Health Protection Scotland
Food Standards Agency (Scotland)

Acknowledgements
The authors would like to thank the Graphics Department of Health Protection Scotland for their valuable assistance with the production of this document.

Health Protection Scotland is a division of NHS National Services Scotland.

Health Protection Scotland website: http://www.hps.scot.nhs.uk

Published by Health Protection Scotland, NHS National Services Scotland, Meridian Court, 5 Cadogan Street, Glasgow G2 6QE.

First published August 2013

© Health Protection Scotland 2013

Reference this document as:

Contributing Authors: Cowden J M, McElhiney J, Smith-Palmer A, Locking M, Browning L, Home J and Brownlie S.

Health Protection Scotland has made every effort to trace holders of copyright in original material and to seek permission for its use in this document. Should copyrighted material have been inadvertently used without appropriate attribution or permission, the copyright holders are asked to contact Health Protection Scotland so that suitable acknowledgement can be made at the first opportunity.

Health Protection Scotland consents to the photocopying of this document for professional use.

All other proposals for reproduction of large extracts should be addressed to:

Health Protection Scotland
NHS National Services Scotland
Meridian Court
5 Cadogan Street
Glasgow G2 6QE
Tel: +44 (0) 141 300 1100
Email: nss.hpsenquiries@nhs.net
Foreword

This is the first joint annual report published by Health Protection Scotland and the Food Standards Agency (Scotland), focussing on the key pathogens identified by the UK Foodborne Disease Strategy.

Although most of these data are available in other publications, including the UK Zoonoses Report and Health Protection Scotland’s Weekly Report outputs, both organisations felt it would be worthwhile to produce a comprehensive report on infectious intestinal disease associated with foodborne illness in Scotland and detail the initiatives that are planned or underway to reduce foodborne disease.

Whilst the primary focus is 2012, we have included data for previous years to provide context where appropriate.
# Table of Contents

1. **Foreword** i
2. **Introduction** 1
3. **Campylobacter** 5
4. **Listeria** 9
5. **E. coli O157 and other VTEC (VTEC/O157)** 13
6. **Salmonella (non-typhoidal)** 21
7. **General outbreaks of Infectious Intestinal Disease** 26
8. **Conclusion** 29
1 Introduction

Infectious intestinal disease (IID), including foodborne disease, is a major cause of illness in the UK population, affecting an estimated one million people each year. Although most cases of IID are mild, it is at best unpleasant and at worst can lead to hospitalisation or death. Foodborne IID puts a massive demand on healthcare services, costing the UK economy an estimated £1.9 billion annually (http://www.foodmanufacture.co.uk/Food-Safety/Sound-science-key-to-protect-against-1M-foodborne-illness-FSA).

The Food Standards Agency in Scotland (FSAS) aims to reduce foodborne disease in Scotland through the UK Foodborne Disease Strategy (FDS) (http://www.food.gov.uk/multimedia/pdfs/fds2015.pdf). The FDS targets those pathogens responsible for the greatest burden of foodborne IID in the UK, and those areas of the food chain where controls can offer the greatest improvement to public health.

The key pathogens addressed in the FDS are:

- Campylobacter, the most commonly reported bacterial cause of potentially foodborne IID in the UK,
- Listeria monocytogenes, the potentially foodborne pathogen with a high case fatality rate.

These pathogens are targeted through specific risk management programmes which are aimed at reducing the risks of contamination with these pathogens at all stages of the food chain.

The FDS also addresses the following pathogens:

- Verotoxigenic Escherichia coli and E. coli O157 (VTEC/O157) which can cause haemolytic uraemic syndrome (HUS), the commonest cause of acute renal failure in children in the UK,
- Norovirus (NV), the most commonly reported viral cause of potentially foodborne IID in the UK,
- Salmonella (non-typhoidal), which, although declining in incidence, continues to be an important foodborne pathogen.

Health Protection Scotland (HPS), among its other duties, is responsible for the surveillance of all these pathogens. Laboratory notification is the foundation of HPS’s
surveillance of all potentially foodborne pathogens, including those addressed in the FSA’s FDS. Since 1 January 2010 the Public Health etc (Scotland) Act 2008 has required directors of clinical microbiology laboratories in Scotland to notify the identification from humans of specified pathogens to their NHS Boards. Because all NHS laboratories in Scotland already report the identification of pathogens to HPS via the Electronic Communication of Surveillance in Scotland (ECOSS), their continuing to do so is deemed to constitute notification. Laboratories not using ECOSS (for example private laboratories) are legally obliged to notify the identification of pathogens to their NHS Board in writing.

The information reported via ECOSS includes a range of demographic fields, including: age, sex, and postcode of residence (where known by the submitting laboratory). In addition there may be some information relating to symptoms, exposure risks, or recent foreign travel, but this is not consistently provided.

HPS collects supplementary microbiological information on the confirmation and detailed typing of:

- Listeria monocytogenes, from the Laboratory of Gastrointestinal Pathogens at Colindale
- VTEC/O157, from the Scottish E. coli O157/VTEC Reference Laboratory
- Salmonella, from the Scottish Salmonella, Shigella, and Clostridium difficile Reference Laboratory

HPS collects supplementary epidemiological, clinical, and exposure information (“enhanced surveillance”) on cases of infection with Listeria and VTEC/O157.

