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Review

Carbohydrate ingestion, blood glucose and mood

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Abstract

A series of studies have reported that a high carbohydrate meal, or diets high in carbohydrate, were associated with feeling less energetic. However, after a drink containing pure sugar most studies report no effect. Meals almost exclusively carbohydrate increase the availability of tryptophan and hence serotonin synthesis in the brain, however, a small amount of protein blocks this mechanism making it an uncommon response. In many individuals, poor mood stimulates the eating of palatable high carbohydrate/high fat foods that stimulate the release of endorphins. There is a tendency for those with lower blood glucose, when performing cognitively demanding tasks, to report poorer mood. In a range of situations an association between a tendency for blood glucose levels to fall rapidly, and irritability, has been found. Differences in the ability to control blood glucose levels influence the association between carbohydrate intake and mood. There is a need in future research to contrast the impact of carbohydrate on mood in those distinguished because of their pre-existing psychological and physiological functioning.

Keywords: Blood glucose; Carbohydrate; Endorphins; Hypoglycaemia; Mood; Sugar; Tryptophan

Contents

1. Introduction

Data are reviewed that deal with the intake of carbohydrate, a related physiological parameter the level of blood glucose, and mood. In a series of popular books,

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sugar ingestion and blood glucose have been related to a range of behavioural problems [1,33,39,55]. Sucrose in particular has been suggested to influence behaviour by a range of mechanisms including the induction of swings in blood glucose levels. Buchanan [19] described sugar as "The most ubiquitous toxin" and Fredericks and Goodman [39] stated:

> For one person in every ten, sugar is a deadly food, paving the way towards a hundred distressing physical symptoms, plus all the tortures of neurotic and even psychotic symptoms

.

Others suggest that the intake of carbohydrate improves mood [148], particularly in subsets of the population, those suffering with seasonal affective disorder (SAD), the pre-menstrual syndrome (PMS) or carbohydrate-craving obesity.

Studies that have attempted to relate either dietary sugar or blood glucose levels to mood are reviewed. It is an area where a number of extravagant claims have been made that are apparently believed by many of the general public. A recent example is that the intake of refined sugar is associated with a short-term improvement in mood, a 'sugar rush'. A popular suggestion is that after a rapid rise there is a subsequent rapid fall in blood, a 'hypoglycaemic reaction', with a corresponding decline in mood.

2. Short-term effect of carbohydrate intake on blood glucose and mood

There are over a dozen studies of the short-term impact of carbohydrate on mood that vary in their approach. Some have contrasted sugar- or starch-based foods with those offering high levels of protein, whereas others have given either sucrose or glucose containing drinks and compared them with a placebo.

Various studies have contrasted the effects of protein-rich foods, such as turkey breast, with carbohydrate-rich foods and reported changes in the extent to which individuals felt energetic [22,104,117,120]. A frequent explanation for carbohydrate being more sedative than protein is the suggestion that it reflects an increased amount of tryptophan entering the brain [53,120] (see below for evaluation). Other studies, that did not use a control meal, have similarly found that a carbohydrate containing snack or meal resulted in decreased subjective energy [125,146].

The picture with studies that have contrasted the taking of a sugar with a calorie-free placebo is equivocal. There are two reports that the sugar containing drink resulted in increased energy [13,15], although in three other studies the findings were non-significant [18,99,129].

A major variable in this area is the time when mood was assessed. The majority of studies measured mood about 2 h after the taking of the drink or meal, something true of all the studies that reported an association between decreased subjective energy and the consumption of a meal. In contrast, the two studies that found that subjective energy increased after a sugar containing drink measured mood after 14 or 30 min [13] or 60 min [15]. Particularly in the Benton and Owens study [13], that used by far the largest sample sizes in this area, the timing of mood measurement may explain the result. They commented that the short term increase in reported energy seemed a robust phenomenon as they replicated the finding; it was, however, a small effect that might not be observed with a small sample size.

In summary, it appears that carbohydrate intake is associated about 2 h afterwards with feeling less energetic, although there may be a short term small increase in energy before this takes place. The finding of Thayer [125] that following a sugary snack a short term increase in energy was followed by a longer-term fall in subjective energy is consistent with this two stage effect. The evidence concerning the differential effects of macronutrients is mixed, although some studies have found meals that were almost entirely carbohydrate had a more sedative effect than those rich in protein. The inconsistency between the impact of meals and sugar containing drinks led Reid and Hammersley [100] to conclude that the effects of carbohydrate on behaviour are mediated via psychological factors.

3. Blood glucose and mood under demanding conditions

The studies discussed above examined the association between meals and the mood of subjects who were sitting quietly. In contrast, the possibility that the raising of blood glucose maintains mood when sustained demands are placed on an individual has been little considered. Given the brain's high metabolic rate it seems possible that mood will be more influenced by the supply of glucose when performing cognitively demanding tasks, as these will increase the brain's need for glucose [92,101].

Owens et al. [89] hypothesised that under conditions of cognitive demand there would be a stronger association between mood and blood glucose levels. Mood was assessed before and after performing three cognitively demanding tasks; a test of hand-eye co-ordination; the Stroop task; a rapid information processing task that required subjects to monitor stimuli every 600 ms for 30 min. With all three tasks falling levels of blood glucose were associated with lower self-reported energy. That the tasks in this series of experiments placed greater cognitive demands on subjects, than in previous studies, may be an important factor in the association between falling blood glucose and falling levels of subjective energy.

Benton and Owens [13], during both short term (30 min) and longer (2 h) experiments, demonstrated that subjects whose blood glucose remained low reported greater tension than those whose blood glucose was higher. Negative mood

states have also been associated with low blood glucose in insulin-dependent diabetics, whereas positive moods tend to be related to higher levels of blood glucose [44]. An insulin-clamp to induce hypoglycaemia has been used to manipulate mood in normal subjects; a state of tense-tiredness resulted that persisted for 30 min after euglycaemia was restored [43]. Two physiological reactions may underlie the changes in mood associated with low levels of blood glucose. Activation of the autonomic nervous system, in an attempt to return blood glucose to normal levels, may account for increased feelings of tension, whereas neuroglycopenia may be responsible for the lowering of subjective energy. You can speculate that the association between falling blood glucose and falling subjective energy reported by Owens et al. [89] may be the consequence of localised neuroglycopenia, a result of the demanding cognitive tasks that had been performed.

4. The incidence of hypoglycaemia

Many in the general population believe that although carbohydrate may give a short-term boost of energy, several hours later there can be a hypoglycaemic response and an associated lowering of mood. The suggestion is that there is a carbohydrate-induced release of insulin that, over time, causes low levels of blood glucose resulting in irritability or aggression. In part this reflects popular books [111] that state that diets containing large amounts of refined sugar are associated with a tendency to develop low levels of blood glucose.

