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Review

Mood state effects of chocolate

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Abstract

Background: Chocolate consumption has long been associated with enjoyment and pleasure. Popular claims confer on chocolate the properties of being a stimulant, relaxant, euphoriant, aphrodisiac, tonic and antidepressant. The last claim stimulated this review.
Method: We review chocolate's properties and the principal hypotheses addressing its claimed mood altering propensities. We distinguish between food craving and emotional eating, consider their psycho-physiological underpinnings, and examine the likely 'positioning' of any effect of chocolate to each concept.
Results: Chocolate can provide its own hedonistic reward by satisfying cravings but, when consumed as a comfort eating or emotional eating strategy, is more likely to be associated with prolongation rather than cessation of a dysphoric mood.
Limitations: This review focuses primarily on clarifying the possibility that, for some people, chocolate consumption may act as an antidepressant self-medication strategy and the processes by which this may occur.
Conclusions: Any mood benefits of chocolate consumption are ephemeral.

Keywords: Chocolate; Chocolate consumption; Negative mood state; Depression; Atypical depression

Contents

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1. Introduction

If any man has drunk a little too deeply from the cup of physical pleasure; if he has spent too much time at his desk that should have been spent asleep; if his fine spirits have become temporarily dulled; if he finds the air too damp, the minutes too slow, and the atmosphere too heavy to withstand; if he is obsessed by a fixed idea which bars him from any freedom of thought: if he is any of these poor creatures, we say, let him be given a good pint of amber-flavored chocolate....and marvels will be performed. — Anthelme Brillat-Savarin (1755–1826).

Chocolate is cheaper than therapy and you don't need an appointment. — Unknown.

It is commonly claimed that chocolate has the capacity to lift spirits, to create highs and make people feel good. In an earlier review of atypical depression and its constituent feature of hyperphagia (Parker et al., 2002), we noted the capacity of carbohydrates (including chocolate) to have a comforting effect and to also promote 'feel good' sensations through the release of multiple gut and brain peptides. Others have argued that carbohydrate craving (including chocolate craving) in atypical depression (Moller, 1992) and in seasonal affective disorder (SAD) (Wurtman and Wurtman, 1989) is a form of self-medication and, in having an impact on brain neurotransmitters have antidepressant benefits.

Publication of our earlier paper (Parker et al., 2002) led to considerable media attention and inquiries both from those with a mood disorder and the general public as to whether chocolate actually was an antidepressant, so encouraging a literature review. Use of the single word 'chocolate' in search engines identified an extraordinarily broad literature warranting integration and encouraging the current review. While we had an agnostic view about chocolate having any antidepressant properties, it became apparent that there were many other topic components of equal importance, resulting in this review going beyond the initial simple objective. As noted by one journal assessor, aspects have some significance for our understanding of the biology of depression, and for the concept of 'atypical depression' in particular.

In this overview, we consider why people crave and eat chocolate, particularly in the context of food cravings and emotional eating, and chocolate's mood state effects. In the many comprehensive reviews (e.g., Bruinsma and Taren, 1999; Christensen, 1993; Ganley, 1989; Mercer and Holder, 1997; Ottley, 2000; Pelchat, 2002; Rogers and Smit, 2000; Weingarten and Elston, 1990), explanations of chocolate and carbohydrate cravings vary widely and include self-medication, homeostatic correction, hedonic experience, addiction to psychoactive substances and emotional eating theories, but it may be possible to integrate the disparate theories. We will argue that chocolate 'craving' and chocolate 'emotional eating' are two separate phenomena — although they can co-exist in the same individual — and that any mood state effects of chocolate are as ephemeral as holding a chocolate in one's mouth.

2. Chocolate

2.1. Chocolate composition

Chocolate is manufactured from cocoa mass (the base product produced by the processing of the cocoa bean), cocoa butter (the natural fat from the cocoa bean) and added sugar. Cocoa butter melts at room temperature to provide the creamy "melt in the mouth" sensation. Europeans added sugar to appeal to their palate when chocolate was introduced from America. Dark chocolate contains these three elements, milk chocolate contains extra milk solids and fats, and white chocolate is akin to milk chocolate without the cocoa base.