As well as laboratory based surveillance and enhanced surveillance, HPS carries out clinical surveillance of HUS, and integrates the results with the laboratory and enhanced surveillance of VTEC/O157.

Finally, HPS collects information on general outbreaks of IID via ObSurv. ObSurv is the system established in 1996 for the surveillance of all general outbreaks of IID in Scotland.

Details of these systems (based on reference laboratory information, enhanced surveillance, clinical surveillance, and outbreak surveillance) are provided in more detail in the relevant sections of this report.

Not all cases of infection are captured by laboratory surveillance. In order for cases to be reported to HPS, they must have had a specimen taken, usually by their General Practitioner (GP), and the laboratory must identify the pathogen and notify
it. GPs take specimens to inform clinical management, not to ensure the accuracy of national surveillance. GPs are therefore probably more likely to take specimens when illness is severe or protracted, when cases have returned from abroad, or when patients are particularly vulnerable, e.g. the very old or the very young.

Accepting that ascertainment in different population groups may vary, the IID2 study (http://www.foodbase.org.uk//admintools/reportdocuments/711-1-1393_IID2_FINAL_REPORT.pdf) calculated population average factors by which national (UK) surveillance data should be multiplied to give more accurate estimates of the true number of cases in the community. These multipliers (and their 95% Confidence intervals) are presented in the relevant sections.

The fact that these multipliers are for the UK as a whole, but in reality differ between different sub-groups of the population emphasises the need for caution in their interpretation – especially when they have wide confidence intervals, e.g. that for VTEC/O157. Nevertheless they are the most accurate measure we have of the unavoidable under-reporting to laboratory surveillance systems which are not designed to test a representative sub-set of cases of any infection, but are the results of clinically prompted investigation.

HPS’s laboratory surveillance systems are not representative surveys of infection. They utilise data generated from clinical investigations, and are influenced by laboratory and clinical practices, and the behaviour of the public. A difference in the number of laboratory reports of a pathogen from year to year may therefore result from factors other than a true change in incidence. However, in the absence of any evidence of, say, a new laboratory protocol, or of a change in the behaviour of GPs or the public, the under-ascertainment of a particular pathogen is likely to be constant from year to year, and changes in annual totals a fair reflection of actual trend.

Although the pathogens addressed in this report are all potentially foodborne, infection can occur by other routes, e.g. person to person spread, or acquisition from the environment, water supplies or direct animal contact. It is very difficult to put a precise figure on the proportion of cases of infection with each pathogen which is foodborne. An extension to the IID2 study has estimated this proportion, but has not been published at the time of going to press. The estimates of the proportion of infections which are foodborne presented in the relevant sections are the best currently available (http://gut.bmj.com/content/51/6/832/T1.expansion.html), although this study was based on data from England and Wales, these proportions can be applied in Scotland. It is also true that not all cases of foodborne infection with these pathogens are acquired in Scotland, or indeed the UK. Again the best currently available estimates are presented in the relevant sections.
Because of all these caveats, this report addresses laboratory reports, and does not attempt to describe Scotland or UK acquired foodborne illness. As it is unlikely that the proportion of cases acquired indigenously or from food has changed in recent years, and as there is no evidence of changes in rates of ascertainment, year to year comparisons of laboratory reports are probably justified.
2 Campylobacter

Campylobacter is the most commonly reported cause of bacterial IID in Scotland, the UK, and the rest of the developed world. HPS’s surveillance consists of laboratory reporting without the collection of additional epidemiological or microbiological information. Laboratory reports probably underestimate its incidence by a factor of 9.3 (95% CI, 6-14.3) (http://www.foodbase.org.uk//admintools/reportdocuments/711-1-1393_IID2_FINAL_REPORT.pdf). It is estimated that 79.7% of Campylobacter infection is foodborne and 78.4% acquired in the UK (http://gut.bmj.com/content/51/6/832/T1.expansion.html). Infection typically causes a severe illness lasting between 24 hours and over a week characterised by diarrhoea (which is often bloody), abdominal pain, and sometimes fever. Campylobacter can occasionally lead to serious, long term complications including neurological, rheumatological and renal problems, the most important of which is Guillian-Barré Syndrome (GBS), a demyelinating disorder resulting in acute neuromuscular paralysis. The risk of developing GBS after Campylobacter infection is about 1 in 1000 (J Infect Dis. 1997 Dec;176 Suppl 2:S125-8).

The number of laboratory reported cases of Campylobacter infection has remained stubbornly high at around 6,000 a year since 2009 (Table 1). The increase from just under 5,000 to over 6,000 between 2008 and 2009 was probably real, but remains unexplained (Figure 1).

<table>
<thead>
<tr>
<th>Year</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>2008</td>
<td>4878</td>
</tr>
<tr>
<td>2009</td>
<td>6415</td>
</tr>
<tr>
<td>2010</td>
<td>6601</td>
</tr>
<tr>
<td>2011</td>
<td>6366</td>
</tr>
<tr>
<td>2012</td>
<td>6333</td>
</tr>
</tbody>
</table>

Campylobacter infection is almost always apparently sporadic. Since 1996, ObSurv has identified only 34 general outbreaks of Campylobacter. From 1996 to 2012 there have been 93,896 laboratory reports of Campylobacter, and 445 cases reported from the 31 outbreaks for which forms are available, of whom 155 (35% of cases reported in outbreaks and 0.16% of all confirmed cases) were laboratory confirmed. This minute percentage of outbreak cases has remained consistent over the years.
In 2012, ObSurv recorded one general outbreak of both Campylobacter and Cryptosporidium infection (Figure 21). The mode of transmission was waterborne.

