THE EFFECTS OF CARBOHYDRATES ON MOOD AND EATING BEHAVIOUR

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Abstract

Some central issues from the literature on the psychophysiological effects of carbohydrate (with protein and fat as comparators) on humans are reviewed and various methodological issues are discussed. From the review chapters the main questions that emerged were:

1. Does carbohydrate, in the form of sucrose alter mood state in humans?
2. Does sucrose increase or delay hunger and subsequent food intake?
3. Does carbohydrate intake in the form of sucrose induce carbohydrate-specific hunger or carbohydrate craving?
4. Can the obese and non-obese regulate nutrient intake physiologically when cognitive cues are held constant?
5. Can humans compensate in the short-term after a sucrose preload?

From these central issues a series of experiments was conducted on obese and non-obese adults where it was found that:

1. When administered blind the ingestion of sucrose did not have any significant influence on mood state in either obese or non-obese subjects. This lack of effect suggests a weak relationship between carbohydrate intake and mood, at least when moderate size preloads are given to obese and normal individuals.

2. When cognitive factors are held constant in the laboratory and preloads are administered blind, sucrose can delay hunger and subsequent food intake in both obese and normal-weight subjects in a natural environment. This is taken to be due to physiological regulation in both obese and non-obese individuals.

3. Although the delay in eating suggests some form of physiological regulation, there was no evidence of change in size of the subsequent meal.

4. There was no evidence that carbohydrate in the form of sucrose led to carbohydrate craving or increased hunger for carbohydrate-rich foods in any way. This applies to both obese and non-obese subjects.

It is concluded that physiological mechanisms operate effectively in humans when psychological factors are controlled. Compensatory processes, however, do not seem to operate as effectively in the short-term. From these findings it may be argued that the obese are no more or no less responsive to internal signals than normals. It may also be argued that if sugar intake has adverse effects on hunger (i.e. carbohydrate-craving, increased hunger) then such effects are more likely to be caused by psychological factors, rather than to any physiological effects. Although there remains little doubt that nutrients do influence mood and behaviour, improvement in methodology and more elaborate methods of measuring changes in behaviour are required.

Contents

Chapter 1- The Effects of Carbohydrates and Protein on Mood: A Review of the Literature

Introduction

The general purpose of this chapter is to examine the effects of carbohydrates and protein, on human mood and behaviour. The review will consider important psychological and physiological factors and concentrate largely upon the literature of 1980-1991.

Human studies on the relationship between diet and behaviour have placed increasing importance upon the effects of food of varying nutritional content on both the physiological and psychological states of an individual. Despite growing interest, to date only a relatively small number of experiments have been carried out in this field with few producing any firm conclusions. Much of the initial research was prompted by folk wisdom which tended to focus on the adverse effects that certain nutrients, particularly sugars have on mood and activity levels of adults and children. Some of these studies have shown alterations in behaviour and mood subsequent to the ingestion of various foodstuffs, (Spring et al., 1983; Lieberman, Spring and Garfield, 1986; Smith and Leekam, 1988; Pivonka and Grunewald, 1990; Christie and McBrearty, 1979). Psychological changes following consumption of carbohydrate include an increase in drowsiness, feelings of calmness and impaired concentration (Spring et al., 1983; Lieberman et al., 1986; Smith and Leekam, 1988), or conversely, an increase in arousal (Duffy, 1975), anxiety and tension (Fredericks, 1969) and even hyperactivity in children. Claims of aggression and antisocial behaviour in both adults and children have also been made (Green, 1969; Von Hilsheimer and Powers, 1973;

Printz, Roberts and Hartmann, 1980). However, the validity of the latter findings has been questioned (Spring et al., 1986; Rumsey and Rapoport, 1983; American Dietetic Association, 1985) and such extreme negative reactions may only occur in a minority of the population, if at all.

Physiological changes have also been observed in various studies. Christie and McBrearty (1979) found that the ingestion of lunch induced physiological effects including a rise in blood glucose, a change in heart rate and body temperature. Further, a great deal of research on the diet-behaviour connection has concentrated on the effects of food on human performance levels although this will not be considered here.

The purpose of this chapter is to review some of the more recent studies investigating the relationship between nutrition and mood. Focus is primarily on short and long-term studies with normal subjects. Brief reference will be made to some of the most frequently cited studies examining the effects of tryptophan on subjective mood state, because it has been postulated that there is a link between tryptophan, ingested carbohydrate and mood. Most of the literature involving animal experimentation, hyperactivity in children, food allergies, and aggressive and antisocial behaviour in humans has been excluded. Some reference will be made to sufferers of depression and carbohydrate cravers, although a thorough evaluation will not be given. Dietary effects on mood related to food choice and acceptance will not be discussed in depth but will be mentioned in relation to the "carbohydrate craving syndrome" proposed by Wurtman et al. (1981). A review of the theories on emotion (i.e. Schachter's theory of emotion (1964); Liebert and Morris' (1967) and Morris, Davis and Hutchings' (1981) two component models of anxiety; or Oatley and Johnson-Laird's (1987) theory of emotion based on five basic emotions) will not be given for it is beyond the scope of this chapter (but see Rogers, Green and Edwards, in press). Nevertheless, the contribution such theories have made to the phenomenology of mood as an emotional state is acknowledged.

The intention of the literature review is to provide a better understanding of the effects of certain nutrients on mood and to illustrate the principal methodological problems and inconsistencies in the literature. Diet-behaviour studies are so diverse and so often methodologically inadequate that it is difficult to comprehend exactly when, how and why mood might be affected. By highlighting these inadequacies it is intended that a better grasp of the main issues and difficulties involved in diet-behaviour studies will be obtained.

Methodological Considerations

A number of methods have been borrowed and modified from other related disciplines. Common methods such as double-blinding techniques and the use of placebos are sometimes used in nutritional experimental designs. In diet-behaviour studies the most common research designs used are within-subjects design or repeated measures, correlational studies and cross-sectional methods and each one has its own strengths and weaknesses.

Methodological Problems

Although there have been several studies of the effects of food or food constituents on mood, there are many inconsistent results in the literature which are probably due to methodological problems. The nutritional composition of foods used as preloads and after a preload tends to vary and is often uncontrolled. Prior nutritional state is another potential source of variation; depending on the quality and the type of nutrients previously ingested, physiological effects of meals which are administered later will vary, as may neuroendocrine functions (Slag et al., 1981; Ishizuka, Quigley and Yen, 1983). Standardising food consumption before the experiment will contribute to control of this variation. One can attempt to monitor compliance by weighing residual food, or having the volunteers keep daily diaries.

Diurnal Variation in Mood

Endogenous rhythms may interact with the effects of the food such as to maximise or indeed minimise the behavioural effects in certain subjects, and are an important factor when deciding the time at which to conduct diet-behaviour tests. Diurnal variation may be found in some physiological and behavioural characteristics such as feelings of fatigue, vigour, alertness and short-term memory efficiency. Craig (1985) indicated that circadian rhythms in the morning are more on the upswing when compared to some rhythms in the afternoon, and this fact alone may cause differential effects on mood between studies of breakfast and lunch.

The extent to which diurnal variation affects mood and behaviour may influence the level to which food will exert its nutrient effects. For example, it has been hypothesised (Craig and Richardson, 1989) that if a person's mood has reached an optimal level due to some diurnal influence or other contributory factors then it is indeed questionable whether the alleged changes induced by certain nutrients will have much effect or not. Indeed there remains some uncertainty about whether or not reported alterations in mood in the early afternoon are necessarily triggered by the consumption of lunch. In their consideration of post prandial behavioural effects, Hildebrandt, Rohmert and Rutenfranz (1974) suggested that these effects reflect endogenous rhythms, rather than the effects induced by food ingestion. However, Christie and McBrearty (1979) found a change in mood in the afternoon induced by the consumption of a meal as opposed to a change in behaviour caused by circadian rhythms.

Time Course of Mood Alterations

Different studies assess mood at very different times; some immediately after intake (Smith and Leekam, 1988), whilst others have measured mood twenty to ninety minutes subsequent to food intake (Brody and Wolitzky, 1983; Thayer, 1987; Spring et al., 1986, 1989; Lieberman et al., 1986; Pivonka and Grunewald, 1990; Craig and

Richardson, 1989). Others have waited as long as two to four hours following food ingestion to initially assess mood (Spring et al., 1983; Keith et al., 1990; Michaud and Musse, 1991). Some studies measure mood on one or two occasions only (Spring et al., 1983; Michaud and Musse, 1990; Pivonka and Grunewald, 1990; Smith and Leekam, 1988; Keith et al., 1990; Craig and Richardson, 1989). In some, mood has been assessed three to five times (Spring et al., 1986, 1989; Thayer, 1987; Leathwood and Pollet, 1983; Brody and Wolitzky, 1983).

There are also those studies that have failed to include a baseline for mood scores (Michaud et al., 1991; Pivonka and Grunewald, 1990) which could affect the conclusions drawn on mood. Furthermore, some of these studies have examined the long-term effects of certain nutrients on behaviour (De Castro, 1987; Rosen et al., 1982; Thayer, 1987; Keith et al., 1990), whilst others have focused on short-term effects (Brody and Wolitzky, 1983; Smith and Leekam, 1988; Craig and Richardson, 1989; Spring et al., 1983, 1989; Lieberman et al., 1986; Pivonka and Grunewald, 1990). Therefore, it is understandable that there is divergence in the findings.

Mood Measures

Validated self-report mood questionnaires such as the Profile of Mood States (POMS) McNair, Lorr and Droppleman (1971); the Visual Analogue Mood Scales (VAMS) of Folstein and Luria (1973) and/or the Stanford Sleepiness Scale by Hoddes, Dement and Zarcone (1972) are the most common way of measuring mood in diet-behaviour studies. Ad hoc measures have also been used in some past studies. Alternative approaches to assess mood is to use performance based measures (the "emotional" Stroop task) or to employ a mood induction technique (e.g. the Velten induction procedure (VIP) (Velten, 1968), whereby a specific mood state is induced. However, to date such approaches have rarely been used in diet-behaviour studies (see Rogers et al. in press for a review). For the purpose of this chapter discussion will be restricted

to mood questionnaires since none of the studies under review have used performance based measures of mood or mood induction techniques.

Subject Expectation

One of the main problems with using mood questionnaires is that it is almost impossible to avoid drawing attention to the examination of mood. Uncontrolled subject expectation is a well-known problem with behavioural studies. The mere fact that subjects are aware that their mood is being measured could increase subject expectations and increase the influence of "demand characteristics". Thus, any dietary-induced effects on mood may be primarily psychological effects; knowledge, beliefs, expectations and emotions about particular foodstuffs, rather than their direct physiological effects. "Impression management" is another problem associated with mood questionnaires (Rogers et al., in press). According to Rogers et al. (in press) there is "the tendency of subjects to tailor their questionnaire responses in order to present themselves in the best possible light" as opposed to appearing over anxious or depressed. However, this problem can be partially overcome by providing an appropriate cover story, in which subjects are informed, for example, that the questionnaire is designed "to control for differences in mood" (Rogers et al. in press)

Individual Variation

So far there has been uncertainty in the literature whether individual differences will influence the effects of diet on a person's mood state. However, given that alterations in performance after food intake are frequently linked with mood changes (Smith, 1985) then it seems possible to hypothesise that varying characteristics of individuals, such as age, sex, personality, and eating habits will alter mood state to some extent. Differential effects on mood as a function of sex and age (Spring et al., 1983) as well as eating habits have been cited (Craig and Richardson, 1989) although the reasons for these variations are far from clear. Personality has also been cited as a possible source for variation in people's behavioural response to food. The particular state that an

individual is in is also a part of the context in which a dietary challenge is carried out. For example, in order to claim that tryptophan or high-carbohydrate consumption induces fatigue this state should be measured in an alert well rested person as opposed to a fatigued individual (Dews, 1982/3). Similarly, Rogers et al. (in press) claimed that dietary effects on mood may be strongly related to the person's basal mood state and, thus, the dietary effects on mood may be considered as being "state dependent". This reinforces the need for baseline measures prior to examining the effects of food on mood.

Experimental Design

The utilisation of different designs may result in different effects. For example, the use of certain designs such as within-subjects design where repeated measurements are common practice, could influence the apparent effects that a specific nutrient has on behaviour. For example, when people are exposed repeatedly to alternative treatments they tend to learn what is expected of them and adjust their behaviour accordingly. Participation in an experimental procedure over days with repeated measurement of mood could also substantially increase the effects of general test fatigue. This might prove to be a critical influence of the carbohydrate-mood effects, especially when these have been in the direction of reduced arousal. The importance of order effects should be considered at this point; all that would be required is for an unbalanced number of subjects to receive carbohydrates last in the design, thereby producing an effect that is an artifact of the experimental design rather than a true effect of carbohydrate on mood. Although using a between subjects design may account for order and practice effects (see Millar, 1983 for a review) other problems may be encountered. For instance, it has been argued that subjectivity of diet reporting and mood reporting is likely to result in substantial inter-individual variation and thus true treatment differences may not be detected if a between subjects design is used, whereas a cross-over design may be more sensitive to individual variation. It seems apparent that different designs have different strengths and weaknesses.

Statistical Analysis of Diet-Induced Effects on Behaviour

A related problem is poor statistical analysis of the findings. Most studies have examined several mood scale scores, often at several time points. The procedure most commonly used has been multiple univariate comparisons. The difficulty with this type of statistical analysis is that correction for multiple comparisons is rarely made. For example, if the POMS were assessed twice then this would involve twelve comparisons, but no correction is usually made for multiple comparisons. Rather a solitary "significant difference" is interpreted as an effect of carbohydrate on mood, although on 11/12 comparisons no effect may be found. Furthermore, rounding up, 1/12 comparisons would be "significant" at $p<0.05$ by chance alone. Thus, many of the inconsistent findings in the literature could be mere statistical error.

Another problem is that few studies have found consistent or stable effects of carbohydrate on mood. Typically, one or more measures of mood are affected, but other measures which ought logically to be affected are not. It is problematic that there is a lack of any systematic effects on mood over time, or across different scales measuring related things. When data are reported as simple means, or means and standard deviations, it is difficult to ascertain whether certain effects such as an increase in fatigue is representative of all the subjects as a group or to a few subjects who differ qualitatively from the other subjects in the direction of a strong effect of sugar on fatigue.

Sample Size

A related problem is the small number of subjects used in past experiments. Some of these studies use repeated measures on less than twenty subjects (Smith and Leekam, 1988; Thayer, 1987; Lieberman et al., 1983; Craig and Richardson, 1989), whilst others have used as few as ten or less (Christensen et al., 1985; Keith et al., 1990; Spring et al., 1986, 1989; Rosen et al., 1982). When small samples are used then the

atypical responses of a small number of subjects may distort results. Furthermore, biased sampling may occur because those who come forward for these studies may have a specific interest in food and behaviour and the experimental results can quite easily be influenced by their preconceived beliefs and attitudes. Finally, such designs are probably insufficiently powerful to detect any but the largest effects and procedures. The Boneferoni correction for multiple comparisons would probably show an absence of effects. It is quite likely in such cases that the results will not generalise to the population at large.

Defining Carbohydrate-Rich Food

Finally, it is important to mention at this stage that to date much reference has been made to the study of "carbohydrate-rich" or "high-carbohydrate" food without establishing a clear criterion of what is meant by these terms. It is possible, for example that high carbohydrate foods are also high in fat and thus, the term "energy-dense" food is perhaps more appropriate. Furthermore, often a distinction is not made between the type of carbohydrate administered which can vary from simple sugars to starches to complex foodstuffs including dietary fibre, sugars and starches. Here, these terms will be used when authors use them without explanation, but where possible the exact amount and type of carbohydrate ingested will be provided in the review tables. It will be left to the reader to derive his/her own conclusions as to whether the foods described as carbohydrate-rich foods merit this label or not. Any precise definition would be somewhat arbitrary.

These are just some of the methodological issues which should be attended to when conducting diet-behaviour research. Other factors, specifically related to the studies reviewed in this chapter will be discussed throughout and at the end of this chapter.

Table 1.1 summarises the design of the main studies examining the diet-behaviour relation and illustrates the sheer variability in methodology. Among the most common

variations in the designs used in the above experiments are sample size (ranging from 5 subjects to 603 subjects), rating scales, time of testing and variation in type of designs used. It is assumed that cognitive factors were not adequately controlled since solid food was frequently administered to subjects making it difficult to control for orosensory factors and expectations.

TABLE 1.1: Methodology of studies of the effects of food on mood

Authors	Design	Sample	Type of Food/	Time of Food	Ratings of Mood	Interval between intake and testing	Short or Long Term
Brody & Wolitzky, 1983	single-blind design	53 normals	liquid	08.00h	NIMH	baseline, 20mins, 4hrs after drink	short
Spring et al. 1983	cross-sectional, single-blind	184 normals 129 M 55 F	solids	breakfast: 07.15-08.30h, Lunch 11.00-1300h	POMS, VAMS, SSS	2hrs	short
Spring et al. 1986, 1989	repeated measures designs	7 F	solids	12.15h	POMS & VAMS	baseline, 30, 75, 120, 150mins after	short
Leathwood & Pollit, 1983	double-blind design	60 normals	solids & pills	08.00h	own scales	baseline, 9am, 10am, 11am	long
Thayer, 1987	randomised within-subject	18 normals 15 F 3 M	solids	morning, mid-afternoon, evening	ADACL	baseline, 30, 60, 120 mins after	long
Keith et al. 1991	within subjects design	7 female cyclists	solids	NA (natural setting)	POMS	3-4hrs and at end of each diet week only	long
Lieberman et al., 1986	repeated measures	40 M	solids	12.00h	SSS, POMS, VAMS	baseline, 120,180, 240, 300mins	short
Pivonka & Grunewald, 1990	within-subjects design	120 F	liquid	15.30-17.00h	SSS, VAMS POMS	up to 60mins	short

Smith et al.1985	crossover design	48 subjects	solid/fast	lunch time	Thayer Adjective Checklist	before & after lunch	short
Smith et al.1988	within-subjects design	12 subjects 6 M 6 F	solids	standard breakfast 08.15h, lunch: 1300h	Visual Analogue Mood Scales	baseline, 60mins after lunch	short
Smith et al. 1991	between-subjects design	35 F	solids	lunch time	VAS	baseline & 1hr after	short
Christie & McBrearty, 1979	within-subjects design	20 subjects	solids/fast	1300h	MACL	up to 2hrs	short
Hartmann et al. 1977	repeated measures	12 subjects	solids	2000h	SSS	15 min interval for 2hrs	short
Rosen et al.1982	between-subjects design	8 obese in-patients	solids	not specified	STAI-T, BDI Daily self-ratings	baseline & at end of 6wk test diet. Before lunch & dinner	long
Rosen et al. 1985	within-subjects design	20 obese out-patients	solids	not specified	MAAC, TSC	baseline & at 2wk intervals	long
Monello & Mayer, 1967	NA (observational study)	603 subjects	solids free-feedings	not stated	Hunger-Satiety Questionaire	before,during, after main meal	short

De Castro, 1987	NA (observational study)	38 normals	free-feeding	not specified	Likert-like bipolar mood rating scale	prior to each meal	long
Wells et al. 1993	expt.1 counterbalance expt.2: cross-over	8 M 5 M	solid lunch lipid infusions	12.45h not specified, but 2 consecut-ive 3h infusions	alertness rating scales (20 point) same as above	baseline, and at hourly intervals after lunch not stated	short short
Wells & Read, in press	single-blind cross-over design	16 M	solids	10.30h or12.30h	POMS, VAS	baseline, 30, 60, 90, 120, 180, 240 mins after meal	short
Benton & Owens, 1993	expt.1: between-subjects. expt.2: same expt.3: same	354 normals 157M, 197F 53 F 96 subjects 48 M 48 F	liquid liquid liquid	morning or afternoon same as above same as above.	AD ACL same AD ACL VAMS	baseline,15 or 30 min after preload baseline, 30, 60, 115min after preload baseline, 15 mins after drink	short short short
Craig & Richardson, 1989	crossover design	24 M	solids	12.00-13.00h	own bi-polar scales	pre-lunch measures 11.15-11.45h, post-lunch 13.15-13.45h	short
Michaud et al. 1991	within-subjects design	319 adolescents	solids	08.00-09.00h	VAS	3-4hrs after breakfast	short
Christensen et al. 1985	single-subject experiment	3 subjects 2 F 1 M	liquid/ sugar challenge	not stated	self-report symptom rating scale POMS, BDI	baseline, 5, 10, 15, 20, 25, 30, 35, 40, 45 mins (2 subjects) baseline, 1hr, 24hr (1 subject)	long

Mechanisms for the Effects of Carbohydrates on Mood

It is not certain which mechanism serves as the mediating link between food intake and the effects it has on behaviour. To date, several mechanisms have been put forward to explain the diet-behaviour relationship. The first mechanism previously offered as an explanation for the behavioural effects induced by certain nutrients, particularly carbohydrate, is hypoglycaemia.

Hypoglycaemia and Carbohydrate Intake

Karlan and Cohn (1946) suggested that hypoglycaemia may underlie the increase in blood insulin following the ingestion of certain nutrients, specifically carbohydrate. The ingestion of carbohydrate may trigger an insulin surge which may lead to a severe reduction in blood sugar levels which may, in turn affect brain glucose, resulting in reactive hypoglycaemia. Hypoglycaemia causes the secretion of adrenaline, which affects brain glucose level, in that glycogen is broken down into glucose and may result in clinical problems involving trembling and fatigue. It has been postulated that the syndrome "hypoglycaemic fatigue" a term coined by Karlan and Cohn (1946) to describe the feelings of tiredness and dizziness experienced by certain individuals may be related to the low energy, deactivated mood experienced after the ingestion of carbohydrate-rich food. Symptoms such as depression, fatigue, confusion, headaches, nausea, irritability, anxiety, convulsions, have all been cited as possible symptoms of hypoglycaemia. It has been claimed that the severe drop in blood glucose levels subsequent to excessive ingestion of sugar plays a predominant role in triggering these symptoms. The fact that insulin treatment by injection or insulin administered exogenously may lead to hypoglycaemia renders the hypothesis that sugar intake leads to insulin-induced hypoglycaemia more plausible. Furthermore, the claim that sugar can induce hyperactivity in children has been attributed to insulin-induced hypoglycaemia caused by the ingestion of sugar.

However, there does not appear to be substantial evidence available to support this theory. Spring et al. (1986) conducted a study where they were unable to attribute any dietary-induced changes in behaviour to hypoglycaemia. For example, fatigue, one of the main features of hypoglycaemia, did not correspond to the time when a fall in plasma glucose appeared after eating a carbohydrate-rich meal. Spring et al. (1986) rejected the hypoglycaemic hypothesis partly on the grounds that the association with carbohydrate intake and elevated plasma glucose levels does not account for the reported behavioural changes, specifically tiredness, and loss of alertness manifested by some individuals one to two hours following the ingestion of carbohydrate-rich food.

Furthermore, although the carbohydrate-induced hypoglycaemia hypothesis is attractive there is evidence to suggest that the reduced blood sugar levels fail to trigger the various symptoms attributed to the hypoglycaemic state in many subjects. It has been demonstrated in past research that a fall in blood glucose levels for many normal individuals is nowhere near the criteria for hypoglycaemia (which is below 50 mg). Thus, the secretion of adrenaline which affects brain glucose levels, and may initiate the hypoglycaemic state need not occur in many healthy individuals following the ingestion of carbohydrate food (DeFronzo, Hendler and Christensen, 1980). Even for those individuals who have claimed to suffer from some of the hypoglycaemic symptoms, it has been found that the timing of the alleged symptoms does not correspond exactly with the time when a fall in blood glucose appears (Johnson et al., 1980).

Rumsey and Rapoport (1983) were unable to support the theory that childhood hyperactivity is caused by the ingestion of refined sugar. Rather, they found that a drop in activity occurred following the administration of sugar drinks for two groups of children; one group of normal children and one group of disturbed children. This

'slowing' effect took place one hour after the sugar drink in the disturbed group and three hours later in the normal group.

Diet and Brain Neurotransmitters

The second mechanism offered as a possible explanation for the link between certain nutrients and behaviour comes from the proposal that various food constituents are neurotransmitter precursors and can consequently change the levels of brain neurotransmitters that are connected with animal and human behaviour. Animal studies initially established a connection with serotonin-mediated properties and sleep; sleep latency was significantly curtailed when animals were given a substance that blocks serotonin synthesis. Since then human studies have shown that sleepiness, sleep latency and drowsiness are enhanced subsequent to the ingestion of tryptophan (Hartmann, 1983; Lieberman et al., 1982/1983; Greenwood et al., 1975) or carbohydrate (Spring et al., 1983; Lieberman et al., 1986) which apparently increases brain tryptophan and serotonin much more than the ingestion of protein-rich meals.

Furthermore, a change in human mood state may be induced by an increase in the synthesis and release of the nerve impulse transmitter serotonin subsequent to ingestion of certain nutrients such as carbohydrate (Fernstrom and Wurtman, 1972). Fernstrom and Wurtman (1972) have claimed that the brain is sensitive to changes in the blood following the ingestion of food or alternatively during a period of abstinence from food which may, in turn affect mood state. Serotonin is synthesised through the neurotransmitter tryptophan which increases in ratio to the other large neutral amino acids (LNAA) crossing the blood brain barrier subsequent to carbohydrate ingestion. Animal experimentation has shown that carbohydrates initiate an insulin surge which, in turn induces a decline in the amino acids leucine, valine and isoleucine by 40-60% and tyrosine by 15-30%. These amino acids are then principally taken up to muscle which facilitates tryptophan's entry into the brain and increases the brain serotonin synthesis (Fernstrom, 1971).

Although protein-rich food is high in tryptophan content, food high in protein does not increase the ratio of tryptophan in the brain, rather a reduction in tryptophan occurs as does the synthesis of the brain neurotransmitter serotonin (Cohen and Wurtman, 1979). This paradox comes about because a protein-rich meal contains other amino acids such as leucine, valine, isoleucine which compete with tryptophan for access into the brain and reduce the influx of tryptophan into the brain (Fernstron and Wurtman, 1972), whereas the consumption of carbohydrate-rich food creates an increase in serotonin in the brain, despite the deficiency of tryptophan in carbohydrate.

The hypothesis that the alteration of the central nervous system serotonin induced by dietary changes may underpin behavioural change is a strong one. Several studies carried out in this area lend support to the notion that neurotransmitters may be one of the candidate mechanisms in the diet-behaviour relationship (Spring et al., 1986, 1989; Lieberman et al., 1986). The research however, implicating neurochemicals in the diet-behaviour relation remains inconclusive and there are also various weaknesses in this theory which will be elucidated later on in this chapter. Serotonin seems to be associated with both normal and abnormal aspects of behaviour including appetite regulation, pain, sleep, aggression, mood (depression versus euphoria), hyperactivity in children, food cravings, pre-menstrual tension, seasonal affective disorders, drug abuse. It then becomes difficult to ascertain the role serotonin plays in the diet-behaviour relation.

Psychological Factors as Causes of Mood Changes

It has also been proposed that behaviour change subsequent to nutrient ingestion is induced principally by psychological stimuli, rather than by physiological effects of food. Knowledge, beliefs, expectations and emotions about particular foodstuffs are all psychological factors that may have a direct affect on a person's eating and behaviour. Rosenthal and Jacobson (1968) reported that food could induce

behavioural changes not necessarily due to the macronutrient composition of the food but to acquired cognitive expectations. Sensory and hedonic aspects associated with certain foods may also play an important role in the consumption of various foods and affect mood and behaviour. Some foods can often be distinguished from others due to their different orosensory properties and this may make it possible to attach different expectancy effects to meals differing in nutrient composition.

It has already been established that the smell, taste and palatability of food influence peoples' eating behaviour in general. It is quite possible that sensory properties of certain palatable foods override any physiological or neurochemical mechanisms which are presumably linked to changes in mood and behaviour. For example, chocolate consumption is renowned for promoting pleasure, elevating mood and is frequently used for "comfort-eating". The hedonic properties of chocolate are likely to produce some of these positive effects (Hill, Weaver and Blundell, 1991). Blass (1991) reported that the sensory properties of sugar and not the nutrient have a calming influence on new born infants. Other studies, however, have been unable to report such findings. Spring et al. (1986) found no evidence that deactivated mood and reported feelings of sleepiness in subjects are attributable to the role of expectancies or to the hedonic properties of the foods ingested since the carbohydrate and balanced lunches used in their study were of similar taste and texture, yet only the carbohydrate-rich food increased feelings of sleepiness in subjects.

In terms of underlying mechanisms it would seem that diet-induced effects on behaviour can not be attributed to one single mechanism.

Alterations in Human Behaviour Subsequent to the Administration of Tryptophan

In the light of evidence that nutrients such as carbohydrates influence human behaviour by altering the brain tryptophan ratio, a brief mention of the research which has

involved the examination of the amino acid tryptophan on behaviour, specifically sleep and mood should first be considered.

Tryptophan /Carbohydrates and Depression

Over the last thirty years or so tryptophan has been given in pill form to normal subjects (Smith and Prockop, 1962; Hartmann and Greenwald, 1984) and psychiatric patients because tryptophan has been alleged to possess an antidepressant action (Coppen and Wood, 1978; Cooper, 1979). It has been proposed that subjects suffering from affective disorders such as depression and seasonal affective disorder may display a deficiency in central serotonin metabolism (Van Praag, 1980; Fernstrom and Wurtman, 1972). Some studies have shown that depressed state may be relieved by increasing serotoninergic function (Wurtman et al., 1981). However, there appears to be no consensus on the antidepressant effects of tryptophan (Cooper, 1979) and some researchers have found no indication that tryptophan has antidepressant actions (Chouinard et al., 1983). There have also been claims in some of the earlier literature that tryptophan induces a mood elevating effect or an euphoriant action (Smith and Prockop, 1962; Oswald et al., 1966). This effect, however, was not replicated in the study by Greenwood et al. (1975) nor in that of Lieberman et al. (1983). In a more recent experiment conducted by Young et al. (1985) no euphoriant effect of tryptophan supplementation in normal subjects was found on examining the psychotrophic effects of tryptophan. However, in the same study Young et al. (1985) found that in normal male humans tryptophan-free amino acids mixtures caused a depletion of plasma tryptophan by five hours and a mild depressive state as assessed by the Multiple Affect Adjective Checklist when compared to subjects who were given amino acid drinks which were either balanced or contained excess tryptophan. Young et al. (1985) postulated that this effect was mediated by a lowering of 5HT in the brain and that low 5HT might be one contributory factor in the onset of depression in some patients.

Carbohydrate Cravings/Serotonin and Disorders of Mood

It has also been revealed that ingestion of carbohydrate can elevate brain tryptophan to help alleviate depression in sufferers of seasonal affective disorder (Rosenthal et al., 1989), premenstrual tension (Wurtman et al., 1989; Reid, 1986), eating disorders (Wurtman and Wurtman, 1984) and "Carbohydrate-Craving Obesity" (Wurtman et al., 1985). Wurtman et al. (1981) claimed that all of these individuals were suffering from a specific pathology, i.e. the "Carbohydrate Craving Syndrome"; an abnormal manifestation of carbohydrate intake which has been associated with a deficiency in serotonin level in the brain. Wurtman et al. (1981) theorised that such individuals experience an improvement in mood following carbohydrate intake and ingestion of the drug D-fenfluramine, where the CNS release of serotonin following carbohydrate intake has been reported to enhance mood and reduce subsequent carbohydrate consumption in such individuals. It has been suggested that many sufferers of affective disorders have unconsciously realised that by ingesting carbohydrate-rich food they will improve their mood, and are thus practising self-medication to a certain extent. The use of serotonin re-uptake inhibitors in human experiments has demonstrated effects upon mood and appetite which are consistent with these hypotheses (Wurtman et al., 1981; Wurtman et al., 1985). Whilst the postulation that some people may select food by inadvertently learning that certain substances may improve mood state is interesting, few of these studies have directly studied the effects of nutrients in an adequate fashion. Although it is not the intention to provide a full methodological critique of these studies, the more obvious methodological defects will be highlighted at the end of this chapter when considering the consequences of methodological problems.

Sleep and Drowsiness

The hypothesis that food high in carbohydrate and low in protein can induce sleepiness and reduce arousal stems principally from several studies which have shown that in entirely normal subjects drowsiness, sleepiness and sleep latency are enhanced and a

fall in alertness occurs subsequent to the ingestion of tryptophan (Lieberman et al., 1982/1983; Hartmann, 1983; Greenwood et al., 1975). Although some studies have failed to report sleep-inducing effects subsequent to tryptophan ingestion (Adam and Oswald, 1979; Nicholson and Stone, 1979).

In an earlier study by Smith and Prockop (1962) it was cited that tryptophan possessed hypnotic and sleep inducing properties when five of their seven patients fell asleep subsequent to the ingestion of 6 g of oral tryptophan, administered during the day time to subjects. Greenwood et al. (1975) found that after administering 5 g of L-tryptophan to ten normal subjects at 09.30h, self-reported feelings of drowsiness, clumsiness, muzziness and mental slowing were much greater than when compared to a placebo ingestion. Contrary to the study by Smith and Prockop (1962) no subjects actually fell asleep, and not enough changes were found in subjective mood ratings, as assessed by (VAMS), to demonstrate any great urge to fall asleep.

Lieberman et al. (1983) administered 50 mg of tryptophan or placebo to twenty normal male volunteers at 06.15h or 07.15h after fasting overnight. Self-reported mood tests were issued two hours after administration. Their findings indicated that alertness significantly decreased after the ingestion of tryptophan (assessed by VAMS) and an increase in fatigue and a decrease in vigour was also found (POMS). These results demonstrated that tryptophan induces sleepiness in the morning as well as evening. Yuwiler et al. (1981) reported that acute and chronic effects of L-tryptophan induced differential effects on mood. For the acute doses, it was reported that tryptophan significantly increased drowsiness which started about thirty minutes after ingestion and returned to baseline several hours later. Chronic effects of L-tryptophan produced greater feelings of lethargy with a longer duration often persisting until evening time. Other findings suggest that tryptophan may have a sedative and calming effect on newborn babies. Yogman, Zeisel and Roberts (1983) found that two to three-day-old infants who were given tryptophan in their bottles of formula fell into a peaceful sleep

more quickly compared with babies who were given the amino acid valine. Most of these findings lend support to the theory that serotonin acts as a sleep-inducing neurotransmitter.

The Effects of Carbohydrate and/or Protein on Mood State

Some studies have shown that carbohydrate induces similar effects on mood state as tryptophan (i.e. fatigue and a decrease in alertness) although the effects are more subtle (Spring et al., 1983; Lieberman et al., 1986). The most relevant studies examining the effects of carbohydrate and/or protein on mood given as breakfast, lunch or a snack will be reviewed. Short-term studies reporting significant results will be reviewed first. It should be mentioned that the duration of short-term studies can vary, for example, sometimes subjects act as their own control group in within-subjects design and so have to come to the laboratory more than once (Pivonka and Grunewald, 1991; Craig and Richardson, 1989). However, for the purpose of this review they will still be classed as short-term experiments, since eating behaviour or mood state has not been monitored over a long-term period. Long-term studies examining the effects of carbohydrate and/or protein on mood state will be reviewed thereafter. A separate section will be used to review those studies which have administered a well balanced conventional meal for breakfast or lunch, since they are not so much interested in the effects of specific nutrients on mood as on the effects of food in general.

Short-Term Studies

Spring and colleagues (1983) examined the alterations in mood and performance subsequent to the ingestion of certain nutrients. They conducted a comparative experiment on the effects of single meals containing either pure protein or pure carbohydrate, administered at breakfast or lunch time to subgroups of the population who varied as a function of age and sex. One hundred and eighty-four adults aged between eighteen and sixty-five years took part in this between-subjects design where subjects were randomly assigned to eat the isocaloric protein or carbohydrate meals

either at breakfast or lunch. The carbohydrate-rich meals contained 47 g sucrose, 10 g corn starch and 4 g of fat as sherbet with no protein and the protein-rich meal (turkey breast and mayonnaise) contained 57 g protein, 4 g fat and 1 g carbohydrate. All the meals were a fixed size (300 g). Subjects were expected to fast overnight if they were assigned to the breakfast condition (between 07.15-08.30h) and those who were in the lunch condition (between 11.00h-13.00h) were given a standard breakfast of black coffee and pastry (containing 3 g protein, 33 g carbohydrate and 7 g fat) before lunch. Mood evaluation occurred two hours after eating, as the serotonin hypothesis would predict that the largest effects should occur at that time. No baseline assessment of mood was taken.

Although no main effects of food type were found, there were interactions for the different measures. For example, their results demonstrated that dietary-induced effects on both mood and behaviour varied as a function of sex and age. The male subjects reported feeling calmer after the carbohydrate meal, whilst the females reported an increase in sleepiness subsequent to high-carbohydrate meals, compared to the high-protein meals as assessed by the Stanford Sleepiness Scale, although the effects were small. There was no differential effect of age nor of the time that the meal was eaten which led Spring et al. (1983) to suggest that carbohydrate might have more powerful effects on females than males. However, differences in body weight between sexes were not compensated for when meal portions were being distributed. This could, in part explain why males were not affected in the same way by the meal as females. A reluctance by male subjects to report feelings of sleepiness in the work place was one explanation offered by Spring et al. (1983) for these differences between male and female subjects. Results also varied with respect to age; older subjects (forty years or older) reported feeling more irritable following the protein-rich breakfast (as assessed by POMS) than after the carbohydrate-rich breakfast where a calming effect was reported. This raises the question as to whether age may be a useful determinant of the dietary effects on mood and the time of day the food is consumed.

Although these data have been cited as evidence that carbohydrates affect mood there are various problems. The fact that the fixed size meals failed to correlate with age, sex, weight and metabolic rate meant that some subjects were undereating, whilst others were overeating. There was no control group in this study nor any crossover phase which limits the validity of their findings. No baseline conditions were used to control composition of food for the previous week or to assess mood the week before or the day of the experiment, prior to food intake. Psychological factors rather than physiological changes could have influenced results since subjects were not fully blind to what they were consuming. The foodstuffs administered to subjects in this study were not balanced, typical meals. This must be borne in mind when considering the results of the study as pure protein or carbohydrate foods are generally not consumed in a natural setting and the effects that they may have on a subject's mood and behaviour may differ somewhat from the behavioural effects induced by conventionally balanced meals. The absence of main effects of food type is another cause for concern, especially as there is no obvious neurochemical reason why women, but not men should become sleepy. The effects were not systematic across the different measures of mood. For example, it is not clear why sleepiness was detected by the Stanford Sleepiness Scale but not by the other mood scales, namely the Profile of Mood states and the Visual Analogue Mood Scales.

The US Army Laboratories in Natick, MA conducted a study similar to that of Spring et al. (1983). Their findings lent some support to those reported by Spring et al. (1983), in that they reported that some differential effects on behaviour were found subsequent to protein versus carbohydrate-rich foods. They also found that sensitivity to nutrients varied according to age and sex, although all of the effects were apparently "subtle", i.e. non-significant.

Lieberman et al. (1986) carried out a similar study to the one conducted by Spring et al. (1983). In this study Lieberman et al. (1986) used a within-subject design with forty young males in the study, aged between eighteen to twenty-eight years. Lieberman and his co-workers (1986) were primarily concerned with finding out whether a carbohydrate-rich meal would induce similar effects of sleepiness in male subjects as it had on the female subjects in the cross-sectional study by Spring et al. (1983). In this study, results were also reported on work conducted by Lieberman et al. (1986) with respect to the relation between the ingestion of nutrients such as protein and carbohydrate and the ingestion of food constituents like tryptophan and tyrosine with regard to some of their effects on mood state and behaviour. They considered whether dietary effects on behaviour would differ if a meal comprising primarily of complex carbohydrate such as starch was administered to subjects, rather than a carbohydrate meal, rich in sucrose content as used by Spring et al. (1983).

Lieberman et al. (1986) administered pure protein and carbohydrate foods to their subjects who were requested to fast overnight, ate a standard breakfast at 07.30h in the laboratory and returned to the laboratory to consume lunch at noon. All subjects were given a protein-rich meal (turkey salad which consisted of 79.3 g of protein, 0.8 g of carbohydrate, and 37.3 g of fat) and a carbohydrate-rich meal (wheat starch which contained 152 g of a pita-bread like substance which comprised of 104.5 g carbohydrate, 27.1 g fat, and 0.7 g protein) to eat in counterbalanced order, on two separate days one week apart.

Mood was assessed using the Profile of Mood States (POMS) and the Stanford Sleepiness Scale (SSS) along with other behavioural assessments at hourly intervals over the course of five successive hours. On both days of the experiment, their findings revealed that sleepiness increased at one to three hours subsequent to carbohydrate intake and subjects reported feeling less vigorous. These results concur

to some degree with the findings reported by Spring et al. (1983) and lend further support to the notion that carbohydrate-rich foods induce sleepiness.

With regard to the serotonin-carbohydrate relation, Lieberman et al. (1986) postulated that an increase in the plasma tryptophan ratio, which apparently leads to changes in brain serotonin synthesis, should coincide with a decrease in alertness subsequent to the ingestion of a high dose of sucrose. On separate days six male volunteers were given either the protein-rich meal or the carbohydrate-rich meal which were used in the first experiment, or an isocaloric preload of sucrose. Their findings showed that the ingestion of the protein-rich meal had substantially reduced the tryptophan ratio one hour after intake when compared to the starch or sucrose meal. It was found that the sucrose or starch meals had a similar effect on the ratio of plasma tryptophan and an elevation in the ratio of tryptophan was noted two hours after lunch in both cases. This increase in the ratio of plasma tryptophan continued until four hours after lunch for the sucrose meal and five hours for the starch meal. Based on these results, Lieberman et al. (1986) concluded that carbohydrate-rich food affects mood in a way similar to tryptophan, in that both induce a decrease in alertness in human subjects.

