FOOD, MOOD AND APPETITE

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INTRODUCTION

"The carbohydrate cravers were significantly less depressed after snacking, whereas noncravers experienced fatigue and sleepiness. These findings suggest that carbohydrate cravers may eat snacks high in carbohydrates in order to restore flagging vitality, much as some people will pour another cup of coffee when they feel that their energy level or attention span is flagging." (Wurtman & Wurtman, 1989, p. 53).

"...just as foods determine our moods so do our moods determine what we eat." (Lyman, 1989, p. 44).

As the title of this review and these quotes suggest, the link between eating and mood includes both effects of foods and drinks on mood and influences of mood on eating and drinking. The purpose of the review is to evaluate current knowledge of these inter-relationships. In the space available it is not possible to cover this subject exhaustively, but particular attention will be paid to the mechanisms by which eating can affect mood and *vice versa*, and to likely directions for future research.

MOOD, EMOTION AND AROUSAL

According to Schachter's (1971) theory of emotion there are two components to any affective state, namely the state of arousal of the individual experiencing the emotion and the cognitions surrounding the emotion state. The cognitive component determines how the change in arousal level is interpreted, and there is little psychological differentiation between emotion states. Although this view has been extensively modified in subsequent work, changes in arousal form an important component of most models of mood and emotion.

It is not always possible to make a clear distinction between mood and emotion. Mood states are usually regarded as lasting for at least several minutes and being more diffuse than emotional reactions generated by specific cognitive evaluations. In addition, moods may lack reference to specific objects or situations, and there appear to be relatively few fundamental moods.

Investigations of the psychometric structure of mood have suggested dimensions variously related to energy, tension, hedonic tone (pleasure–displeasure) and perhaps also general arousal (Matthews *et al.* 1990). In psychological research mood is usually assessed by adjective checklists on which the respondent rates how well the mood descriptors apply to their present mood, or even their mood 'during the past week' (Rogers *et al.* 1994*b*).

A close relationship between mood and arousal is integral to Thayer's (1989) recent conceptualization of mood, which proposes the existence of two arousal systems termed energetic and tense arousal. The energetic arousal system regulates the sleep–wakefulness cycle, and appears to be driven largely by endogenous factors according to a circadian rhythm, with peaks in energetic arousal occurring normally during the late morning and more variably in the late afternoon. However, external events, physical exercise, food constituents and pharmacological agents can also strongly influence energetic moods. The tense arousal system is normally activated when some real or imagined danger is present, and functions to prepare the organism to make the appropriate responses. This includes preparation for 'fight or flight' as well as the regulation of restraint and inhibition. The energetic and tense arousal systems also interact so that, for example, a moderate amount of tension can raise feelings of energy, whereas high levels of tension can reduce energy. On the other hand, increasing energy can often have a tension reducing effect. In addition, it is argued that energetic and tense arousal are at the core of many other commonly identified moods. So, for example, low energy and high tension are basic components of agitated

depression, and high energy and low tension are associated closely with optimism, happiness and pleasurable bodily feelings.

Most of the evidence supporting this model comes from studies on self reports of mood related bodily sensations and subjective feelings. Energetic arousal is recognizable by subjective feelings of energy, vigour and liveliness ('calm-energy'), and tense arousal by feelings of tension, anxiety and fearfulness ('tense-energy'). Nonetheless, energetic and tense arousal are presumably associated with different patterns of physiological and neural activity, and therefore might be identified by, for example, neurochemical or electrophysiological analysis (Thayer, 1989). This in turn is one way in which food may influence mood. Thus a recurring theme of the present review is the effects of dietary constituents on particular neurotransmitter systems and the involvement of those same neurotransmitters in the regulation of mood and appetite.

EFFECTS OF DIETARY CONSTITUENTS ON MOOD: SOME EXAMPLES

CARBOHYDRATE *VERSUS* PROTEIN

Meal composition, serotonin and mood

The most prominent and extensively tested idea concerning effects of food on mood is the hypothesis proposed by R. J. Wurtman and colleagues linking carbohydrate and protein intake, brain serotonergic (5-hydroxytryptamine, 5-HT) function, and mood and behaviour. This was based on the results of studies carried out mainly on rats (e.g. Fernstrom & Wurtman, 1971*a*, *b*, 1972; reviewed by Wurtman *et al.* 1981). In outline the hypothesis is as follows. Consumption of a high carbohydrate meal increases the ratio of the plasma concentration of tryptophan relative to the other 'large neutral amino acids', e.g. tyrosine, phenylalanine, leucine, isoleucine and valine (Trp:ΣLNAA). This occurs because insulin released in response to the carbohydrate load facilitates the uptake of most amino acids, but not tryptophan, into peripheral tissues such as muscle. Uniquely for amino acids, tryptophan is bound to albumin in the blood stream, and the affinity of albumin for tryptophan actually increases in response to insulin as free fatty acids (FFA) are stripped off the circulating albumin (owing to the effect of insulin on the removal of FFA from the circulation). Tryptophan is the precursor of the neurotransmitter serotonin and, since tryptophan and the other large neutral amino acids compete for entry into the brain and the rate limiting enzyme for serotonin production (tryptophan hydroxylase) is not fully saturated with substrate under normal conditions, an increase in plasma Trp:ΣLNAA concentration leads to an increase in brain serotonin synthesis and, in turn, to increased serotonergic neurotransmission. In contrast, consumption of a meal high in protein can be expected to have the opposite effect, primarily because most dietary proteins contain relatively little tryptophan (Wurtman *et al.* 1981).

The behavioural consequences which have been predicted to follow from these diet induced changes in neurotransmission include altered food choice and food intake, and changes in pain sensitivity, aggressiveness, mood, alertness and cognitive performance. Specifically, the relative increase in brain serotonergic activity supposedly occurring after consumption of a high carbohydrate *v.* a high protein meal is hypothesized to give rise to, for example, decreased alertness, and consequently a decline in performance efficiency (see Spring *et al.* 1987; Young, 1991, for reviews). Interestingly, this contradicts the popular notion that consumption of carbohydrates, especially sugar, will normally have an energizing effect.

The evidence for such effects is mixed. Several studies found differences in the effects of

high carbohydrate and high protein meals in the direction of greater drowsiness, sleepiness and calmness after carbohydrate (Spring *et al.* 1987), but these were not consistent across all subject groups, and a majority of mood and performance measures were unaffected by meal composition. Also, more recent studies (Deijen *et al.* 1989; Christensen & Redig, 1993) failed to show any definite carbohydrate *v.* protein effects on mood, despite confirmation in the latter study of significant effects on Trp:ΣLNAA concentrations. Pivonka & Grunewald (1990) found that a sugar sweetened drink increased sleepiness and decreased alertness compared with the effects of the same drink sweetened with aspartame or the same volume of water. These differences emerged about 30 min after the drinks were consumed. However, other similar studies have not revealed such clear results (e.g. Brody & Wolitzky, 1983), and these experiments did not, of course, test the effects of protein or other nutrient loads. This is also true of a larger number of studies which have examined the effects of oral glucose loads on various aspects of cognitive performance. Nonetheless, contrary to the prediction of the carbohydrate–serotonin hypothesis, performance efficiency is, if anything, improved after consuming glucose compared with a non-nutrient-containing control drink (reviewed by Rogers & Lloyd, 1994).

Carbohydrates and depression

In addition to these effects of meal composition on alertness, Wurtman & Wurtman (1989) have developed a further hypothesis which proposes that carbohydrates can relieve depression. The hypothesis relates specifically to three disorders, carbohydrate craving obesity (CCO), premenstrual syndrome (PMS) and seasonal affective disorder (SAD), the characteristics of which include depressed mood and supposedly a craving for and increased intake of high carbohydrate foods. Together with evidence implicating deficient serotonergic function in the aetiology of depression (e.g. Cowen *et al.* 1992; Møller, 1992; Maes & Meltzer, 1995), this has led to the suggestion that the increase in carbohydrate intake constitutes self medication to relieve the depression (see below for further discussion).

