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The contribution of behavioural science to nutrition: appetite control

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Abstract

Behaviour and nutrition are inextricably linked. The behaviour of eating is the agency through which nutrients enter the body and exert their effects on physiology, metabolism and health. It is therefore inevitable that the study of eating behaviour (or appetite in general) is essential to an understanding of the discipline of nutrition, and therefore to describing the ways in which nutrients can begin to exert their effects.

The fact that humans are omnivores, with the potential to eat a huge diversity of foods, clearly denotes the importance of behaviour for nutrition. The roles of culture and biology in determining what foods people put into their mouths highlights the centrality of food choice for nutrition. In turn, behavioural science has made a huge contribution to defining the mechanisms responsible for food choice. This scientific approach has also specified the roles of homeostatic and hedonic principles (and their interactions) in controlling the amount and type of food (nutrition) ingested. A substantial focus has been the investigation of the processes of satiation and satiety, with implications for understanding routes to overconsumption and obesity. All of these investigations have been incorporated within a generally accepted and well described behavioural science methodology that involves the application of objective scientific principles to the study of eating behaviour. This methodology has been heavily implicated in the search for commercially viable functional foods for satiety. In recent years, behavioural science has engaged with the fields of energy balance and physical activity; recognising that nutrient intake is not independent of nutrient utilisation. This approach has been fostered by the pervasive problem of obesity and by its dependence on the interaction between over nutrition and under activity.

The diversity of foods in the omnivore’s repertoire is matched only by the diversity of humans themselves. This diversity is reality, and a future track for behavioural science seems destined to lead to understanding and managing individual differences.

Behaviour and nutrition are inextricably linked

It is important to recognise that behaviour and nutrition occupy separate domains in the psychobiology of human functioning, but they are inextricably linked. The behaviour of eating is the agency through which nutrients enter the body and exert their effects on physiology and metabolism. This means that any factors that influence the behaviour of eating have the potential to influence the impact of nutrition on health.

As Rozin (1998) has pointed out 'Because behaviour is so central to nutrition, the behavioural sciences play an especially important role in the understanding of what we eat and why we eat it. The study of what is in food is extremely important, but all of this knowledge amounts to little if we cannot persuade people to eat what is good for them and to avoid what will do them harm'. However our knowledge of what is in food and how it affects the body is far more advanced than our understanding of what makes people eat some foods and not others, and what makes us start and stop eating at particular moments. The behaviours related to nutrition are extremely complicated; whilst we should not be daunted by this complexity in seeking understanding, we should be aware of what we are up against.

Humans are omnivores

The fact that humans are omnivores is of huge significance for both behaviour and nutrition. Humans are not restricted in their food habits to the same extent as herbivores or carnivores and, consequently, they are capable of consuming a huge range of nutritional materials. Humans are generalists rather than specialists. Of course this ability has been of enormous evolutionary significance and has enabled humans to colonise a wide variety of environments and habitats. Just as different groups of humans can exist on widely divergent types of foods (profiles of nutrition) in different parts of the world, so the patterns of behaviour that bring these nutrients into the mouth can differ widely. It can be appreciated that developing a science that encompasses such complexity is a daunting proposition. A science has therefore developed around a more restricted range of environments and behavioural types. Not surprisingly this science has focussed on the nutrition and behavioural types relevant to technologically industrialised societies in which we live and to the preoccupations of people living in these societies. Over the last 50 years, the issue of obesity has provided a dominant framework for understanding the intimate link between behaviour (appetite control) and nutrition. This is relevant when considering that behaviour can be seen as the agency that mediates in meeting two nutritional demands; namely, what to eat and how much to eat. Both are important for obesity. The problem of what to eat arises because of a combination of our omnivorous nature and the abundance of foods in the environment. This is the issue of food choice and involves the conscious or automatic selection among potential edible materials. Interestingly, this food choice is not strongly programmed biologically but is dependent upon factors such as geography, climate, religion, ethnicity, economics (price, affordability), social class and culture. The issue of how much to eat has always been conceptualised in regards to homeostatic principles of energy requirements of the body, with a stronger link to biology. This snapshot of behavioural science will concentrate on the most prominent ways in which these strands of appetite control have influenced nutrition over the last 50 years. This means that appetite control is a central feature of the behavioural science of nutrition. For theoretical and methodological reasons, appetite control can be divided into issues of food choice and satiety.

The enigma of food choice

The complexity of the issue of human food choice has been elegantly described by the many years of work of Paul Rozin, whose research has made a monumental contribution to this field and whose studies stand as a landmark. Rozin's behavioural science approach has first of all defined the problems by exposing human food selection as the interaction of biology, culture and individual experience (Rozin 1982). Later, the approach was extended to the analysis of contextual influences on food choice and acceptability (Rozin & Tuorila 1993). These studies described that choice depended on the context of food itself (whether it was a snack, a course or a full meal), as well as the non-food contextual features such as the label, package, colour of utensils and aspects of the surroundings. This type of work resonates with the recent approach of Spence (Spence & Youssef 2016) in relation to the chemistry of food. Other contextual effects include the role of expectancies and remembrances of food, which can be seen as antecedents of the work of Brunstrom (Brunstrom *et al.* 2008) and Higgs (Higgs 2005; Birch *et al.* 2003) on expectation and memory respectively. A further innovative contribution of Rozin's behavioural science are the studies on food avoidance and the development of feelings of disgust (Rozin *et al.* 1999). This analysis elucidates how an apparently irrational rejection of certain foods arises from the integration of learning and cognitions, and how such habits become embedded in the culture. The role of learning (conditioning, tolerance) remains a central pillar of Rozin's work and illustrates how psychological processes are instrumental in determining food preferences – including the fascinating issue of liking for apparently aversive flavours such as the burn of chilli peppers. A similar behavioural science approach to children's food preferences used by Leanne Birch has been fundamental in demonstrating how particular likes and dislikes develop according to learning principles (Birch *et al.* 2003).