In the study by Lieberman et al. (1986) there was, however, no real indication that subjects' mood was greatly sensitive to ingestion of carbohydrate compared to protein meals. An increase in sleepiness was also noted for those who had consumed the protein-rich meal in the three hours subsequent to lunch. Moreover, the data revealed that although the postprandial increase in sleepiness was higher following ingestion of the carbohydrate-rich meal (i.e. starch) within the first two hours of lunch, the differences failed to reach statistical significance. There was no statistically significant differences in the scores on the Vigor scale of the Profile of Mood States and the Stanford Sleepiness Scale between the groups of subjects who had consumed either the protein-rich meal or the carbohydrate-rich meal. Similar to the study by Spring et al. (1983), the meals administered to subjects in the study by Lieberman et al. (1986)

were not balanced meals. Furthermore, there was no significant evidence to suggest that male subjects were more sensitive to the behavioural effects induced by carbohydrates which raises the question as to whether females are more responsive to carbohydrate-induced alterations in mood as proposed by Spring et al. (1983) or whether men are more reluctant to report such changes in mood subsequent to carbohydrate intake.

These studies fail to produce any conclusive evidence that substantial alterations occur in mood state of normal humans subsequent to consumption of certain nutrients such as carbohydrate. For example, there is evidence in the literature that protein-rich meals may lead to feelings of tiredness in individuals (Smith and Leekam, 1988). Craig and Richardson (1986) found that a combination of nutrients when served in large quantities (1000 kcal or more) decreases alertness in normal subjects.

Spring et al. conducted a follow-up study in 1986, 1989 and investigated which mechanisms might explain the carbohydrate/mood association. The three candidate mechanisms they considered have been cited at the beginning of this chapter; hypoglycaemia, hedonic properties of foodstuffs, and changes in the ratio of brain neurotransmitter precursors, specifically the tryptophan/large neutral amino acids (LNAA) ratio.

In the study (1986 and 1989) a within-subjects design was used. Seven female subjects, in counterbalanced order, fasted overnight then on the day of the experiment consumed a standard breakfast and at lunch time were assigned to one of four conditions: a carbohydrate-rich meal (105 g carbohydrate, 7 g protein, 42.7 g fat), a protein-rich meal (105 g protein, 33.3 g fat), a balanced meal containing both protein and carbohydrate (76 g carbohydrate, 27.7 g protein, 40 g fat) or fasting. Both the carbohydrate meal and the balanced lunch were very sweet and tasted very similar,

whereas the protein-rich meal did not share the same hedonic properties of the first two meals and was very bland in taste.

By using the two meals similar in hedonic properties, Spring et al. (1986) wanted to test whether it was the nutrients in the foodstuff or the hedonic taste properties that underpinned the subsequent increase in fatigue. It was also hypothesised that both the carbohydrate-rich meal and the balanced meal would produce similar effects on the plasma glucose level but differential effects on the rates of tryptophan (as it has already been established through animal experimentation that even small amounts of protein can reduce tryptophan ratio (Fernstrom and Wurtman, 1971).

A fasting control group was included in the experiment to determine whether behavioural changes are induced purely as a result of ingestion of certain foodstuffs, irrespective of their nutrient composition or whether other variables such as time of day may influence behavioural changes as much as the food itself. It has been previously cited in the literature that mood fluctuations may be attributable to extraneous variables which are non-dietary in nature, circadian rhythms being a prime example (Hildebrandt, Rohmert and Rutenfranz, 1974).

Spring et al. (1986) measured mood using the Profile of Mood States (POMS), the Visual Analogue Mood Scales (VAMS) and the Stanford Sleepiness Scale (SSS), before lunch and at thirty, seventy-five, one hundred and twenty and one hundred and fifty minutes after eating. Blood samples were also taken at the same time to measure plasma glucose, insulin and plasma amino acids. Their results supported previous findings (Spring et al., 1983) that carbohydrate-rich meals increased fatigue two hours after the meal, whilst the protein-rich meal, the balanced meal and the fasting condition failed to produce drowsiness to this degree. According to Spring et al. (1986) the fact that both the balanced and unbalanced carbohydrate meals shared similar hedonic

properties eliminates the possibility of psychological factors contributing to this carbohydrate effect on behaviour.

Nor, according to Spring et al. (1986), could the effect of carbohydrate on mood be accounted for by its effect on blood glucose and serum insulin levels which were elevated after ingestion of a snack either high in carbohydrate content or of a more varied, well balanced nutrient content. However, the snack rich in carbohydrate was found to induce fatigue after consumption. The fact that the glucose level after both snacks increased implies that the sleepiness can not be associated with hypoglycaemia. Spring et al. (1986) noted that sleepiness was induced roughly at the time when it was expected that the tryptophan ratio level would be increased due to the ingestion of the carbohydrate meal.

Out of the three possibilities presented as candidate mechanisms underlying the carbohydrate behaviour relation, Spring et al. (1986) concluded that the alteration of the central nervous system serotonin induced by dietary changes is the most likely. The fact that they measured alterations in plasma amino acids strengthens their argument as this makes it more possible to determine whether any changes in mood are related to changes in plasma tryptophan levels.

As with their previous research there appears to be a number of problems with this work. First, it should be noted that the preloads differed in fat content; the protein-rich preload contained 10 g fat more than the carbohydrate-rich preload. It may be that this difference had some effect on the overall results. Second, whilst Spring et al. (1989) describe the design as "counterbalanced" this would have required a minimum of twenty-four subjects, but there were only seven subjects, so there may well be hidden order effects in the data. The results of their mood analyses were not reported which may imply that as in their past study (Spring et al., 1983) there were no significant effects of food type on subjective mood state. Furthermore, contrary to past findings

(Spring et al., 1983), this time it was found that the ingestion of the carbohydrate-rich meal led to a significant increase in POMS fatigue, compared with fasting but this effect was not reported for SSS sleepiness or VAMS. As with the previous study, it is problematic that there is an absence of any systematic effects on mood over time, or across different scales measuring related factors. Even assuming that fatigue is induced by carbohydrate intake why should one study find that SSS sleepiness is increased after a high-carbohydrate breakfast and another study by the same authors find that POMS fatigue (but not SSS sleepiness) is increased after lunch?

Benton and Owens (1993) conducted three short experiments in which they examined the effects of glucose preloads and their relation with blood glucose on mood. In experiment 1 using a counterbalanced double-blind design, three hundred and fifty-four normal subjects were given either a glucose drink (50 g) or a placebo drink (sweetened with aspartame and acesulfame K). Both drinks were very similar in taste and appearance. The drinks were administered in the morning or the afternoon (the exact time was not specified). Baseline mood scores (as assessed by the AD-ACL) and blood glucose were taken prior to the preload and fifteen or thirty minutes after the preload. Results showed that both the glucose preloads and higher blood glucose levels were related to a reduction in tension. Higher blood glucose levels were also associated with an increase in feelings of energy. It is unfortunate that prior food intake was not controlled for in this study since this might have proved to be quite informative.

In the second study again using a counterbalanced double-blind design with fifty-three subjects all subjects consumed three beverages; either three glucose preloads (containing 50 g, 25 g, 25 g respectively) or three placebo drinks (same as in study one). This was carried out in an effort to stabilise blood glucose over a two-hour period. The drinks were consumed either before or after 12.00h and the second and third drinks were consumed forty-five minutes and seventy-five minutes after the first

preload. Mood ratings and blood glucose were taken prior to the experiment and thirty, sixty and one hundred and fifteen minutes after the first preload. Their findings demonstrated that subjects with sustained high blood glucose felt less tense throughout the two hours, although this time no correlation was found between high blood glucose and energy levels. No correlation was found between the glucose preloads, tension and energy levels. In this study, as in experiment 1, no restriction was placed on food consumption prior to the study, although this time subjects did record if they had consumed a meal prior to the study.

In the last study the effects of glucose on mood were examined in ninety-six subjects who were confronted with a frustrating performance task (stressful video game). Mood (as assessed by VAMS and AD-ACL) and blood glucose were taken prior to the drink and fifteen minutes later. Subjects then performed six trials of the frustrating video task and mood was measured after each trial using VAMS. Blood glucose was taken at the end of the six trials and the AD-ACL was completed thereafter. Although no subjects were asked to fast or restrict food intake prior to the study fifteen subjects tested in the morning had fasted overnight and skipped breakfast. These subjects' results showed that the glucose preload helped them perform better in the frustrating task, in that they were less tense and they showed less negative responses after the experimenter's negative statement at the beginning of the fifth trial. The blood glucose levels of the glucose group increased during the performance task, whilst those of the placebo group did not. This experiment is of interest, in that a mood induction technique is employed to some extent, whereby behaviour is observed, manipulated and categorised into negative behaviour scores. As mentioned previously, this technique is rare in diet-behaviour studies (see Rogers et al., in press for a discussion on mood induction).

This study confirmed the findings of the past two studies, in that subjects with higher blood glucose reported feeling less tense. Benton and Owens (1993) proposed that

failure to make this association in past studies may have been attributable to smaller sample size (i.e. Brody and Wolitzky, 1986). However, Benton and Owens (1993) did indicate that the effects found in their studies were rather "subtle". The fact that a large sample size and an elaborate design was used in these experiments may have determined the findings. This leads one to suspect that whilst diet-induced effects on mood may exist, their effects have perhaps been over-exaggerated. Indeed, irrespective of their positive findings, Benton and Owens (1993) suggested that the relation between sugar ingestion and hypoglycaemia may be an "over reaction".

Late Afternoon Studies

The study by Pivonka and Grunewald (1990) adds further support to the neurochemical hypothesis where the serotonin-carbohydrate connection is thought to be related to sleepiness and fatigue. Pivonka and Grunewald (1990) were concerned with the effect that various dietary components, namely sugar and the dipetide sweetener aspartame, had on subjective mood state. They tested one hundred and twenty female students between ages eighteen to thirty years using a within-subjects design. All subjects consumed three standardised test drinks at 15.30h on three separate days separated by one week intervals. Mood was assessed prior to consumption and up to sixty minutes after the drinks, using the SSS (administered five times over an hour) the VAMS and POMS (both administered at sixty minutes). Subjects were administered one of the three test drinks:- approximately 373 g of water, 373 g of aspartame sweetened beverage which contained between 180-280 mg of aspartame, or 373 g of a sugar sweetened beverage which contained 50 g of sucrose (200 kcal).

Mood findings for one hundred and twenty subjects indicated that, on all three days of testing, after consumption of the sugar sweetened drink subjects reported feeling sleepier than prior to the drink, whilst the other two beverages did not have this effect. They found that SSS sleepiness was increased thirty minutes after the consumption of

the sugar drink and was maintained for the remainder of the one hour observation period. The fact that this effect was consistent over time is important since it makes it less likely that the result is a statistical artifact. Pivonka and Grunewald (1990) suggested that if a sugar sweetened drink of 220 kcal can produce feelings of sleepiness in female subjects then this may have serious implications for the ingestion of the mid-day snack. Their findings lent little support to the psychological theory since both the sucrose and aspartame sweetened beverage shared similar sensory properties. Yet only the sucrose drink induced a significant increase in sleepiness. These results support data by Spring et al. (1986) where reported fatigue could not be attributed to psychological factors.

One methodological weakness of the study is their failure to control prior dietary intake which would have enabled them to control the quality and quantity of meals consumed earlier that day or even that week. No pre-baseline session occurred for the measurement of mood in the week prior to the study. Although Pivonka and Grunewald (1990) claimed that the sucrose preload and the aspartame sweetened drink were of similar taste there can be no certainty that both drinks were indiscriminable in terms of taste and texture since no pilot work preceded the actual experiment. The fact that the taste of the beverages was not masked under experimental conditions immediately raises the question as to whether subjects guessed what they were drinking or not. Although they found that SSS sleepiness was increased over time (thirty to sixty minutes after sucrose ingestion), as with findings by Spring et al. (1983) the absence of effects on VAMS and POMS on similar scales is of concern.

Although little focus will be directed to the effects of food on affective disorders such as depression, nonetheless, the results from the study by Christensen et al. (1985) which examined the relation between diet and emotional distress will be reported since it seems to be of immediate relevance to the relation between carbohydrate and reduced arousal. Christensen et al. (1985) studied four subjects in a single-subject design. The

main aim of the study was to test the hypothesis that, for the four sensitive individuals studied, diet was a cause of emotional distress which could be reduced by altering the dietary components in their diet. In this study the authors also wanted to examine whether behavioural problems or at least the onset of emotional and behavioural distress, might be attributable to certain dietary properties found in some foods. Subjects who were recognised as suffering from behavioural and psychological symptoms were used in the study. Subjects were selected based on their scores from the Behavioural Index of Metabolic Imbalance (BIMI) and a subsequent interview. The BIMI is an instrument composed of a series of descriptive symptoms; cognitive, behavioural and somatic symptoms (Christensen et al. 1985). It was hoped that it would help to identify the need for dietary change in certain individuals and identify the connection between behavioural and emotional malfunction and dietary components. Three of the four subjects were asked to adhere to a diet which was rich in protein content and poor in carbohydrate, particularly sugar which was completely eliminated from the diet as was caffeine. Only sugar and caffeine were removed from the fourth subject's diet. Changing diet appeared to reduce distress in the long-term, even when subjects were misled about the expected effects of the change.

The Minnesota Multiphasic Personality Inventory (MMPI; Hathaway and McKinley, 1967) and the Profile Of Mood States were used to assess mood and emotional state in all the subjects except the first subject who gave a self-report of the symptoms experienced. The findings from mood scores from each experiment indicated that during the baseline period all subjects experienced more emotional distress in comparison to two weeks after the treatment diet. The results indicated that after the two-week dietary intervention subjects scored significantly less on the MMPI scores on the depression, psychasthenia and schizophrenia scales. The same applied to the POMS. Apparently their scores during baseline were indicative of an individual suffering from much emotional distress, whilst following the two-week treatment diet

their scores on the MMPI and POMS were representative of an individual suffering from much less distress.

Subjects were also challenged with the substances being tested, including sucrose. One subject reported headache, weakness and light-headedness thirty minutes after ingesting sucrose under double-blind conditions. Another reported reduced vigour and increased confusion and fatigue between one and twenty-four hours after the same challenge. This study suggests that sucrose may affect some people's mood, but it is not known whether this represents the extreme of a normal response, or whether it is a specific pathology or abnormality.

Nonsignificant Results on Mood Changes Following Carbohydrate Loading

The studies reviewed so far have been able to demonstrate some relation between carbohydrate and reduced arousal, although the validity of their data is indeed questionable. Not all studies, however, have reported such a relation.

Brody and Wolitzky (1983) conducted a study to test the hypothesis that the excessive consumption of carbohydrate, specifically sugar, not only results in a decrease in blood sugar level but has a negative effect on mood. They used a between-subjects design with fifty-three normal-weight subjects who were divided into three groups. The first group was administered 8 oz of an orange drink, rich in sugar content (75 g of cane sugar added to 25.8 g of orange drink which together comprised 97% sugar). This solution was then added to water, whilst the second and third groups were given a saccharin solution (52 mg saccharin in 8 oz of water with 1 g of unsweetened orange drink) or water respectively. The first two groups were informed that they were consuming "an extra sweet sugar-sweetened orange drink." All subjects were asked to fast overnight before participating in the experiment.

Brody and Wolitzky (1983) were interested in assessing mood states, cognitive efficiency and neuroticism in their subjects. Only their findings on mood will be reported here. Mood was assessed using the National Institute of Mental Health (NIMH) Mood Scale. The Eysenck Personality Questionnaire (EPQ) was used as a self-report measure of neuroticism. Subjects assessed their mood prior to the ingestion of the drinks and twenty minutes and four hours following ingestion.

Their results indicated that twenty minutes subsequent to the ingestion of the drinks no depression or elevation of mood was noted in the group which had consumed the sugar beverage nor in the other two groups. The data from the four hour mood ratings revealed no significant differences to the initial ratings for all three groups, except for a decrease in reported anxiety for the sugar group when compared to the other two groups. The findings of this study fail to concur with the theory that sugar consumption has detrimental effects on mood state or contributes to the onset of the condition hypoglycaemia. In effect, Brody and Wolitzky (1983) suggested that mood ratings of the sugar group appear to be moving more in the direction of improved mood state, rather than any deterioration of mood. The problems with this study are that the NIMH mood scale may not be sensitive to the mood changes perhaps caused by eating and that twenty minutes is earlier than other studies have reported effects, whereas four hours is later.

The Effects of Mixed Nutrients on Mood

Not all studies have examined the effects of specific nutrients on mood, some have focused on the effects of mixed nutrients on behaviour which in some respects is more representative of everyday eating. One of the more recent studies conducted by Michaud and Musse (1991) examined the effects of breakfast on behaviour and mood in adolescents (aged between thirteen and nineteen years). Particularly interest concerned potential effects that a larger than normal breakfast might have on their subjects. Concern regarding breakfast-size or total avoidance of breakfast has

previously been raised in the literature, in that there is reason to believe that missing breakfast could have detrimental effects on behaviour especially amongst adolescents (Politt, Leibel and Grenfield, 1981; Stewart-Truswell, 1985).

Three hundred and nineteen adolescents took part in the study. Each subject was expected to record their food intake at breakfast on two days, separated by fourteen days. Subjects were instructed to eat as normal on the first experimental day, whilst they were asked to consume more on the second experimental day than what they would normally eat. The food eaten on the second day was provided by their school (an increase of 63% over habitual breakfast intake was recommended).

Mood and performance tests were given to subjects approximately three to four hours following breakfast, at 11.00h, and blood glucose levels were measured at 11.30h. The participants measured their mood using 18 bipolar Visual Analogue Rating Scales (VASs) devised by Herbert et al. (1976). Subjects were expected to rate their mood by putting a vertical stroke on a 10 cm horizontal line in relation to the 18 pairs of adjectives. Their analysis of the mood ratings revealed no significant differences between the mood ratings of the first day where the normal size breakfast was consumed and the second day where a larger than normal breakfast was consumed.

This study suffers from several common weaknesses. For example, Michaud and Musse (1991) failed to measure subjects' mood before the consumption of breakfast on both occasions so they had no indication of how they were feeling before the ingestion of the food. Furthermore, subjects waited as long as three to four hours subsequent to food intake before assessing mood; a period which is longer than that of other studies which have reported significant effects. It is possible that other food or snacks could have been ingested in between which could have affected mood. This procedure apparently went uncontrolled. Admittedly, the need to assess mood over longer intervals subsequent to the ingestion of food is required so as to detect whether

the effects are consistent over time or short-lived. It is possible that it may take some time before the nutrients ingested have any significant effect on mood state (De Castro, 1986).

In the study by Michaud and Musse (1991) subjects were asked to consume more on the second day of the food experiment but the size or indeed nutrient content of the food was uncontrolled. This is an obvious disadvantage of studying everyday eating as a loss of control over food presentation and of precision in the measurement of what is eaten is inevitable. However, these losses are offset by the possible artefacts induced by the laboratory studies of eating which may increase the amount of extraneous factors influencing the results.

In a short-term study by Monello and Mayer (1967) sensations of hunger and satiety were assessed in 192 men (20-61 yrs), 165 women (20-67 yrs), 101 boys (11-15 yrs) and 145 girls (9-20 yrs, with most falling between 12-17 yrs). Subjects were given the opportunity to measure their own personal experience of hunger and satiety by filling in a questionnaire with multiple choice questions (the Hunger-Satiety Questionnaire) during extreme hunger (defined as "the hungriest you can ever remember being"; during ordinary hunger (defined as 30-120 minutes before a main meal); immediately before, during and after a main meal. The questionnaire was completed in total six times throughout the day and assessed mood, desire to eat, thoughts of food and physical feelings with respect to hunger and satiety. Four-point rating scales were used to measure the severity of desire to eat and preoccupation with thoughts of food ranging from "none" to "extremely strong".

A variety of adjectives were provided to assess mood and subjects were expected to tick those which best described their mood at a specific time. The same adjectives were used to describe mood at different times of the day. The adjectives used were: depressed, apathetic, irritable, nervous, tense, cheerful, excited, calm, relaxed,

contented. The adjectives were divided into four sub-scales: "negative-active mood"; "negative-passive mood"; "positive-passive mood" and "positive-active mood".

The results demonstrated that the majority of subjects (69%) both adults and adolescents, male and female, reported a negative mood state during the extreme hunger condition, whilst only 31% of subjects reported this negative mood state during episodes of ordinary hunger. As might have been predicted, the number of subjects reporting positive mood was considerably lower during episodes of extreme hunger than during ordinary hunger periods. More subjects reported "positive-passive mood" (feeling relaxed, calm, contented) during all conditions than "positive active mood". A marked sex difference was found in relation to mood scores during the extreme hunger condition: 31%, of females experienced more negative mood than males (19%) who seemed to report more physical sensations in relation to hunger. These scores varied as a function of age, in that more women reported these negative feelings compared to girls. After the meal a greater proportion of adults of either sex (65%) reported feelings of sleepiness and warmth than adolescents and the same was found for the scores on calm-contented, although it was noted that a greater percentage of males reported this sensation in both groups than females.

However, the validity of these findings may well be questioned, in that the checklist of adjectives used to describe mood state in this study might not have been suitable for testing mood repeatedly under experimental conditions. Perhaps in this type of experiment mood changes are too slight to be detected by this questionnaire. Most studies assessing mood have used more elaborate rating scales to measure mood state such as the Profile of Mood States (POMS) or other rating scales including the Visual Analogue Mood Scale (VAMS); The Stanford Sleepiness Scale (SSS); National Institute of Mental Health (NIMH). It should be pointed out here that relatively small differences in experimental methods can contribute to some of the inconsistent findings so far reported in this chapter.

One advantage that Monello and Mayer's study (1967) may have over some studies is that it reports findings from a study where natural eating was permitted using a relatively large sample of people.

Short-term Studies on the Effects of Lunch on Mood State

One problem with diet-behaviour studies is variation in the time of the day chosen to conduct the experiments; some studies have examined the effects of breakfast (Brody and Wolitzky, 1983; Michaud and Musse, 1991; Leathwood and Pollet, 1992), whilst others have concentrated on the effects of lunch (Lieberman et al., 1986; Craig and Richardson, 1989; Smith and Leekam, 1988). Yet it has been claimed that differential effects on mood and behaviour subsequent to food intake may be found depending on the time of day the food is administered. Presumably, certain nutrients interact with circadian rhythms to create changes in mood and behaviour (Craig and Richardson, 1989). Therefore, it is possible that breakfast will not have the same effects as lunch. Physiological changes (Christie and Mc Brearty, 1979) and psychological changes (Smith, 1985; Smith and Leekam, 1988) as well as cognitive changes (Smith and Miles, 1986; 1986a, 1986b; Craig, 1986; Spring et al., 1983; 1986) have been reported following the ingestion of lunch.

Post Lunch Dip

Christie and McBrearty (1979) investigated diurnal variation in mood state as well as in various other states (cognitive functioning, heart period, body temperature) in twenty male and female subjects after eating lunch. Three consecutive studies were carried out using the Mood Adjective Checklist (MACL) to examine diurnal variation in mood states of normal individuals. In the second study, their findings indicated that in the period of one to two hours after consumption of lunch there was a notable slump or deactivation in mood state as observed in the MACL activation or deactivation dimensions. In the third study which involved counterbalanced conditions twenty

subjects were tested on two separate days one week apart. On one of the experimental days, lunch and coffee were administered at 13.00h to subjects, whilst on the second day only coffee was offered. This study also found that ninety minutes after lunch a significant increase in mood deactivation scores was noted, whereas these scores decreased for the no lunch condition. It was concluded that post prandial testing was best conducted ninety minutes after the beginning of lunch.

In a study by Smith (1985) the focus of interest was the effect that lunch may have on subsequent mood state and performance efficiency. For the purpose of this chapter only the results which were found as regards mood are considered of immediate relevance. Forty-eight subjects participated in the study. There were two test sessions each on separate days. Each subject was requested to appear for testing in the late morning and early afternoon. Half of the volunteers fasted instead of lunch, whilst the remaining half were given a three-course lunch to consume. The testing time was different for half of the group of subjects, in that some were tested earlier than others (from 10.45h -12.00h V's 12.00-13.15h before lunch and from 13.15-14.30h V's 14.30-15.45h after lunch). The testing included performance tests, before which subjects were asked to assess their mood by filling in the Thayer Adjective Check List (Thayer, 1967).

Smith (1985) reported that subsequent to consumption of lunch, subjects felt less anxious and less alert than prior to the lunch, whilst those who had postponed eating lunch reported an increase in alertness but also in anxiety in the afternoon attention task session, compared to how they felt in the late morning session. In this study however, common flaws arise, in that there was no control over dietary intake prior to the study which could have affected results.

In a later experiment Smith and Leekam (1988) examined the effects that the different nutrient composition in meals had on subjects' behaviour and mood. All subjects were

given a standard breakfast on their three visits which made up a quarter of the required daily intake. For lunch, Smith and Leekam (1988) administered protein and carbohydrate meals which were isocaloric in nature and comprised one third of total daily energy intake, (the carbohydrate meals were either high in sugar content providing 75 g sugar, 28 g starch, 26 g protein, 24 g fat or high in starch content providing 75 g starch, 28 g sugar, 26 g protein, 24 g fat, whilst the protein-rich meals contained 97 g protein, 28 g starch and 24 g fat) to six male and six female subjects. Each subject was required to visit the laboratory on three separate days and received a meal differing in nutrient content on each occasion. Smith and Leekam (1988) also made sure that food size commensurate with the age, sex and weight of the individual. Pulse rate and blood pressure were taken one hour before and after the lunch which was served to the subjects at 1300h. Subjects were requested to measure their mood prior to the performance tests before and after lunch (approximately forty-five minutes beforehand and around 14.15h. Subjects used the Visual Analogue Rating Scales to self-rate their mood.

Their results showed that although differences in performance were noted after carbohydrate meals and protein meals, no differential effect on mood was observed after the consumption of the different food types. An effect on mood was found, however, after lunch, but it appeared to be attributable to the ingestion of food rather than to the dietary components of the lunch. Subsequent to lunch the reported feelings of boredom, muzziness, clumsiness, lethargy, and feeling mentally slow increased quite significantly, compared to the ratings prior to lunch.

The assessment of mood in this study is limited because the researchers investigated the post-prandial condition only in subjects immediately after food consumption. It could be that the reason for finding no effect of meal composition on mood is due to the fact that not enough time had elapsed since ingestion of the meal. In contrast to studies reviewed previously (Spring et al., 1983; 1986; 1989; Lieberman et al., 1986;

Pivonka and Grunewald, 1991), Smith and Leekam (1988) assessed mood immediately afterwards but failed to reassess mood over time.

Using a counterbalanced design on eight male subjects Wells, Read and Craig (1993) served subjects one of two isoenergetic lunches on two consecutive days at 12.45h. The lunches were similar in taste, appearance and in protein content but varied in fat/carbohydrate ratio, (i.e. the lunch was either high in carbohydrates and low in fat content or vice versa). Performance tasks and 20 point rating scales used to assess alertness were completed at regular intervals (every hour). Their findings showed that approximately two hours after the lunch high in fat content a decrease in alertness was found compared to baseline scores, whilst no post-prandial drop in alertness occurred following the carbohydrate-rich lunch. Similar effects were found for mental performance. These results were supported in a later study by the same authors (Wells et al., 1993) when they infused lipid into the duodenum of five male subjects and compared the effects of isotonic saline infusions in paired studies. Using a cross-over design two three-hour infusions; one containing lipid, the other saline, were administered blind to subjects. Their results demonstrated that the lipid infusions induced drowsiness and a decrease in alertness compared to the saline infusion. These findings are of interest, particularly since they lend no support to the carbohydrate-serotonin relation and in fact indicate that fat and not carbohydrate is the key nutrient responsible for the induction of drowsiness and impaired performance in humans.

In a later study Wells and Read (in press) showed in a single blind cross-over design on eighteen male subjects that high-fat, low-carbohydrate meals have more of an effect on mood when ingested in the morning as opposed to at lunch time. Subjects who were given the high-fat, low-carbohydrate meal in the morning (10.30h) reported feeling more dreamy, tired and weak and less friendly after the meal when they were asked to rate their mood using POMS and a "series" of bipolar visual analogue scales (exact amount of scales unspecified), whilst the meal high in carbohydrate content and

low in fat did not have this effect despite the fact that the latter was higher in energy content than the high-fat, low-carbohydrate meal. In contrast, when both meals were administered at lunch time (12.30h) both groups reported feeling more dreamy, weak and tired with no significant differences between the two meals. One problem with this study is that they claim to have used a single blind cross-over design, but the fact that there were apparently nine subjects in each group would make cross-over sessions difficult, if not impossible four conditions would require twenty-four permutations which would require twenty-four subjects.

The Effects of Lunch Size on Mood (with mixed nutrients)

Craig and Richardson (1989) examined the effects that two different size lunches had on various aspects of subjects' behaviour and mood as well as on subsequent hunger. Twelve male volunteers who were accustomed to eating a large lunch (1200 kcal) received a small lunch (less than 300 kcal) or a large lunch (1300 kcal or more) to eat on two separate days in separate weeks. The other twelve male participants who generally consumed a small lunch (less than 300 kcal) followed the same procedure. Data on eating behaviour was collected prior to the experiment from a questionnaire on their dietary habits. The nutrient composition of all experimental lunches was based on a protein-to-carbohydrate ratio of approximately 2 : 3.

Performance tests were conducted before and after the ingestion of the food as were mood ratings and hunger states. Bipolar rating scales were used to measure mood and hunger level. The scales used were: Tense-Calm, Drowsy-Alert, Hungry-Satiated, and Full-Empty. These scales were completed by the subjects along with various other tests between 11.15-11.45h before the test lunch and 13.15-13.45h after the lunch. The test lunch was eaten between mid-day and 13.00h. Results from these tests were compared to baseline measures which were obtained from subjects either on the day before, or after the test lunch.

The subjective scores of alertness decreased subsequent to the ingestion of the larger lunch when compared to the pre-lunch alertness ratings, whilst the opposite effect was found following the ingestion of the lighter lunch. These effects were found in both the light and heavy lunch eaters for the habitual lunch and the test lunch. Similar findings were reported for the subjective tension scores, in that a decrease in feelings of tension was noted after the large lunch, but an increase in tension was found after the consumption of the small lunch.

In view of these results the current impression is that the size of a meal plays an influential role in determining a person's mood. The fact that alertness and tension ratings were affected both by the size of the test lunch, and by the size of the lunch generally consumed, supports this point. Yet as already mentioned, many past studies have failed to take size of test meals into consideration, particularly in relation to weight and height. This experiment, like that of Smith and Leekam (1988) suffers from a common flaw, in that mood was assessed only once after food intake and no dietary history was recorded prior to the study. Breakfast or indeed any food ingested prior to lunch was not controlled for which could have inevitably affected their results.

Long-term Studies

Until now, there has been a paucity of studies in the literature of the long-term effects of nutrition on behaviour. Most of the studies have examined mood immediately after food ingestion or several hours later on the experimental day, but rarely several days later. Yet long-term effects are important because certain nutrients may have different effects in the short and long-term or certain nutrients may have to be ingested over a long-term period before they have any significant effects on subjective mood state (De Castro, 1987; Fernstrom, 1979). The few diet-behaviour studies that have been conducted over a longer time span will be reviewed.

The Effects of Sugar on Mood compared to Exercise

Thayer (1987) compared the effects of eating 1.5 oz of a "Candy bar" of subjects' choice with exercise (which consisted of a brisk walk lasting ten minutes) using a counterbalanced within-subjects design. The experiment took place on twelve separate days over a three-week period. Eighteen subjects participated in the study (fifteen females, three males). They were requested to fast one hour prior to the experiment, and to avoid any form of exercise or food other than the snack bar for the two hours of the experiment. Subjects were randomly selected to either eat a snack or participate in a brisk ten minute walk.

Mood was assessed by using the Activation-Deactivation Adjective Checklist devised by Thayer (AD ACL; 1967, 1978a, 1986). The AD ACL allows subjects to self-rate their energy, tension and tiredness levels. The self-report test forms were completed in a natural setting before the experimental condition and twenty, eighty and one hundred and forty minutes after the experimental condition.

Findings revealed that eating the carbohydrate-fat mixture in a candy bar increased tension between thirty and one hundred and twenty minutes afterwards, compared to walking. In the morning it also increased rated energy over time, although not as much as did walking. In the afternoon the snack slightly decreased reported energy. These results supported Thayer's (1987) hypothesis that the ingestion of the sugar snack would induce an upsurge in energy,when a great number of the subjects reported feeling more energetic after the sugar snack. However, it was found that this early energy increase was short-lived. When subsequent mood measurements were made one and two hours after ingestion of the snack, energy levels dropped quite significantly (to lower than baseline level) and an increase in tiredness and tension one and two hours after the snack was observed. This finding supported Thayer's (1987) second hypothesis that the sugar snack would induce fatigue and tension as secondary effects. It also supports the previous research findings of Spring et al. (1983; 1986)

and Lieberman et al. (1986). Thayer (1987) has claimed that it is possible that two distinct states of arousal are experienced at the same time by an individual. Thayer has developed a theoretical model called the multidimensional activation model (1978b, 1985) which aims to demonstrate that two states of arousal operate interdependently: energetic arousal (i.e. feelings of vigour, energy) and tense arousal (ie, feelings of tension, anxiety). According to Thayer (1987) both systems interact considerably so that some level of tension can increase energy levels, but if tension increases too much this will decrease energy levels. In this study Thayer (1987) has proposed that the fatigue which replaces the temporary energy surge creates a reactive "high-arousal state", in that the subject attempts to combat any feeling of tiredness or sleepiness by continuing to be awake and active.

Thayer (1987) was also interested in determining what motivated people to eat sugary snacks. He postulated that a desire to increase energy may encourage someone to ingest a carbohydrate-rich snack and this motive may even be on a sub-conscious level. The fact that it has been recognised that sugar intake induces an upsurge in blood glucose as shown by the blood sugar response curve (Brobeck, 1979) could account for the "energy raising motive" described by Thayer (1987). However, the fact that it has been claimed that aroused mood apparently occurs well before blood sugar peaks (Spring et al., 1986) would tend to suggest that blood sugar levels are not the principal explanation for mood changes after eating.

The proposal that refined carbohydrates are eaten in an attempt to increase energy immediately raises the question as to whether people already possess knowledge about sugar and the feeling of high energy it presumably creates, despite the fact that few empirical studies confirm this hypothesis. It is therefore possible that people have expectations as regards the desired high energy effect following ingestion of sugar snacks. The effects may, therefore be psychologically induced, as opposed to being physiologically based. This would lend some support to the psychological theory

discussed at the beginning of this chapter as one of the three candidate mechanisms underlying the effects of food on mood.

Thayer (1987) found that consumption of a snack high in sugar content led to a rapid increase in energy and then an increase in fatigue thereafter. However, because the snack eating occurred in a natural setting and subjects were permitted to select their own sugary snack there is no indication of the caloric content or nutrient composition of the snacks ingested. It is possible, given common composition of snack bars that it was high in fat as well as sugar. The lack of control of this experimental condition makes clear interpretation of Thayer's findings (1987) difficult. Time of testing also varied between morning and mid-afternoon which may have affected the findings, in that diurnal variation could have influenced their behaviour. However, Thayer's theoretical model (i.e. the energetic-arousal system) apparently controls the sleep-wakefulness cycle, and is greatly influenced by endogenous rhythms, with energetic arousal generally peaking in the morning and late afternoon (the two times in which the experiment was conducted). External factors such as exercise and food (as used in this study, Thayer, 1987) may also impact "energetic moods"

Although this was a long-term study the effects of sugar on mood over the long-term (i.e. days later) were not analysed. There was no control group in this study, instead subjects were randomly assigned to one of the two experimental conditions from day to day. As in other diet-behaviour studies, multiple comparisons were used in Thayer's study (1987) when analysing the data. However, unlike some past studies (i.e. Spring et al., 1983, 1989) there is a consistent pattern to the results which may suggest that they are not statistical artefacts.

Keith et al. (1990) studied the effects of ingesting different quantities of carbohydrate, protein and fat on athletes' mood. They reported that subjects who were fed low-carbohydrate diets for a seven-day period felt more depressed, tense, angry, tired,

confused and less energetic, based on the Profile of Mood States, than when moderate carbohydrate or high-carbohydrate diets were consumed. These results tend to suggest that eating behaviour can affect the mood of athletes and possibly laymen in an adverse way over a one-week period.

The findings by Keith et al. (1990) differ to those of Lieberman et al. (1986), Spring et al. (1983, 1986) and Pivonka and Grunewald (1990) and hence lend little support to the neurochemical hypothesis. In the latter studies, consumption of carbohydrate-rich food, rather than protein-rich food induced sleep, and fatigue in subjects immediately after the meal. Conversely, Keith et al. (1990) found that subjects reported an increase in vigour after consuming the meals which were moderate in carbohydrate or high in carbohydrate.

These differences may be attributed to the fact that in the above mentioned studies the effects of carbohydrates on behaviour and mood state were measured shortly after consumption (thirty to ninety minutes), whilst in the study by Keith et al. (1990) subjects waited three to four hours after the meal before the effects were measured. It is expected, however, that measurement of mood three to four hours after food intake is more likely to detect 5HT mediated effects. The study by Keith et al. (1990) took place over the long-term, assessing mood only once at the end of each diet week in the evening using the version of the POMS which asks subjects to describe how they felt on that day and for the past week. In contrast, the other studies were short-term studies and rated mood at a given time. Furthermore, in this study, subjects participated in physical activity, whereas in the other experiments (Spring et al., 1983, 1986; Lieberman et al., 1986; Pivonka and Grunewald, 1990) no physical exercise was involved, thus it is possible that there was a diet-exercise interaction in the results reported by Keith et al. (1990) which may help to explain the different findings. It is also possible that the palatability of the moderate and high-carbohydrate meals in this study had more of an effect on mood than the low-carbohydrate meals.

Sherman (1987), Pate and Brun (1989), support the findings of Keith et al. (1990) where feelings of depression, tiredness, and irritability were reported in each study after the consumption of diets low in carbohydrate content for a period of three days. Proposals for such a severe change in mood states have been made although they are totally hypothetical in nature. For example, it has been suggested that perhaps the fact that the subjects were not accustomed to consuming such a poor carbohydrate diet could explain in part why it had such adverse effects on the subjects' mood. Work carried out by Rosen et al. (1982, 1985) lends some support to this theory. In contrast, Morgan et al. (1988) did not find any dietary effect on athletes' mood state. Mood disturbances and feelings of dysphoria were induced by an increase in physical activity, but not by any changes in macronutrient selection.

The fact that mood was assessed at the end of each week over a three-week period, three hours after the last meal immediately casts doubt over the statistical significance of the results by Keith et al. (1990). No pre-baseline was obtained for mood before the onset of the experiment. Any changes in mood could have been attributable to a variety of extraneous factors rather than the independent variable being tested. Nonetheless, Keith et al. (1990) have to be commended for the design of their study, whereby an attempt was made to measure the long term effects of nutrients consumed in a natural setting on athletes' mood state.

The Effects of Carbohydrates on the Mood State of the Obese

Although it is not my intention to focus on the effects of nutrients on the behaviour of the obese in this chapter, two obese studies conducted by Rosen et al. (1982; 1985) will be reviewed since they appear to be of some relevance. In the first experiment Rosen et al. (1982) conducted a comparative study to investigate the effects that two different kinds of diets (a carbohydrate-rich diet versus a carbohydrate-poor diet) would have on subjects' psychological state as well as on subsequent appetite after

being fed a baseline diet for two weeks where adlibitum eating was permitted. The subjects involved were eight female obese in-patients and weighed between 63-108 kg (they were between 24%-98% above ideal body weight, as determined by the Metropolitan Life Insurance Company Norms). Subjective mood was measured by using the State-Trait Anxiety Inventory (STAI-T: Spielberger, Gorsuch and Lushene, 1970) and the Beck Depression Inventory (BDI: Beck et al, 1961), before and after the administration of the test food.

Their findings illustrated that the mood state of the subjects on both the isocaloric carbohydrate containing diet (fat 36%, protein 35%, carbohydrate 29%) and the protein-rich, carbohydrate-poor diet (fat 64%, protein 35%, carbohydrate 1%) did not differ significantly. There was no evidence that the carbohydrate restricted protein-supplemented diet enhanced mood any more than a carbohydrate containing diet. Although, Rosen et al. (1982) did find that, during the first seven days, both diets had more detrimental effects on subjects' psychological state; more feelings of dysphoria and negative attitudes were reported when compared to their psychological state during an ordinary, weight-maintaining baseline diet. However, by the end of the treatment programme, day-to-day fluctuations in mood and other psychological effects were not significantly different to those reported during a pre-treatment free-feeding diet.

A later experiment (Rosen et al., 1985) confirmed their previous findings to a large extent. Again, obese patients (this time twenty out-patients who weighed between 70 and 112 kg and were between 30-113% above ideal body weight) were given diets which were either carbohydrate restricted (820 kcal: 58% protein, 42% fat) or contained some carbohydrate (1000 kcal: 42% protein, 30% fat, 28% carbohydrate) over a sixty-four day period. A within-subjects crossover design was used which meant that the experimental diets had two reverse sequences. The first sequence consisted of ten weeks where ten of the twenty subjects were expected to follow the folowing diet procedure: two weeks of baseline diet (in which free-feeding was

permitted), two weeks of a minimal-carbohydrate diet, two weeks of a carbohydrate-containing diet, then subjects returned to two weeks of the minimal-carbohydrate diet and two weeks of the carbohydrate-containing diet. The fact that each subject was given two different diets, both for two experimental phases meant that responses to the two diets could be compared in the same subject. The other ten subjects followed the other sequence which contained the same diets but in reverse order. This procedure meant that order effects and time effects could be controlled for to some extent.

