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Editorial

Beyond Burkitt—is diverticular disease more than just cereal fibre deficiency?

Diverticular disease is a common disorder in the Western world affecting between 30% to 55% of the population over 60 years of age.^{1,2} Although most diverticula remain asymptomatic, their high frequency means complications are a common clinical problem. About 6500 patients per year are admitted to British hospitals for treatment of diverticulitis,³ although the number with life threatening complications such as perforation is unknown. Furthermore, many more patients require treatment for milder disease as an outpatient and undergo investigations to confirm the diagnosis.

The most well known and substantiated theory on the aetiology of diverticular disease is that of a deficiency of dietary cereal fibre. This hypothesis was advanced by Painter and Burkitt who observed that diverticular disease was uncommon in African countries where dietary fibre consumption was high.⁴ They contrasted this with Western countries with a higher incidence of disease and a lower consumption of cereal fibre and stated that “the refining of flour and other cereals is the primary cause of diverticulosis”.⁴ This theory is biologically plausible and is substantiated by epidemiological investigations,^{5,6} animal experiments,⁷ and a trial of fibre replacement.⁸ However, the few available studies show that two other dietary factors, which have received little attention, may be important. These are excess red meat^{5,9} and fibre deficiency from both fruit⁹ and vegetables,^{5,6,9} all of which increase the risk of symptomatic disease.

Beef meat consumption nearly doubled the risk of symptomatic disease and lamb meat consumption almost quadrupled it in a large case-control study.⁵ This finding was confirmed in a large cohort study of 48 000 US male health professionals where red meat significantly increased the risk by 1.5 times.⁹ Both these studies showed a persistence of the effect of red meat after correcting for fibre intake.^{5,9} This epidemiological evidence would be strengthened by a plausible biological mechanism as to how red meat induces symptomatic diverticular disease. Currently this is unknown, although it would be valid to investigate whether aromatic heterocyclic amines, produced by cooking red meat, are involved. These compounds can induce neoplasia in animal models of colonic cancer,¹⁰ although whether they provoke

diverticular inflammation is unknown. The validity of investigating this hypothesis is supported by the finding that cooked white meat and fish,¹¹ which contain less amine than red meat, are not associated with diverticular disease.^{5,9}

Fibre deficiency from fruit and vegetables appears to be at least as important in the aetiology of diverticular disease as a lack of cereal fibre. The case-control study reported similar protective and independent effects of several foods containing either cereal or vegetable fibre.⁵ Surprisingly, the cohort study found that fruit and vegetable fibre was protective but not fibre from cereals.⁹ The former reduced the risk of symptomatic disease by about a third. The data from the prospective study are more reliable as they eliminates recall bias of diet by measuring food intake before the development of disease. Following the unexpected results of the cohort study, further investigations are required to see if it is actually the fibre from fruit and vegetables which is important or some other constituent of these foods such as vitamins.

The epidemiological studies of diverticular disease may have implications for patients with symptomatic disease. Most clinical studies have concentrated on treating patients with predominantly cereal fibre often with bran or ispaghula supplements.^{12–14} These interventions are supported by physiological evidence showing that bran reduces intraluminal pressures,¹⁵ by data from uncontrolled trials,^{12,16} and a randomised trial of bran supplementation.⁸ Although bran is undoubtedly beneficial, it does not abolish all symptoms^{8,12,14,16,17} and now may be the time to investigate if other dietary modifications have a role such as a restriction of red meat. Furthermore, although all clinicians would encourage their patients to increase their cereal fibre intake, it is unknown whether advice is given to consume more fruit and vegetables.

Recently there has been little research on diverticular disease, although the epidemiological and trial evidence suggests this is an area to develop. This would require cohort studies investigating aetiology and randomised controlled trials of different dietary modifications. Such information may expand Burkitt and Painter’s pioneering

work and reduce the incidence in the new millennium of what they called a 20th century disease.¹⁸

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Medical Anniversary

Dorothy Hodgkin, 17 May 1910

Dorothy Mary Hodgkin (nee Crowfoot) (1910-94) was born near Beccles and was educated at Somerville College, Oxford. She became an *x* ray crystallographer, and this new technique enabled her to discover the structure of penicillin, vitamin B12, and insulin. She was awarded the Gold Medal of the Royal Society (1956) and the Nobel Prize for chemistry (1964), and became a member of the Order of Merit (1965). She held a Royal Society Research Professorship at Oxford (1960-77) and was Chancellor of Bristol University (1970-88). Her husband Thomas died in 1982 and she died on 29 July 1994. She is survived by two sons and one daughter—*D G James*