The prediction is, therefore, that consumption of a high carbohydrate, low protein meal should have different effects in depressed and non-depressed individuals. Relatively few studies have tested this directly, although consistently carbohydrate has been found to be significantly less sedating in CCO, SAD and PMS subjects compared with controls (Lieberman *et al.* 1986*b*; Rosenthal *et al.* 1989; Wurtman *et al.* 1989). Lieberman *et al.* (1986*b*) also demonstrated the expected effects on depressed mood in CCO. In PMS subjects a carbohydrate meal was found markedly to improve several aspects of mood, including tension, anger, depression and confusion, all of which were unaffected in control subjects (Wurtman *et al.* 1989). Both these latter studies, however, failed to establish whether the effects on mood were specific to carbohydrate, because the response to other meals (e.g. high in fat or protein) was not tested.

In a study of spontaneous meal patterns, de Castro (1987) found a significant negative correlation between self reported depression and the proportion of carbohydrate (% energy) consumed over a 9 day period. In addition, depression was positively correlated with proportionate protein intake, and feelings of energy were positively correlated with proportionate carbohydrate intake. These relationships, however, were not apparent in an analysis of meal to meal mood and food intake. Nor is the direction of the effects as predicted by Wurtman and colleagues, since the subjects were not depressed to begin with. High carbohydrate, low protein intake is supposed to increase somnolence, but not affect depression, in non-depressed individuals.

Dietary effects on plasma amino acid ratios and serotonergic function in human subjects

Another test of the carbohydrate–serotonin hypothesis is to measure the effects of meal composition on physiological variables. As predicted, studies on human subjects have shown statistically significant differences in plasma Trp:ΣLNAA concentrations following meals with a very high carbohydrate *v.* those with a high protein content (e.g. Ashley *et al.* 1985; Lieberman *et al.* 1986*a*: Teff *et al.* 1989*a*; Christensen & Redig, 1993). Similar effects have been confirmed in SAD and obese subjects (Rosenthal *et al.* 1989; Pijl *et al.* 1993). The practical significance of these results, however, is uncertain. In particular, it appears that the magnitude of the effects is probably too small to produce functionally significant changes in brain serotonergic activity (Ashley *et al.* 1985; Leathwood, 1987; Young, 1991). The actual changes observed were, for example, small increases in Trp:ΣLNAA plasma concentrations following high starch and high sugar meals and a larger decrease in this ratio after high protein meals. Differences in Trp:ΣLNAA concentrations did not reach their maximum until 2–3 h after these meals, with almost no change occurring within the first hour after a carbohydrate meal (e.g. Lieberman *et al.* 1986*a*; Rosenthal *et al.* 1989). Furthermore, the range in macronutrient compositions of meals typical of most diets is unlikely to produce any very distinct differences in post-prandial plasma amino acid ratios, because the presence of a small amount of protein (perhaps as little as 4% energy as protein) in a high carbohydrate meal is sufficient to block any meal induced increases in the Trp:ΣLNAA concentration (Teff *et al.* 1989*a*, see also Wurtman *et al.* 1981).

Even more striking are the results of a study by Teff *et al.* (1989*b*) in which samples of cerebrospinal fluid were collected from human subjects 2·5 h after they had consumed either 100 g carbohydrate, a high protein load, or water. Compared with the effects of water, the nutrient loads did not alter the cerebrospinal fluid concentrations of either tryptophan or the serotonin metabolite 5-hydroxyindoleacetic acid. On the other hand, plasma Trp:ΣLNAA concentrations were affected in a similar manner to previous studies. This result would therefore support the view that in human beings differences in meal macronutrient composition do not alter plasma amino acid ratios sufficiently to cause appreciable changes in brain tryptophan levels or serotonin synthesis (but see Fernstrom, 1994).

On the other hand, there is still considerable interest in possible dietary influences on brain serotonergic function. In particular, it is likely that the relationship between diet and depression will continue to be investigated. For example, mechanisms have been suggested linking cholesterol lowering diets, brain serotonin and behaviour which, if confirmed, could have very significant epidemiological implications (Muldoon *et al.* 1991; Engelberg, 1992; Kaplan *et al.* 1994). Furthermore, dieting (i.e. energy restriction) has been shown to affect both plasma Trp:ΣLNAA concentrations and an indirect measure of serotonergic function in human subjects (Goodwin *et al.* 1987, 1990) and another study found significant correlations between carbohydrate intake, Trp:ΣLNAA concentrations and mood during weight reducing diets (Schweiger *et al.* 1986). These observations may help to explain, at least in part, some of the psychological consequences of dieting, including the association of weight loss with depression (Cowen *et al.* 1992) and the finding of an impairment in cognitive performance during dieting (Green *et al.* 1994; Green & Rogers, 1995).

Finally, Newsholme and colleagues (Newsholme *et al.* 1992; Hassmén *et al.* 1994) have argued that increased brain serotonin is a factor contributing to the development of mental fatigue during sustained exercise. The relevance of this to the present review is that the mechanism proposed involves an increase in plasma FFA concentration during exercise, which raises the free plasma concentration of tryptophan, causing in turn increased entry of tryptophan into the brain and increased brain serotonin activity. This differs from the

mechanism suggested by Fernstrom and Wurtman which depends on effects on the total plasma concentration of tryptophan, where a large percentage of tryptophan is normally bound to albumin (Wurtman *et al.* 1981).

TRYPTOPHAN

The influence of tryptophan on mood and behaviour is of interest because this can provide a test of the limits of the diet–serotonin hypothesis (see previous section). According to this hypothesis, administering tryptophan alone or in combination with carbohydrate will be expected to have substantial effects on plasma Trp:ΣLNAA concentrations, thereby altering brain serotonergic function and behaviour. Confirmation of such effects on amino acid ratios, mood and behaviour has come from a variety of studies (reviewed by Young, 1986; Spring *et al.* 1987; Hill & Blundell, 1988; Steinberg *et al.* 1992). For example, in adults doses of tryptophan ranging from 1·5 to 5 g have been found to induce feelings of drowsiness and fatigue, to decrease sleep latency, and to decrease appetite including rated hunger and actual food intake.

Tryptophan can also be an effective antidepressant, both when given alone and in combination with other treatments. Mildly or moderately depressed people appear to benefit most from treatment with tryptophan, although it is less potent than standard antidepressant drugs (Young, 1986). This would be consistent with the view that a deficit in serotonergic activity is important as a vulnerability factor, but is not the proximate cause of depression (Maes & Meltzer, 1995). In relation specifically to PMS, a preliminarily study has shown marked improvement in symptoms, including negative mood, in women taking 6 g tryptophan daily during the premenstrual and menstrual phase of their menstrual cycle. These subjects were recruited from a premenstrual syndrome clinic and met the proposed standard diagnostic criteria for 'late luteal phase disorder' (Steinberg *et al.* 1994). Tryptophan has also been found to improve depressive symptoms in SAD (McGrath *et al.* 1990).

Another strategy has been to study the effects of administering mixtures of amino acids devoid of tryptophan. In a seminal study Young *et al.* (1985) fed subjects either a balanced amino acid mixture, or amino acid mixtures that were either tryptophan free or tryptophan supplemented. One measure of mood was based on distractibility during a proofreading task. The subjects performed this task while listening to tapes of varying emotional content over headphones. They were tested under no distraction, low distraction (readings from a statistics textbook), high distraction (eyewitness accounts of the bombing of Hiroshima) and dysphoric distraction (themes of hopelessness and helplessness). In the tryptophan depleted group proofreading performance was significantly worse with the dysphoric distracter than with the low distracter. On the assumption that individuals who are depressed will be more distracted by dysphoric themes than people who are not depressed, this result supports the prediction that the tryptophan free mixture would depress mood, which was further confirmed in standard self report measures of mood. Subsequent results have shown that lowering of mood following tryptophan depletion is most likely to occur in subjects with high baseline depression scores or in subjects with a family history of depression (reviewed by Young, 1991, 1993; Benkelfat *et al.* 1994).