Mood states were measured by using the Multiple Affect Adjective Checklist (MAACL). The three main scales being anxiety, depression and hostility. The Tennessee Self Concept Scale (TSC) was also used to assess self-esteem or psychological well-being. Analysis of mood scores on the MACCL showed that both the low-calorie diets caused changes in the patients' mood state (although the changes were small), when compared to their habitual eating behaviour, as shown in the baseline period. For example, a greater decrease in anxiety, depression and hostility was reported on the MAACL during the weight loss programme when compared to the baseline diet scores. Any fluctuations in mood during the dieting phase were not correlated with the macronutrient composition of the diet. The most positive effect on mood was reported after the first two weeks of dieting, irrespective of the experimental diet being used.

Furthermore, the data from the TSC revealed that the scores on self- esteem increased during the weight-losing period when compared to the scores during the baseline phase. Similar to the findings for mood fluctuations, the alterations in self-esteem were more related to the duration of the weight loss diets, as opposed to the nutrient properties in either of the experimental diets. The findings from this study and from that of Rosen et al. (1982) are of some relevance, in that mood fluctuations were not attributable to the macronutrient content of the diet. Of particular interest was that the high-protein diets did not cause mood deterioration in the obese subjects as would be

expected according to the 5-HT hypothesis. This lends little support to the proposal by Wurtman et al. (1981) that carbohydrate ingestion improves mood in some obese subjects, particularly those suffering from Carbohydrate Craving Obesity (CCO), whilst high protein foods do not.

Unfortunately, the results by Rosen et al. (1985) are not representative of the general population, in that a non-dieting control group was not included in the study. This makes it difficult to draw any conclusions about food and its effect on normal psychological states. From these data it is possible, for example, that the enhanced mood reported by the obese subjects is attributable to other psychological factors related to improved body image due to weight loss. Nevertheless, the studies are of some relevance, in that they lend little support to the carboydrate-serotonin hypothesis relative to obese individuals.

De Castro (1987) postulated that if the ingestion of certain nutrients cause alterations in subjective mood state, then it is feasible that the actual quantity of the nutrient consumed will correlate strongly with mood state. For example, high doses of a specific nutrient should have a strong correlation with any change in mood, whilst a reduction of this particular macronutrient should induce a relatively strong negative correlation with mood. In short, the consumption of a specific nutrient should be significantly related to the individual's psychological condition thereafter.

In this study which took place in a natural setting thirty-eight student volunteers were requested to self-monitor their food intake by writing down everything they consumed in a diary (including the precise time of food intake, as well as the exact amount and how food was prepared) for nine days before submitting the diaries to the experimenter for macronutrient analysis. Subjects were also requested to measure their mood on the elated-depressed scale, tired-energetic scale, and anxious-tranquil scale, using a three seven point Likert-Like bipolar mood rating scale prior to each meal.

After conducting a variety of bivariate and multiple correlation analyses on the relation of the three mood scales with nutrient consumption, it was revealed that each mood scale as well as the sum of the three mood scales failed to show any significant correlation with prior or subsequent macronutrient ingestion. It had been suggested that perhaps there was not enough variance in macronutrient intake to influence mood state. However, De Castro (1987) pointed out that this was not the case and that the average standard deviation for fat, protein and carbohydrate was 58 kcal, 132 kcal and 122 kcal.

De Castro (1987) did report a direct association between macronutrient intake and subjective mood state over a long-term period, (nine days). The total intake of each nutrient ingested by subjects over nine days was correlated with the average scores of each mood scale over nine days for each subject. De Castro (1987) found that a high intake level of protein concentrations in subjects resulted in an increase in depression on subjects' self-rated mood scores whereas the ingestion of carbohydrate-rich food was correlated with low depression over the long-term. Furthermore, De Castro's findings (1987) on the consumption of high-carbohydrate foods failed to support the neurochemical hypothesis that carbohydrate-rich food induces feelings of sleepinesss and fatigue in normal subjects. Rather, De Castro (1987) found the opposite was true, in that after carbohydrate consumption subjects' self-rated energy scores increased. Similar findings in relation to the effects of carbohydrate on psychological state have been reported by other researchers (Keith et al., 1990) who also recorded mood scores over the long-term. These results do not concur with some of the results from short-term studies (Spring et al., 1983, 1986, 1989; Lieberman et al., 1986; Pivonka and Grunewald, 1990). It may be that any mood alterations induced by nutrient intake in the short-term may differ from those induced as a result of long-term intake of a particular nutrient or nutrients.

De Castro's conclusion (1987) that food rich in a particular macronutrient has to be consumed over a long period before it induces significant effects on the overall mood state of an individual is supported by previous findings of Fernstrom et al. (1979) and Gelenberg et al. (1982/3). In a long-term study, high doses of protein in a diet fed to subjects over a five-day period resulted in a fall in the tryptophan and tyrosine levels and produced feelings of depression (Fernstrom, 1979). This is in accordance with the theory that protein-rich food reduces the plasma tyrosine ratio and brain catecholamines which ultimately induce a depressed mood state, whereas carbohydrate-rich food produces the opposite effect.

It is difficult to form any sound conclusions based on these findings, however, as it is not certain how realistic it is to provide long-term results on mood induced by nutrients, as so many other extraneous factors can interact with mood, particularly over the long-term. De Castro (1987) himself indicated that these findings are based on correlational analysis which has many limitations particularly with repect to inferring casual relationships. In this study results were derived from subjects' diaries and self-reported mood questionnaires, and therefore no measurement of individuals' plasma levels or neurotransmitter levels could be made. This means that one cannot unequivocally conclude that any fall in tryptophan or tyrosine levels was induced by the ingestion of high protein foods as the fall in tryptophan levels is mere speculation.

Consequences of Methodological Problems

The difficulties outlined in several of these studies make it difficult to establish if any firm relation exists between dietary components and mood. After reviewing the literature it appears that there is reason to conclude that the carbohydrate-serotonin relationship in humans is less well established than it is in animals. In addition to the methodological problems outlined above there are a number of other important points to note.

First, it has been found that the increase in tryptophan ratio subsequent to carbohydrate consumption in humans, when compared to the recognised change in the trypotophan ratio in rats is relatively small, and may not be great enough to have a significant impact on tryptophan levels (Ashley et al., 1985; Young, 1991).

Secondly, there have recently been claims that the evidence supporting the theory that a meal high in carbohydrate enhances brain serotonin is merely circumstantial and that alterations in brain tryptophan are not necessarily directly associated with food or any behavioural change that food might induce (Young, 1991).

Third, often when high-carbohydrate food has been cited as inducing alterations in behaviour in normal subjects or those with affective disorders, no control group has been included or protein food has served as the control for carbohydrate food. Yet, apparently, carbohydrate increases brain tryptophan levels, whilst protein decreases it. Protein does, however, increase the amount of tyrosine entering the brain. Such findings may ultimately result in an interaction of both effects (Spring et al., 1986). Thus, there is insufficient evidence that changes in behaviour are specific to carbohydrates.

Fourth, the validity of the questionnaires used to define the "Carbohydrate Craving Syndrome" by Wurtman et al. (1981) is questionnable and there are to date few studies of carbohydrate ingestion in an unselected population.

Fifth, alterations in plasma amino acids has rarely been measured in normal subjects or in patients with disturbances of affect to determine if changes in behaviour are mediated or correlated with a reduction or increase in plasma tryptophan levels.

Sixth, when examining diet-behaviour relations the greater context of any dietary manipulation should be borne in mind. A dietary component is unlikely to exert its

behavioural effects in isolation (Anderson, 1986). For example, a diet in which the amount of protein is altered immediately brings about a change in the fat and/or carbohydrate ratio. Thus, such a study might equally be regarded as examining the effects that an increase in carbohydrate exerts on behaviour given that the decrease in protein has increased the carbohydrate ratio.

Seventh, it has also been established that the ingestion of other nutrients such as protein (as little as 4%) with carbohydrate will dramatically reduce the increase in serotonin (Young, 1991). This fact is of great relevance when examining the carbohydrate-mood connection in normals and subjects with affective disorders and lends support for the rejection of the "Carbohydrate Craving Syndrome" (Wurtman et al., 1981). On examining the carbohydrate intake of so-called carbohydrate cravers there is evidence that their carbohydrate level is not "abnormally" high. Although sugar has been cited as the major nutrient craved, sugar is frequently consumed in combination with dietary fats (Drewnowski and Greenwood, 1983). In studies by Wurtman et al. (1981, 1984) it has been revealed that as much as 68-98% of the nutrients ingested were derived from a mixture of fat and carbohydrate.

Lieberman et al. (1986) proposed that carbohydrate craving played a significant role in the onset and maintenance of obesity. In their study of seventy obese subjects they concluded that fifty-one of them suffered from carbohydrate craving. However, on analysing the food consumed in the study by Lieberman et al. (1986) it seems apparent that most of these foods contained more fat than carbohydrate (fat content 40-54% calories). This would presumably prevent any significant alteration in the TRP/LNAA ratio and cast some doubt over the use of the term "carbohydrate craving" which is perhaps a misnomer. The fact that the experimental data are contradictory makes it difficult to make an objective review of the literature on carbohydrate and mood.

Eighth, the problem with the administration of dietary constituents such as tryptophan is that the behavioural changes which tryptophan has been reported to induce may be of little significance outside the laboratory. Many of the studies which administered tryptophan to patients, who reported an increase in drowsiness and tiredness thereafter, received 1 g or more of tryptophan which may be essential to induce sleepiness in humans. Yet, this amount almost equates the recommended daily amount to be ingested by humans (Young, 1991). One of the most obvious limitations of studies of single amino acids such as tryptophan, which are similar to psychopharmacological experiments of pure substances is that they do not reflect how people eat under natural conditions. In these studies there is also the difficulty of ascertaining that the placebo used does not induce any behavioural effect. There have been claims, for example, that valine or leucine placebos can influence the amount of tryptophan entering the brain and the placebo aspartame used as a placebo in sugar studies can increase brain tyrosine (Young, 1991).

The mere fact that several of the human food studies which have reported food-mediated alterations in the plasma tryptophan ratio have indicated that the effects are subtle (Lieberman et al., 1986) suggests that the relation between the consumption of carbohydrates and serotonergic transmission has been spuriously inflated and any reports of sleepiness subsequent to carbohydrate consumption may be connected to a third variable; something other than tryptophan and serotonin. In effect, there are several other peripheral mechanisms which could affect the brain subsequent to carbohydrate intake and which may explain changes in mood and behaviour. For example, it has been proposed that the physiological effect of peptides (derived from food proteins) may significantly impact mood and behaviour (Smith and Gibbs, 1976).

Conclusion

This review chapter has considered the relation between food constituents, namely tryptophan, protein and carbohydrate and whether they promote psychophysiological effects on human behaviour. Based on some of the findings it would appear that food and food constituents can induce behavioural effects. Nonetheless, the possibility that everyday eating behaviour would have any dramatic effects on an individual's behaviour when normal amounts of food are eaten may seem unlikely. Rather, the effects seem at best to be subtle and not of clinical importance. Extraneous factors that may also interact with nutritional effects on behaviour should always be taken into consideration when studying the diet-behaviour relation. Perhaps it is for this reason that there is no consensus among scientists and laymen regarding the effects of food on behaviour.

Experimental findings have shown that the three principal hypotheses which have been proposed as the underlying mechanisms of the carbohydrate-mood relation (i.e. hypoglycaemia, psychological effects and the role of neurotransmitters), each have their limitations, some more than others. Moreover, while at first glance, there appear to be more empirical findings which support the relation between carbohydrates and neurochemicals and accompanying changes in mood and behaviour, inspection of the literature indicates that there are several methodological flaws in some of the studies which purport to support such a hypothesis.

By reviewing the relevant studies on diet-behaviour relations, several methodological problems have emerged which merit attention in future studies if a greater understanding of the effects of food on behaviour is to be gained. The literature reveals weaknesses of inadequate design, poor control of psychological factors, failure to include control groups and/or a fasting control group, absence of baseline measures and lack of control of prior food intake. Moreover, methodological issues concerning test devices used to measure behavioural change, the timing of mood assessment, nutrient

dosage, endogenous processes, normal eating habits, individual variation all require careful consideration before conducting a study on food and behaviour. The measures used to assess behavioural change and the selected timing of mood testing (as earlier or later mood assessment may miss the main effects) are of major importance in this research.

Chapter 2-The Effects of Carbohydrates on Satiety

Introduction

Much effort has been made over the years to gain a deeper understanding of the specific characteristics of feeding behaviour in humans. Focus has been placed on certain nutrients which affect food intake (Blundell and Hill, 1985; Booth, Campbell and Chase, 1970a; Booth, Chase and Campbell, 1970b; Hill and Blundell., 1986; Rolls, Hetherington and Burley, 1988) as well as an attempt to discover the exact processes which work to control human feeding behaviour (Le Magnen, 1971; 1969; Bellisle, 1979; Bellisle and Le Magnen, 1980; Stricker and Verbalis, 1990). Before the onset of research in this field (Hashim and Van Itallie, 1965; Jordan, 1969), it was more or less accepted that nutrient intake was regulated predominantly by physiological mechanisms (Bernard, 1872). This assumption was partly based on empirical evidence suggesting that other organisms regulate feeding physiologically. Animal studies have shown that although intake regulation in animals is dependent on many physiological mechanisms, it is stable and efficient (Adolph, 1947; Booth, 1972a; McHugh and Moran, 1978). Some human studies have found similar effects with normal-weight subjects. For example, Schachter (1967) proposed that the control of body weight and the regulation of food intake in normal-weight individuals operated on a physiological level which was precise and automatic.

There is, however, much debate as to whether nutrient regulation in normal-weight humans is due to their sensitivity to recurrent physiological cues. Some investigations have revealed that such views may be more applicable to the animal regulatory system than to human regulation, which is rather fallible. There is some evidence that humans have the ability to regulate their energy intake in the long-term so as to accommodate

the effects of different preloads, although it has been recognised that the amount is not always accurate or perfect (Porikos, Booth and Van Itallie, 1977; Porikos, Hesser and Van Itallie, 1982; Stockley, Jones and Broadhurst, 1984; Hill and Blundell, 1987). Several days may elapse before caloric adjustment occurs and the energy regulatory process may differ according to the individual, some being more susceptible than others to caloric manipulation (Campbell, Hashim and Van Itallie, 1971; Wooley, 1971; Spiegel, 1973; Porikos et al., 1977). Regulation appears more accurate when cognitive cues and self-monitoring are curtailed by preloads being administered orally or intragastrically using feeding machines (Hashim and Van Itallie, 1965; Campbell et al., 1971; Jordan., 1969; Walike, Jordan and Stellar, 1969). The regulatory process differs as a result of short and long-term energy intake. The satiating effects of certain foods may not operate immediately, but may be effective over several days or so.

Thus, caution must be exercised when interpreting the results from short-term studies on the human regulatory process. Conflicting results have been found in short-term studies. Some experimenters have shown that food intake is modified after a caloric preload (Hill, Leathwood and Blundell, 1986; Birch and Deysher, 1986; Kissileff et al., 1984; Pliner, 1973; Booth et al., 1970a, 1970b; Booth, Lee and McAleavy 1976; Hill, 1974; Nisbett, 1968a), whilst others have failed to find any compensatory behaviour after preloads varying in energy value (Sylvestre et al., 1989; Wooley, Wooley and Dunham, 1972; Rolls, Jacobs and Hetherington, 1986; Spiegel, 1973). Other studies have found effects of cognitive factors rather than biological mechanisms (Woolcy, 1971; Nisbett and Storms, 1975). While other findings suggest that humans will compensate more readily for decreases than for increases in energy intake (Mattes, Pierce and Friedman, 1988).

What has emerged from these studies is the recognition that human ability to regulate consumption of food or nutrients is not straightforward. It seems apparent that eating

behaviour in humans is determined not solely by physiological processes, but by psychological factors.

Specific nutrients, namely carbohydrate, protein and fat may have different effects upon a person's food intake. However, while it has been proposed that the macronutrient composition of particular foodstuffs are responsible for the onset of satiety for specific nutrients, there is ambiguity regarding the exact role of macronutrients in the satiety process or the specificity of satiety whereby satiation is attained for one nutrient or food, but not for others (Rolls et al., 1981).

A review will be made of the literature of 1960-1994 on the satiating effects of carbohydrates, with protein as a comparator. Prior to reviewing the literature in detail, it is necessary to consider various internal and external processes that have been proposed as factors influencing human food regulation. Although these mechanisms are too extensive to consider in depth, the fundamental processes implicated in feeding behaviour will be described. Physiological mechanisms will be considered as well as orosensory, conditioning processes and psychological components. Basic methodological issues and conceptual issues will be discussed and concluded with a detailed review of the literature. The animal literature is largely excluded from this review for it cannot be assumed that results from animal studies will automatically apply to human eating behaviour. Although some reference is made to studies using obese subjects, space does not permit a full study of this literature, thus the emphasis of this review will be studies using normal-weight subjects.

Defining Satiety

The verb to satiate according to dictionary definitions means to "supply to satisfaction or capacity" and the noun satiety refers to the state of being satiated or the feeling of having eaten to excess. There is, however, no general consensus on the technical definition of satiety. Thus, in this chapter definitions of satiety will be used in their

broadest sense, that is satiety refers to the state achieved by the organism after food intake. In the literature reference is frequently made to the satiating "efficiency" or "power" of a particular foodstuff. Thus, satiating effects will be interpreted as meaning that the ingestion of a specific nutrient or nutrients induce satiety and that certain nutrients may speed up or slow down the onset of this state. Because the concept of satiety cannot be directly measured, the cessation of eating in the presence of food is frequently taken to reflect the state of satiety in food experiments. Finally, satiety can be either specific or general. Satiety for a specific foodstuff may mean that the desire to eat that particular food substance has stopped but other foods may be still be eaten (Rolls, 1986). Although nutrient-specific satiety will be referred to later on in this chapter the main focus will be on general, and not specific satiety.

Mechanisms of Satiety

Physiological Mechanisms

In humans physiological or internal factors which play a major role in hunger and satiety include physical feelings such as stomach contractions, stomach or gastric distension and rate of gastric emptying (Bray et al., 1976). Food deprivation tends to induce changes in gastric sensations and in blood sugar concentration. There is evidence that hormones affect hunger and satiety. The release of gastrointestinal hormones such as cholecystokinin, somatostatin and glucagon are released peripherally from the gut during eating and can act indirectly on the brain via the vagus nerve (Smith and Gibbs, 1976), whilst other hormones/peptides such as bombesin released from the gut in response to nutrient intake affect the brain directly (Smith and Gibbs, 1976). These hormones are released in response to the presence of nutrients at different sites in the gut, mainly in the small intestine. Glucose and amino acids have affects in the duodenum, but fat is particularly effective and in addition increases satiety if present in the terminal ileum (Welch et al. 1985). Growth hormones such as oestrogen, and the secretion of insulin from the pancreas, have also been cited as a

contributory factors in signalling appetite and satiety (Friedman and Stricker, 1976). Insulin induces an increase in consumption of other nutrients and increases the absorption of glucose. Insulin promotes storage of nutrients as fat and high levels of insulin are correlated with obesity (Lovett and Booth, 1970), even although the absorption of glucose which precedes insulin release suggests that the enhanced levels of insulin should correlate with the termination of eating.

Central Processes

Various brain mechanisms have been proposed as principal factors in signalling the onset and cessation of eating. It has been theorised that the hypothalamus has receptors which influence food intake and it also controls various physiological conditions, such as glucose levels and apparently fat reserve levels correlate with the biological set point (Mayer, 1955). However, more recent research has shown that central processes such as hypothalamic receptors do not control feeding and satiety (see Friedman and Stricker, 1976 for a review), although it is possible that they act in conjunction with peripheral processes to exert a primary influence on eating.

Peripheral Processes

Peripheral mechanisms such as hepatic and duodenal glucoreceptors have been proposed as processes which signal hunger and satiety to the brain via the vagus nerve (Woods et al., 1980), whilst other evidence favours hepatic oxidative metabolism and not glucose as the principal mechanisms which relays signals to the brain (Friedman and Stricker, 1976).

To date it is not entirely clear which physiological mechanisms control feeding behaviour. Physiological interpretations concerning regulation of food intake and body weight in humans are so varied and so far no specific physiological theory indicating which physiological processes are involved in the food control process has been widely accepted. However, from the literature it may be assumed that humans can

regulate food intake "internally" by relying solely on physiological mechanisms, but the hypothesis requires to be tested. Non-physiological factors are frequently present and interact with the regulatory process. Alternative explanations for food regulation and for the effects of specific nutrients on satiety have been offered. Amongst the most common non-physiological processes are conditioning, orosensory and psychological processes.

The Process of Conditioning

The process of conditioning implies that humans partly regulate their intake with an anticipatory regulatory system based on past learning experiences. The physiological post-ingestive effects of a specific nutrient or nutrients are anticipated and conditioning may even stop eating before satiation is fully reached. Some researchers have proposed that conditioning in humans ultimately determines how much a person will eat in anticipation of his or her nutritional requirements (Booth, 1986a; Le Magnen, 1967; Rozin, 1982). Similarly, Le Magnen (1967) proposed that rats, for example, are able to regulate the amount of food ingested due to an anticipatory regulatory mechanism, in that the quantity of food consumed is determined greatly by factors such as the length of time before the next meal so that they consume enough nutrients to satisfy their nutritional requirements until the next feeding period.

Booth et al. (1976a) found that consumption of a test meal with a distinctive flavour could be conditioned by starch drinks differing in starch content (65% versus 5%). This implies that the subjects learned to anticipate the post-ingestive effects of starch and alter food intake. According to Booth (1976a) this was due mainly to an increased sensitivity of the richness of the foodstuff rather than any conscious awareness of the satiating effects of the food thereafter. Conditioned satiation by starch is probably easier with unfamiliar foods (Booth, 1976a). Subjects were more likely to reduce intake towards the end of the meal, possibly because the yogurts served as dessert were similar in flavour to the starch drinks.

Insulin release may be induced in humans in response to the sight and thought of food (Rodin, 1978; Sjostrom et al., 1980; Sahakian et al., 1981). This physiological condition is apparently elicited through a conditioning mechanism which also operates in animals (Booth, 1986a; Booth et al., 1976). A rise in gastric secretion has also been found in humans by merely thinking about food (Moore and Schenkenberg, 1974). Wooley and Wooley's work on salivation (1973) provided evidence suggesting that salivation may operate as an anticipatory conditioned response to food that was visually available. They found that the sight of food elicited salivation and that the level of salivation varied depending on the palatability of the food and level of deprivation (Wooley and Wooley, 1973). It was also suggested that the volume of saliva may be useful as a measure of appetite (Wooley and Wooley, 1973).

Sensory and Internal Determinants: a Possible Overlap Between The Two

Sensory-Specific Satiety

Oropharyngeal factors exert a large influence on satiation in humans, which perhaps to some extent overrides the influence of physiological mechanisms. Wooley (1972) found that humans are more responsive to the sensory properties of food than to its nutrient properties. This finding would appear to support Le Magnen's theory (1963) that food regulation in animals is due to a conditioning process. Cabanac (1971) proposed that the sensory characteristics of foods may often override their macronutrient content or caloric concentration. Sensory effects are elicited through the smell, taste, texture and temperature of food. Le Magnen (1967) coined the term "sensory-specific satiety" which embodies the concept that the sensory properties of a food contribute greatly to the cessation of ingestive behaviour and temporarily inhibit eating, particularly for foods which share similar sensory characteristics. In Le Magnen's words (1967):

"the excess eating of a specific food with the repetitive oral stimulation it represents induces an immediate decay of response to the same food, or in other words, a sensory-specific satiety". (p.25)

It is possible that satiety may be achieved for one particular foodstuff but not another, in that satiety may be specific only to the sensory characteristics of a specific foodstuff. This may result in reduced responsiveness to and intake of that particular food compared to other foods (Le Magnen, 1967; Rolls et al., 1981a, 1982). The development of satiety is more rapid when one food is consumed in isolation, rather than in combination with other foods (Katz, 1934). It may be assumed that sensory-specific satiety is significant in helping the organism maintain a healthy, well-balanced diet with various nutrients.

In an effort to control for orosensory factors in experiments with oral preloads, feeding machines have been used where the sensory qualities of foods are minimised (Hashim and Van Itallie, 1965; Jordan, 1969; Campbell et al., 1971). Intragastric feeding, where different types of liquids varying in nutrient composition are fed directly to subjects' stomachs via tubes has also been carried out by several researchers (Grinker, Cohn and Hirsch, 1971; Jordan, 1969; Spiegel and Jordan, 1978). Jordan (1969) found that human capacity for food regulation varied according to oropharyngeal and gastric factors. It was also found that oropharyngeal factors (which may to some extent ignore the gastric mechanisms in operation after food intake) were strongly correlated to the regulation of appetite and to the production of satiety.

The Alliesthesia Phenomenon

Cabanac (1971) claimed that the pleasantness of tastes, odours and other stimuli change according to the metabolic state of the body. In other words, as biological needs are met by a certain nutrient or nutrients, alterations in subjective pleasantness

may arise which consequently produce alterations in the internal state of the organism. This phenomenon has been termed "alliesthesia". This concept emerges from the earlier work of Schachter (1967, 1968) who differentiated between 'internal' and 'external' factors in relation to food control in humans. The development of alliesthesia is gradual with the maximal alteration in hedonic stimuli taking place after about forty-five minutes (Cabanac, 1979) as absorption of the nutrient occurs and the need for a specific foodstuff is met.

In his earlier work (Cabanac, Minaire and Adair, 1968) it was demonstrated that intragastric loads and oral loads had very little difference on alliesthesia, in that both were equally potent. This implies that the sensory stimuli are not necessary to change one's perception of pleasantness. In a later study (Cabanac and Fantino, 1977) it was suggested that alliesthesia was mediated by duodenal receptors, in that once the food was emptied from the stomach duodenal receptors controlled the signals which produce changes in pleasantness of certain foods. Scherr and King (1982) reported a greater reduction in the pleasantness ratings for foodstuffs high in energy density compared to low energy density foods. These results supported Cabanac's (1971) theory that the caloric content of the preloads is controlled by gastric and duodenal nutrient receptors, and changes in the hedonic response to the food are induced by the physiological processes in operation. However, the fact that orosensory and psychological factors were uncontrolled in Scherr and King's study (1982) makes it difficult to interpret these results as evidence that gastric and duodenal receptors monitor the energy content of foods.

Further, it seems apparent from Cabanac's findings that external factors play a significant role in feeding. It has been found, for example, that response to specific tastes can be influenced by, or themselves influence, internal nutritional states. Cabanac (1973) referred to "negative alliesthesia" to describe the phenomena of food aversion subsequent to intake of a specific food substance, the most common example

being the aversion to sugar loads following consumption of carbohydrate-rich food. However, it is thought that, although this change is dependent on the orosensory qualities of a food and the palatability of the foodstuff, the mechanism involved in the "negative alliesthesia" phenomena is also related to a physiological condition. The fact that many highly palatable foods can only be ingested in relatively small quantities in comparison to foods which are less palatable supports the hypothesis that there is some internal mechanism in operation which is related to the satiation process. It is expected that after the ingestion of rich foods (such as foods high in sugar and fat) the onset of satiation is faster than after the ingestion of less rich foods. The alliesthesia phenomenon has significant implications for the understanding of palatability since it appears to demonstrate a complex interaction between external and internal factors.

Booth (1976) provided further support for the interaction between external and internal factors when he showed how the palatability of a certain foodstuff together with its orosensory characteristics may be modulated depending on the state of satiety in an individual at a given time. Volunteers were requested to rate their willingness to consume a certain type of food before or after ingestion of a meal. Foods that were considered highly palatable before the meal were regarded as less attractive after the meal. In contrast, the food items that were seen as less palatable before the meal, were seen as more palatable after the meal. However, Booth (1976) did find that some of the highly palatable foods, rich in texture, high in calories and characteristic of the kind of foods eaten to terminate a meal, were still considered extremely palatable even after the subjects had reached satiation. Pliner, Polivy, Herman and Zakalusny (1980) in their study also showed that palatability is reduced when a single foodstuff is ingested in large quantities.

Other researchers have not been successful in replicating the alliesthesia phenomenon, in that non-nutritive, sweet-tasting substances such as mannitol (Cabanac and Fantino, 1977), cyclamate (Wooley et al., 1972a) and aspartame (Blundell and Hill, 1986b)

have also produced alliesthesia to some degree. Such findings undermine the role of physiological factors in altering the hedonic response to subsequent food. Booth et al. (1970a) proposed that the sensory characteristics of food are more likely to have an immediate impact on altering hedonic response and on subsequent intake, since the time course of the metabolic effects of food consumption is not immediate. Thus, whilst alliesthesia is associated with alterations in pleasantness for a food related to the metabolic consequences of ingesting that food this effect may take longer to achieve than sensory-specific satiety which is the result of alterations in pleasantness produced by the orosensory properties and cognitive factors of the food consumed. Further, whilst the alliesthesia effect is gradual, that of sensory-specific satiety is rapid and short-lived. It has been proposed that the orosensory and cognitive processes associated with sensory-specific satiety direct and guide food intake and selection in the short-term (specifically during the meal), whilst physiological processes related to the alliesthesia phenomenon exert more influence over feeding in the long-term.

Considering the importance of orosensory factors, one would imagine that control of the taste and smell of specific nutrients would be fundamental in food experiments. Some experimenters have attempted to reduce external influences by holding constant sensory and cognitive signals when administering preloads. In such cases subjects often fail to alter subsequent caloric intake accurately on a physiological basis (Pliner, 1973; Spiegel, 1973; Wooley, 1972). Some of the more recent studies where cognitive factors have been controlled fail to concur with earlier results in studies where cognitive and sensory factors were uncontrolled and caloric compensation was recorded (Schachter, 1967; Schachter, 1968). This seems to support the fact that, when uncontrolled, sensory stimuli may interact with the learned anticipatory regulators of eating described by Booth (1977b) which may operate on an unconscious level.

Specific Macronutrient Satiety

Evidence has already been presented that satiation for a specific food may be independent of hunger or satiation for another food (Rolls et al., 1981a, 1982a). Thus, the ingestion of carbohydrate-rich foods may ultimately reduce subsequent intake of that macronutrient or foods with similar hedonic properties such as taste, texture, or macronutrient composition, but foods of a different macronutrient composition, such as those high in protein, fat or different in flavour and taste may not necessarily be affected (Hill and Blundell, 1987; Wurtman and Wurtman 1982). The same may apply for the ingestion of protein-rich food, or food more savoury in nature. This implies that humans have the ability to select a nutritionally balanced diet. After conducting a study on infants where it was observed that macronutrient regulation operated in infants (Davis, 1928), Davis (1939) proposed that the self-selection of a nutritionally adequate diet in humans may be attributable to "....the existence of some innate, automatic mechanism...of which appetite is a part".

The neurochemical hypothesis discussed in chapter 1 with respect to mood (Fernstrom and Wurtman 1972; Fernstrom, 1985) has also been proposed in relation to appetite and macronutrient regulation (Blundell, 1985; Spring, 1986; Wurtman and Wurtman, 1986). It has been postulated that the brain is able to regulate intake with the correct nutrient ratios as well as serotonin ratio. According to this theory, protein intake, for example, should decrease subsequent protein consumption, rather than carbohydrate or fat intake, whilst carbohydrate ingestion should reduce subsequent carbohydrate intake and not protein or fat. Furthermore, the differential effects of various nutrients on satiety and subsequent food choice and food consumption have been examined (Kissileff, 1984). Responsiveness to nutrient composition has been shown to some degree in terms of differential satiating effects of various nutrients, such as protein which may be more satiating than carbohydrates, (Hill and Blundell, 1986; Booth et al., 1970b) which have been reported to stimulate an increase in hunger and food intake (Geiselman and Novin, 1982; Rodin et al., 1985).

This theory suffers from the same limitations as those of the neurochemical hypothesis relative to mood (see chapter 1). The data in this area are controversial and other studies have failed to support specific-macronutrient satiety. Further studies are required to examine more closely whether humans have the ability to choose foods with protein or carbohydrate or fat ratios which correspond to their previous energy intake. Rolls, Rolls and Rowe (1983) failed to detect any sign that specific macronutrients in the preloads were strongly correlated to satiety. However, they did find that the sensory characteristics of the food ingested exerted a strong influence on the development of satiety and the ratings of pleasantness for ingested food were significantly lower in comparison to food that had not been consumed, thereby supporting previous work conducted by Cabanac (1971, 1979). According to Booth (1976) macronutrient-specific hunger in humans is also an illustration of conditioning.

Psychological Factors Related to the Onset of Satiety

It has been proposed that the "psychological experience of hunger or satiety" overrides the physiological condition (Wooley, 1972). That is, the post-ingestive satiation of certain nutrients or foods is determined primarily by individuals' cognitions and beliefs, irrespective of the caloric content of food. Wooley et al. (1972a) reported that even visual cues of milk shakes, whereby some appeared richer in caloric content than others, were more influential in determining subsequent intake than the nutrient components of the drink, or its other properties such as nutrient density or bulk. Pudel and Oetting (1977) have also shown how people's beliefs about what they "think" they have eaten may affect their feeding behaviour, particularly when subject to cognitive manipulations by the experimenter. If the satiating effects of certain nutrients is related to subjects' beliefs about the nutritional value of some foods, then it is understandable that some people may feel that satiety "should" be (and therefore is) experienced after consuming food such as steak. Since steak is considered to be a nutritious foodstuff that will produce certain metabolic effects. It is likely that the

higher the expectations regarding metabolic effects, the less food consumed thereafter. Often individuals feel that they have reached a level of satisfaction well before any metabolic or caloric effects have had time to operate effectively. Wooley (1972) found little evidence to support the viewpoint that the caloric density or nutrient composition of the food consumed influence the internal state of depletion in humans on a metabolic level. Wooley (1976) claimed that it takes approximately forty-five minutes before the nutrients ingested have an effect. This would suggest that food ingested, irrespective of nutrient or calorie content, should not affect intake shortly afterwards. Wooley (1976) also claims that it is not possible for a person to internally regulate food intake based on what has previously been ingested.

Conclusion

The evidence available seems to indicate that physiological cues alone may be inadequate in controlling nutrient intake and macronutrient selection in the short-term (Booth, 1986a). Factors associated with learning, conditioning, cognitions, sensory factors and environmental cues may contribute significantly to the regulation of food intake before the metabolic and caloric factors have full impact on human physiology (Blundell and Hill, 1986c; Booth, 1986a).

Figure 2.1 shows the complex mechanisms involved in the control of energy and macronutrients. Signals arrive in the brain via several sources and such information is organised by the brain and integrated in a way that results in quantitative regulation of energy and nutrient intake.

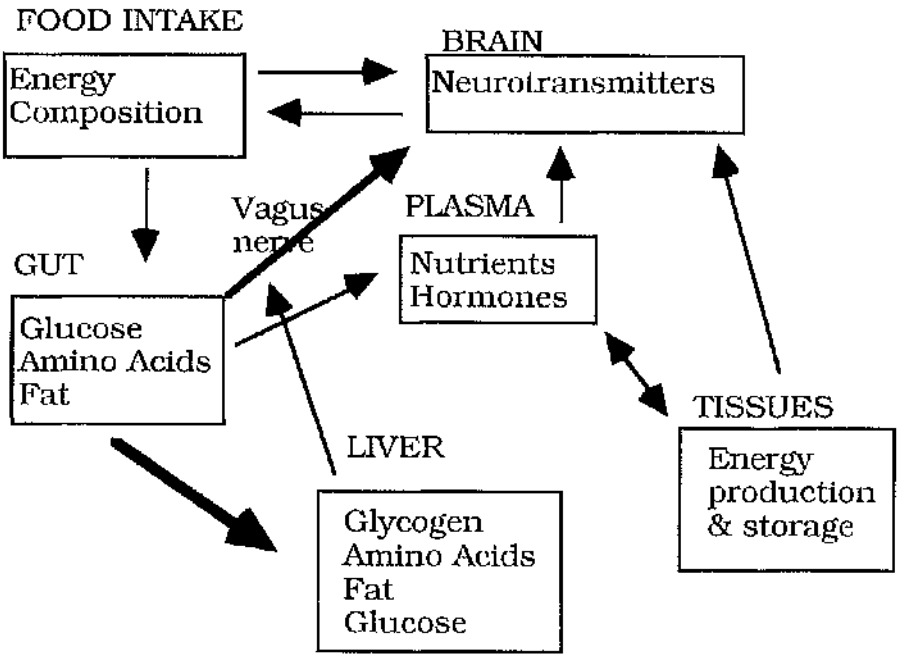

FOOD INTAKE
Energy
Composition
BRAIN
Neurotransmitters
Vagus
nerve
PLASMA
GUT
Nutrients
Hormones
Glucose
Amino Acids
Fat
TISSUES
LIVER
Energy
production
& storage
Glycogen
Amino Acids
Fat
Glucose

FIGURE 2.1

Methodological Issues

In order to address the question of the relative satiating effects of different macronutrients, methods have varied from using preloads of foods rich in one macronutrient to administering capsules containing pure macronutrient; from adding or substituting the nutrient of interest in a meal (Hill and Blundell, 1986); to using liquid nutrients (Friedman et al., 1986) and infusions of macronutrients directly into the bloodstream of participants (Shide et al., 1992). Such diverse approaches may provide different information regarding human eating behaviour.

Short-term and Long-term Studies

Some studies are conducted over the short-term, whilst other findings are from long-term studies. Whilst it may be useful to examine how long and short term regulation are related, it cannot be assumed that short-term regulatory behaviour is the same as the regulatory process for longer-term energy intake. It is necessary to make a distinction between the two when interpreting the results, since both may provide quite different information regarding human eating behaviour.

Most of the studies which will be reviewed in this chapter are short-term studies, although a few long-term studies will be examined. Long-term studies generally provide more information on the physiological mechanisms underlying eating behaviour and on nutrient regulation, as well as on the compensatory processes involved in feeding. They may also provide information concerning the macroanalysis of eating habits and patterns over a relatively long time span (Blundell and Hill, 1986). In contrast, short-term studies are more useful for disclosing information regarding the influence of immediately available internal and external cues on eating behaviour (i.e. factors which are present before, throughout and shortly after a meal).

Use of Preloads

Most of the studies under review examine the effect that a preload has on subsequent food consumption. The preload may be of liquid or solid composition or a combination of both. The purpose of the preload is to examine whether the physiological mechanisms are able to adjust accurately for ingestion of the preload by reducing energy intake in the main meal. The caloric value of the preloads are also frequently manipulated and the effects on subsequent energy intake are assessed. Preloads are often delivered to subjects after a period of deprivation, although this need not always be the case and often vary in size (ranging from as little as 17 kcal; Rolls et al., 1986 to as much as 900 kcal; Wooley, Wooley and Woods, 1975). It is expected that preloading will continue to be used in order to understand capacity to regulate on the basis of nutrient or calorie intake.

Timing of Preloads

The time the preload is delivered to the subjects in relation to the time of the main meal may play a decisive role in subsequent energy intake and regulation. The interval between the preload and the test meal varies across studies and may be as little as ten minutes (Kissileff et al., 1984). Yet presumably it takes at least twenty minutes for the first post-absorptive effects of the food consumed to affect the new energy level (Booth, Toates and Platt, 1976) and perhaps even as long as forty-five minutes (Woolcy, 1976). It is therefore possible that people eat on the basis of other cues in the twenty minutes which elapse from the beginning of ingestion, rather than on the basis of internal sensations of hunger. In contrast, other studies may wait as long as four hours (Spiegel, 1973) to deliver a test meal following a preload. This is possibly too late to assess the post-absorptive effects of the preload. Booth et al. (1970a) found that when glucose was administered immediately before the ingestion of a meal it was less efficient at reducing subsequent food intake than when it was given twenty minutes before the meal. Walike et al. (1969) reported that suppression of subsequent food intake was greater after a fifteen to thirty-minute time gap following the preload.

However, this finding was only applicable to half of the subjects. For the other half, a smaller time gap (one to five minutes) between the preload and the test meal led to a curtailment of subsequent intake. In a later study by Booth (1976) similar results were found. It is possible that such time differences reflect the different objectives of the experiments; short-term effects may be gastric and orosensory whereas waiting several hours to assess post-meal effects may reflect post-absorptive ileal and colonic effects.

Orosensory Factors and Palatability

The palatability of preloads is perhaps the most consistent variable affecting the amount consumed but has often been poorly controlled. If people choose or avoid carbohydrate-rich food this may be due to variations in palatability and/or satiation with, or stimulation by, the taste of the preload. In contrast, it is possible that people eat less when offered protein-rich foods because they are less palatable. Furthermore, food delivered in experiments varies between liquid and solid foods which will undoubtedly affect the eating behaviour of the subjects involved as well as the palatability and attractiveness of the food.

Sample Composition and Selection

Many studies have used small numbers of subjects, some fewer than ten (Kinsell et al., 1964; Pilkington et al., 1960; Yudkin and Carey, 1960; Rosen et al., 1982; Sunkin and Garrow, 1982; Booth et al., 1970b). When small samples are used then the atypical responses of a small number of subjects may distort results. Most studies have used students, university or hospital staff, or obese patients. The latter group are of interest, but their eating behaviour may be atypical, while students are typically in late adolescence, poor, intelligent and radical, compared to the general population. Thus, they are likely to have atypical eating patterns and awareness of dietary issues. Similar problems of generalisability apply to hospital staff. Gender differences are another potential source of variation, especially in food experiments where the differences could be attributable to physiological or psychological factors. Finally, the

majority of subjects have been under thirty and hardly anyone over forty has been studied. Sample composition and selection therefore are important when considering such factors as the reliability of the reported findings of food studies.

Prior Deprivation

Another possible source of variation is the deprivation level of the subjects. It is probable that a subject's prior nutritional state will influence subsequent eating, particularly the metabolism of food intake (Jenkins et al., 1982). Yet some studies include a fasting condition of varying length, whilst others do not.