Hedonic appetite control

As an addition to the complexity of human food preferences provided by the work of Rozin, a common perception about food choice is that it is dominated by the attribute of palatability. In simple terms this means that people eat for pleasure. Indeed there are strong logical and biological reasons why the pleasurable taste of food should influence preference and consumption, and it is clearly a major issue in the manufacture and appeal of food products in the commercial market. This introduces the field of food hedonics. Some extreme ideas in this area have been disseminated recently through books such as Kessler's 'The end of overeating' (Kessler 2010). Although the title is a complete misnomer, this book raised the idea that foods could be blessed with the quality of 'hyperpalatability', designed with a combination of manufactured tastes, textures and mouthfeel, and exert effects on brain neurotransmitters similar to (but much weaker than) the effects generated by drugs. These ideas gave public support to claims for the existence of food addiction. However, the application of critical reviews and analyses are now showing that this concept of food addiction lacks strong evidential support and is much different from drug addiction (*e.g.* Rogers 2011; Long *et al.* 2015; Rogers 2017). However, an important contribution of behaviour science in this area has been to demonstrate that, under controlled scientific conditions, specific tastes and flavours can exert matching effects on liking and consumption of foods (*e.g.* Yeomans 1998) and that these effects can be mediated by certain brain peptides (Yeomans & Gray 2002). This sensory science approach explains how the positive (hedonic) sensations generated by tastes can inexorably exert an influence over food choice. Indeed it is valuable to point out the many papers in recent years that highlight the significant role of sensory properties, such odour and taste quality, and food texture in moderating energy intake within meals (McCrickerd and Forde 2016; Chambers *et al.* 2015b)

A significant advance in the area of hedonics came about with the objective demonstration that in animals the notion of pleasure was not a unitary process (Berridge & Kringelbach 2008). Of course, in this area, the terms pleasure, reward and reinforcement have particular meanings and it is important to be semantically clear. However, a key distinction made concerns separate identities for 'liking' and 'wanting'. Liking is defined as a source of pleasure or reinforcement, whilst wanting is regarded as having a motivational component (technically referred to as incentive salience). It follows that a food that generates a combination of liking plus wanting would exert a strong influence over food choice. It is immediately apparent that a person can have a liking for a food but not want (to eat) that food at that particular time or place. Therefore, the distinction between liking and wanting is meaningful. Importantly, a procedure has been developed to simultaneously measure both liking and wanting for foods in humans (Finlayson *et al.* 2007). The procedure avoids semantic confusion by using a non-verbal technique to measure wanting and also incorporates a covert (non-conscious) element known as implicit wanting (Finlayson *et al.* 2008). With this procedure, food choice can be tracked to changes in liking or wanting independently or to combinations of both. As Mela (2006) has pointed out, this type of procedure allows a behavioural discrimination for foods that may underlay obesity and is a powerful device for investigating the level of risk associated with the consumption, and overconsumption, of certain foods and nutrients.

A further issue in this area concerns the ecology of food choice. The global diversity of diets across our planet should tell us that it is unlikely that any universal laws of food choice can ever be established. Within our current societies the vast range of food products available - reflected in the terms 'Cocacola-isation' or 'Tescopoly' (*e.g.* Simms 2012) – clearly remind us that behavioural science contributes not only to the choice of eating foods but also to the purchasing of foods in response to marketing and advertising. Before people can eat food it normally has to be purchased – and this indicates the contribution made by social scientists in understanding choice and eating behaviours.

The foods actually chosen are promoted to meet the requirements of the market as much as for the well-being of the consumer. In this area, the behavioural science of food choice has been derived from social scientists working in the field of behavioural economics, giving rise to the idea of the 'nudge' (Sunstein & Thaler 2008). The central concept of 'choice architecture' (Thaler *et al.* 2014) is primarily effective in determining what people buy (which they will later eat) and reminds us that the word consumer has a dual meaning. The power of the brand plus promotional sales strategies clearly demonstrates an impact of this other aspect of behavioural science on nutrition; and its importance should not be underestimated. Social scientists make various contributions to the diet that is selected and eaten, but it is a sobering thought that these selections are not always of positive benefit to individuals. In certain cases the procedures of social scientists have been exploited for the promotion of foods in the market place.

