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.

[1] Department of Health and Social Security and Medical Research Council, *Research on Obesity*. London, HMSO, 1976.
[2] Womersley, J, and Durnin, J V G A, *British Journal of Nutrition*, 1977, in press.
[3] Siri, W E, *University of California Radiation Laboratory Publication No 3349*. Berkeley, University of California, 1956.
[4] Khosla, T, and Lowe, C R, *British Medical Journal*, 1971, **4**, 10.
[5] Goldbourt, U, Medalie, J H, and Neufeld, H N, *Journal of Chronic Diseases*, 1975, **28**, 217.
[6] Weinsier, R L, *et al*, *American Journal of Medicine*, 1976, **61**, 815.
[7] Keys, A, *et al*, *Journal of Chronic Diseases*, 1972, **25**, 329.
[8] Mann, G V, *New England Journal of Medicine*, 1974, **291**, 178.
[9] Mann, G V, *New England Journal of Medicine*, 1974, **291**, 226.
[10] Holland, W W, and Humerfelt, S, *British Medical Journal*, 1964, **2**, 1241.
[11] Keys, A, *et al*, *Annals of Internal Medicine*, 1972, **77**, 15.
[12] Dyer, A R, *et al*, *Journal of Chronic Diseases*, 1975, **28**, 109.
[13] Ashley, F W, and Kannel, W B, *Journal of Chronic Diseases*, 1974, **27**, 103.

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.

[1] Pickerill, H P, *The Prevention of Dental Caries and Oral Sepsis*. London Baillière, Tindall and Cox, 1912.
[2] Hall, J T, *Some Chewable Foods for the Promotion of Mastication and the Prevention of Pyorrhoea*. Bournemouth, Bournemouth Guardian, 1929.
[3] Finlayson, D A, and Wilson, W A, *British Dental Journal*, 1961, **111**, 103.
[4] Baker, C, and Thomas, J, *Dental Health*, 1969, **8**, 23.
[5] Wallace, J S, *The Physiology of Oral Hygiene and Recent Research*, 2nd edn. London, Baillière, Tindall and Cox, 1929.
[6] Arnim, S S, *Journal of Periodontology*, 1963, **34**, 227.
[7] Lindhe, J, and Wicen, P O, *Journal of Periodontal Research*, 1969, **4**, 193.
[8] Reece, J A, and Swallow, J N, *British Dental Journal*, 1970, **128**, 535.
[9] Wade, A B, *Dental Practitioner*, 1971, **21**, 194.
[10] Longhurst, P, and Berman, D S, *British Dental Journal*, 1973, **134**, 475.
[11] Birkeland, J M, and Jorkjend, L, *Community Dentistry and Oral Epidemiology*, 1974, **2**, 161.
[12] Slack, G L, and Martin, W J, *British Dental Journal*, 1958, **105**, 366.
[13] Averill, H M, and Averill, J E, *New York State Dental Journal*, 1968, **34**, 403.
[14] Stephan, R M, *Journal of the American Dental Association*, 1940, **27**, 718.
[15] Graf, H, *Schweizerische Monatsschrift für Zahnheilkunde*, 1969, **79**, 146.
[16] Edgar, W M, *et al*, *Journal of the American Dental Association*, 1975, **90**, 418.
[17] Dawes, C, and Jenkins, G N, *Journal of Physiology*, 1964, **170**, 86.
[18] Geddes, D A M, *et al*, *British Dental Journal*, 1977, **142**. In press.
[19] Rugg-Gunn, A J, *et al*, *British Dental Journal*, 1975, **139**, 351.