Basic Experimental Design

In within-subjects designs the order of presentation of conditions is often not fully counterbalanced across subjects. Thus, if presentation of one condition precedes another condition for more subjects then results may be influenced greatly by these order effects rather than by the nutrient itself. Many studies fail to include baseline measures of prior food consumption which is one way to control for individual variation concerning eating behaviour. In the absence of baseline measures, differences between various preloads are difficult to interpret as they could be attributable to individual variations between groups or practice or fatigue effects, particularly when crossover trials are being used. This problem is compounded with the use of small samples and absence of placebo conditions. Even if standardised pre-test meals are given this does not provide an accurate pre-meal baseline measure if subjects still consume other food prior to the test meal.

Expectancy Effects

A further problem is that the observed effects of specific nutrients such as carbohydrate or protein may be attributable to placebo or expectancy effects. In particular, when subjects are aware of what they are eating they may behave according to the "well-known rule" (Poulton, 1989). For example, eating too much sugary

snacks causes irritability or makes children hyperactive. There are also liable to be subtle experimenter effects which may affect eating behaviour differently in different studies.

Applicability of Laboratory Findings to Everyday Eating Behaviour

Studies set in the laboratory often fail to provide realistic alternative activities to eating. People (and rats) may eat more, or differently, when few other behaviours are possible. Subjects are also forced to ingest a preload and have little or no control over the type of preload or dose ingested. Subsequent to the preload they are then encouraged to eat a test meal of some sort whether they are hungry or not, within a fixed time period. Thus, considering the artificiality of these experiments one can only speculate as to the validity of the findings for everyday eating.

Food and Metabolism/Individual Variation

The dynamics of food metabolism, which may vary as a function of individual differences, compounds preceding problems. The effects of age, weight and gender probably affect the metabolism of food. Differences in caloric intake have been noted between the sexes (male subjects often consuming more than the females). Fixed-size preloads may not always be appropriate for subjects' metabolic rate. It might be more useful to scale down energy intake so that it is commensurate with age, weight and sex before determining whether results are significant or not. Further, many of these studies refer to carbohydrates as if all types of carbohydrates are metabolised in a similar way. The absorption of different sugars has been shown to vary and affect subsequent food intake differently (Spitzer and Rodin, 1987; Rodin, Reed and Jamner, 1988; Rodin, 1990). Furthermore, the absorption of starches may be a much slower process than the absorption of sugars and have different effects on the appetitive processes. The role of dietary fibre may also have different effects on satiety. Although fibre will not be discussed here its role in aiding digestion of certain carbohydrate-rich foods and speeding up the satiety process is acknowledged (see

Blundell and Burley, 1987 for a review of the satiating effects of fibre on food intake). There is a need for food researchers to be aware of these differences when theorising about the satiating powers of various nutrients.

Effects of Nutrients in Mixed Meals

Another problem is that some studies reporting the superiority of protein over carbohydrate concerning the delay of hunger have permitted adlibitum ingestion of fat as well as protein (Stock and Yudkin, 1970; Yudkin and Carey, 1960). It could well be argued that the fat content of the diet combined with protein contributed to the satiating power of the protein-rich, carbohydrate-poor foods. It is possible that such an effect might not have been achieved by the ingestion of protein alone. Similarly, Hodgson and Greene (1980) reported that carbohydrates had poor satiating effects, but the carbohydrates in this study were administered in combination with fat (i.e. maltesers). Ingestion of single nutrients may differ considerably from ingestion of mixed nutrients which tends to be the way food is consumed in a natural environment.

Measuring Devices

Most of the studies reported in this chapter have used hunger rating scales and/or food diaries as a means of assessing appetite after ingestion of a particular foodstuff. The validity of such procedures is limited since hunger scales frequently have a low correlation with actual food consumption (Mattes, 1990). According to Le Magnen (1985) evidence relating subjective hunger ratings to eating behaviour has often been relatively weak. Hunger ratings are based on verbal reports yet people differ in their perception of hunger. Spiegel (1973) proposed that some people may not have the ability to distinguish between feelings of hunger and those of satiety. Hence feelings of hunger and satiation are not always cut and dried for many people and the validity of such ratings, which constitute the bulk of some experimental findings, ultimately rests with the individual.

Conditions in which hunger scales are used tends to vary across studies. For example, in some experiments subjects rate their hunger when food is present which may increase their reports of hunger (Wooley, 1976), whilst other studies may have food available at the onset when hunger ratings are being recorded before consumption of the food, but absent later on in the study when hunger is being again rated. The problem is that information about this part of the experiment is often omitted as is the statistical analyses of the results. Nonetheless, it is likely that hunger scales will continue to be used as a proxy for measuring food intake. In assessing appetite it is becoming more common for studies to combine the use of measuring devices such as hunger ratings with the direct measurement of food consumption.

Self-reports of food intake using diaries (Stock and Yudkin, 1970; Yudkin and Carey, 1960; Rosen et al, 1982; 1985) can also cause problems. There is inevitably a loss of control over food presentation and of precision in the measurement of what is eaten. The latter problem may be caused by forgetting the exact amounts eaten or reluctance to report the true amounts consumed, or failure to measure portion sizes accurately. Training on the use of food diaries may overcome these problems to some degree ((Klesges et al., 1988; Mahalko et al., 1985). The very act of recording food intake may trigger a reactive effect which could affect subsequent food intake (Levitz, 1975). Nonetheless diaries are necessary to study food intake in a natural setting.

There now follows a review of the literature on the effects of different nutrients on satiety; however rather than give a full study-by-study analysis, table 2.2 will summarise the methodology of the key experiments. Conclusions from this literature will be drawn at the end of the chapter where an attempt is made to reach a consensus regarding these findings.

Table 2.2: Methodology of studies on the effects of different nutrients on satiety

Authors	Time between preload and test meal	Sample composition	Food composition	Deprivation period	Psychological dependent measures	Control of cognitive factors	Duration of eating	Monitoring of subsequent intake	Baseline measures
Booth et al.1970a	2.5-3h later supplement meal was given after test meal	normals n=9	lunch:protein-rich or protein-poor; 337-1136 kcal. Supplement meal (of average protein composition)	several hours fast between breakfast and test meal	no	no	lunch-30mins 15mins for supplement meal	food weighed after test meal	no
Booth et al. 1970b	preload given 20 mins bef immed. bef immed. after meal	normals 11 M 1 F	Preload: glucose Vs saccharin. Test meal: crisp bread + savoury spread. Supplementry snack: dessert (3 hrs later).	since breakfast, although food not controlled in-between	no	Sometimes a control drink used instead of glucose	not stated	same as above	no
Hodgson & Greene 1980	NA	normal 23 F	6 maltesers consumed 4h or 1/2h after b/fast or lunch	4 hr or 1/2 hr	hunger rating-& salivation measures	no	10 mins	hunger ratings used10 mins after eating	no
Spring et al. 1982/3	NA	normals 55 M 129 F	protein-rich 57g protein, 4g fat 1g CHO. CHO-rich 57g CHO, 4g fat, 0g protein	morning group overnight fast/ afternoon group-standard breakfast.	visual analogue scales	not really	not stated	2 hours after intake using VAS & food weighed after intake	no
Geliebter,1979	70 mins	12 M	Preloads: 283 kcal: (1) 63.5g protein/ 31.5g fat/ 67.4g CHO (2) 54.1g each. (3) 0 kcal - 70% kaolin and 30% cellulose. Test Meal(Sustacal Mead Johnson)	overnight fast (13 hrs)	appetite questionnaire bef/after and halfway through the 1 hr waiting period between preload and meal	yes	approx. 27 mins for test meal	food weighed after test meal	no

Rogers & Blundell, 1989	approx. 1hr	normals 6 M 18 F	Preload:1) 210g yogurt & 25g fruit (131kcal). 2) 210g yogurt, 25g fruit, 163mg saccharin (131kcal). 3)170g yogurt, 25g fruit, 50g starch (295kcal). 4)170g yogurt, 25g fruit, 50g glucose (295kcal). ***2nd cohort of subjects received a 5th preload: 5) 170g yogurt, 25g fruit, 50g starch, 163 mg saccharin (295kcal). Test meal: sandwich, digestives, apple pie, fruit.	fast from 09.30h; 1hr fast between preload and test meal	ratings of motivation to eat using VAS and diary. VAS for sensory and affective ratings of preloads	yes	3-7 mins for preload Time not stated for test meal	up to bedtime using diary	yes
Guss & Kissileff, 1988	30 mins	normals 8F	four 500ml preloads lemon flavoured with 1% or 10% fructose or glucose (all with aspartame or 500ml of water. Test meal: macaroni & beef.	180 min fast	not stated	control of sensory factors, all preloads were equally sweet	not stated		not stated
Kissileff et al.1989	135 min	not stated	10% fructose or glucose preloads in 500ml preload lemon flavoured	180 mins	hunger ratings; 150 mm analogue scale	preloads were equally sweet	not stated	not stated	not stated
Blundell & Hill, 1986	NA	normals 95 M & F	1) 50g glucose in 200ml preload 2)162mg aspartame in 200ml preload 3)water loads-control group.	not stated	hedonic ratings and VAMS	2 preloads equivalent sweetness	not stated	ratings made every 10 mins up to 60 mins	not stated

Rolls & Hetherington 1988	120 mins	normals n=10	Preload 300 kcal high protein, high fat, high starch or mixed fat & sucrose. Test meal-mixed food and drink (adlib intake)	overnight fast	VAS & Food Preference Checklist	no	not stated	Food weighed after test meal	no
Tordoff & Alleva, 1990	NA	normals n=30	1150g soda sweetened with aspartame or high fructose corn syrup, or no drink	NA	daily diaries for 9 wks	yes, but not clear if fully blind .	NA	diaries	diaries for 2-3 days for practice
Spitzer & Rodin 1987	2.25 hrs	normals 21 M 19 F	500 ml of (1) water (2) plus fructose 50g (3) glucose 50g (4)glucose 50g/aspartame flavouring. Test meal - adlib intake of mixed foods	overnight fast	intensity, pleasantness & aversion ratings of preloads	control of sensory/ cognitive factors	preloads: 5 mins; lunch: 20 mins	food weighed after test meal	no
Teff, Young, Blundell, 1989	3 hrs	normals 32 F	Preload: protein-rich pudding (210kcal)-45g protein, 12g CHO, 3g fat.CHO-rich pudding (400kcal)-100g CHO. Test Meal: solid food (adlib basis)	overnight fast and 3hr deprivation between preload and lunch	no	both preloads looked and tasted similar	30 mins for test meal	left-overs from lunch weighed 30 mins after lunch	no
Grinker et al. 1971	Expt.1:- 15mins Expt.2:-15 mins	normals 5 M 1 F normals 8 M	Preloads for expt.1 & 2: Infusion of saline 100ml, glucose 100ml, mannitol 100ml Test meals-expt.1: liquid diet-chocolate nutrament (free feeding)- expt.2 adlib intake of sandwiches & ice cream or spaghetti & ice cream	expt.1-overnight fast/skip breakfast expt.2- 22hr deprivation	Hunger ratings & other rating devices before the infusion, 20mins after onset of infusion & on termination of eating	not clear	expt.1: 30mins free feeding for test meal expt. 2: not stated	hunger ratings immediately after eating	yes- 2 control days- 1 day of saline & mannitol; 2 days of glucose

Shafer et al. 1987	60-90 mins later	normals 14 M 5 F 2nd group- 5 F	solution with 25g of xylitol, glucose, fructose or sucrose or water preload and buffet test meal 250mg/100ml aspartame or xylitol (5g, 15g, 25g) & buffet test meal	fasted condition not stated how many hours	none	not stated	30 mins for buffet meal	food intake measured during the 30mins meal	not stated
Brala & Hagen, 1979	approx.90 mins	normals n=118	milkshake (with glucose 429 kcal; aspartame 198kcal or placebo (198kcal) Test meal: snack foods adlib basis.	overnight fast	hunger ratings and sucrose rating scales	use of placebo drink	30mins from test meal	food intake measured after food tray retrieved	not stated
Booth et al. 1982	presumed immed after	16 normals 8 M 8 F	soup-starch augmented soup (210kcal); control soup (25kcal) sandwiches adlibitum; dessert gelatin (300ml); augmented dessert (25g sucrose+starch 300kcal) control dessert (0.0g saccharin +5g sucrose =20kcal) NB.low glucose maltodextrin was used as starch in soup and dessert & 50g extra in augmented portions	not stated	pleasantness ratings	subjects were unaware of caloric manipul-ation	not stated	not stated	one with lunch session for familiarization purposes with lunch

Hill & Blundell, 1986	NA	normals 13 M & F	sandwiches-500kcal. high-protein;- fat 28.5g; protein 41.1g; CHO 30.3g. high-CHO: fat 18.8g; protein 17.8g; CHO 68.1g	overnight fast	VAS; Food Preference Checklist	no	not stated	60 mins	no
De Castro, 1987	NA	normals 8M, 30F	Regular meals /free feeding in a natural setting.	none	9 day diary and mood ratings	no	NA	9 days	1 day trial
Booth et al.1976	immediately after preload	university staff and students- exact no. not stated	preloads: 1) 100ml of lemon flavoured squash; 2) 100ml of concentrated load (65g maltodextrin, 3g cornflour, 6.5g citric acid, 6.04g sodium saccharin, 0.4g lemon essence 3) 100ml of dilute load(4.4g pregelled starch, 5ml of double cream, 2g sucrose, 0.4g citric acid, 0.01g sodium saccharin, 0.45 lemon essence Test meal: variety of foods	not specified	none	measure sensory factors in preloads and control drink	up to 30 mins for test meal	food weighed after test meal	questionnaire on subjects habitual eating
Rogers et al. 1987	1 hr	normals 8 M 4 F	Preload: 200ml water plus 1) 145mg saccharin or 2) 162mg aspartame or 3) 240mg acesulfame-K or 4) 50,000mg glucose (188kcal) or 5)200ml water. Test meal: mixed foods (adlib basis).	3hrs prior to preload; 1hr fast between preload and test meal; 3hr fast after test meal	ratings of motivation to eat and Food Preference Checklist	yes	1 min for preload Time not stated for test meal	up to 3 hr after test meal	yes

Rodin, Reed & Jamner, 1988	NA 95 mins	expt.1: 18 obese, 20 lean. expt.2: 19 obese, 20 lean	expt.1: 50g fructose/ glucose in 500ml water (197kcal) expt.2: breakfast (20g bread,5g margarine, 50g egg, 150g low fat milk, 50g muffin containing 15 g of glucose or fructose. Snack: 2 cookies (150kcal) 23% fat, 43% simple sugar (25gfructose/glucose)34 % complex CHO.	overnight fast	blood samples	no	10 mins	not stated	Yes: blood sample & 24-hr diaries + extra 150-200g CHO day before study
Rodin, 1990	38 min after preload	12 obese, 12 normal	Preloads: (1) 50g fructose, (2) 50g glucose, (3) plain water, (4) aspartame & lemon flavoured water. Buffet lunch (adlib intake).	overnight fast	blood samples 10-48 min after preloads	sensory control of drinks	10 min for preload/ lunch not stated	blood samples & food weighed after lunch	same as above

Table 2.2: Methodology of studies on the effects of different nutrients on satiety in obese and non-obese.

Sunkin & Garrow, 1982	4 hr, 45 mins 5h10min-6hrs	expt. A- 2 obese F expt.B- not stated expt. C- 4 obese subjects	fixed breakfast (200kcal); lunch-high or low protein formula (adlib intake); supper (200kcal) breakfast (50kcal); lunch /protein meal(500kcal) high protein: 3 1.9% of kcal protein V's low protein:6.5 protein kcal% Evening meal-adlib diet of milk,yogurt, and bread.	not stated	hunger ratings	partly-formula delivered in a concealed container in expt A but not in expt B, C	expt.A-2hrs for test lunch expt,B & C not stated	expt. A. calculate the amount consume of the formula expt B & C the amount eaten for evening meal calculat	expt.A-no expt. B & C- 1 wk baseline diet (800kcal)
Fryer et al. 1955	NA	11 M obese	isocaloric (180kcal); low carbohydrate (23%), low fat (2.5%), low protein (8%), moderate protein (13%)	NA	hunger ratings and diaries	not blind	not stated	3 week adaptation period then 1 day normal eating	no
Yudkin & Carey, 1960	NA	6 obese 4 F 2 M	adlib intake of protein & fat but only 50g of CHO over 4 weeks	NA	intake records	not blind	not stated	intake records for 4 weeks	yes 2 wk baseline
Stock & Yudkin, 1970	NA	11 normals 4F 2M	same as Yudkin & Carey, 1960 except additional 20.02mg of milk & vitamins over 2 weeks	NA	intake records	not blind	not stated	intake records	yes 2 wk baseline
Kinsell et al.1964	NA	5 subjects, 4 very obese, 1 slightly obese	800 -1200 kcal per day of formula diet. Nutrient content varied: Fat 12-83%, Protein 14-30%, CHO 3-64%	2 hr interval between meals	none	no	not stated	not stated	not stated

Rosen et al. 1982	NA	8 obese inpatients	827Kcal:1) 25% protein, 30% fat, 29% CHO restricted 2) 35% protein, 64% fat, 1% CHO	overnight fast	VAS before lunch & dinner	not stated	NA	VAS before lunch & dinner	11 days free feeding
Rosen et al. 1985	NA	20 obese out patients 1 M 19 F	(1) 800kcal-CHO restricted: 58% protein, 42% fat. (2) 1000kcal CHO containing-42% protein, 30% fat, 28% CHO	overnight fast	VAS before dinner at home	subjects knew CHO content of diet was being manipul-ated, but did not know when	NA	food records checked every 2wks	2 weeks but no record of intake

The Effects of Carbohydrates on Subsequent Feeding

It has been suggested that carbohydrate intake increases appetite not only generally but specifically for carbohydrates (Geiselman and Novin, 1982). Novin, Vandeweele and Rezek (1973) proposed that the sight, smell, taste and ingestion of carbohydrates, especially refined sugars, leads to high levels of blood insulin, and consequently lowers the level of blood sugar which may induce increased hunger and appetite. This theory suggests that the macronutrient composition of certain foodstuffs characterised by high calorie and sugar content may provide a physical basis for craving more of these "junk" foods. Animals consume more calories when fed a diet high in refined sugar than when fed a diet high in starch content (Kanarek and Marks-Kaufman, 1979). Geiselman and Novin (1982) infused rabbits with glucose intraduodenally. When infusions occurred at a slow rate (1 millilitre per minute) there was a reduction in subsequent food consumption. Conversely, a higher rate of glucose infusion resulted in a significant increase in feeding specifically during the first half hour when intake was practically doubled. Geiselman and Novin (1982) concluded that when glucose reaches the duodenum at a fast rate absorption is more rapid and appetite is stimulated.

The Effects of Different Carbohydrates on Food Intake

Different carbohydrates may have different effects on hunger, in that they may affect the absorption of certain nutrients, so foods high in refined sugars may increase, or fail to reduce, hunger and appetite more than diets high in complex carbohydrates which are absorbed and metabolised at a slower rate (Cohen, Teitelbaum and Rosenmann, 1977; Naismith and Rana, 1974). This effect is possibly attributable to the more rapid sugar transport, the elevated insulin levels and the resultant hypoglycaemia occurring in response to the ingestion of simple sugars (Karlan and Cohn, 1946). Geiselman and Novin (1982) proposed that rather than increasing appetite and hunger in general, insulin induced hypoglycaemia may specifically increase appetite and hunger for carbohydrates, especially sugars.

In a study by Spitzer and Rodin (1987) where the effects of glucose were compared with fructose on subsequent food intake, the intake varied as a result of ingestion of different types of sugar which do not necessarily possess the same nutritional value. They gave forty normal-weight subjects 500 ml of plain water or equicaloric preloads of fructose, glucose, or glucose flavoured with aspartame so that it tasted as sweet as fructose diluted in water. Both the fructose and glucose drinks were almost identical in appearance and tasted the same, each contained 10% sweetener. Thus, orosensory and psychological factors were controlled to a large extent. Approximately two and a quarter hours later subjects were given a buffet meal. Those who had glucose preloads ate on average 253 kcal more in the buffet meal than subjects who had consumed plain water, whilst those who had received the fructose preload consumed on average 226 kcal less in the buffet than those in the water condition. This suggests that when high glucose meals are consumed the metabolic sequelae may increase hunger, whereas fructose suppresses subsequent intake.

These results were replicated in a study by Rodin et al. (1988) on obese and lean subjects. Fructose, but not glucose, preloads (50 g) reduced subsequent intake in a buffet meal administered two and a quarter hours later. Later, however, when the two different sugars were fed to subjects as part of a mixed meal for breakfast, no differential satiating effects in intake were observed. This time testing occurred approximately one and a half hours later, when biscuits were served as a snack containing 25 g of the same sugar used in the mixed meal earlier on.

One hypothesis concerning the differential effects of different sugars, if such exists, is that they affect blood insulin differently. In a later study Rodin (1990) again showed that 50 g of fructose in 500 ml of lemon-flavoured water reduced overall intake and fat consumption at a buffet lunch served thirty-eight minutes later, when compared to 50 g of glucose, plain water or lemon flavoured water containing aspartame (0.25 g). Rodin

(1990) reported similar results to those found in the 1987 and 1988 studies. This time using a within-subjects design with twelve obese and twelve non-obese fasted subjects. According to Rodin (1990) the fact that the differential effects of glucose and fructose were found in a shorter time period; thirty-eight minutes later on subsequent intake, compared to two hours (Spitzer and Rodin, 1987) or two and a quarter hours (Rodin et al., 1988) makes time variation an unlikely explanation of different findings across studies. Furthermore, although glucose and fructose have differential rates of gastric emptying (Elias et al., 1968; Moran and McHugh, 1982) an association between rate of gastric emptying and differential satiating effects of the two sugars was not found. Fructose which has a more rapid gastric emptying rate, did not stimulate hunger and subsequent intake within the first thirty to sixty minutes. Rodin et al. (1988) showed a direct relationship between peak insulin concentrations after sugar preloads and subsequent food intake at thirty to forty-five minutes and subsequent food intake two and a quarter hours later. In Rodin (1990) blood samples were taken every eight minutes between ten and forty-eight minutes after preload ingestion. The non-caloric preloads resulted in lower insulin and glucose concentrations before the buffet meal and increased intake. After the ingestion of the glucose preload, levels of plasma insulin which increased over time were observed. These were higher than for fructose or non-caloric beverages. A relation between increased food intake and peak insulin concentrations (thirty minutes after the preload) was observed for glucose but not fructose preloads.

Several studies have failed to find different effects of different sugars. Booth et al. (1970a) reported that glucose ingested twenty minutes before lunch suppressed subsequent intake at lunch and three hours later when given a snack. Shafer et al. (1987) found no differential effects in equicaloric preloads of glucose (25 g), fructose (25 g) or sucrose (25 g) when food intake was assessed by administering a buffet meal one hour later. All three sugar solutions failed to suppress intake and ingestion of an aspartame solution (250 mg/100 ml) also failed to have any satiating effect. In contrast

25 g of xylitol, a pentose sugar, significantly reduced intake compared to glucose, fructose, sucrose, aspartame or water preloads. Guss, Kissileff and Pi-Sunyer (1988) using identical preloads to Spitzer and Rodin (1987) on eight normal females, reported no difference in the satiating effects of glucose versus fructose on subsequent intake. Intake was measured thirty minutes later in the form of a macaroni and beef test meal. They found no difference in intake after the different sugars but they reported an increase in food consumption after ingestion of the aspartame sweetened preloads containing only 1% fructose and 1% glucose, when compared to drinking plain water. Using similar preloads (10% solutions) Kissileff, Guss and Pi-Sunyer (1989) were unable to replicate Spitzer and Rodin's results (1987) although they did report a trend in that direction.

These findings raise the question as to whether differential effects of different sugars on appetite exist or not. Blundell et al. (1988) proposed that the greater satiating effects of fructose found in Spitzer and Rodin's study (1987) and in the study by Rodin et al. (1988) may be attributed to the use of the artificial sweetener aspartame in the glucose preload to make it as sweet as fructose and indiscriminable under experimental conditions. This is important since some past studies have shown that artificial sweeteners such as aspartame may increase hunger (Blundell and Hill, 1986; 1987) and feeding (Brala and Hagen, 1983). However, the fact that Spitzer and Rodin (1987) included two other conditions make this hypothesis unlikely, since it would have been expected that aspartame would have increased hunger for all conditions, rather than just the glucose condition. There are various other procedural differences across these studies which could account for diverse findings. For example, in these studies administration of test meals ranged from twenty to one hundred and eighty minutes after the preload. Nutrient dose, deprivation and palatability also varied considerably. These discrepancies may have some importance to the different results, particularly when repeated exposure and ingestion of the same test meals is common in studies using within-subjects design.

Turning to studies of glucose, without fructose conditions, Grinker et al. (1971) found no evidence that glucose increased hunger and food intake. In two separate experiments they examined the effects of different intravenous infusions of glucose, saline and mannitol (isosmotic with glucose) had on fasted subjects' eating behaviour and self-reported hunger levels. Both studies varied in level of food deprivation, the time the food was administered and composition of foodstuff. In experiment 1, after being food-deprived for twelve hours, six subjects were administered food at 08.45h in the form of a liquid diet using a feeding machine, fifteen to twenty minutes after the infusion. In experiment 2, six subjects fasted for twenty-two hours and adlibitum consumption of solid palatable food was provided on a tray at approximately 16.15h, fifteen minutes after the infusion. There was a significant reduction in hunger ratings after intravenous glucose but not after saline or mannitol. This was not found after the shorter first experiment. There was no significant reduction of caloric intake in either study. According to Grinker et al. (1971) this may be attributable to subjects' relatively high level of food deprivation. Variations on hunger ratings in both experiments could have been related to the ingestion of liquid compared to solid foods in the two studies and different deprivation levels. Perhaps the most important finding was no increase in appetite or intake after glucose ingestion.

Carbohydrate and a Possible Priming Effect

It has been suggested that the desire to eat may be related to the nutritional value of food previously eaten (De Castro, 1987). One possibility is that high-carbohydrate food has a priming effect. Hodgson and Greene (1980) found that a person may experience greater hunger after eating a small piece of chocolate, especially if they were originally feeling very hungry. There were two experimental conditions carried out in their study. Twenty-three subjects were either given a small amount of chocolate to eat thirty minutes after consumption of either breakfast or lunch, or alternatively four hours subsequent to food intake. Salivation was measured to determine the

priming effect, as well as hunger ratings (using five-point and one hundred-point hunger scales). Chocolate induced more hunger in subjects who had undergone a four-hour deprivation period as opposed to the half-hour deprivation condition. This is consistent with predictions that carbohydrate ingestion can enhance appetite rather than reduce hunger. The problem is that chocolate does not consist of pure carbohydrates, but rather is made up of a high proportion of fat and carbohydrate.

Carbohydrates Suppress Appetite

Booth and colleagues have found that carbohydrate intake can decrease food intake. Booth et al. (1970a) examined twelve subjects' eating behaviour after ingestion of a 100 ml drink containing 50 g of glucose administered twenty minutes before lunch, or immediately before lunch or after lunch, delivered to subjects in a counterbalanced between-subjects design. The consumption of glucose (188 kcal) twenty minutes before lunch caused a reduction in food intake both at lunch and during a supplementary snack given three hours later, when compared to the control group (saccharin placebo). Orosensory factors were not fully controlled although Booth et al. (1970a) claimed that because subjects were asked to "gulp" the liquid solution "from the back of the throat" most subjects were unaware of the sweetness of the drinks. When glucose was ingested immediately before lunch, suppressing effects were still noted at lunch (although only half as much as that caused by the twenty-minute glucose preload), but not for the supplementary snack. When glucose was ingested immediately after the test meal it had little effect on the snack. Booth et al. (1970a) concluded that the satiating properties of glucose may be effective from about fifteen minutes subsequent to food intake and may last up to three hours. There was no fasting condition in this study and extraneous eating was uncontrolled.

Similar results have been reported for starch by Booth et al. (1976) who found that a 100 ml liquid preload containing concentrated starch ingested before lunch made subjects eat less at lunch, than when they consumed a diluted starch preload (see Table

2.2 for exact amount). Booth et al. (1976) concluded that the satiating properties of food is partly obtained through "Association of its sensory characteristics with some consequence of ingesting the nutrients which are consistently taken with or in that food. This consequence may be rapid intestinal absorption of glucose during or after sensory input". This suggests that sensory factors and not just the nutrients have a significant influence on later intake.

In a later study Booth, Mather and Fuller (1982) found that at lunch starch (low-glucose maltodextrin) added to soups suppressed subsequent food intake (sandwiches), compared to control soup. However, when the starch was added to dessert, no subsequent suppression of eating was observed although it did decrease pleasantness ratings for most foods thereafter. It was concluded that starch suppresses appetite if presented at the beginning of the meal. These findings support the hypothesis that the process of conditioning would be less effective with starch augmentation at the end of a meal (dessert) as opposed to earlier because the sensory factors of the food take precedence over the digestive effects of starch.

The Role of Sugar compared to Artificial Sweetener on Subsequent Food Intake

The data on the effects of artificial sweetener on hunger compared with sugar are also controversial. The main issue in these studies concerns the possible separate effects of sweet taste which may increase appetite compared with the energy effects of nutrients such as carbohydrate which may inhibit appetite. Whilst Blundell and colleagues have shown that aspartame can stimulate hunger in humans (Blundell and Hill, 1986; Blundell and Hill, 1987; Blundell, Rogers and Hill, 1988) other findings have shown no evidence of this (Rodin, 1990; Tordoff and Alleva, 1990). Blundell and Hill (1986) found that in ninety-five subjects appetite ratings, as assessed by VAMS up to an hour after preload ingestion, increased significantly after an aspartame load (162 mg/200 ml), particularly in the last thirty minutes of the rating period. In contrast after 50 g of

glucose there was a significant decrease in the ratings for desire to eat and an increase in the fullness ratings. Ratings for the water group did not differ from baseline scores. While these findings question the role of aspartame on satiety, it cannot be presumed that hunger ratings on their own can predict eating behaviour without measurement of subsequent intake.

Brala and Hagen (1983) investigated the effects of equivalent loads of sucrose and aspartame in milkshakes. A placebo drink (plain milkshake) was included as a third condition. When sweetness was suppressed by gymnemic acid in a milkshake preload, food intake in the test meal was not as high as when sweetness could be tasted. Then increases in overall food consumption and in sweet foods in particular were observed. Such findings suggests that taste alone, irrespective of nutrient content, can increase intake, at least in the short-term. Nevertheless, it was found that the sucrose group consumed more calories than the aspartame or placebo groups. Another interesting finding was that the aspartame group actually consumed more calories (60 kcal more) in the test meal than the placebo group, although this effect failed to reach statistical significance.

Using a blind repeated-measures design with twelve subjects, Rogers et al. (1987) reported differential effects on appetite following preloads containing intense sweeteners (aspartame, saccharin and acesulfame-K) compared to glucose or just plain water. Contrary to some of the past studies on carbohydrates where it has been proposed that carbohydrates such as glucose may stimulate hunger and appetite, Rogers et al. (1987) demonstrated a significant reduction in food intake following glucose ingestion (169 kcal; revealing almost perfect compensation) compared to the water control group. In contrast, no significant reduction in food intake was found following preloads containing the intense sweeteners, although a slight reduction was shown. With respect to ratings of motivation to eat and the food preference checklist the intense sweeteners appeared to facilitate these responses, in that a positive shift

from baseline was reported, whilst the glucose preload showed a marked reduction in both subjective ratings.

In a later study using a repeated measures design on twenty-four subjects, Rogers and Blundell, (1989) confirmed the above findings when it was found that non-nutritive substances such as saccharin do not have the same satiating properties as carbohydrates (i.e. glucose or starch). A significant reduction in food intake and motivational ratings was found for the carbohydrate preloads when compared to saccharin. There was some evidence that saccharin stimulated appetite after lunch by increasing hunger and subsequent intake. Further, Rogers and Blundell (1989) demonstrated that increases in hunger and intake may be found by adding saccharin to starch supplemented preloads, compared to starch preloads without the addition of a sweet substance. The findings from this study also lend support to the differential satiating effect of starch compared to glucose (Rolls et al., 1987), in that the yogurt preload containing starch suppressed intake slightly more than the yogurt supplemented with glucose. This reduction, however, did not reach statistical significance.

In the study by Rodin (1990) reviewed earlier, there was no evidence that aspartame in lemon flavoured water increased food intake in the short-term, compared to drinking plain water. In a long-term study Tordoff and Alleva (1990) compared the effects of a 1150 g of soda containing aspartame or high-fructose corn syrup (530 kcal per day) or no drink over a nine-week period. Using a counterbalanced design, thirty normal male and female subjects were appointed to one of the three conditions for a three-week period. Both drinks were similar in weight and contained equal amounts of water, carbon dioxide, and caffeine. Drinks were to be consumed daily under natural conditions. It was found that aspartame compared to the no beverage condition decreased total caloric intake (by 7%) and a reduction in body weight was observed for males but not females, whilst high-fructose corn syrup increased total caloric intake

(by 13%) and body weight for male and female subjects. Both beverages resulted in an overall reduction of sugar ingestion (33% decrease) but did not affect fat or protein intake. Sensory factors were not masked and the sweetness, rather than the nutrient effects of the aspartame and high-fructose corn syrup beverages, could have influenced subsequent sugar ingestion (Tordoff and Alleva, 1990). This, however, does not explain the overall reduction in food intake following ingestion of the aspartame drink, but not the high-fructose drink. The fact that subjects failed to compensate efficiently for the high caloric drink (37% compensation only) supports past work, where poor compensation has been reported following covert manipulation of caloric intake (Mattes et al., 1988; Porikos et al., 1982).

Lack of consistency in results could be attributed to procedural variation. Tordoff and Alleva (1990) report long-term findings, whilst other findings reporting an increase in appetite and intake following aspartame ingestion were short-term studies. Tordoff and Alleva (1990) have postulated that any aspartame-stimulated effects on appetite reported in these studies may be short-lived and may not have the same effects over the long-term, possibly due to a readjustment taking place after repeated consumption of the aspartame beverage.

Differential Effects of Carbohydrates compared to Protein on Hunger in Normal-Weight Subjects

An experiment conducted by Booth, Campbell and Chase (1970b) found that carbohydrates are not as effective as protein in appetite suppression. Since then there has been a substantial body of investigation on the possible satiating effect of various nutrients (Teff, Young and Blundell, 1989; Hill and Blundell, 1986; Blundell, 1987; Jen, 1985; Rolls, Hetherington and Burley, 1988). Many of these findings suggest that carbohydrate differs from the nutrient protein in its contribution to the control of appetite and food intake in humans. However, some of the findings are quite

contradictory in nature and not all of them lend support to the greater satiating power of protein.

Booth, Campbell and Chase (1970b) administered a lunch and snack of solid composition in the laboratory which were either high in protein or low in protein (varying between 337-1136 kcal). A protein-rich meal decreased subsequent food intake up to three hours later by 26% more than a carbohydrate-rich isocaloric meal. However, although subjects were asked to fast several hours prior to the test meal no other data on food intake prior to the experiment was obtained and only eight subjects were used in this study which introduces various problems, discussed above.

In a short-term study by Rolls, Jacobs and Hetherington (1986) normal-weight females showed little if any response to caloric manipulations in their diet when administered high or low-calorie foods (in the form of pudding or jello), sweetened with either glucose or aspartame. Consumption of the preloads was adlibitum and results showed that, irrespective of the caloric content of the preloads, subjects consumed the same weight of foods. Subjects who had consumed the glucose, as opposed to the saccharin in their foods, failed to compensate later on at lunch one or two hours later. Rolls et al. (1986) found that despite the caloric variation in the jello preloads, subjects' hunger ratings were more or less the same for both the high and low calorie foods. From these findings it seems that weight rather than the caloric content of a meal has more influence over hunger and satiety in normal-weight subjects, at least in the short-term. Their findings fail to support the theory that glucose is more satiating than a non-nutritive substance like saccharin when added to a particular foodstuff.

Rolls, Hetherington and Burley (1987) studied the satiating effects of equicaloric preloads (approximately 300 kcal) on ten females over five days. They found that following the ingestion of preloads rich in either starch or protein, ratings for hunger

decreased and ratings for fullness sharply rose, when compared to the preloads high in sucrose, fat or of mixed nutrients (high in fat and sucrose). When a selection of food was offered two hours later on an adlib basis it was found that total caloric intake was lower after the protein and starch preloads, particularly for carbohydrate-rich food or food high in fat content. In contrast to some other studies (Blundell and Hill, 1987; Wurtman et al., 1982) Rolls et al. (1988) found no significant correlation between intake of a particular macronutrient and subsequent suppression of that macronutrient. Although their findings demonstrated that a high-protein foodstuff was more satiating than a high-sucrose diet it is important to note that the same was not found for the high-starch food which is also a form of carbohydrate. This is of significance, as often studies refer to "carbohydrates" without specifying if starch or a particular type of sugar was used which could affect the findings in general on the satiating effects of carbohydrates.

Again, sample size was small in this study and although differential effects were found between the various preloads, it is possible that differences in orosensory characteristics of the preloads as well as different temperature, weight and bulk of the foods (see Kissileff et al., 1984) influenced the satiating properties of the foods. Cognitive factors may also have contributed to the greater satiating effects of protein and starch preloads compared to the sucrose or the sucrose and fat preloads. For example, the starch (pasta) and protein (chicken) preloads are foods which are generally consumed as part of a main meal, whilst the foods used for the sucrose (turkish delight) and the mixed preloads (chocolate bar) are generally regarded as snacks. Thus, subject expectation and beliefs of certain foods could have biased their results to some extent.

Using a within-subjects design Hill and Blundell (1986) investigated the effects that protein or carbohydrate-rich equicaloric meals (approximately 500 kcal) had on subsequent self-reports of satiety and on desire to eat. Order of presentation was

counterbalanced over two days, one week apart. Sandwiches were administered at lunch to thirteen normal-weight students who had fasted overnight and avoided breakfast. Hunger, fullness, appetite and food preferences were assessed five minutes and sixty minutes after food intake.

The protein-rich meals induced feelings of satiation in subjects considerably more than did the carbohydrate-rich meal, and on the self-rating scales subjects' desire for further intake was lower after the protein-rich meal. Of particular interest was the reduced preference for protein-rich foods following ingestion of the protein-rich preloads, indicating some evidence of nutrient-specific satiety. However, those who had consumed the carbohydrate-rich preload revealed no reduction in desire for carbohydrate foods. Nonetheless, irrespective of the preload ingested there was a greater preference for carbohydrate-rich food when in a state of satiety. These findings differ from what Rolls et al. (1988) reported concerning subsequent nutrient preference and intake.

No baseline measures were obtained in this study and cognitive factors went uncontrolled since food was not administered blind. Thus, changes in preference could have been due to conditioned sensory factors. The preloads also differed in fat content; the protein-rich preload contained more fat than the carbohydrate-rich preload. It may be that the combination of both nutrients (i.e. fat and protein) contributed to the greater satiating effect of the protein-rich preload. Another problem with this study is that both starch and simple sugars were used in the carbohydrate-rich preload. Yet, based on past literature differences in the ingestion of these carbohydrates exist (Rolls et al., 1988). As mentioned previously, it may be more fruitful to make a distinction between both types of carbohydrates and examine each in isolation.

Using a double-blind counterbalanced crossover design with thirty-two fasted subjects, Teff, Young and Blundell (1989) examined how a palatable breakfast high in

protein (approximately 210 kcal), or carbohydrate (400 kcal) would influence satiety levels and lunch intake three hours later. Seventeen subjects received the protein-rich breakfast and the remaining fifteen the carbohydrate-rich breakfast. Thirty minutes after an adlibitum lunch, the left-overs of food were measured and their findings indicated that food intake at lunch was similar, irrespective of the preload consumed.

These findings are of significance since the protein-rich breakfast contained approximately half the calories of the carbohydrate meal. There was no evidence of macronutrient specificity in this study, although the carbohydrate-rich breakfast did reduce the selection of apple during the lunch session. But no control group was included in this study, so it is even possible that neither preload had an effect on lunch. The problem is that no baseline measures of prior food consumption were taken, therefore no comparison can be made between lunch on the days of the experiment and under everyday conditions.

Spring et al. (1983) compared the effects of carbohydrate-rich and protein-rich meals in a between-subjects design on a hundred and eighty-four normal-weight men and women (see chapter 1 for a more detailed review). Other than dietary-induced effects on mood, it was found that subjects reported a greater level of fullness when asked to rate their hunger (using Visual Analogue Mood scales) following consumption of a protein-rich meal (57 g protein, 4 g fat, 1 g carbohydrate) than following consumption of a carbohydrate-rich meal (57 g carbohydrate, 4 g fat, 0 g protein).

Nonsignificant Results for Different Satiating Powers of Carbohydrates, Protein and Fat

Past research has failed to find any evidence that various foodstuffs or nutrients have different satiating powers. Geliebter et al. (1979) found no evidence in their study to support the notion that carbohydrate consumption enhanced appetite more than protein or fat intake one hour after food consumption in humans. In an animal experiment

conducted by the same authors it was reported that carbohydrate or protein-rich preloads delayed caloric intake longer than an equicaloric preload of fat. In the human study twelve male adults of normal weight were given one of six preloads between 11.00-12.30h. Four of the six preloads were caloric preloads, each containing 283 kcal, but differing in nutrient content, whilst the remaining two were non-caloric in content. Covered containers were used to contain the liquid formulae so that subjects were unable to see the preloads. Sensory cues were also eliminated by the use of lidocaine, an anaesthetic solution prior to the meal and nose clips were worn during the consumption of the preloads. All subjects were in a fasted condition prior to the preload.