Taken together, these studies of tryptophan supplementation or depletion add significantly to the evidence indicating a role for serotonin in the aetiology of depression. They also confirm that everyday variations in protein and carbohydrate intake have relatively very small effects on plasma amino acid ratios, and therefore presumably will have little impact on serotonergic function.

This conclusion does not, of course, rule out the possibility that carbohydrate and

protein might exert effects on mood and behaviour *via* other mechanisms. One suggestion is that the elevation of plasma tyrosine in relation to other LNAA after a high protein meal might influence the synthesis of the catecholaminergic neurotransmitters dopamine and noradrenaline (e.g. Spring, 1986). Tyrosine and phenylalanine are precursors of these neurotransmitters. Tyrosine has been shown to improve depressed mood (van Praag, 1990) but, as with tryptophan, the effects of normal diets are probably too small to be of practical significance. On the other hand, meals could influence brain functioning through effects on gut hormones released in response to eating (Young, 1993; and see next section). These are known to act both directly and indirectly, *via* the vagus nerve, on the brain, and may, for example, mediate the potent effects on satiety of orally administered aspartame (a dipeptide of phenylalanine and aspartic acid) and phenylalanine itself (Rogers *et al.* 1991; Rogers & Blundell, 1994).

CARBOHYDRATE *VERSUS* FAT

To date there have been relatively few studies which have investigated the possible psychoactive effects of dietary fat. This is of interest, however, because of Governmental recommendations to reduce fat and increase carbohydrate intake (e.g. Department of Health, 1992), and also because of concern about possible adverse behavioural consequences of consuming low fat, cholesterol lowering diets (e.g. Muldoon *et al.* 1991).

Recently, we have examined the acute effects of manipulating the fat:carbohydrate ratio of individual meals. Subjects' mood and cognitive performance were measured following their consumption of isoenergetic meals, similar in appearance and orosensory properties, but varying in fat and carbohydrate content (e.g. 27 and 62%, 44 and 47%, and 56 and 34% energy as fat and carbohydrate). In the first study, simple reaction time and mood were found to be better (e.g. less drowsy and muddled) in the afternoon following a medium fat medium carbohydrate lunch compared with either a high or low fat lunch (Lloyd *et al.* 1994). In contrast, in the second study morning mood was found to be relatively improved following a low fat high carbohydrate breakfast compared with either a medium or high fat breakfast (e.g. less dejected, drowsy, muddled) (Lloyd & Rogers, 1994). Many of these effects were apparent within 30 min of eating and, perhaps most significantly, in both studies optimal mood and performance were associated with the meal which was closest in macronutrient composition to the subjects' typical intake at that particular time of day.

This is likely to have implications for the immediate effects of making dietary changes. For example, preliminary evidence from a longer term study indicates that changing to a low fat diet (in the absence of weight loss) was associated initially with increased tiredness, even though after several weeks mood was actually improved overall (H. M. Lloyd and P. J. Rogers, unpublished observations). This is perhaps consistent with the adaptation of gastrointestinal and other physiological responses to food known to occur as a result of medium to longer term changes in diet composition (e.g. Cunningham *et al.* 1991; French *et al.* 1995).

The appearance of certain mood and performance effects within half an hour of eating suggests that preabsorptive and/or early postabsorptive mechanisms are implicated. At present, however, the nature of these mechanisms is unclear, although one study has demonstrated substantial, dose-related effects of intravenously administered cholecystokinin on alertness and performance (Stacher *et al.* 1979): subjects felt more relaxed, drowsy, sluggish and inert with increasing doses of the hormone. This is of particular interest, because fat and protein are more potent releasers of cholecystokinin than carbohydrate (Liddle *et al.* 1983). Therefore cholecystokinin may provide part of a mechanism mediating effects of meal composition.

CHOCOLATE

Chocolate is regarded as highly palatable, and in Britain is probably the most discussed and written about of all foods. The question of why people like, consume and crave chocolate has also been addressed in the scientific literature; much of this work has been largely speculative and has contained relatively little actual empirical research. It is clear, though, that eating chocolate can have significant influences on mood, generally leading to an increase in pleasant feelings and a reduction in tension, although increased guilt may be a penalty for some individuals (e.g. Hill *et al.* 1991; Hill & Heaton-Brown, 1994; Macdiarmid & Hetherington, 1995). Also, craving for various foods, including chocolate, is associated with negative moods (e.g. boredom, tension, anger, depression and tiredness) (Schlundt *et al.* 1993; Rogers *et al.* 1994*a*).

A recurring issue concerns the notion of chocolate addiction and the effects of potentially psychoactive constituents of chocolate. Of all foods, chocolate is certainly the most frequently craved, especially among women (Weingarten & Elston, 1991; Rodin *et al.* 1991). However, in itself this and other circumstantial evidence, for example the existence of groups such as Chocoholics Anonymous, is not sufficient to demonstrate addiction to chocolate (Rogers, 1994; and see below for further discussion). Indeed, it is obvious that very many people eat chocolate regularly without becoming addicted.

Serious reviews have generally found little or no support for the suggestion that the liking for chocolate is related to the presence of psychoactive constituents (Tarka, 1982; Max, 1989; Rozin *et al.* 1991). For example, although chocolate can contain relatively high concentrations of theobromine, this is a relatively weak central nervous system stimulant and does not have strong subjective effects (Mumford *et al.* 1994). The related methylxanthine, caffeine, is also found in chocolate but in much lower concentrations. Compared with coffee and tea, chocolate is an insignificant source of dietary caffeine (Gilbert, 1984). There also appears to be a lack of any generally significant relationship between reported chocolate craving and the liking and consumption of other xanthine-containing substances (Rozin *et al.* 1991). Other substances present in chocolate which have been discussed as potentially significant pharmacologically include histamine, serotonin, tryptophan, phenylethylamine, tyramine, salsolinol and magnesium (Cockcroft, 1993; Michener & Rozin, 1994), though many of these exist in higher concentrations in other foods with less appeal than chocolate (Robinson & Ferguson, 1992). The same is true for certain bioactive peptides, such as casomorphins which can act as opioid agonists and occur in a variety of foods (e.g. milk and gluten). These peptides have been shown to influence intestinal function and some evidence indicates that they might be absorbed and then affect the central nervous system (Morley *et al.* 1983; Gardner, 1984; Meisel & Schlimme, 1990).

Unfortunately, very few studies have attempted to investigate directly the psychoactive effects of these various substances administered orally, either alone or in combination, in amounts relevant to dietary intakes of chocolate. A notable exception is a recent report by Michener & Rozin (1994), who provided chocolate cravers with sealed boxes containing either a milk chocolate bar, a bar of white chocolate, capsules containing cocoa (and therefore many of the presumed psychoactive ingredients of chocolate), placebo capsules, white chocolate plus cocoa capsules, or nothing. The subjects consumed, in random order, the contents of one of these boxes when they experienced a craving for chocolate, and they rated the intensity of their chocolate craving just before, just after and 90 min after this. The results showed that only consumption of chocolate itself, either white or brown, substantially reduced the craving, suggesting that there is "no role for pharmacological effects in the satisfaction of chocolate craving" (p. 419). As the authors themselves point out, this cannot be a definite conclusion because the subjects may have had different

expectations for the effects of the substances they were asked to consume, nor does it exclude the possibility that pharmacological reinforcement is a significant factor influencing the acquisition of liking for chocolate (Michener & Rozin, 1994). Nevertheless, it appears highly probable that liking for chocolate and its effects on mood are due largely to its principal constituents sugar and fat, and their related sensory and physiological effects (Rogers, 1994).

SWEETNESS

Certain taste preferences appear to be present at birth. The facial expressions of human neonates indicate acceptance and a positive hedonic response to sweet stimuli, while bitter stimuli evoke rejection coupled with negative expressions (Steiner, 1987). These innate biases could help promote adaptive food selection, because for instance bitter tastes tend to be correlated in nature with the presence of toxins (e.g. alkaloids in plants), and sweet tastes will normally signal a ready source of energy in the form of sugars. Perhaps even more significant, the almost universally enthusiastic response to sweetness may be an important factor facilitating the infant's early acceptance of its mother's milk (Blass, 1991; Beauchamp, 1994).

