Proceedings of the Nutrition Society (2002), **61**, 497–507 DOI:10.1079/PNS2002187

The physiological–psychological dichotomy in the study of food intake

Marion M. Hetherington
Department of Psychology, University of Liverpool, Eleanor Rathbone Building, Liverpool L69 7ZA, UK

Human food intake is driven by necessity. We eat to live, but as Brillat-Savarin and others have noted throughout history, in affluent societies eating is a pleasure and becomes more than a means to an end.Eating signifies lifestyle choice and it has considerable meaning in our society beyond the acquisition of essential energy and nutrients. Thus, it is that the study of human food intake, particularly food choice, in contrast to food intake in other animals, tends to be skewed towards measures of behavioural, social and environmental influences rather than on precise physiological processes reflecting metabolism and nutrient partitioning. The dichotomy between physiological and psychological measures is a false one, since all behaviours are necessarily expressed through physiological systems. However, in the field of human food intake research the dichotomy refers to the divergent strands of interest in either psychological or physiological processes underlying intake and appetite. The present review considers both psychological and physiological measures in promoting our understanding of the human appetite system. The overall conclusion is that the burgeoning interest in identifying appetite suppressant drugs to combat obesity and in genotyping alongside behavioural phenotyping will close the gap between psychological and physiological perspectives on human food intake.

Appetite: Food preference: Pleasure: Meal size: Methodology

A recent episode of the venerable 'Food Programme' on BBC Radio 4 timed precisely for Valentine's Day discussed 'Foods of Seduction', featuring the question of whether aphrodisiac foods influence the mind or the body. This question illustrates the still popular, Cartesian view of mind and body as separate entities. Mass media is littered with examples of dualism, and yet mainstream scientists, including behavioural scientists interested in the factors influencing food intake, behave in the laboratory as determinists, i.e. by understanding the principles of physiology, all behaviour can be explained (Carlson, 2000).

The scientific history of ingestive behaviour research can be traced from Descartes in the 17th century, with his first physiological model of behaviour, to European physiologists such as Claude Bernard in the 19th century, describing the principles of homeostasis, and from the American pioneer Walter Cannon, who conducted the first empirical investigation of eating in human subjects in 1912, to Curt Richter, who first characterised feeding in rats in 1922 (for a concise review of the American tradition, see Smith, 1997). Scientific determinism seeks to unravel general laws (nomothetic rules) in order to predict the behaviour of complex systems whilst taking into account individual variation (idiographic principles). Clearly, the complexity of the human brain makes the task of applying general rules to the prediction of even quite straightforward behaviours such as eating an enormously difficult one. Some scientists might say it is an impossible task. Nevertheless, the discovery in the late 20th century of the hormone leptin has encouraged a wealth of experimentation on the signalling pathways between the brain and adipose tissue (see Blundell *et al*. 2001; Trayhurn, 2001) providing new insights into the long-term regulation of body weight and short-term control of appetite.

In their first study of gastric contractions and the experience of hunger, Cannon & Washburn (1912) demonstrated several crucial features of the psychobiological approach to human appetite. The first feature was to record subjective sensations alongside physiological monitoring in order to extrapolate general characteristics of the experience of hunger. Thus, Cannon involved a medical student and

***Corresponding author:** Professor Marion M. Hetherington, fax +44 151 794 2945, email m.m.hetherington@liverpool.ac.uk

assistant, Washburn, as a subject who swallowed a small rubber tube as far as the stomach, with a balloon attached which could be inflated and subsequently pulled upwards to the fundus. This procedure enabled Cannon to introduce a water manometer into the balloon and to record pressure changes caused by gastric contractions. Washburn registered the experience of hunger by pressing a key, and this action was compared against the frequency and magnitude of gastric contractions. Self-reported experience of the sensation of hunger was recorded simultaneously with pressure changes in the stomach. The second essential principle Cannon demonstrated in this approach was to take account of individual differences by comparing hunger pangs experienced by Cannon during a 20 min period with that of his student. Although there are several weaknesses in this study, not least of which are demand characteristics where both subjects knew exactly the objective of the work, this report remains a classic in demonstrating the interrelationship between behavioural and physiological measures in identifying controls of food intake. Here there is no clear dichotomy, self-report of hunger is assumed to reflect the experience of an empty and contracting stomach.

Investigations of food intake by modern behavioural scientists still apply the basic principles illustrated by Cannon 90 years ago (Cannon & Washburn, 1912), coupling objective measures of gastric function (e.g. emptying or capacity) with reports of sensations (e.g. fullness, discomfort) with the additional behavioural measure of food intake (for review, see French & Cecil, 2001). Cannon's original research examined the 'concomitance of contractions and hunger in man' (Cannon & Washburn, 1912), measuring both physical changes in the stomach and the simultaneous interoceptive experience of those changes, suggesting an inductive approach to the research by generating theoretical inferences from these observations. Most current research approaches to ingestive behaviour follow a hypothetico-deductive model to test specific theoretical accounts of the controls of food intake (for example, see Smith, 1996). These approaches can be categorised into scientific traditions adopting experimental or correlational strategies (Blundell, 1975). Experimental strategies seek to change physiological or psychological systems and measure the consequences of that change. Investigations of pharmacological agents on food intake illustrate this method. Depending on the particular theoretical framework under scrutiny, a specific drug with known tissue or neurotransmitter targets is administered at different doses to the animal and the amount of food, or pattern of eating, is recorded relative to a placebo.

The neurotransmitter serotonin is thought to be involved in appetite regulation. Thus, administration of agents which inhibit serotonin re-uptake in the synapse of serotonergic neurons, such as D-fenfluramine (Vickers *et al*. 2001) or fluoxetine (Blundell, 1995), have been administered in a variety of animal species, including human subjects, in order to assess effects on the development of satiety (Halford, 2001), food intake (McGuirk *et al*. 1991) and binge eating in patients with bulimia nervosa (Walsh & Devlin, 1995).

Although the experimental approach rests on inference and observation, because the approach can be broken down into different levels of analysis from systemic to molecular, and across different psychological systems, the experimental approach offers the most powerful but most invasive strategy for understanding controls of food intake.

Correlational approaches, in contrast, compare changes in physiological variables occurring during some behavioural state or psychopathological disorder. For example, the correlational approach to assess serotonergic function in appetite regulation is exemplified by measuring metabolites of serotonin such as 5-hydroxyindoleacetic acid in the cerebrospinal fluid of patients with anorexia nervosa (Kaye, 1997). From this approach, serotonin activity is inferred from levels of 5-hydroxyindoleacetic acid for patients with known aberrations of hunger and appetite expression.

Correlational approaches are less powerful than experimental approaches insofar as the changes in 5-hydroxytryptamine binding or metabolites of 5-hydroxytryptamine in different patient groups represent associations between physiological and behavioural systems, but do not necessarily reflect a causal relationship. An example of this difference occurs in comparisons of gastric capacity in patients with bulimia nervosa, who binge and purge frequently, with that of obese and normal-weight controls (Geliebter & Hashim, 2001). Although gastric capacity, as measured by several different indices, is greater in bulimic and obese patients who binge eat relative to controls and those who do not binge eat, because binge size and frequency is related to gastric capacity (Geliebter *et al*. 1992), this relationship could suggest either that binge eating is caused by larger stomach size and lower sensitivity to satiety, or that binge eating causes the stomach to stretch, thereby increasing capacity and blunting satiety responses. It can only be inferred that the direction of causality is that episodic binges increase stomach size and promote further binge eating through diminished negative feedback from the stomach (Geliebter *et al*. 1992).