2.2. Chocolate craving

Chocolate is the most commonly craved food and, for most chocolate cravers, non-chocolate substitutes are inadequate (Weingarten and Elston, 1991). Chocolate is not a natural product, and thus its appeal depends on its individual constituents and their unique combination. Chocolate is sweet, raising the possibility of confusion between chocolate craving and sweet craving, but it also contains fat (Rozin et al., 1991). Similarly, other foods that are commonly craved (e.g., ice cream, doughnuts, cakes, biscuits) also taste sweet and might be misjudged as rich in sugar, but most of their calories are provided by fat. Drewnowski et al. (1992) have therefore suggested that the term 'carbohydrate craving' is a misnomer when applied to such foods.

2.3. Psychoactive properties of chocolate

Because chocolate craving has some features of addiction, attempts have been made to identify any psychoactive ingredients. Several candidates have been identified (the biogenic stimulant amines caffeine, theobromine, tyramine and phenylethylamine), but their concentrations are too low to have a significant psychoactive effect and they are also present in higher concentrations in non-craved foods (BNF Nutrition Bulletin, 1998; Hetherington and Macdiarmid, 1993; Hurst et al., 1982; Rozin et al., 1991; Weingarten and Elston, 1991; Ottley, 2000).

Comparisons of subjects ingesting milk chocolate, dark chocolate, white chocolate and cocoa powder (powdered cocoa mass with some cocoa butter extracted) have demonstrated that milk chocolate is most preferred. If psychoactive substances were involved, then cocoa powder should equally satisfy craving and dark chocolate should be the most preferred (Michener and Rozin, 1994).

Chocolate contains two analogues of anandamine similar to the cannabinoid responsible for euphoria from cannabis. However, any association with pleasure from chocolate is likely to be indirect as the analogues inhibit breakdown of *endogenously* produced anandamine (di Tomaso et al., 1996).

3. Chocolate and neurotransmitter systems

Chocolate may interact with a number of neurotransmitter systems (including dopamine, serotonin and endorphins) that contribute to appetite, reward and mood regulation. Some relevant literature is now considered.

3.1. Dopamine

The mesolimbic dopaminergic system is involved in most drugs of misuse except benzodiazepines, with increased levels of dopamine in the nucleus accumbens central to mediating the rewards of positive reinforcement (Koob and Le Moal, 2001). The positive reinforcing effects of stimulation are remembered for long periods, being underpinned by the activation of brain dopamine systems. Rewards from food and from drugs may depend on similar substrates for motivational processes (Pelchat, 2002), with the role of dopamine recognized as critical in anticipation as well as in withdrawal (Lingford-Hughes and Nutt, 2003). It has been argued that, in modern society, humans are driven to eat less by internal energy deficits and more by the anticipatory pleasure of eating (Pinel, 1990). The contribution of the dopaminergic system to chocolate craving and eating is, however, likely to be general rather than chocolate-specific.

3.2. Serotonin

The many roles of serotonin include influence over sleep, appetite, impulse control and mood elevation. A number of specific serotonergic contributions to chocolate eating have been proposed, and a link between serotonin, mood and craving has become part of the 'folklore' of the psychology of eating (Rogers and Smit, 2000). A popular hypothesis is that chocolate or carbohydrate craving addresses serotonin deficiency, particularly in depressed individuals who attempt 'self-medication'. Studies of seasonal affective disorder and atypical depression provide some support for this hypothesis (Moller, 1992; Wurtman and Wurtman, 1989). The latter authors suggested that serotonin acts through a biofeedback mechanism to regulate carbohydrate consumption. The amino acid tryptophan normally circulates in low concentrations, but when carbohydrates enter the bloodstream, they stimulate insulin production and facilitate the uptake of most amino acids, excluding tryptophan, into the tissues. The proportion of circulating tryptophan increases relative to other amino acids, facilitating its passage across the blood–brain barrier, where it is converted into serotonin. Variations in serotonin levels in turn influence carbohydrate intake. Wurtman and Wurtman (1989) hypothesized that, in some cyclic conditions such as seasonal affective disorder, there is a disturbance of the feedback mechanism between serotonin and carbohydrate intake in which the desire for carbohydrates persists. Additional consumption of

carbohydrates may be necessary to lift serotonin levels and thus mood.

