Costed extension to the Second Study of Infectious Intestinal Disease in the Community: Identifying the proportion of foodborne disease in the UK and attributing foodborne disease by food commodity

Project B18021 (FS231043)

Funder: UK Food Standards Agency

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ACKNOWLEDGEMENTS

We should like to thank the Food Standards Agency for funding this work (Project B18021/ FS231043) and the information scientists at the four national surveillance centres for providing the outbreak data on which some of the analyses are based.

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	priors from published studies and models using a vague Dirichlet prior for the proportion of cases attributable to each food commodity

LIST OF ABBREVIATIONS/GLOSSARY

ACMSF	Advisory Committee on the Microbiological Safety of Food
CI	Confidence Interval: Using frequentist statistical methods a 95% confidence interval means that, with repeated sampling, 95% of the time the ("real") average value will lie within the interval that we calculate. This interval assumes that the parameter (e.g., the average) is fixed and that the observed data are uncertain.
CrI	Credible Interval: Using Bayesian methods a 95% credible interval means that, given the data, there is a 95% probability that the value is within the interval. This interval assumes that the data are fixed (i.e., real) and that the parameter is uncertain.
DALY	Disability adjusted life year
ECDC	European Centre for Disease Prevention and Control
EFSA	European Food Safety Authority
FSA	Food Standards Agency
GBS	Guillain-Barré syndrome
GP	General Practice
HPA	Health Protection Agency
HPS	Health Protection Scotland
HUS	Haemolytic Uraemic Syndrome
ICD	International Classification of Diseases
IID	Infectious Intestinal Disease
IID1	The First Study of Infectious Intestinal Disease in the Community
IID2	The Second Study of Infectious Intestinal Disease in the Community
LSHTM	London School of Hygiene and Tropical Medicine

MeSH	Medical Subject Headings
NDNS	National Diet and Nutrition Survey
ONS	Office of National Statistics
PHA-NI	Public Health Agency- Northern Ireland
RR	Rate Ratio
UoL	University of Liverpool
VTEC	Vero cytotoxin-producing *E. coli*
WHO	World Health Organisation

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CHAPTER 1

EXECUTIVE SUMMARY

1.1 AIM

The main aims of this research were to estimate the burden of UK-acquired foodborne disease in 2009, when the Second Study of Infectious Intestinal Disease (IID2 study) was undertaken, and to quantify the contribution of various food commodities to total foodborne disease burden.

1.2 OBJECTIVE

The objectives were to:-

- determine the burden of foodborne disease that is UK-acquired;
- estimate the burden of foodborne disease caused by contaminated food commodities using a point-of-consumption attribution model.

1.3 METHODS

The study took place between 1st April 2011 and 31st March 2012. To meet the first objective, we developed a model to estimate the number of cases, general practice (GP) consultations and hospital admissions of indigenous foodborne disease due to the major enteric pathogens. We used several different data sources to obtain information on model parameters and their associated uncertainty. We obtained data on pathogen-specific rates of disease from the first and second studies of infectious intestinal disease (IID1 and IID2 studies). We used data from reported outbreaks in the UK and the published literature, obtained from a systematic review that we conducted, to inform estimates of the proportion of IID cases attributable to foodborne transmission. The IID1 and IID2 studies and outbreak data also provided information on pathogen-specific hospitalisation rates.

We incorporated information on these parameters into the model to estimate pathogen-specific numbers of cases, GP consultations and hospital admissions in 2009. We used two modelling approaches: Monte Carlo simulation, which is the standard method that has been used by several research groups worldwide, and a

Bayesian approach, which is novel in this field. We generated three modelling simulations – one using Monte Carlo methods and two using Bayesian methods.

To meet the second objective we extended the food attribution model to estimate, by pathogen, the number of cases, GP consultations and hospital admissions attributable to different food commodities. Information on the proportion of cases attributable to different food commodities was obtained from an analysis of UK outbreak surveillance data and published food attribution studies. We used 12 food commodity groups, which were seafood, dairy, eggs, red meat, game, beef and lamb, pork, poultry, grains and beans, oils and sugars, produce, complex and other foods.

1.4 RESULTS

The three modelling simulations produced broadly consistent results. We report here point estimates but it should be noted that the credibility intervals around all the estimates were wide, indicating a high degree of uncertainty. There were over 500,000 cases of foodborne disease due to known pathogens. *Campylobacter* remained the most common foodborne pathogen in the UK, accounting for approximately 280,000 cases of foodborne illness and 40,000 foodborne illness-related GP consultations. Despite this, *Campylobacter* was responsible for only around 600 acute hospital admissions, reflecting a generally lower level of acute disease severity compared with other bacterial pathogens. Other common foodborne pathogens included *Clostridium perfringens* (around 79,000 cases), norovirus (around 73,000 cases) and *Salmonella* (around 34,000 cases). There were fewer than 10,000 estimated cases of foodborne *E. coli* O157 and fewer than 200 estimated cases of foodborne listeriosis.

Salmonella accounted for approximately 10,000 GP consultations and 2,500 estimated hospital admissions, the largest number of any single organism and reflecting the relatively high hospitalisation rate as estimated from outbreak data and the IID1 study. It should be noted, however, that uncertainty around these hospitalisation estimates was large. *E. coli* O157 ($n \approx 2{,}000$) accounted for almost as many estimated hospital admissions as *Salmonella* ($n \approx 2{,}500$). Viruses caused less than 1,000 hospital admissions.

For a sub-set of foodborne illnesses to which it was possible to attribute a food commodity, poultry was the most common source, accounting for approximately 250,000 cases, 34,000 GP consultations and less than 1,000 hospital admissions. This equated to approximately 50% of all cases and GP consultations, and 20% of hospital admissions for foodborne illness being attributable to poultry. According to our estimates, a person under the age of 65 years, with average consumption patterns, is nearly 40 times more likely to acquire foodborne illness through contaminated poultry than through grains and beans, representing a considerably higher risk compared with other food commodities. Eggs, a well-documented vehicle for *Salmonella* infection, accounted for fewer cases, but were associated with greater disease severity; egg-related infections accounted for only 5% of cases of foodborne illness (n ≈ 31,000), but more than 30% of hospital admissions (n ≈ 1,800). Other important food vehicles included beef and lamb (n ≈ 74,000), seafood (n ≈ 32,000) and produce (n ≈ 49,000).

1.5 DISCUSSION

A major strength of this analysis to determine the burden of foodborne disease is the availability of directly observed pathogen-specific incidence data from the recently completed IID2 study in the UK. However, the use of outbreak data to attribute cases of IID to foodborne transmission relies on certain assumptions - principally that outbreak cases reflect the epidemiology of apparently sporadic cases, particularly that the proportion of foodborne outbreak cases of infection with a particular pathogen is similar to the proportion of apparently sporadic cases infected in the same way by the same pathogen. The present analysis updates a previous burden of foodborne illness study and expands the methods used to incorporate uncertainty. Due to differences in the estimation methods, the two sets of estimates are not directly comparable.

For most food commodities, there was a high degree of uncertainty and estimates should be interpreted with extreme caution. This is particularly true for the hospital admission estimates. The models incorporate data from a range of information sources that were not collected for this purpose, and this accounts for both statistical uncertainty and uncertainty in terms of the current knowledge regarding the role of

different food commodities in transmission of foodborne pathogens; uncertainty is compounded in more complex models with a greater number of parameters.

A further limitation of our analysis is that we were unable to distinguish between illness resulting from direct consumption of foods and that resulting from subsequent person-to-person spread.

Our approach to modelling food attribution is also novel in incorporating both data from outbreaks, as was done previously, with food attribution estimates from previous studies for the estimation of the proportion of foodborne illness attributable to different commodities. This maximises the available information, and the use of published data is useful for informing estimates where data from outbreaks or other sources are not available. Given the declining trend in foodborne outbreaks, it is possible that they will become less useful for food attribution analyses in future.

Finally, our modelling approach can provide a useful summary of the current state of knowledge and models can be updated as new information becomes available.

1.6 CONCLUSIONS

Campylobacter remains the most common foodborne pathogen in the UK. Other common foodborne pathogens include *C. perfringens*, norovirus and *Salmonella*.

Contaminated poultry is the most common contributor to foodborne illness but other important food vehicles included eggs, beef and lamb, seafood and produce.

1.7 RECOMMENDATIONS

1.7.1 Recommendations for future research

- Further work is needed to obtain better estimates of hospitalisation, including length of hospital stay, and deaths from foodborne disease in the UK. This could draw on methods currently being employed by the WHO Foodborne Disease Epidemiology Reference Group study (Kuchenmüller *et al*, 2009). However, it should be noted that, for the majority of pathogens, deaths are associated with vulnerable patients and other underlying diseases.
- Future work should include estimates of disease burden (e.g. DALYS) and costs to help prioritise food safety policy measures. These should take into account the

long term sequelae which, for many foodborne pathogens, outweigh the acute disease burden.

- Better data are needed to be able to attribute illness to foods and to perform food commodity attribution. Alternatives to outbreak data, which are declining, are expert elicitation in the UK context, case-control studies of sporadic illness and molecular subtyping. Generating alternative methods for future use could be undertaken in an international context.
- The use of more complex approaches rather than uniform distributions for modelling the proportion of foodborne illness could be explored.
- Further work is also needed to explore differences in outbreak-associated versus sporadic foodborne illness so that these can be qualitatively or quantitatively incorporated into future models.
- Additional work is required to generate adjusted attribution estimates for the total UK population to accommodate differences among population subgroups, because pathogen incidence is not uniform across age/gender groups and these groups comprise varying proportions of the total population.
- Future work should attempt to determine the extent to which illness follows consumption of foods in which primary contamination has not been effectively dealt with versus consumption of foods that have been cross-contaminated or contaminated by infected food-handlers.
- Estimates of foodborne disease associated with specific food groups could be reviewed in the light of evidence from food surveys.

1.7.2 Recommendations for Policy

- Given the burden of illness, there needs to be a continued focus on reducing foodborne illness by *Campylobacter* and *Salmonella*.
- Although *C. perfringens* outbreak reports to national surveillance have been declining it is clear from these analyses that *C. perfringens* continues to cause a considerable illness burden and so its control is an important policy issue.
- Contamination of eggs, produce and red meat are also important policy issues given their contributions to foodborne disease.

CHAPTER 2

BACKGROUND AND OBJECTIVES

2.1 REDUCING THE BURDEN OF FOODBORNE DISEASE

Food safety has been a major purpose of the Food Standards Agency since its inception in 2000. Reducing levels of foodborne disease are the tangible outcomes of improving food safety. Despite a considerable decline in levels of foodborne disease since 2000, the cost and burden are considered to remain unacceptably high (FSA, 2011). Thus in the Foodborne Disease Strategy to 2015 (FSA, 2011) the FSA has underlined the need to ensure food is safe to eat and that consumers understand about safe food. Reducing foodborne disease should lead to decreases in morbidity, mortality and demands on healthcare services, a drop in school absence, or loss of productivity at work and increased consumer confidence in food. Progress in cutting levels of foodborne disease is being measured over the period 2010 to 2015 using laboratory-report based surveillance data for five key pathogens: *Salmonella*, *Campylobacter*, *Escherichia coli* O157, *Listeria monocytogenes* and norovirus.

2.2 INTERNATIONAL BURDEN OF ILLNESS STUDIES

2.2.1 Burden of acute gastroenteritis

Several methodological approaches have been developed for estimating the incidence of acute gastroenteritis including retrospective cross-sectional surveys (telephone surveys of self-reported illness, door-to-door or postal questionnaire surveys) or prospective, population-based cohort studies (Table 2.1 (O'Brien, 2012)).

2.2.1.1 Telephone surveys

Retrospective telephone surveys of self-reported illness have the advantage that large samples of the population can be contacted and interviews are relatively short so participation rates tend to be good. The major disadvantage of telephone surveys and other types of surveys seeking information on symptoms is that the aetiology of symptoms is not captured. They are also prone to inaccurate recall, especially if the recall period is fairly long.

Table 2.1: Population-based studies of the incidence of acute gastroenteritis in countries in the top quartile of the Human Development Index (classified as possessing "very high human development") published since 2001 (O'Brien, 2012)

Lead Author (Year published)	Study Design	Year(s) of Study	Country	Incidence Estimate expressed as rate per person per year (95% CI)
de Wit (2001)	CS	1998-9	Netherlands	0.28 (0.25 – 0.32)
Frühwirth (2001)	PS	1997-8	Austria	0.05 (NR) [children ≤ 48 months]
Herikstad (2002)	TS	1996-7	US (FoodNet sites)	1.4 (NR) [diarrhoea]
Kuusi (2003)	QS	1999-2000	Norway	1.2 (NR)
Imhoff (2004)	TS	1998-9	US (FoodNet sites)	0.72 (NR)
Majowicz (2004)	TS	2001-2	Hamilton, Ontario,	1.3 (1.1 – 1.4)
Scallan (2004)	TS	2000-1	Northern Ireland & Republic of Ireland	0.6
Thomas (2006)	TS	2002-3	British Columbia, Canada	1.3 (1.1 – 1.4)
Gauci (2007)	TS	2004-5	Malta	0.42 (0.09 – 0.77)
Jones (2007)	TS	1996-2003	US (FoodNet sites)	0.6 (NR)
Sargeant (2008)	TS	2005-6	Ontario, Canada	1.17 (0.99 – 1.35)
Karsten (2009)	PS	2004	North West Germany	0.04 (0.019 – 0.067)
Cantwell (2010)	TS	2006-7	US (FoodNet sites)	0.9* (NR) [acute diarrhoeal illness]
Ho (2010)	TS	2006-7	Hong Kong	0.91 (0.81 – 1.01)
Thomas (2010)	D-DS	2007	Gálvez, Argentina	0.49*(0.31–0.68) [High season]
Adlam (2011)	TS	2006-7	New Zealand	1.11 (1·00 – 1·23)
Hauri (2011)	TS	2004-6	Hesse, Germany	0.86 (0.72-1.03) [children ≤ 15 years]
Kubota (2011)	TS	2006-7	Miyagi Prefecture, Japan	0.44 (NR)
Thomas (2011)	D-DS	2008	Metropolitan region,	0.98* (0.89-10.7)
Ziv (2011)	TS	2005	Israel	1.49 (NR) [children < 17 years]
Baumann-Popczyk (2012)	TS	2008-9	Poland	0.9 (0.8-1.0)
Doorduyn (2012)	QS	2009-10	Netherlands	0·96 (0·81–1·11)
Müller (2012)	TS	2009	Denmark	1·4 (1·2-1·6)
Scavia (2012)	TS	2008-9	Italy	1·08 (0·90-1·14)
Tam (2012a)	CS PS	2008-9	United Kingdom	0.27 (0.25 – 0.3) 0.018 (0.014 – 0.022)
Van Cauteren (2012)	TS	2009-10	France	0·33 (0·28-0·37)

NOTE: * = 30 day recall period; CS = prospective, population-based cohort study; D-DS = cross-sectional, door-to-door survey; PS = prospective study of presentation to healthcare; QS = retrospective, cross-sectional survey; TS = retrospective, cross-sectional telephone survey; NR = not reported.

Rates of self-reported illness in the general population across Europe ranged between 1.4 cases per person per year in Denmark to 0.33 cases per person per year in France. Comparing rates across nations can be difficult. Differences in case definitions, study designs, periods of recall of symptoms and the populations studied can all hamper incidence rate comparisons. For example, one of the studies

highlighted in Table 2.1 only involved children. Nevertheless, using a standardised, symptom-based case definition enabled better comparison of rates between countries and as the use of this case definition becomes more widespread some of these difficulties in interpreting rates between studies should diminish.

As well as determining disease rates information on healthcare usage in this series of co-ordinated, cross-sectional telephone surveys of self-reported illness was used to estimate under-reporting and under-diagnosis in the national surveillance systems of the countries taking part. Overall, under-reporting and under-diagnosis were estimated to be lowest for Germany and Sweden, followed by Denmark, The Netherlands, UK, Italy and Poland. Across all countries, the incidence rate was highest for *Campylobacter* spp. and *Salmonella* spp. Adjusting incidence estimates for biases inherent in different surveillance systems provides a better basis for international comparisons than relying on reported data (Haagsma *et al*., 2013).

2.2.1.2 Prospective, population-based cohort study

Prospective studies are uncommon, perhaps because of their expense. Three such studies have been conducted in Europe – one in the Netherlands and two in the United Kingdom. The major advantage of cohort studies is the ability to obtain samples from patients with infectious intestinal disease (IID) to confirm aetiology, which is important if one of the aims is to calibrate national surveillance systems. A major drawback is that participation rates can be low and losses to follow-up may be high but there are several strategies to try to overcome both of these important limitations.

In the UK illness burden has been estimated in a population-based prospective cohort study and a prospective study of presentations to primary care (the Second Study of IID in the Community (IID2 study)). Up to 17 million people (around 1 in 4) in the UK were found to be suffering from IID in a year (annual incidence = 0.27 cases of IID per person per year). There were approximately 3 million cases of norovirus infection and 500,000 cases of campylobacteriosis. The estimated time taken off from work or school because of IID was nearly 19 million days. Around one million people presented to their primary healthcare team and the leading causes were norovirus infection (130,000 cases) and campylobacteriosis (80,000 cases) (Tam *et al*., 2012a).

As well as defining illness burden, a secondary objective of the IID2 study was to re-calibrate national surveillance systems, i.e. to estimate by how much the number of laboratory-reported cases of infection with specified pathogens needed to be multiplied to establish the actual number of infections in the community. So, for every case of IID reported to national surveillance centres in the UK, 147 cases had occurred in the community. For *Campylobacter* the ratio of disease in the community to reports to national surveillance was approximately 9 to 1, for *Salmonella* the ratio was around 5 to 1 and for norovirus the ratio was almost 300 to 1 (Tam *et al*., 2012a).

2.2.1.3 Health economics assessments

The estimated costs of diarrhoeal disease are in the region of 345 million EUR in The Netherlands, 270 million EUR in Australia and 2.8 billion EUR in Canada (Tam *et al*., 2012b).

2.2.1.4 Disability Adjusted Life Years

In the Netherlands in 2009 the burden of norovirus infection alone was estimated to be 1,622 (95% CI: 966–2650) disability-adjusted life-years (DALYS) in a population of 16.5 million, which is a large amount for what is generally held to be a very mild and self-limiting illness (Verhoef *et al*., 2013).

2.2.2 Burden of food-related illness

Having ascertained the burden of acute gastroenteritis, it is then possible to apportion illness burden by transmission route, namely foodborne transmission. Once again, several methodological approaches are available, including epidemiological and microbiological approaches, intervention studies, expert elicitation, health economics assessments and systematic reviews.

2.2.2.1 Source attribution using outbreak data

Outbreaks that have been meticulously investigated, i.e. where the evidence linking the outbreak to a food vehicle is strong, can provide useful information for subdividing diarrhoeal disease by transmission route. However, there are several limitations when interpreting the results. The first is the robustness of evidence incriminating a food vehicle in an outbreak in the first place. For example, in the

EFSA/ECDC Report published in 2013 presenting outbreak data reported in 2011, only 701 of 5,648 outbreaks were considered to provide strong evidence of a link to a food vehicle. Secondly, it has to be accepted that the distribution of food vehicles implicated in outbreaks is the same as the distribution of food vehicles responsible for sporadic cases of infection and this is a major assumption.

In the UK, in an attempt to estimate the impact of disease risks associated with eating different foods, over 1.7 million cases of UK-acquired foodborne disease per year resulted in almost 22,000 people being admitted to hospital and nearly 700 deaths (Adak *et al*, 2005). *Campylobacter* infection caused the greatest impact on the healthcare sector (nearly 161,000 primary care visits and 16,000 hospital admissions) although *Salmonella* infection resulted in the most deaths (over 200) (Adak *et al*., 2005).

In France it has been estimated that foodborne pathogens cause between 10,000 and 20,000 hospital admissions per year. *Salmonella* is the most frequent cause of hospital admissions, followed by *Campylobacter* and *Listeria* (Vaillant *et al*., 2005).

2.2.2.2 Health economics assessments

The UK's Food Standards Agency estimates the cost of foodborne illness in England and Wales annually by assessing the resource and welfare losses attributable to foodborne pathogens. The overall estimated cost of foodborne illness annually in England and Wales has remained relatively constant since 2005 at around GBP 1.5 billion. For comparison, in New Zealand and the US the costs are 216 million NZD, and 152 billion USD respectively (Tam *et al*., 2012b).

2.2.2.3 Disability Adjusted Life Years

In the Netherlands foodborne disease burden due to 14 food-related pathogens has been estimated using DALYs. This method for determining disease burden includes estimates of duration and takes into account disability weights for non-fatal cases and loss of statistical life expectancy for fatal cases. In total there were an estimated 1.8 million cases of diarrhoeal disease and 233 deaths, of which approximately 680,000 cases and 78 deaths were allocated to foodborne transmission. The total burden was 13,500 DALYs. At a population level, *Toxoplasma gondii*, thermophilic

Campylobacter spp., rotaviruses, noroviruses and *Salmonella* spp. accounted for the highest disease burden (Havelaar *et al*., 2012).

Similarly, the public health effects of illness caused by foodborne pathogens in Greece during 1996-2006 have been calculated. Around 370,000 illnesses/million people were judged to have occurred because of eating contaminated food. Nine hundred illnesses were found to be severe and 3 were fatal. The corresponding DALY estimate was 896/million population. Brucellosis, echinococcosis, salmonellosis and toxoplasmosis were the most common known causes of foodborne disease and accounted for 70% of the DALY estimate of 896 DALYs/million people (Gkogka *et al*., 2011).

2.2.2.4 Expert elicitation

Expert elicitation employs expert opinion to apportion pathogens according to foodborne transmission or transmission via other routes. An example of this is the Delphi method, which usually involves experts answering questionnaires in two or more rounds. After each round, a facilitator provides an anonymous summary of the experts' forecasts from the previous round as well as the reasons they provided for their judgments. The experts can then modify their earlier answers in response to the replies of other members of their panel. The range of the answers in each round tends to decrease so that the panel will converge towards a "correct" answer. The Delphi technique is predicated on the basis that forecasts (or decisions) from a structured panel of people is more accurate than those from unstructured groups. Panels do no need to meet in person for the method to work.

Using structured expert elicitation almost half of the total burden of diarrhoeal disease in the Netherlands was attributed to food. *T. gondii* and *Campylobacter* spp. were identified as key targets for additional intervention efforts, focussing on food and environmental pathways. Not surprisingly, perhaps, a very high proportion of toxin-producing bacteria (*Bacillus cereus, C. perfringens* and *Staphylococcus aureus*) were considered to be predominantly foodborne. By contrast multiple transmission routes were assigned to the zoonotic bacterial pathogens and the protozoan parasite *T. gondii* although the food pathway was considered to be the most important (Havelaar *et al*., 2012).

2.2.2.5 Sero-epidemiology

An alternative way to assess the incidence of foodborne pathogens is to investigate exposure to them. Pioneered in Denmark and the Netherlands, an approach to studying infection pressure has been developed using serum antibodies to *Campylobacter* and *Salmonella* as biomarkers to estimate sero-conversion rates. This shows that infections are much more common than clinical disease, probably because the majority of infections are asymptomatic. A great advantage of this method is that the assessment of incidence is independent of surveillance artefacts. The method confirms that comparing the incidence of reported incidence between countries can lead to a totally false impression, even within the European Union (Ang *et al*., 2011; Falkenhorst *et al*., 2012; Teunis *et al*., 2013). However, it should be noted that this method does not account for the proportion of infections that are food-related.

2.2.3 Food-related illness by food commodity

To pinpoint and then prioritise food safety interventions the burden of food-related illness needs to be allocated to food commodities. Again, several methodologies exist.

2.2.3.1 Interventions

The most persuasive evidence for the role of contaminated food items probably comes from studies that demonstrate the impact of interventions on human disease burden. For example, in the UK, where two population-based prospective cohort studies have taken place 15 years apart, there has been a marked fall in non-typhoidal salmonellosis in the community. The fall in incidence coincides closely with a voluntary vaccination programmes in broiler-breeder and laying flocks and suggests that these programmes have made a major contribution to improving public health, demonstrating the success of such concerted, industry-led action (O'Brien, 2013).

Natural experiments also illustrate the importance of poultry contamination as a major source of human *Campylobacter* infection. For example, in the Netherlands widespread culling of poultry that took place because of an avian influenza outbreak was followed by a decrease in *Campylobacter* infection in people, particularly in the

areas where culling had taken place (Friesema *et al*., 2012). When contamination with dioxins caused poultry to be withdrawn from the supermarket shelves in Belgium the incidence of laboratory-confirmed *Campylobacter* infection in people fell (Vellinga & Van Loock, 2002). Similarly, during the 2001 epidemic of foot and mouth disease (FMD) in livestock in England and Wales, reports of cryptosporidiosis in people fell by more than a third over the time spanning the period from the first and last cases of FMD when mass culling of livestock was taking place (Smerdon *et al*., 2003).

2.2.3.2 Microbiological Source Attribution

The main applications of source or reservoir attribution using microbial subtyping have been to *Salmonella* and *Listeria*. Serotyping and phage-typing data tend to be used for this purpose. The underlying philosophy is that controlling pathogens in the source or reservoir will avert subsequent human exposure, whatever transmission route or vehicle. Comparing results from animal and human surveillance programs provides insights about the major sources of disease in people.

In Denmark a source attribution model has been developed to quantify the contribution of major animal-food sources to human salmonellosis. This showed that domestic food products accounted for over half of all cases, with over one third of cases being attributable to table eggs. Nearly a fifth of cases were travel related and in a similar proportion no source could be pinpointed. Nearly 10% of cases were attributed to imported food products and the most important source was imported chicken. Multidrug- and quinolone-resistant infections were rare in Danish-acquired infection and were caused more frequently by imported food products and travelling abroad (Hald *et al*., 2007).

2.2.3.3 Source attribution using outbreak data

Information from well-conducted outbreak investigations can be very useful for so-called point of consumption attribution since they are gathered at the public health endpoint and can, therefore, be considered to be a direct measure of attribution at the point of exposure. One of the difficulties with using outbreak data, however, is that foods implicated in reported outbreaks are often complex foods, containing several ingredients or food items, any one of which might be the specific source of

the pathogen. The method works best for pathogens for which outbreaks are relatively common, and for which food is an important route of transmission. So, for example, it is more robust for Shiga toxin-producing *E. coli* and *Salmonella* than it is for *Campylobacter*, because *Campylobacter* outbreaks are rarely recognised. Using EU outbreak data, 58% of *Salmonella* cases that could be allocated to a source were attributed to contaminated eggs and 29% of *Campylobacter* cases that could be allocated to a source were attributed to contaminated poultry (Pires *et al*., 2010). However, for both pathogens the majority of cases could not be attributed to a source, illustrating another limitation of using outbreak data for these purposes.

In the UK, using outbreak data for point of consumption attribution showed that the most important cause of UK-acquired foodborne disease was contaminated chicken and that red meat (beef, lamb, and pork) contributed heavily to deaths (Adak *et al*., 2005). The prioritisation exercise that this type of analysis allowed showed that reducing the impact of UK-acquired foodborne disease was mainly dependent on preventing contamination of chicken.

2.2.3.4 Systematic review and meta-analysis

Several case-control studies of sporadic salmonellosis and sporadic campylobacteriosis have been published, often using different methodologies and conducted in difference settings. Systematic reviews consist of a formal process for literature review focused on a specific research question. In a systematic review of case-control studies and meta-analysis of 35 case-control studies of sporadic salmonellosis travelling abroad, underlying medical conditions, eating raw eggs, and eating in restaurants were the most important risk factors for salmonellosis in the meta-analysis (Domingues *et al*., 2012a). Similarly in a systematic review and meta-analysis of 38 case-control studies of sporadic campylobacteriosis foreign travel, undercooked chicken consumption, environmental sources, and direct contact with farm animals were all significant risk factors (Domingues *et al*., 2012b).

