

Laboratory surveillance of bacterial gastroenteric pathogens in The Netherlands, 1991–2001

W. VAN PELT¹*, M. A. S. DE WIT¹, W. J. B. WANNET², E. J. J. LIGTVOET³,
M. A. WIDDOWSON⁴ AND Y. T. H. P. VAN DUYNHOVEN¹

¹ Department of Infectious Diseases Epidemiology, National Institute of Public Health and the Environment, Bilthoven, The Netherlands

² Diagnostic Laboratory for Infectious Diseases and Perinatal Screening, National Institute of Public Health and the Environment, Bilthoven, The Netherlands

³ Streeklaboratorium voor de Volksgezondheid, Haarlem, The Netherlands

⁴ Viral Gastroenteritis Section Centers of Disease Control and Prevention, Atlanta, GA, USA

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SUMMARY

Results of the Dutch laboratory surveillance of bacterial gastroenteritis between 1991 and 2001 are presented and compared with recent findings in general practices and in the community. Between 1996 and 2000 the mean annual number of stools screened by sentinel laboratories was about 1000 samples/100 000 inhabitants, which is 4% of the estimated annual incidence of gastroenteritis in the Dutch population. *Campylobacter* (36/100 000 inhabitants) and salmonella (24/100 000 inhabitants) were the main pathogens isolated. Since 1996, the incidence of laboratory confirmed salmonellosis decreased by 30%, predominantly among young children. The incidence of campylobacter was highest in urban areas and *Salmonella* Enteritidis emerged as the predominant serotype in urban areas. Between 1991 and 2001, multi-resistant *Salmonella* Typhimurium DT104 emerged to comprise up to 15% of all salmonella isolates in 2001. Reported rates of *Shigella* spp. and *Yersinia* spp. varied little, with average annual incidences of 3·2 and 1·2 cases/100 000 inhabitants, respectively. *Escherichia coli* O157 (90% STEC) was scarcely found (0·26/100 000).

INTRODUCTION

In developed countries gastrointestinal diseases represent a major public health burden, although the related mortality is low [1, 2]. The epidemiology of gastrointestinal infections has changed considerably in the last 10–20 years due to the global trade in food with changes in the area of primary food production, food processing and resultant changes in eating habits. In the last 10 years, epidemiological studies in The Netherlands in general practices [3, 4] and the general

population [5, 6] have provided insight into the incidence of gastroenteritis, the magnitude of the attendant health burden and the risk factors for infection. These formal epidemiological studies, however are not able to provide a detailed insight into the relative importance of rare bacterial species, serotypes and phage types and their trends. In order to implement and evaluate appropriate control measures for these pathogens, continuous surveillance and assessment is necessary. For instance, detailed information on the circulating sero- and phage types of *Salmonella* spp. in humans and farm animals in The Netherlands has proved to be extremely valuable in outbreak detection and linking animal reservoirs to human infections [7].

* Author for correspondence: National Institute of Public Health and the Environment, Department for Infectious Diseases Epidemiology, PO Box 1, 3720 BA Bilthoven, The Netherlands.

Surveillance of gastroenteritis in The Netherlands consists of several systems: (1) laboratory surveillance of bacterial pathogens by the regional public health laboratories, (2) numbers of gastroenteritis consultations by a network of sentinel GPs, (3) surveillance of foodborne outbreaks reported to Food Inspection Agencies and (4) the mandatory notification of foodborne outbreaks and some specific pathogens. In addition, since 1999, the system is supplemented by an intensified surveillance of STEC O157 [8].

Data on salmonella and STEC O157 are contributed on a monthly basis to the ENTERNET network [9] and reported on a yearly basis in addition to campylobacter to the Federal Institute for Health Protection of Consumers and Veterinary Medicine (BgVV), Berlin, which collects data on zoonotic pathogens in European countries (under directive 92/117/EEC).

This study presents the results of the laboratory surveillance of *Campylobacter* spp., *Salmonella* spp., *Shigella* spp., *Yersinia* spp. and *Escherichia coli* serotype O157 by the Dutch regional public health laboratories from 1991 to 2001.

MATERIALS AND METHODS

Laboratory surveillance system

The Dutch laboratory surveillance network for gastroenteric pathogens started in 1987 and consists of 15 out of 16 regional public health laboratories (PHL) serving mainly general practices but also distinct and university hospitals as well. For each patient, a standardized form is completed for the first isolates of *Salmonella* spp., *Shigella* spp. and *Yersinia* spp. Since April 1996, *Escherichia coli* serotype O157 (sorbitol-negative isolates that agglutinate with *E. coli* O157 antiserum) that may produce verocytotoxin, has also been reported. PHLs send the forms by mail or fax to the Department of Infectious Diseases Epidemiology (CIE) at the National Institute of Public Health and the Environment (RIVM), where since 1991 the data have been entered into a computer and thus available in electronic format. Data accompanying the laboratory results include information on age, sex, place of residence, recent stay abroad of the patient, type and date of sampling, probable source of infection and information on the strain (species and serotype, if determined). First isolates of *Salmonella* spp. and *E. coli* O157, from human and non-human sources, are sent to the National Reference Centre at the

RIVM, for confirmation, further serotyping, and/or phage typing and/or molecular typing, and sensitivity testing to antibiotics of relevance. Since April 1994, a weekly form is sent by each laboratory summarizing the number of all first isolates of the pathogens (genus level) identified that week, and the number of isolates sent to the RIVM for further typing. The weekly form includes the total number of stools screened which serves as a proxy for the number of consultations sought by gastroenteritis patients in the region. From April 1995 onwards, *Campylobacter* spp. has been included in the weekly form and since April 1996 *E. coli* O157 and the number of stools tested for this pathogen has been included. In addition to the weekly-summarized laboratory data on *Campylobacter* spp., data on campylobacter infections in individual patients has been obtained electronically from two laboratories for the years 1996–2001. The collection of laboratory data on *Yersinia* spp. stopped in January 1997.

