INTESTINAL INFECTION

# Trends in indigenous foodborne disease and deaths, England and Wales: 1992 to 2000

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**Background:** Commitment to food safety is evidenced by high profile governmental initiatives around the globe. To measure progress towards targets, policy makers need to know the baseline from which they started.

**Aim:** To describe the burden (mortality, morbidity, new presentations to general practice, hospital admissions, and hospital occupancy) and trends of indigenous foodborne disease (IFD) in England and Wales between 1992 and 2000.

**Methods:** Routinely available surveillance data, special survey data, and hospital episode statistics were collated and arithmetic employed to estimate the burden and trends of IFD in England and Wales. Adjustments were made for underascertainment of disease through national surveillance and for foreign travel. The final estimates were compared with those from the USA.

**Results:** In 1995 there were an estimated 2 365 909 cases, 21 138 hospital admissions, and 718 deaths in England and Wales due to IFD. By 2000 this had fallen to 1 338 772 cases, 20 759 hospital admissions, and 480 deaths. In terms of disease burden the most important pathogens were campylobacters, salmonellas, *Clostridium perfringens*, verocytotoxin producing *Escherichia coli* (VTEC) O157, and *Listeria monocytogenes*. The ratio of food related illness in the USA to IFD in England and Wales in 2000 was 57:1. Taking into account population rates, this ratio fell to 11:1 and converged when aetiology and disease severity were considered.

**Conclusion:** Reducing IFD in England and Wales means tackling campylobacter. Lowering mortality rates however also requires better control and prevention of salmonellas, *Cl perfringens*, *L monocytogenes*, and VTEC O157.

The UK Food Standards Agency (FSA) has set a target of reducing foodborne illness by 20% by 2006.[1] In order to monitor progress, and to set priorities for the development of control strategies, the FSA requires reliable measures of the burden (morbidity, health service usage, and mortality) of indigenous foodborne disease (IFD).

Previous attempts to estimate the burden of foodborne disease were limited by using data from unrepresentative sources,[2] failing to account for the diversity of pathogens causing foodborne disease,[3–6] or through reliance on expert opinion.[7] The Centers for Disease Control and Prevention (CDC) developed an approach overcoming these limitations, producing pathogen specific morbidity, hospital admission, and mortality estimates for foodborne infections in the USA.[8] We have refined the CDC method to account for imported infections and to describe recent trends in the burden of IFD in England and Wales.

## METHODS

### Sources of data

Data sources are shown in box 1.

### Estimating all infectious intestinal disease (IID)

The IID study[13] established that one in every 5.8 cases of IID in the population present to general practice. This ratio was applied to the annual rates of presentation to general practice for IID[10] to produce estimates for the annual number of cases of all IID in England and Wales. The figure for 1995 was calculated using the point estimate for the rate of IID in the community from the IID study.

### Aetiology

Only pathogens causing gastrointestinal symptoms,[13 18] and therefore likely to be diagnosed as food poisoning by clinicians in England and Wales, were included in these analyses (box 2). Foodborne botulism and *Trichinella spiralis* infection were excluded because of their extremely low incidence.

Pathogen specific rates for illness in the population were derived from the IID study.[13] These were used to estimate all illness in England and Wales in 1995 due to each pathogen (box 3, worked example, salmonellas, step 1). Ascertainment ratios (AR), or multipliers, were then calculated by dividing the estimated number of cases due to each pathogen by the corresponding number of LabBase[9] reports received in 1995 (box 3, step 2). For each of the other years, the total number of cases due to each pathogen was calculated by multiplying the annual total of laboratory reports by the appropriate AR (box 3, step 3). Annual figures for total IID of unknown aetiology were calculated by subtracting the total number of cases of disease due to known pathogens from the estimate for all IID.

### Adjusting for travel associated infection

Data from LabBase and special studies[14–16] were used to determine the percentage of travel associated infection for each

**Abbreviations:** AR, ascertainment ratio—ratio of the estimated number of cases of illness in the population due to specific pathogens to the number of laboratory reports in the national database for laboratory confirmed infections; CDC, United States Centers for Disease Control and Prevention; CDSC, PHLS Communicable Disease Surveillance Centre; HES, hospital episode statistics; GSURV, national database for the surveillance scheme for general outbreaks of infectious intestinal disease; FSA, UK Food Standards Agency; IFD, indigenous foodborne disease; IID, infectious intestinal disease; LabBase, national database for laboratory confirmed infections; NLV, Norwalk-like viruses; NS, national statistics; PHLS, Public Health Laboratory Service; py, person years; RCGP, Royal College of General Practitioners; VTEC, verocytotoxin producing *Escherichia coli*.

**Table 1** Estimated cases of infectious intestinal disease and cases, hospitalisations, and deaths due to indigenous foodborne disease in England and Wales in 1995