2.3 AIMS

The main aims of this research were to estimate the burden of UK-acquired foodborne disease in 2009, when the Second Study of Infectious Intestinal Disease in the Community (IID2 study) was undertaken, and to quantify the contribution of various food commodities to total foodborne disease burden.

2.4 OBJECTIVES

The objectives were to:-

- determine the burden of foodborne disease that is UK-acquired;
- estimate the burden of foodborne disease caused by contaminated food commodities using a point-of-consumption attribution model.

CHAPTER 3

METHODS 1 - ESTIMATING THE BURDEN OF FOODBORNE ILLNESS

This chapter describes the methods used for meeting the first objective, that is, to determine the burden of foodborne disease that is UK-acquired.

3.1 LITERATURE REVIEW

We conducted a systematic review of the literature on the proportion of disease due to the major gastrointestinal pathogens that is attributable to foodborne transmission. Although a multitude of pathogens can cause foodborne illness, of necessity our review focused specifically on eight pathogens for which disease burden in the UK is known to be high (Tam *et al*., 2012a), that are priority pathogens in terms of control, and for which foodborne transmission is a recognized and potentially important route of transmission. These eight pathogens were: *C. perfringens, Campylobacter*, *E. coli* O157, *Listeria, Salmonella*, norovirus, *Cryptosporidium* and *Giardia*. Although our review focused on these pathogens, we included data from other pathogens where available, for example, from food attribution studies that presented data on multiple pathogens. We searched the electronic databases MEDLINE, EMBASE, Web of Science for articles published between 1 January 2001 and 31 December 2011. In addition, we reviewed a database of projects funded by the Food Standards Agency (FoodBase) to identify potentially relevant studies.

3.1.1 Eligibility Criteria

We included the following studies in the review:

a) Studies that reported the proportion of human cases attributable to different risk factors (e.g. animals, food, water or other sources). These were expected to be mainly case-control studies, or case-control studies nested within cohort studies.
b) Studies that attempted to attribute human cases to different sources/food vehicles. These might use a variety of methods, including expert elicitation, outbreak data, or genetic/other typing methods.

We excluded the following:

a) Studies published in languages other than English
b) Analytical studies done as part of outbreak investigations
c) Analyses of data from surveillance of outbreaks
d) Studies involving site testing including animals (e.g. hatcheries, production facilities, abattoirs)
e) Studies in special populations (e.g. immunocompromised patients with the exception of *Cryptosporidium* and *Listeria*, long-term care facilities, infants, travellers, drug users, armed services, natural disaster or conflict zones, prisons,)
f) Studies in countries where the distribution of risk factors was unlikely to reflect that in the UK (e.g. countries outside Europe, North America, Australia, New Zealand and Japan)

3.1.2 Search Strategy

We conducted the literature search in three steps using various combinations of key search terms to maximise sensitivity and capture the greatest number of relevant articles. We used MeSH search terms for MEDLINE and EMBASE databases, and free text for Web of Science and FoodBase databases. Utilising *Campylobacter* as an example, the final search strategy employed in the systematic review is listed below.

I. Campylobacter*
II. “sporadic” OR “case-control” OR “cohort”
III. “risk factor*” OR “attribut*” OR “$etilog*”

Step 1: Search I. independently for results

Step 2: Search II. “OR” III. together for results

Step 3: Combine Step 1 “AND” Step 2 searches

A full list of search terms for individual pathogens is given in Appendix 1.

3.1.3 Study Selection and Categorisation

We maintained individual EndNote libraries for each pathogen. Initially, one trained reviewer (TL) reviewed the title and abstract of articles and categorised them as "Include" or "Exclude". A third category, "Other", was created for reports that did not fit the inclusion criteria, but could provide some useful information such as relevant references. Where there was insufficient information in the title and/or abstract to categorise an article, the full text was retrieved.

The categorisation was validated by a second reviewer (CCT). We selected a random sample of 180 abstracts from the *Campylobacter* EndNote library and compared the results of independent categorisation by the two reviewers. In addition, the list of included articles was compared with a list of case-control studies identified as part of a separate review of case-control study methods for enteric infection conducted by colleagues at the Centers for Disease Control and Prevention (Fullerton *et al*., 2012). The lists were compared to determine whether all case-control studies identified in the CDC review had also been captured by our search.

Finally, reference lists from included articles were scanned to identify further relevant articles.

3.1.4 Data Extraction

We retrieved the full text of all included studies. One trained reviewer (TL) extracted relevant data from the article using a standardised extraction form. A second independent reviewer (CCT) evaluated the completed databases and provided continued feedback. Where available, we extracted data into the following eight data fields: author, publication year, country, study design, data collection period, case definition, age groups included, and attributable proportion.

3.2 MODELLING APPROACH

We developed a model to estimate the number of cases, GP consultations and hospital admissions of indigenous foodborne disease due to 13 major enteric pathogens: *C. perfringens, Campylobacter,* Vero cytotoxin-producing *E. coli* O157 (VTEC O157), *Listeria, Salmonella* (non-typhoidal)*, Shigella, Cryptosporidium, Giardia,* adenovirus, astrovirus, norovirus, rotavirus and sapovirus. The choice of

pathogens is predicated on the priority pathogens specified above and the availability of data from the different data sources; only pathogens for which incidence data from the IID1 and/or IID2 studies and outbreak data for food attribution were available could be included in the analysis. We excluded three organisms from our analysis: *Bacillus*, *Staph. aureus* and *Yersinia*. These were not identified in the IID2 study, and data from the IID1 study indicated that these organisms are found with similar frequency among IID cases and asymptomatic controls.

The basic model is given below:

$$F_p = N \cdot c_p \cdot \pi_p$$
$$G_p = N \cdot g_p \cdot \pi_p$$
$$H_p = F_p \cdot \gamma_p$$

where F_p, G_p and H_p represent, respectively, the estimated number of indigenous foodborne disease cases, GP consultations and hospital admissions for pathogen *p* in 2009. c_p is the UK rate of infectious intestinal disease (IID) due to pathogen *p*, and g_p is the rate of IID-related GP consultations due to pathogen *p*. The two parameters, π_p and γ_p, represent the proportion of IID cases due to pathogen *p* that are attributable to foodborne transmission, and the hospitalisation rate (the proportion of cases hospitalised) for pathogen *p*. The constant, *N*, is the mid-2009 population of the UK.

In the model, we assume that the likelihood that an IID case consults a GP or is hospitalised as a result of their illness is not influenced by mode of transmission (that is, cases who acquired their infection through food are no more or less likely to consult their GP or be hospitalised than cases who acquired infection through other routes).

We used the data sources detailed below to obtain information on model parameters and their associated uncertainty. Specifically, we obtained data on pathogen-specific rates of disease from the IID1 and IID2 studies, two large longitudinal studies of acute gastroenteritis in England and the UK respectively. We used data from reported outbreaks in the UK and the published literature to inform estimates of the proportion of IID cases attributable to foodborne transmission. The IID1 and IID2

studies and outbreak data also provided information on pathogen-specific hospitalisation rates.

We incorporated information on these parameters into the model to estimate pathogen-specific numbers of cases, GP consultations and hospital admissions in 2009. We used two modelling approaches: a Monte Carlo simulation approach and a Bayesian approach. The two approaches retain the same basic model structure, but differ in how the parameters π_p and γ_p are specified. In the Monte Carlo simulation, π_p and γ_p are derived from outbreak data only, whereas in the Bayesian model, these parameters are given priors informed by, respectively, published studies and hospitalisation data from the IID1 and IID2 studies.

3.3 PATHOGEN-SPECIFIC RATES OF IID (c_p, g_p)

We obtained data on the pathogen-specific rates of IID and GP consultations from the IID2 study. For *Shigella,* there was no information from the IID2 study, but incidence data were available from the IID1 study. We inferred the overall rate of *Shigella* disease by applying the reporting ratio estimated in IID1, that is, the ratio of community to laboratory-confirmed cases reported to national surveillance, to the number of cases reported in 2009. This was then divided by the mid-2009 UK population to obtain the overall rate of IID. The rate of GP consultation was similarly estimated by applying the ratio of GP to reported incidence to the number of laboratory reports in 2009. Uncertainty in incidence estimates was accounted for by assuming a log-normal distribution for rates. For *Listeria*, no data on incidence were available from either the IID1 or IID2 studies; the number of laboratory reports for listeriosis in 2009 was used as a conservative estimate of population incidence.

3.4 PROPORTION OF CASES ATTRIBUTABLE TO FOODBORNE TRANSMISSION (π_P)

3.4.1 Outbreak data

The national surveillance centres in the four UK countries provided data on general outbreaks of IID occurring between 1 January 2001 and 31 December 2008. This timeframe was chosen so as not to overlap with the previous analysis by Adak *et al*. (2002). Data after 31 December 2008 were excluded because changes in reporting

after this time made it unclear whether data were comparable with earlier years. For each outbreak, information was available on the following: outbreak setting, number of cases affected, number of cases hospitalised, main mode(s) of transmission, pathogen identified and, for outbreaks involving contaminated foods, the implicated food vehicle (where this was identified). For the purposes of this analysis, point source or disseminated outbreaks involving contaminated food, and outbreaks involving contaminated food with subsequent person-to-person transmission, were considered to be foodborne. We excluded from this analysis outbreaks that took place in the armed services. We did not explicitly exclude outbreaks involving infected food handlers. Evidence of infected food handler involvement in the outbreak data was largely speculative and often difficult to interpret. In addition, among food attribution studies identified in the literature review, only one had a specific category for infected food handlers.

3.4.2 Estimating the proportion of cases attributable to foodborne transmission

From the outbreak data, we used the proportion of cases involved in foodborne outbreaks as an estimate of the proportion of cases attributable to foodborne transmission. For each pathogen, we obtained distributions for the proportion of cases involved in foodborne outbreaks using a two-step approach. In the first step, we used bootstrapping methods to repeatedly sample, at random and with replacement, 4,999 sets of *n* outbreaks from the data, with *n* equaling the total number of outbreaks reported for each pathogen. This bootstrapping approach was used to obtain an empirical distribution for the proportion foodborne. The number of required replications was determined from an initial analysis in which variation in the estimated standard error was plotted against the number of bootstrap replications. This was done for norovirus and *Campylobacter*, two organisms with, respectively, a high and moderate number of reported outbreaks and a low and high proportion of foodborne outbreaks. We plotted the standard error against the number of replications, and identified the minimum number of replications at which the standard error stabilised. For pathogens with a very small number of reported outbreaks, this is an unnecessarily high number of bootstrap replications, but the same number was used for consistency between pathogens.

For *Cryptosporidium* and *Giardia*, the proportion of cases involved in foodborne outbreaks gave unrealistically high estimates for the proportion of cases attributable to foodborne transmission. This is because, while the number of reported outbreaks for these two pathogens was small, foodborne outbreaks were, on average, considerably larger than non-foodborne outbreaks. For these two pathogens, we used the same bootstrapping approach outlined above, but instead used the proportion of outbreaks that were foodborne as the estimate of the proportion of cases attributable to foodborne transmission.

In the second step, we fitted smooth Beta distributions to the bootstrapped data. The Beta family of distributions is a flexible group of distributions that can capture a wide range of unimodal distributions within the range >0 to <1 using two shape parameters, *a* and *b*. For this reason, they are useful for modelling proportions. We used maximum likelihood methods to estimate *a* and *b* parameters for π_p. Bootstrap estimates with fitted Beta distributions by pathogen are shown in Appendices 2.1 and 2.2.

3.4.3 Prior distributions for π_p

Prior distributions for the π_p parameters were obtained from the literature review described in section 1.1. We divided retrieved articles into two categories: food attribution studies and other pathogen-specific studies. Food attribution studies were those that attempted to estimate the proportion of cases attributable to foodborne transmission for a variety of pathogens, either through expert elicitation or other retrospective reviews of data. Other pathogen-specific studies were primarily case-control studies of pathogen-specific risk factors, or studies using typing methods for source attribution. For these two categories of studies, we defined uniform distributions for π_p, based on the minimum and maximum values for the proportion of cases attributable to food estimated by these studies, for pathogens for which at least two published studies had been identified. Where the observed proportion from outbreak data fell outside the limits of this uniform distribution, we arbitrarily allowed the lower or upper limit of the distribution to extend by 0.1 beyond the observed value.

3.5 PATHOGEN-SPECIFIC HOSPITALISATION RATES (γ_p)

Data on hospitalisation in outbreaks were only available from England and Wales. For each reported outbreak in the England and Wales dataset, excluding outbreaks that occurred in hospitals and residential institutions, we calculated the hospitalisation rate as the number of cases hospitalised as a proportion of all cases. We calculated this by causative organism and separately for all outbreaks and for foodborne outbreaks only. There was no major difference in hospitalisation rates between all outbreaks and foodborne outbreaks and subsequent estimates of hospitalisation are based on data from all outbreaks. To account for uncertainty in these parameters, we used a two-step approach as detailed above for π_p. We obtained an empirical distribution for the hospitalisation rate by bootstrapping 4,999 replicate samples. Many reported outbreaks involve few cases and are therefore unlikely to involve hospitalised cases. The small number of larger outbreaks, on the other hand, is potentially more informative for estimating hospitalisation. For this reason, in each bootstrap replication we calculated the mean hospitalisation rate across all outbreaks for a particular pathogen, weighted by the outbreak size. We then fitted a Beta distribution to the bootstrapped data and estimated the corresponding *a* and *b* parameters using maximum likelihood. Bootstrap estimates with fitted Beta distributions by pathogen are shown in Appendix 2.3.

This method of calculating hospitalisation rates relies on a number of assumptions:

1. That hospitalisation rates in outbreaks are similar to those among sporadic cases. This might not be true if, for example:
    a. an outbreak is confined to a specific age group or vulnerable population in which hospitalisation is more likely
    b. the outbreak is associated with more severe illness (perhaps because outbreaks involving more severe illness are more likely to be investigated and reported)
    c. the outbreak investigation identifies cases which are so mild that they would not have been identified if sporadic.

2. That the numbers of cases and hospitalisations are accurately recorded in outbreak reports. It is possible, however, that more severe cases involving hospitalisation are likely to be recorded more accurately in outbreak investigations.

3.5.1 Prior distributions for γ_p

We obtained prior distributions for γ_p from the GP presentation components of the IID1 and IID2 studies. We pooled the data from these two studies to calculate the proportion of GP cases that are hospitalised and, hence, the annual number of hospital admissions for each IID pathogen. The ratio of annual estimated hospital admissions to total cases was used as an estimate of the proportion of cases hospitalised, γ_p. We used data from the GP presentation components of the IID1 and IID2 studies because of the much greater number of person-years of follow-up and greater number of cases compared with the population cohort components. This approach requires the following assumptions:

1. The proportion of GP cases that are hospitalised has not changed markedly between the IID1 and IID2 studies

2. All hospital admissions are also associated with a GP presentation, i.e. there are no hospital admissions for which a GP consultation would not also be recorded. If this is not the case, then the values of γ_p are likely to have been underestimated

We took 100,000 random samples from the distributions of the overall rate, c_p, and the proportion of GP cases hospitalised, to estimate the total cases and hospital admissions for pathogen *p*. The ratio of these two numbers was the hospital admission rate and variability around γ_p was accounted for by fitting a Beta function to the resulting distribution. The estimated parameters from this Beta distribution were used to inform the prior values for γ_p in the Bayesian approach. For pathogens for which hospitalisation information was not available from the IID1 and IID2 studies, namely VTEC O157 and *Listeria*, we used a non-informative prior defined by the distribution Beta(1,1).

3.6 PATHOGEN-SPECIFIC IID CASES, GP CONSULTATIONS AND HOSPITALISATIONS (F_p, G_p, H_p)

3.6.1 Monte Carlo approach (Model 1)

We obtained estimates of F_p, G_p and H_p using Monte Carlo simulation, each time drawing at random from each parameter distribution in the model. We carried out 100,000 simulations, discarding the first 10% and retaining the model outputs for every 10th simulation. We checked model convergence graphically by plotting parameter values over time to verify adequate mixing, plotting autocorrelograms and comparing density plots for outcome variables by tertile of the simulation chain. The model and associated parameter distributions are described below:

$$F_p = N \cdot c_p \cdot \pi_p$$
$$G_p = N \cdot g_p \cdot \pi_p$$
$$H_p = F_p \cdot \gamma_p$$

$$\log(c_p) \sim N(\mu_{cp}, \sigma_{cp})$$

$$\log(g_p) \sim N(\mu_{gp}, \sigma_{gp})$$

$$\pi_p \sim \text{Beta}(a_{\pi p}, b_{\pi p})$$

$$\gamma_p \sim \text{Beta}(a_{\gamma p}, b_{\gamma p})$$

From the ensuing distributions of F_p, G_p and H_p, we used the median and central 95% of the distributions as the point estimates and 95% credible intervals respectively. A full description of model parameters is given in Appendix 3.1 and a worked example using *Campylobacter* is shown in Appendix 4.

3.6.2 Bayesian approach (Models 2 and 3)

In the Bayesian approach, we included parameters for the prior distributions of π_p and γ_p. These priors were used, together with the outbreak data to obtain posterior distributions for these parameters, which were then used in the model as described overleaf:

$$F_p = N \cdot c_p \cdot \pi_p$$
$$G_p = N \cdot g_p \cdot \pi_p$$
$$H_p = F_p \cdot \gamma_p$$

$$\log(c_p) \sim N(\mu_{cp}, \sigma_{cp})$$

$$\log(g_p) \sim N(\mu_{gp}, \sigma_{gp})$$

$$f_p \sim \text{Binomial}(\pi_p, o_p)$$

$$\pi_p \sim \text{uniform}(u_{\pi p}, v_{\pi p})$$

$$h_p \sim \text{Binomial}(\gamma_p, m_p)$$

$$\gamma_p \sim \text{Beta}(a_{\gamma p}, b_{\gamma p})$$

For each pathogen, *p*, the parameters f_p and o_p represent the number of cases involved in foodborne and all outbreaks respectively (or the number of foodborne and all outbreaks in the case of *Cryptosporidium* and *Giardia*, as described in Section 3.4.2 above). Similarly, h_p and m_p represent the pathogen-specific number of hospitalisations and GP consultations as observed in the IID1 and IID2 studies. The prior values for parameters π_p and γ_p are defined by uniform and Beta distributions respectively as described in Sections 3.4.3 and above. In Model 2, the uniform distributions for π_p were informed by data from published multi-pathogen food attribution studies (Table 5.1). We used a further model, Model 3, with the same structure as Model 2, but with parameters for the prior distribution of π_p being derived from pathogen-specific studies identified in the literature review. A full description of parameters for models 2 and 3 is given in Appendices 3.2 and 3.3.

For each model, we carried out 100,000 simulations to obtain posterior distributions for F_p, G_p and H_p, discarding the first 10% and retaining the model outputs for every 10th simulation. We checked for model convergence as described for the Monte Carlo approach above.

We conducted the analyses using Stata 12, WinBUGS and Microsoft Excel software. We used the `winbugsfromstata` module in Stata to carry out the simulations (Thompson *et al*., 2006).

CHAPTER 4

METHODS 2 - ESTIMATING THE BURDEN OF FOODBORNE ILLNESS BY FOOD COMMODITY

This chapter describes the methods used for meeting the second objective, which is to estimate the burden of foodborne disease caused by contaminated food commodities using a point-of-consumption attribution model

We extended the food attribution model to estimate, by pathogen, the number of cases, GP consultations and hospital admissions attributable to different food commodities. Ten pathogens were included in this analysis: *C. perfringens*, *Campylobacter*, *E. coli* O157, *Listeria*, *Salmonella*, *Shigella*, *Cryptosporidium*, *Giardia*, norovirus and rotavirus. Adenovirus, astrovirus and sapovirus were excluded because there was insufficient data on commodity-specific food attribution to allow estimation.

The general model is based on Model 2 in the food attribution analysis and is described below:

$$[F]_p = N \cdot c_p \cdot \pi_p \cdot [pc]_p$$
$$[G]_p = N \cdot g_p \cdot \pi_p \cdot [pc]_p$$
$$[H]_p = F_p \cdot \gamma_p \cdot [pc]_p$$

Here, $[F]_p$ is a vector of 12 quantities representing the estimated cases attributable to each food commodity. The vectors $[G]_p$ and $[H]_p$ are interpreted analogously for the number of GP consultations and hospital admissions respectively. The quantity $[pc]_p$ represents a vector of probabilities that a case of IID due to pathogen *p* acquired infection through each of the 12 food commodities. This vector of values is assumed to be independent of disease severity, e.g. a case infected through consumption of poultry products is not more likely to be hospitalised than a case infected through consumption of other foods. Information on the proportion of cases attributable to different food commodities was obtained from an analysis of UK outbreak surveillance data and published food attribution studies. These data sources are described in the next two sections. The remaining parameters in the model are identical to those described in Model 2 (see section 3.6.2).

4.1 OUTBREAK DATA

The outbreak dataset is described in Section 3.4.1. For this analysis, we used the subset of 446 outbreaks that involved foodborne transmission. For each such outbreak, we obtained information, where available, on the causative organism, the setting and the food vehicle(s) implicated. To classify implicated food vehicles, we used a scheme modified from that recommended by Painter *et al*. (2009). Three independent reviewers were asked to review records of individual outbreaks and classify them into one of 19 possible food commodity groups (see Painter *et al*., 2009) for the full list of food commodities. To aid in classification, reviewers were provided information, where available, on the outbreak setting, causative organism, implicated food vehicle(s) and type of evidence to support the implicated vehicle. For more than half of all outbreaks, evidence to support the implicated vehicle was descriptive or circumstantial; 27% of outbreaks had microbiological evidence in which the same organism was identified in patients and in a sample of the implicated food, 15% had epidemiological evidence from a case-control or retrospective cohort study, and 3% had both microbiological and epidemiological evidence pinpointing the implicated food. Reviewers were asked to consider this information when classifying outbreaks, but outbreaks were not excluded on the basis of the type of evidence available. The purpose of using three reviewers was two-fold: to minimise the number of outbreaks in which no food commodity was specified, and to capture uncertainty in the classification of food vehicles, particularly in instances where the implicated food could result in ambiguity, e.g. meat pies, or in the case of complex foods, for which several ingredients could potentially have been responsible for transmission. The 19 food commodities were consolidated into 12 categories: seafood, dairy, eggs, unspecified red meat, game, beef and lamb, pork, poultry, grains and beans, oils and sugars, produce, complex and other foods (Table 4.1). In particular, subcategories of fruits and vegetables were grouped into a single ‘produce’ category, crustaceans and molluscs were consolidated into a single ‘seafood’ group, and beef and lamb were combined into one category. This consolidation was necessary because food attribution studies identified in the literature review (described below) did not always classify food commodities beyond this level of detail.

4.2 LITERATURE REVIEW

We used the food attribution studies identified in literature review, described in Section 3.1. For each study, we extracted the estimates of the proportion of cases of foodborne illness attributable to specific food commodities by pathogen to construct a series of vectors. For each pathogen, the vector comprised a series of values corresponding to the estimated proportion of foodborne illness cases attributable to each of 12 food commodity groups. In some instances, a study gave more than one set of estimates, in which case separate sets of vectors were constructed. For example, a study by food commodity attribution based on outbreak data by Pires *et al*. (2010) gave separate estimates from analyses of foodborne outbreaks, and cases involved in foodborne outbreaks. A similar study by Greig & Ravel (2009) gave separate estimates for *Salmonella* Enteritidis and other *Salmonella* types. Some studies included categories for beverages, while one study, by Havelaar *et al*. (2008) additionally included a category for infected food handlers. These categories were excluded from our analysis, and the proportions re-scaled so as to sum to unity (i.e. one).

4.3 CLASSIFICATION OF FOOD COMMODITIES

Twelve food commodity groups were used: seafood, dairy, eggs, unspecified red meat, game, beef and lamb, pork, poultry, grains and beans, oils and sugars, produce, complex and other foods (Table 4.1). This scheme was a simplified version of that recommended by Painter *et al*. (2009) and was based on the need to consolidate the slightly different food classifications used in the different studies, as well as information available from outbreak data. In particular, the category “unspecified red meat” includes foods such as processed meats that were not ascribed by the original studies to a specific animal source and the “complex food” category comprised foods consisting of two or more types of commodities. In addition, several studies in the literature review reported estimates for beef and lamb together, so these were grouped into a single category.

Table 4.1: Food types included in food commodities

Food Commodity	*Specific foods*
Seafood	Finfish, crustacean shellfish, molluscan shellfish, other seafood, seafood dishes, mixed/unspecified
Dairy	Milk, milk products, dairy, cheese, butter, cream, ice cream, dairy substitute, other dairy products
Eggs	Eggs, egg dishes, egg products
Unspecified red meat	Red meat products for which animal source could not be defined, including tongue, luncheon meats, other meats, other meat dishes, mixed/unspecified
Game	Game, game bird
Beef/Lamb	Beef, ground beef, other beef, beef dishes, whole muscle beef, veal, including. processed and non-processed beef (sausages, steak tartare, hamburgers, etc.), lamb and mutton
Pork	Pork, bacon, ham, other pork, pork dishes, processed and non-processed pork products (sausages, luncheon meats, etc.)
Poultry	Chicken, turkey, duck, goose, dove, ostrich, other poultry, poultry dishes, mixed/unspecified, processed and non-processed poultry products (chicken wings, marinated chicken, confits, etc.)
Grains and beans	Rice, breads, bakery products, cooked and dry cereals, grains and beans
Oils and sugars	Oils and sugar
Produce	Salad vegetables, cooked vegetables, fruit, nuts, seeds (including sprouting seeds), produce dishes, almonds, halva, nuts/dry fruits, peanut butter, peanuts, sesame seeds, tahini
Complex and other	Consisting of ingredients from two or more categories and all other foods that are not listed above, including sandwiches, pre-packed mixed vegetable salads, rice/beans/stuffing/pasta dishes, sauces, other multi-ingredient foods, home canned goods, confectionery, spices, desserts

Source: Modified from Painter *et al* (2012)

4.4 BOOTSTRAPPING OF OUTBREAK DATA

To obtain estimates of the relative frequency of different food commodities in foodborne outbreaks, we first combined the three datasets classified by the independent reviewers. We then obtained, for each pathogen, 10,000 bootstrap estimates of the frequency with which each food commodity was observed. For each replicate in the simulation, we sampled o_p outbreaks with replacement, with o_p equalling the total number of outbreaks recorded for pathogen *p*. For each outbreak sampled, however, the record selected could come from any one of the three datasets from independent reviewers, so as to capture variations in classification. For each bootstrap replication, the number of cases involved in outbreaks attributed to the different food commodities was determined. The frequencies across the 10,000 bootstrap replicates were summarised using the arithmetic mean. The arithmetic means did not markedly differ from the medians and have the advantage that under conditions of random sampling, the sum across food commodities should equal the total cases observed. These summarised frequencies were then used to create pathogen-specific data vectors to use in the models.

4.5 MODELLING APPROACH

We used two sets of models to estimate the number of cases, GP consultations and hospital admissions attributable to specific food commodities. The two types of model have the same general structure, but differ in how the food commodity-specific attributable proportions a parameterised.

4.6 BAYESIAN APPROACH COMBINING OUTBREAK AND PUBLISHED DATA

In this set of models, we used a Bayesian approach combining outbreak data with prior information from published food attribution studies to estimate the posterior distribution of $[pc]_p$. This approach was used for *C. perfringens*, *Campylobacter*, *E. coli* O157, *Salmonella* and norovirus, for which sufficient data from outbreaks were available. Values for the data vector were assumed to come from a multinomial distribution and were obtained from bootstrapping of outbreak data as described above. For a given pathogen, the vector values correspond to the expected number of cases involved in outbreaks attributable to each food commodity.