Homeostatic appetite control: the challenge of satiety

It is possible that the issue of satiety is the most heavily researched phenomenon in appetite control relevant to nutrition. It is conceived as being fundamental to the control over how much people eat and is therefore crucial in the attempt to understand food consumption (and over-consumption) underlying obesity and the gain of adipose tissue. In its simplest form, the issue of satiety is about the feeling of fullness and the suppression of hunger and eating. A formulation devised 30 years ago (Blundell *et al.* 1987) – called the 'Satiety Cascade' – created a framework for thinking about the problem. In fact this formulation identifies two distinct elements namely satiation and satiety. Satiation refers to the operation of those processes ongoing during an episode of eating (such as a meal) and which bring that episode to an end. Satiety refers to the inhibition of eating (and the suppression of hunger and augmentation of fullness) when an episode of consumption has ended. This is what people normally have in mind when they speak of satiety. In principle, the sequential operation of satiation and satiety influence the size and frequency of eating episodes – including the susceptibility to snacking between meals. Both of these processes are crucial for the control over the amount of food energy ingested. However, the two processes are not influenced equally by the same factors (De Graaf *et al.* 1999). The original model of the Satiety Cascade has been adapted and amended (Kringelbach 2004; Halford & Harrold 2012; Van Kleef *et al.* 2012).

A significant feature of the Satiety Cascade is the identification of different – but overlapping – psychological and physiological processes in the control of eating. These include physiological sensory factors arising from the smell and taste of food, psychological factors such as cognitions beliefs and expectations, and physiological factors in the stomach and other parts of the gastrointestinal tract. Much attention is focussed on the release of the gastrointestinal (GI) peptides such as ghrelin, CCK, PYY, GLP1 and others. Although these agents are often referred to as appetite peptides, it should be remembered that they all have other physiological functions concerning growth, metabolism or the management of nutrients through the GI tract. The Satiety Cascade has provided a rationale for thinking about the profiles of these peptides in relation to changes in amplitude of hunger and fullness and the amount of food eventually consumed (Gibbons *et al.* 2013). A recent development has been the use of behavioural science methods to measure the action of nutrients influencing small chain fatty acid (SCFA) receptors in the colon (Chambers *et al.* 2015a).

A major influence on thinking has been the effort to distinguish the relative strength of the influence on satiety of the macronutrients (Stubbs *et al.* 1995). These studies have suggested a hierarchy in the order of decreasing strength of protein, carbohydrate and fat (Stubbs 1995). This type of work has been influential in identifying the strong satiating properties of protein (Johnstone *et al.* 2008). The roles of the macronutrients together with effects of dietary fibre have formed the basis for the development of functional foods for satiety (Chambers *et al.* 2015b).

Measurement and methodology

One noticeable contribution of behavioural science to the study of nutrition has been the development of a widely used methodology and set of experimental procedures. At the centre of this methodology is a system called the 'preload - test meal paradigm'. This is a parsimonious experimental device in which a fixed amount of food (of known composition and structure) is obligatorily consumed, normally under strictly controlled conditions. The effect of this consumption (on the strength of satiety) is assessed by the amount of food freely consumed at an eating test following a fixed period of time. This rudimentary strategy has been used a countless number of times and has given rise to a substantial inventory of factors that influence the amount of food eaten. A strong addition to these studies has been the use of the visual analogue scale (VAS). Initially used for hunger (Silverstone 1976), the procedure was expanded to include four scales (Rogers & Blundell 1979) – hunger, fullness, desire to eat and prospective consumption - which have endured for almost 40 years. Sometimes the scales are summed to give an overall appetite score (*e.g*. Bellissimo *et al.* 2008). The applicability of these scales is based on their demonstrated validity (Flint *et al.* 2000; Stubbs *et al.* 2000b). Surprisingly, at least for some people, these scales are highly informative and they form part of a satiety tool box that is a central part of the methodology of appetite control (for review see Blundell *et al.* 2010). This summary statement by experts working in the field remains valid despite the conclusion reached by a recent poorly conducted review (Holt *et al.* 2017).

For obvious reasons, this type of methodology has been deployed within controlled environments such as a laboratory, research unit or clinic. The procedures are highly stylised and both the presentation and consumption of foods are carried out under carefully controlled conditions. Such scientific conditions constitute one of the requirements for the evaluation of the claims of satiety power for functional foods imposed by the European Food Safety Authority (EFSA). It should be pointed out that the structure of these procedures constitutes one of the advantages but also one of the limitations of this form of appetite methodology. The need for scientific precision in measurement means that the food tests are carried out in an unnatural eating environment, such as a laboratory, rather than in the home, restaurant or school. The question arises whether or not the recorded behaviour represents eating that would occur in more normal surroundings. This has been conceived as the laboratory vs. free-living dilemma. In the laboratory, measurement of eating is precise but not natural, whereas a home setting (with food intake measured by some form of self-report or recall) would be natural but much less precise. In the behavioural science of nutrition it is recognised – though not always admitted – that the outcomes of studies represent a compromise between precision and naturalness.