FIGURE 1: Laboratory reports of Campylobacter in Scotland, 2008-2012

FIGURE 2: Number of laboratory confirmed cases of Campylobacter in 2012 and the rate per 100,000 by age band and sex

In all age bands, apart from those aged 20-24 years, the rate per 100,000 was greater in males than females (Figure 2).

In Scotland the overall rate of Campylobacter infection in 2012 was 120.9 per 100,000 compared to 121.9 per 100,000 in 2011 (Figure 3). At an NHS Board level the rate of Campylobacter declined in seven Boards – Ayrshire and Arran, Borders, Fife, Greater Glasgow and Clyde, Lanarkshire, Tayside and Western Isles. In the other seven NHS Boards there was an increase in the rate per 100,000; the greatest increase in rates were observed in Orkney and Shetland. The rates in the Island NHS Boards should be viewed with caution due to the effect of their small population size.
Among the mainland NHS Boards the rates of Campylobacter ranged from 77.3 per 100,000 in Fife to 155.0 per 100,000 in Borders.

FIGURE 3: Campylobacter in Scotland 2012 (and 2011), Rates per 100,000 by Health Board Area
The seasonality of reports of Campylobacter follows the same general trend every year – with most reports made in the summer months (Figure 4). There is occasionally a second, less pronounced peak in the autumn.

The main source of Campylobacter infection in humans is raw poultry meat, with as many as 60-80% of cases in Scotland attributed to exposure to chicken (Sheppard et al, 2009, Clinical Infectious Diseases, 48, 1072-1078). In the UK, 65% of chicken at retail is contaminated with Campylobacter (http://www.food.gov.uk/multimedia/pdfs/fds2015.pdf).

The focus for the Campylobacter Risk Management Programme (CRMP) (http://food.gov.uk/policy-advice/microbiology/campylobacterevidencedprogramme/) is to reduce the levels of this pathogen in poultry. This programme of work is being taken forward by FSA, in partnership with producers and retailers, through a Joint Government/Industry working group which aims to reduce the percentage of chickens produced in UK poultry slaughterhouses with the highest levels of Campylobacter contamination from 27% (the baseline in 2008) to 10% by 2015. If successful the FSA estimates that this could lead to a reduction in Campylobacter foodborne infection of up to 30%, which is equivalent to about 111,000 actual cases of Campylobacter food poisoning per year.
3 Listeria

In 2002 HPS introduced enhanced surveillance for laboratory confirmed cases of Listeria species including Listeria monocytogenes. For every laboratory confirmed case reported, HPS sends a surveillance form (either pregnancy or non-pregnancy) for completion by the appropriate NHS Board’s Health Protection Team. Due to the high mortality rate of approximately 30% and the presence of serious underlying medical conditions in a large proportion of cases, it is not always possible for all the data items on the form to be completed. In line with ECDC reporting requirements, pregnancy associated cases where Listeria is isolated from both the mother and infant are counted as one case. The epidemiological data is linked to the laboratory data obtained via ECOSS (Table 2) and any additional typing results available from the laboratory of Gastrointestinal Pathogens at Colindale.

Table 2: Listeria in Scotland 2008 – 2012. Human laboratory reports

<table>
<thead>
<tr>
<th>Year</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>2008</td>
<td>15</td>
</tr>
<tr>
<td>2009</td>
<td>17</td>
</tr>
<tr>
<td>2010</td>
<td>17</td>
</tr>
<tr>
<td>2011</td>
<td>14</td>
</tr>
<tr>
<td>2012</td>
<td>11</td>
</tr>
</tbody>
</table>

Because of the low number of cases, the IID2 study was unable to calculate a multiple for the under-ascertainment of Listeria infection (http://www.foodbase.org.uk/admintools/reportdocuments/711-1-1393_IID2_FINAL_REPORT.pdf). However due to its severe clinical presentation, including meningitis and septicaemia, it is unlikely that many symptomatic cases fail to seek medical attention or have appropriate samples taken for laboratory diagnosis. Listeria infection is thought to be foodborne in 99% of cases and 100% acquired in the UK (http://gut.bmj.com/content/51/6/832/T1.expansion.html). Due to the long incubation period of up to 90 days, assigning the country where infection is acquired is more problematic than for most other foodborne pathogens, and evidence from the enhanced surveillance in Scotland suggests that over the past five years a few of the cases may have acquired their infection outside the UK.

In 2012, there were 11 laboratory confirmed cases of L. monocytogenes, a slight decrease on the 14 cases reported in 2011. This is the third consecutive year in which reports have declined and is the lowest annual total recorded in the past 10
years. The low number of cases each year makes the interpretation of trends difficult (Figure 5). From the small number of cases reported each year, there is no detectable seasonality of Listeria infection (Figure 7).

Six (54.5%) of the 11 cases reported in 2012 were aged 65 years and over (Figure 6), which is similar to 2011 when 50% of cases were 65 years and over.

All 11 cases reported in 2012 were admitted to hospital, and one death was reported (information on mortality was available for eight cases), a case fatality rate of 9%.