An alternative method employed by investigators to further our understanding of the systems that control appetite regulation is the application of genotyping to the characterisation of behavioural phenotypes. Sequencing the human genome offers unparalleled opportunities to link molecular and behavioural approaches in the discovery of the causes of human obesity and eating disorders. Efforts to identify single nucleotide polymorphisms or insertion and/or deletion sequence features in relation to the expression of appetite and energy metabolism are flourishing. Examples of these efforts can be found in studies of 5-hydroxytryptamine 2A promoter polymorphisms in susceptibility to the restricting subtype of anorexia nervosa (Nacmias *et al*. 1999), large-scale linkage studies identifying an anorexia nervosa susceptibility locus on chromosome 1p, again for the restricting subtype of anorexia nervosa (Grice *et al*. 2002), and the oestrogen receptor 2 gene in susceptibility to anorexia nervosa (Eastwood *et al*. 2002).

Much of the work on complex multifactorial traits, including obesity and eating disorders, is in its infancy, and the correspondence between genotype and phenotype is considerably less than that in monogenic traits (McCarthy, 2002). Nevertheless, identification of major susceptibility

Table 1. Examples of the psychobiological approach to ingestive behaviour

Approach	Psychological domain	Physiological domain	Reference
Correlational	Measure (e.g. hunger sensations)	Measure (e.g. gastric contractions)	Cannon & Washburn (1912)
Correlational	Measure (e.g. obsessional traits)	Measure (e.g. 5-HIAA in CSF)	Kaye (1997)
Experimental	Measure (e.g. food intake)	Manipulate (e.g. alcohol preload)	Hetherington *et al.* (2001)
Experimental	Manipulate (e.g. food odour)	Measure (e.g. brain activation)	O'Doherty *et al.* (2000)
Genotyping	Describe phenotype (e.g. anorexia nervosa restricting subtype)	Define genotype (e.g. 5-HT2A promotor polymorphisms)	Nacmias *et al.* (1999)

5-HIAA, 5-hydroxyindoleacetic acid; CSF, cerebrospinal fluid; 5-HT, 5-hydroxytryptamine.

genes in advancing our knowledge of the molecular basis of human behaviour is gaining momentum, and it is likely to change the scientific approach not only to the study of health but also to the basic principles of human biology.

The psychobiological approach to ingestive behaviour is summarised in Table 1, giving examples of correlational approaches through to genotyping.

As an adjunct to the advances in genotyping, the role of behavioural scientists in providing tools to characterise and measure specific features of appetite regulation, food intake, choice and preference has become increasingly important. Given the complexity of human ingestive behaviour, and the number and types of competing influences on intake, it is parsimonious to identify specific components of the behaviour and to examine the methods applied to measure each component before attempting to integrate each element into the whole. In the remainder of the present review it is assumed that the study of human food intake does not involve a dichotomy between physiological and psychological variables beyond the age-old mind–body dilemma. Whereas the physiologist is interested in characterising systems such as those involved in energy regulation, the psychologist shares this interest but adopts a different path to its understanding. Psychological approaches to the study of human food intake place an emphasis on behaviour as it is expressed in self-report, sensory or hedonic judgements and cognition (learning, memory, beliefs, attitudes, knowledge). It is assumed that psychological processes reveal elements of the underlying physiological system.

Verbal reports as data

Before embarking on a consideration of the methods applied to the study of human food intake, it is crucial that the putative dichotomy between psychological and physiological processes is tackled. When Cannon asked Washburn to report on the experience of hunger during his experiment on recording gastric contractions, he made the assumption that verbal reports from his subject constituted valid data. In the same way behavioural scientists treat verbal and written responses as veridical in clinical and laboratory contexts. Reliance only on verbal report can be problematic, since the accuracy of the report cannot be easily ascertained. (Radical changes in psychology, particularly social psychology, have seen an emphasis on qualitative research methods, with some psychologists abandoning quantitative methods entirely; for a qualitative analysis of eating in a family context, see Wiggins *et al.* 2001). For instance, when patients or volunteers in a research study are instructed to weigh all foods and fluids consumed for a diet record, unless the investigator has another means of verifying the record (e.g. covert observation of the patient or volunteer), the data is treated as a true reflection of food intake. A physiological index of energy balance can be used to gauge accuracy of the record by comparing energy expenditure from indirect calorimetry or doubly-labelled water against reported intake. In this way, evidence of accurate, under- or over-reporting can be established. Reasons for under-reporting may vary from simple forgetfulness to intentional deception (Muhlheim *et al.* 1998).

Herein lies another problem, it is assumed that indirect calorimetry, and indeed other physiological variables, are necessarily more accurate than verbal or written reports. The physiological variable is taken as more reliable than the volunteer's report, which can be more easily discredited. Even the most reliable procedures to assess energy expenditure, such as doubly-labelled water techniques are subject to some error (Speakman *et al.* 1993; Goran *et al.* 1994). In order to judge the accuracy of the written report, it is often overlooked that the measurement of 'hard' physiological data can be indirect, subject to variation and error, and at times reliant on inference and judgement. Although diet records have been labelled as 'flawed data' (Black & Cole, 2001) because of the discrepancy between reported energy intake and BMR, until a precise biological marker of how much energy and in what form that energy was consumed is discovered, verbal and written reports will continue to be used.

Certainly, from a behavioural scientist's perspective, it is rather interesting and important to understand which macronutrients or types of foods are under-reported (Poppitt *et al.* 1998), why some consumers consistently under- or over-report (Black & Cole, 2001), and what circumstances induce under-reporting (Goris *et al.* 2000). An elegant example of combining self-report and biomarkers of energy balance is illustrated by Vuckovic *et al.* (2000), whose volunteers underwent a 10 d doubly-labelled water protocol to measure total energy expenditure, a 7 d written diet record and a food-frequency questionnaire. This group found through focus groups that volunteers identified two main factors that influenced their self-report, namely honesty *v.* social acceptance and simplifying food intake for the record. It is evident that even if volunteers report their energy intake with accuracy, they may have changed their diet substantially as a function of keeping the record.

Introspective reports gathered by subjective ratings, or in the form of diet records or questionnaires, should be regarded as complementary not competitive with physiological data.

https://doi.org/10.1079/PNS2002187 Published online by Cambridge University Press

Weingarten & Gowans (1991) once described the meal as a 'stream of sensations', and to access these sensations, the great advantage of working with human subjects is their capacity to engage in introspection which, despite the difficulties, enhances and completes the picture. Ingestive behaviour is best understood by combining self-report data from ratings, records and questionnaires with more objective measures of energy intake, food selection and physiological measures of appetite and metabolic status. Above all, since food intake in human subjects is complicated by its capacity to be influenced by a range of factors beyond the basic biological need for fuel, it is incumbent on researchers to utilise all measures and methods at their disposal including self-report.

The test meal

The meal is central to our understanding of ingestive behaviour. Eating occurs in bouts, as meals or snacks following periods of deprivation, and these episodes tend to occur at particular times of the day. The size and macronutrient composition of this meal predicts the timing and size of the next meal (Blundell, 1995). In adults, the inter-meal interval is determined by the previous meal (postprandial pattern), whereas in young babies, who are depletion-driven (Weingarten, 1985), the size of a meal is determined by the duration of the period of depletion (larger meals follow longer periods of deprivation). Thus, in adults, eating is likely to occur when the inhibitory effects of the previous meal have dissipated (Rogers, 1999) rather than as a function of deprivation. Indeed, eating in some children and adults can be initiated immediately following a large meal (Cornell *et al*. 1989; Birch & Davison, 2001).

