# The triple whammy

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**JANE WARDLE** *on how environments, genes and behaviour interact to cause obesity – and what psychologists can do about it.*

OBESITY is associated with serious health risks. The odds of contracting diabetes, coronary heart disease, cancer and arthritis are markedly higher in obese than in normal-weight adults (Bray, 2004). Life expectancy may be reduced by as much as nine years at the highest levels of obesity. Recent estimates suggest that obesity could soon overtake smoking as the primary cause of chronic disease and premature death (Mokdad *et al.*, 2004).

The processes linking adiposity (fatness) with disease are only beginning to be understood. In the past it was assumed that adipose tissue itself was inert and that obesity-related pathology was determined largely by the underlying eating and activity habits. Current thinking is that the adipose cell mass – particularly the abdominal fat depots – is an endocrine organ, secreting a wide range of hormones that affect many bodily systems (Trayhurn & Wood, 2004). With this shift towards a more biological understanding of obesity, does psychology still have a role to play?

## The obesity epidemic

Adiposity is usually indexed by the ratio of weight to height in epidemiological research and clinical practice. In 1997 the World Health Organization recommended a standard definition of obesity in adults, based on the body mass index (BMI) (weight(kg)/height(m)$^2$). A BMI of 30 or more was defined as obese and 25–29.9 as overweight.

In most countries mean BMI has been steadily increasing, and with it, the proportion of the population who exceed the obesity threshold (James *et al.*, 2001). According to National Audit Office figures (see weblinks), 6 per cent of British men and 8 per cent of women were obese in 1984, but nine years later this had doubled to 13 per cent of men and 16 per cent of women; the latest figures from the Health Survey for England put the prevalence close to 23 per cent for both men and women (see weblinks). Increases in obesity in children have been almost as great (Lobstein *et al.*, 2003). To make matters worse, obesity prevalence figures underestimate the magnitude of the clinical problem because adiposity has increased disproportionately at higher levels of BMI. There are not only more obese people, but they are fatter than they used to be.

## What causes obesity?

Obesity develops when energy intake persistently exceeds the energy expended in metabolism and activity; this allows excess energy to be stored as fat. Behind this simple equation are the more complex questions of: (a) why some people maintain a state of positive energy balance while others regulate intake against expenditure, and (b) why many more people are in positive energy balance now than in the past. The epidemic increase in obesity over the past 30 years points to the powerful 'obesogenic' influence of the environment, because genes could not have changed over that period. But there is also enormous variation within populations. English women in the highest decile of BMI in 2001 weighed more than twice as much as those in the lowest decile, with no difference in height. This indicates that other factors – either environmental or

## WEBLINKS

International Obesity Task Force: *www.iotf.org*

National Institute for Diabetes and Digestive & Kidney Diseases weight loss article: *www.niddk.nih.gov/health/nutrit/nutrit.htm*

National Audit Office obesity figures: *www.nao.org.uk/pn/00-01/0001220.htm*

Health Survey for England 2003: *www.publications.doh.gov.uk/public/summary.htm*

genetic – make individuals who share the same broad external environment, store different amounts of fat.

## Environments and adiposity

For humans, environments can be seen as influencing either the opportunities or the pressures for overconsumption. Behavioural research shows that people tend to eat more when food is more palatable, more varied, served in larger portions, and higher in energy density (Kral & Rolls, 2004). Over the past few decades the food supply has shifted along all these dimensions, and in addition, has become more accessible, more vigorously promoted (including directly to children) and cheaper than ever before (Jeffery & Utter, 2003).

To compound the energy balance problem, physical activity has been increasingly engineered out of daily life: mechanisation has reduced the need for manual work in homes and workplaces and motorised forms of transport have replaced walking. At the same time, the variety and sophistication of sedentary activities has increased out of all recognition. Recent years have seen modest increases in the amount of leisure-time physical activity in adults, but these are dwarfed by the reductions in energy expenditure resulting from changes in lifestyle. Expressed in economic terms, physical activity has shifted from being something that people were paid to do (in that activity was required for work) to something that people have to pay for by giving up other enjoyable leisure activities to make time for it. At the same time, food has gone from being the major household expense to a pocket-money option.

Modification of the environment to tip the balance away from energy intake and towards expenditure is one of the principal challenges to public health in the 21st century, and it's likely to be an exceptionally tough one. If palatability, price and availability are among the principal determinants of overconsumption, politicians and the public need to sign up to foods being less pleasant, less accessible or more expensive. If energy-saving environments and easy access to transport are determinants of underactivity, we need to accept more inconvenient lives and less efficient ways of getting around. None of these are likely to be vote-winners.