Energy density and portion size - dietary variables that influence behaviour

All features of foods (taste, texture, smell, palatability, amount, colour, variety) have the potential to influence food choice, the perception of hunger and eating itself. It appears obvious that the properties of foods exert a major influence over how much food energy will be consumed. In recent years the dietary variables of portion size and energy density have received attention because of their potential to lead people to overconsume more (food) energy than is either wished for or required, and therefore to cause weight gain or obesity. Because of the nature of these dietary variables, their actions will be exerted during the actual process of eating (rather than after consumption) and the effects are therefore on satiation rather than satiety. Portion size can be represented in a number of forms such as the size of an entire meal, the amount of an element within the meal, or the size of an individual unit of food that could be eaten either within or separate from a meal (such as a snack item). Although it is logical that portion size should be one factor contributing to overeating, the evidence from controlled laboratory studies (*e.g.* Rolls *et al.* 2002) is stronger than evidence from long-term field trials (*e.g.* French *et al.* 2014). However, the belief generated by behavioural science studies has been sufficiently convincing for health agencies and the food industry to take measures to reduce portion size as part of the UK government's Responsibility Deal (Knai *et al.* 2015).

Since portion size may be said to be a visible extrinsic property of foods, its effect on satiation is likely to be mediated through psychological processes which interfere with the cognitive judgement of what is an appropriate amount of an item or meal. However, whereas portion size is an overt cue, energy density is usually covert. It is assumed that any effect of portion size is exerted at a subconscious level. Portion size forms part of the 'choice architecture' and its effect can be regarded as a type of sub-conscious 'nudge', which could decrease, but more often increase, the amount eaten. Studies on portion size by social scientists have led to some counterintuitive, but also celebrated, experimental outcomes (*e.g.* Wansink & Kim 2005) – such as the demonstration that some US moviegoers consumed a large amount of stale popcorn when it was offered in large buckets. It is not clear that these research results have enhanced confidence in the role of portion size, or in the types of investigations carried out by certain experts in marketing and applied economics.

In contrast there is much broader agreement on the effects of energy density on appetite control, although its action is also mediated at the sub-conscious level. This is because energy density is heavily dependent on the macronutrient composition of foods, and most people are unaware of the nutritional composition of much of what is eaten and have a tendency to consume food based on weight or volume rather than on the nutrient composition (which is not easily perceived). Energy density, expressed as energy per unit of weight, is a property of every single food. It has been demonstrated by Stubbs *et al.* (2000a) that fat has the strongest positive relationship with energy density and the water content of foods has the strongest inverse association. The contribution of fat to the overconsumption of energy was experimentally demonstrated by Stubbs and others (Stubbs *et al.* 1995). This phenomenon was termed high-fat hyperphagia (Stubbs & Whybrow 2004) or passive overconsumption (Blundell & MacDiarmid 1997). In a long series of studies, the landmark work of Rolls (see Rolls 2017 in this issue) investigated energy density usually by manipulating diets by 25-30 g of fat per day (Ello-Martin *et al.* 2007). In a field trial, this mandatory reduction of the fat content of foods had the effect of reducing dietary fat of the diet by 37% and energy density by 19%, thereby achieving an obligatory 29% reduction of energy intake. Just as reducing the fat content of diets can reduce energy intake, so increasing the fat content raises energy density and increases energy intake. This is true in free-living studies when individuals are free to choose their own foods (see Fig. 2 in Hopkins *et al.* 2015). Since people do not exhibit any active drive to overconsume energy when exposed to (or when they sub-consciously choose) high energy dense foods, the process must be passive. Normally, people are not actively endeavouring to consume more energy; this is unwanted energy intake that happens as a consequence of the property of the foods chosen. **'**This passive overconsumption of energy leading to obesity is a predictable outcome of market economies predicated on consumption-based growth**'** and has been identified as a major contributor of the obesogenic environment on weight gain (Swinburn *et al.* 2011). Therefore, although much of the work on energy density has been carried out under laboratory conditions, the effect is potent under free-living conditions where, it has been argued, it can plausibly account for the different effects of fast-food diets compared with traditional African diets (Prentice & Jebb 2003).

It is noteworthy that the effects of fat and energy density are also observed in children. When energy density was raised by doubling the amount of fat in meals (but keeping protein and carbohydrate constant) a highly significant effect on energy intake was observed (Fisher *et al.* 2007). When the fat content was increased by approximately 100%, the energy density was raised by 40% and the children increased the energy consumed in a meal by one third. For the high energy dense meal, the fat intake was increased by 15 g and 30 g respectively for the standard and large portions. Interestingly, this effect of increased fat density on energy intake interacted additively with portion size to promote meal energy intake. This study indicated that for children a particularly damaging scenario would be to be served a large portion of a meal in which the energy density has been raised by a large increase in fat. This could easily be achieved in some fast food outlets (see above) or in some ready-to-eat take home meals.

A further extension of this diet-induced effect on behaviour can be seen in combinations of nutrients and tastes. Here, a potent combination is the sub-category of high energy dense foods comprising high fat and high sugar products. This category of food items exerts a particularly strong effect on consumption through actions on both explicit liking and implicit wanting. In this particular case, there is an active drive to eat these foods (induced by the potent combination of taste and texture) which is strongly apparent in binge eaters (Dalton & Finlayson 2014). The examples of energy density and portion size illustrate well how behavioural scientists have contributed to an understanding of the effect of dietary-based variables on eating behaviour.