| | Infectious intestinal disease | | | | | | Infectious foodborne disease | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Pathogen | Incidence/per 1000 person years | Estimated cases | Laboratory reports | AR | Indigenous (%) | Indigenous cases | Foodborne (%) | Cases | GP presentations | Hospital admissions | Hospital bed days | Deaths |
| Bacteria | | | | | | | | | | | | |
| *Aeromonas* spp | 12.4 | 642 557 | 635 | 1011.9 | 67.1 | 431 069 | 0 | 0 | 0 | 0 | 0 | 0 |
| *Bacillus* spp | 0.4 | 20 628 | 87 | 237.1 | 100.0 | 20 628 | 100.0 | 20 628 | 8251 | 50 | 129 | 0 |
| *Campylobacter* spp | 8.7 | 451 315 | 43 817 | 10.3 | 78.4 | 353 831 | 79.7 | 281 826 | 134 203 | 13 286 | 49 159 | 67 |
| *Clostridium perfringens* | 2.4 | 124 385 | 342 | 363.7 | 100.0 | 124 385 | 94.4 | 117 370 | 61 774 | 494 | 7314 | 124 |
| *Cl difficile* cytotoxin | 1.6 | 82 771 | 7664 | 10.8 | 100.0 | 82 771 | 0 | 0 | 0 | 0 | 0 | 0 |
| VTEC O157 | 0.03 | 1584 | 792 | 2.0 | 88.2 | 1396 | 63.0 | 879 | 879 | 333 | 1899 | 19 |
| Non O157 VTEC | 0.003 | 176 | 0 | n/c | 88.2 | 155 | 63.0 | 98 | 98 | 37 | 212 | 2 |
| Other *Escherichia coli* | 19.47 | 1 008 940 | 0 | n/c | 75.0 | 756 705 | 8.2 | 62 050 | 13 850 | 319 | 1561 | 6 |
| *Listeria monocytogenes* | 0.003 | 170 | 85 | 2.0 | 100.0 | 170 | 99.0 | 168 | 168 | 168 | 3007 | 59 |
| Salmonellas non-typhoidal | 2.2 | 115 904 | 29 719 | 3.9 | 88.2 | 102 227 | 91.6 | 93 651 | 66 894 | 3412 | 19 787 | 268 |
| *S paratyphi* | 0.01 | 340 | 170 | 2.0 | 26.0 | 90 | 80.0 | 72 | 72 | 23 | 144 | 0 |
| *S typhi* | 0.01 | 530 | 265 | 2.0 | 26.0 | 138 | 80.0 | 110 | 110 | 45 | 306 | 0 |
| *Shigella* spp | 0.27 | 13 984 | 4113 | 3.4 | 75.0 | 10 488 | 8.2 | 860 | 860 | 20 | 103 | 0 |
| *Staphylococcus aureus* | 0.27 | 13 989 | 59 | 237.1 | 100.0 | 13 989 | 96.0 | 13 429 | 5372 | 338 | 406 | 0 |
| *Vibrio cholerae* O1/O139 | 0.0004 | 20 | 10 | 2.0 | 0 | 0 | 90.0 | 0 | 0 | 0 | 0 | 0 |
| *V cholerae* non O1/O139 | 0.02 | 880 | 44 | 20.0 | 11.4 | 100 | 90.0 | 90 | 45 | 4 | 14 | 0 |
| Other vibrios | 0.01 | 680 | 34 | 20.0 | 41.2 | 280 | 65.0 | 182 | 91 | 3 | 10 | 1 |
| *Yersinia* spp | 6.8 | 352 374 | 281 | 1254.0 | 92.5 | 326 040 | 90.0 | 293 436 | 25 080 | 1404 | 12 359 | 8 |
| | | 2 831 227 | 88 117 | | | 2 224 463 | | 884 849 | 317 655 | 19 935 | 96 409 | 554 |
| Parasites | | | | | | | | | | | | |
| *Cryptosporidium parvum* | 0.81 | 42 017 | 5678 | 7.4 | 95.00 | 39 916 | 5.6 | 2219 | 1168 | 42 | 156 | 4 |
| *Cyclospora cayatenensis* | 0.04 | 1976 | 52 | 38.0 | 26.92 | 532 | 90.0 | 479 | 252 | 1 | 5 | 0 |
| *Giardia duodenalis* | 0.54 | 28 290 | 6150 | 4.6 | 79.19 | 22 402 | 10.0 | 2240 | 1179 | 7 | 24 | 0 |
| | | 72 283 | 11 880 | | | 62 850 | | 4938 | 2599 | 50 | 185 | 4 |
| Viruses | | | | | | | | | | | | |
| Adenovirus 40/41 | 3 | 155 456 | 158 | 983.9 | 100.0 | 155 456 | 0 | 0 | 0 | 0 | 0 | 0 |
| Astrovirus | 3.8 | 196 915 | 273 | 721.3 | 100.0 | 196 915 | 10.7 | 21 168 | 4811 | 14 | 56 | 5 |
| NLV | 12.5 | 647 701 | 2351 | 275.5 | 100.0 | 647 701 | 10.7 | 69 628 | 11 052 | 44 | 172 | 11 |
| Rotavirus | 7.1 | 367 908 | 17 112 | 21.5 | 100.0 | 367 908 | 2.5 | 9345 | 1558 | 48 | 126 | 5 |
| SLV | 2.2 | 114 002 | 138 | 826.1 | 100.0 | 114 002 | 0 | 0 | 0 | 0 | 0 | 0 |
| | | 1 481 981 | 20 032 | | | 1 481 981 | | 100 141 | 17 420 | 107 | 354 | 21 |
| Unknown agents | 117.3 | 6 078 512 | | | 86.4 | 5 251 834 | 26.2 | 1 375 981 | 174 175 | 1045 | 2926 | 139 |
| Total | | 10 464 004 | 120 029 | | | 9 021 129 | | 2 365 909 | 511 941 | 21 138 | 99 874 | 718 |

Totals are subject to the effects of rounding.
AR, ascertainment ratio—ratio of the estimated number of cases of illness in the population due to specific pathogens to the number of laboratory reports in the national database for laboratory confirmed infections; NLV, Norwalk-like viruses; SLV, Sappora-like viruses; VTEC, verocytotoxin producing *Escherichia coli*.

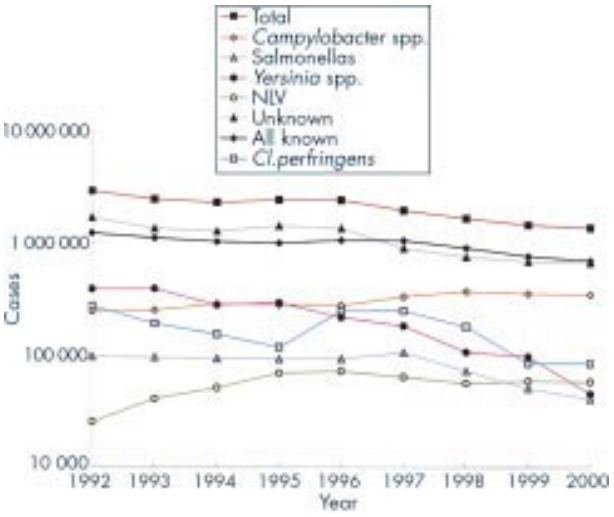


**Figure 1** Trends in indigenous foodborne disease in England and Wales, 1992–2000. NLV, Norwalk-like viruses.

pathogen (including infection of unknown aetiology). For each year the number of travel associated cases was subtracted from the total number of cases to produce pathogen specific estimates for indigenous cases (box 3, step 4).

### Estimating the number of cases of indigenous foodborne disease (IFD)

The pathogen specific percentage of foodborne transmission in outbreaks (includes foodborne plus person to person spread) from GSURV[9] was applied to the corresponding number of indigenous cases to produce pathogen specific estimates for IFD (box 3, step 5).

### Box 1 Data sources

(A) National database for laboratory confirmed infections[9] (LabBase)—for laboratory confirmed infectious intestinal disease (IID) and the proportion acquired abroad.
(B) National surveillance database for general outbreaks of IID[9] (GSURV) (n=4603; response rate=80%)—for the foodborne proportion of IID, hospital admissions, and deaths.
(C) Weekly Returns Service of the Royal College of General Practitioners[10] (RCGP)—for IID presenting to general practice.
(D) Hospital episode statistics[11] (HES)—for hospital admissions and bed occupancy.
(E) National Statistics[12] (NS)—population estimates.
(F) Study of Infectious Intestinal Disease in England (IID study)[13] (study population 495 666)—for adjusting LabBase data for underascertainment.
(G) Campylobacter sentinel surveillance scheme[14] (n=7630; response rate=76%)—for infection acquired abroad and hospital admissions.
(H) Enhanced surveillance of listeriosis in England and Wales[15] (n=409; response rate=75%)—for hospital admissions and deaths.
(I) A case control study of verocytotoxin producing *Escherichia coli* (VTEC) O157 infection in England[16] (n=369; response rate=84%)—for infection acquired abroad, hospital admissions, and deaths.
(J) The UK and Republic of Ireland Collaborative Study of Childhood Haemolytic Uraemic Syndrome[17] (n=413; response rate=100%)—for infection acquired abroad, hospital admissions, and deaths.
(K) Food related illness and death in the USA[8]—for international comparisons, and foodborne proportion of IID, hospital admissions, and deaths for certain pathogens.

### Cases of IFD presenting to general practice

Using the approach described above, data from the general practitioner component of the IID study[13] were used to produce pathogen specific estimates for new IFD consultations to general practice (box 3, step 6).

### Hospital admissions

Data from GSURV, HES, and special studies[11 14–16] were used to estimate pathogen specific hospital admission rates. These were applied to cases of IFD presenting to general practice to produce annual pathogen specific estimates of hospital admissions due to IFD (box 3, step 7).