In measuring food intake it is important to know when and what the previous meal was, or to manipulate the state of deprivation systematically by imposing periods of deprivation and providing fixed meals or snacks (preloads) in advance of the test meal (Hetherington & Rolls, 1987). Having controlled for previous intake, the test meal is the typical unit of measurement in laboratory contexts. Test meals can vary from large portions of a single food item (Kissileff *et al*. 1980), a buffet-style array of foods (Porikos *et al*. 1980; Rolls *et al*. 1998, 1999), vending machines with multiple snacks (Silverstone *et al*. 1980) and liquid or semi-solid food dispensers (Jordan *et al*. 1966; Owen *et al*. 1985).

The choice of test meal is far from arbitrary. Deciding quantities and types of foods to offer in a test meal depends on the theoretical model under scrutiny, but it is essential that the investigator gives due consideration to how and what food is presented. For example, children (Rolls *et al*. 2000) and adult consumers are influenced by portion size (Engell *et al*. 1995). When male volunteers came to the laboratory on three occasions to eat a small (450 g), medium (620 g) or large (790 g) portion of macaroni cheese in random order, there was a linear relationship between portion size and intake. Similarly 5-year-old children responded in the same way to increasing portion size (Rolls *et al*. 2000).

Eating alone or in a group is a powerful determinant of how much is eaten, with estimates of the social enhancement of eating at about 40 % in some cases (de Castro, 1997). Eating with familiar others or with strangers also has an impact on the amount eaten (Shide & Rolls, 1991; de Castro, 1994). In contrast, giving too little food can reduce the sensitivity of the test meal as an index of appetite, since some consumers will 'clean the plate' (Krassner *et al*. 1979).

Pattern of eating

The microstructure of eating during a meal includes meal duration, speed of eating, bite size and frequency, number and duration of chews, and swallowing. The precise pattern of eating can reveal components of the motivation to eat and aberrant eating patterns, and may reflect palatability. Microstructure can be measured in a number of ways: by continuous assessment of eating during a meal using the universal eating monitor (Kissileff *et al*. 1980, Westerterp-Plantenga, 2000; Yeomans, 2000); analysing videotaped eating episodes (Hetherington *et al*. 1993; Tappe *et al*. 1998); the application of telemetric methods such as the edogram (Bellisle & LeMagnen, 1980; Bellisle *et al*. 2000).

The universal eating monitor permits continuous measurement of food intake during a single course meal, coupled with ratings of hunger and food pleasantness (Yeomans, 2000). Yeomans *et al*. (1997) demonstrated that manipulating the flavour of food increases food intake by stimulating reported appetite and increasing the rate of eating. Volunteers were asked to eat from a bowl of food placed on a hidden scale, which was attached to a computer. The consumer paused at intervals during continuous assessment of intake to make subjective ratings. Meals that are interrupted in this way tend to be longer in duration and larger than meals that are uninterrupted (Yeomans *et al*. 1997). Thus, asking subjects to stop eating and make ratings extends the meal and stimulates further eating. Another consideration in adopting this method is that the technique is more suited to measuring intake of a single liquid or semi-solid meal. The advantage of videotaping is that the investigator can map several features of the microstructure of eating, and can record food choice (alternation between multiple items) as well as affect during eating (Hetherington *et al*. 1993). Coding videotapes is, however, extremely difficult and time consuming. Even a clearly structured coding system such as the one developed by Wilson *et al*. (1989) for the eating behaviour rating scale, or that developed by Tappe *et al*. (1998) which counts the frequency of ingestive and non-ingestive behaviours, can provide a relatively crude overview of the meal compared with the fine-detailed analysis of the universal eating monitor.

Beyond the test meal

There are large variations in meal size within a single day. Despite this variation body weight tends to be maintained within a stable range during long periods of adult life (Flatt, 1998). This finding suggests that short-term energy intake (e.g. within a single day), although regulated, is rather weakly controlled relative to long-term energy regulation. Short-term energy intake is registered and integrated with long-term indicators of metabolic status (Smith, 1996). For

measurement purposes, it is important to record all meals and snacks proximal to the test meal, since the greatest impact of the experimental manipulation may be experienced before, during or after the test meal. Investigators record meals and snacks beyond the test meal either using diet records or by providing laboratory meals later in the day and/or the next day (Bell & Rolls, 2001).

Using 7 d food diaries, de Castro (1998) has reported that daily energy intake is regulated following a 2 d delay, such that energy intake on 1 d correlates negatively with intake on the next day, but has a stronger, negative association with the day after. A similar 2 d delay was found for macronutrient intake. de Castro (2000) has reviewed the utility of applying the diet-diary technique to the study of food intake, and describes the method as imperfect but sensitive to a number of influences from environmental to genetic factors.

Given the difficulties of relying on diet records, some studies take place entirely within residential laboratory settings (Foltin *et al*. 1996; Westerterp-Plantenga *et al*. 2002). These facilities offer maximal control over the amount and macronutrient profile of foods available to participants, and enable investigators to manipulate and measure a range of variables involved in appetite regulation, including substrate utilization and energy expenditure (Stubbs *et al*. 1995*a,b*). Several possible drawbacks with this methodology include all those typically associated with laboratory studies (for review, see Meiselman, 1992), e.g. the artificiality of the setting, limited choice of foods, the effects on intake of being observed and the additional issue of the effects of being confined to a small space for long periods. The conundrum for the investigator is to balance the artificiality and limits of the laboratory setting, whether residential or not, against the problems of diet records or recall.

Readiness to eat

Assuming that the investigator has controlled the energy and macronutrient content of the previous meal and the number of hours of deprivation since that meal, and is interested in monitoring preparation to eat, there are a number of behavioural and physiological indices that reflect this state. A series of studies conducted by Campfield and colleagues (Campfield *et al*. 1996; Melanson *et al*. 1999) transferred the continuous monitoring of blood glucose before meal initiation in rats to a paradigm for human subjects. The rationale for this work is based on the crucial role played by carbohydrate metabolism in the regulation of food intake (Mayer, 1953). Carbohydrate is a vital fuel for the central nervous system, and its utilization and storage are tightly regulated (Stubbs, 1999). Thus, the experience of hunger and efforts to initiate eating may be linked to changes in circulating blood glucose. To investigate the relationship between blood glucose dynamics and initiation of eating, Campfield *et al*. (1996) conducted two experiments: the first to examine changes in blood glucose in relation to ratings of desire to eat and requests for a meal in eighteen volunteers; the second to characterise this association following administration of insulin in five volunteers. The first experiment revealed a close correspondence between transient decreases in blood glucose (a drop of 10 % below baseline) and both changes in reported hunger and verbal meal requests. The second experiment demonstrated an increase in hunger following an insulin-induced reduction in blood glucose. This paradigm elegantly demonstrates the utility of combining physiological variables of metabolism, ratings of hunger and desire to eat and verbal requests to eat.

Another index of readiness to eat is the cephalic-phase response. The paradigm for measuring preparation to eat is measured in hungry subjects who are either exposed to the sight, smell and taste of foods or are asked to taste, chew and expectorate a meal (modified 'sham-feed'). In preparation for consuming and digesting this food, salivation (Mattes, 2000), increased heart rate (Nederkoorn *et al*. 2000), secretion of hormones including insulin and pancreatic polypeptide (Teff, 2000) and thermogenesis (LeBlanc, 2000) are observed. Cephalic phase salivation has been linked to hunger state (Wooley & Wooley, 1981) and has been used as an index of palatability, with higher salivary responses reflecting greater hunger and palatability respectively. Generally, salivation is greater in response to food than non-food odours (Nederkoorn *et al*. 2001). In their experiment to compare three different methods of measuring salivary response (dental rolls, electrophysiological recording of swallowing and parotid gland activity) Nederkoorn *et al*. (2001) exposed subjects to chocolate, lemon, lasagne and wood. Swallowing frequency corresponded well with saliva collection via dental rolls, with the most marked increase in saliva recorded following lemon (using both techniques) and the least response recorded with wood chips. It is interesting to note that lemon odour or the taste of lemon juice typically elicits strong salivary responses across experiments, but other foods tend to elicit weaker effects. This finding could be explained in two ways. The first explanation proposed by Lee & Linden (1991) is that salivation may reflect irritation by the stimulus. They compared salivation in response to six pleasant odours: chocolate, vanilla, peppermint, beef, tomato and lemon juice, finding that only the lemon juice stimulated salivation. They subsequently tested subjects using increasing concentrations of chocolate, citric acid and lemon juice, and found again that only citric acid and lemon juice increased salivary flow. They concluded that lemon juice is an irritant and this property causes salivation rather than a cephalic-phase reflex. The second possible explanation noted by Mattes (2000) is that another function of saliva is to dispose of substances that are difficult to clear or are undesirable. Thus, salivation may occur for highly-desirable foods, but may also occur for unpalatable items.

