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eprints@whiterose.ac.uk https://eprints.whiterose.ac.uk/ Behaviour, energy balance, obesity and capitalism

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Background

It is fair to say that much of the landscape in the field of nutrition is dominated by the problem of obesity. Despite a massive surge in research on obesity over the last 50 years, the prevalence of obesity, in the United States, for example, has risen from 18% of the population in 1980 to over 40% today. In a book review published in the Lancet (2011) it was reported that in the previous decade over 22k articles had been published with the word 'obesity' in the title, and over 66k if the search included the title and the abstract. It seems that the more the research being done the worse the problem becomes. Should this be worrying? The existence of obesity is paradoxical in a field apparently governed by principles of regulation and homeostasis; if these principles operate effectively, how can obesity develop? The concept of energy balance (EB), which is a natural way to conceptualise obesity, has been dismissed by many on the grounds that application of these principles is not effective. The widespread acceptance of the principle of biological reductionism plus the idea that obesity is a disease suggest molecular causes.

<u>AQ1</u> <u>AQ2</u> AO3

<u>AQ4</u>

However, the principles of EB can be shown to effectively reduce obesity when applied coercively and with sufficient force. For example, historical evidence has demonstrated that the economic blockade of Cuba from 1980 to 2005 caused a shortage of food and fuel in that country. This meant that people ate less and walked more [1]; daily energy intake (EI) dropped from 2899 to 1863 kcal, and the percentage of physically active people rose from 30 to 67%. These effects caused the prevalence of obesity to fall from 14 to 7% with improvements in diabetes, coronary heart disease and stroke. In addition, the Look Ahead trial, which applied multiple techniques to reduce EI and increase energy expenditure (EE), produced large reductions in BMI and fat mass without any surgical or pharmacological intervention [2]. Therefore, large interventions—either deliberate or accidental—are effective.

Tinkering with aspects of the food supply or physical activity is not enough. Principles of EB must be applied through mechanisms of social engineering. Can this happen in a capitalist system in which key commodities (especially food and transport) are driven by the need to maintain economic growth? Such a system, as argued by scholars such as Herbert Marcuse and Max Horckheimer of the Frankfurt Institute for Social Research more than 50 years ago, controls people's behaviour to the same extent as an overtly authoritarian regime (see ref. [3] for review). If these precepts are valid, it may be questioned whether a scientific solution is possible; that is, can the application of principles in biological sciences disclose mechanisms that could control fat mass, and be applied on the population scale required to deal with a pandemic?

It is argued here that any such procedure would have to be mediated through behaviour—either food intake or physical activity. Is this achievable, and what are the obstacles? It is not argued here that the management of obesity is impossible. There are many examples of smaller-scale, targeted, strategies that bring about weight loss. The issue is whether the pandemic can be overcome by tackling the cultural problem at its source in order to prevent fat mass in normal-weight individuals from being driven up to the level at which they qualify as people with obesity.

A note on biological regulation

One of the dominant explanatory principles in the field of appetite, EB and obesity is the concept of biological regulation. This is usually stated as body weight regulation or, more commonly, adipose tissue regulation. These terms are closely linked to a doctrine of energy homeostasis, which implies that energy (in and out) is controlled in the interests of a higher objective-namely regulatory control of components of body composition. A comparison is often made with the principle of glucose regulation. These terms give the impression of a precise control mechanism operating within a biological system. It is implied that when these mechanisms go awry the result is obesity, which arises from a fault of regulatory or homeostatic principles. However, as Speakman [4] has pointed out 'if body weight is under physiological regulation how come we have an obesity epidemic?'. However, we should keep in mind that biological regulation of a system is not the only principle through which a system can deal with some perturbation or unpredictability. An evolutionary principle is that a system will have adaptive properties, which contribute to biological organisation but which does not need to incorporate a process of regulation. As argued by Bich et al. [5] 'biological systems exhibit a wide range of mechanisms and strategies to ensure their survival under variable conditions'. These writers point out that there is no agreement on what regulation actually means, and the current use of the concept is ambiguous and mixes fundamentally different biological capacities. Adaptation, feedback and dynamic stability might be principles that could be used as alternatives to regulation and which do not embody the absolute certainty of the attainment of some fixed internal state (body mass, fat mass, etc.).

These arguments question whether the notion of regulation (for example, of fat) is essential to an understanding of the control of the EI which, in essence, is the behaviour of food consumption. Much of the justification for the use of terms such as fat regulation, adipostasis and energy homeostasis emanate from studies on laboratory rodents whose uniform laboratory environments and life styles are far removed from the ecological and evolutionary pressures that frame an understanding of the relationship between body fat and food in humans. As eloquently described by Pond [6] and Wells [7] body fat has many functions, which differ according to the life style and evolutionary development of the particular species. For example, the control of adipose tissue in migratory species or in hibernators cannot be used as a model for humans; the functions of body fat show considerable diversity even amongst mammals. In humans adipose tissue has flexible capacity, which allows an adaptation to environmental exigencies and unpredictability; such a function does not embody a requirement for fat to be regulated at some fixed value. One function of human fat is to maximise biological (survival) advantage in the face of environmental uncertainty. Such uncertainty is embodied in the heterogeneous nature of the food environment and the diversity of feeding behaviour.