The prior values comprised a vector of Dirichlet parameters. The values for the Dirichlet prior are positive real numbers. The relative size of the vector values indicates how much more common one element is believed to be relative to another; their order of magnitude is an indication of the degree of confidence in the relative values. Vector values were arbitrarily scaled such that the elements summed to 100. If a particular food commodity was reported in the original studies not to contribute to transmission of a given pathogen, it was given an arbitrarily small value of 0.1. A separate vector of prior values was used for each food attribution study, and the model re-run for each set of vector values in a sensitivity analysis, to see what influence food attribution estimates from different studies had on the results. In addition, we ran the model with a vague prior, in which the same value was given to each food commodity. The food commodity parameters used for each pathogen are given in Appendix 5.

4.7 APPROACH USING ONLY INFORMATION FROM PUBLISHED ATTRIBUTION STUDIES

For some organisms, no foodborne outbreaks were reported, or information on the implicated foods was not available. For this reason, we ran a separate set of models in which the estimates of $[pc]_p$ were derived only from the Dirichlet priors described in the previous section. This approach was used for all ten pathogens.

We fitted the models in WinBUGS software using the `winbugsfromstata` module for Stata 12.0. We ran the models 100,000 times, discarding the first 10,000 simulation runs and retaining the model results for every 10th simulation. Model fit was assessed visually as described in Section 3.6.1.

4.8 SUMMARISING POSTERIOR DISTRIBUTIONS

Food attribution data are complicated by the fact that estimates for individual food commodities are dependent i.e., if for a given pathogen poultry accounts for a larger fraction of cases, the other food commodities must account for a lower fraction. The simultaneous estimation of several dependent quantities comes with a number of difficulties in interpretation. The first is that, while in each simulation the sum of estimated cases across food commodities will equal the total number of estimated foodborne cases, there is no easy way to summarise estimates across simulations.

Indeed, the sum of mean or median values for all food commodities across 100,000 simulations will almost certainly not equal the total foodborne cases. Uncertainty around estimates for individual food commodities should also be interpreted with caution. Thus, although the central 95% of the posterior distribution captures the range of values within which 95% of simulations estimate the value of the parameter of interest to be, this is only true under certain conditions. For example, it is not feasible for all food commodities to simultaneously have very low or very high estimated values. There is, however, no straightforward way to summarise this information across 12 dimensions. For this reason, we have opted to present the median and central 95% of the posterior distributions, since these still have a valid, if limited, interpretation.

A further problem is that there is no established way to combine summaries across studies in a manner analogous to a weighted meta-analysis. Because different attribution studies have used different methodologies, including analysis of outbreak data and expert elicitation, and because there is *a priori* no way to weight the amount or quality of information from different studies, we have opted to instead combine the posterior distributions derived from models using different priors. The rationale for this is that it gives a clear idea of the degree of variability across all studies from which food attribution data were available, although in some instances this gives rise to very complex distributions.

4.9 RATES BY FOOD COMMODITY

To calculate rates of foodborne illness by food commodity, we used data from the National Diet and Nutrition Survey (NDNS). The NDNS is an ongoing cross-sectional survey of persons aged less than 65 years conducted on behalf of the Food Standards Agency and the Department of Health (Department of Health, 2011). We estimated the total annual consumption of each food commodity in the UK from daily average dietary intake values in the NDNS report. We estimated rates by dividing the annual number of cases, GP consultations or hospital admissions attributed to each food commodity by the total annual consumption of that food commodity. Rates are expressed as number of cases per 1,000 persons per year, based on the average per capita annual consumption of each food commodity, together with the corresponding 95% credible intervals. As the NDNS was only conducted among

people aged under 65 years, rates presented correspond to the population of the UK below this age only. In addition, we calculated rate ratios comparing the annual incidence associated with each food commodity relative to category “grains and beans”, with corresponding 95% CrIs.

CHAPTER 5

RESULTS 1 – ESTIMATING THE BURDEN OF FOODBORNE ILLNESS

5.1 LITERATURE REVIEW

5.1.1 Summary of search results

Overall, 24,439 references were identified in the literature search. After removing duplicates, 14,620 references remained, of which 189 were identified as potentially relevant. In total, eight multi-pathogen studies and 27 pathogen-specific studies were included in this report. A detailed breakdown of the search results is given in Appendix 1.

5.1.2 Search Validation

A low level of discordance was found between reviewers (0.026) in the categorisation of "Other" *Campylobacter* articles. All case-control studies identified in the CDC review were captured by our search.

5.1.3 Summary of data from food attribution studies

Figure 5.1 gives a graphical representation of the data abstracted from the eight multi-pathogen food attribution studies identified. Along each of the spokes of the diagram the estimates of the proportion of a particular pathogen that is foodborne are displayed. This can range from 100% in the middle of the plot to 0% at the edge. For a given pathogen, each marker represents an estimate of the proportion of cases attributable to foodborne transmission from one of the identified studies. For *C. perfringens* the estimates from the individual studies are placed close together reflecting consistency amongst the studies in the proportion of *C. perfringens* infections attributed to foodborne transmission. However, for other pathogens like *Campylobacter, Salmonella* and norovirus the diversity of estimates of the proportion of foodborne transmission is evident. The individual studies are summarised in Tables 5.1 and 5.2.

Figure 5.1: Summary of estimates for the proportion of cases attributable to foodborne transmission by pathogen, from the eight multi-pathogen food attribution studies identified in the literature review

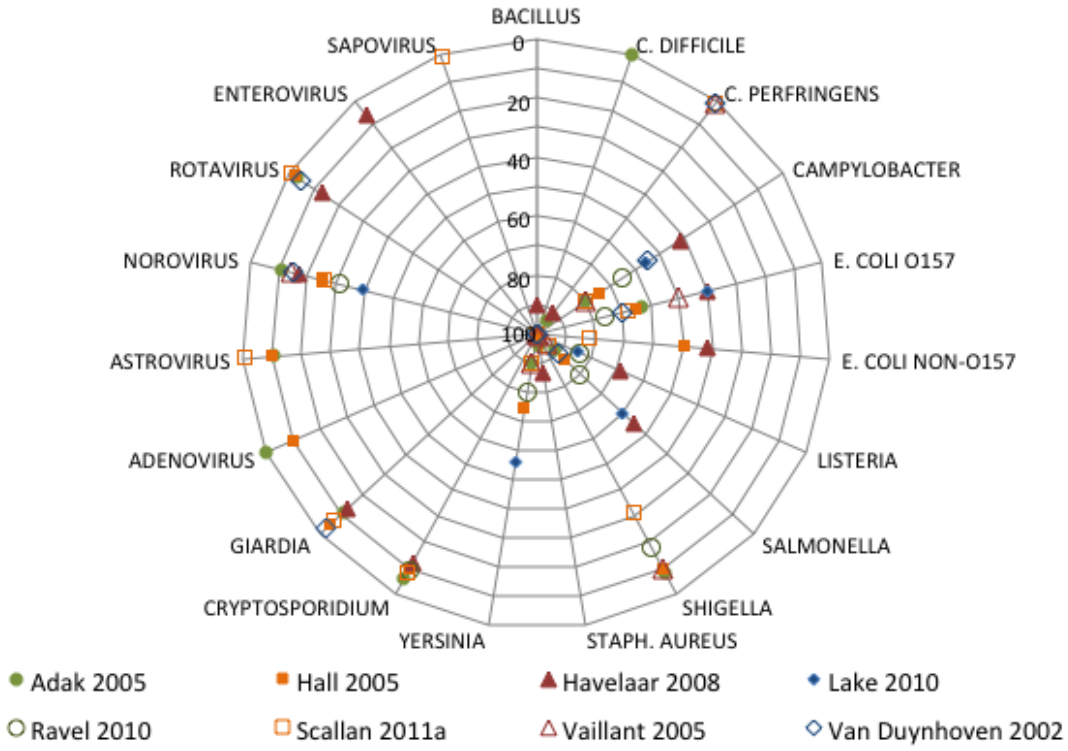


Table 5.1: Proportion of IID attributed to food, summary of results from included multi-pathogen food attribution studies

	Studies (N=8)								
Author	**Adak**	**Hall**	**Havelaar**	**Lake**	**Ravel**	**Scallan**	**Vaillant**	**Van Duynhoven**	
Year	**2002**	**2005**	**2008**	**2010**	**2010**	**2011a**	**2005**	**2002**	
Country	**UK**	**AUS**	**NL**	**NZ**	**CAD**	**USA**	**France**	**NL**	
Period	**1992-2000**	**2000**	**2006**	**2000s**	**2008**	**2000s**	**1990s**	**1990s**	
Data sources*	**O**	**E**	**E**	**E**	**E**	**CC/V**	**V**	**E/CC**	Studies
Travel cases	**Excluded**	**Excluded**	**Included**	**Included**	**Excluded**	**Excluded**	**Included**	**Included**	Identified
Bacillus	1.0	1.0	0.900			1.0	1.0	1.0	6
C. difficile	0.0								1
C. perfringens	0.944	1.0	0.910			1.0	1.0	1.0	6
Campylobacter	0.797	0.750	0.420	0.562	0.680	0.800	0.800	0.550	8
E. coli O157	0.630	0.650	0.400	0.395	0.760	0.680	0.500	0.700	8
E. coli non-O157		0.500	0.420			0.820			3
Listeria	0.990		0.690	0.850	0.840	1.0	0.990		6
Salmonella	0.916	0.870	0.550	0.596	0.800	0.940	0.950	0.900	8
Shigella	0.082	0.100			0.180	0.310	0.100		5
Staph. aureus	0.960	1.0	0.870			1.0	1.0	1.0	6
Yersinia	0.900	0.750		0.562	0.800	0.900	0.900		6
Cryptosporidium	0.056	0.100	0.120		0.090	0.080			5
Giardia	0.100	0.050	0.130			0.070		0.300	5
Adenovirus	0.0	0.100							2
Astrovirus	0.107	0.100				0.005			3
Enterovirus			0.060						1
Rotavirus	0.025	0.020	0.130			0.005		0.050	5
Norovirus	0.107	0.250	0.170	0.392	0.310	0.260	0.140	0.150	8
Sapovirus						0.005			1

* O: outbreak data; E: expert elicitation study; CC: case-control study; V: various data sources

Table 5.2: Proportion of IID attributed to food, summary of results from included pathogen-specific risk factor or modelling studies

Studies (N=27)					Bacteria					Protozoa		Virus
Author	**Year**	**Country**	**Period**	**Data**	***C. perfringens***	***Campylobacter***	***E. coli* O157**	***Listeria***	***Salmonella***	***Cryptosporidium***	***Giardia***	**Norovirus**
Carrique-Mas	2005	Sweden	2001-2002	CC		0.632[a]						
Danis	2009	Ireland	2003-2004	CC		1.0						
Denno	2009	USA	2003-2005	CC		1.0[a]	0.517		0.319			
Doorduyn	2006	NL	2002-2003	CC					0.090			
Doorduyn	2010	NL	2002-2003	CC		0.660[b]						
Doorduyn	2010	NL	2002-2004	CC		0.630[c]						
Effler	2001	USA	1998	CC		0.180[b]						
Evans	2003	UK	2001	CC		0.560						
Fajo-Pascual	2010	Spain	2005-2006	CC		0.606						
Friedman	2004	USA	1998-1999	CC		0.565						
Hald	2004	Denmark	1999	M					0.714			
Kassenborg	2004	USA	1996-1997	CC			0.350					
Kimura	2004	USA	1996-1997	CC					0.280			
Little	2010	UK	2004-2007	M				0.977				
Michaud	2004	Canada	2000-2001	CC		0.460						
Neimann	2003	Denmark	1996-1997	CC		0.396						
Phillips	2011	England	1993-1996	CC								0.020
Rodrigues	2001	England	1993-1996	CC		0.110[b]						
Sheppard	2009	Scotland	2005-2006	M		0.760						
Stafford	2008	Australia	2001-2002	CC		0.314						
Stuart	2003	England	1998-1999	CC							0.400	
Tam	2009	England	2005-2006	CC		0.410						
Toyofuku	2011	Japan	1998-2007	M					0.500			
Unicomb	2008	Australia	1999-2001	CC		0.229						
Varma	2007	USA	2000-2003	CC				0.180				
Voetsch	2007	USA	1999–2000	CC			0.085					
Werber	2007	Germany	2001-2003	CC			0.439					
Wingstrand	2006	Denmark	2000-2001	CC		0.238						
Studies identified					0	16	4	2	5	0	1	1

CC: Case-control study; M: Modelling study; [a]Children only; [b] *C. jejuni*; [c] *C. coli*

5.2 PROPORTION OF CASES ATTRIBUTABLE TO FOODBORNE TRANSMISSION

Table 5.3 presents a summary of the outbreak data used for food attribution by pathogen. Both the number of outbreaks and the number of cases involved in outbreaks are given, together with the number and percentage of these that resulted from foodborne transmission. There was great variability in the amount of data available between pathogens. For norovirus, the data comprised 2,228 outbreaks and 58,855 cases, whereas for *Listeria*, *Shigella*, *Giardia* and astrovirus, there were fewer than 20 outbreaks available for analysis. No outbreaks were reported for adenovirus and sapovirus. For adenovirus, we assumed that the proportion of cases attributable to foodborne transmission was the same as for rotavirus, and used the relevant parameters derived from analysis of rotavirus data for the food attribution calculations. For sapovirus, we used the same parameters as for norovirus.

Table 5.3: Summary of outbreak data for food attribution by pathogen

	FOODBORNE OUTBREAKS			CASES IN FOODBORNE OUTBREAKS		
Organism	Foodborne	All outbreaks	%[1]	Cases	All cases	%[2]
Bacteria						
C. perfringens	45	60	75.0%	1691	1964	86.1%
Campylobacter	31	44	70.5%	373	761	49.0%
E. coli O157	25	86	29.1%	564	1041	54.2%
Listeria	2	2	100.0%	6	6	100.0%
Salmonella	266	308	86.4%	7128	7892	90.3%
Shigella	4	11	36.4%	65	310	21.0%
Protozoa						
Cryptosporidium	4	65	6.2%	415	1375	30.2%
Giardia	1	7	14.3%	106	159	66.7%
Viruses						
Adenovirus[3]	--	--	--	--	--	--
Astrovirus	0	18	0.0%	0	283	0.0%
Norovirus	61	2228	2.7%	1500	58,855	2.5%
Rotavirus	1	136	0.7%	30	2338	1.3%
Sapovirus[3]	--	--	--	--	--	--

[1]Percentage of outbreaks involving foodborne transmission;

[2]Percentage of cases in reported outbreaks that occurred in foodborne outbreaks

[3]No outbreaks reported

Table 5.4 summarises the data used to estimate hospitalisation by pathogen from both reported outbreaks and the IID1 and IID2 studies. Hospitalisation rates could not be estimated from *Listeria* outbreaks, as all of these outbreaks occurred among patients who were already hospitalised. For *Shigella*, *Giardia* and astrovirus, there were fewer than 10 outbreaks with data on hospitalisation. Hospitalisation rates based on outbreak data were less than 4% for most pathogens, with the exception of *E. coli* O157 (22.5%) and *Salmonella* (7.6%). As above, no outbreak data were available for adenovirus and sapovirus, and the relevant hospitalisation parameters for rotavirus and norovirus respectively were used. Hospitalisation data from the GP presentation components of the IID1 and IID2 studies are also shown. It should be noted that these are not directly comparable with hospitalisation data from outbreaks, as they represent hospitalisation among patients presenting to the GP. No hospitalisations were recorded in the IID1 and IID2 studies for *E. coli* O157, *Shigella*, *Cryptosporidium*, adenovirus and sapovirus. For these pathogens, we determined an upper limit for the hospitalisation rate by assuming that the next case observed would have been hospitalised, e.g. for *Shigella*, with 11 cases and 0 hospitalisations, we assumed a mean hospitalisation rate of 1/12=8.3%.

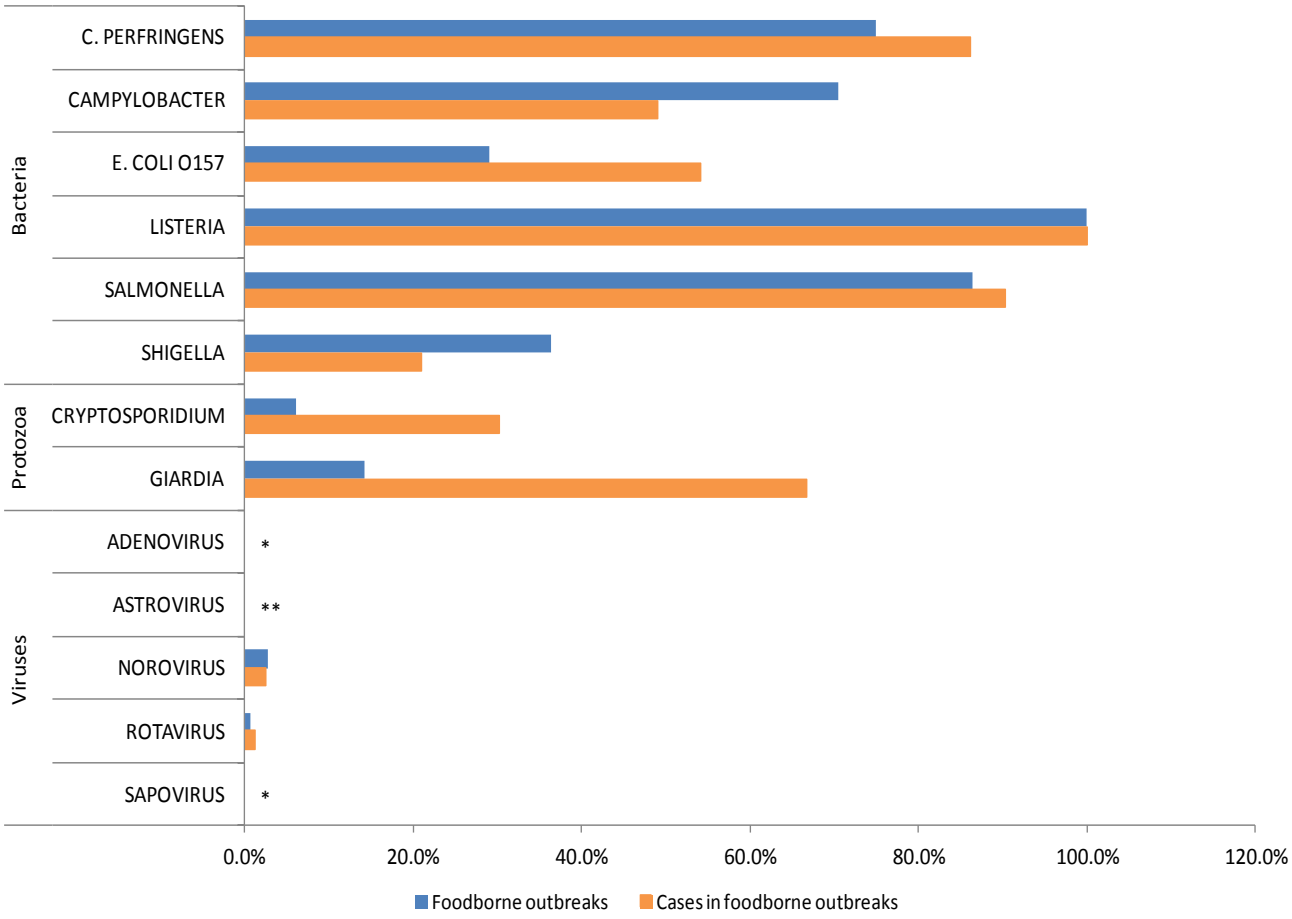
Table 5.4: Summary of hospitalisation data by pathogen

	HOSPITALISATION IN OUTBREAKS					HOSPITALISATION IN IID1 AND IID2 STUDIES			
Organism	Hospitalised	Affected	%	Outbreaks with data	Source	Hospitalised	Affected	%[2]	Source
Bacteria									
C. perfringens	2	1,120	0.2%	21	Outbreak surveillance	2	78	2.6%	IID1 & IID2[1]
Campylobacter	2	424	0.5%	29	Outbreak surveillance	5	441	1.1%	IID1 & IID2[1]
E. coli O157	197	877	22.5%	70	Outbreak surveillance	0	2	33.3%	IID1 & IID2[1]
Listeria	--	--	--	--	Outbreak surveillance	--	--	--	No cases identified
Salmonella	419	5,527	7.6%	217	Outbreak surveillance	4	114	3.5%	IID1 & IID2[1]
Shigella	4	153	2.6%	8	Outbreak surveillance	0	11	8.3%	IID1[1]
Protozoa									
Cryptosporidium	31	836	3.7%	46	Outbreak surveillance	0	50	2.0%	IID1 & IID2[1]
Giardia	1	137	0.7%	5	Outbreak surveillance	1	34	2.9%	IID1 & IID2[1]
Viruses									
Adenovirus	--	--	--	--	No outbreaks reported	0	79	1.3%	IID1 & IID2[1]
Astrovirus	2	88	2.3%	7	Outbreak surveillance	1	67	1.5%	IID1 & IID2[1]
Norovirus	80	12,333	0.6%	342	Outbreak surveillance	2	201	1.0%	IID1 & IID2[1]
Sapovirus	--	--	--	--	No outbreaks reported	0	77	1.3%	IID2[1]
Rotavirus	20	1,211	1.7%	59	Outbreak surveillance	1	64	1.6%	IID2[1]

[1]Data are from the GP presentation component of the IID1 and/or IID2 studies; [2]where no hospitalisations were observed, we determined an upper limit for the hospitalisation rate by assuming that the next case observed would have been hospitalised, e.g. for *Shigella*, with 11 cases and 0 hospitalisations, we assumed a mean hospitalisation rate of 1/12=8.3%

Figure 5.2 shows estimates of the percentage of outbreaks reported in the UK between 2001 and 2008 that involved foodborne transmission (blue bars), and of the percentage of cases in reported outbreaks that were involved in foodborne outbreaks (orange bars). For astrovirus and sapovirus, no outbreaks involving foodborne transmission were identified, whereas for *Listeria* all reported outbreaks involved foodborne transmission. For most pathogens, estimates based on outbreaks and cases involved in outbreaks were similar. For *Cryptosporidium* and *Giardia*, estimates based on outbreak cases were notably higher than estimates based on the percentage of outbreaks that were foodborne. Some differences between the two estimates were also seen for *Campylobacter*, *E. coli* O157 and *Shigella*.

Figure 5.2: Pathogen-specific estimates of proportion foodborne from reported outbreaks, UK 2001-2008

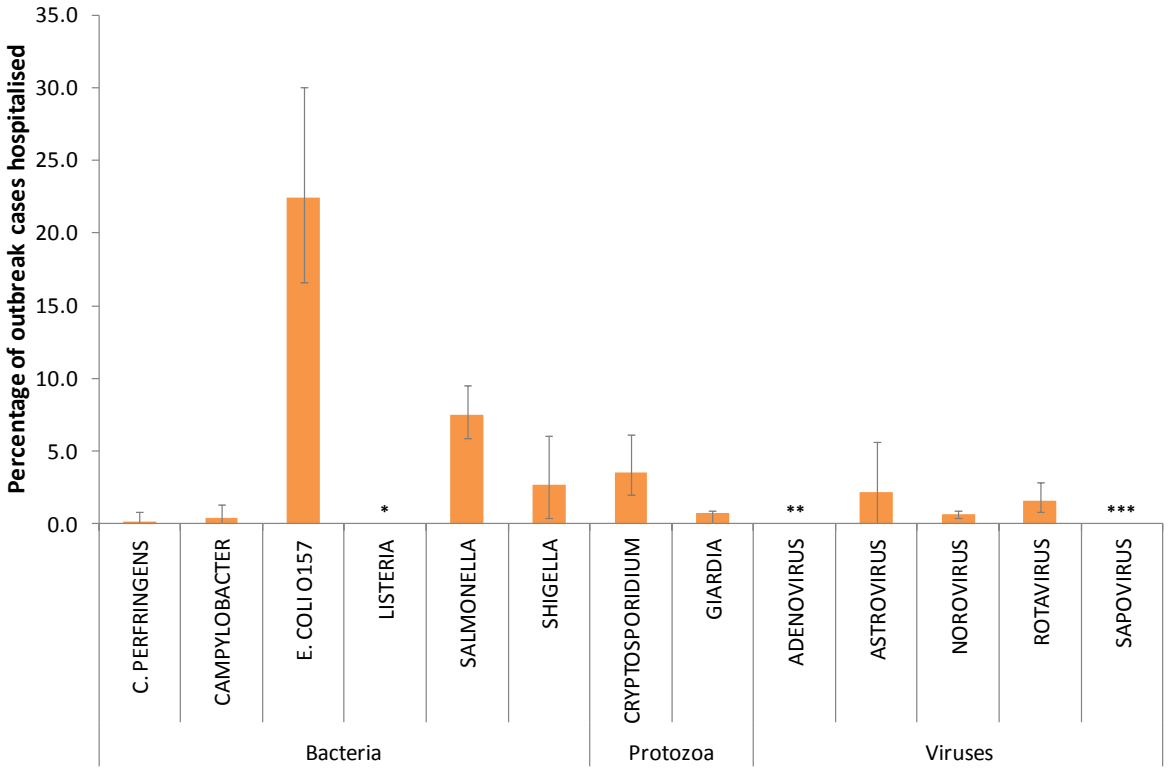


NOTES: Blue bars: Percentage of reported cases involving foodborne transmission; Orange bars: Percentage of outbreak-related cases involved in foodborne outbreaks.

5.3 PROPORTION OF CASES HOSPITALISED

Figure 5.3 shows the estimated hospitalisation rates in reported outbreaks by pathogen. Reported hospitalisation rates were particularly high for *E. coli* O157 (22%). By contrast, hospitalisation rates for *C. perfringens*, *Campylobacter*, *Giardia*, norovirus and rotavirus were all less than 2%.

Figure 5.3: Percentage of cases hospitalised in reported outbreaks, England and Wales 2001-2008.



NOTES: Estimates based on 4,999 bootstrap replications and weighted by outbreak size. Outbreaks in hospitals and residential institutions are excluded; Error bars are 95% confidence intervals.