Salivation as an index of readiness to eat may be best conceptualised in the paradigm developed by Epstein and his colleagues (Epstein *et al*. 1992; Wisniewski *et al*. 1992), in which salivary habituation to repeated exposure of a food stimulus signals reduced attention to that food. Introducing another stimulus, whether a food or non-food stimulus, dishabituates the salivary response (Epstein *et al*. 1997), again suggesting that salivary response may reflect interest in the stimulus and is sensitive to novelty. The association between habituation and interest in food is explored further in relation to pleasure and sensory-specific satiety.

https://doi.org/10.1079/PNS2002187 Published online by Cambridge University Press

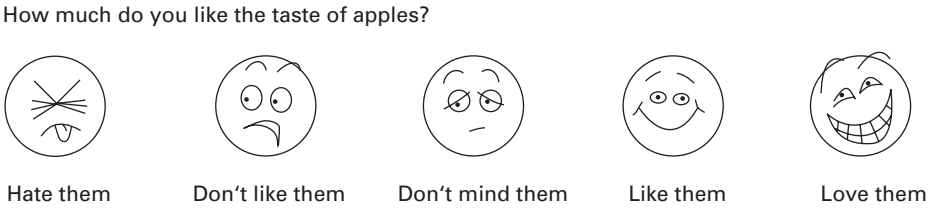

Fig. 1. Facial hedonic rating scale modified from that used by Birch *et al.* (1980).

Acceptance and preference

Consumers acquire a liking for a particular range of foods, they select these foods from those made available, they learn to eat at certain times of the day and the amount they eat will depend on signals associated with the food itself, as well as cues arising from metabolic status. How do consumers know which foods to select from an array of items, and how do scientists measure liking and preference for different foods? Infants are born with an 'innate' liking for sweet solutions, first demonstrated by Steiner (1977) and then replicated and extended to non-human primates using techniques developed by Kent Berridge (Steiner *et al.* 2001). In this investigation Steiner *et al.* (2001) filmed the facial responses of subjects following systematic administration of water compared with sweet, sour and bitter solutions. The tapes were then analysed for affective reactions, indicating that there was a positive hedonic response to sucrose and a generally aversive response to quinine (bitter) across all primate subjects. Apart from this hard-wired phylogentically-old acceptance of sweet and rejection of bitter tastes, consumers acquire preferences for complex foods by associating the orosensory properties of the food with post-ingestive consequences (Birch *et al.* 1987). If a food is tasted and the immediate response is not aversive, that food is consumed, and if the consumer then experiences positive (feelings of satisfaction, provision of energy) rather than a negative (nausea, vomiting) consequences, then that food is likely to be selected again. This observation is consistent with the 'learned safety' hypothesis predicting increased liking for orosensory stimuli that are repeatedly paired with a positive outcome.

Food aversions tend to occur when a particular food is associated with sickness, even when the illness is unconnected with intake of the food. A single aversive experience with a food is sufficient to produce a food aversion which may last many years (Bernstein, 1999). In contrast, acceptance of foods is generally acquired with repeated rather than single exposures. Birch & Fisher (1998) demonstrated increased acceptance of a novel fruit or vegetable in 4–7-month-old infants given the novel food every day for 10 d. Not only did the infants acquire a liking for the target food, but this effect was generalised to other similar foods. The method used to determine acceptance in young infants was to weigh how much of the target, similar or different foods was consumed during a 4 d pre-exposure period compared with a 5 d post-exposure session following the 10 d of repeated exposure to the target food. This study illustrates both acceptance and preference. Acceptance is defined as liking for a food, which is demonstrated in the amount of food consumed, with higher intakes reflecting higher liking. Preference is defined as liking for one food over another. In this case preference for the target food was quantified as the amount of the target consumed relative to similar or different foods. Acceptance is an absolute measure; the food is either accepted or rejected, liked or disliked, and the other is a relative measure of liking one food against another food.

In preschool children Birch (1981) examined acceptance and preference by asking two types of question. The first was to ask the child to taste a food or drink and to evaluate the food on a facial hedonic scale, where a smiling face represents liking the food or a 'yummy' food and a frowning face represents disliking or a 'yucky' food. A face in the middle with a neutral expression represents neither liking nor disliking the food. The second question addressed preference by asking the child to rank-order the items from least liked to most liked. Older children, who are more familiar with using symbols and words to represent objects and feelings, are able to use a more sensitive facial hedonic rating scale, and even a Likert scale (Hayes, 1994). We have recently examined acceptance and preference for fruit and vegetables as well as foods high in fat and/or carbohydrate in children aged 7–11 years following a whole-school intervention to increase intake of these foods (for details, see Anderson *et al.* 2001; Higgins *et al.* 2001). The scale (Fig. 1) was modified from the basic facial hedonic scales used by Birch *et al.* (1980) and by Moskowitz (1985) who described a nine-point Snoopy scale used in preference ratings by 5–7 year olds rating candies.

We assessed liking and ranked preference for twelve items, including fresh fruit (bananas, grapes and apples) and vegetables (carrots and tomatoes), fresh orange juice, cola, apple pie, chocolate, crisps, crackers and sweets at baseline, then 4 months into the intervention and again at 9 months of the intervention. The intervention was successful in increasing fruit intake by a small amount (Foster *et al.* 2001).

Liking for grapes and orange juice increased in the intervention group (n 68) compared with the controls (n 66), whilst ratings of vegetables remained unchanged (see Fig. 2). Reported liking for cola and chocolate declined in the intervention group relative to controls. We returned to one of the intervention schools at the end of the testing period and compared ratings on the facial hedonic rating scale with a Likert scale numbered from 1 to 5. The objective of this re-test session was to find out if younger children were using the entire range of the scale, or if they tended to