Hypoglycaemia has been blamed for a surprising diversity of ailments. Literally meaning low glucose concentration in the blood, it is not a disorder as such, but rather a symptom with a long list of possible aetiologies including organic causes such as islet cell tumours, hepatic insufficiency, adrenocortical deficiency, hypopituitarism and sarcomas. Functional causes include alcoholism, starvation, stress, severe muscular exertion and disorders of intestinal absorption. Chemically induced causes include the influence of insulin and the sulphonylureas [76]. It is, however, reactive hypoglycaemia that has attracted so much popular attention: it is also known as food-stimulated, spontaneous, functional or post-prandial hypoglycaemia. Following a meal the levels of blood glucose increase, stimulating the pancreas to release insulin, that in turn causes glucose to be taken up by the cells. It has been suggested that susceptible individuals, particularly following meals containing high levels of sugars, may produce high levels of insulin for prolonged periods such that the levels of blood glucose fall to an extent that cerebral activity is disrupted.

Blood glucose is normally maintained in the range 70–100 mg/dl. When glucose levels fall insulin secretion stops and steps taken to raise the glucose levels may include releasing catecholamines, glucagon, growth hormone and glucocorticoids. The release of catecholamines is associated with symptoms such as sweating, palpitations, weakness and anxiety, a syndrome that is not specific to hypoglycaemia but occurs whenever high levels of these hormones are released [76]. A blood glucose level of approximately 40 mg/dl is about the point at which this hyper-adrenaline syndrome can be expected, although there are individual differences. If the glucose values fall further then an impairment of brain functioning may occur; confusion, amnesia, blurred vision and bizarre behaviour may be observed [76]. Marks et al. [75] coined the term neuroglycopenia, to refer to the signs and symptoms that develop when the supply of carbohydrates to the neurone is inadequate for normal functioning.

The writings of a few physicians and lay-authors have associated the hypoglycaemic reaction with feelings of fatigue, malaise, inadequacy, anxiety, depression, psychotic disorders, alcoholism and violent crime. There can be few medical topics that have been the subject of more confusion and controversy. Harris [52] introduced the term spontaneous hypoglycaemia when he described non-diabetic patients who presented symptoms similar to those that result from insulin-induced hypoglycaemia. The treatment simply involved the taking of frequent, small, low carbohydrate/high protein meals. Such was the subsequent over-diagnosis that some physicians came to doubt that reactive hypoglycaemia existed. There is, however, no doubt that hypoglycaemia can exist, in fact it can be induced in almost anybody providing that the experimental conditions are chosen appropriately [76]. The debate concerns the frequency with which the condition occurs naturally in the general population and in specific sub-groups. Although it varies from worker to worker, a common definition of hypoglycaemia is less than 40 mg glucose/dl venous blood, within 6 h of ingesting a hyperglycaemic stimulus. Such a definition in no way implies that such a level of blood glucose is necessarily abnormal or pathological, and makes no mention of symptoms. The distinction should be made between reactive hypoglycaemia so defined and ‘essential reactive hypoglycaemia’ in which these glucose values are associated with spontaneous symptoms. Repeated demonstrations of a blood glucose level less than 40 mg/dl, at the time when spontaneous symptoms occur, is necessary for the latter diagnosis.

How many people become hypoglycaemic? There is a marked difference in the frequency with which the writers of books for the lay reader, and many clinicians working in the area, suggest that hypoglycaemia occurs. The former contended that it is the common cause of many problems, whereas the latter contend that it occurs only rarely.

The ability to control blood glucose levels can be assessed using a glucose tolerance test (GTT). After fasting overnight 50 g of glucose are consumed and the initial rise and subsequent fall in blood glucose is monitored. There have been many studies of the metabolism of glucose using the GTT of which Lev-Ran and Anderson [69] was one of the most extensive. They found a large range of glucose nadirs when the test was administered to 650

normal subjects. The median nadir was 65 mg/dl; the 10th percentile was 47 mg, the 5th was 43 mg and the 2.5th was 39 mg/dl. It seems from these data that one in every 40 healthy asymptomatic individuals is likely to have a GTT nadir below 40 mg/dl. The confusion caused by self-diagnosis was illustrated by the study of 135 patients suspected of suffering from reactive hypoglycaemia that found that only four had blood glucose levels sufficiently low to attract the diagnosis [69]. In many of these studies, blood was examined only at hourly or half-hourly intervals. Such a procedure can be misleading as when plasma-glucose is monitored continuously it is relatively common for the nadir to be below 50 mg/dl. It should not be assumed that low blood glucose is necessarily pathological; it occurs relatively commonly, without extreme or in fact any obvious symptoms.

What are normal variations in blood glucose levels? In contrast to the enormous literature describing blood glucose changes under experimental conditions there is surprisingly little evidence of the changes in blood glucose concentrations in healthy subjects during their everyday life. Hansen and Johansen [51] monitored blood glucose throughout the day in five normally fed males; the levels never fell below 71 mg/dl. Genuth [41] in a similar study of eight subjects never found levels below 80 mg/dl. Alberti et al. [2] monitored 19 normally fed individuals and reported "…very little variation in glucose concentration during the day", they commented that the changes are quite different to those obtained during a GTT. The picture that emerges in normally fed healthy subjects is of apparent blood glucose stability. Values rise following a meal and then fall, but typically not to levels that clinicians require to diagnose hypoglycaemia. The evidence is very clear: in normal individuals, fed in the normal manner, a hypoglycaemic response is uncommon. The degree of the elevation of blood glucose depends upon the nature, texture and form of the food ingested but except in diabetics, to a surprisingly small extent upon the actual amount.

In conclusion the widespread assumption within the general public, that a hypoglycaemic reaction is the cause of a range of behavioural abnormalities, has lead a number of professional bodies to consider the question and issue public statements. The American Diabetes Association, The Endocrine Society, and The American Medical Association jointly issued a statement on hypoglycaemia [121]. They said that widespread publicity has: "led the public to believe that there is a widespread and unrecognised occurrence of hypoglycemia in this country. Furthermore, it had been suggested repeatedly that the condition is causing many of the common symptoms that affect the American population. These claims are not supported by medical evidence". The American Dietetic Association endorsed the view that, "Valid evidence is lacking to support the hypothesis that reactive hypoglycemia is a common cause of violent behaviour…".

There seems little reason to disagree with these unanimous views of the professional bodies who have considered the importance of food-stimulated hypoglycaemia. It is an uncommon condition, only rarely the cause of human ailments. Although much has been made of the GTT in popular literature, it is of questionable value when used to diagnose hypoglycaemia. It is extremely unlikely that any individual is going to regularly, if ever, exclusively consume large amounts of sugar on an empty stomach and then wait for 3 h without eating again. A meal tolerance test would be a better reflection of everyday life. In this context, comparisons of meal tolerance and GTT are significant. Hogan [59] contrasted blood glucose levels in patients who had been diagnosed as hypoglycaemic, who received the traditional GTT and on a second occasion an equivalent amount of glucose in a mixed meal. When 33 patients were examined, symptoms associated with low blood levels were observed in 19 during the GTT. In contrast, when administered with a meal, a similar glucose load never resulted in blood glucose levels of less than 75 mg/dl. Of the 33 patients 9 displayed symptoms during the meal, although at the time the blood glucose varied from 78 to 135 mg/dl, that is, they never approached hypoglycaemic values. The meal test appeared valid as hypoglycaemia was observed in two patients with insulinoma (insulin-producing tumours of the pancreas).