* All Listeria outbreaks recognised through patients already in hospital
** No adenovirus outbreaks reported
*** No sapovirus outbreaks involving hospitalisation reported

5.4 CASES, GP CONSULTATIONS AND HOSPITAL ADMISSIONS ATTRIBUTABLE TO FOODBORNE TRANSMISSION (MODEL 1)

Table 5.5 presents estimates of food-related cases, GP consultations and hospital admissions in 2009 from 100,000 Monte Carlo simulations. *Campylobacter* was the

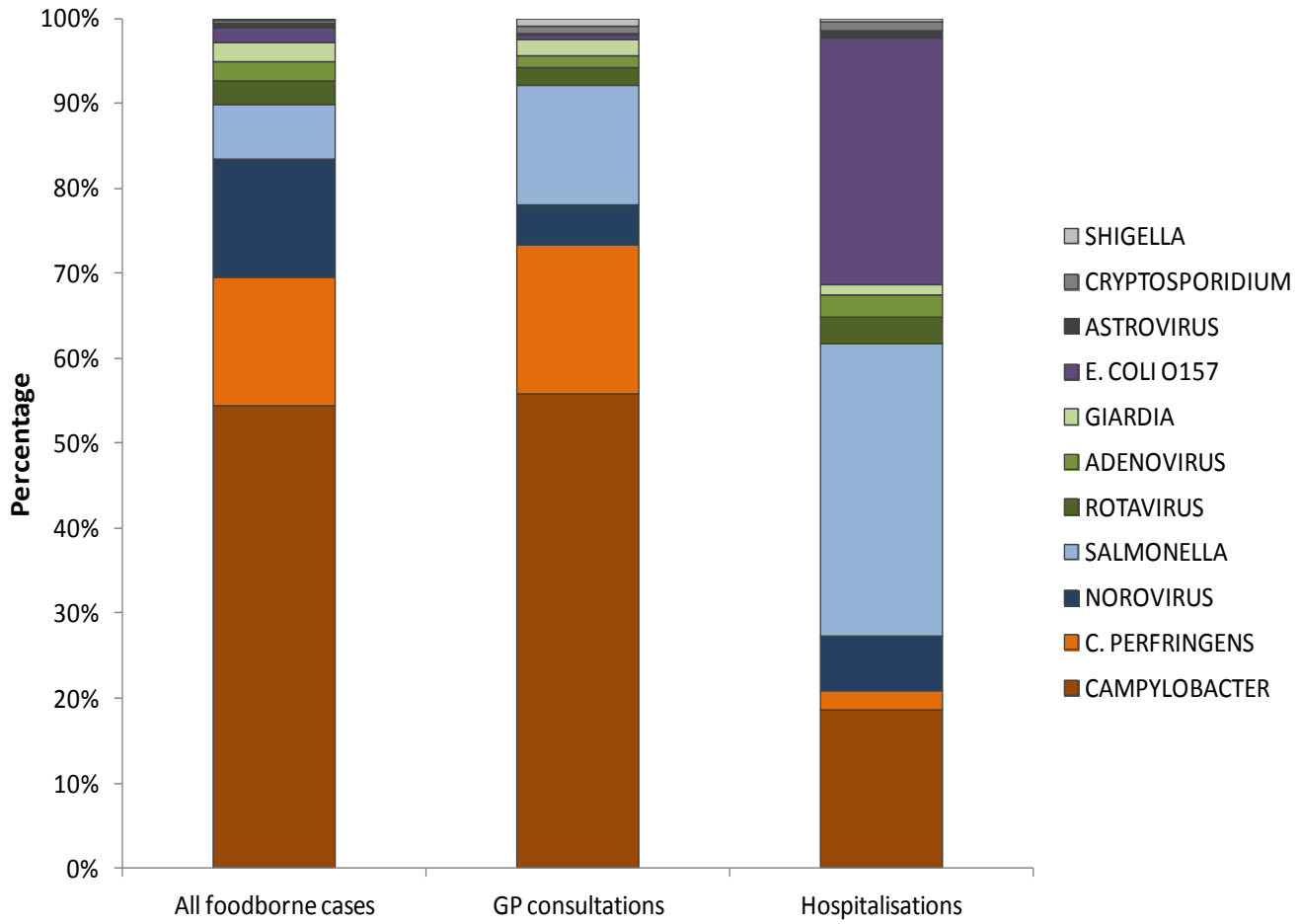
most common foodborne pathogen, accounting for around 286,000 food-related cases and nearly 40,000 GP consultations, but ranked only fourth as a cause of food-related hospital admissions. Similarly, other pathogens such as *C. perfringens* and a number of the viruses, while contributing large numbers of cases and GP consultations, were responsible for a modest number of food-related hospital admissions. It should be noted, however, that there was a large degree of uncertainty around all these estimates, as demonstrated by the wide 95% CrIs.

In Figure 5.4 organisms are arranged in a stacked bar chart according to their proportionate contribution to foodborne illness. The figure allows for comparison of the contribution of each organism to these three components of foodborne illness. As mentioned above, *Campylobacter*, despite contributing the most food-related cases and GP consultations, accounts for relatively few hospital admissions, while *Salmonella* and *E. coli* O157 are more prominent causes of food-related hospitalisation.

Table 5.5: Estimates of food-related cases, GP consultations and hospitalisations by pathogen from Model 1, based on 100,000 Monte Carlo simulations

Organism	Cases	(95% CrI)	GP consultations	(95% CrI)	Hospital admissions	(95% CrI)
Bacteria						
C. perfringens	79,165	(29,310 - 208,688)	12,610	(5,707 - 27,890)	165	(20 - 843)
Campylobacter	286,000	(131,105 - 532,400)	39,750	(18,890 - 69,540)	1,376	(289 - 4,607)
E. coli O157	9,536	(644 - 146,495)	324	(36 - 2,973)	2,141	(143 - 33,237)
Listeria	169	(100 – 215)	169	(100 – 215)	--	--
Salmonella	33,640	(8,286 - 135,798)	10,030	(4,019 - 24,299)	2,536	(608 - 10,400)
Shigella	1,274	(90 - 11,990)	684	(84 - 2,145)	32	(2 - 378)
Protozoa						
Cryptosporidium	2,035	(354 - 10,129)	588	(140 - 2,010)	72	(12 - 395)
Giardia	11,250	(2,239 - 52,878)	1,322	(286 - 4,960)	88	(17 - 415)
Viruses						
Adenovirus	11,920	(3,706 - 28,909)	987	(293 - 2,536)	191	(51 - 559)
Astrovirus	2,362	(594 - 7,180)	180	(41 - 576)	70	(15 - 262)
Norovirus	73,420	(50,320 - 104,000)	3,240	(1,985 - 5,162)	470	(270 - 779)
Rotavirus	14,850	(4,698 - 35,330)	1,603	(494 - 3,856)	237	(64 - 688)
Sapovirus	40,770	(26,661 - 60,230)	2,457	(1,496 - 3,947)	261	(145 - 445)
TOTAL	566,391		73,944		7,639	

Figure 5.4: Proportionate contribution of different organisms to foodborne disease burden, UK 2009: estimates from Monte Carlo simulation approach.



Note: *Listeria* numbers are too small to be displayed on the figure

5.5 CASES, GP CONSULTATIONS AND HOSPITAL ADMISSIONS ATTRIBUTABLE TO FOODBORNE TRANSMISSION (MODELS 2 AND 3)

Estimates of food-related cases, GP consultations and hospital admissions based on the Bayesian approach used in Model 2 are presented in Table 5.6. For this model, there were insufficient data from published studies to enable estimation of the foodborne burden due to sapovirus. For *Campylobacter*, *E. coli* O157, *Listeria* and *Salmonella*, further estimates from Model 3 are presented in Table 5.7. The estimates from the three different models are compared in Figure 5.5. In general, the results from all three approaches were similar for food-related cases and GP consultations. For most organisms, the Bayesian estimates from Model 2 benefit from greater precision. There were differences in the number of food-related hospital admissions estimated by the Monte Carlo and Bayesian approaches for some organisms, notably *Campylobacter*, rotavirus, adenovirus and astrovirus. The differences reflect discordance between outbreak data and data from the IID studies in terms of the hospitalisation rate for these organisms. Where differences were observed, the Bayesian approach gave lower estimates of the number of food-related hospital admissions.

The proportionate contribution of different organisms to food-related cases, GP consultations and hospital admissions, as estimated by Models 1 and 2 is shown in Figure 5.6. The two methods provide comparable estimates, although in Model 2, *Campylobacter* makes a somewhat lower contribution to food-related hospital admissions, while *Salmonella* and *E. coli* O157 account for a slightly greater fraction of hospitalisations compared with Model 1.

Figure 5.7 compares the contribution of the different organisms to all IID (Figure 5.7a) and food-related IID (Figure 5.7b). The number of cases (y-axis) is plotted against the number of GP consultations (x-axis) on a logarithmic scale. For each organism, the area of the corresponding circle is proportional to the ratio of cases to GP consultations and is an indication of the degree of under ascertainment. Thus, a large circle indicates that for that organism there are comparatively more cases in the community for every case that presents to the GP or, equivalently, that a smaller proportion of cases presents to the GP, as is the case for norovirus. Circles near the top-right quadrant of the chart correspond to organisms that account for a large

number of cases and GP consultations. Bacteria are represented by blue circles, viruses by orange circles and protozoa by green circles. Incidence data for these pathogens are derived from the IID2 study. For *Shigella*, represented by grey circles, overall incidence has been estimated by applying reporting ratios from the IID1 study to 2009 laboratory report data. For *Listeria*, incidence has been estimated from the number of laboratory reports only. Comparing Figures 5.7a and 5.7b, it can be seen that, although viral agents rank among the most common causes of IID, they are much less important as causes of foodborne illness, with norovirus ranking lower than *Campylobacter*, *C. perfringens* and *Salmonella* in terms of food-related GP consultations. Similarly, *Cryptosporidium* is much less important as a cause of foodborne disease.

Table 5.6: Estimates of food-related cases, GP consultations and hospitalisations by pathogen, UK 2009. (Estimates based on Model 2)

Organism	Cases	(95% CrI)	GP consultations	(95% CrI)	Hospital admissions	(95% CrI)
Bacteria						
C. perfringens	79,570	(30,700 - 211,298)	12,680	(6,072 - 27,040)	186	(38 - 732)
Campylobacter	280,400	(182,503 - 435,693)	38,860	(27,160 - 55,610)	562	(189 - 1,330)
E. coli O157	9,886	(748 - 142,198)	342	(37 - 3,030)	2,233	(170 - 32,159)
Listeria	183	(161 – 217)	183	(161 – 217)	--	--
Salmonella	33,130	(8,178 - 128,195)	10,060	(4,137 - 24,710)	2,490	(607 - 9,631)
Shigella	1,204	(181 - 8,142)	602	(341 - 1,060)	33	(4 - 270)
Protozoa						
Cryptosporidium	2,773	(562 - 12,200)	800	(233 - 2,386)	94	(18 - 436)
Giardia	7,877	(1,467 - 36,059)	883	(197 - 3,288)	47	(4 - 332)
Viruses						
Adenovirus	8,253	(4,734 – 13,780)	677	(345 – 1,278)	62	(30 – 118)
Astrovirus	3,470	(1,368 - 9,991)	262	(93 - 812)	11	(3 - 42)
Norovirus	74,100	(61,150 - 89,660)	3,276	(2,240 - 4,729)	332	(248 - 440)
Rotavirus	10,295	(6,049 - 16,730)	1,102	(629 - 1,870)	95	(48 - 177)
Sapovirus[1]	--	--	--	--	--	--
TOTAL	511,141		69,727		6,145	

[1]For sapovirus, no studies were identified in the literature review with information on the proportion of cases attributable to foodborne transmission so estimates could not be produced from this model

Table 5.7: Estimates of food-related cases, GP consultations and hospitalisations by pathogen, UK 2009. (Estimates based on Model 3)

Organism	Cases	(95% CrI)	GP consultations	(95% CrI)	Hospital admissions	(95% CrI)
Campylobacter	279,900	(183,100 - 433,098)	38,820	(27,010 - 55,580)	561	(189 - 1,343)
E. coli O157	9,536	(644 - 146,495)	324	(36 - 2,973)	2,141	(143 - 33,237)
Listeria	166	(92 - 214)	166	(92 - 214)	--	--
Salmonella	33,130	(8,178 - 128,195)	10,060	(4,137 - 24,710)	2,490	(607 - 9,631)
TOTAL	322,732		49,370		5,192	

Figure 5.5a: Comparison of estimates from Monte Carlo and Bayesian approaches - Food-related cases (Model 1: Monte Carlo simulation approach, Model 2: Bayesian approach using data from published food attribution studies, Model 3: Bayesian approach using data from published pathogen-specific studies (Error bars show 95% CrI))

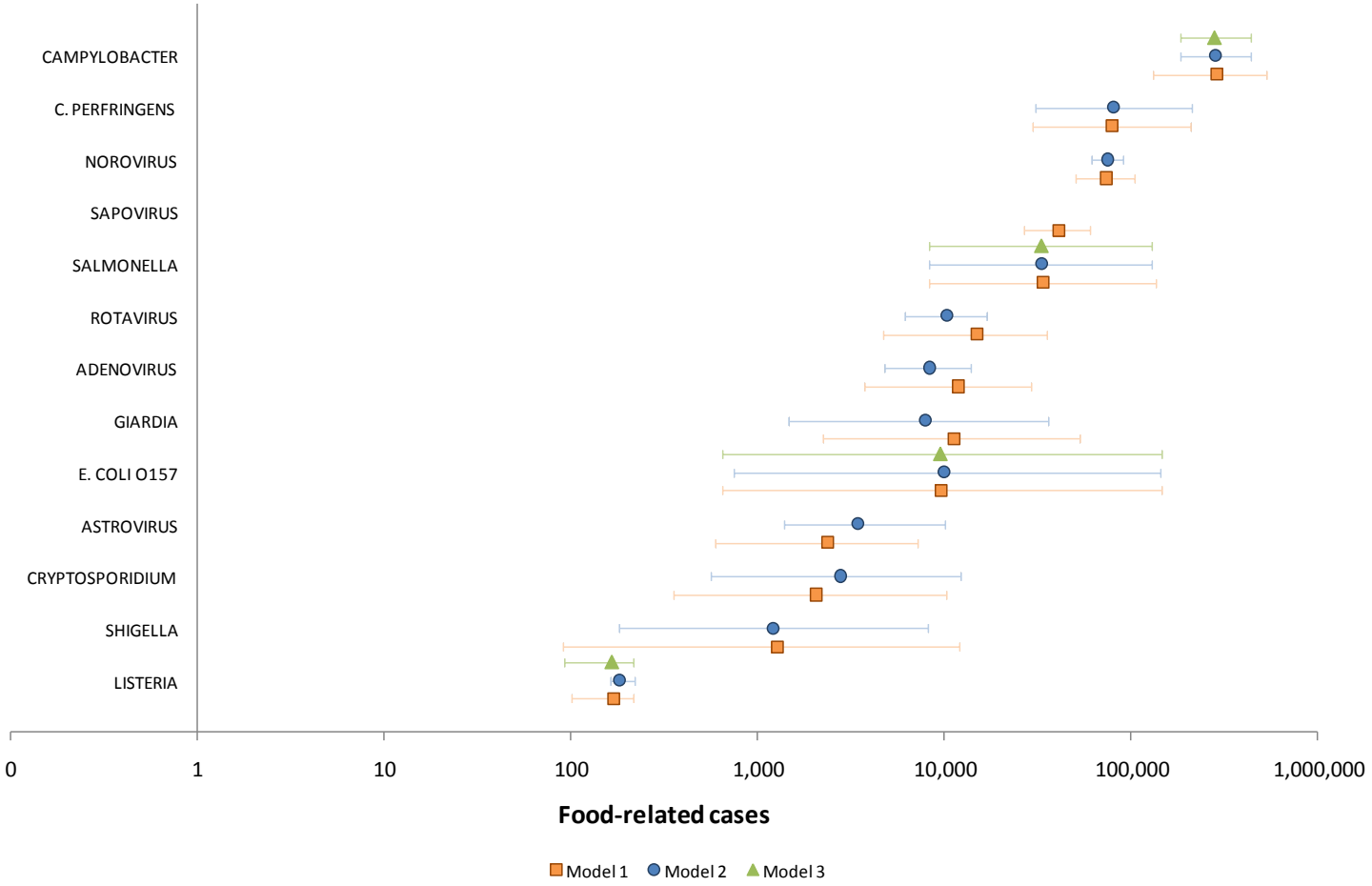


Figure 5.5b: Comparison of estimates from Monte Carlo and Bayesian approaches - Food-related GP consultations (Model 1: Monte Carlo simulation approach, Model 2: Bayesian approach using data from published food attribution studies, Model 3: Bayesian approach using data from published pathogen-specific studies (Error bars show 95% CrI))

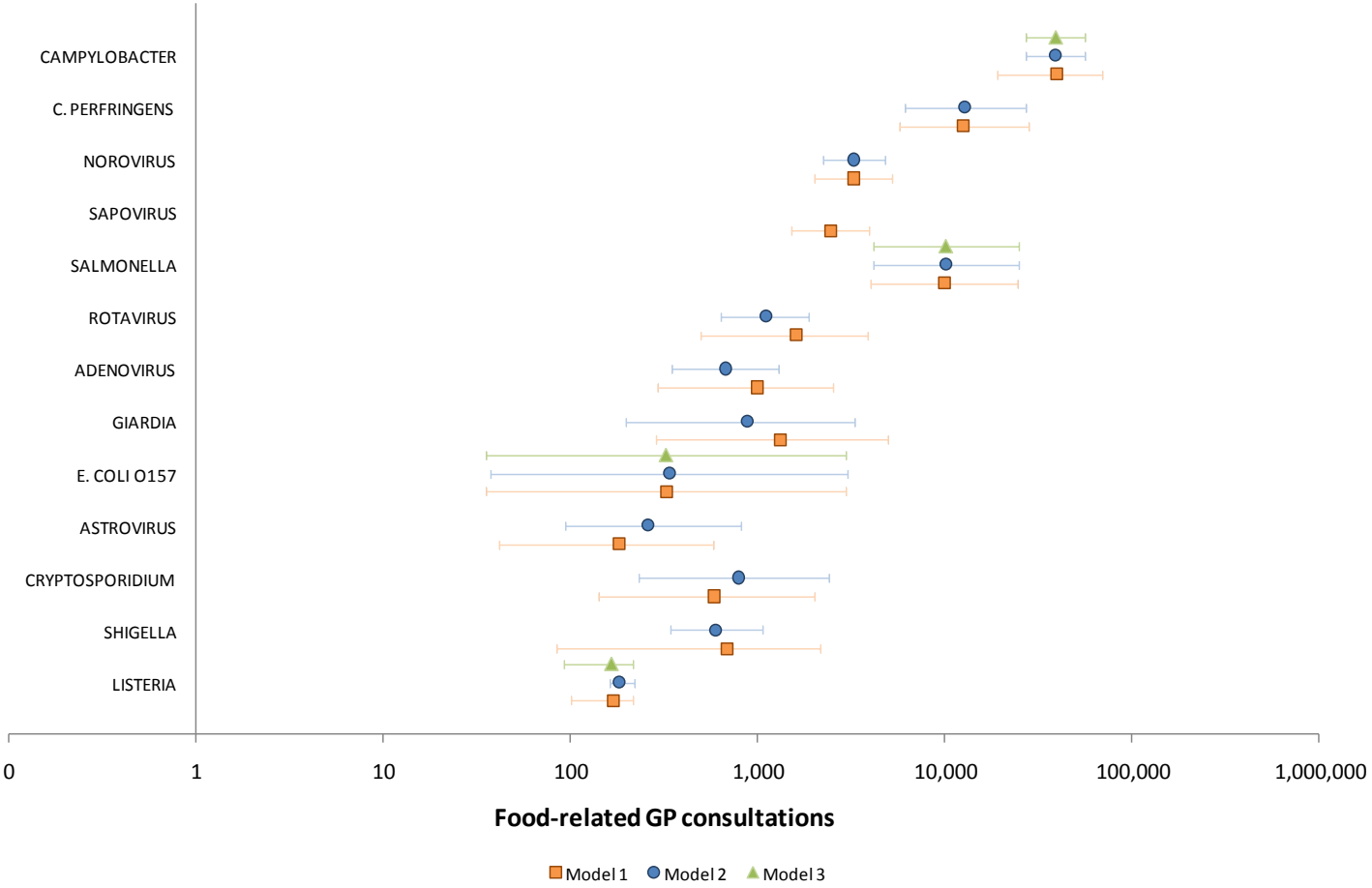


Figure 5.5c: Comparison of estimates from Monte Carlo and Bayesian approaches - Food-related hospital admissions (Model 1: Monte Carlo simulation approach, Model 2: Bayesian approach using data from published food attribution studies, Model 3: Bayesian approach using data from published pathogen-specific studies (Error bars show 95% CrI))

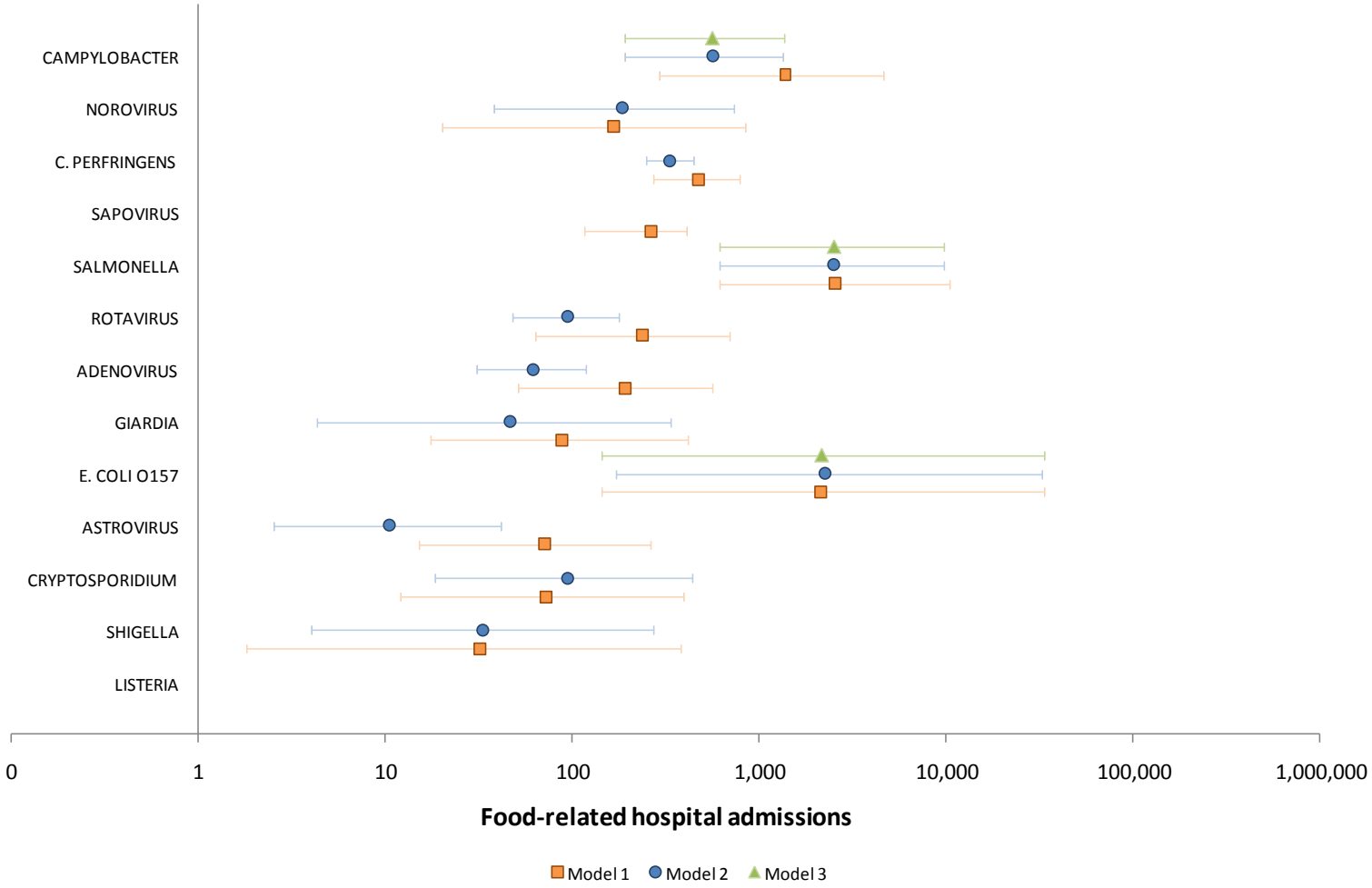
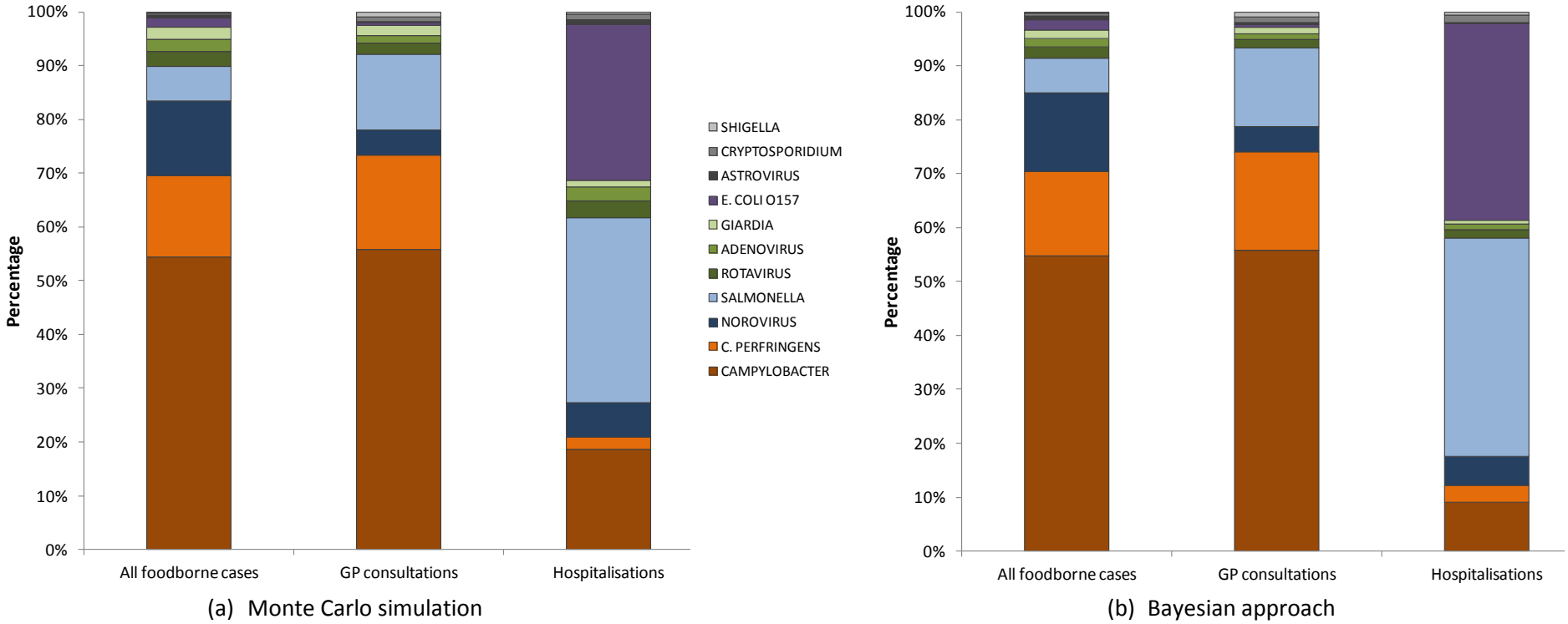
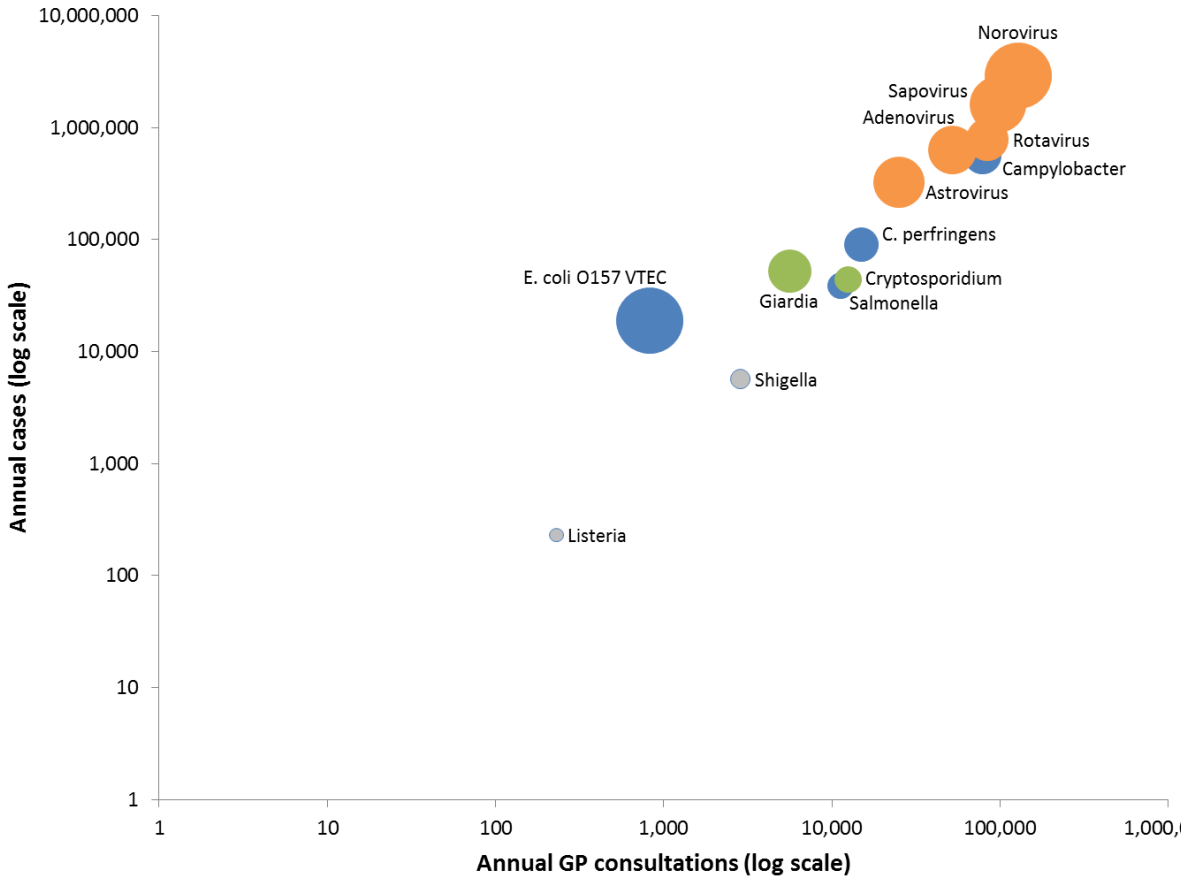


Figure 5.6: Proportionate contribution of different organisms to foodborne illness burden: Comparison of Monte Carlo and Bayesian approaches. (a) Monte Carlo simulation, (b) Bayesian approach.