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Most adults (though probably fewer children) approve of changes in children's environments: taking chips off the school menu, removing vending machines from school premises, and increasing time for sports are measures that attract widespread support. Unfortunately, if protection from obesogenic environments is limited to the early years, it may only postpone the development of problems. Twin and adoption studies show that the environments in which children are reared (of which schools can be considered one facet) may affect their weights while they live within the environment, but have little enduring effect once they leave (Grilo & Pogue-Geile, 1991).

The force of this argument is illustrated by the fact that the 30 per cent of British 55-year-olds who are now obese would almost all have been normal-weight children in the 1950s. Studies of immigrants give the same message: south Asians who moved to Britain as young adults had typically been raised in environments of low food and high activity, but their levels of obesity as adults exceed those in the host population. To prevent obesity in children, childhood environments must be modified, and to prevent obesity in adults, adult environments must be modified.

Psychological research into environmental effects has made progress in understanding the influence of food characteristics (energy density, portion sizes, etc.). There are also exciting new collaborations with geographers and environmental scientists to map characteristics of the built environment (types of food shops, availability of places for leisure activity, or transport options) on to behavioural patterns (Frank *et al.*, 2004). These new approaches don't always confirm the simple associations between adiposity and environmental characteristics that were hypothesised (for example, the concept of poor residential areas as 'food deserts'), but they offer the opportunity to test environmental explanations empirically in a real-world context. There is an urgent need for behavioural research at all levels from the laboratory to the community, as well as an infusion of behavioural expertise within policy making, to work out how environments influence behaviour and how to re-engineer environments to prevent unhealthy levels of fat storage.

## Environments and behavioural choices

While environmental changes should eventually make it easier to eat healthily and be active, control of the obesity

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epidemic in the near future is likely to require people's active cooperation in changing their behaviour. The Wanless Report identified this as the 'fully-engaged scenario', in which the public and the state share responsibility for health promotion and disease prevention (see *tinyurl.com/6stlt*).

The key question is therefore how to motivate people to make healthier choices. This is familiar territory for health psychologists, but while there has been progress in elucidating the contribution of attitudes and social norms to health behaviours (Conner & Norman, 1995), much remains to be learned about how to bridge the gap between people's awareness of the benefits of healthy lifestyles and their day-to-day choices. 'I know I shouldn't' is uttered by millions of people every day as they choose a biscuit over an apple. There are still striking gaps in health knowledge and awareness of the risks of being overweight. A population survey in England showed that fewer than half of the overweight and obese adults in Britain were trying to lose weight (Wardle & Johnson, 2002). More recently, Cancer Research UK reported that only 6 per cent of the population were aware of the link between obesity and cancer.

**How can we motivate people to make healthier choices?**

As with smoking, weight control is linked to education. The most educated sectors of the population have the lowest levels of obesity. Higher socio-economic status is also linked with higher levels of restrictive dieting, more leisure-time physical activity, and stronger weight-control intentions (Wardle & Griffith, 2001; Wardle *et al*., 2004). These disparities highlight the need for research into dissemination of the knowledge and skills to resist the obesogenic environment.

## Genes and adiposity

In contrast to the environmental explanations for the trends in obesity over time, weight differences between individuals are strongly influenced by genetic factors. The primary evidence comes from twin studies, where concordance for BMI is almost twice as high in monozygotic twins (who are genetically identical) than dizygotic twins (who share 50 per cent of their genes on average) (Grilo & Pogue-Geile, 1991). Heritability estimates for adiposity indicate that more than 70 per cent of the variation in adiposity can be attributed to genetic differences between individuals.

Genomic research has yet to find the genes that contribute to variation in adiposity. The discovery of the leptin gene in mice and the subsequent identification of two children with leptin deficiency were important steps, but since that time few single gene causes of obesity have been discovered. It now seems likely that adiposity is determined by many genes, each contributing only a small amount to variance in weight. This will make it more difficult to detect the effects of individual genes.

## If obesity is 'genetic', what role does psychology have?

In 1968 Stanley Schachter published an influential paper in *Science* showing that obese and normal-weight people responded differentially to internal and external cues related to food intake. The 'externality theory' – as it became known – proposed that obesity was a consequence of being more reactive to external cues such as food palatability and less responsive to internal cues related to satiety. Over time this elegant theory was challenged first by Nisbett's hypothesis that externality was a consequence of the episodic food restriction practised by many overweight people who are trying to control their weight (Nisbett *et al*., 1973), and later by Herman and Polivy's (1975) theory which argued that deliberate efforts to control food intake (restrained eating) were the source of disruption of the 'natural' control mechanisms. After this, research into eating behaviour in obesity took a back seat for many years.

