would also be needed to support such a process.

In a similar design in female adolescents, but including genotyping for dopamine D2 and D4 receptor polymorphisms, future weight gain was associated with weaker responding to food images in brain regions mediating incentive value for those with D2 Taq1A A1 and D4...
seven-repeat alleles, whereas for those without these alleles, greater activation in those areas was predictive of future weight gain (Stice et al., 2010). The authors suggest that there may be two distinct neurochemical predispositions for obesity, governed by particular dopamine polymorphisms.

Of particular relevance here, this group has also used the milkshake anticipation and tasting paradigm with functional MRI to dissociate responses of emotional and nonemotional eaters (DEBQ) following negative mood induction (by music and negative situation recall) (Bohon et al., 2009). For activation of brain cortical regions concerned with affective state and anticipatory reward (parahippocampal gyrus and anterior cingulate gyrus), there was an interaction between induced affect and emotional eating status: that is, emotional eaters showed relatively greater activation in those regions, in response to chocolate milkshake, when in the negative mood, but less activation when in the neutral mood. By comparison, emotional eaters also showed greater overall activation of reward areas, including the caudate and pallidum, to the taste of the milkshake, although not its anticipation. Moreover, key regions such as the amygdala and the ventromedial prefrontal cortex were not differentially activated in emotional versus nonemotional eaters in this context, although the study could have been underpowered to detect such effects. Thus, the authors conclude that emotional eaters do not differ in their emotional responses to food, but rather show heightened reward induced by (at least) chocolate milkshake, particularly when in a negative mood. This could in part reflect a learned response cued by negative mood; however, it is also possible that activation of the HPA axis is driving the enhanced reward sensitivity, as discussed earlier. This result is also in agreement with the earlier conclusion that such foods may be particularly effective at distracting emotional eaters from their negative ruminations.

These complex findings make it difficult to consider recommendations for pharmacotherapy for comfort eating, if it were warranted, even just for dopaminergic agents, although they also suggest a future role for pharmacogenomic approaches. For instance, one theoretical approach would be to use dopamine agonists selective for the presynaptic D2 receptor specifically in comfort eaters having the Taq1 A1 allele. Conversely, a therapy targeted at promoting the expression of postsynaptic D2 receptors might be effective in other overeating groups.

The complications of dopamine pharmacotherapy are well illustrated by an analysis of the relationships between dopamine and serotonergic receptor binding affinities of typical and atypical antipsychotic drugs and treatment efficacy (Richtand et al., 2007), the latter in particular being contraindicated for patients at risk of binge eating (McElroy et al., 2010). For example, for typical antipsychotics, the best binding predictor of efficacy (lowest effective dose) was the ratio of 5-HT2C/D2 affinities (r = −0.81), that is negatively related, such that the activation of 5-HT2C receptors is associated with efficacy (through D2 receptor antagonism) at lower antipsychotic doses. The authors suggest that 5-HT2C agonists may improve psychosis treatment by inhibiting mesolimbic dopamine activity. By contrast, for atypical antipsychotics, the relationship between efficacious dose and 5-HT2C binding is in the opposite direction and significantly different from the above, although not itself a significant correlation (r = 0.47). Instead, the best predictors of efficacious dose were combined affinities comprising D2(5-HT2C/5-HT1A) (r = 0.80) and D2(5-HT2C/5-HT1A) (r = 0.78) ratios. It may be that differing doses alter the balance of postsynaptic and presynaptic D2 and 5-HT receptor influences on psychosis, so that linear analyses are too simplistic.

Antipsychotic treatment, particularly atypical drugs, has been associated with weight gain and even with exaggerated binge eating in patients with eating disorders (McElroy et al., 2010). Sentissi et al. (2009) compared eating styles between schizophrenic patients (N = 153) treated with either class of antipsychotic or no treatment. Emotional eating tended to be higher in patients treated with atypical versus typical antipsychotics, but they were also clearly higher in external eating; BMI was higher in both treated groups than the controls, and the highest (but not significantly) in the atypical drug group. These data are limited, but could suggest an association between emotional and external eating and BMI in patients that is better addressed by typical antipsychotics.