If the serotonin hypothesis for carbohydrate and chocolate craving was valid and self-medication effectively corrects a serotonin deficiency, then craving would be a plausible biological response to 'depression'. However, links between depression and appetite are complex. In 'clinical depression', particularly the melancholic sub-type, many patients report decreased appetite and recovery is associated with restoration of appetite and food intake, but the converse pattern is often found in the non-melancholic disorders (Parker et al., 1994). For example, carbohydrate craving can occur in the 'atypical' depressive sub-type, with hyperphagia and weight gain being included in DSM-IV diagnostic criteria. It is also a common response to those with non-clinical depression. For example, patients routinely consulting their primary care physician (Parker and Brown, 1982) were asked to rate their likely behavioural responses to two depressogenic stimuli (a break-up of an important relationship, and increased criticism from someone important to them). Among those who predicted they would experience a depressed mood, 52% reported they would be likely to eat more and only 11% reported that they would eat less, and more thought that increased eating would be effective (33%) rather than ineffective (18%). Thus, appetite loss may be a prototypic *symptom* of some depressive disorders, while appetite increase or food craving may more be a prototypic comfort eating *response* to certain expressions of depression.

An examination of the literature (Hammersley and Reid, 1997); Rogers, 1995; Young, 1991) does indicate that the serotonin account is flawed. First, Benton and Donohoe (1999) reviewed 30 studies of blood amino acid profiles taken after subjects consumed meals varying in their carbohydrate content. Only when the protein component of the meal was less than 2% did the resulting amino acid profile favor a rise in serotonin levels in the brain. Chocolate contains 5% of its calorie content as protein (and ice cream 8%), which would be sufficient to negate any serotonin effect. Second, even extreme dietary manipulations of tryptophan result in physiological changes that are too slow to account for mood effects that are described during or soon after eating (Young et al., 1985). Third, Ottley (2000) has demonstrated that fat slows the absorption of carbohydrate, and commonly craved foods such as chocolate, ice cream and biscuits generally have a high fat content. Fourth, mood changes after carbohydrate ingestion have been reported even in the absence of changes in serotonin synthesis and release (Wurtman and Wurtman, 1989; Rosenthal et al., 1989; Cabellero et al., 1988), suggesting that other or additional mechanisms are involved.

3.3. Opioids

Heroin addicts experience sweet cravings if heroin is not available (Weiss, 1982), suggesting an overlap in the relevant neural systems (Weingarten and Elston, 1991). In addition, conditions such as pregnancy, menstrual cycling, alcoholism and eating disorders that are associated with craving involve altered endogenous opioid peptide (EOP) levels (Mercer and Holder, 1997). On such grounds, Drewnowski (1992) argued that carbohydrate craving is more strongly linked with the opioid rather than the serotonergic system, while Mercer and Holder (1997) have proposed an opioidergic theory of food cravings. Research with vertebrates has shown a relationship between central EOP activity and food intake, and argued that food cravings mediate the opioid-ingestion link.

The opioid system appears to play a role in the palatability of preferred foods (Si et al., 1986), releasing opioids such as P-endorphins as food is eaten which could enhance the pleasure of eating (Ottley, 2000). Opioids released in response to ingestion of sweet and other palatable foods (Blass, 1986; Fullerton et al., 1985) can increase central opioidergic activity, in turn stimulating the immediate release of beta-endorphin in the hypothalamus and producing an analgesic effect that is naltrexone-reversible (Blass, 1986). Similarly, sucrose immediately reduces crying in infants during a painful hospital procedure suggesting that taste, rather than post-ingestive factors, provokes release of EOP (Blass and Hoffmeyer, 1991).