Emerging issue: behavioural science of energy balance

Owing to the attention devoted to obesity in the field of nutrition for the last 20 years, a considerable discussion has been taking place regarding the relative contribution of the behaviour that delivers energy intake (food consumption) and the behaviour that produces energy expenditure (physical activity). It is widely accepted that obesity is a function of energy balance; however, the function is complex (Hall *et al.* 2011) and not simply an algebraic sum of the energy consumed in relation to the energy expended (*e.g.* Hamid 2012). The concept of energy balance as a set of kitchen scales is both incorrect and misleading. There is, of course, considerable evidence regarding an excess of consumed energy – by way of the processes documented in earlier parts of this review: injudicious food choices, weak satiety, hedonic attraction, high energy density and a combination of sensory factors and nutrient composition. Behavioural science has contributed to an understanding of how foods lead to a high intake of energy with little or no effort on the part of the consumer. The process of overconsumption is neither intended nor wanted, but is allowed because of the biological system of appetite control that readily permits a surfeit of energy consumed but defends strongly against an under supply of energy. It has been argued that a year-on-year excess of energy consumed (at least in the US) can account for the secular increase in average bodyweight (and BMI) of the US population (Swinburn *et al.* 2009). In contrast, it has also been argued that the decline in daily energy expended due to the impact of technology on changing patterns of work, can easily account for the increase in adult BMI over the course of 30 years (Church *et al.* 2011). Consequently, there are plausible arguments – and experimental evidence – in favour of both increased energy intake and decreased energy expenditure in the aetiology and maintenance of obesity. During the last decade the British Nutrition Foundation (Watson & Benelam 2012) has emphasised that health is not only a matter of good nutrition (although this is vital) but also requires an optimal level of physical activity (Stensel 2010).

The key to understanding the 'balance' of energy consumed and expended is the recognition that physical activity does not only contribute to the energy expenditure side of the equation, but also influences energy intake. In other words, energy expenditure and energy intake are not independent of each other, they interact. This concept can be traced back to the work of pioneers of nutritional physiology in the UK who postulated that 'the differences between the intakes of food (of individuals) must originate in the differences in the expenditure of energy' (Edholm *et al.* 1955). The landmark study of Jean Mayer with the jute mill workers in Bengal (Mayer *et al.* 1956) demonstrated that dietary intake was related to the energy expended in the physicality of work – though with a U-shaped rather than a linear function. Recent large scale investigations (Shook *et al.* 2015) together with systematic reviews (Beaulieu *et al.* 2016) have supported this. The strong message from this body of work is that people who are habitually physically active have a sensitive control of homeostatic appetite whereas in sedentary people the control is weaker (poor match of energy intake with expenditure). If this is true then one would expect to find positive associations between sedentariness and the amount of surplus stored energy (adipose tissue) and inverse relationships with the amount of moderate and vigorous activity. There is abundant evidence for this (*e.g.* Ekelund *et al.* 2016; Myers *et al.* 2016).

The implication of this picture is that the behavioural science of nutrition should not be conducted in isolation, but should instead be integrated with the behavioural science of energy expenditure. The foods that people put into their mouths (what Rozin referred to as 'what we eat and why we eat it') is not just a matter of the choice architecture of the food-related external environment, but is also influenced by the (reduced) demands for energy expended imposed by the environment. The introduction of energy expenditure into the framework for understanding appetite control also directs attention to the role of metabolism and especially the impact of body composition. Although there will continue to be an interest in the pure psychological approach to food intake in which eating is reported as a function of psychological variables, there is an undisputed role for biological variables. For a quarter of a century – since the discovery of leptin (Zhang *et al.* 1994) - this has been dominated by the belief in a role for body fat (the adipocentric hypothesis). However, there is growing evidence that an underlying metabolic drive to eat is strongly associated with the fat-free mass in the body (lean tissue) and with the body's requirement for energy (resting metabolic rate – RMR) in order to maintain vital biological organ functioning (*e.g.* Blundell *et al.* 2012; Weise *et al.* 2014; Dulloo *et al.* 2016). In addition, recently some anthropologists have introduced evolutionary theory and field data on primitive tribes into the arguments about the overall balance of energy (in and out) (*e.g.* Pontzer *et al.* 2012). Although hunting (for the right product) and gathering (the groceries) in supermarkets is definitely not the same process as the hunting and gathering still carried out by the Hadza people of Tanzania (Marlowe 2010); the question is can we learn anything about the former from studying the latter? Can anthropology throw any light on the behavioural science of nutrition? It is probably better to widen the scientific horizons rather than to make them narrower.

