

## INTESTINAL INFECTION

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

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*Gut* 2002;51:832–841

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Accepted for publication  
12 June 2002

**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.<sup>1</sup> 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,<sup>2</sup> failing to account for the diversity of pathogens causing foodborne disease,<sup>3–6</sup> or through reliance on expert opinion.<sup>7</sup> 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.<sup>8</sup> 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<sup>13</sup> 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<sup>10</sup> 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,<sup>13 18</sup> 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.<sup>13</sup> 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<sup>9</sup> 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<sup>14–16</sup> were used to determine the percentage of travel associated infection for each

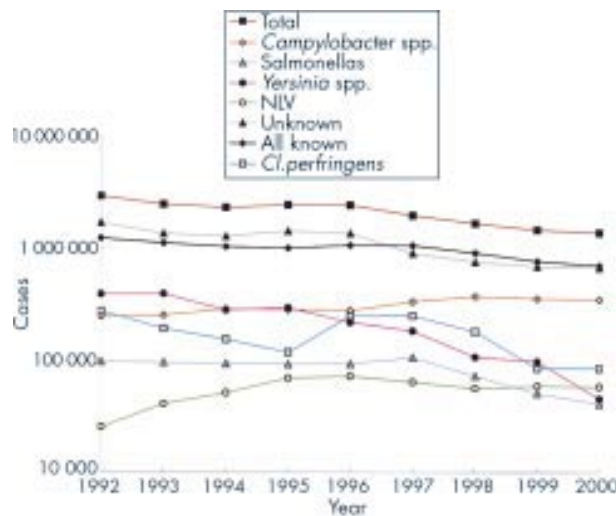
**Abbreviations:** AR, ascertainment ratio—ratio of the estimated number of cases of illness in the population due to specific pathogens to the number of laboratory reports in the national database for laboratory confirmed infections; CDC, United States Centers for Disease Control and Prevention; CDSC, PHLS Communicable Disease Surveillance Centre; HES, hospital episode statistics; G SURV, 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

Pathogen	Infectious intestinal disease					Infectious foodborne disease						
	Incidence/per 1000 person years	Estimated cases	Laboratory reports	AR	Indigenous (%)	Indigenous cases	Foodborne (%)	Cases	GP presentations	Hospital admissions	Hospital bed days	Deaths
<b>Bacteria</b>												
<i>Aeromonas</i> spp	12.4	642 557	635	1011.9	67.1	431 069	0	0	0	0	0	0
<i>Bacillus</i> spp	0.4	20 628	87	237.1	100.0	20 628	100.0	20 628	8251	50	129	0
<i>Campylobacter</i> spp	8.7	451 315	43 817	10.3	78.4	353 831	79.7	281 826	134 203	13 286	49 159	67
<i>Clostridium perfringens</i>	2.4	124 385	342	363.7	100.0	124 385	94.4	117 370	61 774	494	7314	124
<i>Cl difficile</i> 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 <i>Escherichia coli</i>	19.47	1 008 940	0	n/c	75.0	756 705	8.2	62 050	13 850	319	1561	6
<i>Listeria monocytogenes</i>	0.003	170	85	2.0	100.0	170	99.0	168	168	168	3007	59
<i>Salmonellas</i> non-typhoidal	2.2	115 904	29 719	3.9	88.2	102 227	91.6	93 651	66 894	3412	19 787	268
<i>S paratyphi</i>	0.01	340	170	2.0	26.0	90	80.0	72	72	23	144	0
<i>S typhi</i>	0.01	530	265	2.0	26.0	138	80.0	110	110	45	306	0
<i>Shigella</i> spp	0.27	13 984	4113	3.4	75.0	10 488	8.2	860	860	20	103	0
<i>Staphylococcus aureus</i>	0.27	13 989	59	237.1	100.0	13 989	96.0	13 429	5372	338	406	0
<i>Vibrio cholerae</i>	0.0004	20	10	2.0	0	0	90.0	0	0	0	0	0
O1/O139												
<i>V cholerae</i> 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
<i>Yersinia</i> 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
<b>Parasites</b>												
<i>Cryptosporidium parvum</i>	0.81	42 017	5678	7.4	95.00	39 916	5.6	2219	1168	42	156	4
<i>Cyclospora cayatenensis</i>	0.04	1976	52	38.0	26.92	532	90.0	479	252	1	5	0
<i>Giardia duodenalis</i>	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
<b>Viruses</b>												
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
<b>Total</b>		<b>10 464 004</b>	<b>120 029</b>			<b>9 021 129</b>		<b>2 365 909</b>	<b>511 941</b>	<b>21 138</b>	<b>99 874</b>	<b>718</b>