Exogenous opioids generally increase food intake. The mu receptor is central in opiate addiction, but kappa receptor stimulation reduces dopamine function in the nucleus accumbens and may lead to dysphoria (Lingford-Hughes and Nutt, 2003). Injections of mu and kappa agonists may have a biphasic effect, first increasing but then decreasing feeding (Gulati et al., 1991), while low doses may increase consumption and high doses decrease consumption (Triscari et al., 1989; Ramarao and Bhargava, 1989). Together, these findings indicate interactions with other appetite-regulating processes. It has been suggested that EOP enhances dopaminergic activity in the mesolimbic pathways to alter the reward value of food (Cooper and Kirkman, 1993). Although the precise role of EOP in regulating food intake has not yet been established, it is clear it acts in concert with other neurochemicals such as serotonin,

dopamine and cholecystokinin that control eating (Cooper and Higgs, 1994).

4. From pleasure to aversion: a PET study

Small et al. (2001) undertook successive PET scans on volunteers who ate chocolate beyond satiety. Their research model held the sensory stimulus and act of eating as constant, while motivation to eat and reward value were modified by feeding. Differing structural groups were selectively recruited depending whether chocolate was eaten with pleasure and motivation (subcallosal region, caudomedial orbitofrontal cortex, insula/operculum, striatum and midbrain) as against when satiated (parahippocampal gyrus, caudolateral orbitofrontal cortex and prefrontal regions). The authors concluded that results argued for two differing motivational systems being involved — "one orchestrating approach and another avoidance behaviours".

5. Orosensory properties of chocolate and the pleasure principle

The orosensory properties of food, mediated by palatability, have a significant influence over eating. Moment-to-moment, eating is controlled predominantly by the orosensory effects that provide positive feedback, and the post-ingestive effects which provide negative feedback. Effects in the mouth are stimulatory, while the entry of food into the stomach is inhibitory. Positive feedback is 'the stimulation of eating by eating' and its strength is influenced by palatability (Yeomans, 1996), which is defined by the hedonic or affective response to the taste, flavor, aroma and texture of food (Rogers, 1990). Palatability is determined by innate responses such as inborn taste preference for sweets, fat and salt but a dislike for bitter tastes, and by learning (for example, effects on mood). Post-ingestive effects increase gradually with the amount eaten, and eating stops when they outweigh the orosensory effects. However, this satiety mechanism is not infallible, as commonly experienced by chocolate cravers attempting to impose self-restraint. Challenges of self-restraint relate to a 'moreishness' quality (i.e., a desire for more), which is a function of palatability. Craving is experienced during abstinence, but 'moreishness' is experienced while eating.

There is a well-supported view (Weingarten and Elston, 1990; Michener and Rozin, 1994; Rodin et al., 1991; Hetherington and Macdiarmid, 1993) that the orosensory properties of chocolate and the desire for sensory gratification are sufficient to explain the motivation for ingesting chocolate, and that the sight and smell of chocolate is sufficient to trigger cravings. Chocolate has a high hedonic rating, based on the set of extremely appealing sensory characteristics (as previously noted) and with an attractive aroma and unique flavour (Drewnowski and Greenwood, 1983). Its appeal can be argued on the basis of anticipatory pleasure rather than reflecting internal energy deficits (Pinel, 1990). Satiety is somewhat taste-specific. As satiety occurs for one food, the positive-incentive value and palatability of that food declines, but if a new food is offered it can promote further eating (Pinel, 1990). Experiments with rats have demonstrated that increased concentrations of sucrose result in a rise in the 'hedonic breakpoint' after which ingestion decreases. However, the hedonic responses to sweet and fat complexes of 'real foods' are interdependent, with human studies demonstrating that combining sugar with dairy cream or full-cream milk increases the hedonic rating of each (Drewnowski and Greenwood, 1983). Heightened responsiveness to increased palatability is often cited as a major factor in the development of obesity and even rats on a highly palatable high-fat or 'supermarket' diet will overeat to the point of obesity (Drewnowski and Greenwood, 1983). Chocolate provides preferred tastes and texture that have innate appeal and the combinations increase the hedonic rating, challenging the satiety ceiling that might otherwise apply.

