



REPLY

Reply: Is obesity a disease?

PG Kopelman^{1*} and N Finer²

¹*Barts and The London, Queen Mary's School of Medicine and Dentistry, London, UK; and* ²*Luton and Dunstable Hospital and Luton University, Luton, UK*

'Corpulency, when in an extraordinary degree, may be reckoned a disease as it in some measure obstructs the free exercise of the animal functions and hath a tendency to shorten life by paving the way to dangerous distempers.'
(Malcolm Flemyng, 1760)

We are grateful for the opportunity to respond to Drs Heshka's and Allison's paper. In contrast, we consider obesity to be a disease that results from the processes of undesirable positive energy balance and weight gain. To suggest obesity is not a disease suggests expediency that perpetuates the myth that obesity is simply an affliction of modern society without medical consequence. We agree the need for conceptual clarity in science and philosophy and suggest that there are strong reasons for identifying obesity as a disease:

- To promote an understanding that obesity is not simply an inevitable consequence of an affluent and increasingly sedentary society but is a 'malignant' condition of modern life that can be avoided or reversed with substantial health benefit to an individual and society as a whole.
- To highlight to the sufferers, the medical profession and the public at large that obesity (or excessive fatness) is a potentially killing disorder.
- To receive appropriate recognition from healthcare planners, health authorities and local and national governments that obesity is epidemic within society and affects all ages.

We are curious about the reasons why Drs Heshka and Allison wish to declassify obesity from being a disease, particularly as a code for obesity has existed in the International Classification of Diseases since ICD-6 was published in 1948. The clinical modification of ICD-9 (ICD-9-CM) was developed in the United States to provide a way to classify morbidity data: morbid obesity was added to ICD-9-CM in 1995. The latest US mortality figures list obesity as the

underlying cause of death in 1983 cases and a contributing factor in 8761 (US National Center for Health Statistics). Utilizing this data, the National Ambulatory Medical Care Survey (1993) showed that obesity accounted for 8.7% of all patient visits to medical practitioners' offices—a total of 62.7 million visits. The incidence of visits for obesity is greater than for diabetes (5.6%) or asthma (4.9%) or osteoporosis (2.5%). By contrast, HIV accounted for 0.2% of all patient visits. We can only presume that the authors wish to deny the recognized and characteristic pathological processes associated with increasing body weight. They seem to adopt the philosophy of Humpty Dumpty 'when I (we) use a word... it means just what I (we) choose it to mean, neither more or less'.¹

All disease entities are abstract concepts created by the human mind (Feber, 1923).

We suggest that Drs Heshka and Allison's arguments provide persuasive evidence for obesity being a disease: their dilemma appears to be the interpretation of this evidence. We will argue for this firstly from a scientific point of view and then consider more philosophical issues.

Heshka and Allison identify four common and recurring components from definitions of disease (a)–(d). They agree that obesity fulfils the conditions for (a) and (b) but have difficulties with (c) and (d). We now re-examine each component in turn:

(a) 'A condition of the body, its parts, organs or systems or an alteration thereof': Heshka and Allison accept that obesity satisfies this definition. It would be hard to deny that obesity results from an excessive deposition of adipose tissue from which very few organs are exempt.

(b) 'Resulting from infection, parasites, nutritional, dietary, environmental, genetic or other causes.' Heshka and Allison do not deny the pivotal roles played by genes, nutrition and the environment in the current epidemic of obesity worldwide. Obesity is additionally the characteristic, but not only, clinical feature of a number of monogenic syndromes that are associated with other detrimental disease processes and early death (Prader–Willi and Bardet–Biedl syndromes are examples). Furthermore, other rare single

*Correspondence: PG Kopelman, Barts and The London, Queen Mary's School of Medicine and Dentistry, Turner Street, London E1 2AD, UK.
E-mail: p.g.kopelman@mds.qmw.ac.uk

Received 19 February 2001; accepted 22 February 2001

gene mutations cause obesity. For example, a single amino acid deletion in the leptin gene results in obesity from early life, which can be partly reversed by replacement leptin therapy.²