Indeed, the powerful action of sweet taste has been demonstrated in a series of elegant studies by Blass and his colleagues (Smith *et al.* 1990; Blass, 1991; Blass & Hoffmeyer, 1991). In one experiment 2 or 3-day-old infants were given 2 ml 12% sucrose solution to drink immediately prior to blood collection *via* a heel lance. Compared with the same volume of water, the sucrose substantially reduced the initial amount and duration of crying in response to the blood collection procedure. Sweet taste rather than a direct nutritional effect was implicated, since the response was very rapid and the small volume of sucrose was presumably nutritionally insignificant. These observations have been confirmed and extended in subsequent work (Barr *et al.* 1994; Miller *et al.* 1994). In the latter case the cold pressor test was used (in which one arm is immersed in cold water) and analgesic effects of oral sucrose were found in 8–11-year-old children. Similar studies in the rat have indicated that this antinociceptive and/or calming property of sucrose, and possibly other food materials including fats, is mediated by the activation of central opioid mechanisms (Blass *et al.* 1987; Blass, 1991).

Following on from this there is growing evidence that opioid peptides are intimately involved in the mediation of hedonic responses to orosensory stimuli (Kirkham & Cooper, 1991). For example, studies on adult human subjects indicate that the opioid antagonist naloxone decreases taste preferences for sugar–fat mixtures and decreases the consumption of both sweet high carbohydrate and sweet high fat foods in a specific manner (Drewnowski, 1992).

In sum, this evidence shows that sweetness can have marked influences on eating, mood and other behavioural responses independently of any later postingestive effects of the food or drink.

CAFFEINE

The main sources of the caffeine consumed by human beings are coffee and tea, and it has often been pointed out that caffeine is the most widely used psychoactive substance in the world. This is based on an estimated global consumption of 120000 tonnes of caffeine per year (Gilbert, 1984). Accordingly, there is a very large amount of research available on the effects of caffeine on body and mind – one recent review of the central nervous system effects of caffeine cites 656 references (Nehlig *et al.* 1992). It is now generally agreed that

the primary physiological action of caffeine appears to be the blockade of adenosine receptors (James, 1991; Nehlig *et al.* 1992).

Certain aspects of the psychoactive effects of caffeine are also well established. Most of these effects are consistent with a psychostimulant action; for example, caffeine has been found to quicken reaction time, improve vigilance and concentration, and increase feelings of alertness and energy (reviewed by James, 1991). However, other behavioural effects of caffeine are less desirable, including increased anxiety at higher doses or in certain vulnerable individuals, and decreased hand steadiness (James, 1991). Furthermore, with sustained exposure, tolerance develops to at least some of the effects of caffeine, and in many regular users cessation of caffeine consumption is followed temporarily by adverse changes such as increased incidence of headache, drowsiness and fatigue (Griffiths & Woodson, 1988; van Dusseldorp & Katan, 1990; Silverman *et al.* 1992).

In one recent study, for example, we compared morning mood in caffeine users and non-users (Richardson *et al.* 1995). The group of users was divided into 3 matched subgroups who avoided all significant sources of caffeine for either 1·5 h, 13 h, or at least 7 days before testing. Two patterns were apparent in the results. First, the overnight (13 h) deprived group showed markedly increased levels of tiredness and drowsiness, and were more angry and dejected compared with all of the other groups, who did not differ significantly in these moods. The second pattern was less definite, but tended to be characterized by poorer mood (e.g. decreased clearheadedness and cheerfulness) and increased headache in both the 13 h and 7 day groups.

These results show clearly that overnight caffeine deprivation is sufficient to induce significant negative effects, including tiredness, headache and depressed mood. Although it is possible that pre-existing differences in personality or other factors unaffected by caffeine use could explain differences in mood between users and non-users (Smith *et al.* 1991; Rogers *et al.* 1995), this study also found that increased tiredness, drowsiness, anger and dejection were present in caffeine users after overnight caffeine deprivation but not after prolonged deprivation. In other words, the only factor which can reasonably account for the presence or absence of these particular symptoms is the subjects' recent history of caffeine consumption. Similar findings from a smaller study were reported by Bruce *et al.* (1991), who found increased tiredness in 24 h compared with 7 day caffeine deprived subjects. Although non-users were not tested, a further result was that high doses (250 or 500 mg) of caffeine reduced tiredness, and also headache, only in the 24 h group.

Such findings illustrate the difficulty of determining the net effects of caffeine. In a typical experiment most if not all of the subjects have a history of regular caffeine consumption, and they are tested on caffeine and a placebo after a period of caffeine deprivation (usually no longer than 24 h). The problem with relying solely on this approach is that it leaves open the question as to whether the results obtained are due to beneficial effects of caffeine or to deleterious effects of caffeine deprivation (or a combination of both of these) (James, 1991; Rogers *et al.* 1995).

Nonetheless, some results do point to an actual benefit associated with caffeine use. Jarvis (1993), for example, recently reported results showing a strong positive dose–response relationship between habitual caffeine intake and psychomotor performance. This relationship remained even 'after controlling extensively for potential confounding variables' (p. 45). Consistent with this, we found that high users of caffeine given caffeine responded significantly faster throughout a long duration simple reaction time task than did low users. The low users' performance was unaffected by caffeine (Rogers *et al.* 1995). Finally, some studies have found psychomotor performance enhancing effects of caffeine in subjects deprived of caffeine for only 3–4 h (e.g. Smith *et al.* 1994), where deprivation effects are likely to be minimal (Rogers *et al.* 1995).

ALCOHOL

The effects of alcohol depend on many factors including dose, prior experience of alcohol, social context, and the expectations of the drinker. In general terms, alcohol acts as a central nervous system depressant, and in low to moderate amounts can have anti-anxiety and euphorigenic effects (Lowe, 1990; Finnigan & Hammersley, 1992). For example, we found that 8 g, but not 24 g, of alcohol administered double blind significantly improved mood (e.g. less tense and less uncertain) compared with a placebo drink (Lloyd & Rogers, 1995). This occurred in a context where subjects were required to perform a repeated series of cognitive tasks and their performance, especially detection rate on a rapid information processing task, was also improved by the low dose of alcohol. Eight grams of alcohol is equivalent to the alcohol content of half a pint of beer. Similar results have been observed in a variety of previous studies. However, it is also well established that alcohol can impair performance, and this is implicated, together with other behavioural effects, in increased accident rates and antisocial behaviour associated with alcohol (Steele & Josephs, 1990; Finnigan & Hammersley, 1992).

One attempt to understand the diverse effects of alcohol on mood and behaviour has led to the proposal that alcohol produces a specific impairment of perception and thought which Steele & Josephs (1990) call 'alcohol myopia'. This refers to the idea that alcohol increases the tendency for people to attend to immediate, proximal information, while reducing attention to information more distant in space, time and concept. This can explain how alcohol makes social responses more extreme, enhances important self evaluations, and relieves anxiety and depression. On the other hand, it is not clear that the concept of alcohol myopia can be related directly to the relatively diffuse effects of alcohol on the brain (Finnigan & Hammersley, 1992). In terms of the reinforcing and motivational properties of alcohol, these effects would appear to involve actions *inter alia* on dopaminergic, serotonergic and opioid systems (Koob, 1992; O'Brien *et al.* 1995).

HOW MOOD AND THE MOOD EFFECTS OF FOOD CAN INFLUENCE APPETITE

BELIEFS ABOUT THE MOOD EFFECTS OF FOODS AND DRINKS

Knowledge of the supposed psychoactive effects of a food or drink will, at least on certain occasions, influence a person's decision to consume that food or drink. For instance, some readers of this review might decide to alter their dietary habits based on the evidence presented of the mental effects of consuming carbohydrates, chocolate or tryptophan. Advocates of such an approach to food choice can be found in a variety of popular articles and books (e.g. 'Managing Your Mind and Mood Through Food', Wurtman & Danbrot, 1988). Perhaps not surprisingly, though, these accounts tend to overexaggerate the benefits that are likely to be gained from following their advice.