The composite orosensory properties of chocolate outweigh more simple explanations of its role in appetite and satiety. For instance, if a caloric deficit motivates chocolate craving, then milk chocolate and white chocolate should have equal appeal, but they do not. If psychoactive substances or magnesium deficit are the basis of craving, then milk chocolate and unsweetened cocoa powder should have equal appeal, and other foods containing these substances in higher concentrations should be craved, but they are not. If the appeal is the unique sensory combination of chocolate, then chocolate is the only way to satisfy that craving (Michener and Rozin, 1994).

6. A homeostatic hypothesis

One view regards craving for chocolate and carbohydrates as a homeostatic response to dietary deficiencies. For example, chocolate craving has also been proposed as a response to a magnesium deficit. However, while a magnesium supplement has been demonstrated to reduce chocolate craving, certain foods high in magnesium are neither craved nor satisfy craving for chocolate (Michener and Rozin, 1994). Further, several

studies have failed to establish that dieters do experience any greater craving than non-dieters (Weingarten and Elston, 1991; Cohen et al., 1987; Lappalainen et al., 1990) instead, they tend to experience *less* craving.

So far, no support for the homeostatic hypothesis can be provided.

7. Emotional eating

Lyman (1982) has observed that food preference is altered across a range of mood states, with preference for 'junk food' increasing during negative mood states and preference for healthy foods increasing during positive mood states. Reviews by Ganley (1989) and Christensen (1993) detail numerous associations between multiple mood states (stress, boredom, depression, loneliness, social self-doubt, discord, frustration, anger and anxiety) and coping by emotional eating (Weintraub and Aronson, 1969; Taylor et al., 1996; Hudson and Williams, 1981; Hill et al., 1991; Rozin et al., 1991). A review by Ganley (1989) indicated that stress-associated eating (i.e., emotional eating) is more common in those who are overweight or obese but that mixed results in the research means that the direction of the association remains unclear. Clinical observation suggests that, while weight gain caused by emotional eating may exacerbate aversive mood states, the latter may trigger a cycle of further emotional eating and continued weight gain. Such associations are highly dependent on the current emotional state of the subjects. For example, a study by Ruderman (1983) demonstrated that obese women ate significantly less when highly anxious than when mildly anxious.

However, such links do not support the hypothesis that carbohydrate craving is necessarily an attempt to alleviate aversive mood states, or that all obesity is the result of emotional eating, or that stress has a unilateral relationship with food intake. In fact, the natural tendency during stress is for decreased food intake, reflecting activation of the sympathetic nervous system (Schachter et al., 1968), whereby blood is diverted from the digestive system and other systems not required for defense. A study by Stone and Brownell (1994) supported this proposition, showing that subjects were more likely to eat less in response to stress, and with the decrease in proportion to the severity of stress. Although eating when stressed or anxious is inconsistent with the intrinsic physiological stress response, it occurs in real life and is supported empirically with stress thought to disrupt restraint and post-ingestional satiety feedback. Meisel et al. (1990) showed a marked increase in body weight in female Syrian hamsters when socially stressed by being caged in groups compared those housed individually and with the additional finding of greatly enlarged adrenal glands. Morley et al. (1983) showed an increase in sucrose ingestion in response to stress when rats were subjected to a 'pinch-tail test' and suggested that endogenous opioid peptides as well as other neuropeptides played a role in the central regulation of stress-induced eating. As further evidence of the variable associations between appetite and stress, animal model and human volunteer studies of depression demonstrate decreased responsiveness to reward (sucrose solution) under conditions of chronic mild stress, but the opposite when the animals are provided with sweeter pellets (Willner et al., 1998). The latter effect could reflect intrinsic regulatory mechanisms being compromised when concentrations of certain food components are markedly higher than in nature. The high hedonic value of fat and carbohydrate concentrations in certain foods, including chocolate, may overstimulate positive feedback and, together with the learned expectations of alleviation of aversive mood, serve to override normal post-ingestive satiety feedback mechanisms.