(c) 'Having a characteristic, identifiable, marked, group of symptoms and signs'. We suggest that this definition is best addressed by considering other diseases. Ischaemic heart disease is a disease that may have no symptoms or signs. The symptoms of ischaemic heart disease may include chest pain from angina or myocardial infarction, but it is not uncommon for someone to have ischaemic heart disease without symptoms and, indeed, to die from the disease. Where symptoms exist, they may be non-specific and may be mistaken for other conditions such as indigestion or jaw pain. Doctors use the characteristic, but non-specific symptoms of ischaemic heart disease, in conjunction with a detailed medical history, to make a diagnosis. Obesity is more than excess fatness. It has both characteristic symptoms and signs: increasing adiposity is reflected by fatigue, breathlessness, loss of sexual potency, menstrual irregularities, arthralgia and mood change. These symptoms may be accompanied by the signs of tachypnoea on exertion, increased blood pressure, intertrigo, excessive perspiration and arthritis of large joints. Heshka and Allison seek to promulgate a prejudicial view that obesity is merely fatness; careful clinical evaluation will nearly always elicit significant symptoms and signs. In one study of obese subjects, one-third of men and nearly half the women reported shortness of breath on walking uphill or upstairs; approximately a quarter of respondents complained of lower back pain.³ We accept that obesity fits best with the concept of a disease syndrome but, in this respect, it is no different to other diseases defined by clinical rather than genomic or biochemical evidence.

(d) 'Deviation from normal structure or function... etc.' We disagree with the suggestion that 'mild obesity' only 'threatens eventual impairment'—such a statement denies the definite and significant consequences observed in many subjects with upper body obesity who are 'mildly' overweight. 'Mild obesity' should join 'mild diabetes' as a term that is deleted from the medical vocabulary because of the evidence that indicates otherwise. We do not accept the argument that patients with a disease are excluded from being 'competent functioning members of society' nor should they 'feel themselves impaired'. The whole thrust of modern medicine is to avoid stigmatization of groups of patients with disease such as diabetes or even cancer.

We can further support obesity as 'deviating from normal structure or function' by considering the multiplicity of biochemical alterations that attest to 'the impairment of the normal state'. The obesity-associated changes seen in plasma lipids, liver structure and liver and pancreatic beta cell function support 'disorder and derangement'.

The management of many patients with the metabolic syndrome (upper body obesity, type 2 diabetes, hypertension

and dyslipidaemia) will be facilitated by a better understanding by patients and health care professionals of the contribution of visceral adiposity to the development of insulin resistance, glucose intolerance and dyslipidaemia. The premature deaths reported in obese patients with type 2 diabetes in the United Kingdom Prospective Diabetes Study (UKPDS) are irrefutable.⁴ The authors overlook the considerable evidence for adipose tissue being an endocrine organ and the detrimental adaptations observed with increasing adiposity. At present we cannot foretell who will develop an obesity-related health problem but neither can we predict who will die from ischaemic heart disease.

We are similarly unconvinced by the authors' philosophical arguments against obesity as a disease. We consider them counter-productive and merely repeat excuses that have enabled the public and scientists to distance themselves from the reality of obesity—we suggest that the clinical reality determines the disease obesity. There is no escaping the social reality of increasing obesity and its associated morbidity and mortality nor obesity increasingly being seen in young as well as old—type 2 diabetes in early teens and adolescence is a phenomenon of modern society firmly linked to obesity.⁵ We are puzzled by the authors' conjecture that social consensus is required before a label of disease may be applied; this implies that society should conceal a medical reality for fear that doing otherwise implies a society-induced illness. It is crucial for obesity to be taken seriously. This will not happen unless society at large is aware of the penalties that individuals are paying from the current obesity epidemic. We refute the suggestion that a disease label is one of expediency to attract research funding. Moreover, we suggest that measures for the prevention of obesity have failed to date because of a lack of public awareness of obesity as a disease. The immense treatment costs of obesity are not being addressed by health services partly because of puerile arguments about its disease status. We implore all concerned with obesity to put aside any remaining contention about definition or consequence and throw their energies into defeating a major scourge of society at the beginning of the twenty-first century.

References

- 1 Carrol L. *Through the looking-glass, and what Alice found there*, 1872.
- 2 Farooqi IS *et al.* Effects of recombinant leptin therapy in a child with congenital leptin deficiency. *New Engl J Med* 1999; **341**: 879–884.
- 3 Lean MEJ, Han TS, Seidell JC. Impairment of health and quality of life using new US federal guidelines for the identification of obesity. *Arch Intern Med* 1999; **159**: 837–843.
- 4 Stratton IM, Adler AI, Neil HA, Matthews DR, Manley SE, Cull CA, Hadden D, Turner RC, Holman RR. Association of glycaemia with macrovascular and microvascular complications of type 2 diabetes (UKPDS 35): prospective observational study. *Br Med J* 2000; **321**: 405–412.
- 5 American Diabetes Association. Type 2 diabetes in children and adolescents. *Diabetes Care* 2000; **23**: 381–389.