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The epidemiology of foodborne disease in England and Wales

FOUR changes in the epidemiology of foodborne diseases were much publicized in 1989: the epidemic of salmonellosis due to *Salmonella enteritidis* phage type 4, the rise in campylobacter enteritis, the discovery of food as a vehicle of infection in some cases of listeriosis and the large outbreak of botulism in north west England and Wales. However, other important changes took place during the 1980s which attracted less public attention: outbreaks of viral foodborne disease became common, yersiniosis increased, haemorrhagic colitis was first recorded and giardiasis and cryptosporidiosis came into prominence. The main food and waterborne diseases of the past remained rare and those cases that were documented were usually imported.

Salmonellosis is the most important foodborne disease in England and Wales, measured in overall morbidity and mortality. In 1989 there were over 25 000 laboratory reported infections and 57 (provisional) registered deaths. Although usually a mild gastrointestinal infection and often symptomless, about 1.5% of reported cases develop systemic complications such as septicaemia, meningitis and bone and joint abscesses.[1] In the last decade there have been two major epidemics. The first, in the early 1980s, involved *Salmonella typhimurium*. This has now declined and was probably caused mainly by bovine infection.[1] The second epidemic, unprecedented in scale, involved *S enteritidis* phage type 4. This emerged in the mid 1980s and was caused by infection in poultry.[2] Sixty per cent of chickens bought in retail outlets, and the yolk of one in 1000 intact eggs from flocks implicated in outbreaks were shown to be contaminated.[2] Many outbreaks were traced to poultry meat and shell eggs, and case-control studies demonstrated an association of sporadic cases with these foods.[2] Extensive veterinary control measures were introduced[3] and wide publicity given to food hygiene. Although it is too early to assess the effect of these measures it is encouraging that the increase in the epidemic in 1989 and early 1990 was substantially less than that in 1988.

Campylobacter enteritis is now the most common laboratory diagnosed cause of acute gastrointestinal infection — in 1989 there were nearly 33 000 reports in England and Wales. Reports increased as new tests were developed and became widely available in the late 1970s, but the more than threefold rise during the 1980s was probably partly due to a real increase in the incidence of the infection. Although there were a few milkborne and waterborne outbreaks and some were traced to poultry meat, sporadic cases in which no source was identified made up over 95% of reported infections. There is increasing evidence, however, that many of these sporadic cases were caused by poultry, mainly by cross-contamination of other foods eaten raw or without further cooking.[4] The origin of the contamination was probably the intestinal contents of poultry as most of the birds in some broiler flocks were infected. A study in the south of England suggested that the infection was derived from the water supplies and water distribution systems in broiler houses, in which campylobacters were present in non-culturable forms.[5] If further studies confirm that water treatment and cleansing of water systems in broiler houses lead to a reduction of human infection in the area where the poultry is sold, it should be possible to introduce effective control measures at source.

Listeriosis is an uncommon disease usually causing septicaemia and/or meningitis in very young, elderly and immunocompromised people, with an overall fatality rate of about 30% (higher in neonates). Infection is often symptomless. In pregnant women a mild febrile illness may occur which can be followed by fetal infection. In England and Wales laboratory reported infections increased threefold in the 1980s to nearly 300 in 1988, but declined in 1989 and 1990. Four large confirmed foodborne outbreaks were reported outside the UK in the 1980s; one due to raw cabbage in coleslaw salad, one to milk and two to soft cheese. In the UK, however, there was only one cluster of 11 cases, in Cumbria in 1981 which was possibly foodborne; a case control study suggested an association with cream, and in four sporadic cases food was confirmed as the cause — soft cheese in two cases, retail cooked chilled chicken in one case and vegetable rennet in the other.[6]