It seems reasonable to conclude that typical meals rarely stimulate reactive hypoglycaemia although a meal is an appropriate stimulus when examining the possibility of post-prandial hypoglycaemia. Protein triggers hormonal responses that differ to those caused by carbohydrate; fats alter gastric emptying and intestinal motility. Thus a large dose of glucose, in the absence of other nutrients, must be viewed as a grossly unnatural stimulus. An obvious recommendation for future studies is that attempts to diagnose reactive hypoglycaemia should use a stimulus containing protein and fat as well as carbohydrate. In this manner the data obtained will be more indicative of reactions to normal diet.

5. The control of blood glucose and mood

The Quolla Indians live at a high altitude in Peru and are known for their high murder rate and family feuds. Among anthropologists their claim to fame is that "these Andean highlanders are portrayed as perhaps the meanest and most unlikeable people on earth" [90]. Bolton [16,17] noted that many acts of violence appeared to be irrational, even to the Quolla, as they were stimulated by the most minor of events. As a violent response was not culturally approved Bolton wondered if the aggression of the Quolla was a reaction to food. He noticed a strong craving for sugar and considered the possibility that their aggression reflected a tendency to develop low levels of blood glucose. Bolton found that in those ranked as most aggressive there was a tendency for their blood glucose to fall to low values in a GTT.

Virkkunen [130,132–136] studied the control of the blood glucose levels of offenders, who had committed one or more severe violent assault. In a GTT blood glucose tended to rise to abnormally high levels, but it then fell to particularly low values from which it was slow to recover. In some of these individuals blood glucose levels were low enough to attract the clinical diagnosis of hypoglycaemic and neuroglycopenic symptoms were observed. Similarly, a tendency to develop low levels of blood glucose has been found in those with anti-social personalities [131] reflecting the release of high levels of insulin that cause blood glucose to fall rapidly [137]. As many of these offenders had a history of violence when intoxicated, it may be relevant that alcohol enhances the release of insulin and hence the tendency for blood glucose levels to fall.

The study of Benton et al. [12] found that an association between falling blood glucose in a GTT and aggressiveness also occurred in a sample of young male adults from the general population. Those whose blood glucose levels fell rapidly in a GTT were more likely to make aggressive comments when faced with a frustrating situation; they were more likely to think that aggressive acts were justified. A similar study, using young adult females, found that the lower the level to which blood glucose fell during a GTT the greater was the aggressive tendency [28].

Attempts to relate the intake of carbohydrate, hypoglycaemia and violent behaviour attract several types of criticism. One concern is that hypoglycaemia occurs rarely. Gray and Gray [47] argued that there could not be an association between blood glucose levels and aggressiveness unless the levels could be described clinically as hypoglycaemic. Donohoe and Benton [28] demonstrated that very low blood glucose levels were not in fact critical; a nadir of 63 mg/dl or less during a GTT was associated with a greater aggressive tendency. In the three types of study described above the blood glucose of few individuals fell to levels where, in a clinical sense, it could be described as hypoglycaemic. However, it seems that blood glucose levels, higher than those that can be described as hypoglycaemic, can be associated with irritability and even a predisposition towards aggression.

A second concern is that as we have only a correlation, causality cannot be assumed. It is possible that a third factor, maybe a hormone or a brain chemical, influences both aggression and blood glucose. It is possible that one of the hormonal mechanisms that maintain glucose homeostasis may influence this aspect of mood. Virkkunen [131,133,137] reported that violent offenders with a low blood glucose nadir had enhanced insulin secretion during a GTT.

One way of establishing that blood glucose levels predispose towards aggression is to alter blood glucose levels while monitoring irritability. Benton et al. [8] towards the end of a school day gave 6-year-old children a glucose containing drink or a placebo. Those who received the glucose drink were less likely to display signs of frustration and irritability when playing an impossible computer game. Later the same impossible computer game was given to young adults, without any history of anti-social behaviour, and their reaction to the frustration was monitored [13]. In this double-blind study, the taking of a glucose drink decreased irritability. This response occurred when blood glucose levels had not fallen to the low levels needed to diagnose clinical hypoglycaemia. Although pure glucose drinks were given in these studies the influence on irritability was too rapid to be accounted for by changes in the availability of tryptophan (see later).

A neural mechanism has been proposed to account for findings in this area [131,134]. It has been found that impulsive offenders with a tendency to act aggressively, particularly when intoxicated, have a low rate of serotonin turnover, as indicated by a low level of 5-hydroxyindole acetic acid (5-HIAA) in the cerebrospinal fluid. This low level of brain serotonin is associated with enhanced insulin secretion and hence a tendency to develop low levels of blood glucose. The extent, if any, of the role played by counter-regulatory hormones in these phenomena needs to be established.

In summary, a tendency for blood glucose levels to fall rapidly has been found in violent offenders, Peruvian Indians and undergraduates who are aggressive. In two intervention studies, a glucose containing drink reduced irritability in a frustrating situation. Although the literature on the topic is fairly consistent the data are limited. It may prove to be of heuristic value to consider whether irritability and aggression are influenced by individual differences in insulin release, the frequency that meals are eaten and the effect of these meals on blood glucose values.

6. The ratio of carbohydrate to protein and mood

Richard and Judith Wurtman proposed that carbohydrate intake increases the synthesis of serotonin in the brain. They believed that the rate of serotonin synthesis was normally controlled by food intake and that certain individuals eat high carbohydrate foods not for their taste, or because they are hungry, but for the psychopharmacological effects [149].

They proposed that a high carbohydrate meal increases the ratio of tryptophan to ‘large neutral amino acids’ (LNAA; tyrosine, phenylalanine, leucine, isoleucine and valine) in the plasma. Insulin facilitates the uptake of most amino acids, but not tryptophan, into peripheral tissues such as muscle, whereas tryptophan is bound to blood albumin and insulin increases the affinity of albumin for tryptophan. The result is an increased ratio of tryptophan to the other amino acids in the blood. A large protein meal will relatively increase the levels of the other LNAA rather than tryptophan as it represents only 1–1.5% of total protein [147].