Evaluation and challenges

More than 35 years ago in the UK, an entity now called the 'British Feeding and Drinking Group' (BFDG) was established by Professor Trevor Silverstone and a group of colleagues following the 1976 Dahlem Konferenzen on Appetite. The BFDG was dominated by influential psychologists such as David Booth who inaugurated the journal 'Appetite'. The BFDG and Appetite did much to expand research into the effect of behavioural science (especially psychology) on factors influencing eating behaviour and nutrition. What has been the effect of this large body of work? There is no doubt that behavioural science has contributed a litany, a methodology, an enthusiasm and a continuing vital enquiry to the field of nutrition. The impact has influenced dietitians and nutritionists who have themselves adopted the methodology of behavioural science and blended these with their more natural survey procedures. There is now a huge catalogue of outcomes of psychological and environmental manipulations on eating behaviour with implications for the ingestion of nutrients. However, many of these studies are often acute, with single manipulations on single occasions with a variety of contextual factors, but more often than not in a laboratory setting and on many occasions with university students as subjects. One noticeable consequence of this approach has been the demonstration that a momentary inspection of eating behaviour is both labile and extremely volatile. Almost every manipulation can produce some measurable change. This is probably a fair reflection of human eating behaviour which, at any particular moment, may be subject to irrational and often unwanted shifts. These momentary changes can be contrasted with the unhelpful stability of many (disadvantageous) eating habits, which are remarkably resistant to change. Although there exists a wide range of planned behavioural methods that can be deployed to change unhealthy food habits (*e.g.* Abraham & Michie 2008), these can be extremely time consuming and expensive on expertise. At the current time, behavioural science has contributed to our understanding of the complexity of behavioural nutrition but has not produced a behavioural solution for nutritional problems (including obesity).

However, the lack of a present day solution to nutritional problems that exist on a world scale is not the fault of behavioural science. This science has been grappling with the great diversity of global eating patterns and the extremely high degree of individual variability. This is partly why human behaviour is so fascinating (but also frustrating). This individual heterogeneity in behavioural outcomes, apparent in responses to manipulations of energy intake and expenditure, is associated with allelic variation contributing to an underlying biological heterogeneity. Behavioural science cannot be blamed for the complexity of human behaviour. However, one feature that the science can be held responsible for is an insistence on the use of the statistical mean (group averages) to define experimental outcomes. The use of the mean, and the convenient statistical differences between means (at the magical $p < 0.05$ level), has tended to convey an orderliness and neatness to behavioural data and has had the effect of eliminating from enquiry the more important, but inconveniently untidy, issue of human variability. As certain statisticians have pointed out 'The mean is an abstraction. Reality is variation' (Blastland & Dilnot 2008). Behavioural science of nutrition is taking a long time to deal with this issue.

However, the complexity and the individual variability of human behaviour is being addressed. The advent of technological devices for detecting and recording personal data (behavioural and nutritional) together with algorithms in the field of big data analytics are revolutionising the degree to which behavioural diversity can be monitored and utilised for the benefit of individuals. Hence the solution is scalable <u>not</u> on the basis of 'one size fits all' but on the basis of individual solutions for everybody. There is already considerable progress in this area (Martin *et al.* 2016).

The era of the behavioural science approach based on laboratory study has achieved plenty and is coming to an end; the era of behavioural science of larger populations, under more realistic living circumstances is taking over. This approach has the capacity to embrace both energy intake and energy expenditure and to provide a more expansive outlook to issues in nutrition.

References

Abraham C & Michie S (2008) A taxonomy of behavior change techniques used in interventions. *Health psychology* **27**: 379.

Beaulieu K, Hopkins M, Blundell J *et al.* (2016) Does Habitual Physical Activity Increase the Sensitivity of the Appetite Control System? A Systematic Review. *Sports Medicine* 1-23.

Bellissimo N, Desantadina M, Pencharz P *et al.* (2008) A comparison of short-term appetite and energy intakes in normal weight and obese boys following glucose and whey-protein drinks. *International journal of obesity* **32**: 362-371.

Berridge K & Kringelbach M (2008) Affective neuroscience of pleasure: reward in humans and animals. *Psychopharmacology* **199**: 457-480.

Birch LL, Fisher JO & Davison KK (2003) Learning to overeat: maternal use of restrictive feeding practices promotes girls' eating in the absence of hunger. *The American journal of clinical nutrition* **78**: 215-220.

Blastland M & Dilnot AW (2008) *The tiger that isn't: seeing through a world of numbers,* Profile Books.

Blundell J, De Graaf C, Hulshof T *et al.* (2010) Appetite control: methodological aspects of the evaluation of foods. *Obesity reviews* **11**: 251-270.

Blundell J, Rogers P & Hill A (1987) Evaluating the satiating power of foods: implications for acceptance and consumption. *Food Acceptance and Nutrition. Academic Press: London* 205–219.

Blundell JE, Caudwell P, Gibbons C *et al.* (2012) Role of resting metabolic rate and energy expenditure in hunger and appetite control: a new formulation. *Disease Models & Mechanisms* **5**: 608-613.

Blundell JE & MacDiarmid JI (1997) Passive Overconsumption Fat Intake and Short-Term Energy Balancea. *Annals of the New York Academy of Sciences* **827**: 392-407.

Brunstrom JM, Shakeshaft NG & Scott-Samuel NE (2008) Measuring 'expected satiety'in a range of common foods using a method of constant stimuli. *Appetite* **51**: 604-614.

Chambers ES, Viardot A, Psichas A *et al.* (2015a) Effects of targeted delivery of propionate to the human colon on appetite regulation, body weight maintenance and adiposity in overweight adults. *Gut* **64**: 1744-1754.