Seventy minutes later a liquid test meal, comprising of Sustacal, (a nutritional liquid foodstuff), was delivered to subjects from a hidden container. A twenty-seven minute time period was allocated to each subject for food intake. The liquid which remained in the container was weighed thereafter as an indicator of food consumption. This procedure took place over a three-week period. Each individual was administered a different preload for six days, then the preloads were redelivered in that order for the remainder of the experiment.

The findings revealed an increase in food intake after the ingestion of non-caloric preloads when compared to caloric loads. Nonetheless, there was no evidence that equicaloric single-nutrient loads of carbohydrate, protein or fat possessed different satiating effects in later intake or on self-report of hunger, rather all three nutrients seemed to depress food intake to the same degree. It has been proposed that Geliebter's (1979) "extreme" control of sensory factors (nose clips, anaesthesia of the mouth) may have influenced the effects of the preloads and subsequent food intake (Spitzer and Rodin, 1987). The study has also been criticised for monitoring subsequent intake up to seventy minutes only since differences in food intake could

have occurred later on. The latter problem applies to most studies being reviewed in this section.

Stockley et al. (1984) tested whether humans are capable of regulating intake on an internal level after ingestion of a protein supplement. They administered two types of supplements; one was high in energy content (51.39 g carbohydrates; 22.2 g fat; 420 kcal), but contained no protein. The other supplement contained fewer calories (84 kcal), but was high in protein (14 g). The low protein/high energy supplement was given to fourteen adults for seven days. Three weeks later the same group was given a placebo. Another group (n=13) was given the high protein supplement for seven days and a placebo three weeks later. All subjects were requested to retain a detailed record of all food consumed and to weigh food at all times. There was little indication that either of the two supplements lead to a significant decrease in daily food intake. The high energy supplement did reduce the mean caloric consumption for the remainder of the day, but only by 32% of the supplement's energy content which was not statistically significant, whilst the high protein supplement failed to affect subsequent intake. However, interpretation of the findings is complicated by the protein and energy supplements used in the experiment which differ somewhat from the liquid or solid foodstuff used in other studies.

There was no evidence that satiety was nutrient-specific following the protein supplement. Shortly after the ingestion of the high-energy supplement a significant increase in protein intake was found. However, a significant reduction in carbohydrate and not protein, as would be expected, was observed for the protein supplemented group. The explanation offered by Stockley et al. (1984) for the decrease in carbohydrate as opposed to protein was that the composition of the protein supplement differed from the nutrient protein, in that there was a greater proportion of tryptophan in the supplement than is generally found in the nutrient.

Poor compensation for the ingestion of the energy supplement lends no support to the findings of Booth et al. (1982) where a preload high in starch content delayed dietary intake. Stockley et al. (1984) offered an explanation for the discrepancies in the two studies in that both studies differed in their composition of the preloads; Stockley et al. (1984) used fat and carbohydrate, whilst Booth et al. (1982) used solely starch.

De Castro (1987) suggested that human feeding was preprandial in nature; that is the amount ingested in a meal was associated with preceding conditions, such as length of time gone by since last meal and amount of food consumed. The general hypothesis being that prior food consumption should affect subsequent food intake due to the macronutrients remaining in the stomach which would influence the ingestion of that particular nutrient thereafter. In a nine-day study conducted by De Castro (1987) under natural conditions, whilst there was evidence that protein depressed subsequent food intake in thirty-eight normal-weight subjects, and fat was reported to be the least sating, subsequent food intake was unaffected by carbohydrate consumption when food diaries were subject to a computerised macronutrient analysis. Further, although De Castro's (1987) results supported to some extent the hypothesis that the satiating properties of macronutrients may differ, it was nevertheless concluded that all three nutrients reduced subsequent food intake.

Conclusions

To sum up, there are plausible hypotheses that carbohydrate ingestion increases subsequent carbohydrate ingestion and/or energy intake. Different carbohydrates may affect hunger and satiety differently, in that some carbohydrates (i.e. refined sugars versus complex carbohydrates) may stimulate appetite more than others. There is also some evidence that protein possesses more powerful satiating properties than carbohydrate or fat. There are equally plausible hypotheses predicting that refined sugars such as glucose are more or less sating than artificial sweeteners. The combination of small numbers and poor control of psychological factors in these studies make it difficult to disentangle physiological effects from situational, learned

and orosensory factors. Another problem is that most of the studies failed to record baseline measures of prior food consumption. Further, although it seems that insulin may be implicated in feeding behaviour the exact role it plays is still not clear. These differences in studies suffice to make it difficult to guarantee that results from one study will generalise to conditions in other experiments.

The Effects of Weight and Bulk on Satiety

It is still not certain whether the actual nutrients rather than factors such as weight, volume or caloric content are responsible for the satiating effects of certain foods. Kissileff et al. (1984) conducted a series of experiments where they tested the satiating "efficiency" of certain foodstuffs rather than specific nutrients. In one of the experiments it was observed that when preloads of either soup (109 kcal) or crackers, cheese and apple juice (256 kcal) were delivered to ten normal-weight females, caloric intake in the subsequent test meal was reduced on average by approximately 182 kcal more after the soup preload than after the mixed preload. Both preloads were delivered to all subjects on two separate occasions. These findings suggest that the power of satiation in some foods is stronger than that in others and this is quite independent of caloric content.

It had been postulated that because the soup administered in these experiments contained less fat content than the combination preloads and was heavier in weight, these factors were possibly responsible for the more rapid promotion of satiety following the soup preloads. Kissileff et al. (1984) decided to test this hypothesis. Eight subjects were given one of eight soups which varied in nutrient and weight composition. Soups contained either 38.5 kcal or 115.5 kcal. Fat content varied between 16% and 30% and levels of weight varied between 100 g and 300 g. Four subjects received the high-fat soups first and the other subjects were given the low-fat soups. Their findings revealed that for those who had been given the high energy soups (115 kcal) subsequent food intake decreased by 94.2 g less in the main course

when compared to those who had consumed the low energy soup (38 kcal). However, an increase in 39.8 g in the test meal was observed following ingestion of the high-fat soup when compared to the low-fat soup, although this finding was not statistically significant. Lastly, the effect of the weight of the soup preloads was not significant with respect to subsequent food intake.

It was concluded that the effect of the weight or bulk of the soup preloads was not significant with respect to subsequent food intake and that satiating effects of certain foodstuffs were more likely to be attributable to nutrient dispersion. The fact that subjects were not significantly influenced by the bulk of the food is consistent with some findings (Adolph, 1968), but inconsistent with other studies (Spiegel, 1973; Smith, 1962, Jordan, 1969, Monello and Mayer, 1967). Kissileff et al. (1984) also noted in this study that the oropharyngeal factors such as temperature, density of food and palatability could be relevant to the mechanism underlying satiety (Cabanac, 1971; Rolls et al., 1981). However, cognitive factors were not completely controlled for and could have interacted with the internal mechanisms that were operating. Again small numbers were used in this series of studies and so it is possible that the atypical responses of this small group distorted the results. Although pre-tests were given in all four experiments this does not necessarily provide accurate pre-meal baseline measures if subjects consumed food in between the time prior to the test meals.

Long-term Satiating Effects of Carbohydrates and other Nutrients on Obese Subjects

It has been proposed that carbohydrate diets increase appetite and are not as effective in weight reducing regimes as protein and fat. In an early study Fryer et al. (1955) tested the satiating value of carbohydrate, fat and protein on twelve overweight male students wishing to lose weight over a nine-week period. Subjects were placed on all three diets which were isocaloric (1800 kcal), each varying in nutrient composition for a three-week interval. Prior to being placed on an isocaloric diet subjects were fed a

normal diet for one full day. Based on the results from hunger ratings it was concluded that the protein component of the diet played a more integral role in suppressing appetite than either carbohydrate or lipid components. However, as previously mentioned, hunger ratings do not always provide accurate measures of appetite and again sample size was small.

In an experiment conducted in a natural setting with six obese subjects, Yudkin and Carey (1960) found that after a two-week baseline period of normal eating obese subjects, who decreased their carbohydrate intake to 50 g per day for the following fourteen experimental days (but consumed fat and protein adlibitum), revealed an overall reduction in total caloric intake. Yudkin and Carey (1960) suggested that carbohydrate lacks the satiating properties characteristic of other nutrients, particularly protein. Yudkin (1959) claimed that carbohydrate not only prevented weight loss but possibly enhanced hunger as presumably an increase in carbohydrate intake often results in an increase in fat consumption. However, these results may only apply to the obese. In a later study by Stock and Yudkin (1970) eleven normal subjects were studied using the same methodology, except an additional daily 20 mg of milk was recommended along with vitamins together with adlib intake of protein and fat. Their findings revealed that a greater decrease in energy consumption occurred after carbohydrate restriction (by 50 g) than after an overall reduction in nutrients. The problem with these studies is that there are no control groups with which to compare the results.

In contrast, Kinsell et al. (1964) found in a long-term study with five overweight subjects that the determining factor in weight loss was a restriction in total caloric intake rather than a restriction of a specific nutrient. In this study weight loss occurred in subjects, irrespective of nutrients ingested on low-calorie liquid-formula diets (800-1200 Kcal) varying in nutrient content. This is contrary to the prediction that carbohydrate diets stimulate appetite and are not very effective for dieters. It is not

certain whether these conclusions drawn by Kinsell et al. (1964) are applicable only for weight reduction diets.

Null Effects in Obese Studies

Rosen et al. (1982, 1985) found no evidence to support the notion that a carbohydrate-restricted, protein-supplemented fast suppressed appetite and subsequent caloric intake when compared to an isocaloric carbohydrate-containing diet, in a six-week study with a group of obese female in-patients. However, unlike previous studies, the carbohydrate-containing diet was by no means carbohydrate-rich; containing only 29% carbohydrate compared to 39% protein and 36% fat. The fact that a greater amount of protein was contained in the diet could account for the lack of evidence that carbohydrate-containing diets are less satiating than high-protein or high-fat diets as it is possible that the satiating properties of the nutrient protein were still operating.

Sunkin and Garrow (1982) conducted three studies where they failed to find any evidence that a protein-rich formula, administered under blind conditions, depressed obese patients' food intake more than a isoenergetic preload of carbohydrate. In the first part of the study, which was regarded as a pilot trial, two obese inpatients were given a protein-rich (100.6 g protein, 100.9 g carbohydrate, 24.3 g fat) or a protein-poor formula (51.6 g protein, 150.9 g carbohydrate, 23.1g fat) in a concealed container to consume adlibitum over lunch for six days (12.00-14.00h). The containers were collected at 14.00h everyday and were subsequently weighed and hunger ratings were used by the subjects to assess saticty levels after the formula. To control for food consumption throughout the day a standard breakfast was provided to subjects at 09.00h during the six-day period (200 kcal) and at 17.45h a fixed supper (200 kcal) was given to subjects. They found that the hunger ratings for the protein-rich and protein-poor formulas did not statistically differ and although one subject consumed more of the protein-rich formula than the other this was not statistically significant.

In part two of the study, lunches which were high or low in protein (500 kcal) were given to subjects in the second and third week of the study (in counterbalanced order), whilst in the first week a fixed diet was administered (800 kcal). During the test weeks a standard breakfast was included (fruit juice-50 kcal). For the test meal a bland liquid foodstuff was served adlibitum between 17.15-18.45h and the amount consumed was calculated. No significant differences were found in the caloric intake for supper, irrespective of the formulas ingested. The same procedure was used in part three of the study as in part two (with four obese subjects) except that the protein-rich and protein-poor formulas were alternated daily for ten consecutive days rather than weekly as in part two. Their results confirmed the findings from part one and two, in that Sunkin and Garrow (1982) failed to find any evidence that a protein-rich liquid food suppressed intake more than the formula low in protein for three out of four subjects. However, it is difficult to make any firm conclusions about the satiety effects of the nutrient protein when such a small sample was used.

Conclusions

Thus, some obese studies have found that carbohydrates increase appetite and food intake, whilst the ingestion of nutrients such as protein will delay food intake. However, there are equally plausible hypotheses predicting the opposite effects. Currently, high-carbohydrate diets, particularly those high in fibre, are being recommended for weight loss as it is suspected that the dietary fibre may "modulate and sometimes prolong the satiating actions of the food" (Blundell et al., 1994). However, it should be noted that the satiating effects of carbohydrates may differ markedly depending upon whether food is administered blind or not, and the presence of other nutrients (e.g. fat) in the food must also be considered.

Overall Summary and Conclusions

To sum up, the consumption of carbohydrates such as sugars has been shown to influence subsequent appetite and eating, but the directionality of effects has been inconsistent. In normal subjects some studies have found that ingesting "high-carbohydrate" food (which is not defined consistently) increases appetite and/or food intake (Geiselman and Novin, 1982a; Novin et al., 1973; Teff et al., 1989; Rolls et al., 1988). Different carbohydrates may affect hunger and satiety differently, in that some carbohydrates (i.e. certain sugars) may increase appetite more than others. Other studies looking for such an effect have failed to find it (Geliebter, 1979; Stockley et al., 1984). Yet other studies have found the opposite effect; that ingesting "high-carbohydrate" food reduces appetite and/or food intake (Booth et al., 1970b; Rogers et al., 1988; Rogers and Blundell, 1989), although not as much as food high in protein (Booth et al., 1970a). Similarly varied results have been found with obese subjects (Fryer et al., 1955; Yudkin and Carey, 1960; Rosen et al., 1982, 1985; Sunkin and Garrow, 1982).

Interpreting this literature is complicated by (a) the fact that some "high-carbohydrate" food administered is additionally high in fat and the bulk of the foods administered also varies. (b) Food is not always administered blind and the response to it may be a learned reaction to its sensory properties, rather than the direct result of its unconditioned metabolic effects. (c) It is physiologically plausible that carbohydrate metabolism could lead to varied effects on appetite and food intake over time. Most likely is that carbohydrate intake would initially suppress appetite (d) Appetite and food intake also vary with time of day and learned food habits. For example, most people are likely to lunch on specific foods at a specific time and it is questionable as to whether a modest preload of food will substantially affect these habits. Thus perhaps any statements about the effects of carbohydrates on appetite or eating behaviour need to be qualified by exactly what were the constituents of the 'carbohydrate', the extent to which people were aware of what they were eating, how long after ingestion the

effects occurred, and how effects interact with the nutritional habits of the people being investigated.

In a recent paper on carbohydrates and appetite regulation by Blundell et al. (1994) (published after the review work in this thesis had been completed) it was concluded that various carbohydrates (such as glucose, sucrose, fructose, maltodextrins and polysaccharides) decrease subsequent food intake. According to Blundell et al. (1994) most carbohydrates will "suppress later intake by an amount roughly equivalent to their energy value, although the time course of the suppressive action may vary according to the rate at which the carbohydrates are metabolised". Furthermore, there is recent evidence that fat and not carbohydrate, is the nutrient associated with poor satiety, overeating and weight gain (see Rolls, in press for a review).

It is likely that some of the different findings reported in this chapter are due to variations in methodology. These differences in studies make it difficult to guarantee that results from one study will generalise to conditions in other experiments. Despite the methodological difficulties discussed throughout this chapter and the lack of substantiated findings on the satiating effects of carbohydrates there is growing evidence that various nutrients have a different effect on satiety and subsequent intake. However, in order to attain a better understanding of the differential effects of nutrients on satiety (which will ultimately result in better strategies to control food consumption) some systematic measurement procedures which would permit better comparison among experiments need to be developed.

Proposals for Future Research

In order to improve methodology it is proposed that attention be paid to at least six methodological factors some of which have already been mentioned earlier on but will be summarised here. First, further examination of individual variation is required (particularly when such small samples are used). Second, there is a need for more studies to control or standardise subjects' eating behaviour prior to the experiment. Third, the role of expectancies in subjects participating in food studies should also be carefully considered. Folk psychology and lay beliefs about the effects of carbohydrates could influence experimental results, especially since food is rarely administered blind. Fourth, findings from studies which have found that carbohydrates increase appetite and/or eating may be due to the artificial nature of the methodological design used. Fifth, the palatability of some carbohydrate-rich foods or protein-rich foods increase or suppress appetite. Therefore, studies which do not control for palatability and report appetite increasing/suppressing effects following intake of foods high in carbohydrate could be reporting findings that are more related to the unconditioned orosensory components involved in eating than to the physiological effects of the nutrient. Sixth, there is a need to re-emphasise the importance of delay effects of preloads which should be seriously considered before presenting test meals or hunger rating questionnaires at a specific time; five minutes might be too soon, but four hours might be too late to detect any effect. Thus, any effects that are found may be unrelated to the nutrients per se but more attributable to a whole host of extraneous factors.

It is also important to bear in mind that with short term studies these are merely brief exposure tests which normally last for a few hours. Therefore, it would seem premature to make generalisations about the regulation of nutrient intake based on the findings of these studies alone. More long-term studies are needed in order to understand the relation between nutrients, hunger and satiety. Another strategy for

studying macronutrient satiety in humans is to use naturalistic methods more frequently which may help to eliminate problems inherent in laboratory testing.

Chapter 3-The Role of Internal and External Factors in the Regulation of Intake in the Obese and Non-Obese

Introduction

It was proposed in chapter 2 that nutrient regulation in humans is influenced by non-physiological factors which can often interact with or even override the internal mechanisms related to true hunger (Nisbett and Storms, 1975; Wooley, 1972). Cognitive factors probably play a more significant role in human eating behaviour than in animal feeding.

Internal and External Factors as related to Obesity

Schachter (1967) theorised that the mechanisms which regulate food intake operate differently for obese and non-obese individuals. He believed that there was sufficient reason "to suspect that the eating behaviour of the obese is relatively unrelated to any internal gut state, but was governed more by external stimuli". More specifically, he postulated that the internal physiological conditions which vary with levels of hunger and satiety, including gastric motility and arteriovenous blood sugar differences, influence the feeding behaviour of normal-weight individuals, but not the obese. Bruch (1961) believed that the obese were unable to distinguish between nutritional deficit and surfeit on a true physiological level, unlike normal subjects. Furthermore, Stunkard and Koch (1964) found that obese subjects had a significantly lower correlation between self-reports of hunger, and extent of gastric motility than non-obese subjects. Stunkard (1959) postulated that obese people differ from normal-weight people with respect to hunger, in that physiological cues signalling hunger may differ between both groups.

External cues are defined as those factors that are not biologically caused by the content of food ingested, but still have an effect on human food-intake behaviour. Cognitive, emotional, and situational variables are regarded as strong external cues. Orosensory factors such as the taste properties of food, and smell are also considered as external cues which may mediate gastric sensations, and hunger feelings.

Evidence in support of Schachter's theory (1967) stems from a series of studies in the 1960's and 1970's which found differences between obese and normal-weight individuals in relation to externality (Nisbett, 1968a, 1968b; Schachter and Gross, 1968; Schachter, Goldman and Gordon, 1968; Campbell et al., 1971). Schachter and his colleagues attempted to pinpoint the determinants of the apparently different modes of behaviour in the obese and non-obese. Schachter (1967) claimed the over-responsiveness to external cues found in obese subjects played a major role in the psychopathology of obesity. It has also been hypothesised that those suffering from eating disorders such as bulimia or anorexia nervosa or restrained eating are less responsive to the internal physiological stimuli which should play a significant role in food intake regulation.

Dietary Restraint

Much of the research testing the internal-external hypothesis has not considered other factors such as dietary restraint rather than obesity per se as one of the principal components in an individual's behavioural response to internal and external food related cues. On reviewing Schachter's model of obesity Hibscher and Herman (1977) suggested that restraint rather than weight could be a determining factor in the degree of external responsiveness to food. The role of dietary restraint in food regulation has been explained in the "boundary model" devised by Herman and Polivy (1984). It describes how physiological control of eating is used by normal, unrestrained eaters, while food intake is more cognitively controlled by restrained eaters. The boundary model indicates that restrained eaters may "lose the ability to detect the subtle

precursors of satiety....." (Polivy and Herman, 1987). However, although it has been claimed that cognitive control is more prevalent in the obese and dieting populations, it has been found in some studies that not all obese exercise dietary restraint or counterregulate (Ruderman, 1986) and that some normal-weight people may counterregulate on certain occasions (Tomarken and Kirschenbaum, 1984). Other studies have shown that some unrestrained eaters are also highly responsive to external cues (Rodin, 1981a, Wooley, 1972; Nisbett and Storms, 1975).

This chapter reviews the differences between the regulation of intake in the obese and non-obese concentrating principally on the literature of 1968-1984. For brevity, a full analysis of all the studies in this field will not be given. Only the work which seems directly related to the main issues, (i.e. the role of external and internal factors in the regulation of food intake in the obese and non-obese individual) will be fully reviewed. Studies after 1984 have not been reviewed because they have not directly compared the externality of obese and normal-weight subjects. Some studies which have concentrated solely on the effects of food on the physiological regulation system of normal-weight subjects will be briefly reviewed in order to obtain comparative data but a thorough evaluation of the literature will not be given. Brief reference will be made to dietary restraint, but only when there seems some common link between the eating behaviour of restrained eaters and the obese. Tables will be used (see table 3.1 and 3.2) to outline the basic design of most of the studies reviewed.

Each study will be evaluated in terms of type of food ingested by the subjects (liquid versus solid foods), the method of food administration (dispensing machines, hand-held containers, food presented on trays). Deprivation periods where applicable to certain studies will also be examined. The most relevant short-term studies will be reviewed first, starting with those which revealed physiological inadequacies in normal-weight subjects. Short-and long-term studies in the obese and non-obese will be reviewed thereafter.

Furthermore, it is important to mention at this stage that to date much reference has been made to the study of physiological regulation without establishing a clear criterion of what is meant by terms such as "good" or "poor" compensation, "accurate" or "inaccurate" compensation and "efficient" or "inefficient" compensation in the obese and non-obese subject. There is no consensus for the use of any of these terms and different studies may define each term differently. For instance, 55% compensation may be regarded as "efficient" compensation by some investigators, but not by others who may consider 80% as "efficient". Some studies fail to cite the exact amount of compensation attained by subjects and instead use one or more of the above terms to describe their results. However, since it is unclear what the experimenter means by using any of these terms their findings could end up by providing ambiguous or misconstrued data. In the studies reviewed in this chapter the terms used by the experimenters will be cited and whenever possible the exact amount of compensation will be provided, but in the final analysis it is left to the reader to derive his/her own conclusions as to whether "adequate" physiological compensation has been achieved or not.

Table3.1-Studies on physiological regulation in the obese and non-obese (with effects)

Author	Sample composition	Food	Deprivation period	Blind administration	Duration of eating	Baseline measures
Schacter & Gross, 1968	22 normal (mean 18.6 yrs) 22 obese (mean 19.2 yrs)	solid, palatable food (crackers)	not monitored	no	15 mins	no
Nisbett, 1968a	normal, underweight & overweight. number not specified	solid, palatable food (sandwiches, cheese, coke, ice-cream)	4 hrs minimum	no	approx. 15 mins	no
Nisbett, 1968b	students <26 yrs. number not specified	solid, palatable food (sandwiches)	4 hrs minimum	no	not specified	no
Goldman, Jaffa & Schacter, 1968	obese & normal.number not specified.	NA	NA	no	NA	no
Hashim & Van Itallie, 1965	2 normal male, 20 yrs & 5 obese	ad lib intake of formula diet (50% carbohydrate, 20% protein, 30% fat of total calories) with food dispensing device	NA	yes	not specified	no
Campbell, Hashim & Van Itallie, 1971	5 normal male students (20-25yrs), 6 obese (13-30 yrs)	liquid diet from a disperser (45% carbohydrate, 15% protein, 40% fat of total calories	NA	yes	not specified	yes; 1-7 days on standard diet
Porikos et al. 1977	8 obese inpatients	ad lib intake from platter system of normal solid food	NA	no, but covert change in caloric density of diet	not specified	yes
Spiegel, 1973	Expt.A 8 normals, 1F, 7M. Expt.B 15 normals (18-36yrs) 2F,13M	Metrecal liquid preload (between 0.25-1.8 kcal/ml) then Metrecal liquid test meal (1.0 kcal/ml) adlib intake of Metrecal liquid diet (1.0 kcal/ml) 4-9days, then (0.5kcal/ml) 4-14 days	1h between preload and test meal or 3-5h NA	no no	not specified not specified	yes-several days same as above
Pliner, 1973	48 obese, 48 normal	palatable liquid & solid foods (600 or 200kcal liquid preload (45% carbohydrate, 15% protein, 40% fat) 200 or 600kcal solid food (84% carbohydrate, 6% protein, 10% fat)	Fast on day of study	yes	test meal-12 mins, 60 mins after preload	no

Table 3.2 Studies without significant effects

Authors	Sample Composition	Food	Deprivation period	Blind administration	Duration of eating	Baseline Measures
Wooley, 1972	16 obese, 16 normal	600 or 200kcal	several hrs after a standard breakfast	yes	not specified	practice but no baseline
Wooley, 1971	5 normal, 6 obese	adlib liquid diet, high or low energy (lactose, sucrose, water, powdered chocolate)	NA	yes	NA	no
Wooley, Wooley & Durham, 1972	7 obese, 7 lean	liquid meals, high-energy, high-CHO or low- energy, low-CHO	not stated	yes	not specified	yes 5-10 days
Durrant & Royston, 1982	6 lean (4f, 2m), 14 obese (f)	adlib food from food dispensing machine (packet food) & preloads (liquid)	not stated	yes	not specified	1 day of practice, but no baseline
Porikos et al. 1981	Study a: 8 obese Study b: 6 normal Study c: 5 obese, 8 normal	palatable solid conventional foods	NA	covert dilution of energy content of diet	not specified	yes

Irregular Physiological Regulation in Normal-Weight Subjects

Some studies have examined the effects of a caloric preload on subsequent food intake in normal-weight subjects, finding that caloric manipulation of a preload does not always result in accurate compensation of food intake (Speigel, 1973; Rolls et al., 1986; Hill et al., 1987, Porikos et al., 1977). Walike et al. (1969) found that, after seventeen subjects were preloaded with a machine dispensed liquid formula of Metrecal consisting of 20-120% of their spontaneous food intake, they over consumed one to one hundred and twenty minutes later when they were administered a test meal. Spiegel (1973) found both good and poor control of energy intake in normal subjects. In a short-term study eight normals were unable to accommodate the changes in caloric concentration of a preload by altering intake in the adlibitum feeding period efficiently. In the second experiment (Spiegel, 1973b), lasting ten to twenty-one days, only six of the fifteen of the subjects were able to increase the amount of food consumed and dietary regulation took two to five days to occur. In a long-term study (twenty-four days) Porikos et al. (1982) indicated that caloric regulation for normals took at least three days and even then only 40% of the reduction was compensated for.

Further, Sylvestre et al. (1989) found that a 200 kcal difference in a preload administered to normal-weight adolescents was not accommodated in a test meal which followed one hour later. In a short-term study Rolls and Hetherington (1986) found that twenty-four normal-weight female subjects showed no real ability to compensate accurately for caloric changes in their diet when administered high or low-calorie foods. Two different types of foods were used in the study. The first consisted of either a high or low energy soup (0.49 kcals V's 0.07 kcals) and the second was devised from a jello substance (0.54 kcals V's 0.09 kcals). Rolls et al. (1986) found that despite the caloric variation in test meals there was no notable difference in the volume of food ingested and subjects' hunger ratings and stomach fullness were more or less the same for both the high and low calorie foods. Furthermore, the second

course offered one hour later, which consisted of cheese and crackers, demonstrated that subjects showed no real ability to compensate accurately for caloric changes in their diet in the first course, in that no notable differences were perceived regarding consumption in the second courses. Lastly, Hill et al. (1987) reported that, although normals were able to adjust to caloric manipulation by increasing or decreasing intake, subjects were not always very efficient or responsive on an internal basis to the caloric content of certain food. Thus, it seems that normal-weight individuals are unable to regulate food intake well subsequent to caloric manipulation. These findings are of importance when considering Schachter's internal-external theory (1967) and the studies which support this theory.

Irregular Physiological Regulation in Obese Subjects

In support of the theory that external cues predominantly influence eating behaviour in the obese, Schachter and Gross (1968) conducted an experiment where they examined the effect of time as an external cue on the eating behaviour of forty-six normal and overweight male individuals. Schachter and Gross (1968) hypothesised that the eating behaviour of the obese but not the non-obese would be more responsive to the manipulation of time. Two clocks were used in the experiment, one was set to run at half normal speed, whilst the other was set to run at twice normal speed.

Their results revealed that amongst the obese and non-obese subjects who believed that it was one hour past their usual dinner time, only the obese subjects consumed more of the crackers presented to them for adlibitum consumption. In contrast, the obese subjects, but not the normal-weight subjects who were led to believe that they were eating one hour earlier than normal, consumed almost half the amount of crackers consumed by the first group.

The fact that in the obese group there was a tendency for level of hunger to rise in conjunction with what time they thought it was, irrespective of what had previously

been consumed, seems to delineate the importance of cognitive factors, regardless of hunger state, in the obese individual. The time manipulation for normal-weight subjects caused them to behave in the opposite way, in that they were reluctant to eat the crackers for fear of spoiling their appetite for dinner. This phenomenon is interesting since both groups seem responsive to cognitive influence, but react to it differently. In this study it can be seen that for normal subjects cognitive factors are presumably interacting with physiological regulation, for at this point most of them are probably experiencing hunger on a physiological level, yet they reveal imposed self-control by reducing their intake. Further evidence of cognitive effects may be found in Nisbett's study (1968), where subjects deliberately avoided increasing their consumption of ice cream when food was deprived for fear of "making a meal" of ice cream.

Nonetheless, based on Schachter and Gross's findings (1968) it does appear that obese subjects are more influenced by the time shown on the clock as regards eating than normal-weight subjects. However, whether this fact indicates that normal subjects' feeding behaviour is more of a physiological phenomenon is open to question. In effect, these results fail to support the view that the eating behaviour of normal-weight subjects is physiologically controlled. What this study does suggest is that obese individuals are even less influenced by internal factors than the non-obese.

Goldman, Jaffa and Schachter (1968) also found evidence in a series of studies on obese and non-obese Jews that the obese Jews were more responsive to external stimuli than the non-obese on a variety of food related issues. For example, by using a questionnaire they found that overweight subjects are more prone to fast on Yom Kippur than normal-weight subjects. They also found that overweight subjects showed an inverse relation between the "unpleasantness" of food deprivation and the number of hours they were in the synagogue on Yom Kippur. It was hypothesised that this was due to lack of external related food cues, while normal-weight subjects

showed no such relationship. Based on these findings Goldman et al. (1968) claimed that the obese are unable to detect hunger on a physiological level. However, if as Goldman et al. (1968) have proposed that the obese do not seem to find food deprivation difficult (so long as external food related cues are removed) then surely "manipulated food deprivation" in the laboratory may have little bearing on the ability of the obese subject to regulate eating on a physiological level?

The Role of Visual Cues as External Stimuli

In a study with obese, underweight, and normal-weight male students Nisbett (1968a) reported that eating by the obese was more influenced by external factors, such as the visibility of the food, rather than visceral cues which motivated normal and underweight subjects. In this short-term study subjects were asked to fast for four hours after breakfast. There were two experimental conditions in the study; either one or three sandwiches were offered to subjects who were informed that there were "dozens more" sandwiches available from a refrigerator on the other side of the room, and that access was unrestricted. It was found, as hypothesised, that the obese individual, who responds more to external cues, would eat more when presented with three sandwiches than when presented with only one. However, obese subjects consumed less than the normal-weight subjects. After three sandwiches they ate approximately the same amount as underweight subjects when only one sandwich was available (57% less than when offered three sandwiches). The fact that the unseen sandwiches were not visually presented to them seemed to deter them from further consumption.

Nisbett (1968a) concluded that the obese are more influenced by external factors such as the sight of food, rather than internal cues that motivate normal and underweight subjects to eat which concurs with Schachter's theory (1967) regarding obese sensitivity to environmental stimuli.

Nisbett's Set Point Theory

In 1972 Nisbett offered an alternative explanation for obesity based on set point. He theorised that some dieters and obese individuals are underweight with respect to their set point or ideal weight, and are consequently always food deprived. This deprivation may increase their sensitivity to salient external stimuli, which could explain why the external responsiveness of normal and overweight subjects may differ. Nisbett (1972) claimed that there are many similarities between obese and starving organisms. Both are more emotional and more responsive to taste.

A study by Goldman, Jaffa and Schachter (1968) used liquid food, supplied by a food dispenser and kept in another room to prevent subjects monitoring how much they were consuming. Overweight subjects consumed much more of a chocolate milkshake than normals, irrespective of the level of deprivation. However, when the machine was placed in the room, and visual cues indicating the amount consumed were present, then obese subjects significantly reduced their intake, and were more responsive to deprivation levels. Ross (1974) also found that the obese were more influenced by the sight of food; they consumed more cashew nuts when placed in a brightly lit room than when placed in a dim room. Normals, in comparison, consumed the same amount in both conditions.

However, none of these studies controlled for prior food intake by keeping baseline measures and cognitive factors were rarely controlled. Nevertheless, from these studies, despite the usual methodological problems, it would appear that the obese are more externally responsive to food cues than normal-weight subjects.

The Role of Palatability in Eating Behaviour

It has already been mentioned in chapter 2 that calorie or nutrient intake can be largely determined by the palatability of food. Palatability has been known to influence not only which nutrient a subject will ingest, but also in what amount he/she will ingest it;

more food is likely to be consumed if the food available is highly palatable (Hill and McCutcheon, 1975). Several studies have found that people eat more when the foodstuffs provided are highly palatable (Hill, 1974; Nisbett, 1968; Rolls, Rolls, Rowe, 1981), at least on the short-term, although it has been proposed that regulation of food intake over the long-term is not affected by the palatability of food (Levitsky, 1980).

Palatability and Obesity

It has been demonstrated that there is a high correlation between excess weight and palatability of food. When a variety of highly palatable diets are offered to animals (Sclafani and Springer, 1976) and humans (Porikos et al., 1982; Pliner et al., 1980; Rolls et al., 1980) overeating may be induced which contributes to the onset of obesity. Conversely, when the orosensory properties of food are held constant over a period of days, weight is subsequently lost in obese subjects (Hashim and Van Itallie, 1965). Cabanac and Rabe (1976) showed how bland monotonous diets promoted undereating and weight loss in normal-weight males over twenty-one days.

The claim that the obese differ in their control of food intake (Schachter, 1967) was further supported when several studies reported differences in eating behaviour between overweight and normal-weight subjects in response to highly palatable food. The obese are more responsive to highly palatable food (Hashim and Van Itallie, 1965; Nisbett, 1968a; Price and Grinker, 1973), and are more influenced by the visual salience of food (Nisbett, 1968b; Ross, 1974). As yet, it has not been established whether this heightened responsiveness to palatable food is due to a difference in the internal state or in the hedonic properties between the two groups.

Nisbett (1968b) examined the effects of taste on eating behaviour and found that the obese consumed more palatable ice cream than ice cream adulterated with quinine, whereas both normal and underweight subjects were less sensitive to the variation in

taste. The fact, however, that orosensory factors were not masked in this study makes it possible that the normal-weight and underweight subjects were more influenced by the cognitive factors present than the obese and consequently imposed more self-control.

A long-term study by Hashim and Van Itallie (1965) found that obese individuals were greatly affected by the manipulation of taste in their diets. When both normal and obese subjects were placed on a bland, unpalatable liquid diet, supplied by a food dispenser, on an adlib basis, normal-weight subjects maintained their body weight, and their caloric intake during the experiment was approximately the same as their pre treatment intake. In contrast, the obese subjects clearly lost weight due to a dramatic decrease in caloric consumption. This study, however, used fewer than ten subjects and the obese subjects were in-patients, so their results may have been influenced by an underlying desire in the obese to lose weight.

Abnormal Taste Profiles in the Obese

Some researchers have claimed that the taste profiles of the obese are more sensitive to sweet foodstuff than those of normal-weight subjects, and that this difference in taste could partly reflect their physiological state (Rodin, 1975). Similar claims have been made for individuals suffering from eating disorders such as anorexia and bulimia nervosa (Drewnowski et al., 1987; Jirik-Baab et al., 1986). Moskowitz et al. (1974) investigated preferences for 5% versus 30% sugar solutions. Individuals with high blood sugar found the very sweet sugar solution to be less pleasant, whereas individuals with low blood sugar preferred the sweeter solution. Therefore, it is possible that for some obese individuals low blood sugar plays a role in the etiology of obesity.

Other studies however have failed to support this interpretation (Drewnowski et al. 1985; Grinker, Hirsch, Smith, 1972; Rodin, Moskowitz and Bray, 1976). An

alternative proposal is that the heightened responsiveness to palatable food by obese individuals is a result of their restrained, frequently erratic, eating patterns. Cabanac et al. (1975) suggest that following a period of food deprivation, nutritional influences on the physiology of the body are not as effective and the individual may remain highly responsive to sweet taste. Thus, levels of responsiveness to palatable food may differ depending on the level of deprivation and on the state of the organism at a given time. Booth (1972) claimed that the attractiveness of any food, especially palatable food, would increase for any subject who was in a state of hunger.

Defining Palatability

Palatability of food refers to the taste or "the hedonic or affective quality of orosensory stimuli as experienced by the consumer" (Rogers, 1993). Palatability plays a significant role in eating behaviour; it has been known to influence not only which nutrient a subject will ingest but also in what amount it will be ingested. Moreover, the palatability of food is regarded as more of a psychological and/or perceptual component of food (Herman and Polivy, 1988). However, it has also been recognised that palatability is affected by biological or innate factors which may be altered by learning (see Rogers, 1990b for a discussion).

Spitzer and Rodin (1981) claimed that perceptions of palatability differ from individual to individual. According to them, it is difficult to classify palatability as based on internal or external cues. As yet there is insufficient evidence that obese and normal-weight individuals differ with respect to the palatability of food and thus there is no clear support for this element of Schachter's internal-external theory of obesity (1967).

In chapter 1 the "Carbohydrate Craving Syndrome" (Wurtman et al., 1981) with respect to the obese was discussed. Even if sugar is considered an important factor in taste preferences for obese people, it is usually interlinked with other nutrients, specifically fat. Hence, persistent anecdotal reports of a "sweet tooth" or "carbohydrate

craving" among the obese may be somewhat erroneous. Calorie for calorie foods such as chocolate and ice cream contain more fat than carbohydrate (Drewnowski and Greenwood, 1983). Drewnowski and Greenwood (1983) demonstrated that taste preferences of normal-weight subjects were strongly influenced by the lipid content of sweet tasting foods. Heavy cream (37% fat w/w) with 10% sugar was found to be more desirable than a similar substance with the same sugar content but only 0.1% fat.

Perception of Internal Physiological Cues in the Regulation of Eating in the Obese and Non-Obese

Campbell, Hashim and Van Itallie (1971) found that normal-weight, but not obese individuals were able to compensate quite efficiently for caloric depletion or supplementation in their diet on a physiological level by adjusting subsequent nutrient intake. This supported Schachter's theory (1967).

Campbell et al. (1971) fed adult volunteers (five normal-weight subjects, four obese adults and two obese adolescents) with a palatable liquid diet of milk formulae dispensed from a machine for a period of one hundred and twenty-one days. Subjects were not informed that their food intake was being monitored or that the caloric density of the food was being manipulated. Great care was taken to ensure that the subjects' food intake was concealed so as to reduce the possibility of self-monitoring of energy intake in subjects, thereby decreasing the interaction of cognitive cues in the study. The overall implication was that if caloric compensation were to occur, then presumably it would be triggered by internal stimuli as opposed to external cues.

Normal-weight subjects regulated their energy consumption quite accurately. This regulation was achieved by their increasing the volume consumed from the dispensed liquid so as to maintain a constant level of energy intake and no significant increase or decrease in body weight, although there were minor daily fluctuations. Durnin (1961) reported day-to-day changes in body weight of up to 1 kg in forty-four male subjects,

whilst Edholm (1961) found changes over 0.5 kg on 30% of the days when sixty-four soldiers were weighed, and more than 1 kg on 6% of the occasions. In contrast, the obese adults failed to regulate their intake efficiently when their diet was subject to caloric manipulation. They failed to increase the volume of the diet and thus lost weight. Whilst the obese adolescents did not restrain their intake, they failed to increase the volume of the liquid diet.

Many factors may explain why the obese have shown less responsiveness to physiological cues than the normals. One of the most plausible hypotheses is that psychological factors might often override subjects' true internal, metabolic signals. This may frequently result in suppression of intake when hungry and over consumption when sated. Another plausible explanation is that obese individuals do suffer from some type of metabolic disorder, whereby they are unable to regulate energy intake on a physiological basis. However, because of the complexity of this problem it is not within the scope of this thesis to offer nor examine any of the etiological reasons of obesity and this has been done elsewhere (Conn, 1944; Bortz, 1969; Rabinowitz, 1970).

One possible criticism of the findings reported by Campbell et al. (1971) is that they failed to provide any information concerning when the caloric compensation occurred. According to previous findings a period of two to five days should be allowed for caloric adjustment, but it is not clear whether subjects in this study exceeded this time limit or not. Contrary to some past research Campbell et al. (1971) did not state whether compensation occurred as a result of alteration in the size of the meal, i.e. larger portions, or due to meal frequency, whereby subjects would increase or decrease the regularity of their meals.

The fact that the obese subjects consumed so little of the bland milk formula dispensed from a feeding machine makes one question the validity of the test. It seems clear from

this study and that of Hashim and Van Itallie (1965) that obese subjects are not very responsive to the ingestion of bland liquid food dispensed from a feeding machine. However, this does not necessarily imply that they are unable to regulate caloric intake on an everyday basis when solid palatable foods are ingested. Moreover, it is also possible that the obese adults in the study by Campbell et al. (1971) were exercising dietary restraint, so the main question of whether the obese regulate on a physiological basis remains unanswered, since psychological factors might have played a more influential role in their eating.