Surveys have demonstrated that *Listeria monocytogenes* is widespread in the environment so that the isolation of the organism from raw foodstuffs was not surprising, but the recent finding of contaminated ready-to-eat foods was less expected and gave rise to public concern.[7] Some samples of soft cheese and pate contained large numbers of organisms and *L monocytogenes* serogroup 4, the serogroup most commonly isolated from humans, was also the serogroup found most frequently in pate. Although no epidemiological association was demonstrated with human disease, it is possible that the substantial decline in human listeriosis in late 1989 and 1990 may be due to people heeding advice to avoid eating foods that are a potential source of infection. Furthermore, as it is known that *L monocytogenes* will multiply slowly at refrigerator temperatures these foods may now be stored at lower temperatures and with a shorter 'shelf life' than in the past. Improvements in methods of food production have also probably taken place.

Botulism is a very rare disease in the UK — until the outbreak of 27 cases with one death in 1989 only 26 cases with 14 deaths had been reported since the early 1920s. The disease usually presents with nausea, vomiting, dry mouth, blurred vision or diplopia but in the recent outbreak some cases were mild and atypical.[8] The source of the outbreak, yoghurt containing hazelnut conserve, had not been previously associated with botulism but was identified with commendable speed. Nevertheless, the atypical clinical presentation, the novel source, and the wide geographical distribution of cases, made diagnosis difficult.[8] The epidemiology of botulism may be changing, as in the USA,[9] and clinicians should consider the diagnosis in patients with the acute onset of bulbar symptoms or descending paralysis and inform their public health physician colleagues promptly. Information on suspected cases can then be collated centrally and widespread outbreaks quickly detected and investigated.

The incidence of hepatitis A has declined during the past 20 years, but in the 1980s foodborne outbreaks were reported with increasing frequency, most of them traced to shellfish.[1] At the same time, outbreaks of viral gastroenteritis due to small round structured viruses including Norwalk virus also increased, again mainly caused by shellfish.[10] A possible explanation for this changing epidemiology is that the faecal–oral spread of these viruses in children has declined as standards of hygiene have improved, with the result that more people reach adulthood without becoming immune from natural infection, and therefore succumb to illness when exposed to sewage contaminated shellfish or other faecally contaminated foods.

During the 1980s yersiniosis, caused by *Yersinia enterocolitica,* has been increasingly recognized as a cause of gastrointestinal illness often associated with mesenteric adenitis, erythema nodosum and sometimes arthritis. Laboratory reported infections reached around 500 in England and Wales in 1989. Although cases that can be traced to food have only rarely been described in the UK, these have been reported frequently in other countries, where much greater increases in incidence have taken place.[11] The organism has similar growth characteristics to those of *L monocytogenes,* multiplying slowly at refrigerator temperatures, and the same relationship between prolonged refrigeration of food and the change in the epidemiology of the infection has been suggested.[1]

Haemorrhagic colitis is an apparently new disease caused by a vertoxin producing strain of *Escherichia coli,* usually serotype 0 157, and characterized by acute severe abdominal pain, bloody diarrhoea and little or no fever.[12] Several outbreaks and many sporadic cases have been reported in England and Wales, some of them associated with the haemolytic uraemic syndrome. Outbreaks in North America have been traced to beef products and milk suggesting that the infection is a zoonosis, and a recent study in Sheffield suggests that in the UK the infection may also originate from cattle.[13]

Giardiasis and cryptosporidiosis are both protozoal gastrointestinal infections which may be foodborne, although the spread by food has not been substantiated in the UK. Laboratory reports of giardiasis in England and Wales nearly doubled during the 1980s to over 6400 in 1989. Only one waterborne outbreak was detected.[14] Some cases were imported, but in most the source of infection was unknown. Cryptosporidiosis was first recognized in immunocompromised patients in the late 1970s and in previously healthy individuals in the 1980s.[15] In England and Wales laboratory reports rose to over 8000 in 1989. Although some cases were attributed to person to person transmission, some to contact with infected animals and some to importation, there was increasing evidence that many were waterborne.[15] The oocysts of cryptosporidium and the cysts of giardia are both resistant to normal levels of chlorine in water supplies, and as the cryptosporidium oocysts are smaller than the giardia cysts they are more likely to pass into the mains supply if filtration and flocculation are deficient.