Data analysis

Results are presented for the period 1991–2001. For each year, the population size by municipality, stratified by age and sex, was obtained from the National Bureau for Statistics and incidences were calculated with the appropriate denominators adjusted for the degree of coverage. Incidences were analysed by level of urbanization [10], defined on a scale from 1 (large cities: >2500 addresses per km²) to 5 (municipalities in the country: <500 addresses per km²). Each urbanization class represents approximately 20% of the Dutch population.

Denominator population

The coverage of the PHLs was estimated at the community level, based on data compiled since 1984 of isolates of *Salmonella* spp. which it is mandatory to send to RIVM for serotyping and phage typing. For each community and each year the consistency of coverage by a PHL was checked. In this way, communities were noted that belonged to the regular region and hence the regular population covered by a PHL. Including the data from those communities not covered regularly, the effective population of each PHL was estimated. Summing up the 15 PHLs, the surveillance network has a 52.7% regular coverage and 61.8% effective coverage of the Dutch population, figures that varied only slightly between 1991

Table 1. Summary statistics of laboratory submitted faeces and laboratory confirmed cases of gastroenteritis (data are obtained from the weekly and individual forms from the laboratories combined, between 1996 and 2000, supplemented with data on gastroenteritis from the Dutch GP sentinel (1996–9 [4]) and population study (1999 [6]))

	Positive stools (%)	Gastroenteritis cases/100 000 (laboratory surveillance)	Estimated gastroenteritis cases for the whole of The Netherlands		
			Laboratory surveillance	Consultations among GPs [4]	Patients in the general population [6]
Estimated cases				220 000	4 460 000
Stools*	6.19	1037	163 384		
<i>Campylobacter</i> spp.*	3.46	35.9	5650	23 200	107 000
<i>Salmonella</i> spp.*	2.28	23.7	3729	8600	53 500
<i>Shigella</i> spp.*	0.31	3.2	507	n.e.	n.e.
<i>Yersinia</i> spp.†	0.13	1.16	181	n.e.	n.e.
<i>Escherichia coli</i> O157‡ (4.2% stools tested)	0.01 (0.33)	0.26	40	n.e.	n.e.

* 1996–2000 data covering 9.75 million inhabitants.

† 1991–6 data covering 9.50 million inhabitants.

‡ 1997–2000 data covering 5.45 million inhabitants.

n.e.; not estimated.

and 2001. From 1991 to 1995 the mean effective coverage was 9.50 million inhabitants and between 1996 and 2001, 9.75 million inhabitants.

In 2000, all Dutch laboratories were surveyed on their protocol for testing verocytotoxin-producing *E. coli* O157 and numbers of isolates found. This survey showed that the PHLs found 38% of *E. coli* O157 isolates in The Netherlands in 2000. Taking into account that in previous years only some of the PHLs looked for this pathogen in stools, it is estimated that between 1997 and 2000 the surveillance for this pathogen by PHLs covered an average of 5.45 million inhabitants.

For *Campylobacter* spp., the incidences by age are derived from two PHLs that have an effective coverage between 1996 and 2000 of 0.33 and 0.69 million inhabitants respectively. The incidences by level of urbanization are derived from the second laboratory using the coverage from the regularly covered communities, averaging 0.60 million inhabitants between 1996 and 2000.

RESULTS

Summary statistics and general trends

Between 1996 and 2000 the average annual number of stool specimens screened in the 15 PHLs was 1037 specimens per year/100 000 inhabitants, i.e. about 163 000 per year for the whole of The Netherlands (Table 1). This figure for stool specimens has been

fairly constant in this period (Table 2) and covers about 98% of the materials screened for these bacteria. Between 1996 and 2000 bacterial pathogens were recovered in 6.2% the stools screened by the PHLs; *Campylobacter* spp. was the main pathogen isolated 3.5% of stools, followed by *Salmonella* spp. in 2.3%. In the same period the number of isolates of *Campylobacter* spp. (up to 1999) and *Salmonella* spp. decreased, almost 10 and 30%, respectively (Table 2). Over the years, *S. Enteritidis* has progressively emerged as the predominant serotype of *Salmonella* spp., replacing *S. Typhimurium*. Both however have decreased in absolute numbers of isolates reported since 1996. Between 1991 and 2001 multi-resistant *S. Typhimurium* DT104 emerged as the most prevalent salmonella type, whilst the dominant phage type of *S. Enteritidis*, phage type 4, decreased. The number and spectrum of species and/or serotypes of reported *Shigella* spp. (1991–9) and *Yersinia* spp. (1991–6) varied little in the periods indicated (Table 2), with average incidences of 3.2 and 1.2 cases/100 000 inhabitants, respectively (Table 1). In 2000 and 2001 the number of reported *Shigella* spp. was clearly lower than in the 9 years before.

