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How dangerous is obesity?

It is remarkable that we still cannot assign precise risks to health for any degree of moderate obesity. The recent report of the DHSS/MRC Working Group[1] reviewed much of the published data, but, apart from concluding that obesity is associated with conditions such as ischaemic heart disease, osteoarthritis, postoperative complications, poor obstetric performance, and lack of physical fitness and—above certain undefined limits—was likely to reduce life expectancy, it offered no clear guidelines. As the authors themselves state, "as a guide both for prevention and treatment we need more quantitative information about the relation between the degree of fatness and the degree of risk."

Perhaps the most disappointing aspect of our ignorance about obesity is that the nature of the association between obesity and ischaemic heart disease, and between obesity and hypertension, is still far from clear. One difficulty is that of definition. Obesity and overweight are not synonymous: obesity refers to an excess of fat, whereas overweight may be due to other variations in body composition apart from an excess of fat. Nevertheless, in Western society overweight individuals are usually obese, and simple indices of relative weight (weight/height squared almost certainly being the best) appear to give almost as good a measure of body fat, assessed by laboratory methods, as do skinfold measurements.[2] Unfortunately, even the laboratory methods used for assessing body fat, such as body densitometry or measuring total body water or total body potassium, are subject to considerable error unless two or more methods are used together,[2 3] and so there may be little advantage in using these relatively complicated procedures rather than much simpler indices.

A second difficulty is that many important risk factors are interrelated, so that these need to be separated out carefully in analysis. The association between increasing body weight and age is well recognised, but that between body weight and cigarette consumption, though well described,[4] is frequently ignored. Nonsmokers tend to weigh considerably more than smokers, and individuals who have given up smoking may decide to take up the habit again for fear of becoming overweight. Nevertheless, it cannot be emphasised too strongly that the morbidity and mortality for exsmokers is usually considerably lower than for current smokers, which suggests that the untoward effects of (small) gains in weight are much more than compensated by the benefits of not smoking.[5]

The practical importance of these qualifications is shown in a recent case-control study in which "probable or overt ischaemic heart disease" was found not to be related to the body fat content as estimated from measurements of total body water by tritium dilution.) Unfortunately, the smoking habits of the cases and controls were evidently not taken into account, and the apparent lack of association between body fat and coronary heart disease may have been an artefact because probably many of the individuals with signs or symptoms of ischaemic heart disease were cigarette smokers and therefore of relatively low body weight. Conversely, the association between body weight and the incidence of ischaemic heart disease may be exaggerated in studies in which the association between body weight and age has not been taken into account.[7] Since age is obviously a risk factor of prime importance for ischaemic heart disease it is essential to eliminate it as a variable either by considering small age groups separately or by more complex methods of analysis. Similarly, cigarette smokers and nonsmokers should be considered separately, as should the different clinical manifestations of ischaemic heart disease itself.

A further problem is that the sphygmomanometer bag in normal use in Britain (22 cm long) and in the United States (26 cm long) fails completely to encircle the arms of most individuals, thus leading to falsely high blood pressure readings.[8 9] Intra-arterial measurements indicate that there is a real association between increasing obesity and increasing blood pressure,[10] but this relationship has probably been exaggerated because of the short bag used in many studies.

When more complex analysis of the relative importance of different risk factors for ischaemic heart disease has been carried out in large prospective studies to disentangle the influence of individual variables, overweight often emerges as being of importance only by virtue of its association with blood pressure, age, serum cholesterol concentration, and intolerance to glucose.[5 11 12] Nevertheless, this evidence alone is insufficient to undermine the usefulness of overweight as a risk factor, since it is certainly easier to recognise overweight than it is to detect raised blood pressure or raised serum cholesterol levels. And it is important to realise that the factors which are most effective for predicting future disease are not necessarily the most appropriate factors to attack directly in any preventive programme. Blood pressure, serum cholesterol concentration, and blood glucose levels all tend to fall in individuals who lose weight, and for each 10% reduction of body weight we might expect a 20% reduction in the incidence of ischaemic heart disease[13]—though the hypothesis that control of risk factors

will in fact reduce the incidence of disease remains to be tested.