### Hospital occupancy

Mean hospital stay (bed days) for each pathogen was derived from HES.[11] This was multiplied by the pathogen specific number of hospital admissions resulting from IFD to estimate the number of bed days (box 3, step 8).

### Deaths

Pathogen specific case fatality rates from GSURV and special studies[15] were applied to the corresponding number of cases of IFD presenting to general practice to derive annual estimates of deaths (box 3, step 9).

### Quality of evidence

Each of the above steps was classified according to whether the pathogen specific data elements used were direct measures, extrapolations, or inferences in order to evaluate the effects of potential biases on the final estimates produced.

### International comparison

To compare the CDC[8] and PHLS estimates, ratios of the rates of foodborne illness, hospital admissions, and death for all aetiologies, known pathogens, and known bacteria were calculated.

### Box 2 Pathogens

**Bacteria**
*Aeromonas* spp
*Bacillus* spp
*Campylobacter* spp
*Clostridium perfringens*
*Clostridium difficile* cytotoxin
VTEC O157
Non O157 VTEC
Other *Escherichia coli*
*Listeria monocytogenes*
Salmonellas (non-typhoidal)
*S paratyphi*
*S typhi*
Shigella spp
*Staphylococcus aureus*
*Vibrio cholerae* O1/O139
*Vibrio cholerae* non O1/O139
Other vibrios
*Yersinia* spp.

**Parasites**
*Cryptosporidium parvum*
*Cyclospora cayatenensis*
*Giardia duodenalis*

**Viruses**
Adenovirus 40/41
Astrovirus
Norwalk-like viruses
Sapporo-like viruses
Rotavirus

Unknown

**Box 3 Methods formulae**

**Example: salmonellas**
(1) All illness in England and Wales 1995
$N^{1995}=R^{1995}\times P^{1995}$
**115 904=2.24/1000×51 820 222**
$N^{1995}$=number of cases of illness in 1995
$R^{1995}$=rate of illness in the population in 1995 (IID study)
$P^{1995}$=estimated resident population mid 1995 of England and Wales (NS)
(2) Ascertainment ratio
$AR=N^{1995}/L^{1995}$
**3.9=115 904/29 719**
AR=ascertainment ratio
$L^{1995}$=laboratory reports to Public Health Laboratory Service (PHLS) in 1995 (LabBase)
(3) All illness in England and Wales in 2000
$N^{2000}=L^{2000}\times AR$
**58 640=15 036×3.9**
$L^{2000}$=laboratory reports to PHLS in 2000 (LabBase)
$N^{2000}$=number of cases of illness in 2000
(4) Indigenously acquired illness in England and Wales in 2000
$N^{2000,I}=N^{2000}\times(100-t)$
**45 427=58 640×77.5%**
$N^{2000,I}$=number of indigenously acquired cases of illness
t=percentage of illness that is travel associated (LabBase)
(5) IFD in England and Wales in 2000
$N^{2000,I,F}=N^{2000,I}\times f$
**41 616=45 427×91.6%**
$N^{2000,I,F}$=number of cases of IFD in England and Wales in 2000
f=percentage of illness that is foodborne (including foodborne plus person to person spread (GSURV))
(6) IFD presenting to general practice in England and Wales in 2000
$G^{2000,I,F}=N^{2000,I,F}\times g$
**29 726=41 616×71.4%**
$G^{2000,I,F}$=number of new presentations to general practice for IFD in England and Wales in 2000
g=percentage of cases of illness that present to general practice (IID study)
(7) Hospital admissions for IFD in England and Wales in 2000
$H^{2000,I,F}=G^{2000,I,F}\times h$
**1516=29 726×5.1%**
$H^{2000,I,F}$=number of hospital admissions for IFD in England and Wales in 2000
h=percentage of cases of illness presenting to general practice that are hospitalised (GSURV)
(8) Hospital occupancy (bed days) for IFD in England and Wales in 2000
$B^{2000,I,F}=H^{2000,I,F}\times b$
**8793=1516×5.8**
$B^{2000,I,F}$=number of hospital bed days occupied for IFD in England and Wales in 2000
b=mean number of bed days occupied per hospital episode (HES)
(9) Deaths due to IFD in England and Wales in 2000
$D^{2000,I,F}=G^{2000,I,F}\times d$
**119=29 726×0.4%**
d=percentage of cases of illness presenting to general practice that die (GSURV)

## RESULTS

### Overall disease burden (table 1)

There were an estimated 10 464 004 cases of IID in England and Wales in 1995. Nearly 14% were acquired abroad, leaving 9 021 129 indigenous IID cases. Of these, 2 365 909 (26.2%) were estimated to be IFD and 989 928 (41.8%) were attributable to known pathogens. Of the known pathogens, six were responsible for 92.7% of IFD—yersinias, campylobacters, *Cl perfringens*, non-typhoidal salmonellas (salmonellas), Norwalk-like viruses (NLV), and non-VTEC. In 1995, IFD resulted in 511 941 presentations to general practice, 21 138 hospital admissions, 99 874 hospital bed days, and 718 deaths.

### Trends in IFD (fig 1, table 2)

Between 1992 and 2000, IFD fell by 53.3% from 2 869 735 to 1 338 772 cases. In 1992, IFD of unknown aetiology (1 644 515), yersinias (392 753), and *Cl perfringens* (276 266) formed the majority of cases. By 2000 these had all declined sharply. Since 1997 there has also been a fall in salmonellas. IFD due to NLV infection rose by 125.5% and campylobacter by 45.0%.

### IFD presenting to general practitioners (table 2)

Campylobacters were the most common cause of IFD presenting to general practice in 2000.

### Hospital admissions (table 2)

The contribution of campylobacters rose from 54.8% to 81.6%. Salmonellas remained the second most common cause of hospital admission despite a 55.6% fall between 1995 and 2000. In 2000 VTEC O157 infection ranked third among known pathogens.

### Hospital bed occupancy (table 2)

Despite a decline of 19.6% overall, the contribution of campylobacters to bed occupancy rose. In 2000, salmonellas ranked second, *Cl perfringens* third, and *L monocytogenes* fourth for bed occupancy.

### Estimated deaths (table 2)

Deaths fell by 48.1% (principally salmonella and *Cl perfringens* deaths). *Listeria monocytogenes* ranked highly in terms of estimated deaths for the whole period.

### Potential effects of assumptions made on final estimates (table 3)

In general, the effects of extrapolation and inference on the final estimates would be relatively small except for campylobacters and unknown agents.