Any attempt to study the effects on mood following ingestion of carbohydrate is confounded by a range of methodological variables. These include time in relation to eating (i.e., during, immediately after, or later), whether subjects are cravers or non-cravers, and the actual mood state (e.g., depression, anxiety, ennui). Several studies illustrate these difficulties. In one study, non-carbohydrate cravers and lean subjects experienced an increase in fatigue and sleepiness after a carbohydrate-rich snack, while obese cravers experienced improvement in mood (Lieberman et al., 1986; Spring et al., 1982/3). While Wurtman and Wurtman (1989) showed that carbohydrate consumption produced an initial temporary relief from dysphoria, Johnson and Larson (1982) found marked post-binge dysphoria following occasional and transient relief. Thayer (1987) demonstrated a longer-term effect of reduced energy, creating a cycle that contributed to the development and maintenance of depression. In a retrospective study, Hetherington and Macdiarmid (1993) reported that any mood improvement was *during* consumption only, with negative moods returning immediately after eating. In a later study, Macdiarmid and Hetherington (1995) reported that 'chocolate addicts' were more depressed than controls, and their negative mood did not improve after chocolate intake. In those two studies, chocolate eating resulted in a slight increase in contentment whilst eating chocolate, but the authors suggested that this reflected satiety rather than an

improvement in mood, as ratings of depression or feeling relaxed were not affected and, following consumption, ratings of guilt were increased. In their review, Rogers and Smit (2000) suggest that this inducement of guilt in the eating of chocolate stems from the cultural ambivalence surrounding chocolate consumption.

As a corollary, other studies have demonstrated that resisting the craving produced a more positive emotional tone (Hill et al., 1991) and that amelioration of mood was achieved by eliminating refined sucrose and caffeine (Christensen et al., 1985; Christensen et al., 1989; Kreitsch et al., 1988; Christensen and Burrows, 1990), leading to the suggestion that continued ingestion of carbohydrates may contribute to the maintenance of dysphoric mood. Finally, a review by Reid and Hammersley (1999) examining all studies on human subjects since 1983 dealing with the effects of carbohydrates on arousal, found half the studies reported some effect on alertness after consumption of a carbohydrate snack with individuals feeling relaxed or sleepy, but with no specific effect on mood.

Various psychological theories of emotional eating have been proposed. One argued for failure in satisfaction of basic needs (Timmerman and Acton, 2001), proposing that when individuals perceive life situations to be stressful they draw on self-care resources which may be internal (e.g., self-esteem) or external (e.g., social support). If needs remain unmet over a prolonged period of time, then a resource deficit exists and food consumption may be used to supplement the deficit. Another theory is that emotional eating results from confusion and apprehension in recognizing and accurately responding to emotional and visceral states related to hunger and satiety (Van Strien, 2000). A composite 'escape theory' of eating (Heatherton and Baumeister, 1991) argues that eating is an 'escape from self'. Heatherton et al. (1992) hypothesized that distress, by threatening self-image, acts as a trigger for disinhibition, motivating the individual to escape from self-awareness which is aversive when they encounter negative information about self. They avoid meaningful thought and instead, a cognitive shift redirects attention away from unpleasant thoughts about self and towards food cues in the environment. This also has the effect of disengaging restraints and inhibitions that are normally supported by mindfulness.

In summary, it appears that emotional eating fails to produce any real or lasting benefit to psychological and mood states, and that increased or repeated emotional eating may actually contribute to mood dysphoria.

8. Food craving as addiction

Food craving shares some features with drug addiction, including similar neurotransmitter substrates, and terms such as 'chocoholic' and 'sugar addiction' are in common use, but there is no consensus that food cravings qualify as an addiction. The psychoactive substances in chocolate lack such propensities, and chocolate cravers classing themselves as 'chocoholics' identify the orosensory properties as the 'addictive' factor (Hetherington and Macdiarmid, 1993). This interpretation is compatible with positive-incentive theories of acquired addiction, in which the craved substance is desired for its hedonic experience (Pinel, 1990), as opposed to abstinence models that involve a substance being physiologically required to give relief from an aversive state. Expectation models are based on learning about the positive reinforcement of the desired substance, with cravings triggered by stimuli such as the sight and smell of palatable foods which elicit desire for, and subsequent consumption of that food (Cornell et al., 1989).