The large neutral amino acids and tryptophan compete for

a transporter molecule that allows entry into the brain. Therefore, when a high carbohydrate meal increases the tryptophan/LNAA ratio relatively more tryptophan is transported into the brain. Fernstrom and Wurtman [36] fed rats a mixture of carbohydrate and protein, or a mixture of carbohydrate and protein that lacked the five amino acids that share a transport mechanism with tryptophan. Both diets increased the level of tryptophan in plasma, although the level in the brain only increased when the competing amino acids were absent.

In the brain tryptophan is transformed by the enzyme tryptophan hydroxylase into the neurotransmitter serotonin. As tryptophan hydroxylase is normally not fully saturated, any increased availability of tryptophan results in an increased synthesis of serotonin. Serotonergic activity is known to influence aggressiveness and depression.

7. Tryptophan in the blood after carbohydrate intake

A long series of studies have found differences in the ratio of tryptophan to LNAA in the blood after high carbohydrate as opposed to high protein meals. The phenomenon occurs in the general population [3,22,70,71,123], the obese [93] and those suffering with SAD [104]. The question arises as to the importance of these findings?

In a study using rats that had fasted for 19 h, a high carbohydrate meal increased the ratio of tryptophan/LNAA when no protein was consumed. However, as little as 2.5% of protein (by weight) blunted this effect. A meal containing either 5 or 10% protein failed to increase tryptophan levels [154]. The authors concluded that the effect was only exerted with a meal of less than 5% protein. The infrequency that meals containing very low levels of protein are eaten raises considerable doubt as to importance of this mechanism in the majority of diets.

Benton and Donohoe [9] summarised 30 human studies that considered the influence of meals differing in the percentage of calories that came from protein rather than carbohydrate. A meal that is almost totally carbohydrate increased the ratio of tryptophan to LNAA in the blood. When protein offers less than 2% of the calories there is relatively more tryptophan available, although as little as 5% of the calories in the form of protein was enough to ensure that this does not happen. These data cause serious problems for the Wurtman's when they try to explain the choice of food rich in carbohydrate as an attempt to enhance mood. It is difficult to find meals that contain so little protein that the uptake of tryptophan will be increased. Hence serotonin synthesis will not be increased and mood will be improved rarely by this means. In a range of cultures protein intake has been found to be relatively constant, about $13 \pm 2\%$ of daily calories. Similarly the ratio of carbohydrates to protein varies very narrowly in the range 4 or 5 to 1 [27,62,64]. These values are well away from those that would increase the availability of tryptophan.

In foods, which many would classify as high in carbohydrate, for example, potatoes, bread, rice and chocolate, the level of protein is high enough to ensure that tryptophan availability will not be increased. Only with a food that is entirely carbohydrate is an increased availability of tryptophan likely to occur; for example, boiled sweets, cola drinks and lemonade are 100% carbohydrate. Even then the response will only occur when these food items are taken exclusively and so long after the previous meal that protein does not remain in the gut.

Similar data were found using fasted animals where increased serotonin synthesis in the brain, after a carbohydrate-rich/protein-poor meal, occurred only following the consumption of a meal containing little other than carbohydrate [35]. In fact a chronic lack of protein in the diet would decrease rather than increase the levels of tryptophan, as protein is the only source of tryptophan.

The assumption that rats and humans respond to carbohydrate in an analogous manner may not be accurate, the changes in plasma tryptophan levels induced by high carbohydrate meals may be smaller in humans than in the rat. For example, after a breakfast containing 45 g of carbohydrate (data estimated from information presented) with 1.6% of the energy as protein, there was only a 16% increase in the tryptophan/LNAA ratio [3]. If rats and humans respond similarly this increased level of tryptophan would not have significantly increased serotonin synthesis. The tryptophan/LNAA ratio must at least double before reliable increases in serotonin synthesis can be demonstrated in the rat [36]. In fact in humans a 100 g meal of carbohydrate failed to significantly elevate the level of tryptophan in cerebral spinal fluid [124].

The data on serotonin synthesis in primates is limited to two studies in humans [91,124] and one more recently in monkeys [49] that is by far the most informative. Teff et al. [124] contrasted a carbohydrate and protein breakfast and found no differences in their impact on CSF tryptophan and 5-HIAA levels. In monkeys, Grimes et al. [49] progressively decreased the protein content of the diet from 25 to 6%. Both plasma and cerebrospinal fluid concentrations of tryptophan fell as chronic protein intake declined but importantly in this study there was no acute response to meals. The response to a single meal in terms of its influence on CSF tryptophan and the serotonin metabolite 5-HIAA was not observed in monkeys as it had been reported previously in rats. When consumed over a 4-week period, 22 and 16% of protein in the diet produced similar and higher levels of CSF 5-HIAA than diets containing 6 or 10%. This study of the chronic intake of protein produces a very different picture to that obtained following the study of single meals in rats. High rather than low levels of protein intake were associated with greater serotonin synthesis. Importantly changes in the ratio of blood tryptophan/LNAA did not predict CSF tryptophan or 5-HIAA as it does in rats.

8. Does the intake of carbohydrate relieve depression?

Wurtman and Wurtman [148] proposed that an increased intake of carbohydrate was an attempt to relieve depression in those suffering with the PMS, SADs and carbohydrate-craving obesity. They suggested that the consumption of carbohydrate increases the level of brain serotonin and changes food preferences; carbohydrate is no longer liked to the same extent. It was proposed that some individuals suffer from a disturbance of this feedback mechanism so the inhibition of carbohydrate intake fails to occur.

8.1. Pre-menstrual syndrome

The reported increased appetite and food cravings at the end of the monthly cycle have led these to be included as symptoms of PMS. However, although Christensen [21] concluded that "individuals with PMS increase their carbohydrate consumption in the pre-menstrual stage", we must distinguish carbohydrate from a generally increased food intake.

A carbohydrate-rich drink (48 g carbohydrate/no protein) in a double-blind study decreased the depression, anger and confusion of those with PMS [108]. Again it should be noted that such a meal, containing no protein, would rarely if ever be consumed. Wurtman et al. [146] found that the intake of both carbohydrate and fat increased in the pre-menstrual stage. Although these data have been quoted as demonstrating that carbohydrate intake is greater in those with PMS, there was an increased caloric intake that reflected an increased intake of both fat and carbohydrate. When they offered experimental high carbohydrate meals (4% of energy as protein) the mood of those with PMS improved. However, the levels of protein offered were sufficiently high to question whether the provision of tryptophan would have been increased and the authors found that subjects did not differ in terms of their blood amino-acid levels. Critically mood improved in this sample when the level of blood tryptophan had not increased, suggesting that enhanced serotonin synthesis was not the underlying mechanism.