**Table 2** Estimated cases of infectious intestinal disease and cases, hospitalisations, and deaths due to indigenous foodborne disease in England and Wales in 1992 and 2000

| Pathogen | 1992 | | | | | | 2000 | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Laboratory reports | Cases | GP presentations | Hospital admissions | Hospital bed days | Deaths | Laboratory reports | Cases | GP presentations | Hospital admissions | Hospital bed days | Deaths |
| Bacteria | | | | | | | | | | | | |
| *Aeromonas* spp | 475 | 0 | 0 | 0 | 0 | 0 | 372 | 0 | 0 | 0 | 0 | 0 |
| *Bacillus* spp | 182 | 43 152 | 17 261 | 104 | 269 | 0 | 47 | 11 144 | 4458 | 27 | 70 | 0 |
| *Campylobacter* spp | 38 536 | 247 860 | 118 029 | 11 685 | 43 234 | 59 | 55 888 | 359 466 | 171 174 | 16 946 | 62 701 | 86 |
| *Clostridium perfringens* | 805 | 276 266 | 145 403 | 1163 | 17 216 | 291 | 245 | 84 081 | 44 253 | 354 | 5240 | 89 |
| *Cl difficile* cytotoxin | 1681 | 0 | 0 | 0 | 0 | 0 | 14 648 | 0 | 0 | 0 | 0 | 0 |
| VTEC O157 | 470 | 522 | 522 | 198 | 1128 | 11 | 896 | 995 | 995 | 377 | 2149 | 22 |
| Non O157 VTEC | 0 | 58 | 58 | 22 | 125 | 1 | 0 | 111 | 111 | 42 | 240 | 2 |
| Other *Escherichia coli* | 0 | 62 050 | 13 850 | 319 | 1561 | 6 | 0 | 62 050 | 13 850 | 319 | 1561 | 6 |
| *Listeria monocytogenes* | 106 | 210 | 210 | 210 | 3759 | 74 | 98 | 194 | 194 | 194 | 3473 | 68 |
| Salmonellas non-typhoidal | 31 733 | 99 260 | 70 900 | 3616 | 20 972 | 284 | 15 036 | 41 616 | 29 726 | 1516 | 8793 | 119 |
| *S paratyphi* | 125 | 16 | 16 | 5 | 32 | 0 | 154 | 85 | 85 | 27 | 170 | 0 |
| *S typhi* | 198 | 34 | 34 | 14 | 95 | 0 | 165 | 96 | 96 | 39 | 267 | 0 |
| *Shigella* spp | 18 069 | 3778 | 3778 | 87 | 452 | 2 | 966 | 202 | 202 | 5 | 24 | 0 |
| *Staphylococcus aureus* | 112 | 25 493 | 10 197 | 642 | 771 | 0 | 10 | 2276 | 910 | 57 | 69 | 0 |
| *Vibrio cholerae* O1/O139 | 9 | 0 | 0 | 0 | 0 | 0 | 12 | 0 | 0 | 0 | 0 | 0 |
| *V cholerae* non O1/O139 | 56 | 108 | 54 | 4 | 17 | 0 | 23 | 126 | 63 | 5 | 19 | 0 |
| Other vibrio species | 15 | 65 | 33 | 1 | 4 | 0 | 48 | 364 | 182 | 5 | 21 | 2 |
| *Yersinia* spp | 369 | 392 753 | 33 569 | 1880 | 16 543 | 10 | 42 | 45 144 | 3858 | 216 | 1901 | 1 |
| | 92 941 | 1 151 625 | 413 914 | 19 950 | 106 178 | 738 | 88 650 | 607 950 | 270 158 | 20 129 | 86 697 | 395 |
| Parasites | | | | | | | | | | | | |
| *Cryptosporidium parvum* | 5179 | 2024 | 1065 | 38 | 142 | 3 | 5278 | 2063 | 1086 | 39 | 145 | 3 |
| *Cyclospora cayatenensis* | 0 | 0 | 0 | 0 | 0 | 0 | 52 | 992 | 522 | 3 | 10 | 0 |
| *Giardia duodenalis* | 6830 | 2402 | 1264 | 8 | 26 | 0 | 3892 | 1673 | 881 | 5 | 18 | 0 |
| | 12 009 | 4426 | 2329 | 46 | 168 | 3 | 9222 | 4728 | 2488 | 47 | 173 | 3 |
| Viruses | | | | | | | | | | | | |
| Adenovirus 40/41 | 102 | 0 | 0 | 0 | 0 | 0 | 38 | 0 | 0 | 0 | 0 | 0 |
| Astrovirus | 447 | 34 660 | 7877 | 24 | 92 | 8 | 223 | 17 291 | 3930 | 12 | 46 | 4 |
| NLV | 865 | 25 618 | 4066 | 16 | 63 | 4 | 1951 | 57 781 | 9172 | 37 | 143 | 9 |
| Rotavirus | 16 281 | 8891 | 1482 | 46 | 119 | 4 | 16 454 | 8979 | 1497 | 46 | 121 | 4 |
| SLV | 178 | 0 | 0 | 0 | 0 | 0 | 52 | 0 | 0 | 0 | 0 | 0 |
| | 17 873 | 69 169 | 13 425 | 86 | 274 | 16 | 18 718 | 84 051 | 14 598 | 95 | 310 | 17 |
| Unknown agents | | 1 644 515 | 208 166 | 1249 | 3497 | 167 | | 642 043 | 81 271 | 488 | 1365 | 65 |
| Total | 122 823 | 2 869 735 | 637 834 | 21 331 | 110 117 | 924 | 116 590 | 1 338 772 | 368 516 | 20 759 | 88 545 | 480 |

Totals are subject to the effects of rounding.
NLV, Norwalk-like viruses; SLV, Sappora-like viruses; VTEC, verocytotoxin producing *Escherichia coli*.

**Table 3** Classification of evidence used, sources of data, and the effects of assumptions made and bias on final estimates