Species, serotype and phage type distribution

The predominant serotypes of *Salmonella* spp. between 1996 and 2001 were *S. Enteritidis* and *S. Typhimurium*, comprising 75% of all isolates (Table 3). Phage type 4 decreased from more than 90% of all

Table 2. Trends between 1999 and 2001 in the incidence (per 100 000 inhabitants) of reported *Campylobacter* spp., *Salmonella* spp., *Yersinia* spp., faecal samples tested in total and the portion tested for *E. coli* O157 and the number of reported *E. coli* O157 isolates. The percentage contribution of the phage types DT104 and PT4 to their respective serotypes *S. Typhimurium* and *S. Enteritidis* is indicated

	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000	2001
Stools tested*	—	—	—	—	—	1076	1070	1043	1038	978	948
<i>E. coli</i> O157 tests†	—	—	—	—	—	2.4%	2.4%	4.0%	5.4%	5.2%	6.4%
<i>E. coli</i> O157	—	—	—	—	—	3‡	9	13	15	19	24
<i>Campylobacter</i> spp.*	—	—	—	—	—	38.9	37.7	35.2	32.9	34.8	37.0
<i>Salmonella</i> spp.*	29.3	26.5	28.7	29.7	30.1	29.0	25.7	22.7	21.0	20.1	20.1
<i>S. Typhimurium</i>	10.1	9.8	10.2	7.4	8.4	10.1	8.0	6.9	6.7	5.9	6.9
(% DT 104)	4%	6%	8%	13%	18%	21%	26%	27%	32%	29%	43%
<i>S. Enteritidis</i>	10.1	8.9	10.8	14.7	14.5	12.6	11.6	9.7	8.5	9.3	8.6
(% PT 4)	90%	85%	86%	77%	81%	84%	83%	76%	68%	63%	60%
<i>Shigella</i> spp.*	3.2	3.7	3.1	3.3	3.5	3.4	3.6	3.6	3.3	2.4	2.3
<i>Yersinia</i> spp.§	1.07	1.31	1.16	1.36	1.18	0.91	—	—	—	—	—

* 1996–2001 data covering 9.75 million inhabitants.

† 1997–2001 data covering 5.45 million inhabitants.

‡ From 1 April.

§ 1991–6 data covering 9.50 million inhabitants.

Table 3. Distribution of most frequently reported salmonella types in The Netherlands, 1996–2001 from 15 PHLs. The 2001 values for phage types *S. Enteritidis* PT4 and *S. Typhimurium* DT104 are in parentheses

Serotype/Phage type	N	%	Serotype	N	%
<i>S. Enteritidis</i>	5863	43.3	<i>S. Goldcoast</i>	120	0.9
(<i>S. Enteritidis</i> PT4)	4332	32 (25.9)	<i>S. Typhi</i>	106	0.8
<i>S. Typhimurium</i>	4361	32.2	<i>S. Livingstone</i>	97	0.7
(<i>S. Typhimurium</i> DT104)	1267	9.4 (14.8)	<i>S. Derby</i>	86	0.6
<i>S. Infantis</i>	248	1.8	<i>S. Newport</i>	71	0.5
<i>S. Hadar</i>	244	1.8	<i>S. Paratyphi B</i>	68	0.5
<i>S. Brandenburg</i>	214	1.6	<i>S. Agona</i>	64	0.5
<i>S. Bovismorbificans</i>	212	1.6	<i>S. Braenderup</i>	62	0.5
<i>S. Virchow</i>	182	1.3	Other serotypes	1412	10.5
<i>S. Panama</i>	122	0.9	Total	13 532	100

phage types of *S. Enteritidis* in 1991 to 60% in 2001; multi-resistant DT104 was the principal phage type of *S. Typhimurium*, comprising about 30% of all isolates up to 2000 and 43% in 2001.

Between 1991 and 2001, the relative importance of shigella species hardly changed. *S. sonnei* represented 58% of all reported shigella cases, *S. flexneri* 32.1%, *S. boydii* 5.4%, *S. dysenterica* 3.2% and other or unknown species 1.3%.

Between 1991 and 1996 the species and serotype distribution of infections with *Yersinia* spp. hardly changed. *Y. enterocolitica* represented 88.8% of all species, *Y. frederiksenii* 0.9%, *Y. pseudotuberculosis* serogroup-1 0.6%, *Y. intermedia* 0.3%, *Y. kristensenii* 0.2% ($n=1$), and unknown or unreported species

9.2%. Serotyping of yersinia isolates by the PHLs decreased during the 1991–6 surveillance period from almost 100% in 1991 to 60% in 1996. The main serogroups were O:3 (59%) and O:9 (13%), together almost 75% of all *Y. enterocolitica* strains. Slight variations in the occurrence of serogroups O:6 (5%); O:5 (7.5%) and O:7,8 (4%) over time were usually related to small outbreaks. Other serogroups included O:5,27 (2.5%, not included into O:5); O:6,30 (1.5%, not included into O:6) and O:7,13 (1%). The average age of people infected by serogroup O:3 was lower than for the other serogroups combined (22 years compared to 36 years).

Between April 1996 and December 2001 an increasing number of stools was screened for *E. coli*

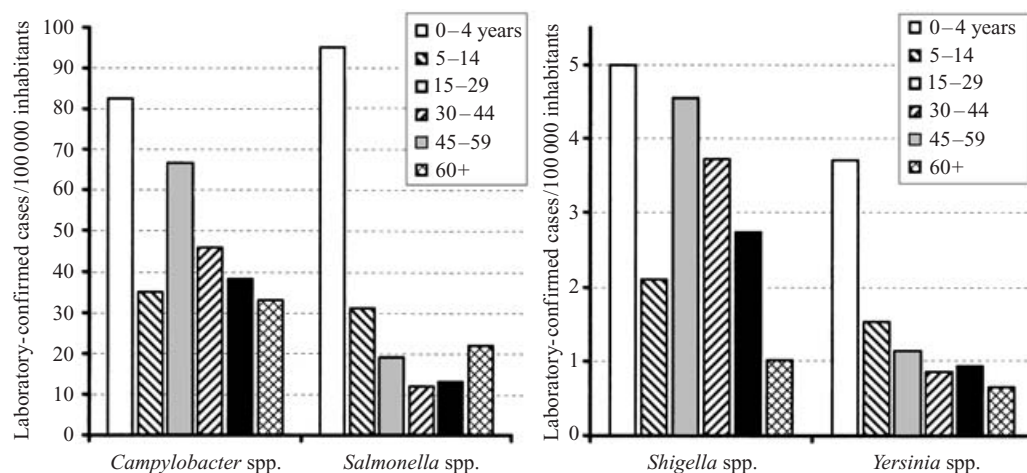


Fig. 1. Incidence per 100 000 inhabitants by age group. Data on *Salmonella* spp. and *Shigella* spp. are for 1996–2001 from 15 PHLs covering 9.75 million inhabitants and on *Yersinia* spp. for 1991–6 covering 9.50 million inhabitants. 1996–2001 data on *Campylobacter* spp. are for two PHLs covering 1.01 million inhabitants.