The normal diet of those suffering with PMS should be considered to see if their diets are distinctive. In the 1950s, surveys began to find that the pre-menstrual stage was associated with cravings for sweet items, particularly chocolate, but there was also a general increase in appetite [138]. A survey of 384 women found that 58% reported an increased appetite and 61% an increased desire for sweet foods in the pre-menstrual period [40]. Vlitos and Davies [138], when they reviewed the topic, listed 13 studies that found a significant increase in energy during the luteal phase that ranged from 87 to 674 kcal/day. There is also substantial evidence of changes in basal metabolic rate over the menstrual cycle of both animals and humans [20,118]. Webb [140] found that eight out of 10 women showed a rise of between 8 and 16% in energy expenditure between the first and second halves of the cycle. Thus there are parallels between increased pre-menstrual appetite, increased energy intake and increased metabolic rate.

The question arises as to whether the anecdotal observation of increased craving for sugary items results in characteristic changes in macronutrient intake? The data of Dalvit-McPhillips [23] gave isolated support for such a view. She found that mean protein and fat intakes were virtually identical in the pre- and post-menstrual stages but that the mean intake of carbohydrate increased from 133 to 257 g. Although there are reports [60,73] of a higher carbohydrate intake in the pre-menstrual stage, when Vlitos and Davies [138] reviewed the topic they found seven other studies that did not. The weight of evidence supports the view that carbohydrate intake is not selectively increased in the pre-menstrual stage.

Is there support for the anecdotal statements that craving for sweet foods increase, even if the intake of total carbohydrate does not rise? There are reports that the intake of sucrose was higher in the luteal phase [45], that the consumption of sweets, cake and chocolate was greater in the pre-menstrual stage [24] and there was a preference for chocolate foods, rather than similar non-chocolate alternatives, during the bleed [127]. Fong and Kretsch [38] found that the consumption of sweets, mainly chocolate, was higher during the bleed than around ovulation. As the foods that are commonly craved are chocolate and cakes, the evidence is that it is pleasant tasting, sweet, high fat/high carbohydrate foods that are craved. The level of protein in chocolate, cakes and cookies is such that the availability of tryptophan in the blood could not be expected to increase.

In summary, there is no consistent evidence that the intake of carbohydrate per se increases in the pre-menstrual stage, rather appetite, metabolic rate and caloric intake all increase. Rather than an increased appetite for carbohydrate as such it is the consumption of pleasant tasting foods, containing high levels of both fat and carbohydrate that rises.

8.2. Seasonal affective disorder

In the winter when the light levels are low those suffering with SAD experience depression, eat more and put on body weight. A survey found that those suffering with SAD consumed more carbohydrate-rich foods (e.g. pasta, rice) in the winter, although the intake of high protein foods (e.g. meat, fish) did not differ with the seasons [64]. Again in this study the high carbohydrate foods contained enough protein to prevent an increased level of tryptophan in the blood. However, Wurtman and Wurtman [148] suggested that one characteristic that distinguished those suffering with SAD is a specific hunger for carbohydrate, a desire to decrease depression by increasing serotonin synthesis.

Rosenthal et al. [104] reported that a carbohydrate-rich protein-poor meal improved mood in those suffering with SAD, although the meal offered high levels of both carbohydrate (105 g) and fat (43 g). As this meal offered only

0.7 g protein (0.4% of energy) it significantly increased the level of tryptophan in the blood. The unusual nature of the meal in this study means that this finding cannot be generalised to more normal meals. The prediction was that the consumption of a high carbohydrate/low protein meal should have a differential impact on those who were and were not depressed. Thus it was not appropriate to use non-depressed subjects for comparison; there was a need to contrast those suffering with SAD and other types of depression. The prediction of the Wurtman's was that carbohydrate would selectively influence those with SAD. Similarly it would be instructive to offer those suffering with SAD snacks that were equally palatable, although they differed in their carbohydrate content. It is possible that there would be a differential response to foods depending on palatability rather than carbohydrate content. Is it important that palatability is often associated with the sweetness? These alternative hypotheses would also predict an increased intake of carbohydrate by those suffering with SAD, but not exclusively carbohydrate.

8.3. Carbohydrate-craving depression

Wurtman and Wurtman [145,149,150] hypothesised that there was a subset of obese patients whose weight problems were associated with depression and uncontrolled carbohydrate intake. It was suggested that they consumed carbohydrate for its predicted psychopharmacological effects. In a study obese individuals who ate almost entirely high carbohydrate snacks were distinguished from those who consumed high fat/high carbohydrate snacks [72]. The snacks eaten between meals were monitored in the former group. It is possible to calculate from the data supplied that the five so-called high carbohydrate snacks provided on average 45% of the total energy as carbohydrate, 5% as protein and 50% as fat. Only one snack contained a level of protein so low (2.5% of energy) that it is reasonable to suggest that, if eaten exclusively, the level of blood tryptophan would have risen. It was, however, improbable that any one snack would have been eaten exclusively and the protein from other food items would have limited the provision of tryptophan. Two hours after eating a high carbohydrate lunch, the mood of the two groups differed [72]. Those who were said to crave carbohydrate felt less depressed and the non-carbohydrate cravers were less alert, more fatigued and sleepy.

However, the existence of 'carbohydrate-craving' patients has been questioned. Toornvliet et al. [126] gave three types of snack to 'carbohydrate-craving' and 'non-carbohydrate-craving' obese patients that offered energy as firstly 100% carbohydrate, secondly 70% carbohydrate, 29% fat and 1% protein, or thirdly 35% carbohydrate, 3% fat and 62% protein. As predicted the tryptophan/LNAA ratio increased significantly after the high carbohydrate and high fat/high carbohydrate meal. Mood was, however, similar after all three snacks and the response of the 'carbohydrate-craving obese' was similar to others. A highly unusual snack that contained virtually no protein did not improve mood, although the level of tryptophan in the blood increased. The difference in results in the only two studies on this topic may well reflect the methodology. Lieberman et al. [72] offered a lunch in the form of palatable cookies that, as with all food, must have been associated with particular expectations. The Dutch study in contrast gave their snacks as liquids of similar taste and appearance [126] and found that the ingestion of a carbohydrate-rich snack did not improve mood. They concluded that "from a therapeutic point of view it was useless to maintain the concept of carbohydrate craving… the existence of carbohydrate-craving patients has never been established…". It is impossible to draw any conclusion on the basis of two contradictory studies.

9. Conclusions concerning the Wurtman hypothesis

There is a good evidence that a meal that is almost exclusively carbohydrate will increase the blood tryptophan/LNAA ratio. However, as little as 5% of the caloric intake as protein will prevent this increased provision of tryptophan [9]. Few diets, unless extremely selective, will regularly increase the availability of tryptophan, although occasionally a particular meal might stimulate the mechanism. Although the hypothesis relies on the study of those suffering with PMS, SAD or 'carbohydrate-craving obesity', the evidence is that such individuals do not normally consume meals with sufficiently little protein to enhance serotonin synthesis. All three disorders are associated with an increased caloric intake that reflects an increased consumption of high fat/high carbohydrate foods that are better described as palatable. Possibly because the question has been little considered in humans, even when the level of tryptophan increases, there is no evidence of an increased release of serotonin [49,124].