Similarly, the recognition of the psychostimulant effects of caffeine may lead the coffee drinker to consume strong coffee at breakfast for its expected alerting effects, but to avoid coffee late in the evening because of the expectation that consumption of caffeine will lead to difficulty in getting to sleep. Indeed, individuals may learn to adjust their caffeine intake very precisely to suit particular circumstances and their own sensitivity to caffeine (Booth *et al.* 1992; Rogers & Richardson, 1993). For example, we have found a positive correlation between caffeine intake and scores on the impulsivity subscale of the Eysenck Personality Inventory (Rogers *et al.* 1995). Highly impulsive individuals are characterized as having relatively low levels of endogenous arousal and they also appear to benefit more from the performance enhancing effects of caffeine (e.g. Smith *et al.* 1991). The benefit or otherwise

of consuming caffeine will probably also influence the actual liking for coffee and tea (see next section).

Closely related to these ideas is the design and promotion of products based on claims of specific functional effects. There is growing interest in this area from the food and drinks industry, with some notable market successes including sports drinks (Leatherhead Food RA, 1994). Other more esoteric products have been promoted with claims specifically promising psychoactive effects. For example, Life Force Trading have advertised soft drinks which "allow you to take control of your own neurochemistry". These products included "Rise & Shine... contains L-phenylalanine as its main ingredient... a powerful natural psycho-stimulant", "Fast Blast... should be used when energy and alertness are at a premium... contains more L-phenylalanine than Rise & Shine and caffeine to aid the stimulating effect", and "Gourmet Choline Cooler... can be used as a tool for cognitive enhancement. Choline becomes metabolized into the neurotransmitter acetycholine, the brain chemical which plays a major role in the function of memory".

Currently, however, the idea of developing foods and drinks to be sold primarily for their beneficial effects on mood or mental performance has not been widely exploited in the UK. If this were to occur, clearly one issue would be whether the claims made could be supported by evidence from controlled studies. For example, it cannot be assumed that inclusion of a neurotransmitter precursor in the formulation of a food or drink, even in relatively high concentrations, will necessarily endow that product with mood altering properties.

LEARNED PREFERENCES REINFORCED BY THE PSYCHOACTIVE EFFECTS OF DIETARY CONSTITUENTS

As suggested above, coffee and tea drinking are to some extent motivated by belief in the beneficial psychoactive effects of caffeine (Rogers & Richardson, 1993). However, if people are asked why they drink coffee and tea, they are more likely to say that this is because they like the taste of the drink. Typically, it is not the case that people consume coffee as if it were a medicine, being prepared to tolerate its taste in the expectation of a benefit. On the other hand, it is fairly certain that human beings are not born with a liking for the taste and flavour of either coffee or tea, at least partly because these drinks contain bitter constituents (including caffeine) and bitterness is innately aversive (Cines & Rozin, 1982). This then raises the question of how people come to acquire a liking for the sensory qualities of these drinks.

One way in which preferences are modified is through the association of the orosensory and postingestive effects of eating and drinking. The most dramatic example of this is the strong and specific aversions which can develop when consumption of a food is followed by gastrointestinal illness (Garcia *et al.* 1974). Similarly, there is now good evidence that association of a taste or flavour paired with positive postingestive consequences can result in increased preference for that specific taste or flavour (e.g. Booth, 1978; Elizalde & Sclafani, 1990; Sclafani, 1990). For example, a small number of studies on human subjects has demonstrated conditioned increases in preference in children and adults for flavours associated with high carbohydrate content (Booth *et al.* 1982; Birch *et al.* 1990) and high fat content (Johnson *et al.* 1991; Kern *et al.* 1993). These changes appear to persist over time and also tend to be state dependent, such that preference is more readily modified if initial exposure occurs during a state of hunger, and is subsequently expressed more strongly when the individual is hungry compared with when they have recently eaten. Another feature of conditioned preferences and aversions is that they appear to be characterized by a changed actual liking for the food or drink. For instance, after aversive

conditioning the food tastes unpleasant, it is not simply that it is avoided because it is expected to cause harm (Booth, 1978).[1]

Studies on conditioned preferences have demonstrated the existence of a capacity to adapt preference for a food according to the benefit or otherwise derived from consuming that food. Not surprisingly, the main focus of this research has been on the nutritional effects (e.g. high *v.* low energy density) of eating and drinking. However, as shown by the previous sections of this review, certain constituents of foods and drinks can also have pharmacological activity and significant effects on mood. Caffeine and alcohol are the most obvious examples. Perhaps, therefore, preferences for coffee, tea, beer, wine, etc. are reinforced by the psychoactive effects of caffeine and alcohol (see also Cines & Rozin, 1982; Zellner, 1991; Rogers & Richardson, 1993).

Recently, we attempted to test this suggestion directly in studies in which caffeine ingestion was paired with the consumption of novel-flavoured fruit juices. Caffeine was given either in the drink or in a capsule swallowed with the drink. A drink of a different flavour was given without caffeine or with a placebo capsule containing a non-pharmacologically active substance such as cornflour. The design of these studies is, therefore, similar in principle to methods used in the work on flavour preferences conditioned by nutrient manipulations, and the straightforward prediction is that if caffeine has beneficial effects on mood, then pairing the drink with the consumption of caffeine should promote increased preference for that drink. Initial results showed that under these circumstances caffeine was not a strong positive reinforcer, although it did have aversive effects (Rogers *et al.* 1992) and, more importantly, it appeared to act as a negative reinforcer by removing or alleviating the negative effects of overnight caffeine withdrawal (Richardson & Rogers, 1993). Results of an earlier study found similar effects in rats (Vitiello & Woods, 1977). Rats which had been previously given caffeine for 12 consecutive days (by injection) developed a relative aversion for a novel taste (saccharin) paired with the absence of caffeine. In addition, aversion for saccharin was also seen when this taste was paired with the injection of caffeine in caffeine-naïve rats. These contrasting effects occurred at doses of caffeine which, at least based on a simple body weight calculation, can be equated to the intakes of caffeine in humans drinking, for example, no more than six cups of instant coffee per day. The rats, however, received the caffeine in a single daily injection.

An implication of these findings is that caffeine will have a positive influence on the consumption of caffeine-containing drinks only after a pattern of fairly frequent, perhaps daily, intake of these drinks has already been established. Initially, therefore, other factors must operate to promote the habit (Cines & Rozin, 1982; Zellner, 1991). Thirst may motivate consumption on some occasions, but in general most people consume tea and coffee, together with other beverages, well in excess of what is needed to maintain adequate fluid balance. Alternatively, preferences for coffee and tea could be acquired through association with the nutritonal benefit derived from added milk, cream and/or sugar, or through a flavour–flavour conditioning process (Zellner *et al.* 1983; Baeyens *et al.* 1990). If nothing else, the use of sweeteners, milk and cream may provide an immediate way of improving the sensory appeal of the beverage for the novice coffee or tea drinker.

Yet a further possibility is that situational influences on mood play a role in reinforcing preferences for certain foods and beverages. Coffee, tea and indeed many drinks are

[1] Liking refers to the palatibility of the food or drink, that is, the hedonic or affective response to the taste, flavour, texture, etc of the item (Rogers, 1990). This can be distinguished from preference. For example, a person may eat more margarine than butter because of price or perceived health benefits, even though they like the flavour of butter more than that of margarine. Therefore, as measured by the amount consumed they would show a preference for margarine, although on a hedonic measure butter would score higher (see also Elizalde & Sclafani, 1990; Forbes & Rogers, 1994). Usually, however, separate measures of liking and preference are not taken, and in most studies changes in liking can only be inferred from measures of preference.

typically consumed in social contexts and during, for example, breaks from work or other activities. Pairing the positive shifts in mood occurring in such situations with consumption of a drink could result in a conditioned increase in preference for that drink. This process is sometimes referred to as evaluative conditioning (Zellner, 1991).