In the past decade there have been between one and 10 reported cases of cholera in England and Wales annually, all of which were acquired abroad; no spread occurred. Over the same period there was an average of 170 cases of typhoid fever per year (87% infected abroad), 55 cases of paratyphoid A fever (95% infected abroad) and 43 cases of paratyphoid B fever (71% infected abroad). Although foodborne infection with these diseases is now a rarity in the UK, outbreaks still occur occasionally. For example, there was a large outbreak of paratyphoid B fever in 1988 following a dinner in Birmingham, the source of which was not discovered. Brucellosis has been almost eliminated since eradication of the disease in cattle in the 1970s; during the 1980s there was an average of 22 cases per year in England and Wales, more than half of them in people infected abroad.

Substantial changes in the epidemiology of foodborne disease have taken place in the last decade. Further changes are likely to occur in the future as microorganisms evolve, as new foods are produced, as novel methods of food production, storage, distribution and preparation are developed, and as social habits of populations alter. General practitioners are well placed to detect these disease changes and should always be alert to 'new' foodborne diseases. They should consult their public health physician colleagues whenever the possibility of foodborne disease arises, so that early epidemiological enquiries can be made and control measures quickly implemented.

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References

1. Galbraith NS, Barrett NJ, Sockett PN. The changing pattern of foodborne disease in England and Wales. *Public Health* 1987; **101:** 319-328.
2. Public Health Laboratory Service. Memorandum of evidence to the agriculture committee on salmonella in eggs. *PHLS Microbiology Digest* 1989; **6:** 19.
3. Corkish JD. Salmonella infections in poultry a statutory approach. *State Veterinary Journal* 1989; **43:** 188-192.
4. Skirrow MB. Campylobacter perspectives. *PHLS Microbiology Digest* 1989; **6:** 113-117.
5. Pearson AD, Colwell RR, Rollins D, *et al.* Prevention of *Campylobacter jejuni* transmission to broiler chickens by farm interventions. In: Kaijser B, Falsen E (eds). *Campylobacter IV: proceedings of the fourth international workshop on campylobacter infections.* Goteborg, Sweden: University Department of Clinical Bacteriology, 1988: 304-305.
6. Gilbert RJ, Hall SM, Taylor AG. Listeriosis update. *PHLS Microbiology Digest* 1989; **6:** 33-37.
7. Social Services Committee. *Food poisoning: listeria and listeriosis.* London: HMSO, 1989.
8. Critchley EMR, Hayes PJ, Isaacs PET. Outbreak of botulism in north-west England and Wales, June, 1989. *Lancet* 1989; **2:** 849-853.
9. MacDonald KL, Cohen ML, Blake PA. The changing epidemiology of adult botulism in the United States. *Am J Epidemiol* 1986; **124:** 794-799.
10. PHLS working party on viral gastroenteritis. Foodborne viral gastroenteritis. *PHLS Microbiological Digest* 1988; **5:** 69-75.
11. Cover TL, Aber RC. *Yersinia enterocolitica. N Engl J Med* 1989; **321:** 16-24.
12. Symonds J. Haemorrhagic colitis and *Escherichia coli* 0 157 — a pathogen unmasked. *Br Med J* 1988; **296:** 875-876.
13. Chapman PA, Wright JD, Norman P. Verotoxin producing *Escherichia coli* infection in Sheffield: cattle as a possible source. *Epidemiology Infection* 1989; **102:** 439-445.
14. Jephcott AE, Begg NT, Baker IA. Outbreak of giardiasis associated with mains water in the United Kingdom. *Lancet* 1986; **1:** 730-732.
15. Casemore DP. Epidemiological aspects of cryptosporidiosis. *Epidemiology Infection* 1990; **104:** 1-28.