There is now renewed interest in whether differences in responses to external (food) cues or internal (satiety) signals could be part of a phenotype that determines obesity risk. Obese children have been shown to be more responsive to food cues and less responsive to satiety signals (Barkeling *et al*., 1992; Jansen *et al*., 2003). Compared with normal-weight groups, obese adults and children will work relatively harder for food than for alternative rewards, indicating that food has a higher reinforcing value (Saelens & Epstein, 1996; Smith & Epstein, 1991). Measures of the reinforcing value of food have also been linked to weight gain after smoking cessation (Epstein *et al*., 2004), adding to an older literature which showed 'externality' to predict weight gain among children attending a summer camp (Rodin & Slochower, 1976). If eating and activity responses are hypothesised to link genes and adiposity, they should also be shown to be heritable. Few studies have investigated the heritability of eating behaviour, but there is evidence from studies of adults that 'disinhibition' (eating in response to external and emotional cues) and responsiveness to palatability are heritable

## DISCUSS AND DEBATE

- Is it right to dismiss regulation of the food supply and the food industry as 'nanny state'?
- Is the food industry providing individuals with choices or overwhelming their power to make decisions?
- Food adverts are generally for foods high in refined sugars and fats, and many of these adverts are aimed at children. Do we *want* to have to exercise choice and resist temptation so often? Do we *want* our children to be offered choice to this extent?
- Is the fast-food culture really to blame for the recent rise in obesity?
- Does the government have a role to play in obesity and should it be doing more to improve the health of the nation?
- Should we be taxing unhealthy food as well as cigarettes?
- If genetic susceptibility plays a part in obesity, do we have an obligation to make the environment less persuasive for these individuals or is it up to them to control themselves?

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(de Castro, 2004), and studies are under way to examine these kinds of traits in children.

A behavioural model of obesity risk conceptualises obesity not as 'determined' by genes, but as an interaction between genes and environments. Differential responsiveness to food cues would lead genetically susceptible individuals to eat more when highly palatable foods are presented. It might also lead these individuals to seek out opportunities for palatable foods – increasing the apparent impact of genes through gene–environment correlations. This model is consistent with the finding that that as food environments become more permissive, adiposity increases most in those who are already obese (Flegal & Troiano, 2000). The behavioural model therefore contributes to understanding not only individual differences, but also population trends.

Research is needed to examine a wider range of eating and activity behaviours, to extend the work to prospective studies, to use genetically sensitive designs to address the heritability of the proposed intermediate variables, and eventually to collaborate with molecular biologists to identify the genes that contribute to these behaviours.

## Behavioural science in prevention and treatment

Reversing the obesity epidemic will require major changes both in the environment and in people's interactions with their environments. Behavioural science will be pivotal, not only for understanding the mechanisms leading to overeating and underactivity, but also for identifying the conditions that achieve change. The much vaunted biopsychosocial model would seem to be ideally suited to the problem. Forging partnerships with biologists on one side and environmental scientists and policy makers on the other will put behavioural science in the centre of the field.

The challenge of prevention should not obscure the pressing need for treatment. Almost one in four British adults are obese, and they are, by definition, the most vulnerable to the obesogenic environment. Psychological treatment – cognitive behaviour therapy – is widely acknowledged to be the gold standard for obesity treatment, but the typical weight loss is a frustratingly small 5–10 per cent, and even that tends to be regained when treatment contact ceases (Wing, 2001). Psychologists should be playing a central role in developing more effective treatments, with longer-lasting results, and finding ways to deliver them on the massive scale that is required.

Lessons could be learned from the smoking field. In the early 1950s almost 80 per cent of British men smoked. When Doll and Hill provided incontrovertible evidence of the link with lung cancer, doctors were in the vanguard of change; now close to half of ever-smokers have quit. Psychological research has been central to understanding nicotine addiction, developing effective treatments, exposing the practices of the tobacco industry, and identifying the range of educational, fiscal and environmental policies which between them have brought smoking levels down. Some of the methods, models and research alliances that proved fruitful in the smoking field could provide templates for obesity research. For example, better collaboration between scientists working on the basic behavioural mechanisms of appetite control, neuroscientists unravelling the biological processes, and public health researchers tracking the population effects, could all provide fertile soil for progress.

There are also striking similarities in the tension between industry and human health. In a system where foods are traded as commodities and industrial profits depend on increasing sales, the struggle to clean up the food environment and encourage people to resist the temptation to eat is likely to be a reprise of the epic struggles with the tobacco industry (Brownell, 2004).

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