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<sup>9</sup> 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<sup>9</sup> (LabBase)—for laboratory confirmed infectious intestinal disease (IID) and the proportion acquired abroad.
- (B) National surveillance database for general outbreaks of IID<sup>9</sup> (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<sup>10</sup> (RCGP)—for IID presenting to general practice.
- (D) Hospital episode statistics<sup>11</sup> (HES)—for hospital admissions and bed occupancy.
- (E) National Statistics<sup>12</sup> (NS)—population estimates.
- (F) Study of Infectious Intestinal Disease in England (IID study)<sup>13</sup> (study population 495 666)—for adjusting LabBase data for underascertainment.
- (G) *Campylobacter* sentinel surveillance scheme<sup>14</sup> (n=7630; response rate=76%)—for infection acquired abroad and hospital admissions.
- (H) Enhanced surveillance of listeriosis in England and Wales<sup>15</sup> (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<sup>16</sup> (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<sup>17</sup> (n=413; response rate=100%)—for infection acquired abroad, hospital admissions, and deaths.
- (K) Food related illness and death in the USA<sup>8</sup>—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<sup>13</sup> 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<sup>11–16</sup> 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.<sup>11</sup> 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<sup>15</sup> 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<sup>8</sup> 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

$$N^{1995} = R^{1995} \times P^{1995}$$

$$115\,904 = 2.24/1000 \times 51\,820\,222$$

N<sup>1995</sup> = number of cases of illness in 1995R<sup>1995</sup> = rate of illness in the population in 1995 (IID study)P<sup>1995</sup> = 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<sup>1995</sup> = laboratory reports to Public Health Laboratory Service (PHLS) in 1995 (LabBase)

(3) All illness in England and Wales in 2000

$$N^{2000} = L^{2000} \times AR$$

$$58\,640 = 15\,036 \times 3.9$$

L<sup>2000</sup> = laboratory reports to PHLS in 2000 (LabBase)N<sup>2000</sup> = number of cases of illness in 2000

(4) Indigenously acquired illness in England and Wales in 2000

$$N^{2000,I} = N^{2000} \times (100 - t)$$

$$45\,427 = 58\,640 \times 77.5\%$$

N<sup>2000,I</sup> = number of indigenously acquired cases of illness

t = percentage of illness that is travel associated (LabBase)

(5) IFD in England and Wales in 2000

$$N^{2000,I,F} = N^{2000,I} \times f$$

$$41\,616 = 45\,427 \times 91.6\%$$

N<sup>2000,I,F</sup> = 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

$$G^{2000,I,F} = N^{2000,I,F} \times g$$

$$29\,726 = 41\,616 \times 71.4\%$$

G<sup>2000,I,F</sup> = 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

$$H^{2000,I,F} = G^{2000,I,F} \times h$$

$$1516 = 29\,726 \times 5.1\%$$

H<sup>2000,I,F</sup> = 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

$$B^{2000,I,F} = H^{2000,I,F} \times b$$

$$8793 = 1516 \times 5.8$$

B<sup>2000,I,F</sup> = 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

$$D^{2000,I,F} = G^{2000,I,F} \times d$$

$$119 = 29\,726 \times 0.4\%$$

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—*Yersinia*, campylobacters, *Campylobacter*, 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), *Yersinia* (392 753), and *Campylobacter* (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, *Campylobacter* third, and *Listeria monocytogenes* fourth for bed occupancy.

**Estimated deaths (table 2)**

Deaths fell by 48.1% (principally salmonella and *Campylobacter* 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
<b>Bacteria</b>												
<i>Aeromonas</i> spp	475	0	0	0	0	0	372	0	0	0	0	0
<i>Bacillus</i> spp	182	43 152	17 261	104	269	0	47	11 144	4458	27	70	0
<i>Campylobacter</i> spp	38 536	247 860	118 029	11 685	43 234	59	55 888	359 466	171 174	16 946	62 701	86
<i>Clostridium perfringens</i>	805	276 266	145 403	1163	17 216	291	245	84 081	44 253	354	5240	89
<i>Cl difficile</i> 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 <i>Escherichia coli</i>	0	62 050	13 850	319	1561	6	0	62 050	13 850	319	1561	6
<i>Listeria monocytogenes</i>	106	210	210	210	3759	74	98	194	194	194	3473	68
<i>Salmonellas</i> non-typhoidal	31 733	99 260	70 900	3616	20 972	284	15 036	41 616	29 726	1516	8793	119
<i>S paratyphi</i>	125	16	16	5	32	0	154	85	85	27	170	0
<i>S typhi</i>	198	34	34	14	95	0	165	96	96	39	267	0
<i>Shigella</i> spp	18 069	3778	3778	87	452	2	966	202	202	5	24	0
<i>Staphylococcus aureus</i>	112	25 493	10 197	642	771	0	10	2276	910	57	69	0
<i>Vibrio cholerae</i> O1/O139	9	0	0	0	0	0	12	0	0	0	0	0
<i>V cholerae</i> 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
<i>Yersinia</i> 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
<b>Parasites</b>												
<i>Cryptosporidium parvum</i>	5179	2024	1065	38	142	3	5278	2063	1086	39	145	3
<i>Cyclospora cayatenensis</i>	0	0	0	0	0	0	52	992	522	3	10	0
<i>Giardia duodenalis</i>	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
<b>Viruses</b>												
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
<b>Total</b>	<b>122 823</b>	<b>2 869 735</b>	<b>637 834</b>	<b>21 331</b>	<b>110 117</b>	<b>924</b>	<b>116 590</b>	<b>1 338 772</b>	<b>368 516</b>	<b>20 759</b>	<b>88 545</b>	<b>480</b>