In 2011, outcome data were available for all 14 cases reported, two of whom died, which is a case fatality rate of 14%. Although these deaths were reported among cases of Listeria infection, many cases had serious co-morbidities which may have either been the primary cause of death or a contributing factor. The surveillance system does not collect information on the cause of death as recorded on the death certificate.

In 2012 there was one pregnancy associated case (where the mother, infant, or both were laboratory confirmed), which is similar to the historical trends observed since enhanced surveillance was introduced in 2002 with between one and three pregnancy associated cases a year.

In 2012, four of the 11 cases were reported to have cancer/malignancy as an underlying medical condition (information on co-morbidity was only available for 8 cases). In 2011 cancer/malignancy was reported for 4 of the 14 cases.

Since the establishment of ObSurv in 1996 there has only been one general outbreak of L. monocytogenes reported, this was in 2005 and was not considered to be foodborne.

The aim of the FSA’s Listeria Risk Management Programme (LRMP) (http://food.gov.uk/policy-advice/microbiology/listeria/) is to reduce the risks of L. monocytogenes in the production, storage and handling of chilled ready to eat foods, which are most commonly associated with human illness. The LRMP will address this firstly by identifying effective measures for controlling L. monocytogenes in the production of these foods, and develop tools to assist food businesses (particularly small and medium enterprises) to minimise the risk of contamination. Secondly, it will develop communication strategies and food safety messaging to ensure information about risk and avoidance is communicated effectively to the individuals who are at highest risk from listeriosis.
FIGURE 5: Laboratory reports of Listeria in Scotland, 2008-2012

FIGURE 6: Listeria in Scotland, 2012 by age band
FIGURE 7: Listeria in Scotland 2012, by four week period
4 *E. coli* O157 and other VTEC (VTEC/O157)

*Escherichia coli* O157 (*E. coli O157*) is the only serogroup of verotoxigenic *E. coli* (VTEC) detectable by faecal culture in local NHS diagnostic laboratories in Scotland. The Scottish *E. coli O157/VTEC Reference Laboratory* (SERL) identifies, types, and sub-types isolates of O157, and non-O157 VTEC from clinically suspicious but locally negative faecal samples. Laboratory identifications of VTEC are notifiable to HPS under the Public Health etc (Scotland) Act 2008. From 2008 to 2012, the average annual total notified was 235 O157 (Table 3), and 30 non-O157 VTEC (Figure 8). Since 2004 HPS enhanced surveillance has supplemented laboratory reports with epidemiological, clinical, and exposure information from NHS Board Health Protection Teams. HPS also conducts clinical surveillance of haemolytic uraemic syndrome (HUS) and integrates the resultant information into the national VTEC dataset.

Table 3: *E. coli* O157 in Scotland: Culture positive cases reported to HPS 2008-2012

<table>
<thead>
<tr>
<th>Year</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>2008</td>
<td>241</td>
</tr>
<tr>
<td>2009</td>
<td>237</td>
</tr>
<tr>
<td>2010</td>
<td>212</td>
</tr>
<tr>
<td>2011</td>
<td>253</td>
</tr>
<tr>
<td>2012</td>
<td>234</td>
</tr>
</tbody>
</table>

Although uncommon compared to *Campylobacter* or *Salmonella*, VTEC/O157 is an important public health challenge in Scotland. Its low infective dose allows it to cause large outbreaks (often foodborne). Although infection can be asymptomatic, it can cause a spectrum of illness ranging from mild diarrhoea, through bloody diarrhoea and haemorrhagic colitis, to HUS or death. Around 60% of cases in Scotland have bloody diarrhoea, and 9% HUS (particularly affecting those aged under 16 or over 60 years).

Some under-ascertainment of cases is likely, but the multiplier of 7.4 and the wide confidence interval (95% CI: 0.5-104.4) reported in the IID2 Study ([http://www.foodbase.org.uk/admintools/reportdocuments/711-1-1393_IID2_FINAL_REPORT.pdf](http://www.foodbase.org.uk/admintools/reportdocuments/711-1-1393_IID2_FINAL_REPORT.pdf)) highlight the danger of generalising from studies to national populations. A multiplier of 0.5 suggests that, statistically, actual case numbers may be as few as half those reported, implying a 50% laboratory false
positivity rate, which – in Scotland - is untenable. As most reported cases have bloody diarrhoea and acute abdominal pain it is likely that most actual cases seek medical help. Furthermore, in Scotland there tends to be assiduous sampling of people linked to confirmed or suspected cases. The higher reported incidence in Scotland compared to the rest of the UK may reflect a higher true incidence or a lower threshold for testing, or a combination of both.

FIGURE 8: E. coli O157 and non-O157 VTEC in Scotland: Culture positive cases reported to HPS, 1984-2012

Ruminants, particularly cattle and sheep are the main reservoir of VTEC/O157. The sources and routes of transmission of O157 and non-O157 VTEC appear similar. Foodborne transmission can occur when the surface of raw meat becomes contaminated during slaughter and processing. Unpasteurised or inadequately pasteurised milk, or raw vegetables, may also become contaminated. Minced beef products pose a particular risk if not cooked properly. Although meat and dairy products have caused large VTEC/O157 outbreaks in the UK, other food vehicles worldwide include salad leaves, white radish and other sprouted seeds, and raw vegetables.