O157 (Table 2). Of isolates sent to the RIVM by the PHLs 83 were confirmed as *E. coli* O157; 90.3% of them were verocytotoxin-producing either VT1 (2.4%), or VT2 (57.8%) or both (30.1%); 85.5% of isolates contained the *E. coli* attaching and effacing gene (*eae*). H-typing showed 39% to be of the H7 type, all but one verocytotoxin producing.

Age

Age-specific incidences for laboratory-confirmed infections with *Campylobacter* spp. between 1996 and 2001 were available for two regions in The Netherlands (Fig. 1). Incidence was highest among very young children 0–4 years of age, followed by young adults 15–29 years of age. Incidence gradually decreased in those over 30 years of age. In the youngest age group (0–4 years), 30% more males were found as females, however 60% more females were found in the age class 15–29 years (data not shown).

Between 1996 and 2001 the incidence of *Salmonella* spp. was clearly highest among young children 0–4 years of age (Fig. 1) but 40% lower compared to the period 1991–5 in the same age group (data not shown). The incidence sharply decreased with age and increased again amongst people 60 years of age and older. *S. Typhimurium* was the dominant serotype amongst children 0–4 years old, and *S. Enteritidis* among people 15–60 years of age (data not shown).

The incidence of infections with *Shigella* spp. was higher among very young children (0–4 years) and young adults (15–29 years) compared to other age groups, especially among females (70% higher among females 15–40 years of age than among males,

irrespective of travel history, data not shown). Between 1996 and 2001, the incidence of shigella infection among the middle-aged (45–59 years) was 50% higher compared to the years 1991–5.

The highest incidence of laboratory-confirmed cases of *Yersinia* spp. infection between 1991–6 was found among the youngest children (0–4 years) and decreased with increasing age. Regards *E. coli* O157, most isolates were found among the youngest children 0–4 years of age (23%), least among adults 15–44 (10%), and increasing again in individuals 45 years old or over.

Seasonality

Between 1996 and 2001, the number of stools tested was higher at the end of the winter as well as in the summer and early autumn (Fig. 2). Between 1996 and 2001, reports of *Campylobacter* spp., *Shigella* spp. and *Salmonella* spp. all had clear peaks in summer. At the end of May reports of *Campylobacter* spp. strongly increased and peaked in early September. Reports of shigella and salmonella peaked a few weeks later than campylobacter and reached a higher peak incidence (Fig. 2). Seasonality of *Yersinia* spp. is least pronounced but is significantly isolated more between the end of March and the beginning of August. Of the 83 *E. coli* O157 isolates 49% were found between June and August (not shown in Fig. 2).

Urbanization

The occurrence of *Campylobacter* spp. by level of urbanization between 1996 and 2001 could be

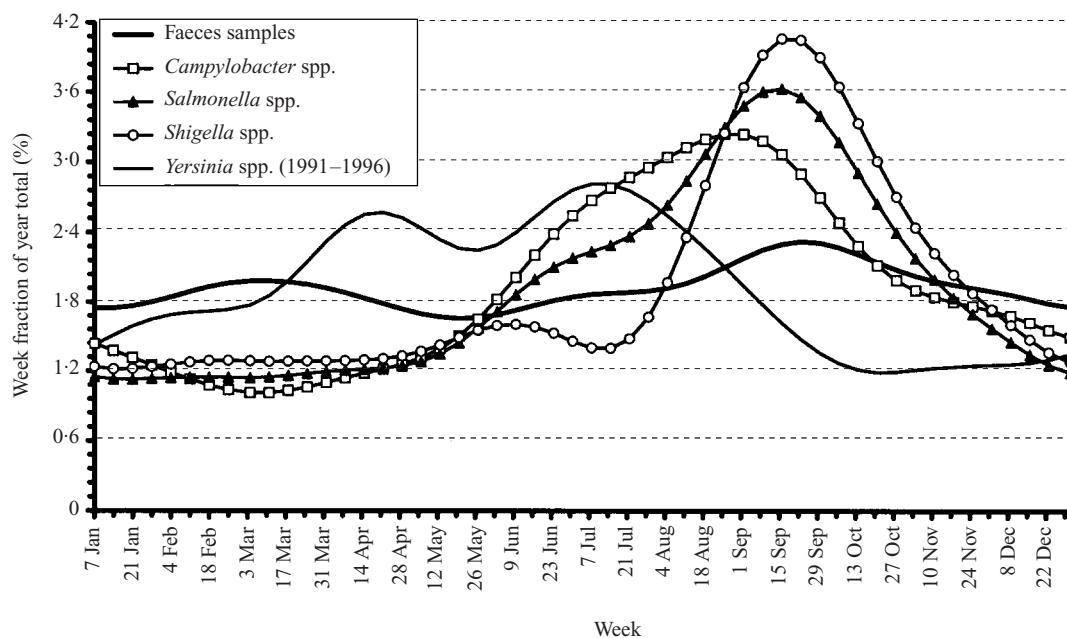


Fig. 2. Seasonal distribution of stools screened and of positive results of first isolates of *Campylobacter* spp., *Salmonella* spp. and *Shigella* spp. between 1996 and 2001. Reports of *Yersinia* spp. are for 1991–6.

