Another 20th century epidemic

Sir,

I greatly enjoyed Dr David Grimes’ review ‘An epidemic of coronary heart disease’.1 An epidemic of a completely different disease, duodenal ulcer, occurred during almost exactly the same time, which seems rather unlikely to have been a coincidence. David Grimes describes the fact that coronary heart disease was rare before the first world war, then became increasingly common in the 1920s and after peaking in the late 1960s, has declined in incidence steadily ever since. Duodenal ulcer disease shows a very similar time scale.

Duodenal ulcers were rare before the early 1900s. Although this was partly due to the lack of radiological diagnosis, dramatic events such as perforation or fatal haemorrhage were obvious to doctors, and such events provide the most reliable data for estimating ulcer incidence around a 100 years ago. The work of surgeons such as Moynihan2 made it possible to recognize the typical symptoms. William Osler, then working in Johns Hopkins Hospital, Baltimore, wrote in the third edition of his textbook of medicine, published in 1898, ‘The disease is much less common in some countries than in others, and in some parts of this country. It is certainly less frequently seen in Baltimore than in Massachusetts or in Canada. In 9 years there were in my wards only 25 instances with a diagnosis of ulcer’.3 The condition was so uncommon at this time as to warrant the publication of individual case reports and series of cases collected from the literature.2,4,5 There was soon a sudden and dramatic increase in duodenal ulcer incidence, which in the UK and Scandinavia, where it was best documented, started around 1920. Within a few years, the number of patients presenting with a perforated duodenal ulcer doubled in the West of Scotland,7 and tripled in Scandinavia.8,9 In England, duodenal ulcer incidence increased ‘by 300–600 percent’ between 1920 and 1950.10 Duodenal ulcer deaths in men in England and Wales rose from 404 in 1911 (the first year in which duodenal ulcer was classified separately in the Registrar General’s statistics) to 1847 in 1950.6 These mortality data provide the best evidence of the time of onset and the speed of the rise in duodenal ulcer incidence in the UK, which mostly occurred over a period of only 15 years, between 1920 and 1935. There was a huge difference in the number of servicemen invalidated out of the British Services in the two World Wars because of peptic ulcers, increasing from 709 in 1914–15 to 23,574 in 1939–41.11 The incidence of duodenal ulcer in the UK reached a peak in the 1950s, at which time it was estimated that nine percent of 4117 medical and surgical beds in 12 London hospitals were occupied by patients with peptic (mostly duodenal) ulcers, and ~10% of the population had an ulcer.10 The incidence has been declining steadily ever since then, but at a slower rate than the previous rate of rise.12–16

Since most duodenal ulcers are caused by chronic infection with Helicobacter pylori,17–21 there is a close correlation between the prevalence of this infection in a community and the incidence of duodenal ulcer.22 The decline in duodenal ulcer incidence seen in many countries in recent years is happening because the number of adults with H. pylori infection is falling, and has been doing so (without any medical intervention) for many years. In North-East Bristol, UK, the proportion of the local population with H. pylori infection is spontaneously decreasing at such a rate that soon Helicobacter-related duodenal ulcers will have almost disappeared in this area.22 This pattern of spontaneous disappearance of the condition seems to be exactly the same as that described by Dr Grimes for coronary heart disease.

Is it possible that these two dramatic 20th century epidemics could be connected? David Grimes poses the question ‘has coronary heart disease been due to an environmental biological factor, which is a micro-organism, a bacterium or a virus?’ Could that micro-organism be H. pylori, or could some environmental change have introduced another micro-organism in parallel to H. pylori?

Helicobacter pylori is usually acquired in the early years of life,23,24 and then persists as a chronic infection of the gastric mucosa. As H. pylori organisms can be detected in the stools, the infection is probably transmitted mainly by the faecal–oral route.25,26 There is some transmission within families, especially from mother to child and between siblings.25–28 Poor social conditions, crowded living and increased numbers of siblings all increase the risk of acquiring H. pylori infection. However, if a rapid increase in the rate of H. pylori infection was the cause of the subsequent epidemic of duodenal ulcer disease, within-family transmission alone could not have accounted for this, and there must have been exposure to some common external source of infection, for example infected drinking water.29,30
Chronic infection has been suggested as part of the pathogenesis of atherosclerosis,\textsuperscript{31} and perhaps the duodenal ulcer story resonates with this.

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References

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