From 1996-2012, 39 (28%) outbreaks of VTEC/O157 were foodborne. Meat or dairy products were suspected in most outbreaks in which a vehicle was reported. Most outbreaks were small: in 37 the average number of cases per outbreak was five. The central Scotland outbreak in 1996, in which the main suspect vehicle was cold, cooked meats, resulted in 296 laboratory-confirmed cases. A Great Britain-wide outbreak in 2011, associated with vegetables, resulted in 250 cases, 44 of which
were in Scotland (17% of all cases in Scotland that year). No foodborne VTEC/O157 outbreaks were identified in 2012.

HPS data suggest that in Scotland 35-40% of VTEC/O157 cases are foodborne, and 11% due to person-to-person spread: a further 15% of cases are likely to have acquired infection outside the UK. The source of the remaining 35-40% is difficult to determine but any environment or water contaminated by animal (or human) faeces is a potential source of infection, as well as direct animal contact.

Almost 50% of cases in Scotland are in children under 16 years of age (Figure 9). Rates of VTEC/O157 infection are highest in children under 5 years, at 15 cases per 100,000 population (2008-2012), compared with a population average of 4.5 per 100,000. On average 41% of all VTEC/O157 cases are hospitalised. Amongst cases with HUS, 85% are under 16 years old. Less than 2% of cases in Scotland died following infection, although data on exact cause of death is often unavailable.

Reports of E. coli O157 generally peak in the third quarter of the year (Figure 10). Reports of non-O157 VTEC are too few in number for seasonality to be estimated.

FIGURE 9: E. coli O157 in Scotland: Culture positive cases reported to HPS, 2012 by age band and sex
FIGURE 10: *E. coli* O157 in Scotland: Culture positive cases reported to HPS, 2012 by four week period

On average 80% of cases are not part of general outbreaks (i.e. they are apparently sporadic cases or single household clusters). Reported incidence of VTEC/O157 varies considerably across Scotland (Figure 11). Although the drivers of VTEC/O157 infection are multifactorial, geographic differences in infection in Scotland may be influenced by the complex relationships between relative densities of cattle and human population. While incidence in NHS Boards sometimes varies substantially from year to year, in most years the highest rates amongst mainland Boards have been reported in Dumfries & Galloway or Grampian, with Borders occasionally reporting higher rates, as occurred in 2012.
Annual case numbers in recent years have remained stubbornly consistent despite the publication in 2001 of 104 recommendations for control measures in the joint Scottish Executive/Food Standards Agency (Scotland) E. coli O157 Task Force Report. This is one of the factors that has led to the Scottish Government commissioning a VTEC/E. coli O157 Action Group (co-ordinated by HPS), tasked in particular to produce a VTEC/E. coli O157 Action Plan for Scotland, which it is anticipated will be published later in 2013, and which will give attention to foodborne routes of transmission.
5 Norovirus

Norovirus (NV) is the second most commonly reported pathogen causing IID in Scotland (after Campylobacter). HPS’s surveillance consists of laboratory reporting without the collection of additional epidemiological or microbiological information. Laboratory reports probably underestimate its incidence by a factor of 288 (95% CI, 239-346) ([http://www.foodbase.org.uk//admintools/reportdocuments/711-1-1393_IID2_FINAL_REPORT.pdf](http://www.foodbase.org.uk//admintools/reportdocuments/711-1-1393_IID2_FINAL_REPORT.pdf)). Another study estimates that 10.7% of NV infection is foodborne and 100% acquired in the UK ([http://gut.bmj.com/content/51/6/832/T1.expansion.html](http://gut.bmj.com/content/51/6/832/T1.expansion.html)). Because of this massive underestimate, NV is therefore a very important foodborne pathogen, despite the small proportion estimated to be foodborne. Symptoms of NV infection usually consist of sudden onset of vomiting, watery diarrhoea and nausea, occasionally accompanied by flu like symptoms, including fever, and lasting 12 to 60 hours. Dehydration may occur, and hospital treatment is sometimes necessary, particular in the very young and very old.

With the exception of bivalve molluscs, the contamination of most food with NV results from an infected foodhandler. NV infection is not a zoonosis.

Since 2008 there has been no discernible trend in the incidence of laboratory reports of NV which have varied between about 1500 and 3000 a year (Table 4, Figure 12). Almost all cases of NV are attributable to outbreaks. This may be partly artefactual as the virus is seldom sought in sporadic cases. In none of the 329 outbreaks of NV infection identified by ObSurv in 2012 was a foodborne component reported.

Table 4: Norovirus in Scotland 2008 – 2012. Human laboratory reports

<table>
<thead>
<tr>
<th>Year</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>2008</td>
<td>2170</td>
</tr>
<tr>
<td>2009</td>
<td>2200</td>
</tr>
<tr>
<td>2010</td>
<td>3109</td>
</tr>
<tr>
<td>2011</td>
<td>1668</td>
</tr>
<tr>
<td>2012</td>
<td>2920</td>
</tr>
</tbody>
</table>
Although NV infection usually results in mild illness it can occasionally be serious, particularly in the elderly, among whom it is especially common (Figure 13).

The seasonality of reports of NV follows the same general trend every year – with most reports made in the winter (Figure 14). Most cases during the winter occur in residential institutions and hospitals, whereas most of those in the summer occur in holiday and leisure settings, including hotels and cruise ships.
In Scotland the overall rate for reports of NV in 2012 was 55.8 per 100,000 compared to 31.9 per 100,000 the previous year (Figure 15). The increase in rates of NV was observed across 12 of the NHS Boards, with the rates in the other two Boards remaining the same. Among the NHS Boards rates ranged from 0 to 154.5 per 100,000. Some of the difference between NHS Boards may be due to differences in clinical or reporting practices. The rates for the Island NHS Boards should be viewed with caution due to the effect of their small population size.