Chambers L, McCrickerd K & Yeomans MR (2015b) Optimising foods for satiety. *Trends in Food Science & Technology* **41**: 149-160.

Church TS, Thomas DM, Tudor-Locke C *et al.* (2011) Trends over 5 decades in US occupation-related physical activity and their associations with obesity. *PLoS One* **6**: e19657.

Dalton M & Finlayson G (2014) Psychobiological examination of liking and wanting for fat and sweet taste in trait binge eating females. *Physiology & behavior* **136**: 128-134.

De Graaf C, De Jong LS & Lambers AC (1999) Palatability affects satiation but not satiety. *Physiology and Behavior* **66**: 681-688.

Dulloo A, Jacquet J, Miles-Chan J *et al.* (2016) Passive and active roles of fat-free mass in the control of energy intake and body composition regulation. *European Journal of Clinical Nutrition*.

Edholm OG, Fletcher JG, Widdowson EM *et al.* (1955) The Energy Expenditure and Food Intake of Individual Men. *British Journal of Nutrition* **9**: 286-300.

Ekelund U, Steene-Johannessen J, Brown WJ *et al.* (2016) Does physical activity attenuate, or even eliminate, the detrimental association of sitting time with mortality? A harmonised meta-analysis of data from more than 1 million men and women. *The Lancet* **388**: 1302-1310.
Ello-Martin J, Roe L, Ledikwe J *et al.* (2007) Dietary energy density in the treatment of obesity: a year-long trial comparing 2 weight-loss diets. *American Journal of Clinical Nutrition* **85**: 1465.
Finlayson G, King N & Blundell J (2007) Liking vs. wanting food: importance for human appetite control and weight regulation. *Neuroscience & Biobehavioral Reviews* **31**: 987-1002.
Finlayson G, King N & Blundell J (2008) The role of implicit wanting in relation to explicit liking and wanting for food: implications for appetite control. *Appetite* **50**: 120-127.
Fisher JO, Liu Y, Birch LL *et al.* (2007) Effects of portion size and energy density on young children's intake at a meal. *The American journal of clinical nutrition* **86**: 174-179.
Flint A, Raben A, Blundell J *et al.* (2000) Reproducibility, power and validity of visual analogue scales in assessment of appetite sensations in single test meal studies. *International Journal of Obesity* **24**: 38-48.
French SA, Mitchell NR, Wolfson J *et al.* (2014) Portion size effects on weight gain in a free living setting. *Obesity* **22**: 1400-1405.
Gibbons C, Caudwell P, Finlayson G *et al.* (2013) Comparison of postprandial profiles of ghrelin, active GLP-1 and total PYY to meals varying in fat and carbohydrate, and their association with hunger and the phases of satiety. *Journal of Clinical Endocrinology and Metabolism* **in press**.
Halford JC & Harrold JA (2012) Satiety-enhancing products for appetite control: science and regulation of functional foods for weight management. *Proceedings of the Nutrition Society* **71**: 350-362.
Hall KD, Sacks G, Chandramohan D *et al.* (2011) Quantification of the effect of energy imbalance on bodyweight. *The Lancet* **378**: 826-837.
Hamid TK (2012) Thinking in Circles About Obesity. *Reflections* **11**.
Higgs S (2005) Memory and its role in appetite regulation. *Physiology & Behavior* **85**: 67-72.
Holt GM, Owen LJ, Till S *et al.* (2017) Systematic literature review shows that appetite rating does not predict energy intake *Critical Reviews in Food Science and Nutrition* 57 (16): 3577-3582
Hopkins M, Finlayson G, Duarte C *et al.* (2015) Modelling the associations between fat-free mass, resting metabolic rate and energy intake in the context of total energy balance. *International Journal of Obesity*.
Johnstone AM, Horgan GW, Murison SD *et al.* (2008) Effects of a high-protein ketogenic diet on hunger, appetite, and weight loss in obese men feeding ad libitum. *The American journal of clinical nutrition* **87**: 44-55.
Kessler DA (2010) *The end of overeating: Taking control of the insatiable American appetite,* Rodale.
Knai C, Petticrew M, Durand M *et al.* (2015) Has a public–private partnership resulted in action on healthier diets in England? An analysis of the Public Health Responsibility Deal food pledges. *Food Policy* **54**: 1-10.
Kringelbach ML (2004) Food for thought: hedonic experience beyond homeostasis in the human brain. *Neuroscience* **126**: 807-819.
Long CG, Blundell JE & Finlayson G (2015) A systematic review of the application and correlates of YFAS-diagnosed 'food addiction'in humans: are eating-related 'addictions'a cause for concern or empty concepts? *Obesity facts* **8**: 386-401.
Marlowe F (2010) *The Hadza: hunter-gatherers of Tanzania,* Univ of California Press.