Possibly we know so little about the dangers of different degrees of overweight because the investigators have not studied this specific problem. They may have spent too much effort in trying to unravel the complicated interrelations between numerous variables which may be risk factors. They now need to find the answer to questions such as, "What is the excess risk to a 40-year-old male nonsmoker or moderate smoker if he is 20%, 40%, or 60% overweight?" Even now, however, we already have enough evidence to suggest that if the onset of obesity is prevented by establishing appropriate patterns of nutrition and physical activity in childhood, and if obesity is corrected at least in those in whom associate risk factors are also present, then much untimely illness and mortality would be avoided. Since the costs and risks associated with such a programme of prevention and treatment need be very small indeed, it is about time that somebody did something.

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Apples and the teeth—"Nature's toothbrush" reappraised

For most of this century ending a meal with a hard food or fruit has been widely advocated for preventing the two major dental diseases, dental caries and periodontal disease.[1] Of these hard foods, apples have been the most commonly recommended, since it has been claimed[2] "that the eating of fresh uncooked apples has a regenerative influence upon the teeth and gums." Apples have commonly featured in dental health programmes[3] and have become, to some extent, a symbol of dental health.[4]

There have been three reasons for recommending apples. Firstly, it was long believed that apple eating after meals cleaned the teeth[2 5]—that it removed food residues and plaque (the bacterial deposit formed on uncleaned teeth thought to be responsible for both caries and gingivitis). However, apples and other fibrous foods in fact do little to achieve this objective.[6-11] Apples may bring about some cleansing, but plaque in the important sites—between the teeth and near the gum margin—is not removed, and most studies have shown no improvement in gingival health. Only two clinical studies of the supposed benefits of apples preventing caries appear to have been carried out; one of them[12] suggested a reduction in caries incidence when children ate apples after meals, but the effect was small and the initial caries scores of the children in the apple and control groups were not well matched. A later study[13] showed a negligible reduction in the incidence of caries when apples were eaten after the evening meal.

Secondly, apples have been promoted as a less damaging food for eating between meals than other snacks with higher carbohydrate contents and a greater tendency to stick to the teeth. Evidence on this point has come from studies of the pH of plaque, which falls within 10 minutes of eating sugar[14] as a result of acid produced by bacterial glycolysis. The pH often reaches levels at which the tooth mineral may dissolve, and the size of this fall in pH has been used to assess the harmfulness of foods to the teeth.[15 16] Apples not only contain sugar but are themselves also very acid; so that after eating them the plaque pH shows a distinct fall—suggesting that they are not without hazard to the teeth, though not so damaging as some other traditional dental enemies in the diet.

A third suggestion, originating from work by Pickerill in 1912[1] but strangely neglected since, was that the benefits of eating apples at the end of a meal arose because the acid taste of apples stimulated the flow of an alkaline saliva. As the flow-rate increases, the pH of saliva rises (typically from resting values of below 6 for parotid saliva to about 8 or more with maximal flow), and the buffering power is greatly increased.[17] So we might expect that any acid formed by the plaque from ingested sugars would be neutralised and buffered by the apple-stimulated saliva. The first, long overdue, experimental investigation of this idea[18] has dealt a final blow to the apple story by showing that eating apples when the plaque pH is already low after a sugary food does not lead to a protective rise in pH. The beneficial effect of the alkaline saliva is roughly balanced by the strong acidity (and perhaps sugar content) of the apple itself.

Even so, the idea of eating something at the end of a meal to stimulate a protective flow of saliva is not dead. In the same paper, and in other publications from the same authors,[19] salted peanuts and cheese have been shown to achieve the desired effect. These foods seem to be beneficial because, in addition to their strong flavour (and hence potent sialagogue effect), they contain little readily fermentable carbohydrate and are not strongly acid. So, while apples must be demoted from their position of eminence as foods "good for teeth," other foods, among them peanuts and cheese, which are harmless to the teeth and help to combat the effects of potentially harmful foods might be recommended both as between-meal snacks and as the last item of the diet at mealtimes.

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