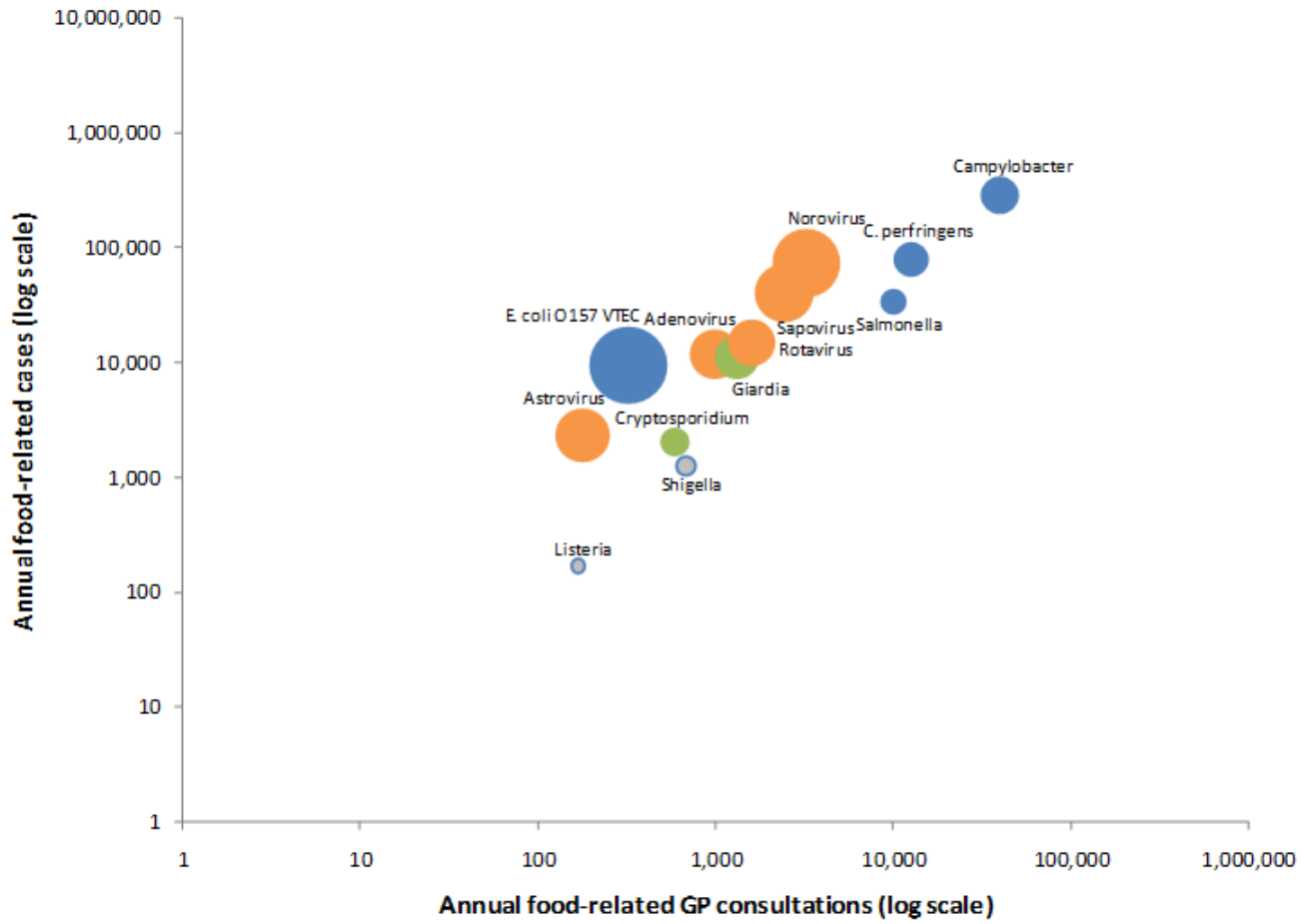
Only organisms for which estimates were available from both models are shown. *Listeria* is omitted, as the number of foodborne cases and GP consultations are too small to be displayed and hospitalisations could not be estimated by either method

Figure 5.7a: Annual estimated cases and GP consultations for all IID by organism (data from IID2 Study) (estimates based on Model 2).



NOTES: Area of circles represents the ratio of all cases to GP consultations. Blue circles: Bacteria, Orange circles: Viruses, Green circles: Protozoa, Grey circles: incidence data for these organisms (i.e. *Shigella*) is based on laboratory reports in 2009 multiplied by reporting ratios estimated in the IID1 Study, except for *Listeria*, for which laboratory reports only have been used.

Figure 5.7b: Annual estimated food-related IID cases and GP consultations by organism (estimates based on Model 2)



NOTES: Area of circles represents the ratio of all cases to GP consultations. Blue circles: Bacteria, Orange circles: Viruses, Green circles: Protozoa, Grey circles: incidence data for these organisms are based on laboratory reports in 2009 multiplied by reporting ratios estimated in the IID1 Study, except for *Listeria*, for which laboratory reports only have been used.

CHAPTER 6

RESULTS 2 – ESTIMATING THE BURDEN OF FOODBORNE ILLNESS BY FOOD COMMODITY

6.1 LITERATURE REVIEW

Table 6.1 summarises eight food attribution studies that we identified from the systematic literature review.

6.2 ESTIMATES FROM PUBLISHED FOOD ATTRIBUTION STUDIES

Estimates of the percentage of cases attributable to different food commodities from published food attribution studies are shown graphically in Figure 6.1. Each radar chart represents one pathogen. Each marker represents the percentage of cases, as estimated by each study, attributable to the corresponding food commodity. Values closer to the centre of the radar chart indicate a higher percentage of cases attributable to that food commodity. Studies are colour coded, such that blue markers represent estimates from outbreak studies and orange markers represent studies from expert elicitation studies. One study from Denmark, by Hald *et al*. (2004) used microbiological typing information to attribute salmonellosis cases to different food commodities. Little *et al*. (2010) used a similar approach for attribution of listeriosis cases in England and Wales.

The charts convey visually how much information there is from previous studies, as well as the degree of variation in estimates between studies. We found only one study with information on *Giardia* and rotavirus, while for *Campylobacter* and *Salmonella* there were six and nine sets of estimates respectively. For some pathogen and food commodity combinations, there was considerable variation between studies in their estimated contribution to foodborne illness. In particular, the percentage of *Salmonella* cases estimated to result from egg consumption varied widely between 13% and 80%. Similarly, the percentage of *Campylobacter* cases thought to be attributable to poultry consumption varied between 35% and 71%. The parameter values from these studies used to construct the Dirichlet priors are given in Appendix 5.

Table 6.1: Summary of included food attribution studies ('x' indicates that information for that pathogen was available from a particular study). The ten pathogens included in the food commodity attribution analysis are in bold

	Studies (N=8)								
Author	**Adak**	**Davidson**	**Greig**	**Hald**	**Havelaar**	**Hoffmann**	**Little**	**Pires**	
Year	**2005**	**2011**	**2009**	**2004**	**2008**	**2007**	**2010**	**2010**	
Country	**UK**	**CAD**	**CAD**	**Denmark**	**NL**	**US**	**UK**	**EU**	
Period	**1996-2000**	**2008**	**1988-2007**	**1999**	**2006**	**2000s**	**2004-2007**	**2005-2006**	
Data sources*	**V**	**E**	**O**	**M**	**E**	**E**	**M**	**O**	Studies
Travel cases	**Excluded**	**Excluded**	**Included**	**Excluded**	**Included**	**Excluded**	**Excluded**	**Included**	Identified
All organisms	X								1
Bacillus			X		X				2
C. difficile									
C. perfringens			**X**		**X**				**2**
Campylobacter		**X**	**X**		**X**	**X**		**X**	**5**
***E. coli* O157**		**X**	**X**		**X**	**X**			**4**
E. coli non-O157					X				1
Listeria		X	X		X	X	X		5
Salmonella		**X**	**X**	**X**	**X**	**X**		**X**	**6**
Shigella		**X**	**X**			**X**			**3**
Staph. aureus			X		X				2
Yersinia		X				X			2
Cryptosporidium		**X**			**X**	**X**			**3**
Giardia					**X**				**1**
Adenovirus									
Astrovirus									
Enterovirus									
Norovirus		**X**			**X**	**X**			**3**
Rotavirus					**X**				**1**
Sapovirus									

* V: various data sources; E: expert elicitation study; O: outbreak data; M: modelling of molecular typing data

Figure 6.1a: Summary of estimates for the percentage of foodborne illness cases attributable to each food commodity by pathogen from food attribution studies identified in the literature

C. perfringens

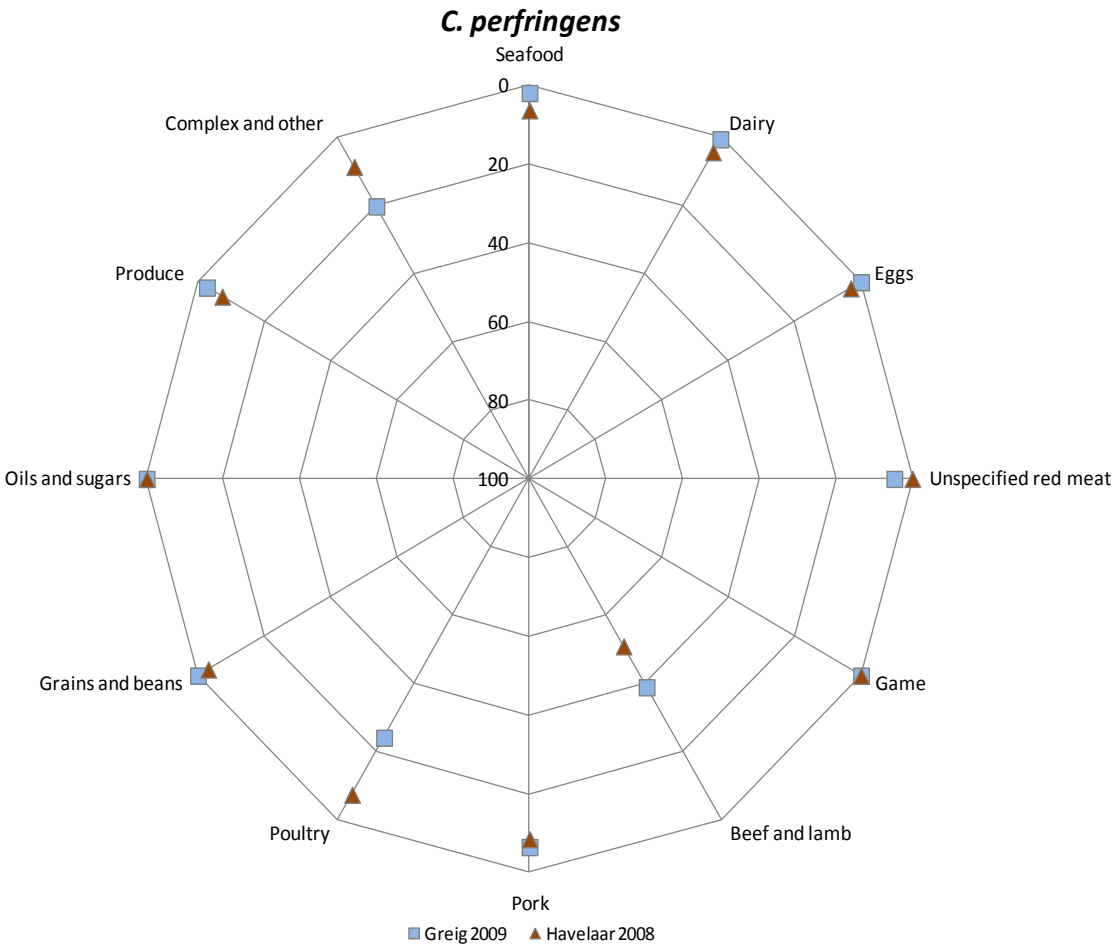
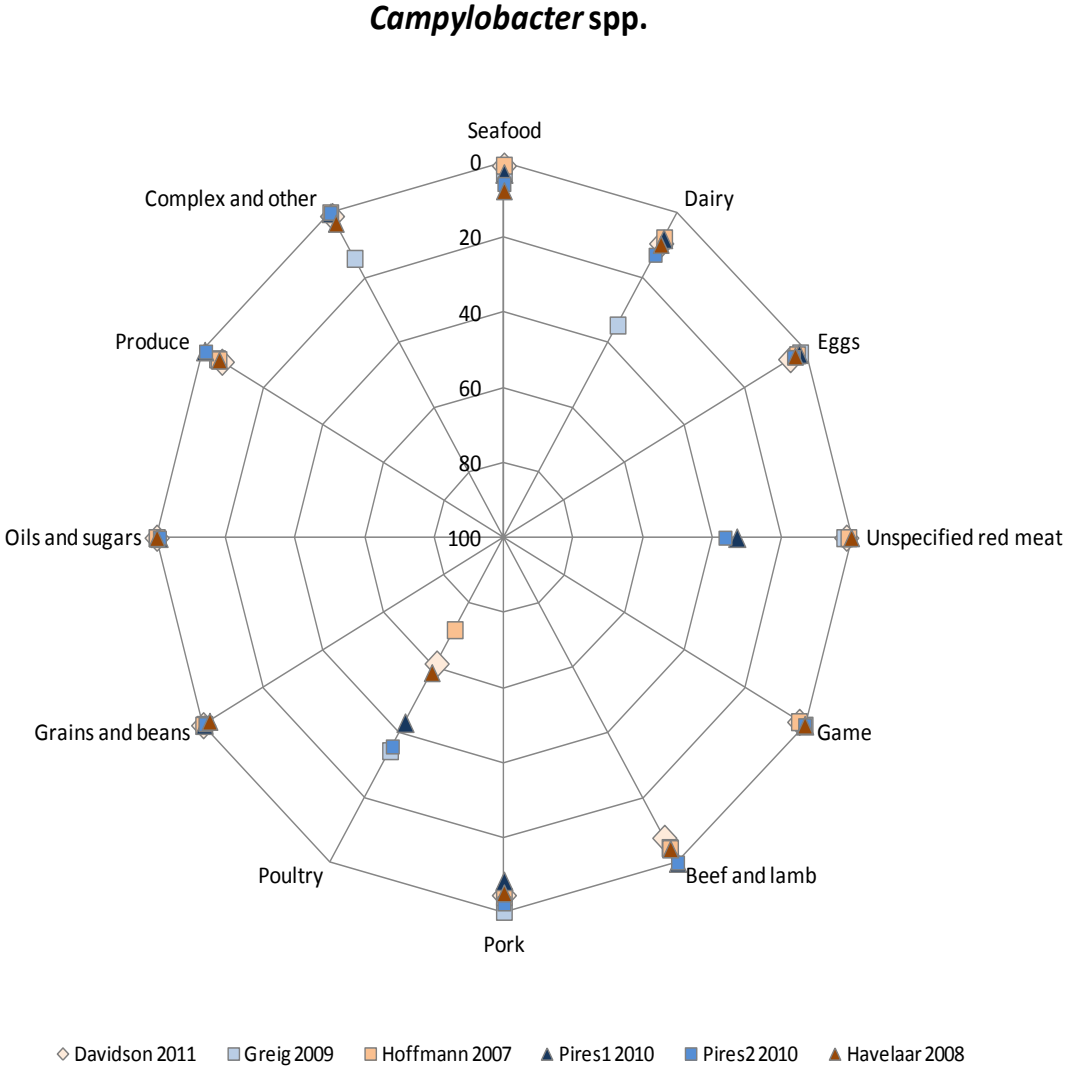


Figure 6.1b: Summary of estimates for the percentage of foodborne illness cases attributable to each food commodity by pathogen from food attribution studies identified in the literature - Campylobacter spp.



NOTES: Pires 1 comprises estimates based on the percentage of outbreaks attributed to different food commodities; Pires 2 comprises estimates based on the percentage of cases in outbreaks attributed to different food commodities

Figure 6.1c: Summary of estimates for the percentage of foodborne illness cases attributable to each food commodity by pathogen from food attribution studies identified in the literature - E. coli O157

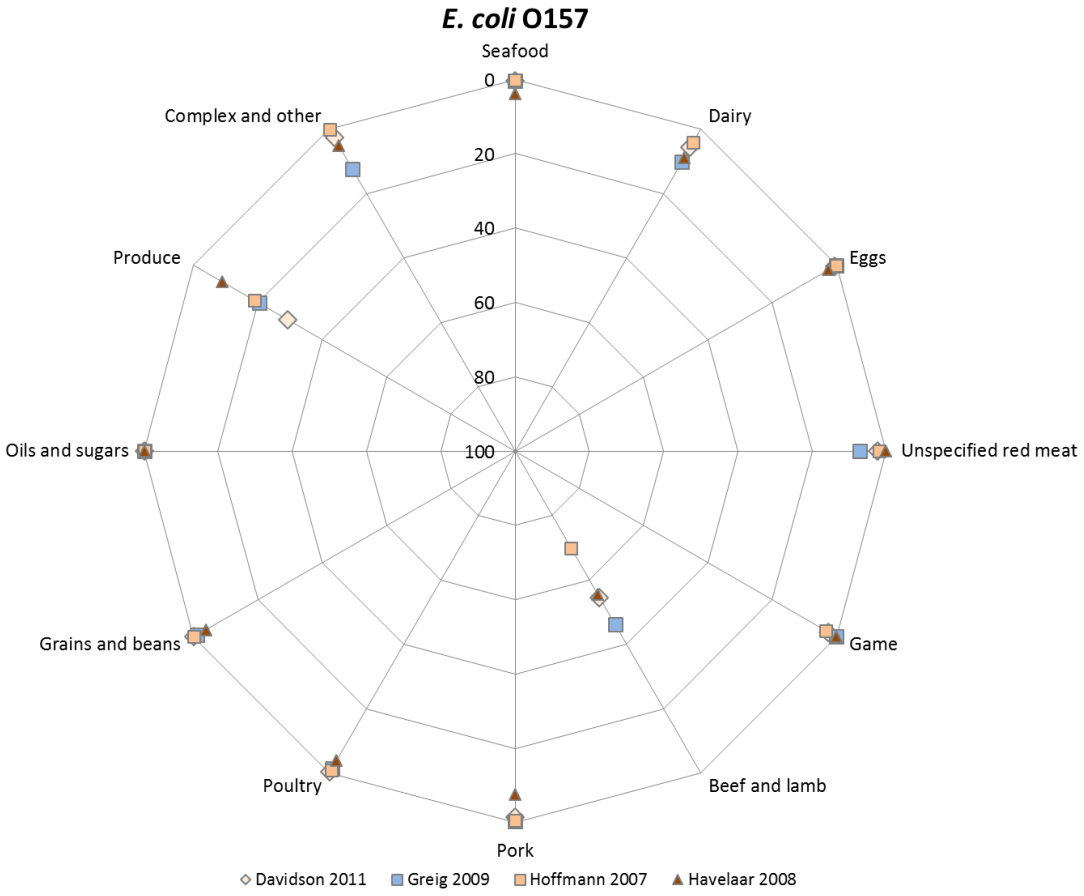


Figure 6.1d: Summary of estimates for the percentage of foodborne illness cases attributable to each food commodity by pathogen from food attribution studies identified in the literature - Listeria

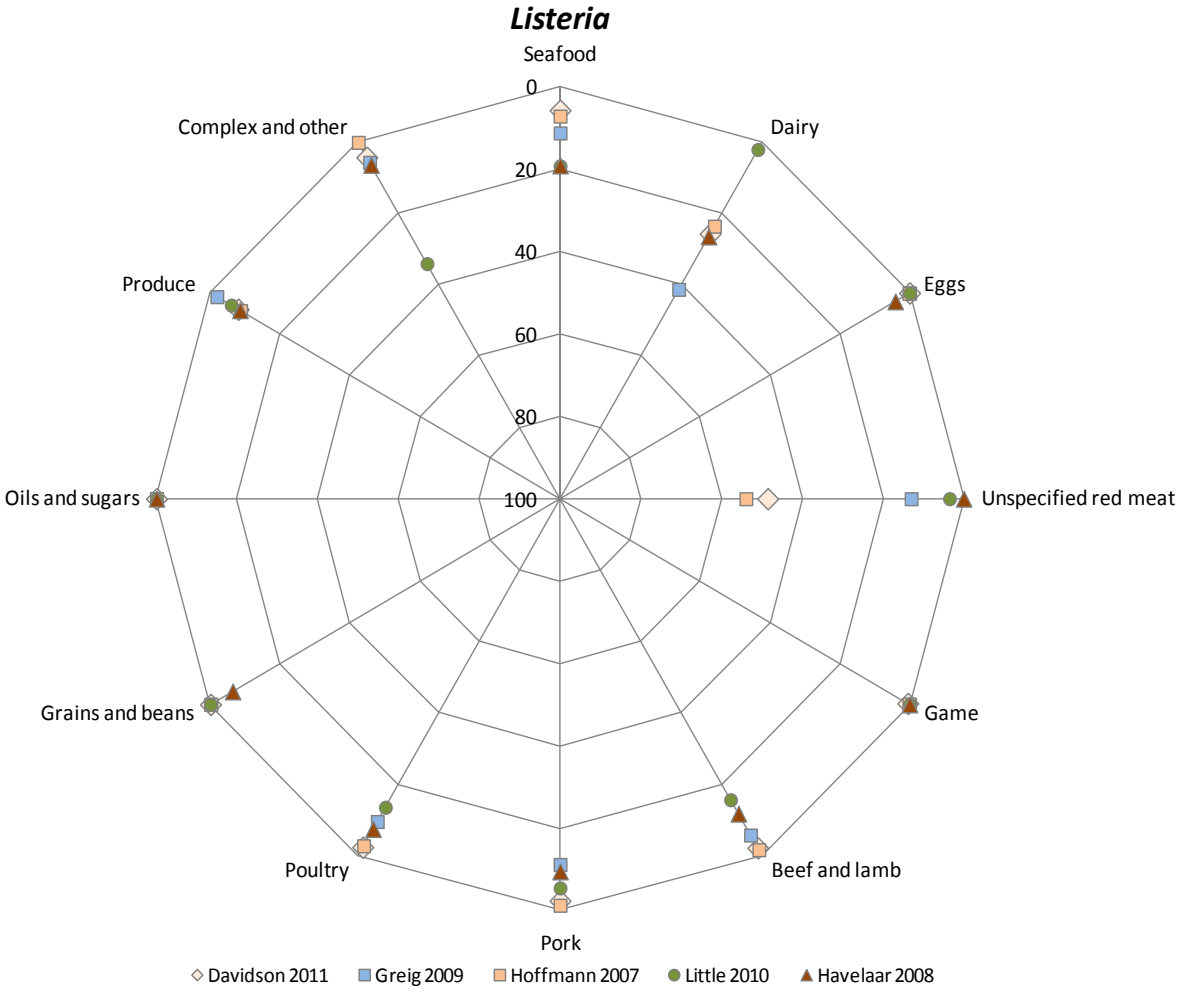
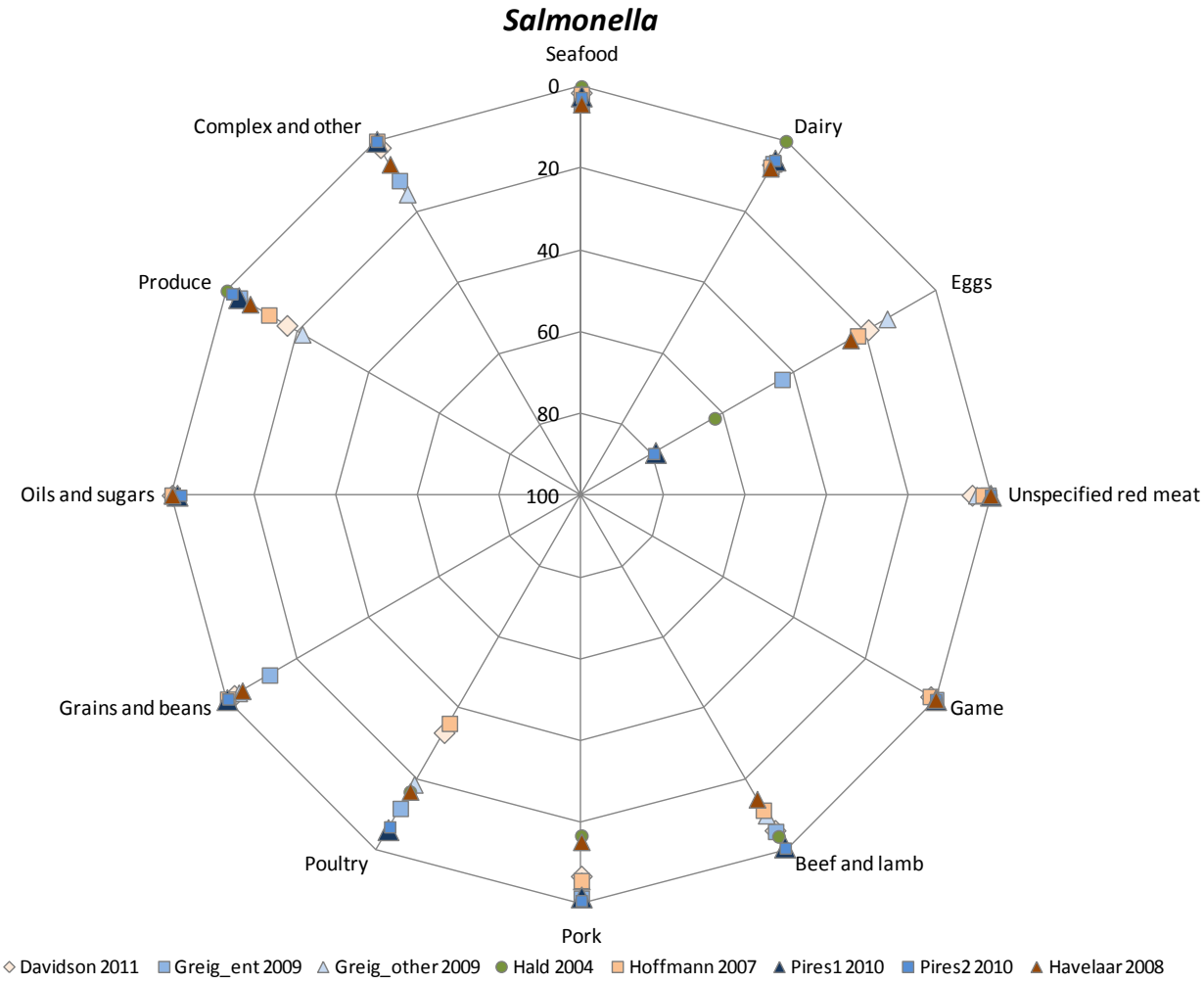