However, it should not be assumed that on occasions a high carbohydrate meal may not influence mood by the Wurtman mechanism. Markus et al. [78,79] argued that the reaction to stress included an increased release of serotonin and that those prone to stress may be serotonin depleted. They produced evidence that a high carbohydrate meal selectively benefited those prone to stress but only when they had been subject to stress. However, the large size of the breakfasts consumed (1200–1300 kcal) makes it unclear whether the results will generalise to more normal-sized meals. These findings suggest the need to explore individual differences as those not prone to stress did not differentially respond to the meals.

10. The chronic intake of carbohydrate and mood

There is little evidence that carbohydrate intake enhances mood by stimulating serotonin synthesis. If future research

does support the proposition then it is likely to be in very carefully selected subjects consuming atypical meals that have been experimentally created. There are, however, several studies not driven by the Wurtman's perspective that find carbohydrate intake to be associated with better mood, although it is not assumed that the provision of tryptophan has been increased. Over 9 days, de Castro [27] asked normal subjects to keep dietary diaries from which he calculated the proportion of calories that came in the form of carbohydrates. He found a significant negative relationship between the proportion of energy consumed as carbohydrate and depression. A higher intake of carbohydrate was also associated with feeling more energetic. Interestingly the associations were not between meals and mood around the time of eating, rather over several days the impact was cumulative. More recently, 686 individuals were asked at mid-day to report their mood and what they had eaten that morning [10]. The more carbohydrate that they had consumed, the happier male subjects reported that they had been during the morning. When a particular intake of carbohydrate was considered the absolute and relative amounts of both protein and fat were not associated with mood. As these observations were made in the general population they conflict with the prediction that carbohydrate only benefits the depressed [149]. Again a positive response to carbohydrate was obtained with diets that contained sufficient protein to prevent an increased availability of plasma tryptophan.

When for a week students ate experimental diets, containing low, medium or high levels of carbohydrate, the consumption of the low carbohydrate diet was associated with increased anger, depression and tension [61]. This report was similar to the finding that the eating of a low carbohydrate (25 g)/high protein (70 g) breakfast for 3 weeks resulted in increased levels of anger [26]. When a group of young German women dieted for 6 weeks those whose meals were based on cereals and vegetables, rather than meat and fish, reported better mood [114]. Thus there are three reports that experimental diets higher in carbohydrate are associated with better mood.

11. Palatability

It has been suggested frequently that stress results in general over eating [48] rather than a selective increase in carbohydrate intake. Although an increase in carbohydrate intake has been predicted at times of emotional distress studies typically report an increased intake of both fat and carbohydrate [72,138,146]. This combination of fat and carbohydrate intake raises the possibility that palatability is the critical factor.

A survey of young Canadian adults found that 97% of women and 68% of men experienced food cravings [142]. These cravings were, however, highly selective, chocolate was by far the most commonly and intensely craved item [58,106,113,142]. There is considerable evidence that animals, including humans, prefer foods that are both sweet and high in fats. Drewnowski and Greenwood [31] considered the palatability of combinations of fat and sugar and found the optimal combination was 7.6% sugar with 24.7% fat. The fat content of chocolate is close to this hedonic ideal and although the sugar content of chocolate is greater than the ideal it may well be counteracting the bitterness of cocoa.

For many dysphoria stimulates chocolate craving and Benton [6] argued that the consumption of chocolate was in many individuals a response to poor mood. The experience of strong food cravings has been associated with being bored, anxious and having a dysphoric mood [58]. Individuals who 'self-medicatate' with chocolate are more likely to have personality traits associated with hysteroid dysphoria [112], a syndrome characterised by episodes of depression in response to feeling rejected. Similarly a desire for chocolate has been reported to be associated with depression, although not with suicidal thoughts [66].

Benton et al. [11] used factor analysis to explore the attitudes of normal subjects to chocolate. A first factor was labelled craving and was associated with two types of question: chocolate was 'overpowering', 'preys on my mind' and you ' cannot get it out of my head'. This weakness for chocolate occurred when under emotional stress; 'when I am bored', 'when I am upset' or 'when I am down'. The coupling of these two groups of questions again suggested a link between negative mood and an intense desire to consume chocolate. A second factor also included two types of question: chocolate was associated with negative experiences. I feel 'unattractive', 'guilty' and 'depressed' after eating chocolate. This guilt was related to weight and body shape: 'I often diet', 'I look at the calorific value of a chocolate snack' and 'if I ate less chocolate I think I would have a better figure'. Only one study has systematically varied mood and looked at chocolate. When music was used to induce either a happy or sad mood, chocolate intake increased in those who heard the sad music [143].

There have been several suggestions that 'drug-like' substances in chocolate improve mood [6]. These include caffeine, theobromine, magnesium, anadamides (that act at cannabis sites) and phenylethylamine (that acts in a similar manner to amphetamine). As these putative 'drug-like' constituents of chocolate are found in cocoa powder, if they are critical to the satisfaction of chocolate craving, it should be satisfied by cocoa powder. In a seminal experiment, it was shown that cocoa powder was unable to satisfy chocolate craving [85]. Rather the critical factors that satisfied chocolate craving were taste and mouthfeel. In fact the suggestion that 'drug-like' substances in chocolate improve mood stands little examination. The levels of these substances in chocolate are several orders of magnitude less than the active dose. It is simply not

possible to eat enough chocolate to produce a pharmacological effect [6].

12. Palatability and the release of endorphins

Although Wurtman used the study of 'carbohydrate-craving depression', PMS and SAD to examine the hypothesis that carbohydrate was consumed to enhance serotonin levels, another more plausible mechanism exists. All three disorders are characterised by depressed mood. It is more plausible that stress or low mood induces eating, not specifically of carbohydrate, but rather the consumption of palatable foods. Palatable foods are often high in both fat and carbohydrate [29]. When ingested palatable foods induce the release of endorphins.

The example of chocolate illustrates the ability of foods containing a high level of both fat and carbohydrate to taste good and for its consumption to be stimulated by poor mood. It is well established that opioid mechanisms influence food intake. In general, opioid agonists enhance and antagonists decrease feeding [67,83]. An important finding is that opiates increase food reward or palatability.

In rats, the consumption of chocolate has been associated with an increased release of beta-endorphin [32]. The palatability of the food appears to be important, as in the rat the opioid antagonist naloxone decreased the consumption of chocolate-chip cookies more than standard rat food [42]. The preference for a sweet taste, and the intake of sweet solutions, was increased by an opiate agonist and decreased by naloxone or naltrexone [98]. In humans opioid antagonists decreased thinking about food, feelings of hunger [119,144] and food intake [128]. Naltrexone reduced the preference for sucrose [34]. Spontaneous eating has been associated with an increased release of beta-endorphin [25]. Opioid antagonists such as naloxone influenced the eating of pleasant tasting food such as chocolate, in both animals [32,42] and humans [30,119,128,144,151–153].