It is relevant that the reaction of body weight to a perturbation is asymmetrical; there is a strong defence against an energy deficit but weak defence against an energy surfeit or positive EB. Over the years there has been a strong urge to explain why people can gain weight with apparent ease but—even with strong motivation—fail to maintain hard-earned weight loss. The reasons are likely to be biological and psychological. There is persuasive evidence from an analysis of the classic study of starvation by Keys that lean mass, rather than fat mass, may be the key component of body composition underlying the recovery from excessive weight loss [8]. This uniquely controlled investigation suggests that biological mechanisms account for weight regain in lean individuals. However, in the quite different landscape of millions of obese people trying to maintain weight loss in an aggressive consumer environment, we should not dismiss the role of strong behaviour habits held in place by psychological mechanisms. It is likely that the respective roles of biology or psychology in weight regain depends on the time, place and cultural context.

Humans are omnivores

The fact that humans are omnivores is of huge significance for both behaviour and nutrition [9]. 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. There is high individual heterogeneity in the rhythm of eating [10] and in nutrient intakes [11]. 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 and affordability), social class and culture.

In this argument it is important not to use the terms EI and feeding behaviour as if they were interchangeable. EI arises from the language of physiology and implies uniformity and mechanistic control. Human feeding behaviour operates according to quite separate principles. The particular language used to describe phenomena inevitably determines the way in which we conceptualise the issues and how these are investigated.

The factor of food choice is not subject to homeostatic principles but is embodied in cultural and social issues. The important factor is that this economically driven overconsumption is not compensated by a large degree of homeostatic feedback (although there is some adjustment [12]). In contrast there is a stronger resistance to under-eating, which can offset the tendency of an energy deficit to induce weight loss.

The dependence of food choice on environmental factors takes on toxic proportions in a consumer-driven society in which continual purchasing of food is required to drive economic growth. This economic growth is underpinned by the industrialisation of food production, the creation of seemingly ever more attractive food properties and clever marketing; all designed to promote excessive purchasing and consumption. The way in which foods can be designed to exploit the natural hedonic brain system further serves to stimulate consumption [13]. In this environment overconsumption is legitimised and even promoted. It has been estimated that in the United States, the increase in mean body weight over a 20-year period was associated with an average increase in energy flux of approximately 500 kcal per day for adults and 250 kcal for children [14]. Other calculations have demonstrated that the difference in daily EI between obese and lean people can be between 300 and 1500 kcal per day (see ref. [15] for comment). These amounts are not trivial and demonstrate the extent to which behaviour in a consumer environment can make an impact on EB. The power of the economic system to promote food consumption cannot be overestimated (although part of the energy gap will be contributed by reductions in EE). For a critical discussion see ref. [16].

However, the phenomenon is not selective to food alone. In a capitalist system a prevailing principle influencing people's lives is materialistic self-interest (eg, [17]) together with a loss of institutional structure [18] and overconsumption is widespread; the issue may be construed as 'commodity fetishism'. People buy too much of everything: shirts, trousers, dresses, furniture, household goods and other commodities, including food. However, of these materials only food has an effect on EI and promotes weight gain. Therefore, the food industry should not be singled

out for special blame; culpability rests on the wider political system that demands economic growth to promote wealth, employment and profit. The automobile industry produces ever more comfortable and seductive motor cars, which increase the tendency for people to drive instead of walk; and the visual media industry produces more compelling screens (and entertainment) to keep people in a sedentary rather than an active state. Where does this leave biology? It could be argued that despite the ubiquity of overconsumption (it applies to everything), principles of regulation and homeostasis should prevent the body from assimilating the energy surfeit contributed by increased food intake and sedentary behaviours; quite obviously this does not happen. A slightly different interpretation is that biological control may exist but it is overshadowed by external factors favouring consumerism [19].

Appetite behaviour in an EB framework

The classical approach in EB research has been to demonstrate that physiological adjustments form adaptive responses to perturbations in order to maintain stability. Studies normally measure physiological variables in response to fixed energy loads. Behavioural output has not normally been a feature of interest. However, it should be kept in mind that EI is 100% behaviour (eating) and for EE, let's say, behaviour is between 30 and 50% of TEE (depending of course on the amount of activity performed). Both of these behaviours are extremely volatile, show considerable individual heterogeneity and are difficult to measure. These factors may have been a deterrent to the investigation of the natural expression of these in relation to EB, and may have suppressed the examination of the role of EE as a factor in EI (appetite control).