The reinforcing effects of caffeine, alcohol and a variety of other psychoactive substances used by human beings have been studied extensively (e.g. Stolerman, 1992; Griffiths & Mumford, 1995; O'Brien *et al.* 1995). However, these effects have not generally been considered in relation to their impact on orosensory preference (liking), and at present rather little is known about how preferences for coffee, beer and cigarettes, for example, are learned. Further study of the role of conditioning in the acquisition of dietary habits is therefore clearly warranted. This can be expected to confirm a significant influence of the mood effects of dietary constituents on preferences for particular foods and drinks.

EMOTIONALITY, DIETARY RESTRAINT AND BINGE EATING

Emotional eating and the psychosomatic theory of obesity

Stated simply the psychosomatic view is that obesity is due to overeating that occurs in response to emotional stimuli. While the most common response to arousal states, such as anger, fear and anxiety is the loss of appetite, it is argued that some individuals react by eating excessively. In turn, eating modifies the emotional state, for example it reduces anxiety. Overeating is thus a learned behaviour which can be viewed either as a coping response, or as resulting from a confusion of internal cues associated with activation and stress and natural hunger cues (Kaplan & Kaplan, 1957; Bruch, 1961; Robbins & Fray, 1980). These and related ideas have been investigated extensively and have received some empirical support. For example, individuals who are overweight are more likely to report eating when depressed or anxious (Plutchik, 1976). Furthermore, both experimentally induced and naturally occurring anxiety have been shown to lead to overeating in the obese, whereas the same manipulations inhibited eating in lean subjects (Slochower, 1983).

A difficulty with much of the research testing the psychosomatic hypothesis, however, is that it has failed to take into account the possible interaction between effects related to obesity and the effects of dieting (see next section). Thus, for instance, current dieting appears to be a better predictor of the amount eaten when depressed than is obesity (Baucom & Aiken, 1981). These relationships can also be studied by examining the intercorrelations between the three subscales of the Dutch Eating Behaviour Questionnaire (van Strien *et al.* 1986) which purport to measure restrained eating (example item: "If you put on weight, do you eat less than you usually do?"), emotional eating (e.g. "Do you have a desire to eat when you are irritated?") and external eating (e.g. "If the food tastes good to you, do you eat more than usual?" and "Do you eat more than usual when you see others eating?"). A consistent finding is a fairly strong correlation between emotional eating and restraint, a similar correlation between emotional eating and externality, and a non-significant correlation between restraint and externality (van Strien *et al.* 1986; Wardle, 1987; Hill *et al.* 1991; Rogers & Green, 1993). Thus externality and dietary restraint (or dieting) may have rather separate influences on (over)eating behaviour, while emotional eating perhaps arises partly as a consequence of dieting and partly as a consequence of the effects of emotional stress.

In addition, the idea that eating in response to emotional stress occurs because it is reinforced by, for example, a reduction in anxiety has been challenged on theoretical grounds. Instead, it is argued that eating is activated by non-specific effects of the stress (Robbins & Fray, 1980). From their extensive review of the literature these authors

concluded that "stress-induced eating is definitely fact and not fiction, but... the eating is *not* produced as a coping response to aversive correlates of the (emotionally) activating situation. Rather the organism responds to activation by focusing on salient external cues, which for the obese, are particularly compelling foods" (p. 129, their italics).

Dieting and dietary restraint

The concept of dietary restraint as developed by Herman & Polivy (1980) has had a major impact on research investigating human eating behaviour. Using the Revised Restraint Scale, which assesses concern with dieting and weight, and short-term weight fluctuation, they were able to show that self-control of food intake in 'restrained eaters' is highly susceptible to disruption. In what is now seen as a classic study, subjects were given preloads of either two glasses of milkshake, one glass, or none, and were then required to rate the taste of various ice-creams (Herman & Mack, 1975). Whereas the intake of ice-cream was inversely related to the size of the preload in so-called unrestrained subjects, restrained subjects responded in a counter-regulatory fashion, that is their intake of ice-cream increased as the size of the preload increased. A similar result was obtained by manipulating subjects' beliefs about the calorie content of the preload consumed while keeping its actual calorie content constant. Restrained subjects tended to overeat after a preload identified as high calorie, but ate rather little following a preload identified as low calorie (Polivy, 1976; Spencer & Fremouw, 1979). These results have been interpreted in terms of a process of disinhibition. The preload, by forcing the perceived intake of calories above a critical threshold (or 'diet boundary'), causes normally restrained eaters to suspend their self imposed restraint, thereby releasing their underlying desire to eat due to hunger, emotional or other reasons (Herman & Mack, 1975; Herman & Polivy, 1984).

In addition to a food preload, induction of negative mood states, including anxiety and depression, can precipitate breakdown of restraint and overeating in restrained eaters (reviewed by Ruderman, 1986). In contrast, unrestrained eaters tend to respond to anxiety and depressed mood by undereating. In a study of clinically depressed patients unrestrained eaters reported significant weight loss and restrained eaters significant weight gain after the onset of their depression (Polivy & Herman, 1976). Increased anxiety may also be responsible for precipitating overeating after food preloads, since this manipulation in itself will be emotionally arousing for the restrained eater. The same is probably true for the disinhibiting effects of exposure (without eating) to palatable food (Rogers & Hill, 1989).

However, by no means all studies have found that restrained eaters overeat in response to anxiety or depression. For example, Steere & Cooper (1992) reported that restrained eaters markedly reduced their food intake if they were both hungry and anxious, and we observed no effect of dietary restraint on premenstrual mood related increases in food intake (P. Jas & P. J. Rogers, unpublished).

Recent research has continued to refine the concept of restraint and to investigate the psychological and nutritional consequences of dieting (Herman & Polivy, 1991; Lowe, 1993; Westenhoefer *et al.* 1994). One aspect of this is that overeating triggered in laboratory studies by a preload, dysphoric moods or other events appears to have similarities with naturally occurring eating binges. A common behaviour of many dieters is the alteration of strict adherence to their (unrealistic) diet broken by periodic episodes of overeating. Accordingly, it has been argued that dieting is a pivotal causal factor in the development of binging, bulimic behaviour and anorexia nervosa (Polivy & Herman, 1979; Hill, 1993).[2]

[2] Note that although the terms dieting and restrained eating have often been used interchangeably, the various restraint scales used to date do not assess current dieting; rather they measure the extent to which an individual exerts conscious control over eating in relation to concerns about weight (Polivy *et al.* 1979; Rogers & Green, 1993). This distinction is not merely semantic, because the effects on eating and psychological functioning of

Bulimia

The primary symptom of bulimia or bulimia nervosa is binge eating, that is, the 'rapid consumption of a large amount of food in a discrete period of time' accompanied by the feeling that the eating is 'out of control' (American Psychiatric Association, 1994). Typically, extremely large amounts of food (> 12·5 MJ (> 3000 kcal)) are consumed during bulimic episodes, with most items being energy dense high fat, high carbohydrate foods (e.g. Mizes, 1985; Hetherington *et al.* 1994; van der Ster Wallin *et al.* 1994). Binging is also usually followed by purging, for example, self induced vomiting, abuse of laxatives or compulsive exercise that is aimed at avoiding weight gain. The highest incidence of bulimia occurs in young women, and a majority of these women with bulimia are of normal weight.

Bulimia tends to begin during a time of personal stress. In addition, many reports indicate that it is very common for actual bulimic episodes to be preceded by dysphoric mood states, and that mood is improved during binging (e.g. Mizes, 1985; Cooper & Bowskill, 1986; Hetherington *et al.* 1994). Cooper & Bowskill (1986) found a strong association between deterioration of mood and the onset of self reported episodes of overeating in patients with bulimia, and also in women students who were highly restrained eaters and were on a diet. This effect was much weaker in non-dieting high restrainers. These observations have led to the suggestion that binging is negatively reinforced by the emotional relief (e.g. Mizes, 1985).