Figure 6.1e: Summary of estimates for the percentage of foodborne illness cases attributable to each food commodity by pathogen from food attribution studies identified in the literature - Salmonella spp



NOTES: Greig (SE) comprises estimates for *Salmonella* Enteritidis; Greig (Other) comprises estimates for other *Salmonella* types; Pires 1 comprises estimates based on the percentage of of outbreaks attributed to different food commodities; Pires 2 comprises estimates based on the percentage of cases in outbreaks attributed to different food commodities

Figure 6.1f: Summary of estimates for the percentage of foodborne illness cases attributable to each food commodity by pathogen from food attribution studies identified in the literature - Shigella

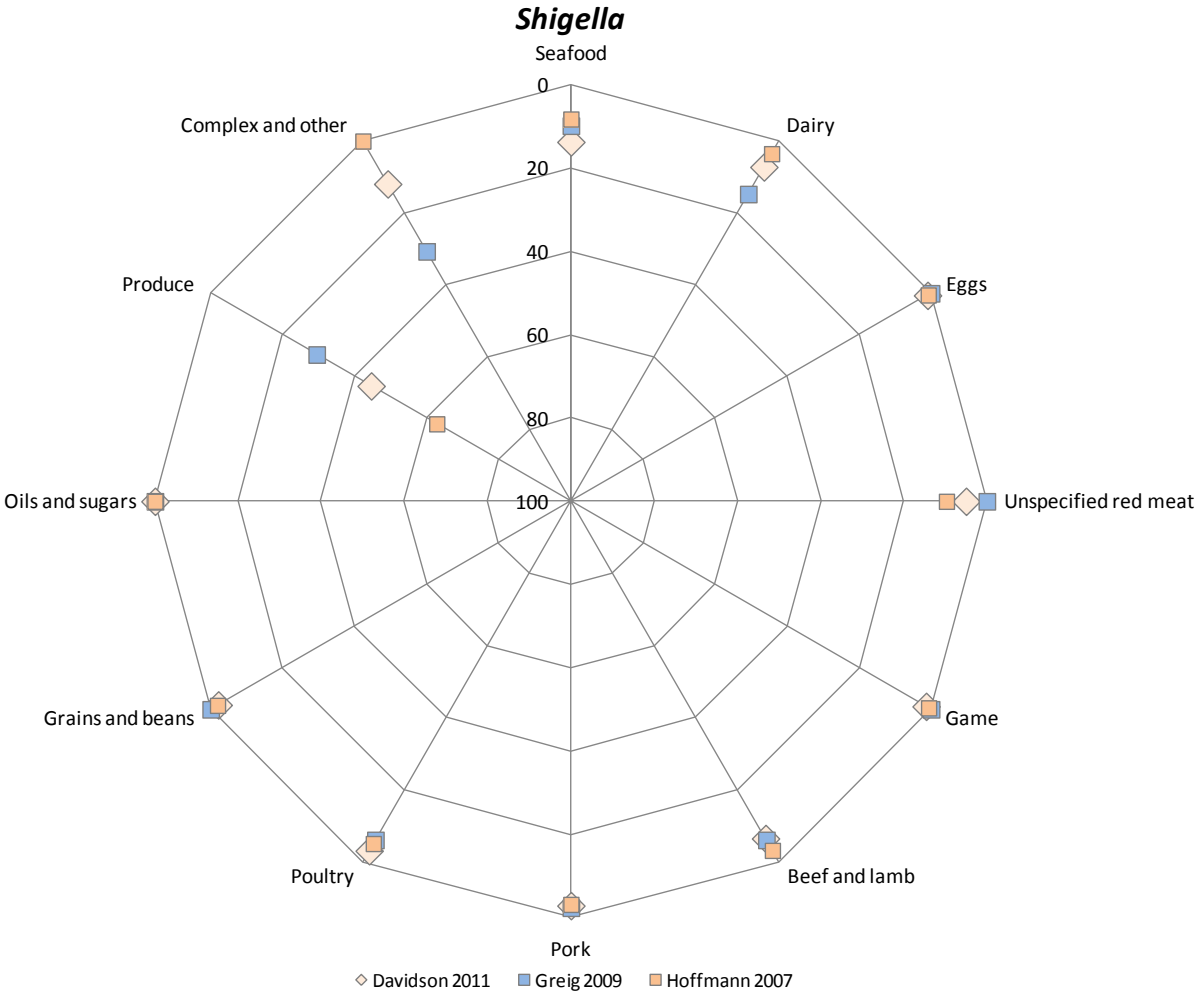


Figure 6.1g: Summary of estimates for the percentage of foodborne illness cases attributable to each food commodity by pathogen from food attribution studies identified in the literature - Cryptosporidium

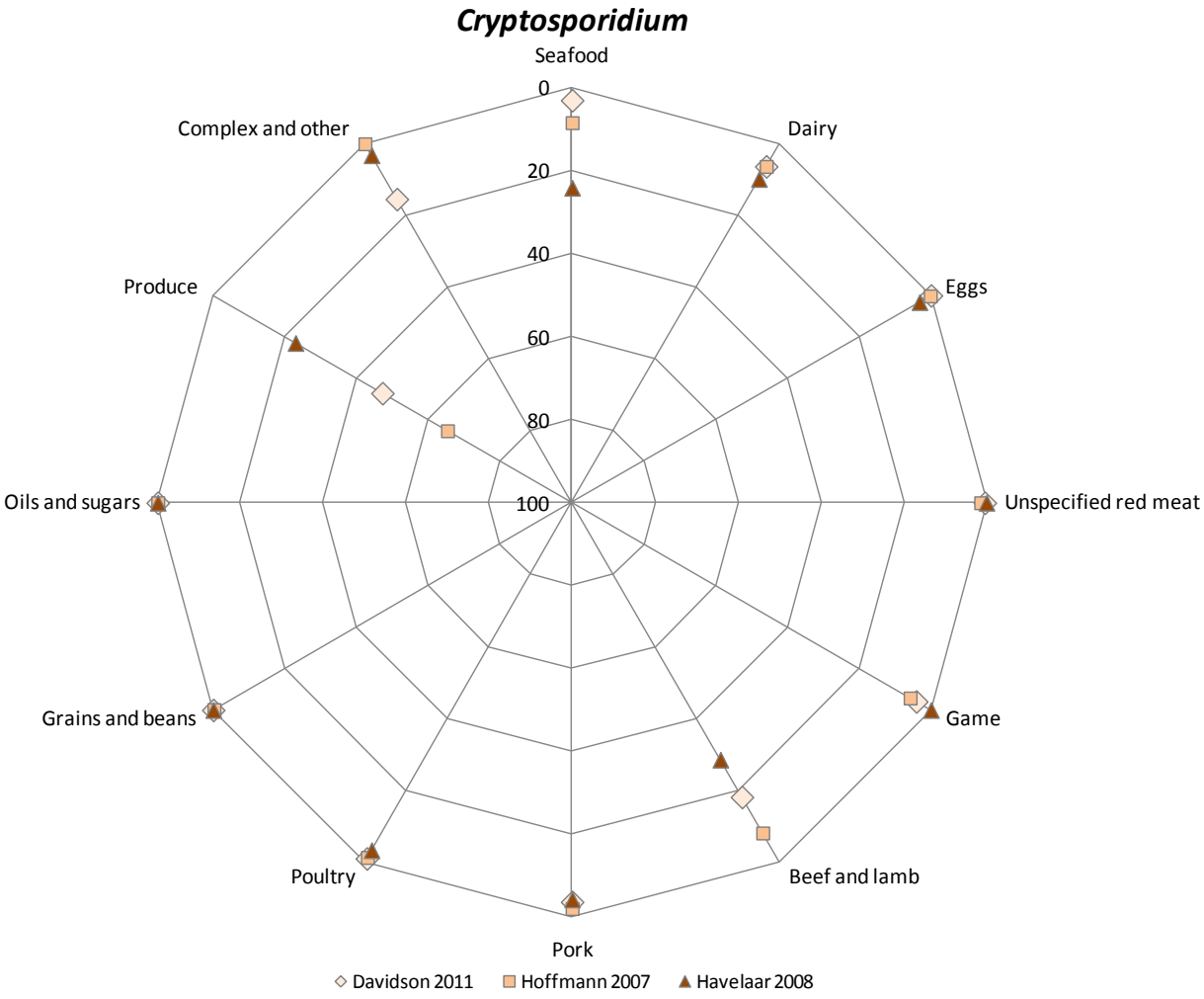


Figure 6.1h: Summary of estimates for the percentage of foodborne illness cases attributable to each food commodity by pathogen from food attribution studies identified in the literature - Giardia

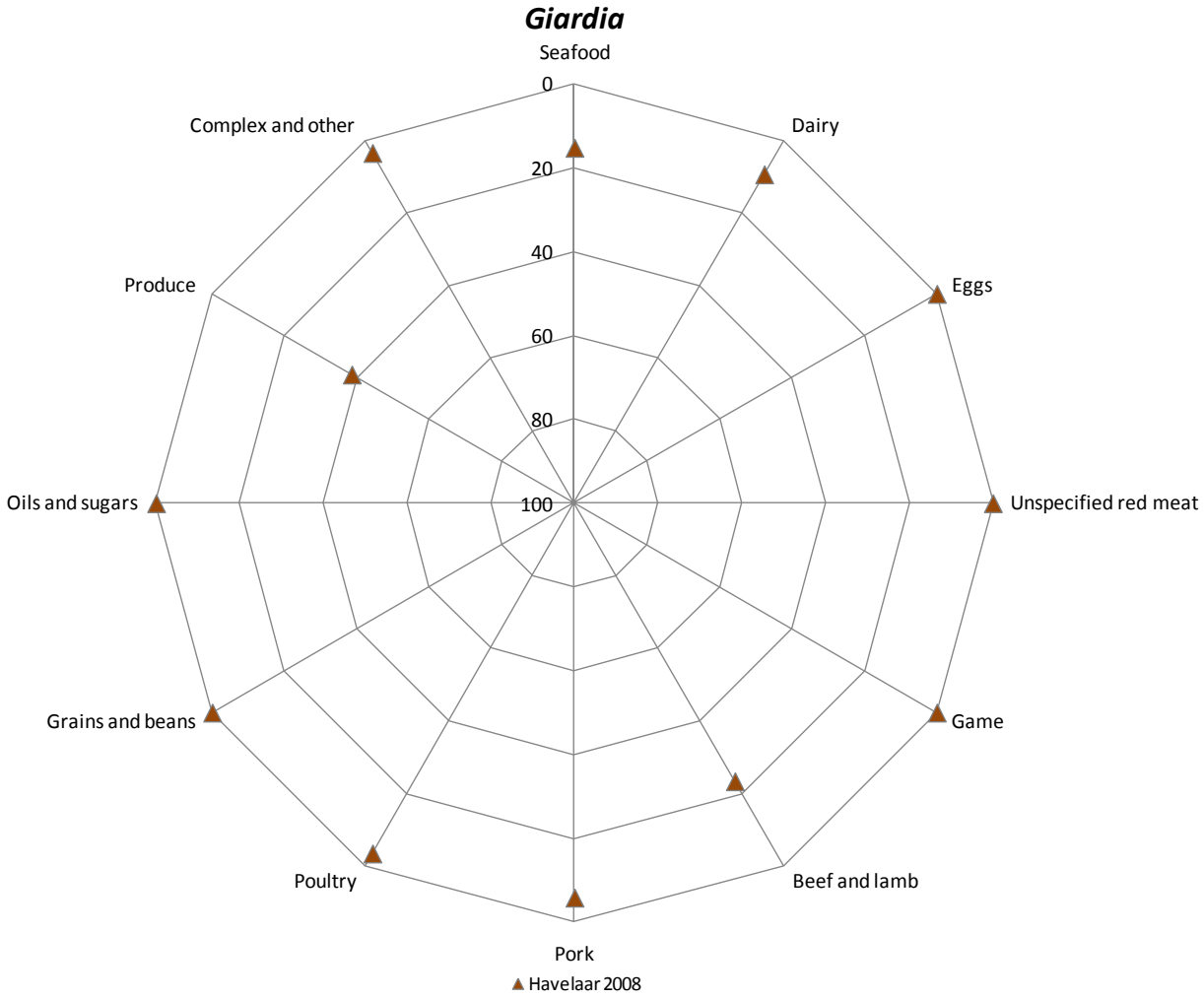


Figure 6.1i: Summary of estimates for the percentage of foodborne illness cases attributable to each food commodity by pathogen from food attribution studies identified in the literature - Norovirus

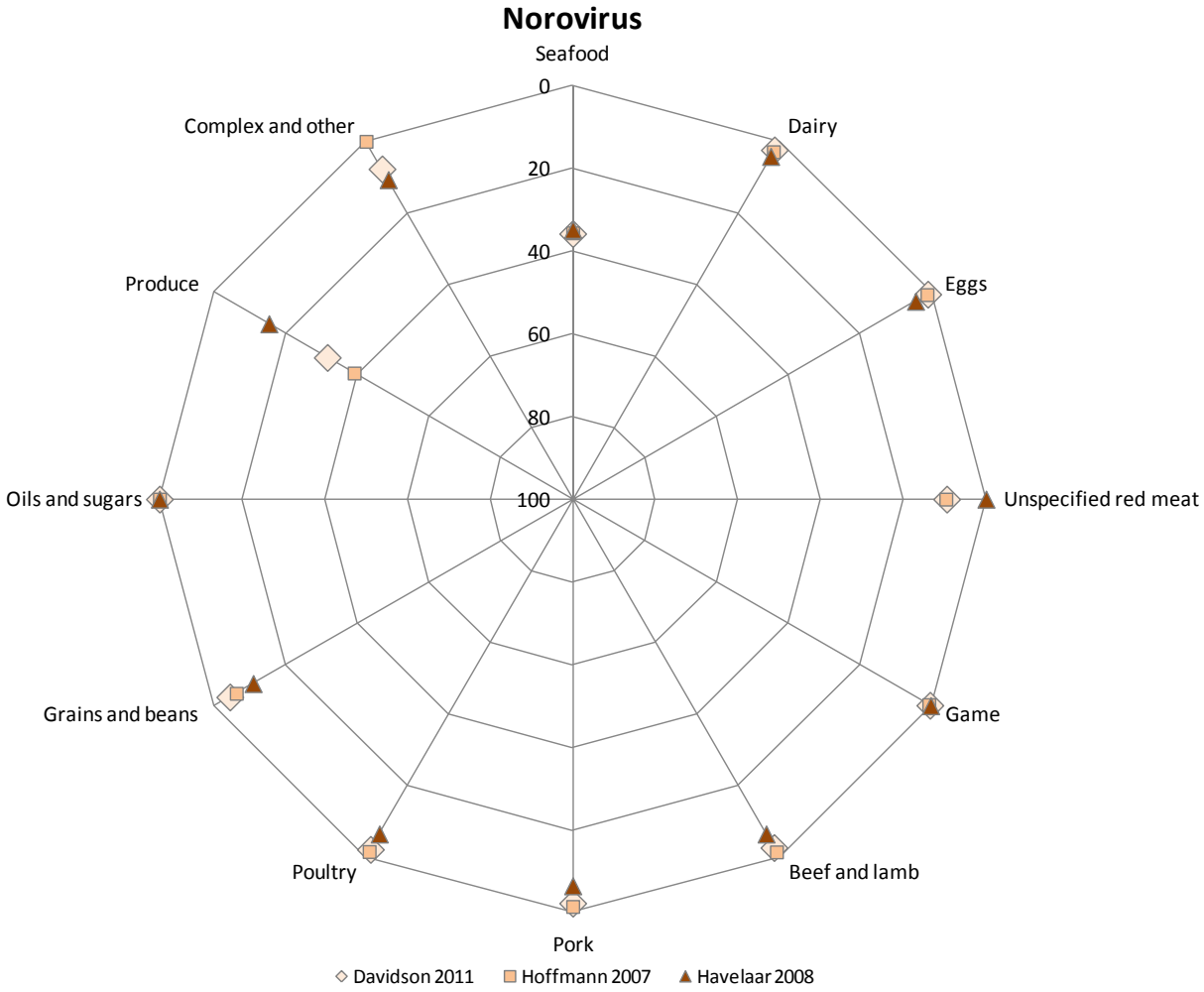
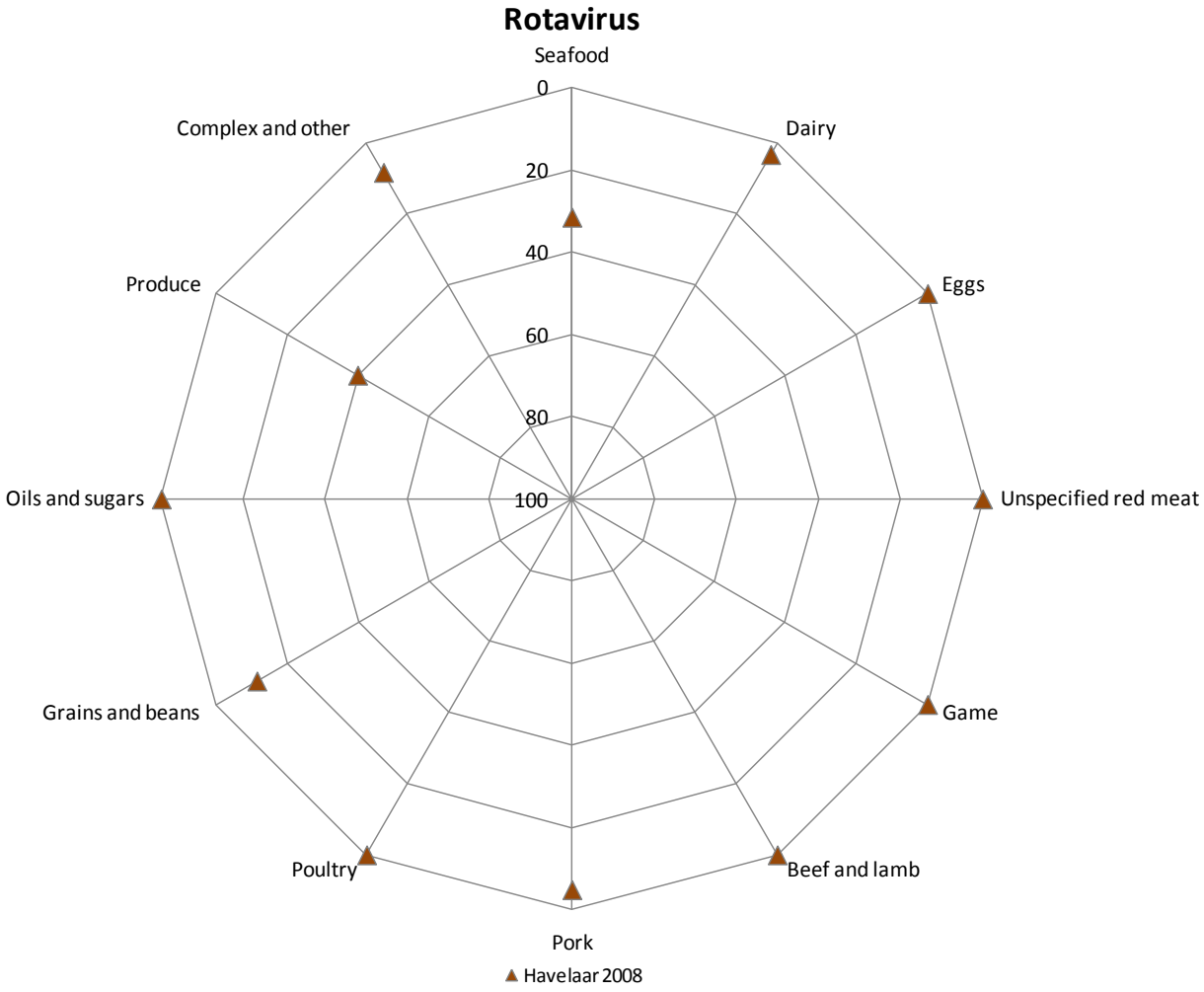


Figure 6.1j: Summary of estimates for the percentage of foodborne illness cases attributable to each food commodity by pathogen from food attribution studies identified in the literature - Rotavirus



6.3 CONTRIBUTION OF DIFFERENT FOOD COMMODITIES TO PATHOGEN-SPECIFIC IID

The posterior distributions obtained from models using the Bayesian approach are shown in Appendix 6.1 for the pathogens *C. perfringens*, *Campylobacter*, *E. coli* O157, *Salmonella* and norovirus. For each figure, each panel corresponds to a separate food commodity. The densities plotted correspond to the posterior densities estimated using Dirichlet priors from individual studies and are colour coded according to the scheme in Figure 6.1. The dashed black line corresponds to the

density of the combined distributions, while the grey line corresponds to the posterior distribution obtained from a model with a vague prior, as described in section 4.6.

Despite variation in the priors used, the results from the different models are quite similar, as the outbreak data are given more weight in this analysis. A clear exception is the role of poultry in *Campylobacter* transmission, for which variation between studies results in a wider range of estimates.

Appendix 6.2 shows the posterior distributions obtained from models in which prior information only, with no outbreak data, was used. As can be seen, the posterior distributions are now much more variable, as estimates are influenced much more heavily by variations between studies. This is particularly true for the role of beef and lamb in *C. perfringens* transmission, poultry in *Campylobacter* transmission, beef and lamb and produce in *E. coli* O157 transmission, eggs and poultry in *Salmonella* transmission. For other pathogens, including *Shigella*, *Cryptosporidium* and norovirus, there is considerable variability in estimates of the role of produce.

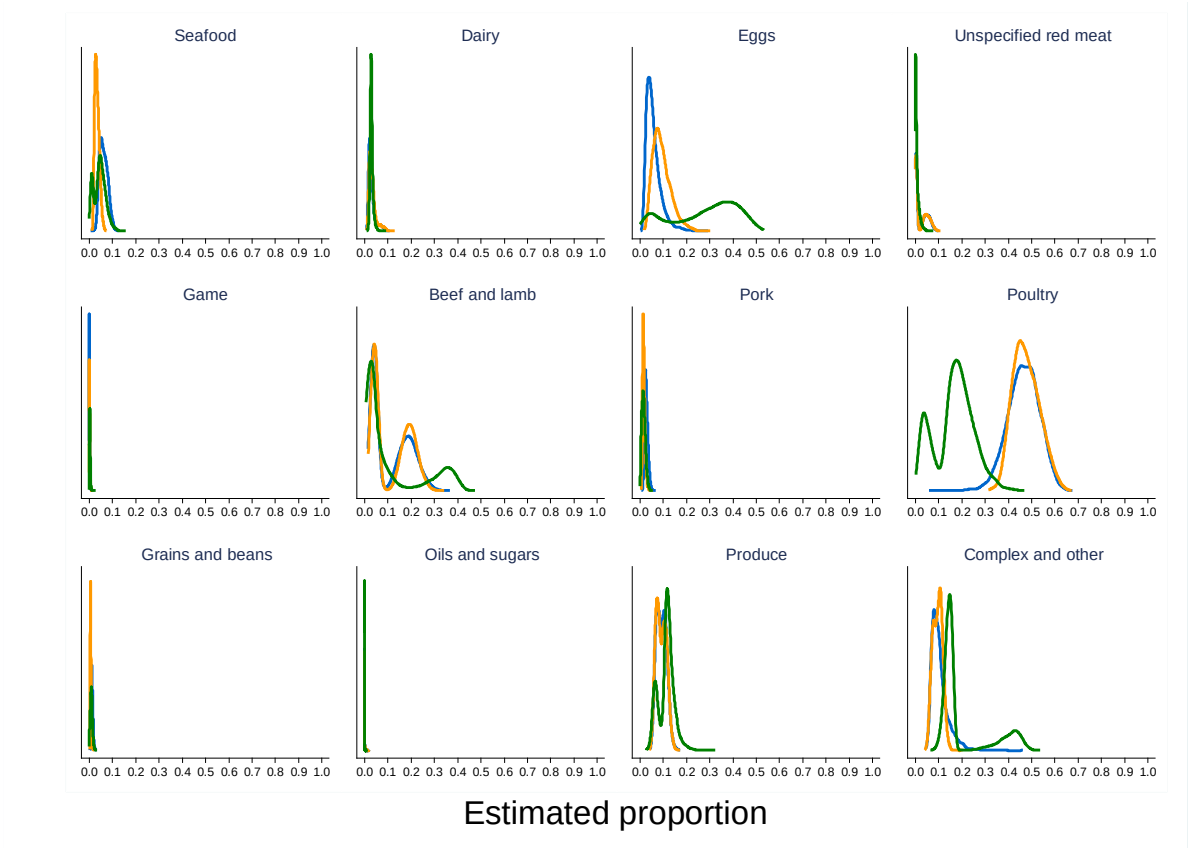
6.4 PROPORTION OF FOODBORNE ILLNESS ATTRIBUTABLE TO DIFFERENT FOOD COMMODITIES

Figure 6.2 shows density plots for the contribution of different food commodities to overall IID caused by the nine pathogens included in the analysis. The densities correspond to the combination of posterior distributions summed across pathogens. For *C. perfringens*, *Campylobacter*, *E. coli* O157, *Salmonella* and norovirus the combined distributions from the models using the Bayesian approach were used, while for the remaining pathogens, the combined distributions from the models with only prior information were used. The blue lines represent cases, the orange lines GP consultations and the green lines hospital admissions. For each food commodity, the eventual shape of the distribution is influenced by the frequency of pathogens transmitted through that route and the relative severity of those pathogens. Estimates of the proportionate contribution of each food commodity to cases and GP consultations are similar, because pathogens that cause large numbers of cases also tend to result in large numbers of GP consultations. However, the estimates for hospital admissions are quite different, particularly for eggs and poultry. This is because the main pathogen transmitted through egg consumption is *Salmonella*, which tends to have higher rates of hospitalisation. Conversely, poultry contributes

proportionately fewer hospital admissions than cases and GP consultations, because *Campylobacter*, commonly transmitted through poultry consumption, has lower hospitalisation rates. The shapes of density plots for hospitalisations are also more complex, partly due to the greater uncertainty around estimates of hospitalisation, compounded by variability around the contribution of some food commodities.

Plots comparing estimates obtained through the different modelling approaches are given in Appendix 7.

Figure 6.2: Densities for the combined posterior distributions of the proportion of cases (blue), GP consultations (orange) and hospital admissions (green) attributable to each food commodity



y-axis: posterior density (y-axis values are omitted to allow clearer comparison between food commodities); x-axis: estimated proportion of cases attributable to each food commodity

Estimates of cases, GP consultations and hospital admissions attributable to different food commodities in the UK in 2009 are presented in Tables 6.2a-c. The percentage contribution of each food commodity to all food-related illness is also shown. Note that the totals in these tables do not correspond to the totals in Table 5.6 for the reasons described in Section 4.6 and because they include a smaller set of pathogens. Poultry-related illness accounted for approximately half of all cases and GP consultations, equating to nearly 250,000 cases and 34,000 GP visits. Beef and lamb, produce and complex and other foods each accounted for approximately 10% of cases and GP consultations.