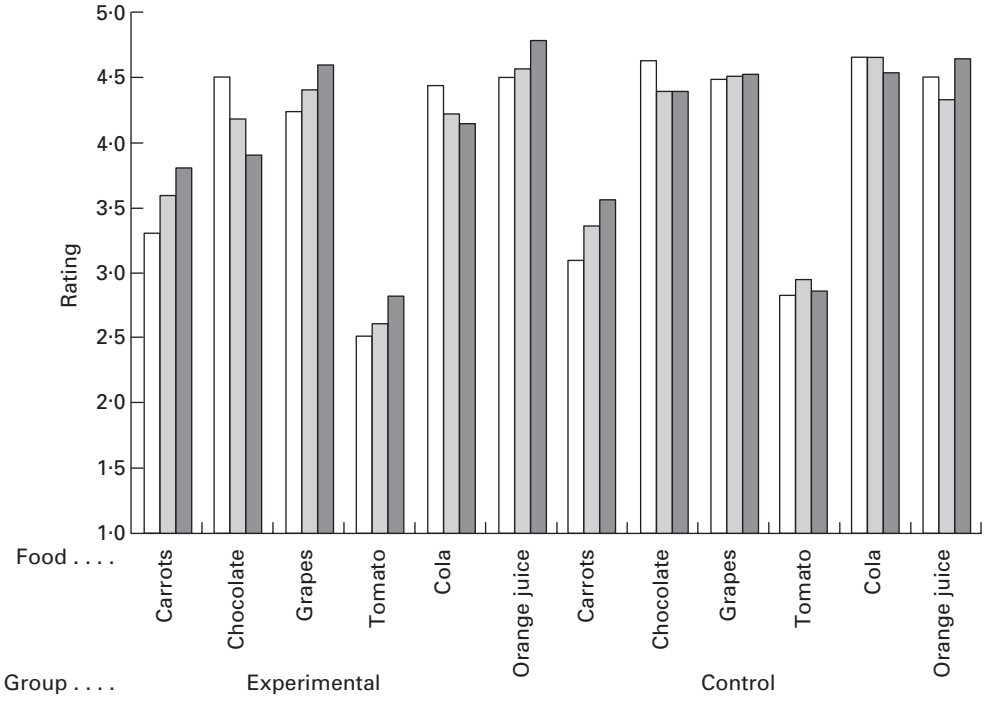

Fig. 2. Preference ratings for individual foods before (□), 4 months (▒) and 9 months (■) into a whole-school intervention to increase fruit and vegetable intake (Data from Anderson *et al.* 2001; Foster *et al.* 2001; Higgins *et al.* 2001.)

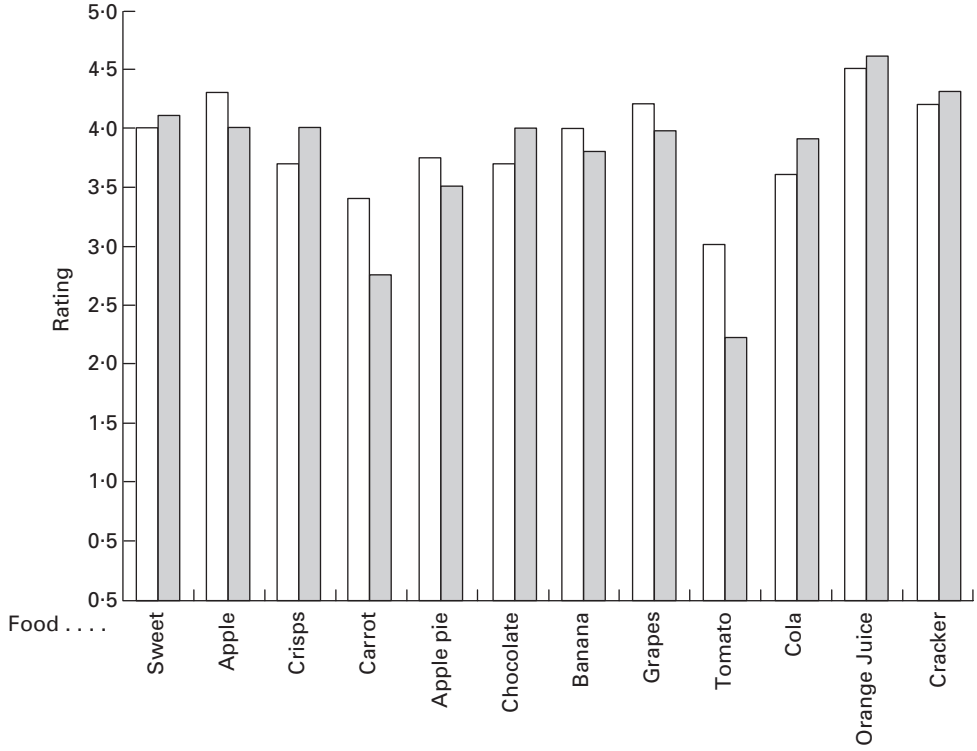

Fig. 3. Preference ratings for individual foods using the facial hedonic rating scale of Birch *et al.* (1980; □) or Likert scale of Hayes (1994; ▒). (Data from Anderson *et al.* 2001; Foster *et al.* 2001; Higgins *et al.* 2001.)

choose the end of the scale, with the 'smiley' face selectively. Children aged 7 years (n 14) completed the same preference test using a Likert scale. The correlations for individual food items were high ($P < 0{\cdot}01$), and no differences between ratings on the facial or Likert scale were found (see Fig. 3).

https://doi.org/10.1079/PNS2002187 Published online by Cambridge University Press

In this study the increased intake of fruit could be accounted for, in part, by an increased liking for some fruit items as a function of the intervention. This observation illustrates the power of combining measures of intake with taste preference assessment in understanding the expression of food acceptance and choice in young children.

Meal size

What are the determinants of how much is eaten within a test meal? Readiness to eat can be gauged in relation to blood glucose dynamics, salivation and self-reported hunger. Hunger ratings reflect the subjective experience of the need to eat. Appetite, as a reflection of the desirability of that particular food, can be measured by the rate of eating (Yeomans, 1996) and physiological variables associated with arousal, including salivation in response to that food, heart rate, heart-rate variability and skin conductance (Nederkoorn *et al*. 2000). Self-report variables of appetite include ratings of the desire to eat that food and ratings of the pleasantness of the appearance, smell, texture and taste of that food. The process of satiation determines when the meal will stop, and this process involves both negative feedback arising centrally (relating to a change in desire and liking for that food) and peripherally from the gut (e.g. gastric fill). Satiety is the state generated by the meal, and is measured in relation to the suppression of hunger and appetite until the next meal. Factors which contribute to satiation involve both direct and indirect controls of meal size (Smith, 1996).

The model of meal size proposed by Smith (1996) distinguishes between direct controls which arise from direct stimulation by food onto pre-absorptive receptors along the gut from mouth to small intestine, and indirect controls which comprise all other environmental, metabolic, learned, hormonal, cognitive and other systems operating outside direct contact between food and gut receptors. Indirect controls modulate the potency of direct controls. This model advocates a particular set of measures that can be used to examine systematically the controls of meal size; for example, some index of direct contact between food and the gut, e.g. various concentrations of a sucrose solution, together with a measure of indirect control of meal size, e.g. depletion level. This model proposes that as a food is eaten signals originating in the mouth and gut interact with information generated by long-term components of metabolic status (fuel oxidation and body fat) and other indirect controls, these factors are then integrated centrally to determine meal size (Smith, 1996). Direct controls of meal size in human subjects, are represented in the 'stream of sensations' arising from the gut as the food is consumed, including sensory experience of tasting, chewing and swallowing food, since these sensory inputs are registered in the oral cavity. As the food is eaten positive and negative feedback signals are generated; the function of positive feedback is to promote eating and the function of negative feedback is to slow eating. Positive feedback is generated in the mouth by direct contact between food and pre-absorptive receptors, whereas negative feedback is generated in the mouth, stomach and small intestine (Smith, 1996). When the potency of negative feedback equates to the potency of positive feedback, the meal is terminated. A number of different methods have been used to characterise direct controls, amongst them sham-feeding in the animal (for review, see Smith, 2000) and modified sham-feeding in human subjects (for example, see Rolls & Rolls, 1997), where consumers see, smell, taste and chew, but don't swallow, the food. The measurement of indirect controls varies from precise monitoring of metabolism 'on-line' (e.g. blood glucose dynamics, insulin levels) to, for example, assessing fuel oxidation and metabolic rate as indicators of long-term metabolic status.