<u>AQ5</u>

However, over 50 years ago, Edholm et al. [2021] and Mayer et al. [22] illustrated that food intake (EI) should be interpreted in the light of the prevailing level of EE. Indeed, it was asserted that 'the differences between the intakes of food (of individuals) must originate in the differences in the expenditure of energy' [20]. This idea was ignored until recently when in 2011 it was initially demonstrated that fat-free mass (FFM) was strongly associated with EI [2324], with subsequent confirmation [25] in quite different cultural groups. Since FFM is a major determinant of resting metabolic rate (RMR), and RMR is (normally) the major contributor to TEE, it was hypothesised that RMR could be a driver of EI. It was subsequently confirmed that RMR was strongly associated with both EI and with the level of hunger [26]. More recently statistical modelling has demonstrated that the effect of FFM on EI is mediated fully by RMR [27].

<u>AQ6</u>

<u>AQ7</u>

This phenomenon can be interpreted through an evolutionary perspective; it makes sense that the energy required to maintain the functioning of major vital organs (heart, liver, kidneys, gastrointestinal tract, brain and skeletal musculature) should constitute the minimal energy demand to drive EI. In other words, to drive foodseeking behaviour that leads to ingestion [28]. The relationship between FFM, RMR and EI is very robust and might even be considered a type of biological rule. All of the initial findings have been replicated [272930] and this has generated an editorial comment in AJCN that 'FFM and RMR constitute major determinants of EI' [31] thus endorsing findings produced 5 years earlier.

EE drives EI: a way of thinking about appetite control

It should be noted that the proposed drive for EI generated by RMR is not a cause of overconsumption. RMR induces a drive to eat but does not determine food choice. As noted earlier, for omnivores eating (and purchasing) in a consumer society, food selection is determined by contextual demands in the food supply of the environment. A major feature of the industrialised food repertoire is energy density (ED) of specific foods (and of the whole diet). Many studies have confirmed that total EI is related to the ED of foods consumed (eg, [27323334]. In other words metabolic requirements (RMR) drive food-seeking behaviour but the energy ingested results from the topography of that behaviour and the nutritional properties of the diet consumed.

This overconsumption is not a regulated endpoint; it is free to vary and does not appear to be subject to principles of strict regulation or homeostasis. The excess intake does not generate significant compensatory energetic adjustments (in later intake or activity) that are sufficient to offset the excess energy taken in (eg, [12]). In other words, the physiological properties of the EB system are not able to offset a major behavioural response (hyperphagia from ED) from causing a prolonged positive EB. The system is exquisitely designed to accommodate, but not to offset. This approach can be regarded as an alternative to the adipocentric idea and other concepts that suggest that eating is driven in order to regulate body fat. As noted earlier (eg, [6]) in humans one function of body fat is that it offers an adjustable resource in the face of environmental perturbations. Therefore, the argument that EI behaviour is driven in response to the body's energy requirements changes the way that we view weight gain and obesity. Indeed, in an economically driven consumer society, in which increasing consumption is an economic necessity, obesity is inevitable. Moreover, as people get fatter, they also develop more FFM and have increased metabolic demands for energy (RMR). The system therefore demonstrates positive feedback. It is argued here that EI is not a variable that is controlled in order to regulate body fat stores; rather adipose tissue adapts to an energy surfeit (or deficit if that should arise) [35]. In addition, it is possible to identify multiple effects of fat on appetite; these include a stimulatory effect via biology, and inhibitory effects through biological and psychological pathways [2736]. However, it should be kept in mind that the endpoint of these functional pathways is a form of behaviour, emitted in the public domain, that is susceptible to enormous modification.

Conclusion

The main argument in this essay concerns the nature of the interplay between behaviour and physiology for the understanding of EB and its relationship to obesity within a potent economic environment. A parallel proposal is that behaviour is the key component that has to be managed if the so-called obesity epidemic is to be overcome. This behaviour is influenced positively and negatively by biology, but not regulated. From a scientific perspective the biggest problem is that behaviour is the variable that is the most heterogeneous, most volatile and the least susceptible to objective quantification. Unlike physiological variables whose measurement can be largely conserved in moving from the laboratory to a freeliving situation, this is not the case for behaviour. Within laboratories behaviour can be quantified and can assume properties similar to physiological measures. However, laboratory behaviour is much calmer, more orderly and less capricious than behaviour (physical activity or eating) that takes place in a psychologically and physically turbulent culture. This is why laboratory experiments give a false impression of orderliness, and why studies in EB should contain some element of a more natural free-living situation. The consumer society in which people conduct their lives is not only an environment replete with millions of food items, motor cars and screens that induce an excess of intake and a lack of expenditure. It is, above all, a culture that actively promotes overconsumption and underactivity in order to drive economic growth; this is more than an environment of abundance, which passively offers foods to be consumed or not. In the face of this power, it appears that the biological system has adaptive properties, which can absorb the impact of culture but which lacks true regulatory properties to prevent damage. One interesting question for researchers is how to conceptualise the role of consumer behaviour (purchaser and eater) in which biology operates in the cultural and political landscape of a complex system [37]. Can biological mechanisms or psychological processes be revealed that are strong enough to resist the political and economic forces of a capitalist system, which is the basis for the world's business? [35].

Compliance with ethical standards Conflict of interest The authors declare that they have no conflict of interest.

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