As mentioned above, the relative severity of different pathogens is partly reflected in the distribution of food commodities. Figure 6.3 shows the percentage contribution of each food commodity to foodborne disease cases, GP consultations and hospital admissions. The figure does not display the uncertainty around these estimates and should be interpreted with caution. It illustrates, however, that eggs accounted for around 5% of cases, 9% of GP consultations, but 32% of hospital admissions, reflecting the greater severity of illness from *Salmonella* infection, for which eggs were the main food vehicle (Figure 6.3). By contrast, poultry accounts for 50% of cases and GP consultations, but only 20% of hospital admissions. For a number of key food commodities, particularly poultry, beef and lamb and eggs, uncertainty around these estimates was high.

Table 6.2a: Estimated cases of foodborne illness by food commodity, UK 2009

Food commodity	Mean	Median	(95% CrI)	% of total
Seafood	32,107	31,761	(25,169-41,207)	6.6%
Dairy	16,445	14,065	(7,304-39,012)	2.9%
Eggs	30,963	25,928	(11,646-81,948)	5.4%
Unspecified red meat	12,725	3,352	(136-39,356)	0.7%
Game	892	546	(87-3,520)	0.1%
Beef and lamb	74,084	43,357	(10,321-217,627)	9.0%
Pork	14,350	14,003	(9,142-21,728)	2.9%
Poultry	248,596	243,988	(151,743-372,961)	50.8%
Grains and beans	6,686	6,532	(4,542-9,784)	1.4%
Oils and sugars	380	127	(2-2,167)	0.0%
Produce	48,868	47,575	(33,035-71,162)	9.9%
Complex and other	61,856	49,416	(24,270-159,642)	10.3%
Total	547,953	480,650		100.0%

Note: Median values from the model are used to generate % totals

Table 6.2b: Estimated GP consultations due to foodborne illness by food commodity, UK 2009

Food commodity	Mean	Median	(95% CrI)	% of total
Seafood	2,334	2,285	(1,587-3,329)	3.4%
Dairy	2,318	2,009	(1,147-5,267)	3.0%
Eggs	6,671	6,068	(2,739-14,250)	9.1%
Unspecified red meat	1,688	373	(19-5,224)	0.6%
Game	147	106	(25-497)	0.2%
Beef and lamb	9,753	7,077	(1,515-24,829)	10.7%
Pork	1,184	1,138	(560-2,087)	1.7%
Poultry	33,980	33,637	(21,544-	50.7%
Grains and beans	497	474	(294-842)	0.7%
Oils and sugars	51	17	(0-292)	0.0%
Produce	6,398	6,292	(4,389-8,996)	9.5%
Complex and other	7,188	6,862	(3,071-13,100)	10.3%
Total	72,210	66,336		100.0%

Note: Median values from the model are used to generate % totals

Table 6.2c: Estimated hospital admissions due to foodborne illness by food commodity, UK 2009

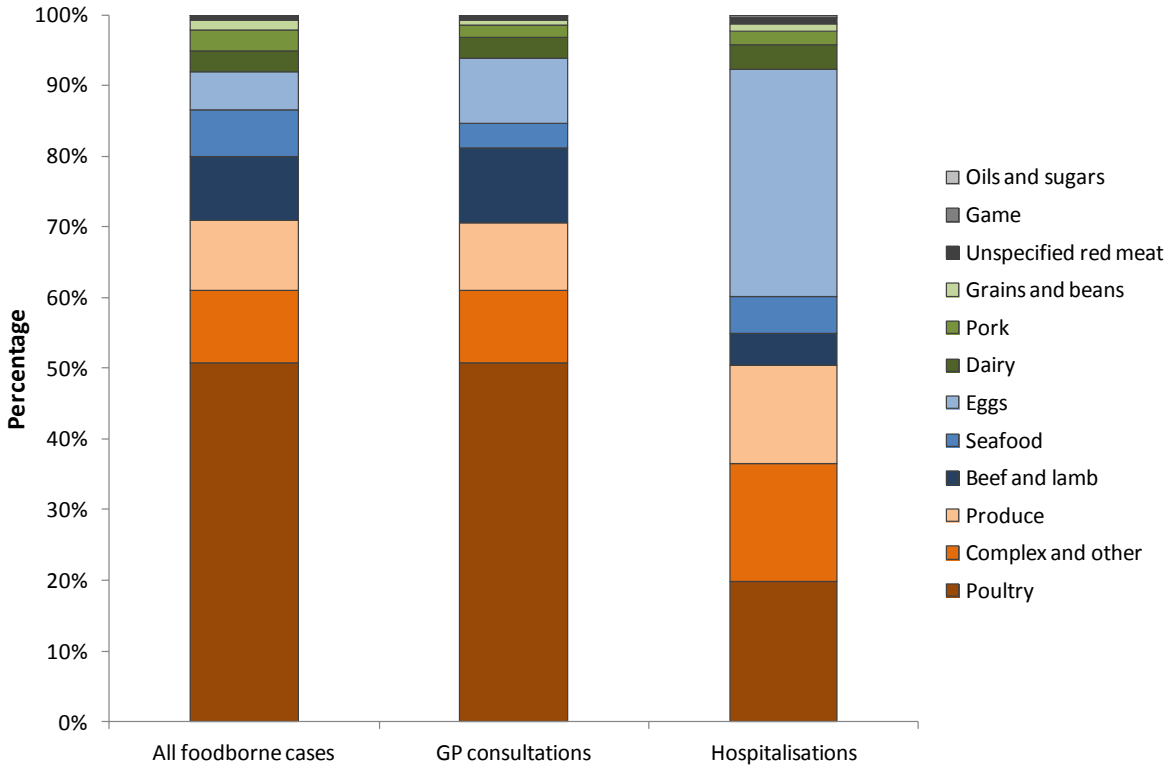
Food commodity	Mean	Median	(95% CrI)	% of total
Seafood	241	226	(154-411)	5.2%
Dairy	291	156	(57-1,264)	3.6%
Eggs	1,785	1,400	(373-5,641)	32.1%
Unspecified red meat	66	37	(1-295)	0.8%
Game	35	19	(4-161)	0.4%
Beef and lamb	2,656	194	(49-18,723)	4.4%
Pork	103	79	(50-312)	1.8%
Poultry	937	869	(411-1,863)	19.9%
Grains and beans	60	48	(29-163)	1.1%
Oils and sugars	3	1	(0-12)	0.0%
Produce	888	610	(263-2,941)	14.0%
Complex and other	3,559	723	(233-22,240)	16.6%
Total	10,623	4,361		100.0%

Note: Median values from the model are used to generate % totals

Appendix 8 presents food attribution estimates for the five pathogens *C. perfringens*, *Campylobacter*, *E. coli* O157, *Salmonella* and norovirus using the Bayesian approach, comparing results from literature-based priors with those obtained using a vague prior that effectively assumes that only the outbreak data are informative. For the major food commodities, the two approaches produce similar results. For certain commodities, notably game, grains and beans, and oils and sugars, the approach

using a vague prior gives higher estimates, indicating that although outbreaks due to these commodities are reported, these have generally been found by studies in the literature to be of relatively lower importance.

Figure 6.3: Proportionate contribution of different food commodities to foodborne illness burden, UK 2009



6.5 RATES OF FOODBORNE ILLNESS BY FOOD COMMODITY

Rates of illness, GP consultation and hospital admission by food commodity are shown in Tables 6.3a-c. Rates are presented as the number of cases, GP consultations or hospital admission per 1,000 persons per year, based on the average annual consumption of the different food commodities in the UK. For each food commodity, the rate ratios relative to the food commodity “grains and beans” are also shown.

Table 6.3a: Estimated rates of foodborne illness in the community by food commodity, UK 2009

	Community			
Food Commodity	**Rate[1]**	**(95% CrI)**	**RR**	**(95% CrI)**
Seafood	0.54	(0.42-0.72)	5.0	(3.4-7.2)
Dairy	0.25	(0.13-0.70)	2.3	(1.1-6.8)
Eggs	0.40	(0.16-1.71)	3.7	(1.4-15.1)
Red meat products	1.61	(0.90-3.33)	15.0	(7.5-31.5)
Beef and lamb	1.11	(0.56-2.77)	10.3	(4.8-25.6)
Pork	0.24	(0.16-0.37)	2.2	(1.4-3.8)
Poultry	4.22	(2.75-6.58)	38.8	(22.7-68.5)
Grains and beans	0.11	(0.08-0.16)	1.0	--
Oils and sugars	0.00	(0.00-0.04)	--	--
Produce	0.82	(0.56-1.29)	7.6	(4.5-12.9)

[1]Rates are expressed as cases per 1,000 persons per year, based on the average annual per capita consumption of each food commodity

Table 6.3b: Estimated rates of foodborne illness presenting to GP by food commodity, UK 2009

	Presenting to GP			
Food Commodity	**Rate[1]**	**(95% CrI)**	**RR**	**(95% CrI)**
Seafood	0.04	(0.02-0.05)	4.9	(3.0-7.6)
Dairy	0.03	(0.02-0.08)	4.2	(1.9-11.9)
Eggs	0.11	(0.05-0.26)	14.7	(5.6-34.8)
Red meat products	0.14	(0.07-0.27)	18.9	(8.3-38.3)
Beef and lamb	0.09	(0.05-0.21)	12.7	(5.6-29.2)
Pork	0.02	(0.01-0.03)	2.2	(1.1-4.6)
Poultry	0.48	(0.32-0.70)	64.7	(35.6-116.3)
Grains and beans	0.01	(0.00-0.01)	1.0	--
Oils and sugars	0.00	(0.00-0.00)	--	--
Produce	0.10	(0.06-0.14)	13.1	(7.1-22.6)

[1]Rates are expressed as cases per 1,000 persons per year, based on the average annual per capita consumption of each food commodity

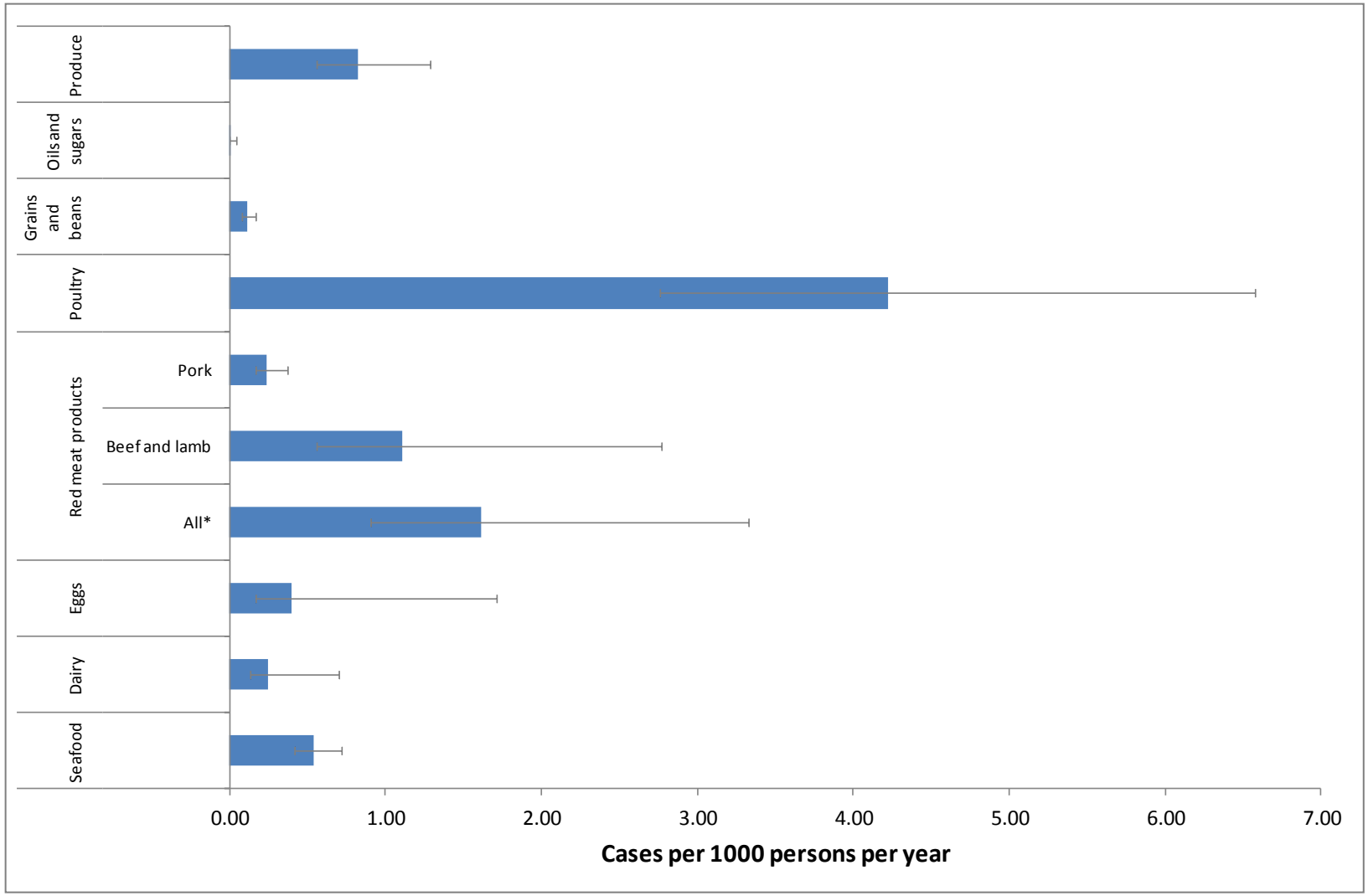
Table 6.3c: Estimated rates of foodborne illness resulting in hospital admission by food commodity, UK 2009

	Hospitalisations			
Food Commodity	**Rate[1]**	**(95% CrI)**	**RR**	**(95% CrI)**
Seafood	0.004	(0.002-0.008)	4.7	(1.8-7.3)
Dairy	0.004	(0.001-0.019)	4.2	(1.8-17.0)
Eggs	0.020	(0.004-0.119)	25.7	(4.0-87.8)
Red meat products	0.025	(0.006-0.283)	30.0	(8.1-276.9)
Beef and lamb	0.023	(0.004-0.274)	26.7	(5.5-267.0)
Pork	0.001	(0.001-0.005)	1.8	(0.9-3.7)
Poultry	0.015	(0.007-0.036)	17.9	(5.2-35.8)
Grains and beans	0.001	(0.000-0.003)	1.0	--
Oils and sugars	0.000	(0.000-0.000)	--	--
Produce	0.012	(0.005-0.049)	14.1	(5.3-50.7)

[1]Rates are expressed as cases per 1,000 persons per year, based on the average annual per capita consumption of each food commodity

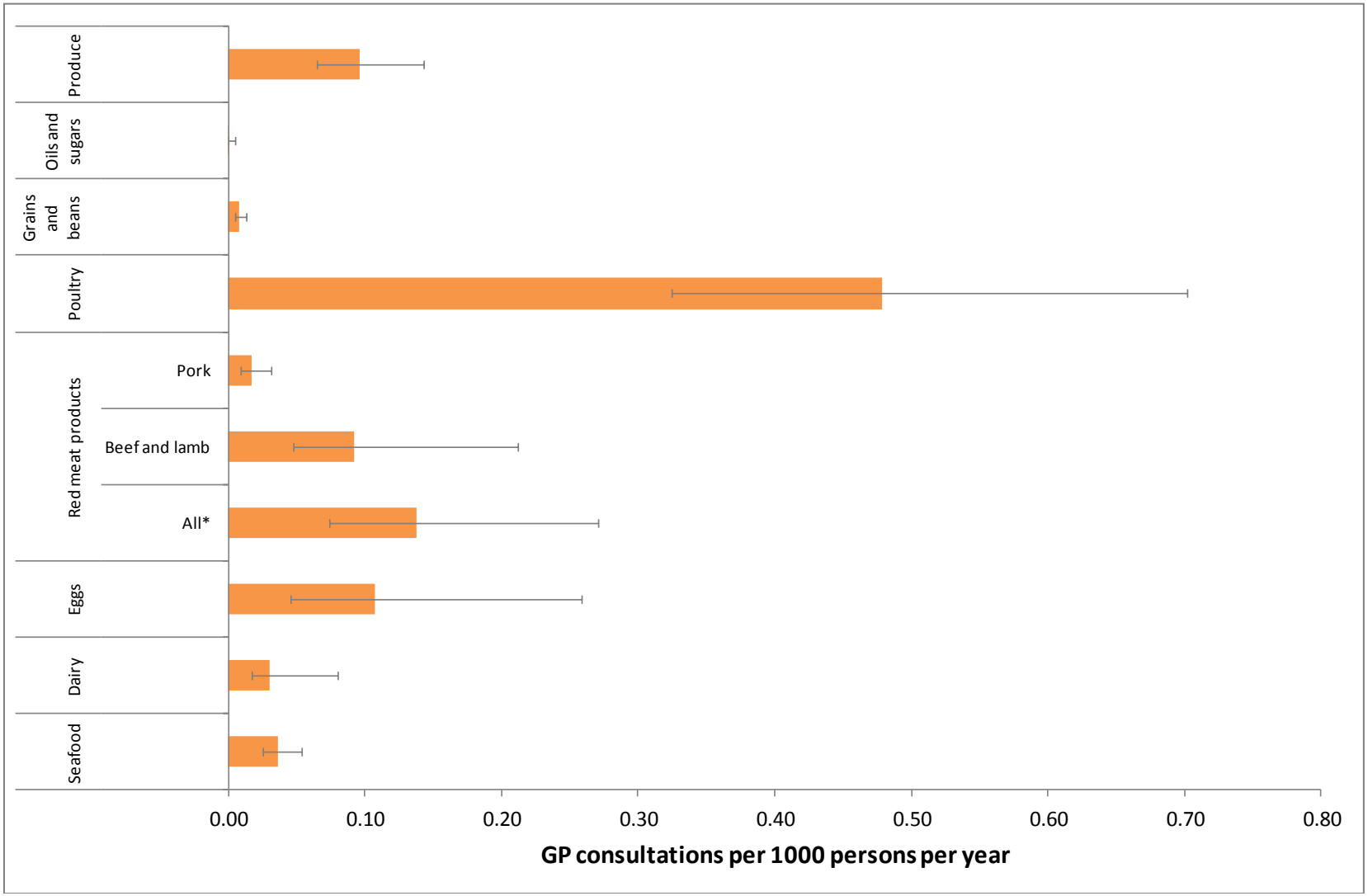
In terms of cases and GP consultations, by far the highest rate of illness was related to poultry consumption (Figure 6.4). We estimate that every year approximately 4 in every 1,000 people acquire food-related illness (4.22 per 1,000 per year, 95% CrI: 2.75 – 6.58) and 5 in 10,000 consult their GP for IID-related conditions (0.48 per 1,000 per year, 95% CrI: 0.32 – 0.70) as a result of poultry consumption. A person with average patterns of consumption is nearly 40 times more likely to acquire foodborne illness from poultry consumption than from consumption of grains and beans (RR = 38.8, 95% CrI: 22.7 – 68.5). Red meat products, eggs, produce and seafood were associated with lower rates of illness and GP consultation compared with poultry (Tables 6.3a and 6.3b). However, rates of hospital admission associated with consumption of eggs and red meat products were higher than those for poultry, although there was substantial overlap in 95% CrIs for hospitalisation rates (Table 6.3c).

Figure 6.4a: Estimated rates of foodborne illness per 1,000 persons per year by food commodity - Cases (Error bars show 95% CrI)



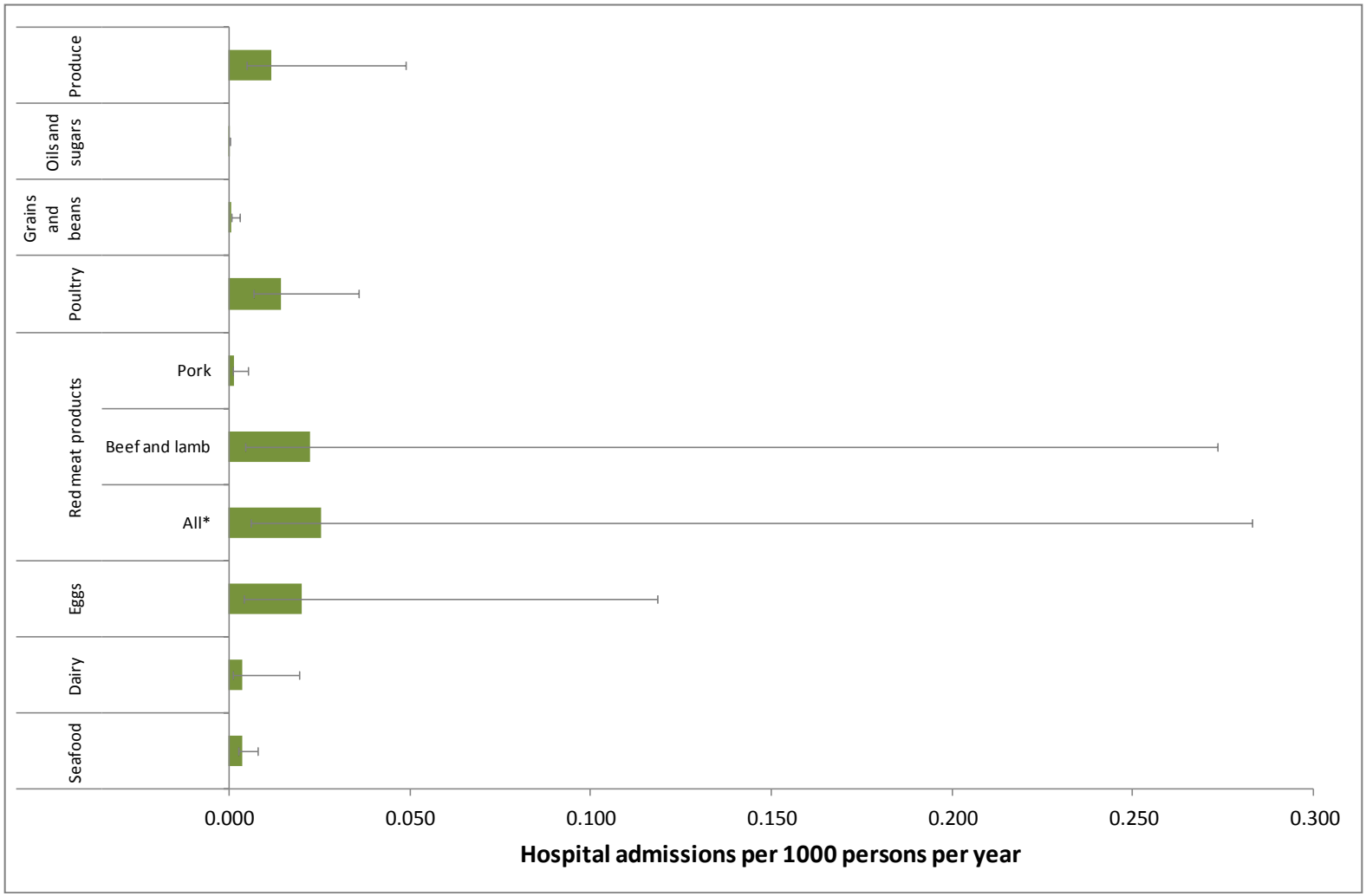
*includes red meat products not otherwise assigned to beef, lamb or pork, e.g. sausages, pies, burgers

Figure 6.4b: Estimated rates of foodborne illness per 1,000 persons per year by food commodity – GP Presentations (Error bars show 95% CrI)



*includes red meat products not otherwise assigned to beef, lamb or pork, e.g. sausages, pies, burgers

Figure 6.4c: Estimated rates of foodborne illness per 1,000 persons per year by food commodity - Hospital admissions (Error bars show 95% CrI)



*includes red meat products not otherwise assigned to beef, lamb or pork, e.g. sausages, pies, burgers

CHAPTER 7

DISCUSSION

7.1 SUMMARY OF MAIN FINDINGS

Campylobacter remains the most common foodborne pathogen in the UK, accounting for approximately 280,000 cases of foodborne illness and 40,000 food-related GP consultations. Despite this, *Campylobacter* is responsible for a small proportion of hospital admissions, reflecting a generally lower level of disease severity compared with other bacterial pathogens. Other common foodborne pathogens include *C. perfringens*, norovirus and *Salmonella*. *Salmonella* accounted for approximately 2,500 hospital admissions, the largest number of any single organism and reflecting the relatively high hospitalisation rate as estimated from outbreak data and the IID1 study. It should be noted, however, that uncertainty around these hospitalisation estimates was large. Viral agents, while being common causes of IID, ranked lower as causes of foodborne illness, particularly where healthcare contact was involved.

An unexpected finding is that estimates of hospitalisations due to *E. coli* O157 were higher than estimated GP consultations for this pathogen, which was not the case for any of the other pathogens investigated. Two possible explanations for the smaller number of *E. coli* O157 GP consultations compared with hospitalisations are that hospitalisation rates tend to be higher in outbreaks (either because outbreaks tend to be associated with more severe illness, or they affect younger age groups in whom hospitalisation is more common), or that this pathogen in general tends to cause more severe illness, leading patients to seek treatment in hospitals directly, without necessarily first consulting their GP. It should be noted, however, that estimates for *E. coli* O157 are based on very sparse data, because this pathogen was rarely found in the IID2 study; only one case was identified in each of the cohort and GP presentation components. As a result, there is a very high level of uncertainty as indicated by the width of the credible intervals from all models, and estimates for this pathogen should be interpreted with extreme caution.

Poultry is the most common source of foodborne illness, accounting for approximately 250,000 cases, 34,000 GP consultations and 850 hospital

admissions. Approximately 50% of all cases and GP consultations, and 20% of hospital admissions for foodborne illness are attributable to poultry contamination. A person with typical patterns of consumption is nearly 40 times more likely to acquire foodborne illness through contaminated poultry than through grains and beans, representing a considerably higher risk compared with other food commodities. Eggs, a well-documented vehicle for *Salmonella* infection, account for fewer cases, but are associated with greater disease severity; egg-related infections accounted for only 5% of cases of foodborne illness, but more than 30% of hospital admissions. Other important food vehicles included beef and lamb, seafood and produce.

7.2 COMPARISON WITH OTHER STUDIES

Our study updates estimates calculated by Adak *et al*. (2002) for England and Wales for the period 1992-2000. Our method expands upon that of Adak *et al*. (2002) by taking into account uncertainty in the estimates. Due to differences in the estimation methods, the two sets of estimates are not directly comparable.

Adak *et al*. (2002) provided estimates for a wider range of foodborne pathogens, which have not been included in this analysis because they do not cause symptoms of IID. Excluding these organisms, the five most common foodborne pathogens in 2000 in terms of cases were *Campylobacter*, *C. perfringens*, norovirus, non-typhoidal salmonellas, and astrovirus. *Campylobacter* and *C. perfringens* also accounted for the most GP consultations, followed by non-typhoidal salmonellas and norovirus, while *Campylobacter* and *Salmonella* accounted for almost all hospital admissions. Our analysis largely supports these earlier results, but indicates that *Campylobacter* is a less important cause of food-related hospitalisation, while *Salmonella* and *E. coli* O157 are more important.

Other studies investigating the burden of foodborne illness caused by a wide range of pathogens have been carried out in Australia (Hall *et al.*, 2005) and the United States (Mead *et al*, 1999; Scallan *et al.*, 2011a; Scallan *et al.*, 2011b). A major feature of the studies by Hall *et al* (2005) and Scallan *et al* (2011a) was the prominence of norovirus, which was estimated to be among the top two most common foodborne disease pathogens in Australia and the US. In the US study, it was also the second most common cause of food-related hospital admissions. The

greater prominence of norovirus in those settings is related to the greater importance of foodborne transmission; approximately a quarter of norovirus IID cases in those two studies were attributed to foodborne transmission, whereas our estimate for the UK is less than 5%. A recent study by Phillips *et al*. (2011), which investigated risk factors for norovirus disease diagnosed by quantitative polymerase chain reaction (PCR) methods, identified only shellfish consumption as a food-related risk factor, accounting for approximately 2% of cases. This suggests that foodborne transmission plays only a minor role in the spread of norovirus in the UK. Despite this, the high frequency of norovirus in the community means that this pathogen still accounts for more than 70,000 cases of foodborne illness in the UK each year.