Typically, the potency of positive and negative feedback during meals in human subjects is measured by proxy, using ratings of how pleasant or how strong the desire to eat that food is during (for example, see Yeomans, 2000) or before, immediately and at intervals after the meal (for example, see Hetherington & Rolls, 1996). These methods provide an on-line appraisal of positive feedback during and after the meal and, together with the universal eating monitor, are sensitive to increased appetite ratings immediately after eating begins, indicating an 'appetiser' effect in the early stages of eating the meal (for a review, see Yeomans, 2000).

Pleasure you can measure

As the food is eaten, and before the food is fully digested and absorbed, consumers report a decline in the pleasantness of the sensory attributes of the food (Rolls *et al*. 1981). This phenomenon is thought to contribute to negative feedback and to the process of satiation. It has been termed sensory-specific satiety, since the appeal of the eaten food decreases, leaving the pleasantness of other foods unchanged or with a greater hedonic value and desirability (Hetherington *et al*. 1989). In a number of different studies of consumers across a wide age range, the pleasantness of the appearance, smell, texture and taste of a food declines relative to other foods that are tasted but not eaten (for a full consideration of this phenomenon, see Hetherington & Rolls, 1996). In essence, the pleasure derived from eating a particular food is high at the beginning of a meal and stimulates further eating, but as eating progresses the hedonic evaluation of the food's sensory properties declines towards the end of the meal, contributing to general satiation and specific satiety for that food. Even when food is chewed, but not swallowed, there is a decline in the pleasantness of the taste and smell of the eaten food compared with other uneaten foods (Rolls & Rolls, 1997). The phenomenon of sensory-specific satiety occurs in the absence of feedback from the lower gastrointestinal tract.

It is thought that the change in liking of a food during a meal reflects a decrease in interest in that food (attentional), a form of sensory habituation (Raynor & Epstein, 2001), and a reduction in the reward value of the food (Rolls & Rolls, 1997). Using functional magnetic resonance imaging technology, O'Doherty *et al*. (2000) reported a decrease in activation in the orbito-frontal cortex in response to the odour of a food eaten to satiety, whereas activation of this area remained unchanged in response to the odour of an uneaten food.

Recently, we have attempted to assess both the attentional component of sensory-specific satiety and the change in the

https://doi.org/10.1079/PNS2002187 Published online by Cambridge University Press

reward value of the food as it is eaten (Hetherington *et al*. 1997). To examine cognitive and affective mechanisms underlying sensory-specific satiety we examined event-related potentials before and after consumption of a meal. This methodology overcomes problems in interpreting self-report by offering a tool that measures brain potential indices of hedonic tone, fatigue, attention, anticipatory processes and sensitivity to novelty without relying exclusively on introspection.

A decrease in N100 amplitude, reflecting early sensory and attentional stages of processing, was found for the eaten food (cheese on cracker), but not the uneaten food (chocolate), during exposure to slide presentations of the foods. A correlation between the magnitude of change in N100 amplitude and decline in desire to eat the eaten food was found. This finding suggests an association between sensory-specific satiety, as recorded in subjective ratings and brain potentials, reflecting early sensory and attentional processing.

Improvements in imaging and refinements of this powerful technology to permit scanning during eating will allow considerable insight into central mechanisms involved in the direct controls of meal size and the processes of hunger, appetite and satiation. An example of this technology being harnessed is in the study of 5-hydroxytryptamine binding using single-photon emission tomography in binge-eating and non-binge-eating obese volunteers (Kuikka *et al*. 2001). Coupled with emerging studies of susceptibility genes for obesity, anorexia and bulimia nervosa, a comprehensive account is developing of the genetic, physiological and behavioural causes of these disorders.

The future of ingestive behaviour research

Current research on human ingestive behaviour is witnessing an important change in direction. Diverse influences on human eating generate various interests and research approaches. Perspectives on food intake and food choice from socio-cultural to psychobiological accounts necessarily split investigators into different camps. However, the development of clear, reliable and valid measures of each component of ingestive behaviour have increased our understanding of the controls of food intake. Such measures complement newly-emerging collaborations between behavioural and genetic scientists in an attempt to present a broad psychobiological explanation of human food intake. It is important to recognise that the ostensible dichotomy in psychological and physiological approaches to food intake arises from fluctuating and varied inputs to the decisions to eat certain foods in particular amounts, from a behavioural perspective, against how these behaviours contribute to energy balance. However, the seeming polarity between a loosely-organised short-term control of food intake and the long-term homeostatic relatively-precise control of body weight simply reflect different components of the same system. The dichotomy is better characterised as a 'mind–body' problem where the same enterprise is under investigation, with parallel methodologies applied to advance theoretical models to account for food intake.

The future will see the science of ingestive behaviour benefiting from the insights generated by investigators from social, psychological and biological specialisms, given the medical and scientific imperatives of understanding the control of food intake in combating both the increased prevalence of eating disorders and the global epidemic of obesity.

References

Anderson AS, Adamson A, Hetherington M, Foster E, Porteous L & Higgins C (2001) Results from a school-based nutrition education intervention aimed at increasing fruit and vegetable intake in primary-school aged children. *Proceedings of the Nutrition Society* **60**, 143A.

Bell EA & Rolls BJ (2001) Energy density of foods affects energy intake across multiple levels of fat content in lean and obese women. *American Journal of Clinical Nutrition* **73**, 1010–1018.

Bellisle F, Guy-Grand B & LeMagnen J (2000) Chewing and swallowing as indices of the stimulation to eat during meals in humans: effects revealed by the edogram method and video recordings. *Neuroscience and Biobehavioral Reviews* **24**, 223–228.

Bellisle F & LeMagnen J (1980) The analysis of human feeding patterns: the edogram. *Appetite* **1**, 141–150.

Bernstein IL (1999) Food aversion learning: a risk factor for nutritional problems in the elderly? *Physiology and Behavior* **66**, 199–201.

Birch LL (1981) Generalization of a modified food preference. *Child Development* **52**, 755–758.

Birch LL & Davison KK (2001) Family environmental factors influencing the developing behavioral controls of food intake and childhood overweight. *Psychiatric Clinics of North America* **48**, 893–907.

Birch LL & Fisher JO (1998) Development of eating behaviors among children and adolescents. *Pediatrics* **101**, 539–549.

Birch LL, McPhee L, Shoba BC, Pirok E & Steinberg L (1987) What kind of exposure reduces children's food neophobia? Looking *v*. tasting. *Appetite* **9**, 171–178.

Birch LL, Zimmerman SI & Hind H (1980) The influence of social-affective context on the formation of children's food preferences. *Child Development* **51**, 856–961.

Black AE & TJ Cole (2001) Biased over- or under-reporting is characteristic of individuals whether over time or by different assessment methods. *Journal of the American Dietetic Association* **101**, 70–80.

Blundell J (1975) Physiological Psychology. London: Methuen.

Blundell JE (1995) *The Psychobiological Approach to Appetite and Weight Control. Eating Disorders and Obesity: A Comprehensive Handbook*, pp. 13–20 [KD Brownell and CG Fairburn, editors]. New York: Guilford Press.

Blundell JE, Goodson S & Halford CC (2001) Regulation of appetite: role of leptin in signalling systems for drive and satiety. *International Journal of Obesity and Related Metabolic Disorders* **25**, Suppl. 1, S29–S34.

Campfield LA, Smith FJ, Rosenbaum M & Hirsch J (1996) Human eating: evidence for a physiological basis using a modified paradigm. *Neuroscience and Biobehavioral Reviews* **20**, 133–137.

Cannon WB & Washburn AL (1912) An explanation of hunger. *American Journal of Physiology* **29**, 441–454.

Carlson NR (2000) *Physiology of Behavior*. Boston, MA: Allyn and Bacon.