The abstinence model may have some relevance to a tendency by those with low blood sugar to report irritability or poor mood while completing cognitive tasks, with such a mood state stimulating consumption of palatable high carbohydrate food that releases endorphins (Benton, 2002). Recognizing that a proportion of carbohydrate cravers report feeling 'alert' and 'calm' after eating a snack, it is reasonable to concede that physiological benefits mediated by the opioid system have relevance to some individuals and in certain circumstances.

8.1. Chocolate craving and emotional eating as separate phenomena

The literature tends to confound concepts of chocolate craving, carbohydrate craving, obesity, emotional eating and self-medication. We suggest it is useful to distinguish clearly between two separate phenomena: first, 'chocolate craving', and second, carbohydrate craving in the context of 'emotional eating'. The two phenomena can, however, co-exist in the same individual by virtue of the dual status of chocolate as being desired specifically for its unique sensory experience and being more generally craved as a carbohydrate at times of emotional eating.

There is some support for the hypothesis that each phenomenon is driven by different motivations, activating different neurotransmitters, and producing different outcomes. In essence, chocolate craving is driven by a desire for hedonic reward, and dopamine (being responsible for reward) is the predominant neurotransmitter

released after eating chocolate for the purpose of a pleasurable sensory experience. In contrast, emotional eating is characterized by carbohydrate craving and is motivated by desire for the comforting effect of opioids (endorphins) to alleviate dysphoria and other negative states.

Support for the differential role of dopamine and opioid systems in chocolate craving and emotional eating is suggested in several studies. Craving for chocolate has been shown to be more driven by a desire for the dopaminergic rewards of consumption rather than to avoid negative consequences of abstinence (Stewart et al., 1984; Wise, 1988; Cooper and Kirkman, 1993; Koob and Le Moal, 2001; Pelchat, 2002; Pinel, 1990). Drewnowski (1992) reported that infusing the opioid antagonist naloxone reduced caloric intake in binge eaters, as well as the taste preference for sweet high-fat foods such as biscuits and chocolate in both binge eaters and in controls, compared with an opioid agonist or saline infusion. The author suggested that endogenous opioid peptides may be involved in mediating taste responses and preferences, and especially for sweet, fatty foods. Benton (2002) reported that poor mood stimulates eating of palatable high carbohydrate food leading to endorphin release, and Fullerton et al. (1985) demonstrated that opioid antagonists can suppress stress-induced eating.

This differentiating model is supported by other lines of evidence. Weingarten and Elston (1991) found a majority of chocolate cravers reported there was no non-chocolate substitute when they craved chocolate, but, when craving carbohydrate, then any sweet-fat food (including chocolate) was consumed. Schuman et al., 1987 found some distinction between chocolate and sweet cravings, while Rozin et al., 1991 concluded that, although craving for chocolate is partly related to sweet craving, the major component is independent of sweet craving. Schuman et al. (1987) investigated 'chocoholics', former alcoholics (since they crave sweet food) and non-craving controls. When subjects were regrouped according to whether they 'self-medicated' or not, 32% of chocoholics were 'self-medicators' compared to 13% of the controls and 23% of the former alcoholics. Chocolate was not greatly preferred over other sweets for self-medication, even among chocoholics. Self-medication occurred most commonly in response to depression, tension and irritability, less often in response to anxiety, and least often to anger.

Chocolate produces a unique effect: when craving chocolate specifically, only chocolate will satisfy that craving. Yet, when experiencing an aversive mood state, any carbohydrate will suffice in an attempt to achieve relief, with chocolate no better than ice cream, cake or potato chips. Anecdotal reports from 'chocoholics' who are also emotional eaters indicate that, as chocoholics, they crave chocolate only. However, when feeling 'stressed', they will crave any carbohydrate, and chocolate is not preferred over any other carbohydrate. Since chocolate has the highest of hedonic ratings and produces rewarding and pleasurable feelings, it might seem reasonable to expect that these feelings would be an affective panacea. However, when experiencing an aversive mood state, the pleasurable feelings unique to the experience of eating chocolate are not what is required, and may be ineffective in ameliorating negative affective states. In such negative states, any carbohydrate may be ingested as a comfort food.