FIGURE 14: Norovirus in Scotland, 2012 by four week period

FIGURE 15: Norovirus in Scotland 2012 (and 2011), Rates per 100,000 by Health Board Area
6 *Salmonella* (non-typhoidal)

Salmonella is the second most commonly reported cause of bacterial infectious intestinal disease in Scotland after *Campylobacter*. The surveillance of *Salmonella* in Scotland relies on reports from the Scottish *Salmonella*, *Shigella* and *Clostridium difficile* Reference Laboratory (SSSC DRL) who receive isolates from all diagnostic routine microbiology laboratories. In addition, SSSC DRL receive animal, environmental, water and food isolates for identification and typing and thus provides a valuable opportunity for the surveillance and comparison of all isolates. Moreover, SSSC DRL subject all human isolates to molecular typing in real time to facilitate the rapid detection of outbreaks. Laboratory reports probably underestimate the incidence of *Salmonella* infection by a factor of 4.7 (95% CI 1.1-18.2) ([http://www.foodbase.org.uk//admintools/reportdocuments/711-1-1393_IID2_FINAL_REPORT.pdf](http://www.foodbase.org.uk//admintools/reportdocuments/711-1-1393_IID2_FINAL_REPORT.pdf)). Another study estimates that 91.6% of salmonellosis is foodborne and 88.2% acquired in the UK ([http://gut.bmj.com/content/51/6/832/T1.expansion.html](http://gut.bmj.com/content/51/6/832/T1.expansion.html)). Common symptoms include diarrhoea, nausea, fever and occasionally vomiting. *Salmonella* is a zoonosis – a disease which can be transmitted between animals and humans. Humans can acquire infection by direct or indirect contact with infected animals, or through the consumption of food.

During the early 1990s the annual totals of *Salmonella* increased most years by around 5% a year until their peak of 3349 reports in 1997. From 1997 to 1998, when vaccination against *Salmonella Enteritidis* was introduced in the poultry industry, there was a 37% decrease in reports and in following years the numbers have continued to drop (Figure 16). There was a small rise in reports in 2004 after which numbers continued to decline although there were slight increases in 2008 and 2010.

In 2012 there were 728 reports of *Salmonella*, a 30% reduction since 2008 (Table 5) and a 78% reduction since the peak in 1997 (Figure 16).

The rate of *Salmonella* infection in Scotland in 2012 was 13.9 per 100,000 population (Figure 19) – a slight decrease on the rate observed in 2011(14.1 per 100,000 population). A decrease in the rate of infection was observed in seven NHS Boards (Dumfries & Galloway, Fife, Forth Valley, Highland, Lothian, Orkney and Tayside). Increased rates were observed in Ayrshire & Arran, Borders, Grampian, Greater Glasgow & Clyde, Lanarkshire, and Shetland though the increase was slight in Grampian. The rates in the Island NHS Boards should be viewed with caution due to the effect of their small population size.
### Table 5: Salmonella in Scotland 2008 - 2012. Human laboratory reports

<table>
<thead>
<tr>
<th>Year</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>2008</td>
<td>1043</td>
</tr>
<tr>
<td>2009</td>
<td>846</td>
</tr>
<tr>
<td>2010</td>
<td>941</td>
</tr>
<tr>
<td>2011</td>
<td>737</td>
</tr>
<tr>
<td>2012</td>
<td>728</td>
</tr>
</tbody>
</table>

### FIGURE 16: Salmonella in Scotland – human laboratory report 1990 - 2012

Most cases of Salmonella were reported in the young and the elderly (Figure 17).

### FIGURE 17: Salmonella in Scotland by age band and sex
The seasonality of reports of Salmonella follows the same general trend every year - with most reports made in the summer months (Figure 18).

**FIGURE 18: Salmonella in Scotland, 2012 by four week period**

![Graph showing the seasonality of Salmonella reports in Scotland, 2012 by four week period.](image)

**FIGURE 19: Salmonella in Scotland 2012 (and 2011), Rates per 100 000 by Health Board Area**

![Map of Scotland showing the distribution of Salmonella cases by Health Board Area.](image)

Salmonella Enteritidis and Salmonella Typhimurium are the most commonly reported serotypes, and the only two serotypes with more than 100 reports in 2012. The next commonest serotype was Salmonella monophasic group B.
Table 6: Commonly reported serotypes in Scotland, 2012

<table>
<thead>
<tr>
<th>Serotype</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>S. Enteritidis</td>
<td>252</td>
</tr>
<tr>
<td>S. Typhimurium</td>
<td>139</td>
</tr>
<tr>
<td>Salmonella monophasic group B</td>
<td>31</td>
</tr>
<tr>
<td>Others</td>
<td>296</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>728</strong></td>
</tr>
</tbody>
</table>

There has been a dramatic reduction in reports of PT4 from the peak in 1997 where 72% of Salmonella Enteritidis were PT4, to the situation in 2012 where 7% of Enteritidis were PT4 (Table 7). PT8 was the most commonly reported type in 2012 (and the previous two years) and laboratory reports suggest around 30% may have been acquired abroad.