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

Pathogen	Cases		GP presentations		Hospital admissions		Hospital occupancy		Deaths	
	No	%	No	%	No	%	No	%	No	%
<i>Bacillus</i> spp	11 144	1.8	4 458	1.6	27	0.1	70	0.1	0	0
<i>Campylobacter</i> spp	359 466	56.6	171 174	62.6	16 946	85.1	62 701	73.4	86	21.1
<i>Clostridium perfringens</i>	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
<i>Listeria monocytogenes</i>	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
<i>S paratyphi</i>	85	<0.1	85	<0.1	27	0.1	170	0.2	0	0
<i>S typhi</i>	96	<0.1	96	<0.1	39	0.2	267	0.3	0	0
<i>Shigella</i> spp	202	<0.1	202	0.1	5	<0.1	24	<0.1	0	0
<i>Staphylococcus aureus</i>	2276	0.4	910	0.3	57	0.3	69	0.1	0	0
<i>Vibrio cholerae</i> non O1 & O139	126	<0.1	63	<0.1	5	<0.1	19	<0.1	0	0
<i>Vibrio</i> (other species)	364	0.1	182	0.1	5	<0.1	21	<0.1	2	0.5
<i>Yersinia</i> spp	45 144	7.1	3858	1.4	216	1.1	1901	2.2	1	0.2
<i>Cryptosporidium parvum</i>	2063	0.3	1086	0.4	39	0.2	145	0.2	3	0.7
<i>Cyclospora cayatenensis</i>	992	0.2	522	0.2	3	<0.1	10	<0.1	0	0
<i>Giardia duodenalis</i>	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
<b>Illness</b>				
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
<b>Hospitalisations</b>				
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
<b>Deaths</b>				
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<sup>8</sup> 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,<sup>19,20</sup> and salmonellas which followed the introduction of a vaccination programme against *Salmonella enterica* serotype Enteritidis in chickens by the British poultry industry.<sup>21</sup> 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.<sup>13</sup> IFD caused by yersinias, aeromonads, and non-VTEC may have been overestimated as not all strains are pathogenic.<sup>13</sup> 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.<sup>22</sup>

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.<sup>14–16</sup> 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.<sup>23</sup> 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<sup>13 16 24 25</sup> 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.<sup>8 13 26–29</sup> *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%<sup>30</sup> at one extreme to 7.6%<sup>31</sup> 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.<sup>16 24 25 32</sup>

Outbreaks of yersiniosis are scarce in England and Wales. However, as yersinias appear to be one of the most common causes of IID,<sup>13</sup> 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<sup>11</sup> 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”.<sup>33</sup> Therefore, special study<sup>14–17</sup> 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<sup>8</sup> was fourfold greater than that used in the PHLS model. The US acute gastroenteritis rate was mainly derived from a retrospective population survey.<sup>34</sup> However, the IID study team performed a comparison of retrospective and prospective methodologies for assessing rates of gastroenteritis.<sup>13</sup> 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<sup>22</sup> 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.<sup>8</sup> Instead, on the basis of a single population study,<sup>35</sup> 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.<sup>8</sup>

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.<sup>36</sup> 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.<sup>13</sup>

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.<sup>5</sup> However, not all cases presenting to general practice are sampled let alone reported.<sup>37</sup> 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.<sup>38</sup> 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.<sup>20</sup>

### 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<sup>10</sup> and microbiological<sup>9</sup> 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.<sup>39</sup> This is borne out by an examination of recent trends. Notifications rose to reach a peak in 1998<sup>40</sup> 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.<sup>36</sup> 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.<sup>41</sup> 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.<sup>13</sup> 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,<sup>13 16 24 25 42</sup> 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.

### ACKNOWLEDGEMENTS

The authors would like to thank the following colleagues for their helpful comments on the manuscript: Martin Wood, Birmingham Heartlands Hospital, UK; Douglas Fleming, Birmingham Research Unit of the Royal College of General Practitioners; Henriette de Valk, Jean Claude Desclos, and Veronique Vaillant, Institut de Veille Sanitaire, St Maurice, France; Mike Painter, Manchester Infection Control and Surveillance Unit, UK; Yvonne van Duynhoven, National Institute of Public Health and the Environment, Bilthoven, the Netherlands; Richard Slack, Nottingham Health Authority, UK; Eric Bolton, Iain Gillespie, Judith Richards, David Tompkins, and Henry Smith, Public Health Laboratory Service, UK; and Stephen Palmer, University of Wales College of Medicine, Cardiff, UK. We also thank the microbiologists, public health physicians, infection control nurses, environmental health officers, general practitioners, RCGP, the staff of the PHLS and National Health Service laboratories and all members of the Gastrointestinal Diseases Division of CDSC without whose work the surveillance schemes would not function.

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