One possible explanation for the different results cited by Campbell et al. (1971) compared to other studies on long-term regulation which will be subsequently reviewed (Wooley, 1971; Porikos et al., 1982; Spiegel,1973) is the variation in time that each study expected subjects to remain on a diluted diet. For example, Wooley (1971) and Spiegel (1973, group A) monitored energy intake on a liquid diluted diet for a period of four to eight days. Yet, four to eight days may be insufficient time to observe any long-term adjustment to changes in caloric concentration of a diet. This viewpoint may be supported by the fact that compensation in both studies was extremely low; 11% and 4% respectively, when compared to 87% compensation in the study by Campbell et al. (1971) and 46% in Spiegel's study (1973, group B), where subjects remained on the diluted diet for five to fifteen days.

However, Porikos et al. (1982) found no evidence in their study to support the notion that caloric compensation is more accurate when the number of days is increased. They reported incomplete compensation following caloric dilution with aspartame over a ten day period. Their findings tended to support those of Wooley (1971) and Spiegel (1973), in that compensation could only account for 40% of the missing calories.

Another possible explanation for the discrepancies could be uncontrolled cognitive factors in some of the studies. In Wooley's study (1971) subjects were able to see the

amount of liquid concentration they were consuming, as was group A of Spiegel's study (1973). This immediately raises the question of whether this led them to rely on self-control techniques to regulate intake. Furthermore, Porikos et al. (1982) used solid, conventional, and palatable food. Even although Porikos et al. (1982) tried to counterbalance psychological stimuli by delineating the fact that they were interested purely with the effects that everyday eating had on the human physiology, it is impossible to ignore the fact that subjects' attention would have inevitably been drawn to their eating behaviour. The mere fact that they were participating in a food experiment would have made them conscious of their eating, and this would have undoubtedly increased any cognitive influences that existed.

In the study by Campbell et al. (1971), and in group B of Spiegel's study (1973) cognitive cues were minimised due to the fact that the machine-dispensed liquid was concealed, thereby preventing subjects from visually monitoring their intake. Campbell and his colleagues (1971) suggested that the only way individuals could have measured their energy consumption would have been by "counting the mouthfuls". The fact that these two studies demonstrated better caloric regulation in lean subjects would tend to suggest that physiologic cues were more predominant in studies where cognitive factors were minimised.

Liquid versus Solid Foodstuff in Food Regulation in the Obese and Non-Obese.

After examining the effects of solid and liquid food on the feeding behaviour of both normal and overweight subjects, Pliner (1973) hypothesised that for some obese individuals the physiological process which operates after food intake may differ depending on whether the foodstuff is of solid or of liquid composition. This experiment, similar to that of Campbell et al. (1971), focused on manipulating internal cues in both groups of subjects. Subjects were preloaded with either 200 or 600 kcal of a liquid or solid preload and were presented with a test meal one hour later. The test

meal consisted of sandwich quarters and subjects were given twelve minutes to eat them.

Normal-weight subjects regulated their intake in accordance with their energy intake from the preloads of liquid and solid foods. They ate on average 3.25 more sandwich quarters after the low calorie liquid preload (7.79 sandwich quarters) compared to after the high calorie solid preload (4.10 sandwich quarters). Furthermore, an average of 3.69 more sandwich quarters were eaten after the low calorie solid preload (8.17 versus 4.92 sandwich quarters). However, although normals preloaded with the low calorie liquid or solid foodstuff ate more afterwards than those who were given the 600 kcal preloads, their total energy intake was still less than the high calorie preload group. Nonetheless, there is no indication from these results that the subjects who were in the low calorie preload conditions underate. It is possible that the other group consumed an excess amount (Stellar, 1967 showed similar findings). Moreover, this study was examining how the internal system responds to short-term changes in food intake. Thus, it is quite possible that the subjects made up for this difference in energy intake later on, possibly even the same day.

In contrast, the obese group showed poor compensation, in that only a difference of 0.12 sandwich quarters was consumed after the low calorie solid preload. Obese subjects given the low calorie preload consumed 7.29 sandwich quarters, while obese subjects given the high calorie preload consumed 7.17 sandwich quarters. Compensation after the liquid preload was better; the obese group preloaded with the low calorie drink consumed 8.08 sandwich quarters, while the obese group given a high calorie drink ate 6.00 sandwich quarters.

These data again indicate that normals can regulate intake when the energy content of the food varies, but the obese cannot. It is interesting that this study revealed that the obese and non-obese groups' eating behaviour was responsive to the physiological

system after the liquid preloads, whilst only the normals were responsive to metabolic signals after the ingestion of the solid preloads. It is not yet clear why these differences exist, but they seem to suggest that the obese are more responsive to liquid manipulation on an internal basis than to solid manipulation. In Pliner's study (1973) there was also a slight solid-liquid difference noted for normal subjects. Despite the fact that Pliner's study (1973) was physiological in orientation, orosensory factors were not controlled for and differences existed in palatability, weight and richness of the preloads. Visual cues were also present when administering solid or liquid foods which may have influenced control of food intake in either group. Nonetheless, this study together with the studies by Campbell et al. (1971) and Hashim and Van Itallie (1965) reviewed earlier, have found physiological regulation to be less accurate in the obese compared with normal-weight subjects when manipulating physiological cues.

These findings support the internal-external theory, but other studies have failed to confirm that obese subjects are more responsive to external cues. Cognitive factors can influence food intake of normal-weight subjects as well as obese subjects (Wooley, 1971; Wooley, Wooley and Dunham, 1972). One study found that the food intake of normal-weight people was in effect influenced more by cognitive factors than was that of obese subjects (Nisbett and Storms, 1971).

Nonsignificant Results against the External-Internal Theory

Durrant et al. (1982), in a long-term experiment, tested the ability of both obese and non-obese subjects to counterbalance the effects of changes in the caloric density of a diet. Subjects delivered food to themselves through a dispensing machine. The experiment took place in a hospital setting over seven days with fourteen obese subjects, and six non-obese subjects. No preloads were administered to the subjects on the first day of the study. All subjects were administered blind, three daily preloads which varied in caloric density. On the second and third day subjects were provided with 600 kcal of food at breakfast, lunch, and dinner. On day four, five, six and seven

subjects were preloaded with a 900 kcal or 300 kcal liquid formula alternatively. Adlibitum consumption from the food dispensing machine was then permitted.

There were differences in overall energy intake between obese and lean subjects. Obese individuals showed a propensity to undereat and lose weight when feeding themselves from the machine. Durrant et al. (1982) proposed that the satiating power of the preloads was stronger for the obese than the non-obese. However, there is no substantial evidence to support this viewpoint. Alternatively, Durrant et al. (1982) postulated that the fact that the obese subjects were in hospital, specifically for the treatment of obesity, probably elicited dietary restraint in these subjects. Apparently, this type of behaviour is quite characteristic of obese subjects when placed under experimental conditions (Nisbett, 1968; Campbell et al., 1971) and is not necessarily typical of their natural eating behaviour.

However, despite undereating, obese subjects compensated for caloric deficit to some degree. This was one of the first studies to be published which found that obese individuals are responsive to concealed changes of caloric content. Compensation was far from accurate (70 kcal compared to an increase of energy intake of 176 kcal in the non-obese group). Although these data suggest a more efficient regulatory system in normal-weight subjects, thereby lending support to Schachter's theory (1967), Durrant and Royston (1982) pointed out that normals' regulation of food intake was also inaccurate.

A pilot study was conducted by Porikos, Booth and Van Itallie (1977) to investigate chiefly the effects of blind dietary dilution on subsequent food intake. Normal solid food was freely offered to eight obese subjects for their daily meals, using a platter system. The experiment lasted fifteen days, with the first three days being a baseline period. After baseline, the caloric concentration of some of the foodstuffs was manipulated, specifically drinks, desserts, jams and syrups, by substituting sucrose in

some of the foodstuff with aspartame. This manipulation lasted six consecutive days, after which the test diet was the same as the baseline period. Apart from the consumption of the three daily meals, subjects were also given unlimited availability to drinks and snacks which were placed in a refrigerator in their room and these were sometimes calorically manipulated. Food consumption was monitored throughout the study.

Their findings revealed that food intake during the sucrose period was significantly greater than during the aspartame dilution period. Furthermore, this increase in energy intake was not attributed to such factors as an increase in energy expenditure which might have partly accounted for the deficit in calories (Porikos et al., 1977). This again shows that humans are not able to compensate accurately for caloric change in their diet. None of the obese subjects compensated for the 25% caloric deficit, instead they continued to ingest the same volume of food as before. Over a six-day period compensation was inefficient, but it is possible that greater accuracy could have occurred over a longer time span. Moreover, these findings are based purely on the eating behaviour of obese subjects.

In 1981 Porikos conducted three studies examining nutrient intake in both normal and overweight subjects. Rather than using a feeding machine, in all three studies solid palatable food was offered to all subjects on a "platter" system over a period of fifteen to thirty days and subjects were unaware that their food intake was being manipulated. Food was weighed before it was delivered to the subjects on a platter, and after they had finished eating.

During the baseline period all subjects exceeded their normal intake of dietary energy and gained weight. This supports past research on animals (Sclafani and Springer, 1976) and humans (Pliner et al., 1980; Rolls et al., 1980) where highly palatable food plays a decisive role in overeating, and hence weight gain. When sucrose in the food

was replaced by aspartame both obese and non-obese subjects were unable to compensate accurately for the 25% caloric decrease in their diet. As was also found in the later study by Porikos et al. (1982), both obese and normal groups increased their dietary energy intake, but not enough to accommodate energy depletion. The fact that both groups overate and gained weight on the adlibitum palatable diet lends further support to the viewpoint (Wooley et al., 1972) that lean subjects are not superior in control of feeding to obese subjects.

Price and Grinker (1973) were also unable to find any evidence of accurate short-term compensatory behaviour in obese or non-obese subjects, following a solid food preload. Therefore, some of studies in this area have found that compensatory behaviour in both obese and non-obese subjects is incomplete. To some degree, this rules out the hypothesis that obese individuals suffer from some defect in the regulatory system, or are "physiologically" different from normal-weight people (Schachter, 1967).

Cognitive Sensory Factors in the Eating Behaviour of the Obese and Non-Obese

Cognitive considerations may involve one's thoughts and beliefs about certain foods. Pudel (1976) and Wooley (1972) have shown that both obese and non-obese subjects are sensitive to cognitive factors such as belief about prior caloric consumption. Although it has been demonstrated in several studies that normal-weight individuals are able to accommodate for preloads on the basis of physiological regulation, this compensation is strongly influenced by cognitive components. For example, compensatory intake after preloads is frequently based on what people believe they ate as opposed to what they actually consumed. It was also mentioned in chapter 2 that there is a tendency to consume less food after eating something that we believed to be sweet, irrespective of its caloric content (Brala and Hagen, 1983). Sensory factors may also contribute to the regulation of food intake. Garrow (1978) proposed that

cognitive considerations such as ideal body weight may influence voluntary food regulation in humans in the long-term. This is particularly true for dieters who voluntarily restrain their intake (Herman and Polivy, 1975) using psychological, external factors to control their food intake which override physiological, internal stimuli.

Results failing to support the External-Internal Theory

A series of studies conducted by Wooley in the 1970's brought the role of cognitions in the eating of obese and non-obese to the forefront. Wooley (1972) hypothesised that a person's perception and beliefs about what they have consumed may contribute to feelings of satiation, irrespective of the actual energy value of the food. She estimated a 40-60% adjustment in eating behaviour attributable to non-physiological regulation, specifically to cognitive regulation.

Wooley (1972) conducted a study to determine whether non-obese subjects are more responsive to caloric change on the physiological level than obese subjects, when the influence of cognitive factors was minimised; Secondly to determine whether cognitive factors are likely to alter intake, and/or affect satiety levels. Sixteen obese and sixteen non-obese student volunteers were preloaded with high or low calorie drinks containing 600 kcal or 200 kcal. The drinks were either a high calorie milk shake or a low caloric diet drink and were administered at noon on four consecutive days. Both the normals and overweight subjects did not control their intake efficiently when presented with their choice of sandwiches twenty minutes after the preloads, thereby indicating that the physiological regulatory system in both groups was inefficient.

However, subjects adjusted intake in accordance with prior information about the caloric content of the preloads. Both obese and non-obese subjects increased their intake of sandwiches after being informed that they had drunk a low calorie diet drink.

Furthermore, after the drink believed to be high in calories, most subjects not only ate less (significant at the .025 level), but reported feeling "fuller".

Of additional interest was the large amount of sandwich consumption by all subjects. When asked to estimate how many sandwiches they had eaten, all subjects underestimated their actual intake. As a group, the average estimated intake was only 74.2 % of the amount eaten. As before, in this study the obese subjects were even less accurate than normals at estimating food intake. This seems to delineate the fact that when cognitive monitoring is faulty then subjects are unable to rely on physiological stimuli to control intake which, in turn results, in overeating on a short-term basis.

Wooley (1972) suggested that, because sandwiches were presented in very small units, subjects were less conscious of how much they were eating, whereas this might not have been the case if the sandwiches were available as whole sandwiches. It is almost as if they had been deceived by their own cognitions. Nevertheless, it is important to distinguish between humans' ability to regulate food intake on an internal level when most external stimuli are not present and regulator performance under different conditions; for example, it is clear in this case that cognitive manipulation overrode energy-based regulation- at least in the short-term.

Wooley's study (1972) may be criticised for lack of baseline data. Furthermore, Wooley (1972) possessed no knowledge of total daily food consumption during the four experimental days, although she did instruct subjects to consume a standard breakfast throughout the duration of the experiment. However, one positive aspect of this study is that, although it focused on short-term regulation, it was spread over four days, unlike a number of other studies (Schachter, 1967; Schachter, 1968; Schachter, Goldman and Gordan, 1968; Nisbett, 1968; Goldman, Jaffa, and Schachter, 1968; Nisbett, 1968), where testing lasted one day only.

Nisbett and Storms's results (1971) concerning the importance of cognitive controls were consistent with Wooley's findings (1972). In this study they found no evidence that obese subjects were more influenced than normal-weight subjects by a) social stimuli which encourage or discourage eating; b) false information about internal state and deprivation level; and c) cognitively supplied information defining the "correct" amount of food to consume. In the first of three experiments both obese and non-obese subjects' eating behaviour was influenced by merely watching someone else eating; both groups ate more food in the presence of a confederate when he consumed a lot of food, whereas when the confederate consumed a minimal amount of food, both groups ate very little.

In the second experiment Nisbett and Storms (1971) found that after misinforming some subjects about the nutritional value of the preloads, subsequent food intake was adjusted accordingly. Some subjects were administered a 750 kcal drink (twenty-five ounces of Nutrament), while others were given diet Pepsi as the non-caloric drink, two hours before they were offered adlibitum consumption of assorted sandwiches and soda. Both normal and overweight subjects decreased their sandwich intake after being informed that they had consumed a high calorie drink (Nutrament) as opposed to a low calorie drink (diet Pepsi), even although they may have been deprived of food for the last two hours. This finding supports the hypothesis that both groups use cognitions as a control mechanism.

In the third experiment both normal and obese subjects consumed less food when it was offered to them in small units than when it was presented to them in larger quantities. However, the obese were only able to do this after they had been given a liquid preload, whereas the contrary occurred if they were food deprived. This finding could possibly have some relation to Pliner's results (1973) where it was found that the obese were more efficient at regulating intake after a liquid preload, but not a solid preload.

Because of the series of studies which have suggested that non-obese subjects are more responsive to physiological cues than obese subjects Wooley (1971) was interested in determining whether normals are superior to the obese in regulating caloric intake, with or without the control of cognitive cues, over the long-term. Subjects (six obese, five normals) were given adlibitum consumption of a liquid foodstuff for a period of fifteen days. For the first five days six of the subjects were given a high calorie preload (comprising of Metrecal, water, sucrose and lactose in equal quantities), while the remaining subjects were administered a low calorie liquid (comprising of Metrecal, water, and artificial sweeteners). For the next ten days subjects were fed either a low calorie drink for five days and then the high calorie drink for the remaining five days, while others carried out the experiment in the reverse order, starting off with the high calorie drink. Subjects were unaware of the difference in calories in the drinks that were almost identical in taste. The first five days of the study were treated as an adjustment period. Every four minutes hunger scales were completed by subjects and their food intake was measured daily by the experimenters.

Both obese and non-obese compensated for changes in caloric density of their liquid food. However, subjects still consumed more on the high-calorie diet, (3,006 kcal per day of the high calorie food, compared to 1,920 kcal per day of the low caloric liquid). There was no significant difference in total caloric intake for either the obese or non-obese groups. These findings fail to lend any support to Schachter's short-term studies on feeding behaviour in the obese and non-obese (Schachter, 1967; Schachter, 1968; Schachter and Gross, 1968). The fact that Schachter's studies (1967; 1968) were conducted principally over a short-term period, measuring food intake for one meal only, could partly account for the different results.

The only major difference between the obese and non-obese in Wooley's study (1971) was that normal-weight subjects' ratings remained low for the entire experiment, while obese hunger ratings progressively increased throughout the study. Wooley (1971) attributed this discrepancy to a different definition of hunger by the two groups. For the obese individuals the sight and smell of non-experimental food in the hospital environment caused them to interpret their internal state as a state of hunger, whilst in reality it was not a physiological hunger, but merely an "appetitive" hunger induced by external stimuli. On the contrary, the normals felt no such hunger as they were influenced by their internal needs and they were not physiologically hungry.

This study, like most of the series of studies investigating food regulation in humans, has a very small sample of subjects, and this makes it difficult to draw any strong conclusions. Furthermore, again there was no data on any of the subjects' previous eating behaviour prior to the experiment which means that no comparison between their food intake under experimental conditions and everyday eating could be made.

In another long-term study Wooley, Wooley and Dunham (1972) examined whether obese and normal-weight subjects were capable of responding to the caloric content of a meal (which might be higher or lower than their habitual food intake), when relying solely on internal metabolic cues. Subjects were also asked to estimate the calories in meals and to rate hunger.

Seven obese and seven non-obese subjects participated in the study. A baseline period lasted five to ten days during which subjects substituted one meal with a liquid foodstuff. Then the test period lasted fourteen to twenty-one days, (it is unclear why the times varied) with the same procedure except that the caloric content of the meals varied; half of the meals were high in energy (with a mean of 516 kcal), while the other half were low in energy (a mean of 275 kcal). The caloric content of the test meals was adjusted for individual intake. Because orosensory factors were carefully

controlled, subjects were unable to distinguish between the high and low calorie meals. They were aware that they would be receiving both meal types on different occasions, but were not informed when.

The actual caloric value of the test meals had little relationship with subjects' hunger, which was more influenced by their perceived caloric content of the food. Both groups reported a greater level of hunger following ingestion of a meal apparently low in caloric content as opposed to the high calorie meal. Some ability to distinguish the high and low calorie preloads was demonstrated but performance was only 7% above chance for the obese and 9% for the non-obese. Both groups were more accurate when judging the caloric content of the meals high in calories. These data are not positive evidence for physiological regulation in humans.

The measures used in this study to examine whether subjects could distinguish between high and low calorie foods, and whether such meals influenced hunger thereafter could be criticised for being unrealistic. The activity of repeatedly guessing the caloric content of food previously ingested is not generally associated with natural eating, and it could have affected subjects' hunger ratings and caloric perceptions. Making their internal feelings of secondary importance. The time variation in this study for both the baseline and test period could also have affected results.

Wooley (1972) claimed that if normal-weight people regulate intake on the basis of internal stimuli this was perhaps strongly influenced by mediating cognitive factors. Perceptions and beliefs about the food being consumed could override the response to metabolic cues in food intake regulation over the short-term. To support her argument Wooley (1972) has referred to the study conducted by Schachter and Koch (1968) where apparently cognitive factors strongly interacted with the reported physiological regulation: When subjects were misinformed that it was near dinner time, they consequently reduced their food intake. Such behaviour would appear to be indicative

of imposed self-control, rather than of any spontaneous regulation of intake. Further evidence of cognitive effects may be found in Nisbett's study (1968) where subjects deliberately avoided increasing their consumption of ice cream when food was deprived.

Regulation of Food Intake in Restrained and Unrestrained Eaters

Although it is not my intention to examine in any depth restrained eating in this thesis, proposals similar to those for the obese have been made concerning food regulation in the restrained eater. Herman and Polivy (1975) conducted an experiment which examined the compensatory eating of restrained and unrestrained female eaters, subsequent to being fed preloads varying in caloric content. Normal eaters revealed an efficient and accurate compensatory behaviour after different preloads, both when left alone to eat and when they were being observed. In contrast, the restrained eaters were shown to compensate efficiently for the nutriment preload only in the presence of the observer. The non restrained eaters in this study appeared to be in no way sensitive or affected by the presence or absence of the experimenter. Instead they appeared on both occasions to be equally responsive to physiological signals of hunger and satiety which seemed to control their subsequent eating behaviour. Some of the findings in this study concur with previous research where significant results as regards regulation of nutrient intake in normal, unrestrained individuals, but not the obese, have been cited. These findings are of importance since they lend support to the hypothesis that restrained eaters show decreased sensitivity to internal cues. The results of this study show that restrained eaters are more sensitive to external stimuli than unrestrained eaters and such findings support to some extent Schachter's model of obesity which emphasises the importance of sensitivity to environmental stimuli rather than internal cues. Furthermore, the results from Herman and Polivy's study (1975) where the behaviour of the observer influences breakdown of self-imposed restraint in the restrained eaters is relevant when considering Nisbett's hungry-obese hypothesis (1972) where it was considered that food deprivation in some dieters and obese

individuals might increase their sensitivity to external cues (such as sight and taste of food, emotional factors and even the influence of others).

Tomarken and Kirschenbaum (1984) conducted two studies where they examined the effects of anticipated overeating of future meals on present food intake. In the first experiment subjects were offered nuts in a taste test. Then both unrestrained and restrained eaters consumed more of a milk shake when they expected a high caloric meal than when they expected a low calorie meal. Similar findings were reported in their second study when all subjects were administered ice cream, then a milk shake, although the restrained eaters consumed more of the milk-shake than the unrestrained eaters.

Summary and Conclusions

Thus some studies have found that the obese are less responsive to physiological signals than the non-obese. Hashim and Van Itallie (1965) and Campbell et al. (1971) found that when administering a liquid diet from a food dispensing machine under blind conditions normal weight subjects but not obese subjects were sensitive to caloric manipulation. Some studies have found that the obese are more influenced by external stimuli such as time of day (Schachter and Gross, 1968), visual cues (Nisbett, 1968a), palatability of food and availability of food (Nisbett, 1968b), and an analogy may be drawn between the eating behaviour of the obese and the restrained eater in terms of externality (Herman and Polivy, 1975) influenced by periods of food deprivation (Nisbett, 1972). However, other studies have not found this effect. Durrant and Royston (1982) found that obese eating is influenced by physiological, internal mechanisms. Work conducted by Wooley and colleagues showed that both the obese and non-obese are unable to regulate intake on an internal basis and both groups are more influenced by psychological factors.

These differences in results make it difficult to reach a consensus about appetite regulation in the obese versus the non-obese. Nonetheless, from the studies reviewed it would appear that the balance of the evidence is tilted towards the obese being less efficient at regulating food intake on a physiological level than normal-weight subjects. Evidence that under certain conditions the obese are more responsive to external stimuli such as sight, smell and palatability of foods than normal weight subjects seems fairly substantiated by some of the studies' findings. However, it also seems obvious that cognitive factors affect control of food intake in both normal and overweight subjects. That the obese are able to regulate intake on a physiological level to some degree also seems apparent. Finally, from this body of research there does not appear to be enough evidence to confirm Schachter's theory (1967) concerning the regulation of nutrient intake in obese and non-obese subjects.

Food and Metabolism

The inconsistencies in the results of these studies may also be attributable to metabolism. So far there is not sufficient evidence to suggest that regulatory physiology operates in the same mode when an increase or decrease in calories occur (Rogers, 1993). Although an increase in intake is expected after caloric reduction in a diet and a decrease in nutritive consumption after a diet has been calorically supplemented, it is possible that food deprivation may influence a subject's metabolic system in quite a different way from the intake of additional calories. An individual may accommodate an excess of calories by an increase in energy output rather than a reduction in food intake. In contrast, an overall decrease in metabolic output (e.g. heat and activity), instead of an increase in spontaneous food intake may accommodate caloric reduction. There is evidence in animal studies that energy expenditure plays a fundamental role in the regulatory process together with energy input (Cowgill, 1928; Adolph, 1947; Janowitz and Grossman, 1949). In human studies of semi-starvation (Keys and Brozek, 1950) it was observed that during periods of food deprivation humans regulated energy balance through changes in the metabolic system, whereby

energy storage i.e. fat reserve supply and any surplus body protein is used. A decrease in spontaneous activity was also reported which compensated for the restriction in energy intake.

The Royal College of Physicians (1983) offered a possible explanation for the common lack of efficiency of the energy regulatory mechanism in humans by proposing that the human physiology can adjust to fluctuations in food consumption of plus or minus 15% by changes in energy expenditure, rather than the expected changes in food intake. This could possibly obviate the need for an efficient and accurate regulatory mechanism.

Individual variation should also be considered, in that individuals may metabolise food differently. Metabolic studies have shown that in people with weight problems, energy intake does not necessarily have to correspond with body weight. Some overweight people actually ingest less caloric intake than someone who is lean (Rose and Williams, 1961). Yet, it is well documented that one of the main factors causing obesity is overeating (Prentice et al., 1989). However, demonstrating that the obese consume more food than normal weight subjects is no easy task (Rogers, 1993), particularly in the laboratory. Furthermore, it has been shown several times that obese subjects under report their food intake (more than normal subjects) and this may affect interpretation of results. It is expected that at some point a surplus of calories in overweight subjects has occurred (Rodin, 1975) so that once the excess body weight develops subjects can decrease intake to their set level but will maintain this new weight.

These results are important when considering the mechanisms underlying control of food consumption. In some of the studies reviewed above inadequate compensation of caloric intake has been attributable, at least in part, to the process mentioned above, where modifications in activity or in metabolic output occurred so as to maintain near

constant caloric balance. Yet, few studies have monitored metabolic rate. Furthermore, laboratory studies may inadvertently decrease activity levels.

Evidence that a significant relationship exists between energy expenditure and caloric intake is inconclusive (Durnin, 1961) although some research with humans has postulated that the processes of regulation of energy balance may be controlled by both energy intake and energy output (Garrow, 1974, 1978). This theory has been used as a possible mechanism underlying some of the etiologic factors in obesity where it has been hypothesised that obesity is induced by excessive energy intake and a malfunctioning energy output system (so that metabolic output and activity is less than what it should be).

An Overlap between External and Internal Factors

Schachter's internal-external hypothesis (1967) becomes less credible when considering the interaction between internal and external factors as discussed in chapter 2. Non-physiological factors such as the process of conditioning, orosensory factors, cognitive factors appear to play a significant role in eating regulation in normal and obese individuals (Sjostrom et al., 1980; Sahakian, 1980; Moore and Schenkenberg, 1974). They may interact with or elicit internal mechanisms. For instance, external stimuli may induce physiological conditions such as hunger, salivation, a rise in gastric secretion, insulin release). Finally, obese eating appears to be influenced by physiological, internal mechanisms.

There are data to support the contention that the mechanism which regulates food intake in humans is not accurate, and some studies have found this applies especially to obese individuals. More specifically, the reported accuracy of compensation amongst the obese and non-obese has varied widely, perhaps again due to variations in method.

Methodological Issues

Because studies have often compared obese and non-obese individuals, subjects have been divided into groups by weight. However, it has since been shown that obesity alone does not suffice to determine degree of external or internal responsiveness in a person (Nisbett, 1972; Price and Grinker, 1973, Rodin, 1981). Other studies have found that people with high and low externality exist in every weight category (Nisbett and Storms, 1975; Rodin and Slochower, 1976; Tom and Rucker, 1975). This evidence casts doubt over Schachter's theory (1967).

Another source of variation is method of food administration. Some studies have used a liquid formula dispensed from a feeding machine over the long-term and have found that the obese are unable to regulate energy intake efficiently, when compared to lean subjects (Campbell et al., 1971; Hashim and Van Itallie, 1965). Others have found that neither the obese nor the non-obese are able to control energy intake efficiently when administered liquid foodstuff (Wooley, 1971, Wooley, Wooley and Dunham, 1972, Durrant et al., 1982). Porikos (1981) found that neither lean nor obese subjects regulated food intake accurately when administered a solid palatable diet.

Thus, the method of food presentation immediately raises the question of the validity of their findings. Being fed a fairly unpalatable or at least monotonous liquid diet under experimental conditions is so far from the reality of everyday eating behaviour that it casts doubt over whether or not such results can be interpreted as a true reflection of human feeding. Using feeding machines to dispense the liquid to subjects either orally or intragastrically increases the artificiality of the eating process and may even make the subjects feel quite uneasy, especially obese individuals. Furthermore, the validity of administering liquid formulas to individuals has also been questioned, mainly because it may confuse eating behaviour with drinking which are two quite different phenomena. However, because psychological factors can influence eating behaviour, caution must also be exercised when administering solid, palatable food.

Even drinking from hand-held containers provides more visual and cognitive influence than being fed from a feeding machine.

It would thus seem that before any assessment can be made on differential eating mechanisms in the obese and non-obese at least three methodological issues should be attended to. First, a consistent and adequate method of food administration should be sought which is suitable for both lean and overweight subjects, in that prior to any dietary manipulation both groups will ingest enough food to maintain their normal level of weight. Second, baseline diets should be used prior to caloric manipulation. Third, cognitive factors should be attended to which means that taste, texture, appearance of food should be indiscriminable from placebo food or calorically manipulated food, and this is particularly important when using solid palatable foods. It has already been demonstrated that the caloric content of solid palatable foods can be manipulated without affecting the palatability of the foodstuff (Porikos, 1981; Porikos et al., 1982).

Conclusion

Several of the studies reviewed in this chapter and in chapter 2 have shown that normal-weight subjects do not rely solely on physiological mechanisms. Cognitive factors, socio-cultural factors and conditioning mechanisms all account for food regulation in both groups. It might be that obese individuals have learnt to consume more than what is culturally prescribed as "normal", while normal-weight subjects may have a better understanding of what to eat and how much. It is also possible that the obese, like normals, are able to regulate their intake using physiological cues, but some choose on occasions to rely more on external food-related cues.

Chapter 4- Carbohydrates and Subsequent Food Intake

In the literature it has already been suggested that the macronutrient composition of a person's diet may be relevant as regards subsequent food consumption and control. So far very little attention has been paid to the specific nutrient properties of food and how it affects an individual's experience of hunger, food intake and satiety. It has been previously cited that the nutrient components of food and palatability of food may influence or in part determine the desire to continue eating. Blundell and Hill (1985) in an experiment showed that factors such as macronutrient composition and the palatability of food exert influence over subsequent food intake. In chapter 2 it was mentioned that certain types of food may trigger some people to eat again soon afterwards. Such foods have been reported to be high in the nutrients carbohydrate and fat, but low in protein. Geiselman and Novin (1982) suggested that carbohydrate intake increases appetite overall as well as specifically increasing hunger for carbohydrates.

Such findings suggest that carbohydrate has a different effect on human eating behaviour and appetite control than fat and protein. It has also been hypothesised that high-carbohydrate food has a priming effect (Hodgson and Greene, 1980). Findings of this kind support the theory that nutrients vary in their degree of importance in the control of food intake and that the nutritional composition of the diet is important where appetite, hunger and satiety are concerned.

In contrast, as reviewed in chapter 2 other past research has been unable to show any indication that carbohydrate intake increases appetite more than protein or fat intake (Geliebter, 1979; Grinker et al., 1971; Rosen et al., 1982; 1985; Sunkin and Garrow, 1982; De Castro, 1987). Furthermore, Booth and colleagues have revealed that carbohydrate intake can in effect decrease food intake. Booth et al. (1970a) affirm that

the satiating properties of glucose may be effective from about fifteen minutes subsequent to food intake and may last just under three hours.

Thus, there are plausible hypotheses that carbohydrate ingestion increases subsequent carbohydrate ingestion and/or energy intake. Nevertheless, there are equally plausible hypotheses predicting the opposite effects and some data to support both positions. It is likely that the inconsistencies in this literature are due to some of the methodological problems discussed in chapter 2.

It was considered useful to study the effects of carbohydrate ingestion on everyday eating behaviour, outside the laboratory, while controlling the administration of carbohydrate so that subjects were blind to what they had ingested. The overall aim of the study which extends the pilot work is to examine the relationship between sugar ingestion and hunger over a two hour period. One of the principal objectives is to ascertain the extent to which sugar loading could reinforce the eating of foods or food combinations with a high glycemic index.

Another aim of the study was to examine whether sugar ingestion has effects on behaviour mediated by physiological effects which are independent of psychological factors (i.e. the sensory qualities related to eating food, subject and/or cultural expectations). Therefore, the control of cognitive cues is considered of major importance (see Hashim and Van Itallie, 1965; Jordan, 1969; Campbell et al., 1971). It was partly for this reason that it was decided to administer sucrose in a dilute liquid solution which enables the control of visual, and orosensory factors by using nose clips and anaesthetic lozenges. In order to eliminate sensory cues, such as taste and texture, subjects were asked to suck a benzocaine anaesthetic lozenge prior to drinking (after Millar, Hammersley and Finnigan, 1992). Nose clips were also worn while drinking, to mask the aroma of the drinks.

Although such elaborate precautions to mask taste and smell may appear somewhat extreme, it was felt important to take every precaution to minimise sensory qualities since the focus was on physiologically mediated effects. In the review chapters it has been shown that eating behaviour in humans may be strongly influenced by cognitive components such as the sight, smell and taste of food as well as subjects' thoughts and beliefs about certain foods (see Wooley, 1972 for a discussion). Furthermore, although the sucrose and saccharin solutions were equisweet it was still found necessary to eliminate taste. The taste properties of sugar rather than the actual nutrient per se has been known to affect satiety (Cabanac and Duclaux, 1971), subsequent food intake and selection (Rolls et al., 1982) and mood (Blass, 1991). Several studies have claimed that the ingestion of sugar increases hunger. However, it is not clear whether such an effect is due to the biological effects of the food ingested or due to the hedonic properties of the test food. Thus, this procedure enables sugar to be administered blind, thereby controlling for expectancy effects to some extent. Using a sucrose preload in liquid form also meant it was possible to incorporate a placebo condition into the experiment which could not be discriminated from the test drink.

The problem of sample composition and selection has already been discussed in chapter 2 where it was mentioned that many studies in the past have used students, university or hospital staff, or obese patients. It has also been found in previous studies that most subjects have been under thirty. Thus, it was felt that using a wider sample which ranged in age (20-55 yrs) was necessary in order to increase the generality of the findings. Including enough subjects (n=52) to compare the effects of sucrose on males and females was also considered a strength. Most subjects were recruited from places of work which made the sample more varied.

In an effort to again address some of the problems from past research, such as the artificiality of laboratory studies it was considered helpful to study the behaviour of non-obese adults at their places of work. Such a procedure was used in the experiment

following the pilot study. Although the study was designed to examine people at places of work in an effort to tap into everyday eating behaviour the obvious disadvantages of studying eating behaviour outside the laboratory were recognised. The most common of which include a loss of control over food presentation and of precision in the measurement of what is eaten. However, these losses are offset in this case by the possible artefacts induced by laboratory studies of eating and food diaries from trained subjects can provide relatively accurate information about intake (Klesges et al., 1988; Mahalko et al., 1985).

It was decided to use a prospective diet diary as a method of monitoring food intake. Day-to-day subjects were asked to write down everything they had to eat or drink. Each day was divided into one hour time slots and subjects were asked to record all food and liquid intake, the amount consumed and the time of day the food was consumed. However, instead of weighing each item subjects were asked to describe the quantities eaten in household measures (e.g. a glass of milk). In order that subjects could make a more accurate estimate of the quantities eaten pictures of various common foods with portion sizes marked A, B, C were attached to the diary booklet for comparison purposes (provided by Annie Anderson, a dietician at the department of Human Nutrition). Studies suggest that the use of food models improves the validity of reported intakes (Bone, 1992). Although this method may be less accurate than weighing, it has the advantage of minimal disturbance of everyday eating behaviour. Moreover, since it is less demanding, it was expected that there would be a higher response rate from those agreeing to participate in the experiment.

Although only ten to fifteen minutes training was given due to time constraints on the part of the volunteers, detailed example sheets of how the diary was expected to be filled in were provided together with the food models mentioned above. Subjects were also informed that should they encounter any difficulty during the 7 day period prior to testing they were to contact the experimenter for advice.

The main purpose of the diary was to acquire information on food intake after preloading (which was achieved by issuing a 24 h diary after the experiment). However, the 7 day diary was used to extract certain information. It also proved to be useful for comparative reasons since it demonstrated that for most of the subjects the 24 hour recall was a realistic representation of their eating patterns from the week before. For example, when calculating the number of subjects who had consumed calorific beverages prior to eating on the day of testing, reference was made to the 7 day diary where habitual consumption of calorific beverages could be examined in terms of: (i) whether or not subjects normally took milk and/or sugar in tea or coffee and (ii) if they had consumed a calorific beverage without solid food on any weekday morning of the previous week. These data provided information on whether beverage consumption prior to eating on the test day was a function of habit or mediated by the sucrose or saccharin preloads.

From the 7 day diary the mean time of the meals on weekdays which occurred after 1000 hrs, but before 1800 hrs (to exclude days when subjects skipped lunch) was also calculated in experiments 1 to 3. Then change in mealtime after the preload was calculated by subtracting time of next meal from mean meal time. On the morning of the experiment subjects' 7 day diaries were collated prior to participation in the study. Whilst they were going through the experimental procedure their diaries were checked for accuracy by the experimenter. If there were any problems or any questions concerning the diaries they were asked then and there, and subjects made changes if necessary. This also ensured that the 24 hour diary would be more accurate and valid.

Although it was recognised that between subject designs may not be as sensitive to individual variation as within subject designs, this disadvantage was offset in this case by the potential problems of within subject designs, namely order effects, and problems of learning: when people are exposed repeatedly to various conditions they

are more likely to learn what is expected of them or correctly guess the hypothesis being tested and adjust their behaviour accordingly (see Millar et al. (1983) for a review on the weaknesses of within-subject design and chapter 1 for more detail on experimental designs). Here, the main phenomena of interest, namely the effects of sucrose on eating behaviour and mood, have not been unequivocally demonstrated, so it was felt premature to seek subtle within-subjects variations. Thus, having carefully considered the strengths and weaknesses of both designs it was decided to use a between-subjects design which takes into account order and practice effects.

The decision to examine the effects of carbohydrates on subsequent eating and mood in the morning was partly due to the fact that an overnight fast condition could be included in the study, thereby enabling the control of prior intake to some extent. This was considered important since it has already been established that prior nutrient intake can influence both neuroendocrine functions (Ishizuka et al., 1983, Slag et al., 1981) and the metabolism of subsequent food intake (Jenkins et al., 1982) and possibly mood. To date several studies have overlooked this important factor and failed to control the prior nutritional state of their subjects either by not asking them to fast or not including a pre-meal baseline whereby food intake is standardised or controlled for in some other way (e.g. self-recording of prior intake through diaries).

Saccharin as opposed to aspartame was used in all the experiments as a placebo on the basis that the placebo aspartame in sugar studies may increase brain tyrosine (Young, 1991), whereas there have been no such reports for saccharin. Furthermore, the saccharin solution contained minimal amount of calories which was considered important for the placebo drink; 56 ml of the saccharin preparation contained 10 kcal; 4.34 g (see overpage for the exact contents of the saccharin tablets).

As part of the measures used in this series of experiments the Profile of Mood states (POMS) was used as the sole device to assess subjective mood state over time for two

reasons: First, the Profile of Mood states (POMS) has been used widely to study the effects of food on mood (e.g. Spring et al., 1983, 1986, 1989; Lieberman et al., 1982/3; Pivonka and Grunewald, 1990, Christensen et al., 1985), which implies that it is sensitive to mood changes caused by eating. Second, although other studies have used a variety of devices to measure mood, namely the Stanford Sleepiness Scale (SSS); the Visual Analogue Mood Scale (VAMS) as well as the Profile of Mood States (POMS) (Spring et al., 1983, 1986; Lieberman et al., 1982; Pivonka and Grunewald., 1990) the effects on mood subsequent to food intake have not been systematic across the different measures of mood. For example, Spring et al. (1986) found sleepiness was affected using POMS but not VAMS or SSS. It has been revealed that this is partly attributable to the selective reporting of isolated "significant" findings from numerous statistical comparisons.

It was decided to conduct a series of short-term studies as opposed to long-term for two reasons: First, more controversy revolves around short-term studies than long-term ones; differences in methodology are more apparent in short-term experiments with poor control of psychological factors being one of the major methodological weaknesses. Second, at this initial stage of experimentation it was considered simpler to design a series of short-term studies well than to design long-term studies well, as there are many more complications attached to conducting a long-term study. Amongst the most common are problems in recruiting enough subjects to participate in a long-term study so as to have an acceptable sample size. A higher drop-out rate of volunteers is another problem as well as the increased risk that subjects guess the purpose of the study.

Lastly, it should perhaps be mentioned that although ethical permission was not sought for any of the experimental work reported in this thesis all subjects gave their informed consent before taking part in any of the experiments.

Pilot Study-The Effects of Sucrose on Subsequent Food Intake in Normal-Weight Female Subjects

This pilot study attempted to experimentally test the effects of a sucrose preload on the subsequent food intake of male and female subjects over a two-hour period. This was accomplished by giving subjects a preload of sucrose or saccharin administered under blind conditions, and then measuring their subsequent food intake by self-monitoring diaries. The central question of the study was whether the sucrose preload produced greater hunger with the passage of time compared to the saccharin preload or delayed hunger.

