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Because we receive many more letters than we have room to publish we may shorten those that we do publish to allow readers as wide a selection as possible. In particular, when we receive several letters on the same topic we reserve the right to abridge individual letters. Our usual policy is to reserve our correspondence columns for letters commenting on issues discussed recently (within six weeks) in the *BMJ*.

Letters critical of a paper may be sent to the authors of the paper so that their reply may appear in the same issue. We may also forward letters that we decide not to publish to the authors of the paper on which they comment.

Letters should not exceed 400 words and should be typed double spaced and signed by all authors, who should include their main degree.

Acute appendicitis and dietary fibre

SIR,—Professor D J P Barker (13 April, p 1125) has propounded an alternative hypothesis to that which incriminates fibre depleted diets in the pathogenesis of acute appendicitis. He suggests that acute appendicitis is primarily a response to systemic infection with viruses or bacteria in an appendix in which the lumen has been narrowed by lymphoid hyperplasia in the wall. This was postulated to have occurred because of a decreased incidence of childhood infections, which changed the pattern of immunity. This probably occurred in Britain towards the end of the last century.

Professor Barker suggested that if the dietary fibre hypothesis was disproved this disorder should no longer be deemed to be a Western disease. This statement reflects a serious misunderstanding of the definition of Western diseases. They have not been defined in terms of dietary change or of deficiency in fibre. They have been defined as those diseases characteristic of modern affluent Western technological communities. Diseases such as cancer of the lung and breast, also pernicious anaemia, were included in a list of 25 diseases.¹ Professor Barker, so far as I know, is the first person to challenge this list. However, he agreed that appendicitis had a low incidence in certain non-industrialised countries but that the incidence had increased in their affluent urban communities. I submit that Professor Barker has acknowledged that appendicitis is a Western disease.

He presented data which he summarised by stating that "daily total fibre consumption per person did not differ between 1880 (when appendicitis was a rare disease in Britain) and 1970 (when it was a common disease)." This is depicted in his fig 3. Clearly the veracity of this statement depends on the accuracy of the 1880 dietary fibre figure. The 1880 dietary fibre intakes had been reported thus: cereals 12.2 g/day per person and vegetables 9.6 g/day per person, a total of 21.8 g/day per person.² This is not the correct figure, for a footnote in the original table stated that total dietary fibre intake in 1880 should be reported as 28.3 g/day per person, nearly one third higher. The original lower figure had not included fibre from oatmeal and rice or from fresh vegetables. To fill these gaps figures were "borrowed" from those of 1909, some 29 years later, because this was the first year to report intakes of

Dietary fibre intakes (g/day per person) from 1860 to 1970

Year(s)	Total	Cereal	Potato	Other
1860	37.47	22.32	10	5.1
1880	28.6	13.9	9.6	5.1
1938	22.3	9.2	6.1	7.0
1942-4	32.39.6	18.7.24.6	7.3.8.9	6.0.7.2
1970	22.7	8.1	7.3	7.3

these foods. Small wonder that the diet of Welsh farm workers in 1870, which included oatmeal and fresh vegetables, reported total dietary fibre intakes of 65 g/day per person in 1870 but 21 g/day per person in 1977.³

The most recent assessment of British dietary fibre intakes in 1860-1970 has been reported as shown in the table,⁴ indicating that fibre consumption in 1880 was significantly higher than that in 1970.

I agree with Professor Barker that the lack of response of the appendicitis mortality rate trends during the years of the wartime national flour 1941-53 (his fig 2) remains inexplicable by the present dietary fibre hypothesis. A very much greater change in fibre intake is necessary to alter significantly the incidence of appendicitis; the relatively small increase in appendicitis in urban compared with rural black South Africans would support this.

Perhaps the two different hypotheses are beginning to converge. It has been noted that moderate voluntary increases of fibre containing foods have not reduced the incidence of appendicitis.⁵ On the other hand, very high intakes of dietary fibre, especially that associated with the starch containing foods, have been associated with extremely low rates of appendicitis.

In sub-Saharan Africa appendicitis has remained rare in rural African communities, in which dietary fibre intakes usually exceed 100 g/day per person.⁴ The disease becomes more common but far below Western rates in urban African communities eating partially Westernised diets. These contain smaller amounts of the starchy foods, which are usually refined and have lower fibre content.⁴

The narrow lumen of the appendix hypothesis has three weak points. Firstly, the hypothesis was propounded by Bohrod in 1946 but still lacks confirmation by pathologists who can measure the

lumen of appendices removed at operation. Secondly, Professor Barker suggested that the decreasing incidence of childhood infections towards the end of the last century in Britain led to increasing lymphoid hyperplasia and this narrowed the lumen of the appendix. This might explain the rise in the appendicitis rates about 1880. It is unlikely to explain the steeply falling mortality rates that started in the 1940s and have continued ever since; these might be attributed to the advent of chemotherapy in the late 1930s. Presumably childhood infections have decreased during most decades of the present century. Thirdly, it has been stated by more than one doctor that lymphoid hyperplasia in the appendix is maximal in the second decade of life, but appendicitis mortality rates have been slightly lower in the age group 5-24 years than the average rate, shown in the all ages group, during 1931-80 (Barker's fig 2).

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- 1 Trowell HC, Burkitt DP, eds. *Western diseases: their emergence and prevention*. London: Edward Arnold, 1981.
- 2 Southgate DAT, Bingham S, Robertson J. Dietary fibre in the British diet. *Nature* 1978;274:51-2.
- 3 Hughes RE, Jones E. A Welsh diet for Britain? *Br Med J* 1979;i:1145.
- 4 Bingham S. Dietary fibre intakes. In: Trowell HC, Burkitt DP, Heaton KW, eds. *Dietary fibre, fibre-depleted foods and disease*. London: Academic Press (in press).
- 5 Walker A, Burkitt D. Appendicitis. In: Trowell HC, Burkitt DP, Heaton KW, eds. *Dietary fibre, fibre-depleted foods and disease*. London: Academic Press (in press).

** Professor Barker replies below.—ED, *BMJ*.

SIR,—I wrote that "daily total fibre consumption per person did not differ greatly in 1880 and 1970." Even when I allow the new data which Dr Trowell quotes, or the footnote to the original data, this conclusion still stands. The difference between the 1880 and 1970 levels in Britain is small in relation