Whether or not such a mechanism does operate in bulimia and other examples of mood and stress associated eating, on its own this would seem to be insufficient to account for the very intense and excessive character of bulimic eating behaviour. Furthermore, after binging, bulimics frequently experience a rapid deterioration in mood, including guilt, self disgust, fear of weight gain and depression. Purging, in turn, can reduce these negative emotional states (thus purging may also be negatively reinforced; Mizes, 1985). It is also difficult to establish the exact causal relationships between the different features of the psychopathology of bulimia. For example, some authors have suggested that the overeating and depression, as well as other symptoms which coincide in bulimia such as substance abuse and impulsive behaviour, share a common pathology, but that they are not necessarily functionally interdependent. The link is thought to be a deficit in brain serotonergic activity (Rosenthal & Hefferman, 1986; Weltzin *et al.* 1994).

Various approaches have been taken in the search for explanations and treatments for bulimia, and increasingly these have made connections with the mood (affective) disorders. Mood and overeating do appear to be closely associated in bulimia, but the development and maintenance of bulimic behaviour will depend on a complex network of influences, including primary aetiological factors and the biological and learned effects of the binging and purging (Booth, 1988; Laessle *et al.* 1988).

FOOD CRAVING

Food craving is usually defined as a strong desire to eat a particular food (crave: 'to long for, to desire intensely, to need greatly or urgently': Hanks, 1986; see also Weingarten & Elston, 1991). Its relevance to the present discussion is that relationships between mood and food craving form the basis of several hypotheses concerning abnormalities of appetite control.

current dieting and weight suppression can differ from those of so-called dietary or cognitive restraint (Lowe, 1993; Green *et al.* 1994).

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Carbohydrate craving

The first of these hypotheses is the suggestion that dysphoric mood is associated with carbohydrate craving. This then is supposed to lead to increased carbohydrate intake, which in turn augments brain serotonergic activity and thereby ameliorates the depression (Wurtman & Wurtman, 1989). Some support for this comes from results showing improvements in mood following the consumption of high carbohydrate meals (see above). In addition, several studies indicate a specific increase in carbohydrate consumption associated with depression in CCO, SAD and PMS (Fernstrom *et al.* 1987; Rosenthal *et al.* 1987; Kräuchi & Wirz-Justice, 1988; Wurtman *et al.* 1989; Wurtman & Wurtman, 1989).

Serotonergic mediation of these effects has also been tested. For example, Wurtman *et al.* (1987) identified a group of obese carbohydrate cravers and a group of obese non-carbohydrate cravers and compared their responses to the drug d-fenfluramine which increases serotonergic neurotransmission. Carbohydrate craving was defined on the basis of the frequent consumption of snacks high in carbohydrate and fat, but low in protein (average of seven such snacks/d). The smaller number of non-carbohydrate cravers had a similar frequency of snacking, divided almost equally between high protein and high carbohydrate snacks. Compared with placebo, treatment with d-fenfluramine had a more immediate and larger effect on the frequency of snacking and snack intake in the carbohydrate cravers, although there was also a significant reduction in snacking in the non-carbohydrate cravers during the third and final month on d-fenfluramine. These results were interpreted as being consistent with the proposal that d-fenfluramine decreases hunger for carbohydrates in the carbohydrate craver by mimicking the effects of carbohydrate consumption on serotonergic functioning. However, although d-fenfluramine also reduced mealtime energy intake in the carbohydrate cravers (but not in non-carbohydrate cravers), this was due to a similar reduction in intake of all macronutrients. In addition, d-fenfluramine did not appear to be superior to placebo in its effects on mood. On the other hand, d-fenfluramine has been found to be effective in the treatment of depression in SAD and PMS (O'Rourke *et al.* 1989; Brzezinski *et al.* 1990) and in the latter of these studies the improvement in mood was accompanied by a suppression of premenstrual increases in carbohydrate and fat intake.

An aspect of the mood, carbohydrate craving and serotonin hypothesis which has not been widely discussed is the mechanism by which depressed mood is supposed to give rise to carbohydrate craving. One suggestion is that the depressed individual recognizes the beneficial effects of carbohydrates and accordingly deliberately increases carbohydrate intake in order to improve his or her mood (Wurtman, 1988). Wurtman & Wurtman (1989) have also proposed that increased carbohydrate consumption may occur because the normal feedback regulation of carbohydrate and protein intake is disrupted in CCO, SAD and PMS. Normally, it is argued, consumption of carbohydrate would be expected to increase brain serotonin activity and inhibit further carbohydrate intake. In depression, when brain serotonin levels are low, this response is reduced and the desire to eat carbohydrates persists. Other variants of these ideas have also been suggested (e.g. Møller, 1992), but none of them appears to have been tested directly.

A further possibility is that appetite for carbohydrates is amplified because of an increased liking for high carbohydrate foods reinforced by their effects on mood (Rogers *et al.* 1992; and see above). Strong conditioned preferences are unlikely to develop, however, if there is any substantial delay between the ingestion of carbohydrate and improved mood. A similar suggestion was made to account for the observation that the carbohydrate:fat ratio normally chosen by subjects at either breakfast or lunch tended to

correspond with the macronutrient composition favouring optimal postmeal alertness (Lloyd & Rogers, 1994). These effects occurred relatively soon after eating.

There is also a problem in the way in which the term craving is used in relation to these ideas. Although there may be an association between lowered mood and increased carbohydrate intake or increased preference for high carbohydrate foods, it is far from clear that these changes in eating behaviour can be characterized as arising from cravings for carbohydrates. Craving suggests a particular intensity as well as specificity of appetite, and while carbohydrate craving is obviously an appealing term, its existence as a distinct form of appetite remains unproven. The most significant difficulty for the carbohydrate craving hypothesis, however, is the evidence suggesting that the actual effect of carbohydrate on brain serotonin synthesis is at best very small and therefore probably functionally insignificant (see above).

Sugar, fat and endogenous opioids

The existence of carbohydrate craving has also been challenged on the grounds that the preferences appear to be for foods high in both fat and (sweet) carbohydrate (Drewnowski *et al.* 1992*b*).[3] This, moreover, has been linked with activity of the endogenous opioid system rather than serotonergic mediation (Drewnowski, 1992).

Recent evidence shows that opioid peptides play a significant role in the regulation of food and fluid intake, and specifically in the mediation of hedonic responses to orosensory stimuli (Kirkham & Cooper, 1991; Drewnowski, 1992). For example, in rats allowed to sham-drink sucrose (i.e. rats fitted with a gastric fistula and drinking with the fistula open) the opioid antagonist naloxone did not alter the animals' willingness to start drinking but instead appeared to reduce their rate of sucrose intake in a similar manner to the effect of lowering the concentration of sucrose (Kirkham & Cooper, 1988). This indicates a dissociation of hunger and palatability, and results of studies on human subjects (e.g. Yeomans & Wright, 1991; Drewnowski, 1992) generally confirm a lack of an effect or minimal effect of opioid antagonists on self reported hunger, while pleasantness ratings and intake of preferred foods and/or sweet and high fat foods are often markedly reduced. Perception of the intensity of sensory stimuli is unaffected. Other findings indicate that opioids are released in response to the ingestion of palatable foods (Fullerton *et al.* 1985; Blass, 1991; and see above).

In turn, these observations have led to proposals linking effects of eating on endogenous opioids with food craving and effects on mood. For example, Drewnowski (1992) has argued that overeating and reported cravings for foods high in sugar and fat may share a common mechanism with opiate drug addiction. Earlier, Blass (1987) suggested that consumption of sweet and other palatable foods may be motivated in part by their capacity to relieve stress, this effect being mediated by the release of endogenous opioids in response primarily to sweet taste. These ideas are supported by results showing that the opioid antagonist naloxone is highly effective in reducing food intake in binge eaters, owing largely to an effect on the consumption of sweet, high fat foods such as chocolate and biscuits (Drewnowski *et al.* 1992*a*). It is also claimed that opioid antagonists suppress stress-induced overeating (Fullerton *et al.* 1985).