Cornell CE, Rodin J & Weingarten H (1989) Stimulus-induced eating when satiated. *Physiology and Behavior* **45**, 695–704.

de Castro JM (1994) Family and friends produce greater social facilitation of food intake than other companions. *Physiology and Behavior* **56**, 445–455.
de Castro JM (1997) Socio-cultural determinants of meal size and frequency. *British Journal of Nutrition* **77**, Suppl. 1, S39–S55.
de Castro JM (1998) Prior day's intake has macronutrient-specific delayed negative feedback effects on the spontaneous food intake of free-living humans. *Journal of Nutrition* **128**, 61–67.
de Castro JM (2000) Eating behavior: lessons from the real world of humans. *Nutrition* **16**, 800–813.
Eastwood H, Brown KM, Markovic D & Pieri LF (2002) Variation in the ESR1 and ESR2 genes and genetic susceptibility to anorexia nervosa. *Molecular Psychiatry* **7**, 86–89.
Engell DK, Kramer M, Zarin D, Birch L & Rolls B (1995) Effects of serving size on food intake in children and adults. *Obesity Research* Suppl. **3**, 381.
Epstein LH, Paluch R, Smith DS & Sayette M (1997) Allocation of attentional resources during habituation to food cues. *Psychophysiology* **34**, 59–64.
Epstein LH, Rodefer JS, Wisniewski L & Caggiula AR (1992) Habituation and dishabituation of human salivary response. *Physiology and Behavior* **51**, 945–950.
Flatt JP (1998) What do we most need to learn about food intake regulation? *Obesity Research* **6**, 307–310.
Foltin RW, Haney M, Comer CD & Fischman MW(1996) Effect of fluoxetine on food intake of humans living in a residential laboratory. *Appetite* **27**, 165–181.
Foster E, Adamson AJ, Porteous LEG, Higgins C, Hetherington MM & Anderson AS (2001) The consumption of fruits and vegetables by Scottish children of primary school age. *Proceedings of the Nutrition Society* **60**, 64A.
French SJ & Cecil JE (2001) Oral, gastric and intestinal influences on human feeding. *Physiology and Behavior* **74**, 729–734.
Geliebter A & Hashim SA (2001) Gastric capacity in normal, obese, and bulimic women. *Physiology and Behavior* **74**, 743–746.
Geliebter A, Melton PM, McCray RS, Gallaher DR, Gage D & Hashim SA (1992) Gastric capacity, gastric emptying, and test-meal intake in normal and bulimic women. *American Journal of Clinical Nutrition* **56** 656–661.
Goran MI, Poehlman ET & Danforth E Jr (1994) Experimental reliability of the doubly labeled water technique. *American Journal of Physiology* **266**, E510–E515.
Goris AH, Westerterp-Plantenga MS & Westerterp KR (2000) Undereating and underrecording of habitual food intake in obese men: selective underreporting of fat intake. *American Journal of Clinical Nutrition* **71**, 130–134.
Grice DE, Halmi KA, Fichter MM, Strober M, Woodside DB, Treasure JT, Kaplan AS, Magistretti PJ, Goldman D, Bulik CM, Kaye WH & Berrettini WH (2002) Evidence for a susceptibility gene for anorexia nervosa on chromosome 1. *American Journal of Human Genetics* **70**, 787–792.
Halford JC (2001) Pharmacology of appetite suppression; implication for the treatment of obesity. *Current Drug Targets* **2**, 353–370.
Hayes N (1994) Foundations of Psychology. London: Routledge.
Hetherington MM, Cameron F, Wallis DJ & Pirie LM (2001) Stimulation of appetite by alcohol. *Physiology and Behavior* **74**, 283–289.
Hetherington MM, Regan MF & Pirie L (1997) Use of brain potential indices to explore sensory-specific satiety. *Appetite* **29**, 398.
Hetherington M & Rolls BJ (1987) Methods of investigating human eating behavior. *Feeding and Drinking*, pp. 77–109 [FM Toates and NE Rowland, editors]. Amsterdam: Elsevier Science.
Hetherington MM & Rolls BJ (1996) Sensory-specific satiety: theoretical framework and central characteristics. *The Psychology of Eating*, pp. 267–290 [ED Capaldi, editor]. Washington, DC: American Psychological Association.
Hetherington M, Rolls BJ & Burley VJ (1989) The time course of sensory-specific satiety. *Appetite* **12**, 57–68.
Hetherington MM, Spalter AR, Bernat AS, Nelson ML & Gold PW (1993) Eating pathology in bulimia nervosa. *International Journal of Eating Disorders* **13**, 13–24.
Higgins C, Hetherington MM, Anderson AS, Porteous LEG, Foster E & Adamson AJ (2001) Children's understanding of fruits and vegetables – implications for nutrition education. *Proceedings of the Nutrition Society* **60**, 2A.
Jordan HA, Wieland WF, Zebley SP, Stellar E & Stunkard AJ (1966) Direct measurement of food intake in man: a method for the objective study of eating behavior. *Psychosomatic Medicine* **28**, 836–842.
Kaye WH (1997) Anorexia nervosa, obsessional behavior, and serotonin. *Psychopharmacology Bulletin* **33**, 335–344.
Kissileff HR, Klingsberg G & Van Itallie TB (1980) Universal eating monitor for continuous recording of solid or liquid consumption in man. *American Journal of Physiology* **238**, R14–R22.
Krassner HA, Brownell KD & Stunkard AJ (1979) Cleaning the plate, food left by overweight and normal weight persons. *Behaviour Research and Therapy* **17**, 155–156.
Kuikka JT, Tammela L, Karhunnen L, Rissanen A, Bergstrom KA, Naukkarinen H, Vanninen E, Karhu J, Lappalainen R, Repo-Tiihonen E, Tiihonen J & Uusitupa M (2001) Reduced serotonin transporter binding in binge eating women. *Psychopharmacology* **155**, 310–314.
LeBlanc J (2000) Nutritional implications of cephalic phase thermogenic responses. *Appetite* **34**, 214–216.
Lee VM & Linden RW (1991) An olfactory-parotid salivary reflex in humans? *Experimental Physiology* **76**, 347–355.
McCarthy MI (2002) Susceptibility gene discovery for common metabolic and endocrine traits. *Journal of Molecular Endocrinology* **28**, 1–17.
McGuirk J, Goodall E, Silverstone T & Willner P (1991) Differential effects of d-fenfluramine, l-fenfluramine and d-amphetamine on the microstructure of human eating behaviour. *Behavioral Pharmacology* **2**, 113–119.
Mattes RD (2000) Nutritional implications of the cephalic-phase salivary response. *Appetite* **34**, 177–183.
Mayer J (1953) Glucostatic mechanisms of the regulation of food intake. *New England Journal of Medicine* **249**, 13–16.
Meiselman HL (1992) Methodology and theory in human eating research. *Appetite* **19**, 49–55.
Melanson KJ, Westerterp-Plantenga MS, Campfield LA & Saris WHM (1999) Appetite and blood glucose profiles in humans after glycogen-depleting exercise. *Journal of Applied Physiology* **87**, 947–954.
Moskowitz H (1985) *New Directions for Product Testing and Sensory Analysis of Foods*. Westport, CT: Food and Nutrition Press.
Muhlheim LS, Allison DB, Heshka S & Heymsfield SB (1998) Do unsuccessful dieters intentionally underreport food intake? *International Journal of Eating Disorders* **24**, 259–266.
Nacmias B, Ricca V, Tedde A, Mezzani B, Rotella CM & Sorbi S (1999) 5-HT2A receptor gene polymorphisms in anorexia nervosa and bulimia nervosa. *Neuroscience Letters* **277**, 134–136.
Nederkoorn C, de Wit T, Smulders FT & Jansen A (2001) Experimental comparison of different techniques to measure saliva. *Appetite* **37**, 251–252.
Nederkoorn C, Smulders FT & Jansen A (2000) Cephalic phase responses, craving and food intake in normal subjects. *Appetite* **35**, 45–55.