| Evidence | Incidence/AR Class[Sources] | Indigenous (%) Class[Sources] | Foodborne (%) Class[Sources] | Presenting to GPs (%) Class[Sources] | Hospital admissions (%) Class[Sources] | Hospital bed days (mean) Class[Sources] | Deaths (%) Class[Sources] | Principal assumption | Potential effects of bias on final estimates | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | | | Cases | Total burden |
| Bacteria | | | | | | | | | | |
| *Aeromonas* spp | Measured[A,F] | Measured[A] | Inferred[K] | n/c-nft | n/c-nft | n/c-nft | n/c-nft | Foodborne % | Moderate | Negligible |
| *Bacillus* spp | Measured[A,F] | Measured[A] | Measured[B] | Measured[F] | Measured[B] | Measured[D] | Measured[B] | — | Minor | Negligible |
| *Campylobacter* spp | Measured[A,F] | Measured[G] | Measured[B] | Measured[F] | Measured[G] | Measured[G] | Inferred[K] | Deaths % | Major | Major |
| *Clostridium perfringens* | Measured[A,F] | Measured[A] | Measured[B] | Measured[F] | Measured[B] | Measured[D] | Measured[B] | — | Moderate | Moderate |
| *Cl difficile* cytotoxin | Measured[A,F] | Measured[A] | Measured[B] | n/c-nft | n/c-nft | n/c-nft | n/c-nft | — | Negligible | Negligible |
| VTEC O157 | Extrapolated[A] | Measured[I] | Measured[B] | Extrapolated[A] | Measured[I,J] | Measured[D] | Measured[I,J] | Incidence | Negligible | Moderate |
| Non O157 VTEC | Inferred*[J] | Inferred* | Inferred* | Inferred* | Inferred* | Inferred* | Inferred* | Incidence | Negligible | Negligible |
| Other *Escherichia coli* | Measured[A,F] | Inferred† | Inferred† | Measured[F] | Measured[D] | Measured[D] | Measured[D] | Pathogenicity | Major | Moderate |
| *Listeria monocytogenes* | Extrapolated[H] | Measured[H] | Inferred[K] | Extrapolated[H] | Measured[H] | Measured[D] | Measured[H] | Incidence | Negligible | Moderate |
| Salmonellas non-typhoidal | Measured[A,F] | Measured[A] | Measured[B] | Measured[F] | Measured[B] | Measured[D] | Measured[B] | — | Moderate | Major |
| *S paratyphi* | Extrapolated[A] | Measured[A] | Inferred[K] | Extrapolated[A] | Measured[D] | Measured[D] | Inferred[K] | Incidence | Negligible | Negligible |
| *S enterica* typhi | Extrapolated[A] | Measured[A] | Inferred[K] | Extrapolated[A] | Measured[D] | Measured[D] | Inferred[K] | Incidence | Negligible | Negligible |
| *Shigella* spp | Measured[A,F] | Measured[A] | Measured[B] | Measured[F] | Measured[B] | Measured[D] | Measured[B] | — | Minor | Negligible |
| *Staphylococcus aureus* | Measured[A,F] | Measured[A] | Measured[B] | Measured[F] | Measured[B] | Measured[D] | Measured[B] | — | Minor | Negligible |
| *Vibrio cholerae* O1/O139 | Extrapolated[A] | Measured[A] | n/c-nia | n/c-nia | n/c-nia | n/c-nia | n/c-nia | — | None | None |
| *V cholerae* non O1/O139 | Measured[A,F] | Measured[A] | Inferred[K] | Measured[F] | Inferred[K] | Measured[D] | Inferred[K] | Foodborne % | Negligible | Negligible |
| Other vibrio species | Measured[A,F] | Measured[A] | Inferred[K] | Measured[F] | Inferred[K] | Measured[D] | Inferred[K] | Foodborne % | Negligible | Negligible |
| *Yersinia* spp | Measured[A,F] | Measured[A] | Inferred[K] | Measured[F] | Inferred[K] | Measured[D] | Inferred[K] | Pathogenicity | Major | Moderate |
| Parasites | | | | | | | | | | |
| *Cryptosporidium parvum* | Measured[A,F] | Measured[A] | Measured[B] | Measured[F] | Measured[B] | Measured[D] | Measured[B] | — | Minor | Minor |
| *Cyclospora cayatenensis* | Inferred[K] | Measured[A] | Inferred[K] | Inferred‡ | Inferred[K] | Inferred‡ | Inferred[K] | Incidence | Negligible | Negligible |
| *Giardia duodenalis* | Measured[A,F] | Measured[A] | Inferred[K] | Measured[F] | Measured[D] | Measured[D] | Measured[B] | Foodborne % | Negligible | Negligible |
| Viruses | | | | | | | | | | |
| Adenovirus 40/41 | Measured[A,F] | Measured[A] | Measured[B] | n/c-nft | n/c-nft | n/c-nft | n/c-nft | — | Negligible | Negligible |
| Astrovirus | Measured[A,F] | Measured[A] | Measured[B] | Measured[F] | Measured[B] | Measured[D] | Measured[B] | — | Negligible | Negligible |
| NLV | Measured[A,F] | Measured[A] | Measured[B] | Measured[F] | Measured[B] | Measured[D] | Measured[B] | — | Major | Moderate |
| Rotavirus | Measured[A,F] | Measured[A] | Measured[B] | Measured[F] | Measured[B] | Measured[D] | Measured[B] | — | Moderate | Moderate |
| SLV | Measured[A,F] | Measured[A] | Measured[B] | n/c-nft | n/c-nft | n/c-nft | n/c-nft | — | Moderate | Moderate |
| Unknown agents | Measured[A,C,F] | Inferred§ | Measured[B] | Measured[F] | Measured[B] | Measured[D] | Measured[B] | Indigenous % | Major | Major |

Total burden includes cases, healthcare usage, and deaths.
Data sources are from box 1 (see text).
*VTEC O157 data.
†*Shigella* spp data.
‡*Cryptosporidium parvum* data.
§All known pathogens.
n/c, not calculated; nia, not indigenous acquired; nft, no foodborne transmission.
AR, ascertainment ratio—ratio of the estimated number of cases of illness in the population due to specific pathogens to the number of laboratory reports in the national database for laboratory confirmed infections;
NLV, Norwalk-like viruses; SLV, Sappora-like viruses; VTEC, verocytotoxin producing *Escherichia coli*.

**Table 4** Morbidity and mortality due to indigenous foodborne disease caused by pathogens under surveillance in England and Wales in 2000

| | Cases | | GP presentations | | Hospital admissions | | Hospital occupancy | | Deaths | |
|---|---|---|---|---|---|---|---|---|---|---|
| Pathogen | No | % | No | % | No | % | No | % | No | % |
| *Bacillus* spp | 11 144 | 1.8 | 4 458 | 1.6 | 27 | 0.1 | 70 | 0.1 | 0 | 0 |
| *Campylobacter* spp | 359 466 | 56.6 | 171 174 | 62.6 | 16 946 | 85.1 | 62 701 | 73.4 | 86 | 21.1 |
| *Clostridium perfringens* | 84 081 | 13.3 | 44 253 | 16.2 | 354 | 1.8 | 5240 | 6.1 | 89 | 21.9 |
| VTEC O157 | 995 | 0.2 | 995 | 0.4 | 377 | 1.9 | 2149 | 2.5 | 22 | 5.4 |
| *Listeria monocytogenes* | 194 | <0.1 | 194 | 0.1 | 194 | 1.0 | 3473 | 4.1 | 68 | 16.7 |
| Salmonellas non-typhoidal | 41 616 | 6.6 | 29 726 | 10.9 | 1516 | 7.6 | 8793 | 10.3 | 119 | 29.2 |
| *S paratyphi* | 85 | <0.1 | 85 | <0.1 | 27 | 0.1 | 170 | 0.2 | 0 | 0 |
| *S typhi* | 96 | <0.1 | 96 | <0.1 | 39 | 0.2 | 267 | 0.3 | 0 | 0 |
| *Shigella* spp | 202 | <0.1 | 202 | 0.1 | 5 | <0.1 | 24 | <0.1 | 0 | 0 |
| *Staphylococcus aureus* | 2276 | 0.4 | 910 | 0.3 | 57 | 0.3 | 69 | 0.1 | 0 | 0 |
| *Vibrio cholerae* non O1 & O139 | 126 | <0.1 | 63 | <0.1 | 5 | <0.1 | 19 | <0.1 | 0 | 0 |
| Vibrio (other species) | 364 | 0.1 | 182 | 0.1 | 5 | <0.1 | 21 | <0.1 | 2 | 0.5 |
| *Yersinia* spp | 45 144 | 7.1 | 3858 | 1.4 | 216 | 1.1 | 1901 | 2.2 | 1 | 0.2 |
| *Cryptosporidium parvum* | 2063 | 0.3 | 1086 | 0.4 | 39 | 0.2 | 145 | 0.2 | 3 | 0.7 |
| *Cyclospora cayatenensis* | 992 | 0.2 | 522 | 0.2 | 3 | <0.1 | 10 | <0.1 | 0 | 0 |
| *Giardia duodenalis* | 1673 | 0.3 | 881 | 0.3 | 5 | <0.1 | 18 | <0.1 | 0 | 0 |
| Astrovirus | 17 291 | 2.7 | 3930 | 1.4 | 12 | 0.1 | 46 | 0.1 | 4 | 1.0 |
| NLV | 57 781 | 9.1 | 9172 | 3.4 | 37 | 0.2 | 143 | 0.2 | 9 | 2.2 |
| Rotavirus | 8979 | 1.4 | 1497 | 0.5 | 46 | 0.2 | 121 | 0.1 | 4 | 1.0 |
| Total (FB pathogens under surveillance) | 634 568 | 100 | 273 284 | 100 | 19 910 | 100 | 85 380 | 100 | 407 | 100 |
| FB paths/All FB IID (%) | 47.4 | | 74.2 | | 95.9 | | 96.4 | | 84.8 | |
| All (FB IID) | 1 338 772 | | 368 516 | | 20 759 | | 88 545 | | 480 | |

Totals are subject to the effects of rounding.
NLV, Norwalk-like viruses; VTEC, verocytotoxin producing *Escherichia coli*.