The suggestion that opiate mechanisms selectively influence the pleasure associated with palatable food was supported by a study in which healthy human males were given a long lasting opioid antagonist [153]. Nalmefene decreased caloric intake by 22% although the subjective ratings of hunger did not alter. The consumption of fat and protein but not carbohydrate decreased. Nalmefene did not influence the intake of particular macronutrients, but rather it influenced the intake of palatable foods, for example, high fat cheese such as brie. The choice was between various savoury food items; chocolate and sweet foods were not on offer. In a similar study naloxone differentially decreased the intake of high fat/high sugar foods [30]. The preference for mixtures of milk, cream and sugar was reduced by naloxone. Naloxone also reduced the consumption of chocolate and cookies in women who binged.

A food-induced release of endorphins would be expected to reduce stress as endogenous opiates modulate the bodily response to pain and pleasure. Similarly stress would be expected to induce the release of endorphins and increase the intake of palatable foods. In the rat a stressor, such as pinching the tail, will induce a naloxone reversible increase in eating [63]. Rowland and Antelman [105] found that rats subjected to mild tail pinching for 5 days gained more weight and preferred palatable foods. Mandenoff et al. [74] pointed to the parallels between stress-induced increases in rodent eating and a stressed human who snacks on palatable foods. As has been discussed above, there is an association between negative mood and an increased intake of high carbohydrate/high fat foods.

The early suggestion that morphine selectively increases rats' preference for high fat diets [77] has been more recently interpreted as evidence of an increased intake of a preferred diet. Gosnell et al. [46] found that morphine increased carbohydrate intake in carbohydrate preferring rats and fat intake in fat preferring rats. They interpreted this as evidence that morphine increased the palatability of preferred foods irrespective of the macronutrient content.

In summary, a major theory is that the eating of palatable foods is associated with the release of endorphins. The blocking of the action of endorphins, with drugs such as naloxone or naltrexone, decrease the intake of palatable high fat/high carbohydrate foods.

13. Individual differences in the psychological response to palatable foods

Much of the data in this area can be accounted for by the assumption that dysphoria or stress induces eating [48], a response associated with the release of endorphins [86]. It is the consumption of palatable foods, containing high levels of carbohydrate and fat that is stimulated preferentially. The possibility that such a generalisation needs to be modified to include individual differences in the response to food has been suggested. Greeno and Wing [48] distinguished two theories. Firstly, a 'general effects' model views emotional eating as a universal reaction. Secondly, an 'individual differences' model views overeating in response to stress as a characteristic of a sub-sample of the population, those who are attempting to control food intake (restrained eaters). The more general model has tended to emphasise physiological mechanisms such as endorphin release that has been considered above. The individual difference approach has tended to take a more psychological perspective.

There are various reports that stressful life events are associated with increased eating [37,110]. A range of individuals in a variety of situations have been shown to respond to distress by experiencing food cravings and eating palatable foods high in fat and carbohydrate. This has been reported to be the case for those suffering with PMS [146]

and SAD [64]. A major stimulus leading to the eating of chocolate is poor mood [6].

During periods of high workload, there was a report of a higher energy intake with a higher percentage of energy consumed as fat [81]. Based on a food diary, both teenagers [84] and university students [141] had a higher energy intake the day before an examination, compared with other times. A general problem with these studies is that it is difficult to distinguish the choice of food from its availability. If you work late your diet may be dictated by the snacks available in the workplace or university canteen, rather than the food you would choose to eat given the opportunity. Given the tendency for snack foods to contain high levels of fat and carbohydrate this in itself could influence macronutrient intake. It is relevant that one of the few studies to include a non-stressed control group did not find that stress-influenced nutritional intake [97]. When the choice of lunch of 686 students was examined the reported stress of the morning did not influence the macronutrient content. However, in this study, only the normal range of daily stressors was compared rather than an acute extremely stressful event [10]. In a well-controlled laboratory study, the stress of preparing to speak in public increased the intake of sweet high fat foods in emotional eaters, those with a tendency to eat more when stressed [88].

13.1. Restrained eating and the reaction to food

There is a growing evidence of individual differences in the tendency to eat when stressed that questions whether we should make generalisations about the relationships between eating and mood. In males, but not females, a stressful film was found to increase the intake of snacks [50]. In a French study, lunch the day before a surgical operation was compared with one several weeks later [5]. There was considerable individual variation so that whereas one man ate 125% more prior to his operation, one ate 53% less. It has been reported that decreased eating was as common a response to stress as increased consumption [87,122]. It is apparent that we need to consider individual reactions to stress rather than making general statements.

Restrained eaters chronically limit their food intake for fear of gaining weight. The distinguishing of those who restrain their eating has proved instructive and has produced pleasingly consistent findings. In typical studies, where participants are asked to taste and rate highly palatable foods such as ice cream, restrained eaters eat more under conditions of high rather than low stress. In contrast non-restrained eaters either do not change their food intake or eat less under stressful conditions [4,54,57,68,95,112].

Although food intake under naturalistic conditions has been less considered, studies have found that restrained eaters were more likely to have reported increased food intake when stressed [87,103]. Workers in a large departmental store ate more fat and sugar during periods of high workload [139]. Restrained eaters ate more when stressed, although there was no difference with non-restrained eaters. Thus, both in the laboratory and real world, those with a high level of dietary restraint ate more when under stress.

Disinhibition has been used to explain the increased food intake of restrained eaters when stressed. Forcing restrained eaters to eat pushes them above the threshold consistent with dieting causing them to suspend their normal restrictions on eating and release their underlying desire to eat [107]. Inevitably it is palatable high fat/high carbohydrate items that are consumed. Other factors that cause the disinhibition of restrained eaters include emotional events, alcohol and the anticipation of overeating later in the day [94].

The possibility that the obese and those of normal weight respond differently when distressed has been considered. When threatened with electric shock normal subjects were found to eat less, although the obese ate a similar amount [109]. Similarly, McKenna [82] induced anxiety and found that normal subjects ate less than when calm, whereas there was a non-significant trend for the obese to eat more when anxious. Threats involving pain or physical discomfort were more likely to decrease the food consumption of non-restrained eaters, while they had little effect on restrained eaters. In contrast ego threats, such as failure or public humiliation, significantly increased the food intake of restrained eaters, while food intake declined a little in non-restrained eaters [54]. It is unclear why the nature of the threat is critical.

Another hypothesis that has been considered [65,102] is that stress makes dieters more susceptible to the taste and appearance of food (shift in externality): there is an increased response to external cues, rather than cues associated with hunger or emotional state [106]. Polivy et al. [96] did not support the prediction that dieters would be more susceptible to external stimuli, as when stressed more cookies were consumed irrespective of whether they tasted good or bad. It appears that stress-induced eating in those who are dieting reflects an internal desire to eat rather than a response to taste. There are various possibilities as to the nature of this internal desire; does eating provide comfort when distressed; is food a distraction; can increased eating be viewed as learned helplessness?