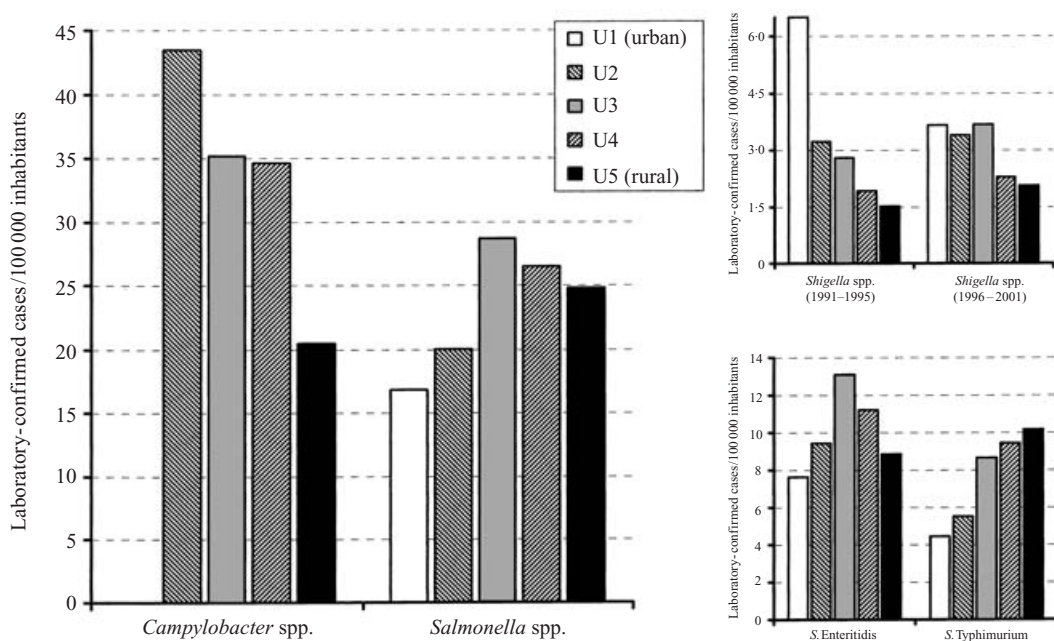


Fig. 3. Incidence per 100 000 inhabitants by level of urbanization. Data on *Salmonella* spp. and *Shigella* spp. are for 1996–2001 from 15 PHLs covering 9.75 million inhabitants and for 1991–6 covering 9.50 million inhabitants. 1996–2001 data on *Campylobacter* spp. are for one PHL covering 0.60 million inhabitants.

determined for one region in The Netherlands (Fig. 3). Although cities with more than 100 000 inhabitants (urbanization grade 1) do not occur in this region, clearly, the incidence is much lower in the rural than the urban areas. The reverse holds for *Salmonella* spp.: between 1996 and 2001 the incidence is significantly

lower in the larger Dutch cities than in the rest of the country. *S. Enteritidis* emerged between 1991 and 2001 as the dominant serotype in most cities whilst *S. Typhimurium* still dominates in the rural areas (Fig. 3). Between 1996 and 2001, the incidence of *Shigella* spp. was higher in the urbanized areas (grades

1–3) compared to more rural areas (grades 4–5). The higher occurrence in the larger cities is considerably less pronounced than it was in the period 1991–5 (Fig. 3). No clear trend was seen for *Yersinia* spp.

Travel history

Between 1996 and 2001, recent travel abroad was reported in 6.3% of the patients with a laboratory-confirmed salmonella infection. Compared to this overall figure the relative risk that an infection with *S. Enteritidis* was contracted abroad was 0.8 and that for *S. Typhimurium* even lower, 0.3. Nonetheless, infections with these two serotypes are responsible for the majority of all infections of salmonella-infected patients that reported travelling. The relative risk (RR) that an infection was contracted abroad was highest for *S. Paratyphi B* (RR=9.4) followed by *S. Typhi* (RR=8.5), *S. Virchow* (RR=2.7) and *S. Hadar* (RR=2.1), all included in the list of most frequently reported salmonella serotypes in The Netherlands (Table 3). Infections with *S. Typhi* have dropped by half since 1991. Mediterranean countries were the most likely source for *S. Enteritidis* and *S. Typhimurium* infections acquired abroad. Indonesia was the most likely source for travel-associated infections of *S. Typhi* followed by Morocco, India and Pakistan. Travel-associated *S. Paratyphi B* infections were most likely contracted in Turkey, followed by Indonesia and Morocco.

Up to 1994, a recent travel history was reported in 33% of patients with a laboratory-confirmed shigella infection, but this increased to an average of 49% after 1994. The majority of reported foreign countries were developing countries, whilst very few cases reported recent travel to Europe (3%) or North America (0.1%). From 1996 to 2001 the countries to which the highest number of cases of shigella infection could be attributed were Egypt (13%), India (12%), Turkey (12%), Indonesia (8%) and Morocco (6%). *S. boydii* was recovered more frequently from patients who visited North Africa (29%) or Central Asia (33%); *S. sonnei* from North Africa (34%); *S. dysenterica* from the Indian subcontinent (35%); and *S. flexneri* from African countries (45%) and Asia (33%). Between 1997 and 2001, the number of *Shigella* spp. isolated in patients returning from Egypt was higher than before 1997: on average 30 as compared to 9 in the preceding 6 years. Apart from Egypt no other major trend by country of travel over time was identified.