O'Doherty J, Rolls ET, Francis S, Bowtell R, McGlone F, Kobal G, Renner B & Ahne G (2000) Sensory-specific satiety-related olfactory activation of the human orbitofrontal cortex. *Neuroreport* **11**, 399–403.

Owen WP, Halmi KA, Gibbs J & Smith GP (1985) Satiety responses in eating disorders. *Psychiatric Research* **19**, 279–284.

Poppitt SD, Swann D, Black AE & Prentice AM (1998) Assessment of selective under-reporting of food intake by both obese and non-obese women in a metabolic facility. *International Journal of Obesity and Related Metabolic Disorders* **22**, 303–311.

Porikos KP, Sullivan AC, McGhee B & Van Itallie TB (1980) An experimental model for assessing effects of anorectics on spontaneous food intake of obese subjects. *Clinical Pharmacology and Therapeutics* **27**, 815–822.

Raynor HA & Epstein H (2001) Dietary variety, energy regulation, and obesity. *Psychological Bulletin* **127**, 325–341.

Rogers PJ (1999) Eating habits and appetite control: a psychobiological perspective. *Proceedings of the Nutrition Society* **58**, 59–67.

Rolls BJ, Bell EA & Thorwart ML (1999) Water incorporated into a food but not served with a food decreases energy intake in lean women. *American Journal of Clinical Nutrition* **70**, 448–455.

Rolls BJ, Castellanos VH, Halford JC, Kilara A, Panyam D, Pelkman CL, Smith GP & Thorwart ML (1998) Volume of food consumed affects satiety in men. *American Journal of Clinical Nutrition* **67**, 1170–1177.

Rolls BJ, Engell D & Birch LL (2000) Serving portion size influences 5-year-old but not 3-year-old children's food intakes. *Journal of the American Dietetic Association* **100**, 232–234.

Rolls BJ, Rolls ET, Rowe EA & Sweeney K (1981) Sensory specific satiety in man. *Physiology and Behavior* **27**, 137–142.

Rolls ET & Rolls JH (1997) Olfactory sensory-specific satiety in humans. *Physiology and Behavior* **61**, 461–473.

Shide DJ & Rolls BJ (1991) Social facilitation of caloric intake in humans by friends but not by strangers. *International Journal of Obesity* **15**, Suppl. 3, 8.

Silverstone T, Fincham J & Brydon J (1980) A new technique for the continuous measurement of food intake in man. *American Journal of Clinical Nutrition* **33**, 1852–1855.

Smith GP (1996) The direct and indirect controls of meal size. *Neuroscience and Biobehavioral Reviews* **20**, 41–46.

Smith GP (1997) Eating and the American Zeitgeist. *Appetite* **29**, 191–200.

Smith GP (2000) The controls of eating, brain meanings of food stimuli. *Progress in Brain Research* **122**, 173–186.

Speakman JR, Nair KS & Goran MI (1993) Revised equations for calculating CO_2 production from doubly labeled water in humans. *American Journal of Physiology* **264**, E912–E917.

Steiner J (1977) Facial expressions of the neonate infant indicating the hedonics of food-related chemical stimuli. *Taste and Development: The Genesis of Sweet Preference*, pp. 176–189 [JM Weiffenbach, editor]. Washington, DC: US Government Printing Office.

Steiner JE, Glaser D, Hawilo ME & Berridge KC (2001) Comparative expression of hedonic impact, affective reactions to taste by human infants and other primates. *Neuroscience and Biobehavioral Reviews* **25**, 53–74.

Stubbs RJ (1999) Peripheral signals affecting food intake. *Nutrition* **15**, 614–625.

Stubbs RJ, Harbron CG, Murgatroyd PR & Prentice AM (1995*a*) Covert manipulation of dietary fat and energy density: effect on substrate flux and food intake in men eating ad libitum. *American Journal of Clinical Nutrition* **62**, 316–329.

Stubbs RJ, Ritz P, Coward WA & Prentice AM (1995*b*) Covert manipulation of the ratio of dietary fat to carbohydrate and energy density: effect on food intake and energy balance in free-living men eating ad libitum. *American Journal of Clinical Nutrition* **62**, 330–337.

Tappe KA, Gerberg SE, Shide DJ, Andersen AE & Rolls BJ (1998) Videotape assessment of changes in aberrant meal-time behaviors in anorexia nervosa after treatment. *Appetite* **30**, 171–184.

Teff K (2000) Nutritional implications of the cephalic-phase reflexes: endocrine responses. *Appetite* **34**, 206–213.

Trayhurn P (2001) Biology of leptin – its implications and consequences for the treatment of obesity. *International Journal of Obesity and Related Metabolic Disorders* **25** Suppl. 1, S26–S28.

Vickers SP, Dourish CT & Kennett GA (2001) Evidence that hypophagia induced by d-fenfluramine and d-norfenfluramine in the rat is mediated by 5-HT2C receptors. *Neuropharmacology* **41**, 200–209.

Vuckovic N, Ritenbaugh C, Taren DL & Tobar M (2000) A qualitative study of participants' experiences of dietary assessment. *Journal of the American Dietetic Association* **100**, 1023–1028.

Walsh BT & Devlin MJ (1995) Pharmacotherapy of bulimia nervosa and binge eating disorder. *Addictive Behaviors* **20**, 757–764.

Weingarten HP (1985) Stimulus control of eating, implications for a two-factor theory of hunger. *Appetite* **6**, 387–401.

Weingarten HP & Gowans SE (1991) Sensory control of eating: The meal as a stream of sensations. *Smell and Taste in Health and Disease*, pp. 381–389 [TV Getchell, RL Doty, LM Bartoshuk, C Pfaffmann and BP Halpern, editors]. New York: Raven Press.

Westerterp-Plantenga MS (2000) Eating behavior in humans, characterized by cumulative food intake curves – a review. *Neuroscience and Biobehavioral Reviews* **24**, 239–248.

Westerterp-Plantenga MS, van Marken Lichtenbelt WD, Strobbe H & Schrauwen P (2002) Energy metabolism in humans at a lowered ambient temperature. *European Journal of Clinical Nutrition* **56**, 288–296.

Wiggins S, Potter J & Wildsmith A (2001) Eating your words: discursive psychology and the reconstruction of eating practices. *Journal of Health Psychology* **6**, 5–15.

Wilson AJ, Touyz SW, Dunn SM & Beumont PJV (1989) The Eating Behavior Rating Scale (EBRS), a measure of eating pathology in anorexia nervosa. *International Journal of Eating Disorders* **8**, 583–592.

Wisniewski L, Epstein LH & Caggiula AR (1992) Effect of food change on consumption, hedonics, and salivation. *Physiology and Behavior* **52**, 21–26.

Wooley OW & Wooley SC (1981) Relationship of salivation in humans to deprivation, inhibition and the encephalization of hunger. *Appetite* **2**, 331–350.

Yeomans MR (1996) Palatability and the micro-structure of feeding in humans, the appetizer effect. *Appetite* **27**, 119–133.

Yeomans MR (2000) Rating changes over the course of meals: what do they tell us about motivation to eat? *Neuroscience and Biobehavioral Reviews* **24**, 249–259.

Yeomans MR, Gray RW, Mitchell CJ & True S (1997) Independent effects of palatability and within-meal pauses on intake and appetite ratings in human volunteers. *Appetite* **29**, 61–76.