Table 7: S. Enteritidis phage types in Scotland 2008 - 2012

<table>
<thead>
<tr>
<th></th>
<th>2008</th>
<th>2009</th>
<th>2010</th>
<th>2011</th>
<th>2012</th>
</tr>
</thead>
<tbody>
<tr>
<td>PT4</td>
<td>84 (21%)</td>
<td>55 (17%)</td>
<td>39 (13%)</td>
<td>13 (6%)</td>
<td>18 (7%)</td>
</tr>
<tr>
<td>PT1</td>
<td>73 (18%)</td>
<td>37 (12%)</td>
<td>47 (16%)</td>
<td>38 (17%)</td>
<td>17 (7%)</td>
</tr>
<tr>
<td>PT8</td>
<td>44 (11%)</td>
<td>35 (11%)</td>
<td>60 (20%)</td>
<td>54 (24%)</td>
<td>78 (31%)</td>
</tr>
<tr>
<td>PT21</td>
<td>52 (13%)</td>
<td>39 (12%)</td>
<td>12 (4%)</td>
<td>13 (6%)</td>
<td>16 (6%)</td>
</tr>
<tr>
<td>PT14b</td>
<td>39 (10%)</td>
<td>16 (5%)</td>
<td>17 (6%)</td>
<td>24 (11%)</td>
<td>34 (13%)</td>
</tr>
</tbody>
</table>

There has been a notable drop in reports of S. Typhimurium DT104. In 2012 there were only 9 reports, a decrease of 58% on the previous year (Table 8). DT193 was the most commonly reported type in 2012, mirroring the situation seen in the previous two years.

Table 8: S. Typhimurium phage types

<table>
<thead>
<tr>
<th></th>
<th>2008</th>
<th>2009</th>
<th>2010</th>
<th>2011</th>
<th>2012</th>
</tr>
</thead>
<tbody>
<tr>
<td>DT104</td>
<td>30 (15%)</td>
<td>18 (13%)</td>
<td>27 (14%)</td>
<td>21 (12%)</td>
<td>9 (6%)</td>
</tr>
<tr>
<td>DT193</td>
<td>4 (2%)</td>
<td>4 (3%)</td>
<td>46 (24%)</td>
<td>52 (29%)</td>
<td>49 (35%)</td>
</tr>
<tr>
<td>RDNC</td>
<td>43 (22%)</td>
<td>23 (17%)</td>
<td>28 (14%)</td>
<td>20 (11%)</td>
<td>16 (12%)</td>
</tr>
<tr>
<td>DT120</td>
<td>14 (7%)</td>
<td>10 (7%)</td>
<td>26 (13%)</td>
<td>30 (17%)</td>
<td>14 (10%)</td>
</tr>
</tbody>
</table>
Salmonella has declined impressively since 1997 (Figure 20), mainly because of the virtual eradication of S. Enteritidis PT4 as a result of the identification in the late 1980s of fresh shell eggs as the most likely vehicle of infection, and the introduction in the late 1990s of vaccination of laying flocks against this serotype.

FIGURE 20: Salmonella in Scotland 1990 – 2012. The decline of S. Enteritidis PT4
7 General outbreaks of Infectious Intestinal Disease

HPS collects information on general outbreaks of infectious intestinal disease (IID) via ObSurv. ObSurv is the surveillance system established in 1996 for all general outbreaks of IID in Scotland. For the purpose of ObSurv an outbreak is defined as an incident in which two or more linked cases experience the same illness or when the observed number of cases unaccountably exceeds the expected number. The system seeks information on general outbreaks, defined as outbreaks affecting members of more than one household or residents of an institution. For foodborne outbreaks the system seeks information not only on the suspected food vehicle, but also the type of evidence supporting the association (microbiological, epidemiological or descriptive) and any issues that are thought to have contributed to the outbreak. ObSurv is a voluntary surveillance system and therefore it is likely that some outbreaks will not be captured.

In 2012 a total of 351 general outbreaks of IID were reported to ObSurv, this is the third highest number of outbreaks reported in a single year since ObSurv was established in 1996. The peak year for outbreaks was 2010 when 371 general outbreaks were reported.

Figure 21 shows the pathogens responsible for general outbreaks of IID in 2012. Of the 351 outbreaks identified:

- 329 (94%) were due to NV, of which none were foodborne
- six (2%) were due to of E. coli O157, of which none were foodborne
- four (1%) were due to Cryptosporidium, of which one was foodborne,
- four (1%) were due to Salmonella (one of S.Typhimurium, one of S. Newport, one of S. Braenderup and one of both Salmonella Group B and C.difficile), of which one (that due to S. Newport) was foodborne and one (that due to S. Braenderup) included a foodborne component
- two (0.5%) were due to C.difficile, none of which none were foodborne
- one (0.25%) was due to Scombrototoxin, and was foodborne
- one (0.25%) was due to rotavirus, and was not foodborne
- one (0.25%) was due to both Campylobacter and Cryptosporidium and was waterborne
three (1%) were of unknown aetiology, one of which was foodborne.

**FIGURE 21:** Pathogen responsible for general outbreak of IID reported to ObSurv in 2012 (n=351)

Completed outbreak report forms were returned to HPS for 348 (99%) of the 351 outbreaks. The remaining three outbreaks for which no ObSurv report forms were available were, based on the preliminary information available to HPS, all NV outbreaks.