Between 1991 and 2001, the incidence of reported cases of shigella in individuals in the age group over 44 years increased from 15% of all cases before 1995 to 23% of all cases after 1995. This increase was found to be almost exclusively due to an increase of cases in this age group with reported travel abroad. Although overall incidence is highest among the youngest children, only 24% of cases under 15 years reported recent travel abroad, compared to 44% in all age groups. Trend analysis showed that the seasonal peak of cases without a travel history occurred two weeks after the seasonal peak of cases with a travel history. When stratified by age, a delay in the seasonal peak was observed for the categories 0–15 years (2 weeks), and 15–40 years (3 weeks), the latter predominantly females. A delay in the seasonal peak could not be demonstrated clearly for older cases.

DISCUSSION

Comparison with studies in general-practices and the community

The relationship between laboratory findings of gastroenteric pathogens, gastroenteritis patients consulting a GP and estimates of the total number of infected patients in the population is poorly studied [11]. Results are likely to differ between western countries. Such knowledge is fundamental for studies on the economic costs and disease burden related to these infections as have been performed in the United States [12] and, for *Campylobacter* spp. and *Salmonella* spp., in The Netherlands [13, 14]. In The Netherlands, several epidemiological studies have been undertaken to estimate the incidence of gastroenteritis and associated pathogens (bacteria, viruses and parasites); a study in four regions in The Netherlands in 1991 [5]; a nationwide community study in 1999 (Sensor) [6], and a study among cases consulting a GP in the periods 1992–3 [3] and 1996–9 [4]. For the comparison of rates derived from the laboratory surveillance with recent findings in general practices and in the community, the number of stools screened were used as a proxy for the number of consulting gastroenteritis cases (GP consulting and hospital cases) and an estimate was made of the populations covered. It seems plausible that the estimated 62% coverage for salmonella applies for campylobacter as well. However, as a minority of the reports concerns hospital cases the coverage of more serious diseases like yersiniosis and shigellosis may be lower and closer to the 38% coverage found for *E. coli* O157.

The 1999 estimate of the incidence of gastroenteritis in the population, was 28 300/100 000 (95% CI 25 200–31 500) inhabitants, or 4.5 million episodes of gastroenteritis per year in the whole population in The Netherlands [6]. Between 1996 and 1999, the annual incidence of gastroenteritis cases consulting a GP in The Netherlands was estimated at 1400/100 000 inhabitants, yielding over 220 000 cases per year (4.9% of total) [15]. Using faecal samples screened by the PHLs as a proxy for consulting gastroenteritis patients (GP consulting and hospital cases), yields an estimate of 163 000 cases per year (3.6% of the estimated total number of cases in the community). The percentage of positive findings in stools screened by the PHLs are much lower in the first quarter of the year (cf. Fig. 2) due to a predominance of faecal samples sent in for viral infection related gastroenteritis, notably rotavirus [16]. On an annual basis, bacterial pathogens were recovered in 6.2% of the stools screened by the PHLs (Table 1), much less than the 15.7% found amongst GP-consulting gastroenteritis cases [4]. Clearly (Table 1), campylobacter was recovered relatively more often than salmonella among gastroenteritis cases included by the GP in the sentinel study as in routinely sent in stools screened by the PHLs. Selection of more severe cases and postponed sampling of stools during a gastroenteritis episode by GPs may reduce the chances of a positive finding more in campylobacter infections than in salmonella infections. Part of the difference, however, can also be attributed to the number of stools from hospital cases (an estimated 25–45%) screened by the PHLs that may be selected by severity of disease as well.

Trends in gastroenteritis and gastroenteric pathogens

An earlier estimate of the incidence of gastroenteritis in the Dutch population in 1991 of 44 700/100 000 (95% CI 38 300–51 100) [5] was higher than the above estimate, for 1999, of 28 300/100 000 suggesting a major decrease over this decade. However, it has been argued that this early estimate is an overestimation [6]. Nevertheless, isolates of the predominant gastroenteric bacteria (*Campylobacter* spp. and *Salmonella* spp.) reported by the PHLs show a decrease since 1996. This is contrary to what is found in most other developed countries, where laboratory reports of *Campylobacter* spp. steadily increased during the 1990s [17] and *Salmonella* spp. levelled off after the *S. Enteritidis* epidemic in the 1980s and early 1990s

[18]. In the United States, however, recent data shows a sustained decrease since 1996 of *Salmonella* spp. and *Campylobacter* spp. as well as *Yersinia* spp. [19]. In fact, salmonella has more than halved since the 1970s in The Netherlands, almost entirely due to the reduction of *S. Typhimurium* and because of a reduction among children between 0–4 years and people older than 60 years of age [20]. The decrease might be due to improved hygiene and more widespread application of HACCP procedures in animal production chains. Also, media attention has increased and might have improved public awareness of food-borne infections and knowledge on how to prevent them. Moreover, meeting European regulations on protection against zoonotic agents [21], preventive measures in poultry in The Netherlands implemented in 1997 and 1998, might have helped to reduce salmonella in recent years. Finally, a lower laboratory consultation rate due to the deferral policy of GPs in The Netherlands for gastroenteritis since 1993 [22] may have played a role as well. However, the latter cannot explain the trends in recent years, as the annual number of faecal samples screened by the PHLs between 1996 and 1999 stayed fairly constant. The predominance of infected patients with *Campylobacter* spp. and *Salmonella* Enteritidis in the larger cities may be related to differences in exposure (food and travel) between people in urban and rural areas. Both agents (contrary to *S. Typhimurium*) are recognized as related to contamination of poultry meat.