Composition of Preloads

One of the central points to be demonstrated in the pilot study was that the taste of the caloric and non caloric drinks should be indiscriminable under experimental conditions. The initial stage of the experiment involved making up mixtures that would be suitable for the study. Several mixtures were given preliminary testing with two members of academic staff who were requested to guess whether the drink contained saccharin or sucrose. They were allowed to change their guesses after tasting both mixtures. The composition of the drinks was considered as being appropriate when the members of staff were unable to distinguish between the two mixtures. The same mixtures used in the pilot study were used in experiment 1, 2 and 3. The nutrient dosage chosen was 40 g of sucrose, as preliminary testing revealed that this amount could not be discriminated from the placebo drink containing saccharin and this quantity was similar to that used by Pivonka and Grunewald, (1990).

Saccharin tablets, manufactured by Boots Company PLC. were used for the placebo drink. The tablets contained sodium bicarbonate, artificial sweetener (saccharin), sodium carbonate, tartaric acid, anticaking agents (stearic acid, magnesium stearate) acacia (tableting aid). It was calculated that 56 ml of the saccharin solution which contained 10 kcal; 4.34 g was equivalent in sweetness to 40 g of sucrose (160 kcal).

It was decided to use diabetic whole orange drink manufactured by Boots Company PLC to make up the sucrose and saccharin preloads principally because it contained a minimal amount of calories and sugar. The typical values for undiluted drink per 100 ml are as follows: energy-5.2 kcal, protein-0.1 g, fat-trace, other carbohydrate-1.2 g, total carbohydrate-1.2 g. The ingredients in the diabetic orange drink were water, orange (10%) citric acid, acidity regulator (E331) flavouring vitamin C, colour (E160a), stabilisers (E412, E405), artificial sweeteners (aspartame containing phenylalanine, sodium saccharin preservatives (E223, E221).

Design

Subjects were randomly assigned to two groups: ten female subjects in the sucrose condition and ten females in the saccharin condition.

Subjects

Subjects were recruited by advertising in Glasgow and Paisley University and were either students or university staff. Volunteers were requested to complete the Eating Disorder Inventory (EDI), a standardised self-report measure which consists of eight subscales related to specific behavioural and psychological dimensions important in Anorexia Nervosa and Bulimia (Garner, 1990). Only subjects falling within the normal range of all eight scales and within 20% of weight norms for their age, gender and height were included in the study. The EDI questionnaire served as a useful indicator for detecting abnormal eating behaviour or health problems that were diet-related such as diabetes. Twenty females took part. The females mean age was 32.0 years (SD=8.59), their mean height was 167.8 cm (SD=9.30) and their mean weight was 62.4 kg (SD=7.93).

Procedure

Subjects were informed that the purpose of the study was to examine the effects of various nutrients on subsequent food intake but were not told that some of them would be administered a placebo drink devoid of any nutrients, in this case sugar. Volunteers

were tested in the university laboratory. At the initial intake interview subjects' height and weight were recorded by the experimenter and they filled out the EDI questionnaire. They then completed a Profile of Mood States form, (POMS) which has six scales measuring composed/anxious, elated/depressed, agreeable/hostile, energetic/tired, confident/unsure and clearheaded/confused. It has been used extensively in studies of the effects of food on mood (e.g. Keith et al., 1990; Spring et al., 1983, 1986, 1989; Pivonka and Grunewald, 1990).

Subjects were then issued a seven-day food diary to complete prospectively for the seven days prior to testing. Each day was divided into one hour time slots. They were given brief (ten to fifteen minutes) training on the use of such diaries and requested to record all solid and liquid foodstuffs, the amount consumed and the time of day the food was eaten. Subjects were instructed to indicate portion sizes and were given pictures of sample portion sizes to use for comparison. All subjects were instructed to fast without breakfast on water only on the morning of the experiment.

Seven days later, on the morning of the experiment volunteers again appeared for testing as soon after 09.00h as possible. Subjects who had not complied with fasting instructions, or who had failed to complete the diaries, were rescheduled or discarded. All subjects were again asked to rate their mood using the POMS.

They were then administered a pint of liquid comprising 110 ml of diabetic dilute-to-taste orange squash and water which was either preloaded with 40 g of sucrose (160 kcal) or 56 ml of saccharin solution (4.34 g; 10kcal). In order to eliminate sensory cues such as taste, and texture, subjects were asked to suck a benzocaine anaesthetic lozenge (Tyrozets, containing 1 mg of Tyrothricin and 5 mg of Benzocaine) prior to drinking which produced a mild anaesthesia of the mouth (after Millar et al., 1992). Subjects also wore nose clips while consuming the drink to mask the aroma of the drink. Subjects were given fifteen minutes to drink their beverage. There were no

reports of nausea or other symptoms by the subjects during or subsequent to the ingestion of the preloads.

Immediately after they had finished drinking, subjects were given another POMS to complete. Subjects were then issued a final food diary which was to be filled in over the next twenty-four hours and returned to the experimenter the following day. Subjects were then free to leave the laboratory setting (see Tables 4.1 and 4.2 which outline the exact procedure of the pilot study). Once their final diaries had been received, subjects were debriefed in writing about the nature and purpose of the experiment. There was no evidence of communication between subjects with respect to the hypothesis being tested. In experiment 1, 2 and 3 this was even more unlikely since subjects were being tested in groups at different places of work throughout Glasgow.

Table 4.1 Week one-Initial interview

Step 1	Step 2	Step 3	Step 4
Weight/Height Measurements taken	Subjects complete EDI	Subjects complete Baseline POMS	Subjects are given prospective 7-Day diary & 15 mins training

Table 4.2 Week Two-Approximate Time of all experimental events

9 00-9.15h	9.15-9.30h	9.30-9.45h	9.45-10.00
1) Collate 7 day diary 2)Administer Tyrozet 3) Complete POMS	Administer Preload (Subjects given 15 Minutes to consume)	Complete POMS immediately after drink	Administer 24hr diary to complete in natural setting **N.B.** subjects complete additional POMS in experiments 1,2,3.

Results

From the food diaries subsequent to the experimental drink were extracted (a) time of next solid food, to the nearest hour. No subject ingested other calorific liquids prior to their next solid food. The time of administration (09.00h-10.00h) did not differ between groups (units of meausurement of time are in hour chunks). Paired t-tests indicated that their pretest POMS scores did not differ significantly from their mood the previous week. Post-test mood scores were calculated as changes from pretest baseline. All mood scales were unaffected by sucrose ingestion (all $p(t)>0.05$) except for the elated scale (t (18 df)=1.98, $p<0.07$) energy scale (t (18 df)= 1.78, $p<0.1$) and agreeableness scale (t (18 df)= 2.15, $p<0.05$). POMS were calculated by using a bipolar form with a handscoring key for each scale which is placed over the completed POMS form. Each item is scored 0,1,2, or 3. Since the scales are bipolar, each scale score is the sum of positive item scores *minus* the sum of negative item scores. The scoring sequence is as follows:

1) add the scores on the 6 positive items: *Sp*

2) add the scores on the 6 negative items: *Sn*

3) subtract *Sn* from *Sp*, (*Sp*-*Sn*). Add the constant 18 to get the total: $St = Sp-Sn + 18$.

The group who received the sucrose preload ate just before noon (mean time 11.50 am (SD=0.316 hrs)), whilst the saccharin group ate earlier (mean time 10.06 am (1.48). There was markedly more variation in time of eating after the saccharin preload and the difference between the two groups was significant ($t(9.82)=3.56, p<0.005$).

Discussion

The experimental procedure developed in the pilot study whereby sucrose was administered in a dilute liquid solution with visual, taste and smell cues masked and controlled proved to be effective. Under these conditions the results of the pilot study revealed that sucrose delays subsequent eating behaviour and may influence mood

state. It was concluded from this study that because the applied methodology was effective in testing the effects of carbohydrates on eating behaviour under blind conditions that the same experimental design would be used in subsequent experiments. Furthermore, it is recognised that the pilot study lacks detail (e.g., in terms of analysis of subsequent intake) when compared to the studies which follow. This was because the main purpose of the pilot work was to test the method. In the experiments to come some information from the diaries is examined in more detail.

Experiment 1- The Effects of Sucrose on Everyday Eating in Normal-Weight Men and Women

Experiment 1 extended the pilot study. The methodology used in this study was the same as in the pilot study with a few differences: this time there was a larger sample size, fifty-two subjects both male and female who were non-students, recruited from places of work. Sex differences were also examined. Testing took place in groups in places of work as well as in the university laboratory. As well as examining the time of the next solid food after the preload, the energy and carbohydrate content of the food were examined.

Design

Subjects were randomly assigned to two groups: twenty-six subjects (male and female) in the sucrose condition and twenty-six subjects in the saccharin condition.

Subjects

Subjects were recruited by advertising in places of employment around Glasgow. Fifty-two volunteers, twenty-eight females and twenty-four males, took part. The females mean age was 32.0 years (SD=8.59), their mean height was 167.8 cm (SD=9.30) and their mean weight was 62.4 kg (SD=7.93). The males mean age was 29.54 years (SD=9.74), their mean height was 173.5 cm (SD=22.19) and their mean weight was 76.9 kg (SD=32.36). The mean BMI for the females was 22.2 (2.7SD). The mean BMI for the male group was 21.5 (4.6SD).

Procedure

The procedure was similar to that of the pilot study with a few exceptions. This time volunteers were either tested in groups at their place of work, or in the university. The analysis of food diaries was more extensive than that of the pilot study. From the food diaries were extracted (a) time of next meal, to the nearest hour. A meal was defined as

all food and drink consumed within the first hour period following the preload during which solid food was consumed. From the 7 day diary the mean time of meals on weekdays which took place after 1000 hrs, but before 1800 hrs (to exclude days when subjects skipped lunch, only applicable to one male), was also calculated. Then change in mealtime was calculated by subtracting time of next meal from mean meal time. (b) Calories of next meal, calculated assuming standard portion sizes from Davies and Dickerson (1991). (c) Carbohydrate content of next meal (carbohydrate intake is defined as simple and complex carbohydrate excluding dietary fibre which had a separate column. (d) Calorific content of any milk and sugar in tea or coffee taken prior to the next meal. All other liquids ingested prior to the next meal contained less than 5 kcal per subject (e.g. black coffee, single can of mineral water). (e) Habitual consumption of calorific beverages; (i) whether or not subjects took milk and/or sugar in tea or coffee and (ii) if they had consumed a calorific beverage without solid food on any weekday morning of the previous week.

Although subjects had fasted on the morning of the test and some complained of caffeine deprivation, paired t-tests indicated that their pretest POMS scores did not differ significantly from their mood the previous week (all $p > 0.05$). Furthermore, baseline scores on all scales were not sufficiently high to indicate a mood state (e.g. a dsyphoria or severe fatigue) that might respond to nutritional intervention. Nor were there any differences in pre-drink ratings between the saccharin and sucrose group. Post-test mood scores were calculated as changes from pretest baseline scores. If subjects had consumed calorific beverages prior to eating, the energy and carbohydrate content of these were added on to the values for their next meal.

Each of the six POMS scales were subject to a 2 x 2 analysis of variance (ANOVA) with preload content (sucrose or placebo) and gender (male or female) as independent variables. Change in meal time, energy content and carbohydrate content of next meal were subject to similar analyses, with the addition of weight as a covariate. However,

males who had received sucrose tended to consume calorific beverages prior to this next meal and this factor necessitated the supplementary analysis of meal times separately by gender. For females, the ANOVA used preload as an independent variable and weight as a covariate. For males, a three-level independent variable was created: saccharin preload, sucrose preload and sucrose preload plus calorific beverage, and weight was included as a covariate.

Results

Full Analyses

Changes in mood scales were unaffected by the sucrose versus the saccharin preload; post-eating changes in mood scales were calculated as changes from baseline mood scores, all, t(51df), n.s.: clearheaded: t=0.61, energetic: t=1.40, confident: t=1.36, elated: t=0.14, agreeable: t=1.83, composed: t=0.09).

Only fourteen out of fifty-two subjects consumed liquids prior to their next meal. Of these, twelve consumed calorific beverages. The majority of them (eight subjects out of twelve) were males who had received a sucrose preload (Table 4.3). The mean energy intake in beverages (excluding subjects with zero intake) was 35 kcal (SD=17), the maximum was 56 kcal. Significantly more sucrose preload subjects than saccharin preload subjects then consumed a calorific beverage prior to eating solid food ($X^2(1)=5.2$, $p<0.05$). This effect was not attributable to prior differences in groups' beverage choices; eleven out of twenty-eight saccharin subjects and seven out of twenty-four sucrose subjects had consumed a calorific beverage without solid food on at least one weekday morning in the previous week ($X^2(1)=0.6$, n.s.). Only three subjects in each group never took milk or sugar in their tea or coffee (Fisher's exact probability=0.33).

Weight affected none of the measures (All F(1,43), $p>0.05$) and is not discussed further. All groups tended to eat their next meal earlier than normal, presumably to

make up for missing breakfast (Fig. 4.1). In comparison to saccharin, the sucrose preload marginally delayed time of next meal ($F(1,43)=3.9$, $P=0.056$). Consuming a calorific beverage also delayed the time of next meal ($F(1,43)=6.6$, $p<0.05$). Gender had no effect ($F(1,43)=0.1$, n.s.) on time of next meal.

Although intake was not meausured in the pilot study it was calculated in this experiment and the experiments to follow. The energy content of the next solid food (including any prior beverages) was increased by prior beverage ($F(1,43)=12.4$, $p<0.001$) and this effect was larger than could be attributed to the energy content of the beverage (Fig. 4.2). Men had higher energy intake than women ($F(1,43)=11.8$, $p<0.001$). Those who had received a sucrose preload tended to have a lower energy intake, although this effect was not significant ($F(1,43)=3.5$, $p=0.068$). There was also a gender by preload interaction ($F(1,43)=12.1$, $p<0.001$) and a gender by prior beverage interaction ($F(1,43)=7.3$, $p<0.05$), but these interactions are distorted by the uneven cell sizes for prior beverage (see Table 4.3). Women's energy intake was unaffected by the preload, whereas the few men (n=5) who received sucrose and did not drink a beverage tended to have a reduced energy intake.

Men ate more carbohydrates than women (Fig. 4.3) ($F(1,43)=14.1$, $p<0.001$), but the preload did not affect carbohydrate intake ($F(1,43)=2.8$, n.s.) nor did prior beverage ($F(1,43)=3.6$). There were, however, significant interactions between gender and preload ($F(1,43)=4.9$, $p<0.05$) as well as gender and prior beverage ($F(1,43)=6.5$, $p<0.05$); again though these interactions are distorted. The trend was for sucrose-preloaded males to eat less carbohydrate.

Female Meal Times

Change in meal times in women was analysed with preload as an independent variable and weight as a covariate. There were no effects of preload ($F(1,25)=0.01$), or of weight ($F(1,25)=2.0$).

Female subjects tended to choose low energy (mean 281 kcal), low-carbohydrate foods (mean 28 g), whether they had received sucrose or not and whatever their weight. These are low proportions of daily energy requirements, which suggest considerable dietary restraint being exercised by these normal females, despite missing breakfast.

Male Meal Times

Change in meal times in men was analysed with preload+beverage as an independent variable and weight as a covariate. There was a marginal effect of preload+beverage ($F(2,20)=3.4$, $P=0.053$) and no effect of weight ($F(1,20)=3.3$, n.s.). The three levels of preload+beverage were compared by post-hoc t-tests. Subjects who drank the saccharin preload ate significantly earlier than those who drank the sucrose preload and a calorific beverage ($t(16df)=3.08$, $p<0.01$), but no earlier than those who drank the sucrose preload but no calorific beverage ($t(14df)=0.53$, n.s.). The two groups who received the sucrose preload did not differ significantly ($t(12df)=2.0$, $p=0.073$), but this was probably due to the small number in the sucrose + no beverage group.

Compared to females, males tended to choose high energy (mean 601 kcal), high-carbohydrate foods (mean 69 g).

Table 4.3:Consumption of Calorific Beverages Prior to Solid Food

	No Calories in Beverages	Calories in Beverages	Proportion
Male/ Sucrose Drink	5	8	0.61
Female/ Sucrose Drink	10	1	0.09
Male/ Saccharin Drink	10	1	0.09
Female/ Saccharin Drink	15	2	0.11

Discussion

In this study the ingestion of sucrose under blind conditions led to effects that differed between men and women. About 60% of male subjects responded to the sucrose preload by soon drinking tea or coffee containing milk and/or sugar. After this combination with the preload, they then delayed their next meal compared to subjects who had ingested saccharin, or, rather, those who consumed saccharin tended to eat earlier than they would normally have done. Sucrose did not "prime" the consumption of a calorific beverage, but rather obviated the need for more solid food, as eaten by the saccharin-preloaded males. The trends in the data suggest also that sucrose primed males either drank a beverage or ate a snack soon afterwards. Few female subjects consumed calorific beverages without solid food. For women, the preload did not significantly affect the timing of the next meal, which was more like an early lunch. However, it should be noted that the labels "snack" and "lunch" are somewhat arbitrary.

There was no evidence that the preload affected the energy or carbohydrate content of subsequent food. Women ate less energy dense, lower carbohydrate meals than men. This finding is of interest since it is recognised that some high carbohydrate foods are less energy dense than low carbohydrate foods. However, this may depend very much on the type of carbohydrate ingested (i.e. starch or fibre versus sugar) and the proportion of fat or protein ingested in combination with carbohydrate. Men tended to have a drink or solid food soon after the preload. It seems that the effects of the preload interacted with normal dietary habits; a phenomenon which would not be apparent in studies where food presentation is controlled. Such habits may also explain why a calorific beverage seemingly increased subsequent energy intake: subjects who took milk and/or sugar in their tea or coffee also tended to eat a more energy-dense lunch.

The novel features of this study were the blind administration of sucrose combined with the study of everyday eating, rather than of food choice in the laboratory; also older, non-obese subjects were studied. These findings suggest the possibility that when previous studies have found that carbohydrates *increase* appetite and/or eating, this may have been due to the somewhat artificial nature of the experimental conditions used. In particular, when subjects are aware of what they are eating they may behave according to the "well-known rule" (Poulton, 1989), in this case that carbohydrates make one hungry. The palatability of some high-carbohydrate foods may also increase appetite. Further, the free availability of food in the laboratory may encourage eating; in everyday life people will tend to eat food which is offered to them.

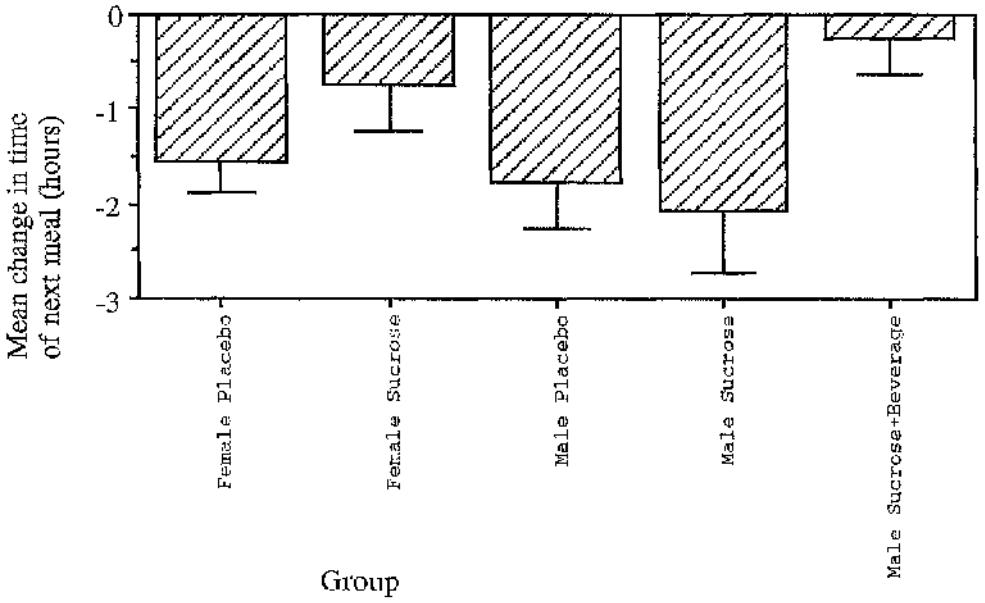
Various studies (Geiselman and Novin, 1982a; 1982b; Novin et al., 1973; Spitzer and Rodin, 1987; Rodin et al., 1988) have suggested that carbohydrates may increase appetite or eating behaviour. The findings reported here suggest that among normal adults the metabolism of carbohydrate, (in the form of sucrose) does not itself increase appetite or eating, but rather when administered blind, sucrose delays eating, but that meal content is as much related to habit (i.e. habitual meal times, time of day, place, normal meal content) as to hunger. The importance of time of day is well recognised in diet-behaviour studies (Schachter and Gross, 1968). It may be that learned sociocultural factors such as times of day set aside for meals and specific location impact eating in a natural setting more than in the laboratory. Various other external cues, namely the sight and smell of food and the influence of others are also considered influential factors on eating behaviour in our everyday environment (Schachter and Gross, 1968; Nisbett, 1986a; Weingarten, 1985). Thus, it is probable that the effects of preloads on eating depend upon the timing of preload and subsequent food (Booth et al., 1970a, b). In particular, studies which present food at a set time after a preload will be insensitive to delay effects and it may be easier to delay eating before habitual meal times, which was the effect found here, rather than to postpone habitual meal times.

Habits are also a function of the individual. Here, men and women differed in their carbohydrate and energy intake and men were more likely to eat and drink early to make up for having missed breakfast. This gender difference could be physiological, or it could be a gender role bias in that women normally try to restrain eating while men normally try to keep up their intake. It is likely that other individual differences will also affect eating behaviour and caution should be exercised in generalising findings from particular groups such as male students or laboratory staff.

Mood immediately after sucrose ingestion was unaffected. This may have been due to completion of the POMS immediately after drinking, for previous studies have not measured mood until thirty minutes after eating or later. Pivonka and Grunewald (1990) found mood elevated at thirty minutes and other studies have found mood changes still later (Lieberman et al., 1986; Spring et al., 1983, 1986, 1989).

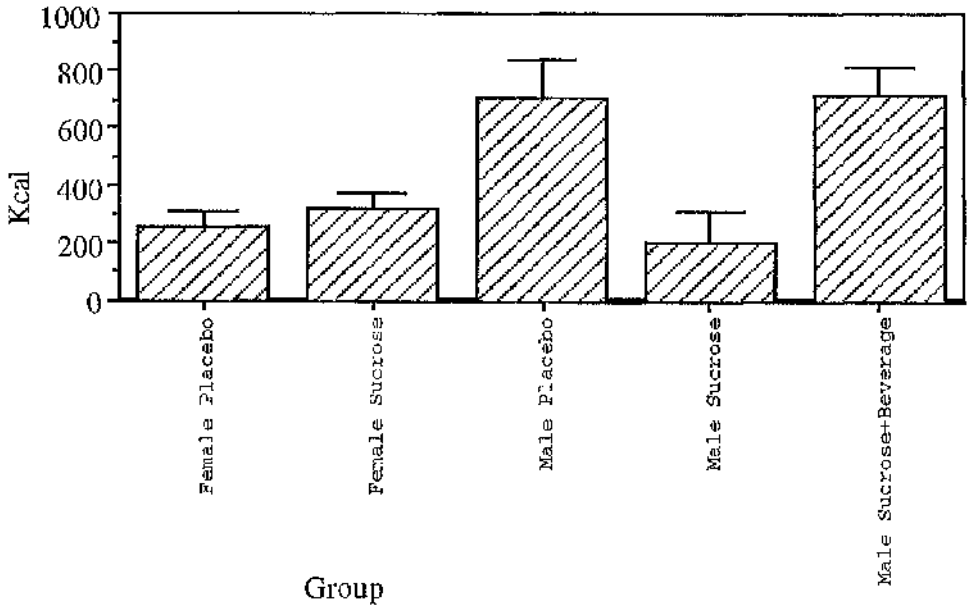
Despite the inherent inaccuracy of diary records of food intake, the methodology used here appears to be a useful supplement to studies of eating under precisely controlled conditions. It would appear that caution must be exercised in assuming that eating behaviour in the laboratory will resemble everyday eating behaviour. In the latter conditions the behavioural effects of a preload may be mediated by habit.

Figure 4.1: Effects of sucrose or placebo preload on time of next solid food.



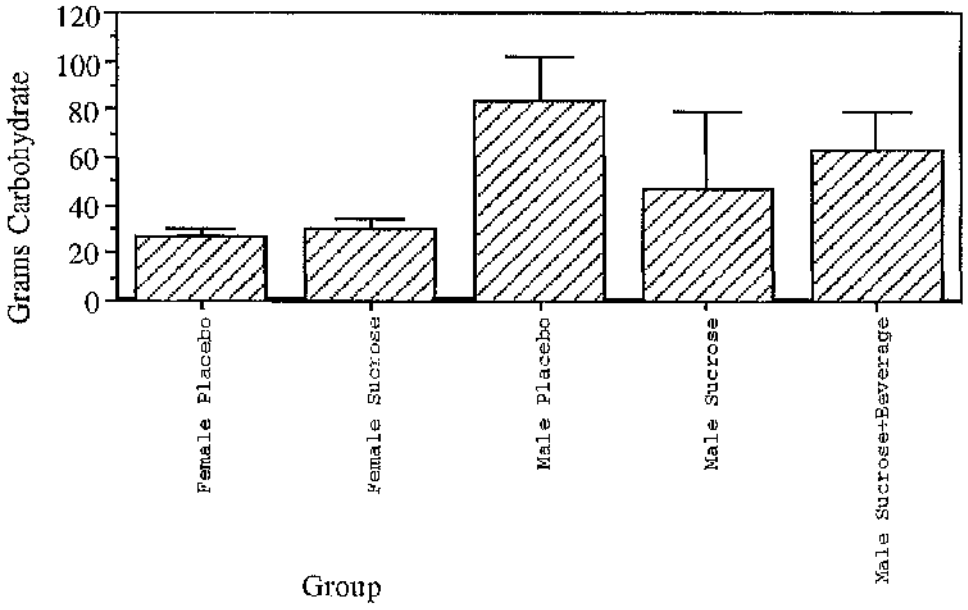
Data are shown as changes in timing from subjects' normal lunch times in hours (calculated from 7-day diary). Then change in meal time was calculated by subtracting time of next meal from mean meal time. As subjects had not eaten breakfast, most ate lunch early. Sucrose significantly delayed eating, compared to placebo ($F(1,47)=7.27$, $p<0.01$). Sex of subjects had no effect ($F(1,47)=0.18$, $p>0.1$), nor did weight ($F(1,47)=0.59$, $p>0.1$). Bars are standard errors.

Figure 4.2. Effects of sucrose or placebo preload on calories consumed in next solid food.



There was no main effect of preload content ($F(1,47)=0.80$, $p>0.1$), but males consumed more calories than females ($F(1,47)=14.81$, $p<0.001$). Weight had no effect ($F(1,47)=0.67$, $p>0.1$). Despite the apparent effect of sucrose in males only, there was no clearly significant preload by sex interaction ($F(1,47)=2.94$, $p=0.09$) and a post-hoc between groups t-test on males only was not significant ($t(22df)=1.36$, $p>0.05$). Bars are standard errors.

Figure 4.3: Effects of sucrose or placebo preload and drinking a calorific beverage on grams of carbohydrate consumed in next solid food.



There was no main effect of preload content ($F(1,47)=1.46$, $p>0.1$), but males consumed more carbohydrate than females ($F(1,47)=15.30$, $p<0.001$). Weight had no effect ($F(1,47)=0.48$, $p>0.1$). Despite the apparent effect of sucrose in males only, there was no preload by sex interaction ($F(1,48)=2.91$, $p=0.10$) and a post-hoc between groups t-test on males only was not significant ($t(22df)=1.34$, $p>0.05$). Bars are standard errors.

Chapter 5-The Effects of Carbohydrates on Mood and Subsequent Food Intake

Experiment 2

In the pilot study and experiment 1 sucrose delayed subsequent food intake. However, despite the promising findings in the pilot study, mood was unaffected by sucrose in experiment 1.

As reviewed in chapter 1 several studies have found that mood is affected by carbohydrate, most commonly leading to sleepiness and low arousal, but many studies suffer from methodological flaws and are incomplete. To recap: Problems include variation in the time of day the experiments are conducted, assessing mood at different times, failure to include baseline measures for mood scores (Spring et al., 1983; Keith et al., 1990), failure to include a control group or comparative group, using small samples, short-term studies versus long-term studies all of which are liable to affect the data on mood and eating behaviour in some way or another.

The aim of this study was to examine the impact of a carbohydrate-rich drink on an individual's mood state and subsequent food intake using the Profile of Mood States, natural eating behaviour outside the laboratory and a between-subjects design, whilst controlling the administration of carbohydrate so that subjects were blind to what they had ingested. Mood was assessed at 0, 30, 60 minutes after the experimental drink after Thayer, (1987) and Pivonka and Grunewald, (1990), on the assumption that liquids are absorbed more rapidly than the solid food used by Spring et al. (1983, 1986) and based on past findings should affect mood earlier than two hours.

If a subject expects to experience a specific psychological effect as a result of the ingestion of a certain foodstuff, then it may be reported as happening (Sprague, 1981).

Consequently, results may reveal an effect that is not caused directly by carbohydrates, but rather is an artifact of the attitudes and prior expectancies of the volunteers. For this reason it was decided to include an unblind control group as well as a placebo group in this design to control for any expectancy factors that might affect the experimental findings.

The literature suggests differential effects of carbohydrates consumed in the morning compared to the afternoon. Most of the studies which have reported significant effects of carbohydrates on behaviour were conducted at lunch time or later on in the afternoon (Spring et al., 1986; Lieberman et al., 1986, Smith and Leekam, 1988; Craig and Richardson, 1989; Pivonka and Grunewald, 1990; Christie and McBrearty, 1979). Fewer studies have been conducted at breakfast reporting effects of carbohydrates. Yet, it may well be that breakfast does not affect mood in the same way, which may in part be attributable to endogenous processes rather than to the nutrient *per se*, as well as to extent of prior fasting. As Craig (1985) has indicated people tend to be more alert in the morning compared to in the early afternoon, and this alone may cause differential effects on mood between studies of breakfast and lunch. This might prove to be a critical influence of the carbohydrate-mood effects reported in afternoon studies, especially when these have been in the direction of reduced arousal. Thus, one of the aims of the present study was to determine the effects of a carbohydrate-rich preload administered blind at breakfast on hunger and mood when compared to a placebo or control condition. Ideally, it would have been more beneficial to test the effects of sucrose in the morning and afternoon (modelled after studies such as Benton and Owens, 1993 and Spring et al., 1983). However, this proved to be impractical in terms of time constraints, in that more subjects (i.e double the number) would have been required if using a between subjects design.

Lastly, although some studies reporting significant findings in relation to carbohydrates and mood (Spring et al., 1986) have tested plasma amino acid ratios

before and during testing periods which might indicate changes in brain serotonin, it was decided in this study not to conduct such testing as it was felt that such intervention would have interfered too much with the naturalistic conditions of the study. Plasma glucose and insulin samples were not measured for similar reasons.

Hypotheses

The present experiment was designed to test the main hypothesis in the literature, namely that the ingestion of sucrose has a differential effect on a subject's mood when compared to saccharin and water, which implies that variations in mood can be physiologically induced by carbohydrate ingestion. More specifically, the literature suggests that carbohydrates may cause a decrease in arousal in the afternoon but have no effect, or may boost energy levels, in the morning. Here, the effects of sucrose in the morning were examined.

Design

In the sucrose condition subjects were administered a sucrose beverage. Placebo was the second condition where subjects received a saccharin beverage. Unblind control was the third condition and subjects in this group were given water to drink.

Subjects

Subjects were thirty-one males and twenty-nine females recruited from several businesses in Glasgow and the surrounding area. Some university undergraduate students also participated in the study. Subjects ranged in age from eighteen to fifty-five years. The mean BMI for the females was 23.1 (3.9SD). The mean BMI for the male group was 24.6 (3.4SD). As in the previous experiment all volunteers were screened for eating disorders, using the Eating Disorder Inventory (EDI) (Garner, 1990) which was also found to be useful in obtaining information regarding whether a subject was taking any medication known to affect appetite and/or mood. The sixty participants were divided into three groups. Two subjects were excluded for feeling unwell on the day of testing, for reasons unassociated with the experiment.

Measures

As in the pilot study and experiment 1 subjective mood was measured using the Profile of Mood States.

Procedure

The procedure was identical to the one used in the pilot study and experiment 1 with a few differences: 1) Subjects in the third group who were given water to drink were informed that they were serving as a control group. 2) As well as completing POMS prior to and immediately after drinking, subjects also completed POMS thirty minutes and sixty minutes after drinking. They were allowed to leave the laboratory environment after the thirty minute mood test, as we wanted to minimise their presence in an experimental environment, the artificiality of which could create an abnormal effect in subjects' mood state. However, subjects were asked to refrain from consuming any food or drink during this time as this could evidently affect their mood ratings for the sixty minute mood test. The remaining POMS questionnaire together with the twenty-four hour diary was given to the experimenter the following day. In order to examine cognitive expectations the following day subjects were asked what they thought they had drunk. Similar to experiment 1 some subjects complained of caffeine deprivation on the morning of test. Again, however, paired t-tests indicated that their pretest POMS scores did not differ significantly from their mood the previous week. Subjects did, however, mention either during or after the experiment that they found the task of completing the POMS very tedious.

Results

Intake of Beverages Prior to Next Solid Food

Figure 5.1 shows the proportion of subjects in different conditions who drank calorific beverages prior to eating solid food. It can be seen that, as in the previous experiment, this behaviour was predominantly engaged in by male subjects who had received sucrose. For this reason beverage consumption was included in the analysis reported

below. Only one female who received sucrose then drank a calorific beverage. For clarity her data are shown as a single data point with zero error in the figures below.

By pair t-tests baseline ratings on all six POMS subscales did not differ from ratings the previous week (all $p > 0.05$). Post-eating changes in mood scales were calculated by subtracting from the respective baseline scores. POMS scores were subject to multivariate analysis of variance with preload (sucrose, placebo or water), gender (male or female) and whether or not subjects had ingested calorific beverages prior to their next solid food (yes or no) as independent factors. Time of test (immediately after eating, thirty minutes or sixty minutes) and scales (composure, elation, energy, clearheadedness, agreeableness, and confidence) were used as within-subjects factors. In this analysis, there were no main effects of preload ($F(2,49)=0.90$,n.s), gender ($F(1,49)=0.11$,n.s.) or beverage consumption ($F(1,49)=0.28$,n.s). There was no effect of time of test ($F(2,48)=0.72$, n.s). Nor were there any significant interactions between scale and time, such as would indicate that sucrose affected only some scales. As the most likely change was a change in rated energy, but a similar analysis to the above on rated energy alone also found that it was unaffected by preload content (see Table 5.1).

Effects of the Preload on Subsequent Eating

Time of next solid food, the carbohydrate content of next solid food and the energy content of next solid food were subject to analysis of variance, using preload (sucrose, placebo, water) and gender (male, female) as independent variables and the number of calories taken in beverages prior to solid food as a covariate. As can be seen in figure 5.2, time of next solid food was significantly delayed by ingesting a sucrose preload ($F(2,51)=11.1$, $p<0.001$). Indeed placebo and water subjects ate almost immediately after the hour's post-preload fast. Placebo and water conditions did not differ, indicating that there was probably no psychological component to this effect. There

was also an effect of energy intake in beverages ($F(1,51)=9.2$, $p<0.005$), but no gender difference ($F(1,51)=0.02$,n.s).

Figure 5.3 shows the energy content (including prior beverages) of the next solid food. This was unaffected by preload content ($F(2,51)=1.9$, n.s.), but marginally decreased by prior beverage ($F(1,51)=4.0$, $p< 0.051$). Women tended to ingest lower-energy food than men ($F(1,51)=5.11$,
$p< 0.05$).

Finally, figure 5.4 shows the carbohydrate content of the next solid food (again including prior beverages). This was again unaffected by preload content ($F(2,51)=1.1$,n.s.), increased by prior beverage ($F(1,51)=4.7$, $p<0.05$) and women tended to ingest smaller quantities of carbohydrate than men ($F(1,51=8.0$, $p<0.01$).

Discussion

As before, subjects who had consumed the carbohydrate drink significantly delayed time of next solid food compared to subjects who had been given saccharin or water. The female sucrose group delayed intake longer than the male sucrose group. Taken alone, these data could suggest cognitive and/or physiological effects of sucrose on food intake, but the inclusion of two other conditions makes this interpretation less likely. The fact that both the placebo and control group ate their next solid food at more or less the same time suggests that cognitive factors did not greatly influence eating behaviour in this study which makes the physiological interpretation more likely.

Furthermore, although some subjects in the sucrose group drank a calorific beverage (mainly tea or coffee with milk and/or sugar) soon afterwards, they still delayed their next solid food longer than those who had been given water or saccharin. Indeed, it is possible that for those who consumed a caffeine beverage after the preload, this was motivated by cognitive factors (such as self-awareness of caffeine deprivation) and learned/habitual cues rather than in direct response to hunger.

As was found in the previous study the ingestion of the sucrose preload did not in itself affect the energy (figure 5.3) or carbohydrate (figure 5.4) content of the next meal, and this applied to both sexes.

Although the carbohydrate-rich preload delayed eating, there were no significant effects on mood in this procedure up to sixty minutes after carbohydrate ingestion (Table 5.1). Since the lack of effect fails to lend support to previously published findings (except for Brody and Wolitzky, 1983), possible explanations for the reported results will be considered.

A possible competing explanation for the lack of effect is that the procedure was not powerful enough to detect mood fluctuations subsequent to the ingestion of sucrose. This is not likely to be true for various reasons. The experiment found that the 40 g of sucrose used in a liquid preload was sufficient to delay subsequent food intake. Therefore, it is likely that the same nutrient dosage would suffice to produce alterations in mood which could be observed with the sample size used. In a study reported since this work was completed, Benton and Owens (1993) found using an elaborate design with a large sample (n=354) that mood effects were very small following glucose ingestion. Thus, it is hardly surprising that glucose had no effects on mood in this experiment with a smaller sample size and no control of blood glucose levels. This would also apply to many other studies of mood, including most alleging positive effects.

Problems in methodology in some previous studies may partly explain reported carbohydrate-mood effects (e.g. failure to measure baseline mood, small sample size). Moreover, the utilisation of designs where repeated measurements are taken could in part account for dietary-induced effects. Participation in an experimental procedure over days with repeated measurement of mood could substantially increase the effects

of general test fatigue which might prove to be a critical influence of the carbohydrate-mood effects, especially when these have been in the direction of reduced arousal. The importance of order effects should be considered at this point; all that would be required is for an unbalanced number of subjects to receive carbohydrates last in the design, thereby producing an effect that is an artifact of the experimental design rather than a true effect of carbohydrate on mood.

Alternatively, it is possible that endogenous processes interact with high-carbohydrate food and test fatigue to produce drowsiness in some subjects. This may be particularly true in the case of the post-lunch dip (Smith et al., 1988). A related problem is that when data are reported as simple means, or means and standard deviations, it is difficult to ascertain whether certain effects such as an increase in fatigue is representative of all the subjects as a group feeling slightly more drowsy than, or to a few subjects who differ qualitatively from the other subjects in the direction of a strong effect of sugar on fatigue.

If carbohydrates affect mood at all, previous evidence being both weak and equivocal with the exception of Pivonka and Grunewald (1990), then the results of this study, as well as the previous study (experiment 1) and Brody and Wolitzky (1983) suggest that this does not occur within the first hour after modest doses (e.g. 40-50 g sucrose) administered in the morning instead of breakfast. Although Benton and Owens' (1993) study showed an effect of glucose on mood up to two hours the magnitude of these effects were very small.

The proposal that the ingestion of foods high in sugar influences mood state in such a way that carbohydrate craving is thereby induced which, in turn enhances appetite was not supported in this study, at least not in the morning. Although different effects were found in men and women subsequent to the sucrose preload administered in the morning, no evidence that the ingestion of sucrose led to cravings for sweet foods or

increased appetite for either group was found. Rather, the findings reported here suggest that the ingestion of 160 kcal of sucrose delayed subsequent eating.

The blind administration of sucrose combined with the study of mood and eating behaviour in a natural environment where subjects rated their own mood on several occasions and followed everyday eating patterns, rather than eating food offered to them under laboratory conditions (which may increase food intake, particularly if the food presented is highly palatable) was considered a particular strength of this experiment. It is possible that when previous experiments reported an effect on mood and an increase in appetite and/or food intake subsequent to carbohydrate ingestion the effect may be an artefact of poor method. For example, the delay effects after the sucrose preload reported in this study and the interaction of eating habits with preloads often go untested in studies where food is administered at a specific time following a preload and the food presentation is controlled. With adequate controls sucrose in the morning delays eating but does not affect mood.