The importance of serotonergic efferents in regulating dopamine pathways has echoes in the treatment of binge eating, both for patients with BN and for those with binge eating disorder, and may be relevant for any attempt to treat comfort eating. Depressive symptoms are known to be common in these eating disorders as well as among emotional eaters (Ouwens et al., 2009; Konttinen et al., 2010; Vögele and Gibson, 2010). Therefore, it is not surprising that the usual pharmacotherapy for BN is antidepressant treatment, albeit at quite high doses; furthermore, this approach is used to treat binge eating disorder, although with less reliability (McElroy et al., 2010). Clearly, one might in principle consider treatment strategies for comfort eating, where health was compromised, on the basis of a range of drug classes that have been shown to have some benefit in binge eating behaviour; for example targeting orexigenic hormones, opioid and cannabinoid hedonic pathways, and restraint of the HPA axis (Pecoraro et al., 2006; Shin et al., 2009). Moreover, as new therapies, such as peptide hormone analogues, become licenced for weight management, some of these may prove to be effective in reducing emotional eating; for instance, the appetite-stimulating

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hormone, ghrelin, has been found to mediate stress-induced eating in mice (Chuang et al., 2011), and human emotional eaters show lack of postigestive decline in ghrelin compared with nonemotional eaters, although their baseline ghrelin level was also lower (BMI did not differ; Raspopow et al., 2010). However, at least for the time being, nonpharmacological interventions such as stress management, relaxation, reward substitution and promotion of physical activity may be preferential (Konttinen et al., 2010).

**Conclusion**

Comfort or emotional eating (similar to stress-induced eating) has been a core theme of explanations for overeating and obesity for decades. Theories of emotional eating and related eating styles have gradually evolved so that there is increasing convergence with animal models of overeating as well as broader aspects of personality theory, such as impulsivity and reward sensitivity. Examination of prevalence suggests that emotional eating is more common in women than men, and in obese than nonobese individuals; nevertheless, emotional eaters typically are in the minority among nonclinical populations. An emerging theme is one of vulnerability to depression, emotional dysregulation and a need to find ways of escaping from negative affect and rumination. A strikingly consistent finding in both animal and human studies is that comfort eating (or stress-induced eating) leads to preferential consumption of sweet, and often fatty, energy-dense foods. The animal studies suggest that eating such foods confers protection against stress, evidenced by suppression of the HPA axis response, possibly mediated by hormonal consequences of accumulating abdominal obesity. However, the human data are less clear, in that the lifting of mood after comfort eating appears to be transient; perhaps stress-induced binges develop in an attempt to prolong mood improvement, although escape from negative rumination is as likely. Also, some studies find comfort eaters to be high reactors to stress, which itself has been linked to abdominal obesity, although not consistently. However, chronic severe stress eventually leads to reduced cortisol response to stress, although this may adversely affect health in the long term. Not surprisingly, many studies associate comfort eating with a greater risk of obesity, although not all studies are consistent in this: one reason may be that, like eating disorders, individuals rarely express just one pure facet of eating behaviour independent of others, and comfort eating might be associated with other personality characteristics that work against the risk of obesity. Still, chronic overeating of energy-dense foods, as may occur with comfort eating, is a risk factor for weight gain, and is likely to be detrimental to health over the long term.

The extent of heritability of comfort eating remains uncertain, as different populations of twin studies have produced quite variable results; nevertheless, genetic predispositions are likely, including polymorphisms such as Taq1A that affect the regulation of dopamine pathways. This variant might contribute to weakened resilience to stress, as its presence promoted the emergence of comfort eating in adolescents reporting high levels of parental control. Similarly, there is increasing evidence that this polymorphism dissociates the risk of overeating and obesity in terms of either overactivation or underactivation of dopamine pathways underlying incentive salience. Emotional eaters in particular seem to show heightened activation of incentive pathways to food stimuli during negative mood.

Even assuming that a patient’s health was being sufficiently compromised by comfort eating, the obvious complexities of neurochemical systems linking stress, emotions, impulsivity and reward sensitivity resist reliable proposals for pharmacotherapy using today’s drugs. At present, cognitive behavioural therapies would be the first approach. Nevertheless, future developments may use pharmacogenomic strategies to provide more personally focused drug therapies to prevent comfort eating from threatening health and well-being.

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**Conflicts of interest**

There are no conflicts of interest.

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