There is good evidence that endogenous opioids are involved in mediating affective responses during eating. Whether or not the same system(s) are implicated in the

[3] Actually, the critical factor in relation to the proposed effects of carbohydrate on brain serotonin is the food's glycemic index, that is, its relative ability to stimulate insulin secretion (Wurtman & Wurtman, 1992). The relevant foods are therefore not only those containing pure or almost pure carbohydrate. They should however contain very little protein, since ingestion of protein will tend to decrease brain serotonin production. Nonetheless, many of the data on craving (e.g. Drewnowski, 1992; Hetherington *et al.* 1994; van der Ster Wallin *et al.* 1994; Hill & Heaton-Brown, 1994) suggest that a high fat content is in itself an essential feature of frequently craved foods.

development of food craving, compulsive eating, or obesity is less certain. However, this is a possibility which is likely to provide a fruitful area for continued investigation.

Learned specific appetites

In an important contribution to the understanding of food craving, Weingarten (1983, 1984*a*) demonstrated the influence of learned associations on basic motivational processes. In these studies rats were fed liquid meals on a preprogrammed schedule giving a total daily intake of 70% of their *ad lib.* intake. During this training phase each meal was signalled with a tone and light (the conditioned stimuli, CS+) presented for 4 min before and 30 s after the food was made available. In the test phase the CS+ was again presented, but the rats were no longer food restricted since a bottle of the liquid food was available *ad lib.* The rats, nonetheless, responded 'robustly and rapidly' to the CS+ by taking a substantial meal, and this behaviour was maintained throughout many subsequent days of testing. This shows that external stimuli previously associated with food consumption can reliably motivate eating in the absence of immediate nutritional need. This is not to say that the animal's internal state has no influence under these circumstances; if it has eaten very recently the likelihood of responding to presentations of the CS+ is reduced (Weingarten, 1984*a*).

The implications of these observations in relation to the control of food intake and food choice, and other appetitive activities including drug use and abuse (*cf.* Stolerman, 1992), would appear to be far-reaching. The basic findings have been replicated in a study on preschool children (Birch *et al.* 1989), and further studies on rats have investigated suggestions for physiological mechanisms mediating external stimulus control of eating (Weingarten, 1984*b*; Weingarten & Martin, 1989). It also appears that a relatively lengthy presentation of the conditioned stimuli is necessary to enhance eating (Weingartern, 1985). However, many questions about this phenomenon remain unanswered.

Of particular interest here is the possibility that presentation of a CS+ which has become associated with consumption of a food may elicit a desire to eat or craving for that specific food or reinforcer (Weingarten, 1985). If this is the case, then external stimulus control of eating, which Weingarten refers to as conditioned meal initiation, might more accurately be considered a conditioned or learned specific appetite. Specific appetites might also become conditioned to salient internal stimuli accompanying, for example, particular emotional states. In fact, more probably this would involve associations formed between eating and a configuration of both internal and external stimuli evoking the emotional response (*cf.* Robbins & Fray, 1980; Wardle, 1990; Booth, 1994). Therefore, as well as providing a mechanism linking mood and food craving, this could help explain effects of mood on eating at an individual level, since these relationships would be shaped according a person's own unique learning history. Also if food cravings are based on learned associations then presumably they can be unlearned (i.e. extinguished), for example, through unreinforced exposure to the context in which the craving normally occurs. The exposure might even involve tasting a small amount of the food but not swallowing it. This cue exposure technique has shown some success in the treatment of binge eating in bulimia nervosa (Jansen *et al.* 1992) and drug abuse (Heather & Bradley, 1990).

Food attitudes, ambivalence and anxiety

The discussion on learned specific appetites suggests that food craving, that is, the desire to eat specific foods in particular contexts or in relation to particular feelings, can be a feature of normal appetite. This contrasts with the widely held view that food craving is indicative of an eating pathology, or even an addiction. One of the questions that this raises is exactly what experiences are regarded as food cravings. The desire to eat cereal at

breakfast and the desire to eat chocolate when relaxing and watching television in the evening may both be examples of learned specific appetites. However, very few people are likely to label the desire to eat cereal as a food craving (unless perhaps this occurred in the evening, which culturally is a less appropriate time for eating breakfast cereal).

This suggests that attitudes to food and eating will play a critical role in the experience of food craving. Chocolate, for example, is a highly palatable food typically consumed in addition to main meals and used variously as a gift, treat, and reward. Thus chocolate is not regarded as a staple food. Indeed, its nutritional value (high sugar, fat and energy content) is often viewed negatively – as the penalty for indulging in the enjoyment of the sensory pleasure it evokes (e.g. 'nice but naughty'). In other words, chocolate is a highly desirable food, but which according to social norms should be eaten with restraint (*cf.* James, 1990). One implication of this conflict is that there may be an attempt to avoid eating chocolate altogether, or at least to limit its consumption before natural satiety occurs. Therefore, the desire to eat chocolate is experienced more intensely because by attempting to resist it the desire persists and becomes more noticeable. In turn, this desire is labelled as craving, rather than say hunger, because of the attitude that chocolate is an indulgence (Rogers, 1994).

The conflict often experienced when eating chocolate and other 'treat' foods, such as cakes, biscuits and also savoury snackfoods, is demonstrated by the results of a number of studies. Some, for instance, have observed a positive correlation between food-induced salivation and dietary restraint, whereas others have found either no difference or reduced salivation in restrained compared with unrestrained eaters. A review of these different findings suggests that they can be accounted for by differences in restrained and unrestrained eaters' emotional responses to food and eating (Hill *et al.* 1995). Specifically, it was suggested that confronting restrained eaters and dieters with palatable, diet prohibited foods when they have recently eaten can be expected to provoke anxiety which in turn will inhibit their salivary responses (Rosen, 1981; Rogers & Hill, 1989). A similar conclusion was drawn from results showing that individuals who reported themselves to be chocoholics displayed, compared to a group of age and sex matched controls, the greatest liking for chocolate, the largest increase in hunger, but the smallest increase in salivation when exposed to chocolate (Rogers *et al.* 1994*a*). Although the results for the salivation measure appear to contradict the other measures, and also the finding that the chocoholics had a much higher frequency of consumption of chocolate, they are consistent with the suggestion that a characteristic of these individuals is an anxiety about eating chocolate (Rogers, 1994).

In an investigation of the relationship between dieting and food craving, subjects kept detailed diaries of their food intake, hunger, mood and food cravings (Hill *et al.* 1991). Chocolate was specified as the craved food on nearly 60% of all occasions on which a craving was noted. Furthermore, dysphoric mood often preceded occurrences of craving, and for the subjects who invariably acted on their craving (i.e. ate the craved food) there was a positive shift in mood after eating, for example, toward feeling calm and relaxed. Significantly, however, subjects who attempted to resist their cravings tended to report a negative shift in mood after eating the craved food. In another recent study subjects rated the appropriateness of 50 foods and beverages on 50 attributes or uses (P. J. Rogers & H. G. Schutz, unpublished). Among these attributes/uses were, "when I am unhappy", "when I want something I really like", "when I want something nutritious", and "a food difficult to resist". Chocolate ranked highest on the attribute "difficult to resist", although it ranked only 17th in terms of frequency of consumption. Foods which were scored high on "difficult to resist" were also highly liked. Furthermore, these foods were rated as appropriate for the mood "when I am unhappy", indicating presumably that eating these

foods can improve mood. On the other hand, there was a low correlation between "difficult to resist" and perceived nutritional value, with chocolate ranked 39th out of 50 on the use "when I want something nutritious". This again indicates an ambivalence about eating chocolate.

Foods which are craved or said to be addictive appear not to possess any unique psychopharmacological activity (see above). Instead, the experience of food craving is probably best understood in terms of the interaction of the normal mechanisms of appetite control, the hedonic effects of certain foods and culturally determined perceptions of the role and use of those foods.

CONCLUSIONS

This review shows that eating and drinking can have substantial effects on mood, mediated by sensory, predigestive and postabsorptive influences of the substances consumed. Some of these effects involve relatively direct action on the central nervous system, although of course ultimately all changes in mood will be encoded by changes in brain activity. In turn, appetite can be affected by mood. The mechanisms by which this occurs are also complex, and include (at a proximate level) the interaction of cognitive factors and basic conditioning processes. The investigation of these mechanisms should be a priority for future research, since this holds particular promise for improving knowledge of the control of normal and excessive appetites.

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