**Table 5** Food related illness and death in the USA compared with indigenous foodborne disease in England and Wales (2000)

| | USA | England and Wales | Ratio (USA:E&W) | Ratio adjusted for population |
|---|---|---|---|---|
| Illness | | | | |
| All aetiologies | 76 000 000 | 1 338 772 | 56.77:1 | 11.22:1 |
| Known pathogens | 13 697 367 | 696 729 | 19.66:1 | 3.89:1 |
| Known bacterial pathogens | 4 174 730 | 607 950 | 6.87:1 | 1.36:1 |
| Hospitalisations | | | | |
| All aetiologies | 320 299 | 20 759 | 15.42:1 | 3.05:1 |
| Known pathogens | 58 153 | 20 271 | 2.86:1 | 0.57:1 |
| Known bacterial pathogens | 36 359 | 20 129 | 1.81:1 | 0.36:1 |
| Deaths | | | | |
| All aetiologies | 4820 | 480 | 10.04:1 | 1.98:1 |
| Known pathogens | 1420 | 415 | 3.42:1 | 0.68:1 |
| Known bacterial pathogens | 1287 | 395 | 3.26:1 | 0.64:1 |

Data for USA do not include the contribution of the following pathogens: *Cl botulinum*; *Brucella* spp; *Toxoplasma gondii*; *Trichinella spiralis*; hepatitis A.

### IFD in England and Wales in 2000 (table 4)

Despite accounting for just under half (47.4%) of all cases of IFD, pathogens under routine national laboratory surveillance represented the majority of cases presenting to general practice (74.2%), hospital admissions (95.9%), hospital occupancy (96.4%), and deaths (84.8%).

### Comparison with the USA (table 5)

There were 76 million cases of food related illness in the USA per year[8] compared with 1.3 million cases of IFD in England and Wales in 2000—that is, a ratio of 57:1. When rates were considered, the ratio for all illness fell to 11:1, and to 1.4:1 for bacterial illness. Taking disease severity into account, the two models also converged. The population adjusted ratio of estimates from the CDC and PHLS models for hospital admissions for food related illness as a whole was 3:1, and for deaths was 2:1. For hospital admissions and deaths due to all known pathogens and known bacterial pathogens, the US rates fall below those of England and Wales.

## DISCUSSION

In 1992 there were an estimated 2 869 735 cases of IFD in England and Wales. By 2000 this had fallen by over half to 1 338 772. Measures of health service usage due to IFD fell less sharply owing to a rise in the incidence of campylobacters. However, there was a reduction of almost half in the number of estimated deaths. This was due in almost equal part to declines in illness caused by *Cl perfringens*, following a decline in the consumption of red meats in the UK,[19 20] and salmonellas which followed the introduction of a vaccination programme against *Salmonella enterica* serotype Enteritidis in chickens by the British poultry industry.[21] Campylobacters, *Cl perfringens*, salmonellas, VTEC O157, and *L monocytogenes* accounted for the greatest disease burden.

### Quality of data

Direct measurements were used wherever possible. However, extrapolation or inference was used when accurate measurements were not available because event frequencies were below the level of detection of epidemiological studies or surveillance. Therefore, the effects of these assumptions on the final model will be minimal.

### Total cases of IID

For this study, it is important that incidence estimates for common pathogens are accurate. The IID study data were robust for those pathogens contributing most to IFD in England and Wales.[13] IFD caused by yersinias, aeromonads, and non-VTEC may have been overestimated as not all strains are pathogenic.[13] Conversely, the role of NLV might have been underestimated as a result of the use of electron microscopy, rather than molecular techniques, as the method of detection in the IID study.[22]

Changes in patient presentation, diagnostic practice in primary care, or improved laboratory methods might affect laboratory trends. There has been no shift in the relative proportions of blood and faecal isolates in LabBase from patients with salmonella or campylobacter infections, suggesting no changes in general practitioner or patient behaviour. The widespread use of immunological assays and molecular techniques for NLV in the future means that the reliability of estimates based on IID study data will decay over time. Periodic incidence measurements for specific infections, such as NLV, will be needed to recalibrate the model.

### Adjusting for travel associated infection

In general, LabBase data underestimate the extent of imported infection and therefore special study data were used where available.[14–16] However, the final estimates for IFD might not fully account for infection acquired abroad.

### Estimating the number of cases of foodborne infection

Using outbreak surveillance data to estimate the proportion of foodborne disease requires care.[23] The validity depends on the extent to which disease transmission in general outbreaks represents all disease transmission. There was however no alternative. Recent national studies of sporadic gastrointestinal infection[13 16 24 25] were not designed to provide attributable fractions for foodborne transmission as a whole.

*Clostridium difficile*, *Shigella* spp, *Cryptosporidium parvum*, adenovirus 40/41, Sapporo-like viruses, and rotavirus are not usually transmitted through food.[8 13 26–29] *Clostridium difficile* and adenovirus 40/41 were included in the PHLS model but, with no foodborne outbreaks reported, neither contributed to the overall burden of IFD. Foodborne transmission rates for *Shigella* spp, *C parvum*, and rotavirus from GSURV were low.

Estimates of foodborne NLV transmission vary from 68%[30] at one extreme to 7.6%[31] at the other. Given its high incidence it is important to use an accurate figure for percentage foodborne transmission. The 10.75% figure in the PHLS model was considerably lower than most other published estimates but is derived from the largest and most contemporary dataset (1992–2000; n=1592 outbreaks).

In the CDC model, 85% of VTEC O157 was considered to be foodborne. In England and Wales 63% of VTEC O157 outbreaks were foodborne, which is consistent with recent studies of sporadic infection where person to person spread and contact with livestock were also important.[16 24 25 32]

Outbreaks of yersiniosis are scarce in England and Wales. However, as yersinias appear to be one of the most common causes of IID,[13] better data on pathogenicity and transmission pathways are needed.

Data on outbreaks of disease of unknown aetiology are held in GSURV. However, this might conceal a wide range of agents with differing modes of transmission and therefore represents an area of great uncertainty, requiring further research.

### Estimating the number of cases presenting to general practice

Mostly, rates of presentation to general practice were taken directly from the IID study. Estimates for *L monocytogenes*, *S paratyphi*, *S typhi*, VTEC O157, and *C cayatenensis* were derived by extrapolation or inference. Given the relatively low incidence of each of these pathogens, the effect of inaccuracies on the final estimates would be trivial.