The epidemiology of shigella is associated with a history of travel abroad. Whilst the total annual number of cases hardly changed between 1991 and 1999, the number of cases reporting a recent visit to a foreign country increased from 33% in the years up to 1994 to 49% after 1994. Thus endemic cases must have decreased between 1991 and 1999. Especially in the two biggest cities in The Netherlands, Amsterdam and Rotterdam, contributing between them 25% of all cases, active prevention of secondary infection through contact tracing proved to be successful [23]. The decrease in these cities to a large extent explains the decrease in 2000 and 2001 and the drop in the incidence of urbanization level one in recent years. The reported travel destination countries associated with shigella infection have also been described in other European surveillance systems [24]. The post-summer peak is likely to correspond to holidays and family visits to shigella-endemic countries. The development of exotic tourism, particularly among people over 44 years may explain the increase over the years

since 1991 in this age group. There is strong evidence for secondary transmission of shigella infections as the seasonal increase appears first in those who travelled. This is most clearly found for young children and is in agreement with a recent Dutch study that found them to be at a higher risk for infection by secondary transmission [23]. A seasonal delay was also found in females in the age group 15–40 years, independent of travel. A predominance of females in this age group has been found in other studies [25] and it has been suggested that secondary infection of females through an infected child might play a role. Incidence of shigella infections was higher in the cities which can be explained either by a high proportion of travellers coming from the city areas or by a higher proportion of immigrants coming from the at risk countries residing in the city areas [26] and a higher secondary attack rate in densely populated areas.

Between 1991 and 1996, the number of isolates of *Yersinia* spp. hardly varied. However, in the first years of the surveillance (1989/90), the number of yersinia isolates was about twice as high as in later years. This decrease in incidence in the early 1990s has been observed in neighbouring countries and been attributed to changes in slaughtering procedures, specifically the prevention of contamination of carcasses by tonsils and tongues [26]. The main serotypes O:3, and O:9 (previously reported in The Netherlands [27]) have often been isolated from pigs (tongue and tonsils) in Northern European countries [28]. Data from the population study and GP sentinel study indicate that 1.5 and 0.7% respectively of the gastroenteritis cases were associated with a *Yersinia* spp. infection. However, all were apathogenic types. About the same figures were found for controls in these studies. This may explain, why contrary to the literature, most yersinia isolates reported by the PHLs were found in the summer months, being a consequence of the larger number of stools screened in the peak season for salmonella and campylobacter.

So far, only two small clusters of STEC O157 infection have been described in The Netherlands [29]. An estimate of 40 laboratory-confirmed STEC O157 infections per year indicates that this infection may be rather uncommon in The Netherlands, especially compared to other European countries [30]. As in other countries [31], a seasonal effect with a peak in the summer months and early autumn is apparent and mirrors the shedding season observed in farm animals in The Netherlands [32]. In the GP sentinel study, one STEC O157 case was observed and none was found

in the community study [4, 6]. However, non-O157 STEC infections were found in 0.4% of the consulting gastroenteritis cases and 0.2% in the community study. Non-O157 serotypes have been rarely associated with HUS in The Netherlands, but there are reports of such an association in other countries [22].

General remarks and recommendations

The number of stools screened was used as a proxy of consulting gastroenteritis patients but we could not differentiate between stools originating from hospitals and those from general practices. An estimate of number of stools from general practice would be extremely useful as a denominator for other national surveillance systems facilitating international comparisons.

Infections by pathogens such as *Shigella* spp., *S. Typhi* and *S. Paratyphi B* are known to be generally acquired abroad, but this is not so evident from the laboratory reports. Although travel information seems highly underreported, it is useful to rank pathogens and countries of origin of the infection. The GP sentinel study suggests that about 20% of *Campylobacter* spp. infections are travel-related. This illustrates how the weekly enumeration of positive laboratory findings of *Campylobacter* spp. without any further patient data is insufficient to understanding the epidemiology of *Campylobacter* spp. To be able to target and evaluate control programmes in The Netherlands, there should be collection of better data on campylobacter infections and subsequent epidemiological analysis. This will be achieved by the steadily increasing number of regions with automated electronic laboratory surveillance, which this study has already shown to be extremely valuable. Associations with travel and secondary transmission are predominant characteristics of *Shigella* spp. infections. During the 1990s travel-associated *Shigella* spp. infections increased mainly in middle-aged and older people and infections became less restricted to large cities and to the late summer. *Yersinia* spp. infections in The Netherlands seem of limited public health significance compared to other enteric pathogens. An unpublished inventory in 2000 among the PHLs showed the same level of laboratory-confirmed *Yersinia* spp. infections as in 1991–6.

Infections with STEC O157 were rare in The Netherlands and non-O157 STEC infections seem of limited public health interest, as yet. However, because of the serious disease that can be caused by STEC infections and developments abroad, all STEC

infections in humans and animals should be continuously monitored.

Weekly reported typing results, basic patient information and simple denominator information have been found invaluable both for outbreak detection and following trends, allowing up-to-date assessment of basic epidemiological characteristics. Desirable improvements such as timeliness and completeness of data, and more detailed denominator information will be supplied in the future by standardized electronic laboratory surveillance, the coverage of which is rapidly increasing.