Although there is considerable evidence that in at least some individuals emotional distress stimulates eating, is there evidence that eating improves mood? Although the comfort hypothesis suggests that food can ease the distress of those who diet, or who are obese, this view has been questioned. Polivy et al. [96] did not find that anxiety was reduced after or during eating. Under stressful conditions, Wardle et al. [139] found that although the food intake of restrained eaters increased, the perception of being stressed did not decrease.

A similar possibility is that eating acts as a distraction [56], for dieters the act of eating is so engrossing that attention is focused away from stressful events. A related idea is that emotional distress may be falsely attributed to

overeating. As overeating can be perceived as a problem that can be resolved later, by decreasing future food intake, this is psychologically beneficial. A final approach is to see the overeating after an ego threat as learned helplessness, the attack on the ego is generalised to food. In a similar manner to learned helplessness the individual gives up and stops trying to escape noxious stimuli, food intake is no longer limited. Polivy and Herman [95] attempted to distinguish between these types of explanation. Although they found some support for the view that eating can be a distraction from a distressing situation, there was no support for the comfort eating explanation, mood was not improved.

14. Discussion

The knowledge that sugar is a potent source of physical energy has lead to the assumption, in sections of the population, that it will also enhance psychological energy. The marketing of 'high energy' sugar containing drinks, whose advertising implies that it will give you a psychological boost, has encouraged such a view. It is easy to speculate that the discussion of a 'sugar rush' is largely, if not entirely, a reflection of the expectations generated by this type of suggestion. In fact, as discussed above, any suggestion that the intake of sugar, and the associated increase in blood glucose levels, is associated with a 'sugar rush', gains little support from the literature. The finding that a candy snack produced a short term increase in subjective energy, that was less than that produced by walking for 10 min, indicates the subtle rather than dramatic nature of the effect [125]. In fact the majority of studies have been driven by the opposite hypothesis, that carbohydrate intake is associated with decreased arousal. There is evidence that a meal containing a high level of carbohydrate compared with one virtually devoid of protein is associated with decreased arousal [53,120].

Where sugar containing drinks have been compared with placebos the influence on mood is inconsistent. The data are equivocal and where the giving of glucose and sucrose containing drinks influenced mood the effects were small. The term 'rush' seems to be totally inappropriate. The term implies a speed and degree of impact that does not correspond with reality. Although a sugar containing drink may be consumed with the mistaken expectation that it will provide a psychological boost, as discussed above the consumption of foods high in both carbohydrate and fat are more typically consumed at times of emotional distress for their presumed calming influences [6].

When relating macronutrient intake to mood there is a need to take into account the demands that are being addressed. Rather than increasing arousal there is evidence that increased blood glucose prevents the decrease in arousal that occurs when individuals perform cognitively demanding tasks [89]. A sugar drink did not boost arousal rather it prevented its decline. Although it is clear that there is no general response to glucose, the possibility that there is a subset of individuals whose mood is particularly susceptible to changes in blood glucose, should not be excluded.

There is a common assumption that any benefits associated with the intake of sugar are short-lived and followed by a rapid fall in blood glucose, a hypoglycaemic rebound, with a resulting lowering of mood. Although there is little doubt that occasional individuals suffer with rebound hypoglycaemia, there is no evidence that it is a common condition [69]. With any normal meal, that contains protein and fat, the release of glucose into the blood stream is slowed and very low levels of blood glucose are even more uncommon than those observed in a GTT.

Whereas there is no doubt that blood glucose levels sufficiently low to satisfy the clinical definition of hypoglycaemia occur rarely, the possibility cannot be excluded that blood glucose levels above those observed with clinical hypoglycaemia maybe disruptive. Thayer [125] reported that although a candy snack increased subjective energy after 20 min, the feeling of energy had declined after 1 and 2 h. Although blood glucose levels rise rapidly after food consumption, in most individuals within an hour they have begun to fall from the zenith. Benton et al. [14] contrasted the influence of breakfasts that offered either 10 or 50 g of carbohydrate (corn flakes). The eating of the larger breakfast was associated with poorer mood, late in the morning, an effect reversed by eating a snack of a further 25 g of carbohydrate one-and-a-half hours after breakfast. Clearly there is a need to take into account the patterns of meals and the interaction between snacks and what has been consumed previously.

A robust finding is that the eating of breakfast, as opposed to fasting, is associated with better mood [14,115,116]. The most extensive study was carried out by Benton et al. [10] who compared subjects after consuming one of eight breakfasts, each offering 50 g of carbohydrate. This study considered foods with different glycaemic indices and reported that breakfasts that provided higher levels of glucose later in the morning produced better mood. The levels of glucose were without exception well into the normal range.

A repeated impression is that individual differences in the ability to control blood glucose levels influence mood [12,28]. Future studies should consider such individual differences and examine differences in the reaction to meals and snacks. As well as taking into account differences in physiology there is a need to consider groups selected for differences in their psychological reaction to food. We need to distinguish those who do and do not diet. Overeating can be expected to be distressing for a dieter. Whether carbohydrate intake is pleasurable for those not dieting needs to be considered. The importance of considering individuals is illustrated by a factor analytical study of the attitudes to chocolate questionnaire [11]. Two major dimensions were distinguished. Firstly, those who craved chocolate tended to do so in response to low mood and scored highly on the external eating dimension of the Dutch Eating

Questionnaire. A second factor weighted heavily on guilt when eating chocolate, low self-esteem, body dissatisfaction and restrained eating [7]. There is a need to distinguish these two reactions. The association between the eating of highly palatable chocolate and low mood was distinct from restrained eating and the experience of guilt after the consumption of chocolate. Such an observation demands a consideration of the impact of palatable foods in individuals distinguished in terms of the tendency to display craving/external eating from those displaying guilt/restrained eating.

Many respond to feeling depressed by craving palatable foods. Having reacted in this manner some do, and some do not, experience guilt [11]. Virtually all the research in this area has treated mood as a single dimension. The failure to distinguish these two emotional dimensions may well have been the cause of confusion. Those who found that stress increased the eating of restrained eaters, but did not improve mood, may have considered those who tend to experience guilt after eating high carbohydrate/high fat meals. Those who respond to a low mood by eating chocolate are not necessarily from the same group, they may or may not experience guilt. This failure to find that eating improved the mood of restrained eaters [96,139] is likely, in part at least, to reflect the guilt felt by this group. The possibility that those not prone to guilt after eating attractive foods respond to palatable food with improved mood should be specifically considered. Future research should establish whether it is possible to select subjects who do not experience guilt and respond to the consumption of palatable food with an improvement in mood.

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