### Estimating the number of cases admitted to hospital, hospital occupancy, and deaths

HES for hospital admissions for IID[11] as a whole were consistent with data from GSURV and PHLS enhanced surveillance schemes. However, a disproportionate number of patients were assigned to generic International Classification of Disease 10 codes such as "diarrhoea and gastroenteritis of presumed infectious origin".[33] Therefore, special study[14–17] and GSURV data were used for acute admissions. Chronic disease or long term sequelae were not considered. Detailed NS mortality figures were poor and therefore enhanced surveillance and GSURV data were used.

### International comparisons

First impressions are that foodborne illness is 11 times higher in the USA, with an additional 69 million cases after adjustment. However, the CDC baseline population estimate for IID[8] was fourfold greater than that used in the PHLS model. The US acute gastroenteritis rate was mainly derived from a retrospective population survey.[34] However, the IID study team performed a comparison of retrospective and prospective methodologies for assessing rates of gastroenteritis.[13] The retrospective method yielded a rate of IID in the population that was 2.8 times the rate derived through prospective follow up of the same cohort. It was concluded that recall bias led to the retrospective method overestimating the rate of IID in the community. Using the prospective method, the Sensor study[22] in the Netherlands also yielded rates of IID much lower than those employed in the CDC model.

CDC used laboratory surveillance data for most pathogens except NLV.[8] Instead, on the basis of a single population study,[35] the proportion of illness due to NLV was estimated to be 11% of total IID, approximately 40% of which was regarded as foodborne. This represented 67% of all foodborne illness caused by known pathogens. This is crucial as the percentage foodborne transmission in known agents was used as a proxy for unknown agents, and these accounted for 82% of all IID in the USA. Thus varying the percentage foodborne transmission of NLV changes foodborne illness due to unknown agents dramatically.[8]

Using a nearly fourfold greater foodborne transmission rate for NLV, alongside a much higher baseline level for IID results, created 59 million extra cases of illness due to unknown agents in the USA—that is, 85% of the difference between the two models. Similarly, an extra nine million cases of foodborne NLV in the USA accounted for a further 13% of the difference.

When illness due to known bacteria is considered, the CDC and PHLS estimates converge. In both, campylobacter was the most common bacterial cause of foodborne disease. Using population rates, the PHLS estimate for IFD due to campylobacter infection in 2000 was 95% of the CDC estimate. However, for salmonellas the PHLS estimate was only 16% of that from the CDC. Part of the explanation is a substantial decline in salmonellas in England and Wales since 1997.[36] Furthermore, CDC used an AR of 38 for both campylobacter and salmonellas. In the IID study the AR for campylobacter was higher than that for salmonellas.[13]

The estimates converged further when disease severity was considered. This is because these data are not directly influenced by the disparity in the baseline estimates for IID. Generally, there were more hospital admissions due to known

pathogens and known bacteria in England and Wales than in the USA. In the PHLS model, hospital admissions rates were applied to cases presenting to general practice rather than to laboratory reports as in the CDC model.[8] However, not all cases presenting to general practice are sampled let alone reported.[37] For hospital admissions due to viruses, the CDC model exceeded the PHLS model. However, in the CDC model, data were extrapolated from a single study.[38] Hospital admissions due to unknown agents were also higher in the USA.

The same arguments hold true for deaths. Both models highlighted the importance of salmonellas, *L monocytogenes*, campylobacter, and VTEC. In the PHLS model more deaths from *Cl perfringens* were due to high numbers reported through GSURV.[20]

### Other evidence from England and Wales

It might appear surprising that we describe a fall in IFD over a period when food poisoning notifications increased. However, our analyses have incorporated data from clinical[10] and microbiological[9] sources which independently show parallel declines over a nine year period. One study has demonstrated that food poisoning notifications are closely bound to the laboratory reporting of salmonellas and campylobacters.[39] This is borne out by an examination of recent trends. Notifications rose to reach a peak in 1998[40] reflecting an increase in the combined laboratory reporting of these pathogens. Since then notifications have fallen in line with declines in the reporting of both salmonellas and campylobacters.[36] The trends in IFD that we have described take into account a much wider range of pathogens and crucially measure the burden of infection due to each of these agents in a way that food poisoning notification data cannot.

In a retrospective survey it was estimated that over five million people per year in the UK suffered from acute gastroenteritis which they ascribed to contaminated food.[41] Our analyses suggest that such a retrospective survey of this type would be expected to produce a figure in this range given that recall bias would result in at least a three fold overestimation in the rate of illness.[13] This might have been compounded by misclassification bias because individuals made subjective judgements about illness causation. Based on symptoms alone, an individual cannot be certain if their illness was due to foodborne, person to person, or environmental transmission,[13 16 24 25 42] with the possible exception of those involved in recognised and proven point source foodborne outbreaks. Some individuals with gastrointestinal illness will inappropriately blame contaminated food.

## CONCLUSIONS

We have developed estimates using five separate criteria for IFD in England and Wales. A wide range of agents was included and, importantly, an adjustment for foreign travel. Between 1992 and 2000, overall illness fell by over half but hospital admissions declined by only 3%.

Illness due to known pathogens, particularly those under routine laboratory report surveillance, caused the most severe disease and greatest health service usage. In 2000, the majority of general practitioner consultations, hospital admissions, and hospital bed days were due to campylobacter infection. Salmonellas were the most common cause of death, also resulting in high levels of health service usage. *Cl perfringens* was second only to salmonellas as a cause of death.

If total IFD were taken as the sole measure of disease burden, the impact of VTEC O157 and *L monocytogenes* would be completely overlooked. Their importance only appeared when hospital occupancy and deaths were considered. By contrast, NLV infection caused far fewer deaths but caused large numbers of cases. We only considered the acute effects of foodborne disease because there is little routine information on chronic disease or long term sequelae.

The pattern of IFD is complex and evolving. Pathogens emerge, laboratory tests improve, and new data streams will require incorporation into the model. The most recent, robust, and reliable data currently available were used, but improvement requires continuous validation of those data sources.

Finally, reducing IFD in England and Wales means tackling campylobacter. Lowering mortality rates however also requires better control and prevention of salmonellas, *Cl perfringens*, *L monocytogenes*, and VTEC O157.