Race, ethnicity and general practice

THE post-war industrial boom in the UK attracted systematic and large scale migration from the ex-colonies of the new commonwealth. Earlier migration had been largely from eastern Europe and Ireland. The context of black migration is important; the new immigrants were used to an inferior status under British rule and had a similar status when they arrived in the UK. Three decades later their relative status has changed little.[1-3]

The terms 'race' and 'ethnicity' require definition. 'Race' refers to the concept that physical, intellectual and behavioural characteristics are inherited and is based on interpretations which allow people of 'specific genetic stock' to be placed in positions of inferiority or superiority. Thus wrote the eighteenth century philosopher David Hume: 'I am apt to suspect the negroes ... to be naturally inferior to whites'.[4] Social Darwinism and the church gave these theories intellectual and moral respectability. However, sociologists have rejected the idea that human groups can be unambiguously defined in terms of their genetic make up. In sociological theory such groups are more commonly defined by reference to shared culture, such as language, customs and institutions,[5] and are referred to as 'ethnic' groups.

There is debate on the appropriate level for defining ethnicity. For example, the concept of a 'Pakistani' ethnicity[6] is apparently inconsistent with the multitude of linguistic, cultural and geographical differences between the various groups of Pakistani origin in the UK. Conversely, those originating from the West Indies and the Indian sub-continent may adopt a politically significant 'black' ethnicity, based on shared experience of racism and racial discrimination. In this context the idea of multi-level ethnic affiliation is useful, with affiliation changing according to circumstances.

In epidemiological studies 'ethnicity' has variously been based on country of birth,[7] interviewer observation,[8] name and religion,[9] or 'self-definition'.[10] Although there is no one appropriate definition of ethnic origin or a guaranteed method for gaining this information, the first two approaches are particularly fraught with problems. The history of official data on ethnic origin is stormy, with changes in philosophy and debate on the need for and use of such data.[1] One criticism is that these data have rarely been used to the benefit of ethnic minorities. The 1991 census proposes to ask an elaborate (but not uncontroversial) question on ethnic origin; significantly, it has the heading 'Racial discrimination and disadvantage'.

Race and ethnicity are important in the context of health and the use of health care. There are diseases (such as thalassaemia) which are more prevalent in some 'racial' groups than others. Concepts of health and illness, attitudes to medicine and health professionals, and the use of health services differ between cultures. In the British context, cultural differences have predominated in explanations of health inequalities between the white majority and ethnic minority groups.[11] The far greater similarities between cultures, and the importance of race and social deprivation in producing and perpetuating health inequalities have largely been ignored.[12,13]

Ethnicity and race are not simply indices of horizontal stratification but are related to socioeconomic status. There is considerable evidence of racism and, despite legislation to the contrary, racial discrimination in employment, housing, education, the judicial system and the workings of immigration policies.[1-3] Ethnic inequalities in health need to be considered against this background.

The relationship between social status and health, as well as the provision of health care, is well documented.[14] The traditional diseases of poverty such as vitamin D deficiency and tuberculosis now have a relatively high prevalence in the socially deprived groups of Asian origin.[12,13] There is a considerable difference between infant and child health in white and black populations in the UK. Differences in the prevalence of heart disease or mental health likewise cannot be accounted for by cultural or behavioural differences; for the latter, differential diagnosis along racial lines is also an important issue.

Primary care is usually the first line of contact with the health services, and most general practitioners in large industrial towns in the UK have some patients from ethnic minorities. Sensitive care by the general practitioner and the primary care team is of paramount importance and this is aided by knowledge of the culture, religion and socioeconomic status of ethnic minority patients. However, caution is required to avoid prejudgement, prejudice or stereotyping. 'All Englishmen wear bowler hats' is an absurd stereotype; yet similar stereotypes, of Asians having