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REFERENCES

- Guerrant RL, Hughes JM, Lima NL, Crane J. Diarrhea in developed countries: magnitude, special settings and aetiologies. *Rev Infect Dis* 1990; **12**: S41–9.
- Bern C, Martines J, Zoysa I de, Glass RI. The magnitude of the global problem of diarrhoeal disease: a ten-year update. *Bull World Health Org* 1992; **70**: 705–9.
- Goosen ESM, Hoogenboom-Verdegaal AMM, Bartelds AIM, Sprenger MJW, Borgdorff MW. Incidence of gastroenteritis in general practices in the Netherlands, 1992–1993. Bilthoven, the Netherlands: National Institute of Public Health and the Environment, 1995; RIVM-report 149101012.
- Wit MAS de, Koopmans MPG, Kortbeek LM, Leeuwen WJ van, Bartelds AIM, Duynhoven YTHP van. Gastroenteritis in sentinel general practices in the Netherlands. *Emerg Infect Dis* 2001; **7**: 82–9.
- Wit MAS de, Hoogenboom-Verdegaal AMM, Goosen ESM, Sprenger MJW, Borgdorff MW. A population-based longitudinal study on the incidence and disease burden of gastroenteritis and campylobacter and salmonella infections in four regions in the Netherlands. *Eur J Epidemiol* 2000; **16**: 713–5.
- Wit MAS de, 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**: 666–8.
- Pelt W van, Giessen AW van de, Leeuwen WJ van, et al. Origin, extent and cost of humans salmonellosis. Part 1: origin of human salmonellosis with respect to pigs, cattle, chicken, eggs and other sources. *Infectieziekten Bull* 1999; **10**: 240–3.
- Duynhoven YTHP van, Jager CM de, Heuvelink AE, et al. Enhanced laboratory-based surveillance of Shiga-toxin producing *Escherichia coli* O157 in the Netherlands. *Eur J Clin Microbiol Infect Dis* 2002; **21**: 513–9.
- Fisher IST. The Enter-net international surveillance network – how it works. *Eurosurveillance* 1999; **4**: 52–5.
- Dulk CJ den, van de Stadt H, Vliegen JM. A new measure for the degree of urbanisation: the regional address density. *Mnd, stat. Bevolk (CBS)*, 1992; 92/7.
- Wheeler JG, Sethi, D, Cowden JW, et al. Study of infectious disease in England: rates in the community, presenting to general practice, and reported to national surveillance. *BMJ* 1999; **318**: 1046–50.
- Mead PS, Slutsker L, Dietz V, et al. Food-related illness and death in the United States. *Emerg Inf Dis* 1999; **5**: 607–18.
- Havelaar AH, Wit MAS de, Koningsveld R van, Kempen E van. Health burden in the Netherlands due to infection with thermophilic *Campylobacter* spp. *Epidemiol Infect* 2000; **125**: 505–17.
- Pelt W van, Giessen AW van de, Leeuwen WJ van, et al. Origin, extent and cost of humans salmonellosis. Part 2: estimation of the extent of human salmonellosis in the Netherlands and related economic costs. *Infectieziekten Bull* 2000; **11**: 4–8.
- Wit MAS de, Kortbeek LM, Koopmans MPG, et al. Comparison of gastroenteritis cases in a general practice based-study and a community-based study. *Epidemiol Infect* 2001; **127**: 389–98.
- Koopmans MPG, Asperen IA van. Epidemiology of rotavirus infections in the Netherlands. *Acta Paediatr Suppl* 1999; **426**: 31–7.
- Takkinen J, Ammon A, Robstad O, Breuer T. European survey on campylobacter surveillance and diagnostics. Report, August 2001. Project financed by the European Commission, DG Sanco IV, agreement no. SI2.137721 (99CVF4-032).
- Saeed AM, ed. *Salmonella enterica* Serovar Enteritidis in humans and animals – epidemiology, pathogenesis and control. Ames: Iowa State University Press, 1999: 19–102.
- CDC. Preliminary FoodNet Data on the incidence of foodborne illnesses – selected sites, United States, 2001. *MMWR* 2001; **50**: 241–6.
- Pelt W van, Wit MAS de, Giessen AW van de, Leeuwen WJ van, Duynhoven YTHP van. Decrease of infections with *Salmonella* spp. in humans; demographic changes and shifts in serovars. *Infectieziekten Bull* 1999; **10**: 98–101.
- Council directive 92/117/EEC, concerning measures for protection against specified zoonoses and specified zoonotic agents in animal and products of animal origin in order to prevent outbreaks of foodborne infections and intoxications. 17 December 1992.
- NHG-Standard on Acute Diarrhoea. *Huisarts en Wetenschap* 1993; 36(9).

23. Vermaak MP, Langendam MW, Hoek JAR van den, Peerbooms PGH, Coutinho RA. Shigellosis in Amsterdam, 1991–1998: occurrence and results of contact tracing. *Ned Tijdschr Geneesk* 2000; **144**: 1688–4.
24. Blystad H. Travel-related notifiable diseases imported to Norway 1998. *Eurosurveillance Weekly* 1999; 30.
25. Lee LA, Shapiro CN, Hargreft-Bean N, Tauxe RV. Hyperendemic shigellosis in the United States: a review of surveillance data for 1967–1988. *J Infect Dis* 1991; **164**: 894–6.
26. Verhaegen J, Charlier J, Lemmens P, et al. Surveillance of human *Yersinia enterocolitica* infections in Belgium: 1967–1996. *Clin Infect Dis* 1998; **27**: 59–5.
27. Stolk-Engelaar VM, Hoogkamp-Korstanje JA. Clinical presentation and diagnosis of gastrointestinal infections by *Yersinia enterocolitica* in 261 Dutch patients. *Scand J Infect Dis* 1996; **28**: 571–5.
28. Butler T. *Yersinia* species, including plague. In: Mandeel GL, Bennett JE, Dolin R, eds. *Principles and practice of infectious diseases*, 5th edn. Edinburgh: Churchill Livingstone, 2000: 2400–13.
29. Heuvelink AE, Tilburg JJHC, Herbes RG, et al. A family outbreak of *E. coli* O157 infections. *Infectieziekten Bull* 1998; **9**: 44–8.
30. Ammon A. Surveillance of enterohaemorrhagic *E. coli* (EHEC) infections and haemolytic uraemic syndrome (HUS) in Europe. *Eurosurveillance* 1997; **2**: 91–5.
31. Decludt B, Bouvet P, Mariani-Kurkdjian, et al. Haemolytic uraemic syndrome and Shiga toxin-producing *E. coli* infection in children in France. *Epidemiol Infect* 2000; **124**: 215–5.
32. Heuvelink AE. The occurrence of Shiga toxin-producing *E. coli* in humans and animals. *Tijdschr Diergeneesk* 1999; **124**: 671–7.