Martin CK, Gilmore LA, Apolzan JW *et al.* (2016) Smartloss: A Personalized Mobile Health Intervention for Weight Management and Health Promotion. *JMIR mHealth and uHealth* **4**.
Mayer J, Roy P & Mitra K (1956) Relation between caloric intake, body weight, and physical work: studies in an industrial male population in West Bengal. *American Journal of Clinical Nutrition* **4**: 169.
Mela DJ (2006) Eating for pleasure or just wanting to eat? Reconsidering sensory hedonic responses as a driver of obesity. *Appetite* **47**: 10-17.
Myers A, Gibbons C, Finlayson G *et al.* (2016) Associations among sedentary and active behaviours, body fat and appetite dysregulation: investigating the myth of physical inactivity and obesity. *British Journal of Sports Medicine* bjsports-2015-095640.
Pontzer H, Raichlen DA, Wood BM *et al.* (2012) Hunter-gatherer energetics and human obesity. *PLoS One* **7**: e40503.
Prentice AM & Jebb SA (2003) Fast foods, energy density and obesity: a possible mechanistic link. *Obesity reviews* **4**: 187-194.
Rogers P & Blundell J (1979) Effect of anorexic drugs on food intake and the micro-structure of eating in human subjects. *Psychopharmacology* **66**: 159-165.
Rogers PJ (2011) Obesity–is food addiction to blame? *Addiction* **106**: 1213-1214.
Rogers PJ (2017) Food and drug addictions: Similarities and differences. *Pharmacology Biochemistry and Behavior* **153**: 182-190
Rolls BJ, Morris EL & Roe LS (2002) Portion size of food affects energy intake in normal-weight and overweight men and women. *The American journal of clinical nutrition* **76**: 1207-1213.
Rozin P (1982) Human food selection: The interaction of biology, culture and individual experience. *The psychobiology of human food selection* 225-254.
Rozin P (1998) *Towards a psychology of food choice,* Institut Danone.
Rozin P, Haidt J, McCauley C *et al.* (1999) Individual differences in disgust sensitivity: Comparisons and evaluations of paper-and-pencil versus behavioral measures. *Journal of Research in Personality* **33**: 330-351.
Rozin P & Tuorila H (1993) Simultaneous and temporal contextual influences on food acceptance. *Food Quality and Preference* **4**: 11-20.
Shook RP, Hand GA, Drenowatz C *et al.* (2015) Low levels of physical activity are associated with dysregulation of energy intake and fat mass gain over 1 year. *The American journal of clinical nutrition* **102**: 1332-1338.
Silverstone T (1976) *Appetite and food intake,* Udchn & Schneider.
Simms A (2012) *Tescopoly: How one shop came out on top and why it matters,* Hachette UK.
Spence C & Youssef J (2016) Constructing flavour perception: from destruction to creation and back again. *Flavour* **5**: 3.
Stensel D (2010) Exercise, appetite and appetite-regulating hormones: implications for food intake and weight control. *Annals of Nutrition and Metabolism* **57**: 36-42.
Stubbs J, Ferres S & Horgan G (2000a) Energy density of foods: effects on energy intake. *Critical reviews in food science and nutrition* **40**: 481-515.
Stubbs R (1995) Macronutrient effects on appetite. *International journal of obesity and related metabolic disorders: journal of the International Association for the Study of Obesity* **19**: S11.
Stubbs R, Harbron C, Murgatroyd P *et al.* (1995) 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 R, Hughes D, Johnstone A *et al.* (2000b) The use of visual analogue scales to assess motivation to eat in human subjects: a review of their reliability and validity with an evaluation of new hand-held computerized systems for temporal tracking of appetite ratings. *British Journal of Nutrition* **84**: 405-415.

Stubbs R & Whybrow S (2004) Energy density, diet composition and palatability: influences on overall food energy intake in humans. *Physiology & behavior* **81**: 755-764.

Sunstein C & Thaler R (2008) Nudge. *The politics of libertarian paternalism. New Haven*.

Swinburn B, Sacks G & Ravussin E (2009) Increased food energy supply is more than sufficient to explain the US epidemic of obesity. *American Journal of Clinical Nutrition* **90**: 1453.

Swinburn BA, Sacks G, Hall KD *et al.* (2011) The global obesity pandemic: shaped by global drivers and local environments. *The Lancet* **378**: 804-814.

Thaler RH, Sunstein CR & Balz JP (2014) Choice architecture.

Van Kleef E, Van Trijp J, Van Den Borne J *et al.* (2012) Successful development of satiety enhancing food products: towards a multidisciplinary agenda of research challenges. *Critical reviews in food science and nutrition* **52**: 611-628.

Wansink B & Kim J (2005) Bad popcorn in big buckets: portion size can influence intake as much as taste. *Journal of nutrition education and behavior* **37**: 242-245.

Watson R & Benelam B (2012) Physical activity: the latest on its contribution to energy balance and health. *Nutrition Bulletin* **37**: 78-85.

Weise CM, Hohenadel MG, Krakoff J *et al.* (2014) Body composition and energy expenditure predict ad-libitum food and macronutrient intake in humans. *International Journal of Obesity* **38**: 243-251.

Yeomans MR (1998) Taste, palatability and the control of appetite. *Proceedings of the Nutrition Society* **57**: 609-615.

Yeomans MR & Gray RW (2002) Opioid peptides and the control of human ingestive behaviour. *Neuroscience & Biobehavioral Reviews* **26**: 713-728.

Zhang Y, Proenca R, Maffei M *et al.* (1994) Positional cloning of the mouse obese gene and its human homologue. *Nature* **372**: 425-432.