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## REFERENCES

1 **Food Standards Agency**. Available at: <http://www.food.gov.uk/news/newsarchive/59179> accessed 8 May 2002.
2 **Hauschild AHW**, Bryan FL. Estimate of food- and waterborne illness in Canada and the United States. *J Food Protect* 1980;**43**:435–40.
3 **Archer DL**, Kvenburg JE. Incidence and cost of foodborne diarrhoeal disease in the United States. *J Food Protect* 1985;**48**:887–94.
4 **Bennett JV**, Homberg SD, Rogers MF, *et al*. Infectious and parasitic diseases. In: Amler RW, Dull HB, eds. *Closing the gap: the burden of unnecessary illness*. New York: Oxford University Press, 1987:102–14.
5 **Todd ECD**. Preliminary estimates of costs of foodborne disease in Canada and costs to reduce salmonellosis. *J Food Protect* 1989;**52**:586–94.
6 **Todd ECD**. Preliminary estimates of costs of foodborne disease in the USA. *J Food Protect* 1989;**52**:595–601.
7 **Hanson S**. Estimating the incidence of food-borne *Salmonella* and the effectiveness of alternative control measures using the Delphi method. *Int J Food Microbiol* 1997;**35**:195–204.
8 **Mead PS**, Slutsker L, Dietz V, *et al*. Food-related illness and death in the United States. *Emerg Infect Dis* 1999;**5**:607–25.
9 **Wall PG**, de Louvois J, Gilbert RJ, *et al*. Food poisoning: notifications, laboratory reports, and outbreaks—where do the statistics come from and what do they mean? *CDR Review* 1996;**6**:R93–100.
10 **Fleming DM**. Weekly Returns Service of the Royal College of General Practitioners. *Commun Dis Public Health* 1999;**2**:96–100.
11 **Hospital Episode Statistics**. Available at <http://www.doh.gov.uk/hes/>.
12 **National Statistics Population Estimates Unit**. *Estimated resident population mid-1995 of England and Wales*. London: National Statistics.
13 **Foods Standards Agency**. *A report of the study of infectious intestinal disease in England*. London: The Stationery Office, 2000.
14 **CDSC**. Campylobacter sentinel surveillance scheme. *Commun Dis Rep CDR Wkly* (serial online) 2001 (cited 30 August 2001); **11** (23). Available at <www.phls.co.uk/publications/CDR/PDFfiles/2001/cdr2301.pdf>.
15 **Smerdon WJ**, Jones R, McLauchlin J, *et al*. Surveillance of listeriosis in England and Wales, 1995 to 1999. *Commun Dis Public Health* 2001;**4**:188–93.
16 **O'Brien SJ**, Adak GK, Gilham C. Contact with the farming environment as a major risk factor for sporadic cases of Shiga toxin (Vero cytotoxin)-producing *Escherichia coli* O157 infection in humans. *Emerg Infect Dis* 2001;**7**:1049–51.
17 **O'Brien SJ**, Adak GK, Lynn R, *et al*. Synergy between clinical and laboratory surveillance for describing the epidemiology of VTEC O157 in the United Kingdom. 4th International Symposium and Workshop on Shiga Toxin (Verocytotoxin)-Producing *Escherichia coli* Infections. Kyoto, Japan, 2000.

18 **Lorber B**. Listeria monocytogenes. In: Mandell GL, Bennett JE, Dolin R, eds. *Principles and practice of infectious diseases*, 5th ed. New York: Churchill Livingstone, 2000:2208–15.
19 **Ministry of Agriculture Fisheries and Food**. *National Food Survey 1999*. London: Her Majesty's Stationery Office, 2000.
20 **Smerdon WJ**, Adak GK, O'Brien SJ, *et al*. General outbreaks of infectious intestinal disease linked with red meats, England and Wales, 1992 to 1999. *Commun Dis Public Health* 2001;**4**:259–67.
21 **Kessel AS**, Gillespie IA, O'Brien SJ, *et al*. General outbreaks of infectious intestinal disease linked with poultry, England and Wales, 1992 to 1999. *Commun Dis Public Health* 2001;**4**:171–7.
22 **De Wit MAS**, Koopmans MPG, Kortbeek LM, *et al*. Sensor, a population-based cohort study on gastroenteritis in the Netherlands: incidence and etiology. *Am J Epidemiol* 2001;**154**:666–74.
23 **Palmer S**, Parry S, Perry D, *et al*. The role of outbreaks in developing food safety policy: population based surveillance of salmonella outbreaks in Wales 1986–98. *Epidemiol Infect* 2000;**125**:467–72.
24 **Parry SM**, Salmon RL, Willshaw GA, *et al*. Risk factors for and prevention of sporadic infections with Vero cytotoxin (shiga toxin) producing *Escherichia coli* O157. *Lancet* 1998;**351**:1019–22.
25 **Locking ME**, O'Brien SJ, Reilly WJ, *et al*. Risk factors for sporadic cases of *Escherichia coli* O157 infection: the importance of contact with animal excreta. *Epidemiol Infect* 2001;**127**:215–20.
26 **Hale TL**. Bacillary dysentery. In: Collier L, Ballows A, Sussman M, eds. *Topley and Wilson's microbiology and microbial infections*, 9th edn. New York: Oxford University Press, 1998:479–93.
27 **Current WL**. Cryptosporidiosis. In: Collier L, Ballows A, Sussman M, eds. *Topley and Wilson's microbiology and microbial infections*, 9th edn. New York: Oxford University Press, 1998:329–47.
28 **Clarke IN**, Lambden PR, Caul EO. Human enteric RNA viruses: calicivirus and astroviruses. In: Collier L, Ballows A, Sussman M, eds. *Topley and Wilson's microbiology and microbial infections*, 9th edn. New York: Oxford University Press, 1998:511–35.
29 **Offit PA**, Clark HF. Rotavirus. In: Mandell GL, Bennett JE, Dolin R, eds. *Principles and practice of infectious diseases*, 5th edn. New York: Churchill Livingstone, 2000:1696–703.
30 **Sekine S**, Okada S, Hayashi Y. Prevalence of small round structured virus infections in acute gastroenteritis outbreaks in Tokyo. *Microbiol Immunol* 1989;**33**:207–17.
31 *Advisory Committee on the Microbiological Safety of Food Report on Foodborne Viral Infections*. London: The Stationery Office; 1998:27–32.
32 **Kassenborg H**, Hedberg C, Evans M, *et al*. Case-control study of sporadic *Escherichia coli* O157:H7 infections in 5 FoodNet sites (Calif., Conn., GA., Minn., Ore.). 1st International Conference on Emerging Infectious Diseases. Atlanta, Georgia, March 1998.
33 **World Health Organisation**. *International Statistical Classification of Diseases and Related Health Problems*,10th revision. Geneva: World Health Organisation, 1992:108–12.
34 **CDC**. Incidence of foodborne illnesses- FoodNet 1997. *MMWR Morb Mortal Wkly Rep* 1998;**47**:782.
35 **Koopmans M**, van Duynhoven Y, van de Heide R. Molecular detection and epidemiology of Norwalk-like viruses and Sapporo-like viruses in the Netherlands. Presented at the International Workshop on Human Caliciviruses, Atlanta, Georgia, USA, 1999.
36 **CDSC**. Trends in selected gastrointestinal infections–2000. *Commun Dis Rep CDR Wkly* (serial online) 2001(cited 8 February 2001); **11** (6): enteric. Available from <http://www.phls.co.uk/publications/CDR/PDFfiles/2001/cdr0601.pdf>.
37 **Chalker RB**, Blaser MJ. A review of human salmonellosis: III. Magnitude of salmonella infection in the United States. *Rev Infect Dis* 1988;**10**:111–24.
38 **Mounts AW**, Holman RC, Clarke MJ, *et al*. Trends in hospitalizations associated with gastroenteritis among adults in the United States, 1979–1995. *Epidemiol Infect* 1999;**123**:1–8.
39 **Atkinson P**, Maguire H. Is food poisoning a clinical or a laboratory diagnosis? A survey of local authority practices in the south Thames region. *Commun Dis Public Health* 1998;**1**:161–4
40 **CDSC**. *1999/2000 Review of Communicable Disease*. London: PHLS, 2002:86–87.
41 **Food Standards Agency**. Consumer attitudes to food standards 2001 (cited 11 February 2002). Available at <http://www.food.gov.uk/news/pressreleases/campaignlaunch>.
42 **Palmer S**, Houston H, Lervy B, *et al*. Problems in the diagnosis of foodborne infection in general practice. *Epidemiol Infect* 1996;**117**:479–84.