Table 5.1 Effects of Preloads on Mood from POMS Scales (Means and SDs of POMS scales)

Agreeable	Before	After	30 mins	60 mins
Sugar 1	27.1	27.6	28.4	27.2
	(5.8SD)	(6.6SD)	(7.2SD)	(6.7SD)
Saccharin 2	28.5	29.9	27.9	28.0
	(5.8SD)	(4.6SD)	(5.4SD)	(5.8SD)
Water 3	29.7	30.3	28.8	28.9
	(6.4SD)	(6.4SD)	(7.2SD)	(7.7SD)

Clearheaded	Before	After	30mius	60 mius
Sugar 1	26.3	25.8	26.7	26.3
	(7.3SD)	(7.6SD)	(7.3SD)	(8.6SD)
Saccharin 2	27.9	28.7	28.6	28.0
	(6.3SD)	(5.0SD)	6.0SD)	(4.6SD)
Water 3	26.8	26.6	26.6	26.4
	(5.2SD)	(5.3SD)	(5.8SD)	(6.2SD)

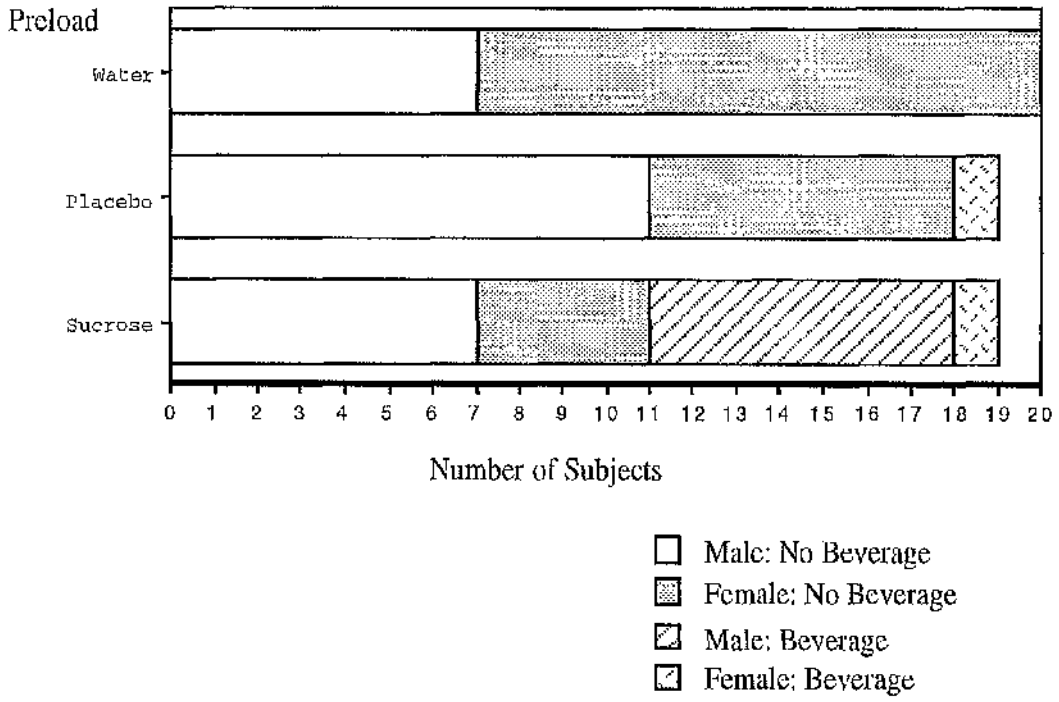
Compose	Before	After	30 mins	60 mins
Sugar 1	23.3	24.4	26.3	27.0
	(5.7SD)	(7.6SD)	(7.5SD)	(7.0SD)
Saccharin 2	25.4	26.5	26.0	25.6
	(5.6SD)	(4.9SD)	(5.8SD)	(5.5SD)
Water 3	25.8	26.2	25.2	26.8
	(6.3SD)	(6.0SD)	(7.4SD)	(7.5SD)

Confident	Before	After	30 mins	60 mins
Sugar 1	23.0	24.7	24.6	25.1
	(8.3SD)	(8.2SD)	(9.1SD)	(7.8SD)
Saccharin 2	25.4	24.4	24.4	24.7
	(6.4SD)	(4.8SD)	(5.7SD)	(4.8SD)
Water 3	25.0	23.8	24.3	24.3
	(5.6SD)	(6.3SD)	(6.0SD)	(6.5SD)

Elate	Before	After	30 mins	60 mins
Sugar 1	24.0	25.7	26.1	26.8
	(7.6SD)	(6.8SD)	(8.4SD)	(6.8SD)
Saccharin 2	26.1	27.6	26.6	26.3
	(6.3SD)	(4.9SD)	(5.6SD)	(4.7SD)
Water 3	26.4	26.8	27.1	26.0
	(5.4SD)	(7.4SD)	(7.1SD)	(7.8SD)

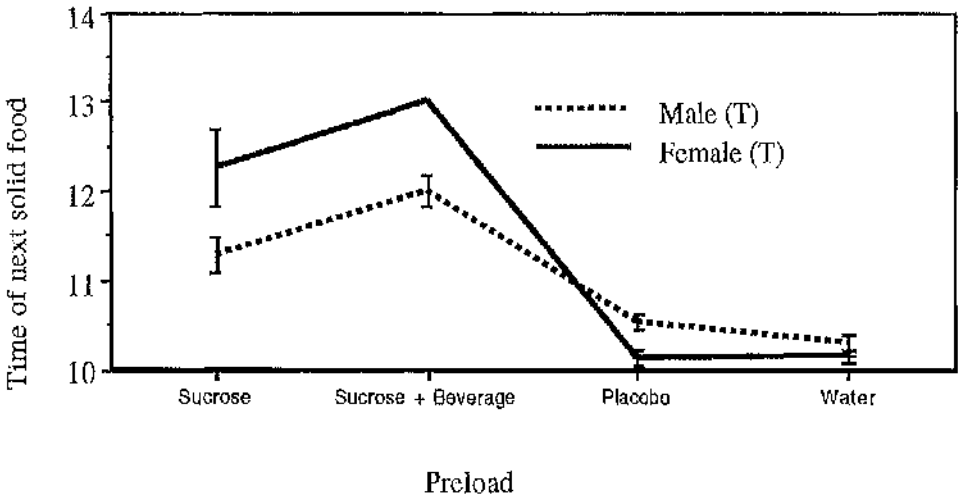
Energy	Before	After	30 mins	60 mins
Sugar 1	24.4	24.2	24.8	24.8
	(8.6SD)	(9.2SD)	(9.6SD)	(8.8SD)
Saccharin 2	24.0	24.7	22.5	23.1
	(5.9SD)	(6.3SD)	(7.9SD)	(6.8SD)
Water 3	24.2	25.3	24.3	24.3
	(6.5SD)	(7.1SD)	(8.1SD)	(7.3SD)

Figure 5.1: The proportion of subjects who drank calorific beverages prior to next solid food.



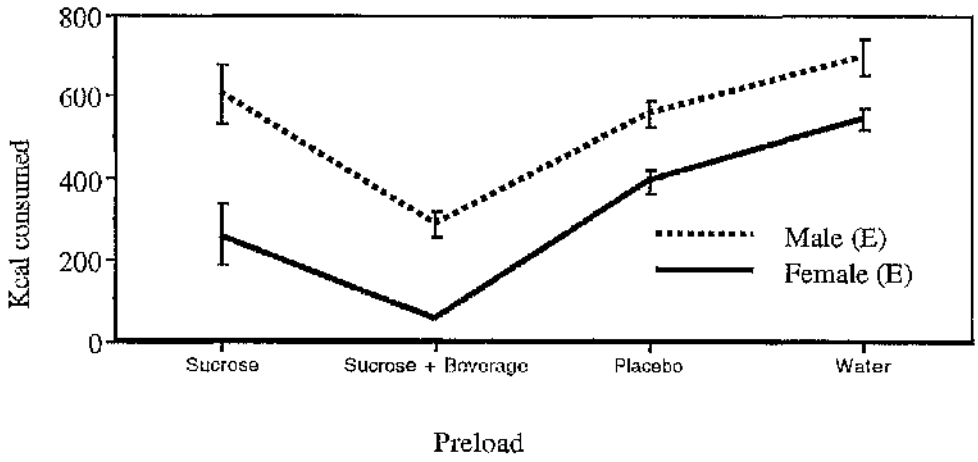
The proportion of subjects in different conditions who drank calorific beverages prior to eating solid food can be seen from the above figure. This behaviour was predominantly engaged in by male subjects who had received sucrose. Only one female who received sucrose then drank a calorific beverage. For clarity her data are shown as a single data point with zero error in the figures above.

Figure 5.2: Time of next solid food



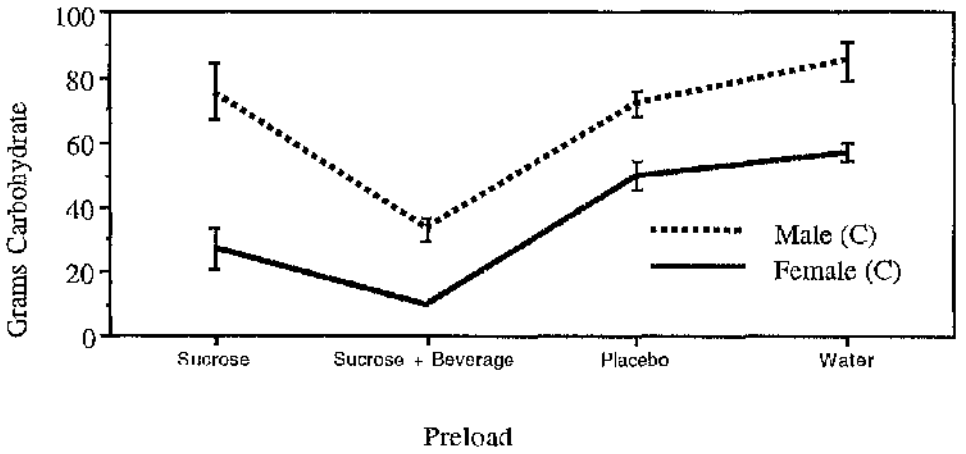
Time of next solid food was significantly delayed by ingesting a sucrose preload ($F(2,51)=11.1$, $p<0.001$). Placebo and water subjects ate almost immediately after the hour's post-preload fast. There was also an effect of energy intake in beverages ($F(1,51)=9.2$, $p<0.005$), but no gender difference ($F(1,51)=0.02$,n.s).

Figure 5.3: Energy content (including prior beverages) of the next solid food



Energy content was unaffected by preload content ($F(2,51)=1.1$, n.s.), but decreased by prior beverage ($F(1,51)=2.9$, $p< 0.05$). Women tended to ingest lower-energy food than men ($F(1,51)=8.0$, $p< 0.01$).

Figure 5.4: Carbohydrate content of the next solid food (including prior beverages)



Carbohydrate content of the next solid food was unaffected by preload content ($F(2,51)=1.9$,n.s.), marginally increased by prior beverage ($F(1,51)=4.0$, $p=0.51$) and women tended to ingest smaller quantities of carbohydrate than men ($F(1,51)=5.1$, $p<0.05$).

Chapter 6-Short-Term Effects of Sucrose on the Eating Behaviour of Obese and Non-Obese Women

It was found in chapter 5 that sucrose delayed appetite and subsequent food intake in normal-weight individuals, but did not have any significant effect on mood up to sixty minutes later. In this chapter the main issues examined are possible variation in eating behaviour and mood in the obese and non-obese following the same sucrose preload. As discussed in chapter 3 some investigators have found that the obese are not as responsive to variations in energy density as normal subjects (Schachter, 1967). According to Wurtman et al. (1981) obesity may be associated with disordered eating behaviour which is characterised by preferential ingestion of carbohydrate-rich snacks. This so called Carbohydrate-Craving Obesity (CCO) is apparently related to a dysphoric mood state which is temporarily alleviated by carbohydrate-rich snacks.

In fact, there is little substantial evidence in support of Schachter's internal-external theory (1967) (see chapter 3) and the findings for the so-called "Carbohydrate Craving Syndrome" (Wurtman et al. 1981) (see chapter 1) are also inconclusive. At the risk of repetition: many of these studies are methodologically inadequate; poor control of psychological factors, lack of control of prior food intake, use of small numbers and artificial nature of the experiments are among the most glaring defects of these studies. The present study had two main objectives: b) to examine if there were any obese-non-obese distinctions as regards short-term food regulation; a) to determine if a caloric preload administered at 11.00h would delay subsequent food intake when compared to a placebo drink or whether subjects would be affected by environmental stimuli and habits (e.g. time of day, i.e. approaching lunch hour).

The decision to examine the effects of a sucrose preload administered at 11.00h was made to test whether subjects would compensate for caloric supplementation by

adjusting nutrient intake at lunch time. The past two studies have shown that compared to after placebo at 9.30 am, sucrose ingestion delays food consumption. However, the implications of these findings are complicated by normal eating habits. Seemingly, sugar could substitute for breakfast, for those who received it waited until lunch time before eating again. Sugar may have different effects when ingested after normal eating. Indeed, one of the problems of the previous experiments was the failure to include data from the 7 day diaries on how many subjects were breakfast eaters. Here, this information was collated and used in the overall analysis. Furthermore, the obese may be particularly responsive to external cues, such as meal time. As in the past two studies an attempt was made to combine eating in the laboratory with eating in a natural setting. This was considered particularly useful for studying eating behaviour in the obese who may be more sensitive to a laboratory setting. It was decided to test females only mainly because of the practical difficulty of trying to recruit obese men; obese subjects were recruited through advertising in health clubs, but the majority who frequented such clubs tended to be obese females with very few obese males.

Hypotheses

The central hypothesis being tested in this experiment was that there are differential effects of sucrose on behaviour in unrestrained obese and unrestrained non-obese females, when compared to saccharin or water. As before natural eating behaviour and rated mood were examined.

Design

The design was similar to experiment 2, in that there were three conditions, the sucrose, saccharin and water conditions. The third group who were given water to drink were informed that they were serving as a control group. There were forty-five obese and forty-five non-obese females, (and fifteen of each weight were randomly assigned to each condition).

Subjects

All subjects who participated in this study were selected according to various criteria. All subjects were to be over 21 years of age and obese subjects were to be from the non-clinical populations. Although obese individuals from clinical settings are of interest they are often over represented in diet-behaviour studies, and it is not clear that their behaviour can generalise to the obese population at large. For example, there is considerable evidence that obese individuals, not selected from psychiatric or medical referrals, are no more psychologically disturbed than the non-obese (McReynolds, 1982). Only those who reported that they were not currently dieting were selected. Subjects also completed the Eating Disorder Inventory Symptom Checklist (EDI-SC) which is a self-report form, separate from the EDI questionnaire. The EDI-SC provides data on dieting, weight history (including current weight), exercise patterns and the frequency of eating symptoms such as binge eating, purging, use of laxatives, diet pills and diuretics. Information on menstrual history and current medication is also obtained from this checklist. Those manifesting disordered eating and/or dieting or participating in vigorous exercise were excluded, as was anyone taking medication related to diabetes or any psychiatric disorder.

Volunteers were then asked to complete the EDI questionnaire which has eight subscales; Drive for Thinness (DT), Bulimia (B), Body Dissatisfaction (BD), Ineffectiveness (I), Perfectionism (P), Interpersonal Distrust (ID), Interoceptive Awareness (IA), Maturity Fears (MF), Asceticism (A), Impulse Regulation (IR) and Social Insecurity (SI). These subscales assess attitudes and behaviour concerning eating, weight, shape, whilst also tapping into more psychological traits clinically relevant to eating disorders. It contains sixty-four items presented in a six-point format requiring respondents to answer whether each item applies "always", "often", "usually", "sometimes", "rarely" or "never".

The EDI provides individual patient profiles that may be compared against norms for eating disorder patients and comparison samples. Calculation of the raw scores and percentile scores showed that when compared against norms for eating disorder patients no subject manifested chaotic dietary patterns or any psychological disturbance. Furthermore, there were no substantial differences between both groups of subjects on any of the scales, except body dissatisfaction with obese subjects scoring marginally higher than the non-obese on this scale. Thus the samples tested consisted of individuals who generally displayed low levels of eating disorder symptomatology.

Obese and non-obese subjects were recruited in different ways. Most normal-weight subjects were recruited by advertising in places of employment around Glasgow and only a few were recruited from health clubs. Obese females were recruited through advertising in health clubs. It was presumed that the average level of exercise of the normal subjects would be greater than the obese group. Thus, by recruiting most normal weight subjects from the workplace rather than health clubs (where participation in exercise is common) it was expected that such differences would decrease. After recruitment, whether or not subjects were actually obese was defined according to their BMI (Body Mass Index) score. Those scoring 30 or more were defined as "obese". Those scoring less than 30 as "non-obese". This cut off was selected to produce two equally sized groups. The mean BMI for the obese group was 31.5 (1.2SD) and the range was 29.4-35.0. The mean BMI for the non-obese group was 21.3 (1.9SD) and the range was (17.9-26.6), (see Table 6.1). Nonetheless, as will be described below, rather than categorise subjects, BMI was treated as a continuous variable in analyses.

The normal females' mean age was 33.24 years (SD=7.76), their mean height was 164 cm (SD=0.04) and their mean weight was 57.30 kg (SD=6.42). The obese

group's mean age was 34.93 years (SD=8.22), their mean height was 163 cm (SD=0.02) and their mean weight was 83.33 kg (SD=2.47) (see Table 6.1)

Procedure

The procedure was the same as before with the exception that instead of fasting subjects were instructed to eat as normal before participating in the experiment which took place at 11.00h. However, subjects were requested to record all prior food intake in a diary on the morning of the experiment so that monitoring of prior intake was maintained. The Profile of Mood states was the device used to measure mood, but this time mood was only rated twice after the beverages were consumed; immediately after they had finished drinking, and thirty minutes after intake (when Pivonka and Grunewald, 1990 found largest effects). Due to the lack of effect on mood scales in the preceding experiments it was decided to not assess mood after a longer period. Complaints of boredom and fatigue with the POMS from previous subjects when completing it several times also influenced this decision. Furthermore, although it was recognised that measurement of mood at 0 and 30 minutes was not likely to pick up 5HT mediated effects, it could still detect sucrose mediated effects at 30 minutes since mood changes have been reported then (Thayer, 1987; Pivonka and Grunewald, 1990). Subjects were requested to fast between zero to thirty minutes after the test drink and were allowed to return to work immediately after the drink and complete the twenty-four hour diary in a natural setting.

Subjects were told that the study was investigating the effects of carbohydrates on eating behaviour but were not informed that some of them would be administered a placebo drink containing saccharin. All subjects were interviewed at the end of the experiment when they were debriefed about the actual purpose of the study. Subjects were also asked at this point if they thought they knew the caloric content of the beverage consumed and the hypotheses being tested. On the basis of on their responses no subject seemed to have correctly guessed the true purpose of the study or that they had consumed saccharin.

From the food diaries were extracted: (a) Time of any solid food, to the nearest hour, prior to the test drink. (b) Calories consumed prior to testing, including all foodstuff, solid and liquid, which is referred to as "breakfast". (c) Carbohydrate content of solid food prior to testing, calculated as for calories. (d) Calorific content of any beverages taken without solid food prior to testing (calculations mainly involved the caloric content of milk and sugar consumed in tea or coffee). (e) Time of next solid food after the test drink, to the nearest hour. A meal was defined as all food and drink consumed within the first hour period following the preload during which solid food was consumed. (f) Calories of next solid food, including all solid and liquid foodstuff, consumed within the respective hour time slot. (g) Carbohydrate content of next solid food. (h) Calorific content of any beverages taken without solid food after testing (again calculations mainly comprised the caloric content of milk and sugar consumed in tea or coffee).

Results

Analyses of covariance were conducted on the following measures of mood, which according to the literature should be most likely to be affected by a sucrose preload: POMS energy and POMS elation, both immediately after eating and at 30 minutes. Analyses of covariance were also conducted on the same measures of food intake as used in the previous experiments: Time of next solid food, carbohydrate content of next solid food, energy value of next solid food. All seven analyses had the same design, using the preload (sucrose, saccharin, water) as a independent variable and the following covariates: Body-mass index, drive for thinness, body dissatisfaction, social insecurity, the time subjects ate breakfast, the carbohydrate content of breakfast, the energy content of breakfast and the energy content of any beverages consumed without breakfast.

None of the mood scales were significantly affected by any of these variables (all $F(1,82)$, $p>0.05$). As in the previous experiments, the time of the next solid food was

significantly delayed by the sucrose preload ($F(1,82)=52.3$, $p<0.001$). There was also a main effect of the energy content of beverages ($F(1,82)=14.3$, $p<0.001$) such that those who had consumed higher-energy beverages tended to eat later. There was also a marginal effect of body-mass index ($F(1,82)=3.9$, $p=0.053$), indicating a very slight tendency for lighter subjects to eat later (but the correlation between eating time and BMI was not significant). There were no interactions which might suggest that obese and non-obese subjects responded differently to the preloads, or that subjects with higher EDI scale scores responded differently. The carbohydrate content of lunch was unaffected by the preload or the other control variables. The energy content of lunch was affected only by the time of eating breakfast ($F(1,82)=4.1$, $p<0.05$). As those who did not eat breakfast scored 0 for "time of eating breakfast", this result means that those who ate breakfast at all tended to report eating more calories for lunch. Again, there was no effect of or interaction involving BMI.

The obese and non-obese groups were also compared on their EDI scores, Despite current dieters being excluded, by one-way ANOVA, obese subjects scored significantly higher on the scales measuring Drive for thinness, Body Dissatisfaction and Social Insecurity, but not on the remaining EDI scales (see Table 6.3).

Discussion

As in the previous two studies, the sucrose preload delayed eating more than the saccharin beverage or the control group and this effect applied to both obese and non-obese groups (Figure 6.1). Furthermore, the findings from this study show that sugar has similar effects when ingested at 11.00 h (i.e. delays eating) when subjects are in a non-fasted state as when ingested at breakfast time, 09.30h in a fasted state.

Furthermore, these results lend little support to the prediction made by Schachter (1967) that the obese are less responsive to internal physiological cues than normal-weight people. Although neither group reduced lunch to compensate for the preload,

both groups delayed lunch after the sucrose drink from which one may infer that physiological mechanisms were in operation.

In this study an attempt was also made to examine the effects of sucrose on mood in the obese and non-obese groups. The data obtained from the mood ratings showed that although changes in mood occurred for the energy and elated scales (as seen in Table 6.2) there was no significant correlation between mood scores and the composition of preloads. Differences in mood were found between the obese and non-obese groups but the reasons for this are not obvious since both sucrose and saccharin caused a decrease on the elated and energy scales for the obese, but the opposite effect was observed for the normal-weight subjects. These effects may have been random variations rather than being attributable to any physiological, or psychological mechanism.

Furthermore, as in the previous two experiments, there was no indication that the consumption of foods high in sugar content influences mood state. So carbohydrate craving cannot be induced and in turn enhance appetite. Here, there was no evidence that the ingestion of sucrose led to cravings for sweet foods or increased appetite in either the obese or non-obese group. Carbohydrate may have different effects on mood and appetite when ingested in combination with other nutrients. In some previous studies carbohydrate has been administered with fat and/or protein. This factor, together with the palatability of the food, may influence mood and eating differently. In studies by Wurtman et al. examining the "Carbohydrate-Craving Syndrome" (1981, 1984) as much as 68-98% (in terms of energy value) of the nutrients ingested were derived from a mixture of fat and carbohydrate. Similarly, most of the food consumed by obese subjects in the study on "Carbohydrate Craving" by Lieberman et al. (1986) contained more fat than carbohydrate (fat content 40-54% energy). Further, it is recognised that the theory on carbohydrate craving probably reflects results from a biased sample of subjects such as those seeking treatment, whilst in this study no

attempt was made to select or even identify subjects who might be considered as carbohydrate cravers.

Finally, although current dieters were excluded, obese subjects' EDI scores suggested that they were less satisfied with their bodies, less socially secure and had a higher drive for thinness. These scores suggest that the obese subjects may have been restrained eaters (dieters) at other times, which in turn suggests that any different effects of carbohydrate preloads on restrained eaters may be a function of their current diet, rather than a permanent personal trait.

In conclusion, it would seem that more systematic controlled research into the control of eating in both short and long-term studies is a prerequisite for a better understanding of the energy control in obese and non-obese subjects and may help to elucidate the etiology of obesity. In some recent studies, for example, the focus is on the nutrient fat and not carbohydrate leading to dysphoric mood (Wells and Read, in press), poor satiation (Lissner et al., 1987), weight gain and health problems particularly within the obese population. Lastly, considering the methodological difficulties involved in past studies on mood and carbohydrates and the various objections made from other investigators in the field (Teff et al., 1989; Young, 1991), particularly regarding proposals made by Wurtman et al. (1981) caution should be exercised when interpreting their findings or when intending to replicate or support them in any way.

Table 6.1: Details of Groups Age, Height ,Weight & BMI (Means and SDs)

Age	Obese	Normal
	34.9 (8.2SD)	33.2 (7.8SD)
Height	1.6 (0.1SD)	1.6 (0.1SD)
Weight	83.3 (2.5SD)	57.3 (6.4SD)
BMI	31.5 (1.2SD)	21.3 (1.9SD)
Min	29.4	17.9
Max	35.0	26.6

Table 6.2. Effects of preload on elation and energy.

Elation	Time	Sucrose	Saccharin	Water
Normal	10 min	2.3 (4.4SD)	0.9 (2.4SD)	2.0 (3.9SD)
Obese	10 min	1.1 (2.8SD)	0.7 (2.9SD)	0.1 (1.7SD)
Normal	30 min	1.9 (4.1SD)	0.7 (4.1SD)	1.7 (3.7SD)
Obese	30 min	0.3 (2.0SD)	-1.4 (3.9SD)	-1.0 (4.3SD)

Energy	Time	Sucrose	Saccharin	Water
Normal	10 min	0.1 (2.9SD)	1.7 -(3.1SD)	2.7 (3.1SD)
Obese	10 min	-0.4 (2.3SD)	1.7 -(3.1SD)	2.7 (3.1SD)
Normal	30 min	-0.6 (2.2SD)	-0.1 (3.7SD)	-0.5 (2.8SD)
Obese	30 min	-1.6 (3.7SD)	-3.8 (6.0SD)	0.4 (5.5SD)

Table 6.3-Difference between obese and non-obese on EDI Scales

EDI SCALE	Non-Obese	Obese
Thinness*	1.9 (3.6)	4.5 (4.6)
Bulimia	1.2 (1.5)	1.6 (2.1)
Body Dissatisfaction*	7.5 (6.6)	16.9 (6.5)
Ineffectiveness	2.9 (2.2)	4.1 (4.0)
Perfectionism	2.7 (3.3)	3.0 (2.7)
Interpersonal Distrust	1.8 (1.9)	2.7 (2.9)
Interoceptive Awareness	1.8 (1.8)	2.8 (3.5)
Maturity Fears	2.8 (2.3)	2.6 (2.5)
Asceticism	1.9 (2.4)	2.8 (2.6)
Impulse Regulation	1.6 (2.1)	2.5 (3.2)
Social Insecurity*	3.7 (2.7)	5.2 (3.7)

*Difference between obese and non-obese $p<0.05$, one-way ANOVA.

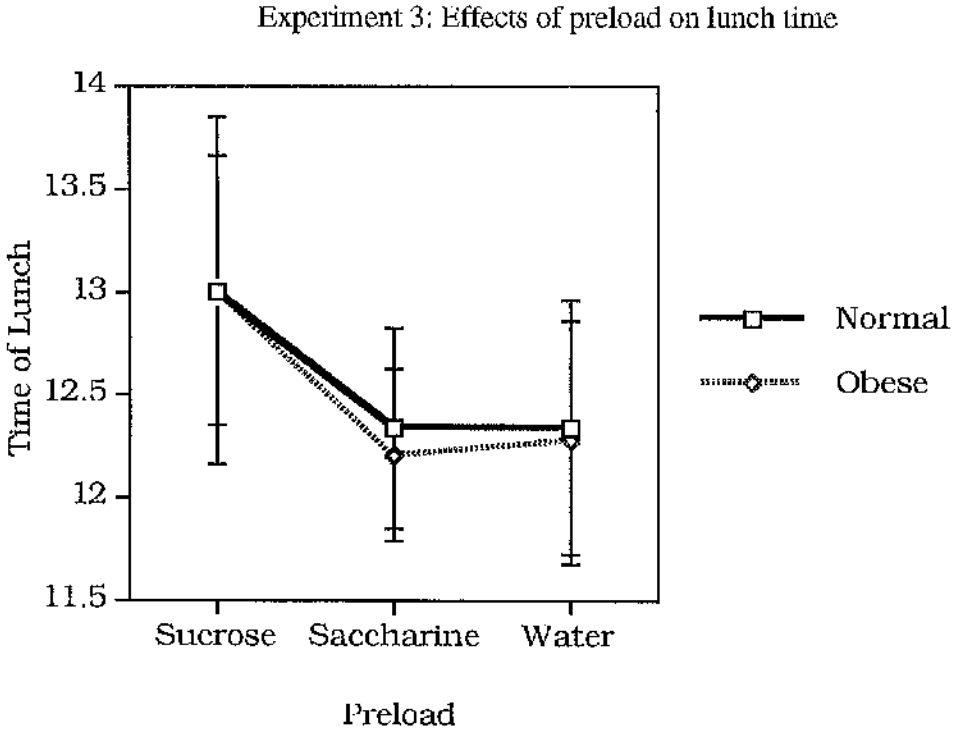


Figure 6.1: Time of the next solid food (lunch) was delayed by drinking a preload containing sucrose ($F(1,82)=52,3, p<0.001$).

Chapter 7- Overall Summary and Conclusions

This chapter will summarise briefly what has been discussed in the preceding chapters and draw conclusions. A review of the principal studies on carbohydrates and protein related to mood state was given in chapter 1. The objective was to ascertain the degree to which the ingestion of these food components is responsible for alterations of mood state in humans. From the studies reviewed several hypotheses have been made concerning the correlation between food or dietary constituents and human mood state. The central hypothesis is that the consumption of nutrients such as carbohydrate or protein lead to behavioural modification due to the apparent changes that they cause. One of the main findings cited in the literature is that the consumption of sugar or other carbohydrates induces feelings of calmness and sleepiness in the subject thirty to one hundred and twenty minutes after intake. This is consistent with the neurochemical hypothesis, whereby there is a relation between brain neurotransmitters and diet. Other possible mechanisms are the hypoglycaemia hypothesis (where emphasis is on the adverse behavioural effects induced by sugar ingestion) and the psychological theory which concentrates on cognitive expectations related to diet-induced behavioural change, as opposed to any physiological changes. There is evidence in the literature that all three mechanisms have their weaknesses and limitations.

Methodological difficulties are prevalent, but methodological issues are rarely considered seriously when some of the interpretations regarding the link between diet and mood are made. There is considerable need to increase diet control. Subject expectancies should also be controlled for effectively. Appropriate scales and devices should be used consistently when measuring behaviour. An adequate sample size is of great importance. Statistical analyses should be carefully conducted (numerous uncorrected statistical comparisons should be avoided) and properly reported. In the general absence of these procedures, no conclusions can be drawn.

In chapter 2 a review of the literature on the satiating effects of carbohydrates compared to other nutrients such as protein were examined. Various physiological and non-physiological processes influencing human feeding behaviour were also considered. From the main issues examined the following conclusions emerged:

1) In the absence of psychological factors normal-weight humans can delay onset of eating after a caloric preload which is taken to be due to physiological regulation. However, compensatory processes, particularly in the short-term may not always be accurate.

2) Specific nutrients such as protein, carbohydrate and fat may affect the regulation of energy intake. There have been claims that the nutrient protein suppresses subsequent food intake more than the two other nutrients, particularly carbohydrate, which has been found to increase appetite and food intake, although the evidence for this is inconclusive. In fact carbohydrates in the form of starch or simple sugars may also delay appetite and decrease subsequent food intake.

3) Factors such as conditioning, orosensory properties of food, and psychological influences can affect feeding behaviour and interact with internal mechanisms in the regulation of eating.

4) The regulatory process differs in the long and short term. Compensatory effects of certain foods may not take place immediately, but may be operated by a control system that functions over several days or so, whilst orosensory factors and cognitive attributes may have more of an immediate effect on eating. Thus, caution must be exercised in interpreting the results from short-term studies on the human regulation process.

5) Considering the importance of orosensory properties relative to feeding some control of these factors should be considered when examining the differential effects of nutrients on hunger and satiety.

6) In reviewing the chapter on the satiating efficiency of carbohydrates compared with other nutrients similar methodological problems to those in chapter 1 arose which ought to be considered when interpreting results as well as for future research in this area.

With these findings in mind chapter 3 was devoted to examining some of the external as well as internal factors involved in food regulation in both normal and obese subjects. The main issues discussed have been summarised and several conclusions have been made:

1) The internal-external theory proposed by Schachter (1967) stated that the eating behaviour of the obese was governed more by external stimuli than internal physiological signals which controlled eating in normals. Schachter (1967) proposed that this over-sensitivity to external food-related cues in the obese may play a significant role in the psychopathology of obesity.

2) Based on some research a relationship between palatability and obesity has been proposed, in that oversensitivity in obese individuals to palatable food might in part reflect their physiological state. Other researchers have found no such relationship. Whilst other studies have found that highly palatable food plays a decisive role in overeating for both the obese and non-obese.

3) There is some evidence that the physiological mechanisms which operate after food intake may differ in the obese depending on whether the foodstuff is of solid or of

liquid composition; the obese are perhaps more efficient at controlling intake after a liquid preload than a solid preload.

4) Some studies have found inaccurate compensatory behaviour following caloric manipulation in the diet of obese and normals both in the short and long-term. There is insufficient evidence that the regulatory system in normals is more efficient than the regulatory system in the obese.

5) Cognitive factors frequently influence eating in both obese and non-obese individuals and there is insubstantial evidence that obese subjects are more influenced by external-food related stimuli than normals. In effect, there is some evidence that cognitive factors play a more important role in eating behaviour than physiological factors in both groups.

6) An interaction of external and internal factors in eating for both normals and obese has been recognised which weakens Schachter's basic theory (1967).

7) Many methodological problems already raised in the previous chapters cast doubt over the validity of some of the findings discussed.

8) Based on the empirical evidence available it may be concluded that Schachter's internal-external hypothesis (1967) is, at best, a partial explanation of obesity.

Summary of Experiments

The empirical work of chapters 4, 5 and 6 examined some issues arising from chapters 1, 2 and 3. A summary of each experiment will be given and it will also be stated whether or not they provided any support to the theories discussed in the review chapters.

The pilot study and experiment 1 which extended the pilot work set out to test whether the ingestion of a carbohydrate-rich preload administered in liquid form would delay subsequent food intake or increase intake in a between-subjects, blind placebo design. According to what was discussed in chapter 2, the regulatory process works more efficiently in the absence of non-physiological stimuli, therefore, cognitive and orosensory factors were controlled for. In chapter 2 several studies which were reviewed found that carbohydrate increases appetite in general as well as for carbohydrate-specific foods and leads people to eat more afterwards (Rodin and Spitzer, 1983; Geiselman and Novin, 1982).

The experiment demonstrated that the sucrose preload delayed hunger and food consumption. The fact that psychological properties were controlled implies that the internal regulation system was effective. The study failed to support the theory that carbohydrates increase appetite and found no relation between the ingestion of the carbohydrate preload and subsequent carbohydrate intake. It may be concluded that if sugar intake increases hunger (and consequently sugar consumption, resulting in obesity, diabetes, hypoglycaemia and eating disorders) then such effects are more likely to be attributable to the sensory factors such as palatability of the food as well as psychosocial expectations, rather than to any physiological effects.

Experiment 2 examined the effects of carbohydrate on subsequent food intake as well as on subjective mood in men and women. The experimental design was similar to experiment 1. The neurochemical hypothesis proposes that the ingestion of carbohydrate-rich food leads to sleepiness and calmness in humans, whilst other views state that carbohydrate, particularly sugar induces hyperactivity, aggression and irritability. Whilst the results on subsequent food intake were the same as those reported in experiment 1, in that sugar delayed hunger, the ingestion of sugar failed to have any significant effect on mood up to sixty minutes thereafter. This suggests that

the carbohydrate-mood relation is not a strong one, at least not when modest size preloads are given to normal individuals in the morning.

Various methodological problems in past research investigating the diet-behaviour relation have been considered in this study which could account for the discrepancies in the findings. The most common ones being, namely failure to administer food in such a way that it controls for the sensory experience of eating, failure to obtain data prior to eating (specifically on mood and food intake) for comparison, and poor statistical analyses of the collected data.

Experiment 3 extended experiment 1 and 2. The effects of carbohydrate on subsequent eating and mood were examined in obese and non-obese subjects. The experimental design was similar to the previous studies except that the preloads were administered at 11.00h as opposed to 09.00h and there was no fasting condition in this study. The results of this experiment lend no support to Schachter's internal-external theory (1967), in that the sucrose delayed eating for both groups compared to the two other conditions. For those who had consumed sucrose, subsequent intake was delayed for approximately two hours, whilst those who had received saccharin or water ate within the next hour or so. Considering that orosensory properties and cognitive attributes were greatly controlled in this experiment implies that the internal regulation system is effective in both obese and normals to a large extent. As in the past studies no evidence of carbohydrate craving was found for either group in this study. From these findings it may be concluded that in the short-term the obese are no more or no less responsive to internal signals than normals.

General Conclusion

The fact that many of these studies of human eating and behaviour provide inconsistent results makes it difficult to reach conclusions concerning the psychophysiological effects of eating in humans. Nevertheless, some information and insight on diet-behaviour relations have been achieved both from the results of the experiments

conducted in this thesis and through the synthesis and commentary on empirical research.

An attempt has been made to address some of the problems of previous research (e.g. poor cognitive control, small sample size, artificial nature of laboratory studies) by conscientiously designing a series of studies that would tap into "natural" eating by older, normal adults, but at the same time which controlled for psychological expectancies. Although it may have appeared as if extreme methods were used so that particular variables might be held constant (e.g. nose plugs and anaesthetic lozenges to mask the flavour of sucrose versus saccharin), it is likely that these extreme but careful efforts have contributed to the findings in the diet-behaviour literature.

Furthermore, although none of the questions raised in these experiments are original, since most of the hypotheses tested in these experiments have been tested elsewhere, the experiments were more carefully conducted than some of the past research which have used weak methodologies and have subsequently assumed that their findings provide sufficient support or not as the case may be to the theories raised in this area.

By drawing attention to some of the limitations which experimenters should be conscious of when conducting research in this area (especially when using the findings from their experimental paradigm to support or provide general theories), an increase in awareness may be attained which will contribute to the investigation of carefully designed studies in the future.

From the central issues examined in these experiments it was found that:

1. When administered blind sucrose does not affect mood state within the first hour after food intake.
2. When cognitive factors are held constant in the laboratory sucrose delays eating after breakfast and at lunch time.

3. Sucrose does not decrease the number of calories in the subsequent meal.

4. There was no evidence that sucrose led to carbohydrate craving or increased hunger for carbohydrate-rich foods in any way.

5. These effects were unaffected by gender or age and there was no evidence that there were any differential effects on obese subjects.

In spite of the lack of effect of sugar on mood found in this work the diet-behaviour connection is still of significant interest and importance and more controlled empirical studies are required to examine eating and its effects on behaviour. Advances in the understanding of the physiological and psychological effects of food is essential as well as more elaborate methods of measuring changes in behaviour.

Limitations of this work

With hindsight it is clear that improvements in the design of the studies conducted for this thesis could have been made, in order to meet the methodological criticisms outlined in the previous chapters. This would have strengthened the findings and the overall conclusions. Among the most obvious weaknesses are:

1) Failure to assess hunger or satiety by using some form of hunger scale measure. Despite the fact that hunger ratings do not always have a strong correlation with food intake it still seems useful to have some measure of hunger.

2) Mood could have been assessed more frequently, perhaps using some other self-report test with fewer scales along with or in place of the Profile of Mood States.

3) Although no physiological measures were taken in any of these experiments, it is intended that in future studies a more elaborate design will be formed which will incorporate some of the measures omitted in these studies.

Clearly, by improving the methodology used this would be more useful in the study of human eating behaviour and mood, in that a greater understanding of the processes involved would be sought.

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Appendix 1: Written Instructions in the Experiments

Written Instructions for the Pilot Study and Experiment 1 and 2.

1. There will be an initial interview one week prior to the actual experiment where your height and weight will be recorded and you will be requested to fill out two questionnaires. The first one will measure your mood and the second will assess your eating behaviour.

2. You will also be issued a food diary which we require you to fill out for one whole week before participating in the experiment. You will be given 10-15 minutes training and detailed example sheets of how we expect the diary to be filled in will be provided. Please complete this diary as accurately as possible and follow the instructions and guidelines provided by the experimenter. However, although we require accurate information about the type and quantity of food eaten please do not adjust your food intake because you are keeping a record. We want facts about your everyday diet not the perfect diet!

3. For this experiment you are asked to refrain from eating overnight and miss breakfast on the morning of the experiment.

4. You will be asked to fill out several questionnaires to assess your mood on the morning of the experiment which will take place at 09.00h

5. After completing the experiment a new 24h food diary will be issued which we require you to fill out for the remainder of the day and which you should post on to us after completion (postage paid).

6. You will be debriefed in writing about the experiment and feedback will be provided on the questionnaire assessing eating behaviour.

Please check that you have understood these instructions. If there is any further information which you require, please feel free to ask the experimenter.

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Written Instructions for Experiment 3.

1. There will be an initial interview one week prior to the actual experiment where your height and weight will be recorded and you will be requested to fill out two questionnaires. The first one will measure your mood and the second will assess your eating behaviour.

2. You will also be issued a food diary which we require you to fill out for one whole week before participating in the experiment. You will be given 10-15 minutes training and detailed example sheets of how we expect the diary to be filled in will be provided. Please complete this diary as accurately as possible and follow the instructions and guidelines provided by the experimenter. However, although we require accurate information about the type and quantity of food eaten please do not adjust your food intake because you are keeping a record. We want facts about your everyday diet not the perfect diet!

3. On the morning of the experiment you are expected to eat as normal before participating in the experiment.

4. You will be asked to fill out several questionnaires to assess your mood on the morning of the experiment which will take place at 11.00h

5. After completing the experiment a new 24h food diary will be issued which we require you to fill out for the remainder of the day and which you should post on to us after completion (postage paid).

6. You will be debriefed in writing about the experiment and feedback will be provided on the questionnaire assessing eating behaviour and on your food diary if required.

Please check that you have understood these instructions. If there is any further information which you require, please feel free to ask the experimenter.

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