9. An evolutionary perspective

Addiction has been modeled as a cycle of progressively-increasing dysregulation of brain reward systems, resulting in compulsive use and loss of control over drug taking. According to Koob and Le Moal (2001) the normal 'limitation of reward' function fails to operate within the normal homeostatic range and forms an allostatic state (i.e., a chronic deviation of the reward set point). An evolutionary model hypothesizes that food craving, overeating and addiction emerge from dysregulation of endogenous systems whereby higher than normal concentrations of certain foods provide excessive stimulation and override regulatory feedback systems.

There are two key innate factors associated with eating that contribute to this dysregulation. First, the natural environment in early human existence was unlikely to have provided a regular and reliable food supply. To adjust to variable periods of plenty and famine, adaptive advantages would have accrued to those with flexibility in appetite and satiety mechanisms, allowing consumption of greater amounts during times of plenty and building of temporary stores of body fat to carry through times of famine (Hammersley and Reid, 1997). Individuals lacking such flexibility would have been less likely to survive. Extreme expressions of 'flexibility' would have been maladaptive. For example an obese individual would be at a disadvantage in hunting food and escaping from predators. In modern westernized society where food is readily available, such flexibility is potentially maladaptive, leading individuals to react to the stacked supermarket shelves as if it were a time of plenty and likely to be followed by famine. In the absence of famine, shopping and food intake require some degree of cognitive restraint to avoid unwanted weight gain.

Second, humans have innate taste preferences for sweet, fat and salt. This ensured that foods ingested in their natural form would provide sufficient calories and other essential nutrients. Body mechanisms that evolved for survival in a natural environment, with appetite and satiety mechanism 'calibrated' to regulate ingestion of natural foods in most individuals, are unlikely to have adapted to accommodate the higher concentrations of calories in processed food. In developed countries, such taste preferences and lack of adaptation operate in an environment with ready availability of processed foods that are calorie-supersaturated and contain high concentrations of sugar, starch, fat and salt. Processed food might also be deficient in other essential elements such as magnesium, thus failing to satisfy needs for these nutrients and craving will not be satisfied. For such cravings, chocolate offers a unique combination of sweetness, creamy texture, characteristic taste and tantalizing aroma — the perfect orosensory experience to seduce the palate.

Endogenous mechanisms that have evolved to respond to natural substances, whether external (food) or internal (neurotransmitters), can be compromised when the body is subjected to substances which are chemically similar but in more concentrated form. Just as the natural function of dopamine and opioid systems can be compromised by exogenous opiates and other illicit drugs, the natural function of appetite and satiety mechanisms can be challenged by calorie-supersaturated processed food. Rather than assume a state of addiction or presence of psychoactive chemicals, a simpler answer may be that the behaviour is either a compromise or a dysregulation of natural endogenous mechanisms. In essence, certain neurotransmitter systems are activated in dysfunctional ways by inappropriate analogues or by concentrations of the factors (including foods) which exceed the evolutionary capacity and override regulatory feedback mechanisms. Such foods do not have to be intrinsically addictive through any 'psychoactive' properties, but simply have the capacity to activate neurotransmitters in a dysfunctional way. High concentrations of sugar and fat, by virtue of their ability to release dopamine and opioids, have the potential to compromise such endogenous mechanisms. Rather than view them (and chocolate specifically) as 'substances of abuse', it might be more appropriate to consider them as abusers of endogenous mechanisms.

10. Conclusion

For most people chocolate invokes anticipatory and consummatory pleasure, and is therefore an indulgence. When taken in response to a dysphoric state as an 'emotional eating' strategy it may provide some transient 'comforting' role but it is more likely to prolong rather than abort the dysphoric mood. It is not, as some would claim, an antidepressant.

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