Of the 348 outbreaks in 2012 for which HPS has a completed outbreak report form, the reported mode of transmission was:

- mainly foodborne in four outbreaks,
- multiple modes of transmission including a foodborne component in one outbreak.

This total is similar to that seen in recent years (Figure 22).
FIGURE 22: General outbreaks of IID with a food component to their mode of transmission 2008-2012

Table 9: Food vehicles reported for those outbreaks with a food component to their mode of transmission, 2012

<table>
<thead>
<tr>
<th>Pathogen</th>
<th>Mode of transmission</th>
<th>Location</th>
<th>Number affected*</th>
<th>Number laboratory confirmed*</th>
<th>Food vehicle</th>
<th>Type of evidence</th>
<th>Strength of evidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cryptosporidium</td>
<td>FB</td>
<td>Community</td>
<td>5</td>
<td>5</td>
<td>Prewashed bagged mixed salad leaves</td>
<td>Analytical Epidemiological</td>
<td>Strong</td>
</tr>
<tr>
<td>Salmonella Newport</td>
<td>FB</td>
<td>Community</td>
<td>4</td>
<td>4</td>
<td>Watermelon</td>
<td>Descriptive</td>
<td>Weak</td>
</tr>
<tr>
<td>Scrombrototoxin</td>
<td>FB</td>
<td>Hotel</td>
<td>3</td>
<td>0</td>
<td>Tuna mayonnaise sandwiches</td>
<td>Descriptive</td>
<td>Weak</td>
</tr>
<tr>
<td>Unknown</td>
<td>FB</td>
<td>Restaurant</td>
<td>4</td>
<td>0</td>
<td>Rice</td>
<td>Descriptive</td>
<td>Weak</td>
</tr>
<tr>
<td>Salmonella Braenderup</td>
<td>Multi incl FB</td>
<td>Restaurant</td>
<td>7</td>
<td>5</td>
<td>None</td>
<td>Not applicable</td>
<td>Not applicable</td>
</tr>
</tbody>
</table>

* total number of persons reported to be ill
** total number of persons reported to be ill and laboratory confirmed
8 Conclusion

The Food Standards Agency in Scotland (FSAS) aims to reduce foodborne disease in Scotland through the UK Foodborne Disease Strategy (FDS) (http://www.food.gov.uk/multimedia/pdfs/fds2015.pdf). The ultimate test of the success of the FDS is a reduction in indigenously acquired foodborne disease in Scotland.

HPS’s surveillance systems are founded on the monitoring of laboratory reports of potentially foodborne pathogens. As a measure of the absolute numbers of cases of foodborne disease these surveillance systems are neither completely sensitive (they miss an unknown number of cases of foodborne disease because of under-ascertainment) nor specific (they include an unknown number of cases of non-foodborne disease arising from other sources). Furthermore, laboratory reporting is an insensitive way of ascertaining whether infection was acquired in the UK or abroad (not all cases will inform their doctor that they have been abroad, nor will the doctor always inform the laboratory if they do).

Special studies can estimate these deficiencies of sensitivity (http://www.foodbase.org.uk//admintools/reportdocuments/711-1-1393_IID2_FINAL_REPORT.pdf) and specificity but they are expensive and are still only estimates. Enhanced surveillance can improve the accuracy of ascribing infection to the UK or abroad, but is also expensive. Despite its shortcomings, laboratory surveillance is a useful and relatively inexpensive way to monitor the success of the FDS, because as long as there are no changes to the admittedly imperfect sensitivity and specificity of the system, it provides a valid measure of the trend of foodborne disease. Those administering the systems are likely to be aware of any changes to them which might alter the sensitivity and specificity of the systems (for example the introduction of a new technology, or laboratory protocol).

HPS’s surveillance systems indicate that there have been no dramatic changes in the incidence of foodborne disease in Scotland in the past five years.

Campylobacter reports have been stubbornly resistant to control measures at around 6,000 a year since 2009.

Listeria reports have declined from 17 in 2009 to 11 in 2012, but it is difficult to attribute a change in such a small number as a trend.

E. coli O157 and non-O157 VTEC reports have remained relatively stable from 2008-2012. Although case numbers in foodborne VTEC outbreaks have been lower in
recent years, a GB-wide foodborne outbreak in 2011 (associated with vegetables) nonetheless accounted for 17% of E. coli O157 cases in Scotland that year, and raised issues that have since been taken on board in the drafting of the VTEC/E. coli O157 Action Plan for Scotland commissioned by the Scottish Government, which is anticipated to be published later this year.

Norovirus reports have varied widely between 1500 and 3000, with no evidence of trend. As the least likely of the pathogens reviewed to be foodborne, this wide variation may have nothing to do with foodborne transmission.

Salmonella reports appear to be stabilising at around 700 a year, following the impressive declines since 1997 resulting from virtual eradication of S. Enteritidis PT4 achieved by the successful vaccination strategy in laying hens. The success of this strategy, and its reflection in the surveillance data, emphasises the value of the laboratory surveillance system in evaluating intervention measures, despite its limited accuracy.

HPS laboratory surveillance of potentially foodborne pathogens is not a precise measure of indigenously acquired foodborne disease in Scotland, but it is sufficient to record substantial changes that may be a result of the FSA’s Foodborne Disease Strategy.