7.3 STRENGTHS AND LIMITATIONS

7.3.1 Estimating the burden of foodborne illness

A major strength of this study is the availability of directly observed, pathogen-specific incidence data from the recently completed IID2 study in the UK (Tam *et al.*, 2012a). Using data from IID2 obviates the need for assumptions about under-ascertainment of disease due to individual organisms in national surveillance and requires fewer assumptions about the rates of healthcare usage among IID patients. Incidence data for *Bacillus*, *Shigella*, *Staph. aureus* and *Yersinia* were not available from the IID2 study, either because these organisms were not included in the study, or because no positive specimens were identified. *Bacillus*, *Staph. aureus* and *Yersinia* were excluded from our analysis because previous data from the IID1 study indicated that these organisms are found with similar frequency among IID cases and asymptomatic controls. This suggests strongly that these organisms are rarely pathogenic and including them in the analysis would grossly overestimate their importance as causes of IID. For *Shigella*, we estimated incidence by multiplying the number of laboratory reports in 2009 by the reporting ratios as estimated in the IID1 study, adjusted for decreases in usage of GP services in the intervening time period. This approach relies on the assumption that reporting ratios for this organism have remained stable since the mid-1990s. Data from the IID2 study indicate that the suitability of this assumption is likely to be pathogen-specific. For example, comparatively fewer cases of salmonellosis in the community are currently reported to national surveillance compared with the 1990s, while the reporting ratio for

Campylobacter IID cases has remained largely unchanged over the same period (Tam *et al.*, 2012b). Given the lack of other data, we believe that our approach provides a reasonable approximation. For *Listeria*, we have no additional data on incidence, and we have based our incidence estimates solely on cases reported to national surveillance. Our estimates of foodborne listeriosis are therefore likely to be an underestimate. Finally, we did not attempt to estimate the burden of illness due to unidentified pathogens since the proportion of illnesses transmitted by food for these illnesses is unknown and may differ from that for known IID pathogens.

Using outbreak data to attribute cases of IID to foodborne transmission relies on certain assumptions. The principal assumption is that outbreak cases reflect the epidemiology in the wider community particularly that the proportion of cases in foodborne outbreaks due to a particular pathogen is similar to that of apparently sporadic cases infected in the same way by the same pathogen. Another potential limitation of using outbreak data is that there might be a bias towards investigation of foodborne outbreaks. However, this does not seem to be the case: there has been a gradual decrease in the proportion of reported outbreaks involving foodborne transmission, which reflects both a reduction in incidence of certain foodborne pathogens, particularly *Salmonella*, and greater investigation of outbreaks in other settings, particularly viral outbreaks in hospitals and residential institutions. Within pathogens, there is also little evidence of a change in the proportion of outbreaks that are foodborne, with the exception of norovirus, for which most outbreaks currently reported involve person-to-person transmission.

We also used outbreak data to estimate hospitalisation rates by pathogen. A potential limitation of this approach is that more severe cases requiring hospitalisation might be more likely to be recorded in outbreak reports, whereas milder cases might be missed. Alternatively, outbreaks with larger numbers of hospitalised cases might be more likely to be investigated. This would tend to overestimate hospitalisation rates. For this reason, our Bayesian models additionally incorporated prior information on hospitalisation rates from the IID1 and IID2 studies. For most pathogens, the two types of model gave similar results. It should be noted, however, that for most organisms, the number of hospitalisations in both sets of data was small, and this is reflected in the large degree of uncertainty in the estimates. For rotavirus and astrovirus, the Bayesian model gave somewhat lower estimates of

hospital admissions, which might indicate that hospitalisation rates for these two pathogens are over-reported in outbreak data. Another possibility is that, for some pathogens, the populations affected in outbreaks might differ in important ways from the general population. For example, outbreaks might occur in specific age groups or people with underlying conditions, in whom disease severity might be different. Outbreak reports, however, do not contain specific information regarding the age groups or populations affected.

We investigated other sources of data on IID-related hospitalisations, such as electronic records of in-patient data. However, we did not find these suitable for this analysis. Although hospital in-patient databases record admissions by International Classification of Diseases (ICD) code, for many pathogens of interest there is no specific ICD code, such as for *E. coli* O157, and it is unclear to what extent hospital admissions for IID-related codes are microbiologically confirmed. In addition, coding of these admissions is sub-optimal, as many of these admissions are classified under non-specific diagnostic codes. Harris *et al*. (2007) employed a regression model to estimate the proportion of hospital admissions for pathogen-specific and non-specific codes attributable to rotavirus in children under five years, using correlations in the seasonal distribution of admissions and laboratory reports. However, this information is not available for a wide range of pathogens across all age groups, and the method is not suitable for pathogens with less marked seasonal patterns.

Our modelling approach allows for use of data from various sources to incorporate the best available information from both UK-specific epidemiological studies and other published sources. This can provide a useful summary of the current state of knowledge and models can be updated as new information becomes available. In our models, information from the literature carried more influence if outbreak data were sparse and we addressed discrepancies between studies in a sensitivity analysis incorporating separate parameters from each study identified. The comparison of models with and without the incorporation of prior information from other studies indicates where there is disagreement between different data sources. In this way, our analysis enables uncertainty in all the relevant parameters to be accounted for. Uncertainty in these models reflects not simply statistical uncertainty in individual parameters, but disagreement between data sources and availability of

information from previous studies. Information from previous studies on the proportion of IID attributable to foodborne transmission was captured using Bayesian uniform priors. This is likely to be a conservative approach, as it presupposes that every value within the specified limits is equally likely. However, for most pathogens, the number of available studies was small and use of more informative priors was not possible. This was particularly true for pathogen-specific risk factor studies, for which very few studies had the necessary information on population attributable fractions for food-related risk factors. The one exception was *Campylobacter*, for which 14 studies had relevant data.

Due to the need to prioritise certain pathogens, it was impossible within the scope of this review to conduct individual literature searches for all pathogens. We may therefore have omitted relevant studies for some pathogens, although we included data for them where available from previous food attribution studies identified in our search. Future work in this area could include more comprehensive reviews for other pathogens, including *Shigella*, the enteric viruses and *Toxoplasma*, among others. We included only English language articles, with the exception of one multi-pathogen study by Van Duynhoven *et al*. (2002), results from which were also reported by Havelaar *et al*. (2008). Although most articles relevant to the UK are likely to be published in English, we excluded one study from Japan with potentially relevant information, as it was reported in a conference abstract and full results were not yet published. To validate our literature search strategy, we compared our search results with those of a recent review of case-control studies of enteric pathogens by Fullerton *et al*. (2012). All relevant case-control studies identified in that study were also captured in our search.

The use of data from risk factor studies, while providing a useful summary of available evidence, presents problems in interpretation. Studies vary widely with respect to the design, methods and risk factors investigated. Consequently, variability between studies in the importance of food-related risk factors is high. The relative importance of different risk factors could also differ between geographical settings.

We could not estimate hospital occupancy, because of a scarcity of reliable data. In the IID1 and IID2 studies, the most comprehensive longitudinal studies of IID in the

UK, hospital occupancy estimates for *Salmonella*, for example, are based on only 3 admissions, while no admissions for VTEC were observed. While data on hospital occupancy are available from electronic in-patient records, these lack specific diagnostic codes for many pathogens, e.g. VTEC and the causative agent is often not specified. For other pathogens, such as *Salmonella*, admissions often present as septicaemia and bacteraemia, and it is impossible to determine whether these are the result of IID or other conditions.

We could not estimate deaths attributable to foodborne illness, due to the lack of reliable data sources on pathogen-specific mortality rates. Death certificates rarely provide information on specific gastrointestinal pathogens involved, while deaths in outbreaks are rare and may not be recorded if they occur sometime after the outbreak investigation is over. More generally, such mortality estimates would be difficult to interpret. Deaths involving enteric and foodborne pathogens are often associated with vulnerable groups that have underlying conditions, and the mortality in these groups may be very different from that in the general population. Where a death occurs, it is difficult to determine the extent to which the foodborne pathogen, rather than an underlying condition, was responsible. In some cases, the pathogen may play a direct role, as is likely to be the case in deaths involving *E. coli* O157, but in others, infection might be merely coincidental. Deaths attributed to foodborne disease are, therefore, not the same as preventable deaths; some deaths might have been precipitated by an episode of foodborne illness, but in many cases death would have occurred even in the absence of an enteric pathogen. The extent to which this is an issue is likely to vary between pathogens, but is currently poorly understood.

Two Scandinavian studies, from Denmark and Sweden, have estimated mortality due to common foodborne bacterial pathogens relative to the general population. These were registry-based studies, in which cases of laboratory-confirmed IID reported to national surveillance were linked to records of all-cause mortality up to one year after occurrence of IID.

In the Danish study, Helms *et al*. (2003) were able to adjust for differences between IID cases and the general population in terms of age and sex distribution, as well as the prevalence of co-morbidities using the Charlson index. After adjusting for these

factors, the authors found higher mortality among cases of IID due to *Salmonella*, *Campylobacter* and *Yersinia* even up to a year after IID occurrence. Most of the excess mortality occurred in the first 30 days after infection. Among those without known co-morbidities, *Campylobacter* IID patients had a two-fold increased risk of mortality in the subsequent 12 months compared with the general population (RR = 2.06, 95% CI: 1.68 – 2.53); the corresponding figure for salmonellosis patients was 2.85 (RR = 2.85, 95% CI: 2.56 – 3.17). The relative mortality was highest for *Salmonella* Dublin, which was associated with a 15-fold higher risk of mortality within one year.

In the Swedish study, Ternhag *et al*. (2005) studied mortality among *Campylobacter* IID cases using standardised mortality ratios (SMR). The SMR compares the mortality observed among IID cases with that which would be expected if IID cases had the same age and sex distribution as the general population. It is thus an estimate of increased mortality that is not accounted for by differences in age and sex. The authors found a higher-than-expected mortality among *Campylobacter* IID patients infected in Sweden up to a year from infection. The highest relative mortality occurred within one month of IID occurrence (SMR = 2.9, 95% CI: 1.9 – 4.0), but there was no excess mortality beyond one year.

The clustering of mortality shortly after bacterial infection with gradually decreasing relative mortality up to one year from infection strongly indicates a frailty effect, whereby the most vulnerable patients die soon after infection, while those who survive had much lower risk of death to begin with. Further evidence for this phenomenon is suggested by the Ternhag study, in which *Campylobacter* IID patients infected abroad had generally much lower risks of death than the general population. This is compatible with a “healthy traveller” effect, as those fit enough to travel are likely be healthier and have lower mortality than the general population (Ternhag *et al*., 2005).

Assuming that the mortality estimates from Norway are applicable to the UK, applying the mortality rates and attributable mortality to the number of laboratory-confirmed *Salmonella* and *Campylobacter* infections reported in the four UK countries in 2009 suggests that approximately 110 deaths due to *Salmonella* and 220 deaths due to *Campylobacter* IID would have occurred. If 90% of salmonellosis

and 50% of campylobacteriosis is foodborne, this suggests that each of these pathogens is responsible for approximately 100 food-related deaths per year.

The two Scandinavian studies provide some of the most robust data on mortality from bacterial IID available in the literature. However, the application of mortality estimates from other countries to the UK is highly problematic, for several reasons. The Scandinavian studies estimated mortality among cases of salmonellosis and campylobacteriosis reported to national surveillance and these mortality estimates cannot be generalised to all cases in the community. In addition, applying mortality estimates from Denmark to the UK makes a strong assumption that the reporting systems in the two settings are comparable; reported cases in the UK and Denmark may differ in important ways, because of differences in health-seeking behaviour or in reporting practices. Lastly, the populations of the UK and Denmark might differ in crucial ways that affect mortality risk. This includes factors such as the age and sex distribution of the population, but also the distribution of co-morbidities and the mortality associated with such co-morbidities. It is therefore unlikely that IID mortality estimates from other countries are directly applicable to the UK and great caution should be taken in interpreting such analyses. Ultimately, robust estimates of IID-related mortality in the UK will require specific studies in the UK and/or the development of methods using UK-specific routinely collected data.

Our estimates of foodborne disease measure burden only in the acute phase of illness. For some pathogens, the long-term consequences of illness can add considerably to their burden, as is the case with VTEC-associated haemolytic uraemic syndrome (HUS) and *Campylobacter*-associated Guillain-Barré syndrome (GBS). Moreover, our estimates are based only on the number of cases of illness, and take no account of the consequences of illness in different sectors of the population. For example, VTEC O157 infection in young children is considerably more costly, both economically and in terms of quality of life, because of the long-term consequences of HUS. Further studies using additional measures of disease burden and taking into account long-term health consequences are therefore required.

7.3.2 Estimating the burden of foodborne illness by food commodity

We defined as foodborne any outbreak in which food was implicated in transmission, regardless of whether a specific vehicle had been incriminated. In classifying outbreaks, three individual reviewers were asked to assign the outbreak to the most likely food commodity, based on the information available. For some outbreaks, several candidate foods might have been implicated, and reviewers were asked to apply their expertise, using the available information, to attribute the outbreak to the most likely food commodity category. This enabled us to capture uncertainty in the categorisation. Where insufficient evidence was available, reviewers could assign outbreaks to a category for "complex and other foods".

We estimated rates of foodborne illness by food commodity. Using rates has the advantage that it accounts for differences in consumption patterns of different commodities. We have chosen to express rates according to the average annual per capita consumption of each food commodity as estimated by the 2008-09 National Diet and Nutrition Survey. This approach is more readily interpretable than rates based on units of consumption, as it requires no assumptions about serving sizes for different types of commodities. For the UK, an average annual pattern of consumption comprises 11.7kg of fish and shellfish, 72.6kg of dairy, 6.4kg of eggs, 21.4kg of beef and lamb, 9.2kg of pork, 11.5kg of other red meat products, 23.4kg of poultry, 79.1kg of grains and beans, 11.5kg of oils and sugars, and 114.5kg of produce (per capita consumption of complex foods is not available). It should be noted, however, that such average consumption represents total consumption divided by the population size, and may not represent any given person's consumption patterns or even a typical pattern of consumption.

Our modelling approach is novel in incorporating both data from outbreaks, as previously done by Adak *et al*. (2002), with food attribution estimates from previous studies for the estimation of the proportion of foodborne illness attributable to different commodities. This approach maximises the available information, and the use of published data is useful for informing estimates where data from outbreaks or other sources are not available. Indeed, in our analysis, outbreak data were only available for five pathogens – *C. perfringens*, *Campylobacter*, *E. coli* O157, *Salmonella* and norovirus – and the number of outbreaks for each combination of

pathogen and food commodity was small. This reflects the small number of foodborne outbreaks currently reported to national surveillance. This recent decline is partly due to changes in reporting mechanisms and partly due to the introduction of layer flock vaccination against *Salmonella* Enteritidis phage type 4, which has had a dramatic impact on the reporting of outbreaks due to this pathogen (Gormley *et al.*, 2011). Given the declining trend in foodborne outbreaks, it is likely that this will become a less useful data source for food attribution analyses in future.

The use of published food attribution studies also has the advantage of helping to summarise the information that is currently available and highlight variation in estimates between studies that may warrant further investigation. This is particularly true for the role of eggs in salmonellosis and poultry in *Campylobacter* transmission, key food commodities for important pathogens for which it is important to obtain more precise estimates. These studies come from a variety of settings in Europe and North America and comprise approaches based on analysis of outbreak data, expert elicitation and molecular typing. Of necessity, we have used studies from other countries that we deemed comparable to the UK in terms of the epidemiology of foodborne diseases, as the only previous studies from the UK were those by Adak *et al*. (2002 and 2005). It is possible that the contribution of food and specific food commodities to transmission of different pathogens differs between countries, because of differences in levels of contamination, consumption or control measures. However, we saw no evidence from published studies of systematic differences in food attribution estimates between countries, with the exception of norovirus, for which estimates from the United States were consistently higher than those from Europe. Studies also differ slightly in the way in which different foods are grouped into food commodities, with red meat products being a particular problem. It is thus unclear whether these differences reflect real differences between settings, differences in the estimation approach or differences in opinion between different groups of experts. Alternatively, differences in the contribution of different food commodities could reflect differences in consumption patterns between settings. Any future international comparison of foodborne disease burden would benefit from an investigation of consumption patterns in different countries. Presenting disease burden as rates by food commodity should enable more meaningful comparison between countries. In addition, it should be noted that for some pathogens, notably

for rotavirus and *Giardia*, information was available from one study. Food commodity attribution estimates for these pathogens should therefore be interpreted with extreme caution as they do not fully account for the low amount of information available.

One complication of our approach is the lack of a straightforward summary of estimates based on priors from different published studies. The variability between studies in estimates and estimation approaches means that there is no clear way to weight and summarise their results in a manner analogous to a meta-analysis so as to obtain an average prior distribution. We have opted instead to combine the posterior distributions across models and provide summaries of their combined density. Although this approach is unconventional and results in complex posterior distributions in some cases, we believe it has a meaningful interpretation, in that it captures overall level of uncertainty and enables the reader to identify pathogen-food commodity combinations for which information is variable between studies and better information is required. Summarising complex posterior distributions is problematic, however, since no single point estimate may be a good summary of the data. We have presented the median and the limits of the central 95% of the posterior distributions, as the median still has a valid statistical interpretation, even if it is not the most common, or even a typical, value.

For most food commodities, there was a high degree of uncertainty and estimates should be interpreted with extreme caution. This is particularly true for the hospital admission estimates. The models incorporate data from a range of data sources that were not collected for this purpose, and account for both statistical uncertainty and uncertainty in terms of the current knowledge regarding the role of different food commodities in transmission of foodborne pathogens; uncertainty is compounded in more complex models with a greater number of parameters.

A further limitation of our analysis is that we were unable to distinguish between illness resulting from direct consumption of foods and that resulting from subsequent person-to-person spread. In some instances, a particular food may serve as the source of infection, but not necessarily the vehicle, as is the case, for example, with cross-contamination from poultry to other foods with *Campylobacter* and *Salmonella*. In other situations, a food may be the vehicle of infection but not the source, such as

in instances of food contamination by infected food handlers. This could explain the relatively high contribution of poultry to norovirus transmission as estimated from our outbreak data; it is possible that these outbreaks resulted from contamination of poultry products by infected food handlers, rather than poultry serving as the source of infection. Although information on infected food handlers may be collected in outbreak reports, only a minority of outbreaks had such information and in most the evidence implicating a food handler was weak. Of the eight published food attribution studies we identified, only one included a category for the contribution of infected food handlers to foodborne illness; we excluded this category from our analyses to make estimates more consistent between studies.

7.4 CONCLUSIONS

Campylobacter remains the most common foodborne pathogen in the UK. Other common foodborne pathogens include *C. perfringens*, norovirus and *Salmonella*.

Contaminated poultry is the most common contributor to foodborne illness but other important food vehicles included eggs, beef and lamb, seafood and produce.

7.5 RECOMMENDATIONS

7.5.1 Recommendations for future research

- Further work is needed to obtain better estimates of hospitalisation, including length of hospital stay, and deaths from foodborne disease in the UK. This could draw on methods currently being employed by the WHO Foodborne Disease Epidemiology Reference Group study. However, it should be noted that, for the majority of pathogens, deaths are associated with vulnerable patients and other underlying diseases.
- Future work should include estimates of disease burden (e.g. DALYS) and costs to help prioritise food safety policy measures. These should take into account the long term sequelae which, for many foodborne pathogens, outweigh the acute disease burden.
- Better data are needed to be able to attribute illness to foods and to perform food commodity attribution. Alternatives to outbreak data, which are declining, are expert elicitation in the UK context, case-control studies of sporadic illness and

molecular subtyping. Generating alternative methods for future use could be undertaken in an international context.

- The use of more complex approaches than uniform distributions for modelling the proportion of foodborne illness could be explored.
- Further work is also needed to explore differences in outbreak-associated versus sporadic foodborne illness so that these can be qualitatively or quantitatively incorporated into future models.
- Additional work is required to generate adjusted attribution estimates for the total UK population to accommodate differences among population subgroups, because pathogen incidence is not uniform across age/gender groups and these groups comprise varying proportions of the total population.
- Future work should attempt to determine the extent to which illness follows consumption of foods in which primary contamination has not been effectively dealt with versus consumption of foods that have been cross-contaminated or contaminated by infected food-handlers.
- Estimates of foodborne disease associated with specific food groups could be reviewed in the light of evidence from food surveys.

7.5.2 Recommendations for Policy

- Given the burden of illness, there needs to be a continued focus on reducing foodborne illness by *Campylobacter* and *Salmonella*.
- Although *C. perfringens* outbreak reports to national surveillance have been declining it is clear from these analyses that *C. perfringens* continues to cause a considerable illness burden and so its control is an important policy issue.
- Contamination of eggs, produce and red meat are also important policy issues given their contribution to foodborne disease.

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APPENDICES

Appendix 1 – Summary of Results from Literature Review

Results: Literature Search

Campylobacter

Six-hundred and thirty references were identified in MEDLINE, 882 in EMBASE, 914 in Web of Science, and 40 in FoodBase (including REMIND projects). In total, 1,443 unique articles remained after duplications were removed. Initial screening of references produced 75 potentially relevant references for which full papers were obtained.

Search terms: *Campylobacter**/or *Campylobacter coli*/ or *Campylobacter jejuni*

E. coli O157

Three-thousand five-hundred and eighty references were identified in MEDLINE, 4,822 in EMBASE, 5,332 in Web of Science, and 43 in FoodBase (including REMIND projects). In total, 8,207 unique articles remained after duplications were removed. Initial screening of references produced 40 potentially relevant references for which full papers were obtained.

Search term: *Escherichia coli**/ or *Escherichia coli* O157

Salmonella

One-thousand four-hundred and ninety one references were identified in MEDLINE, 147 in EMBASE, 1,922 in Web of Science, and 32 in FoodBase (including REMIND projects). In total, 2,509 unique articles remained after duplications were removed. Initial screening of references produced 38 potentially relevant references for which full papers were obtained.

Search terms: Salmonell*/ or *Salmonella* enteritidis/ or *Salmonella* enteric/ or *Salmonella* food poisoning

Listeria

Three-hundred and seventy six references were identified in MEDLINE, 517 in EMBASE, 663 in Web of Science, and 17 in FoodBase (including REMIND projects). In total, 937 unique articles remained after duplications were removed. Initial

screening of references produced 12 potentially relevant references for which full papers were obtained.

Search terms: *Listeria*/* or *Listeria*/ or *Listeria monocytogenes*

Norovirus

Three-hundred and seventy seven references were identified in MEDLINE, 443 in EMBASE, 349 in Web of Science, and 4 in FoodBase (including REMIND projects). In total, 574 unique articles remained after duplications were removed. Initial screening of references produced 9 potentially relevant references for which full papers were obtained.

Search terms: Norovirus/ or exp Norovirus/

C. perfringens

Ninety-seven references were identified in MEDLINE, 172 in EMBASE, 187 in Web of Science, and 5 in FoodBase (including REMIND projects). In total, 277 unique articles remained after duplications were removed. Initial screening of references produced 3 potentially relevant references for which full papers were obtained.

Search terms: *Clostridium perfringens*/ or *Clostridium perfringens* type A

Cryptosporidium

One-hundred and eighteen references were identified in MEDLINE, 182 in EMBASE, 298 in Web of Science, and 6 in FoodBase (including REMIND projects). In total, 383 unique articles remained after duplications were removed. Initial screening of references produced 6 potentially relevant references for which full papers were obtained.

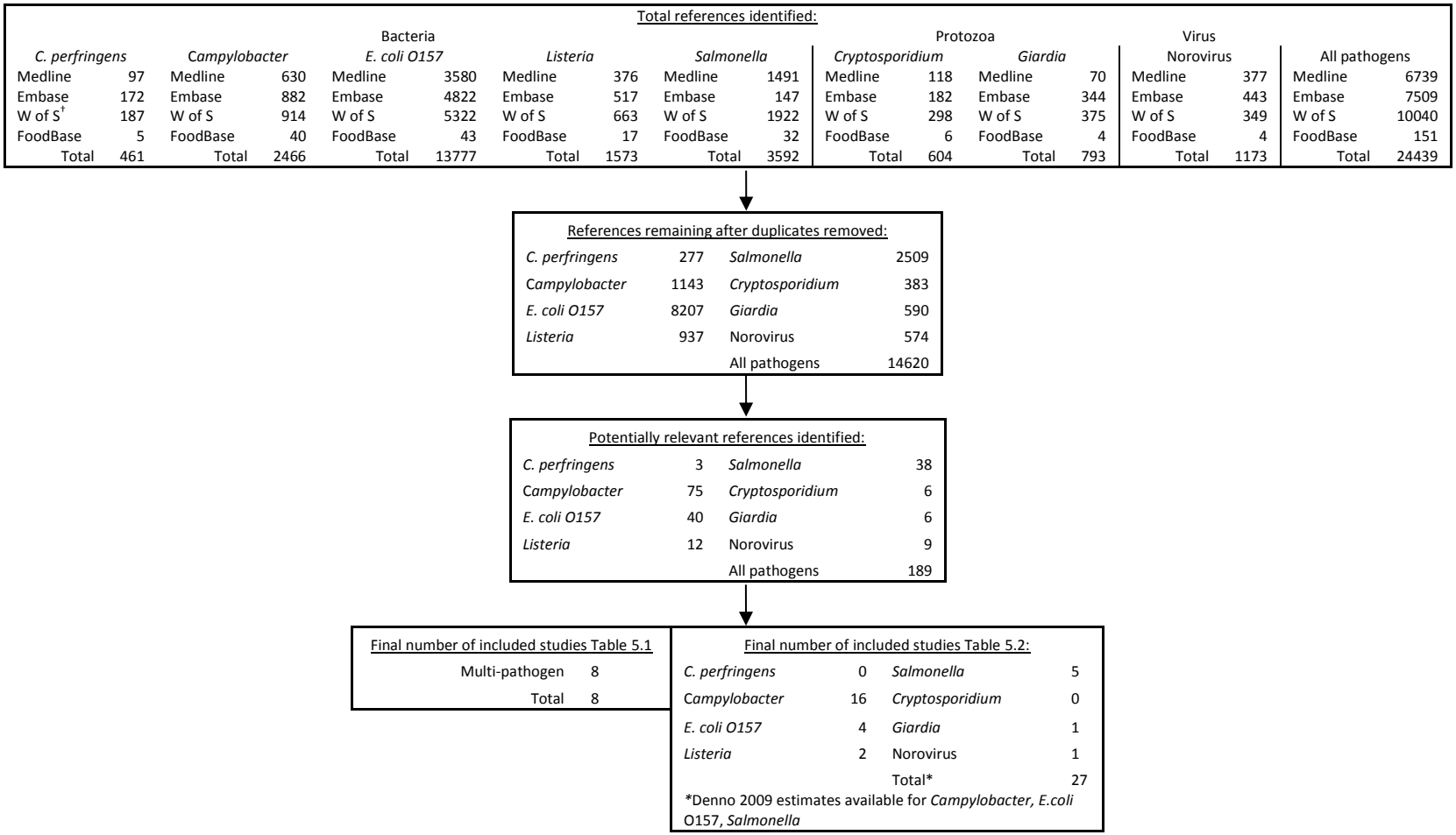
Search term: *Crypto*/* or *Cryptosporidium parvum*

Giardia

Seventy references were identified in MEDLINE, 344 in EMBASE, 375 in Web of Science, and 4 in FoodBase (including REMIND projects). In total, 590 unique articles remained after duplications were removed. Initial screening of references produced 6 potentially relevant references for which full papers were obtained.

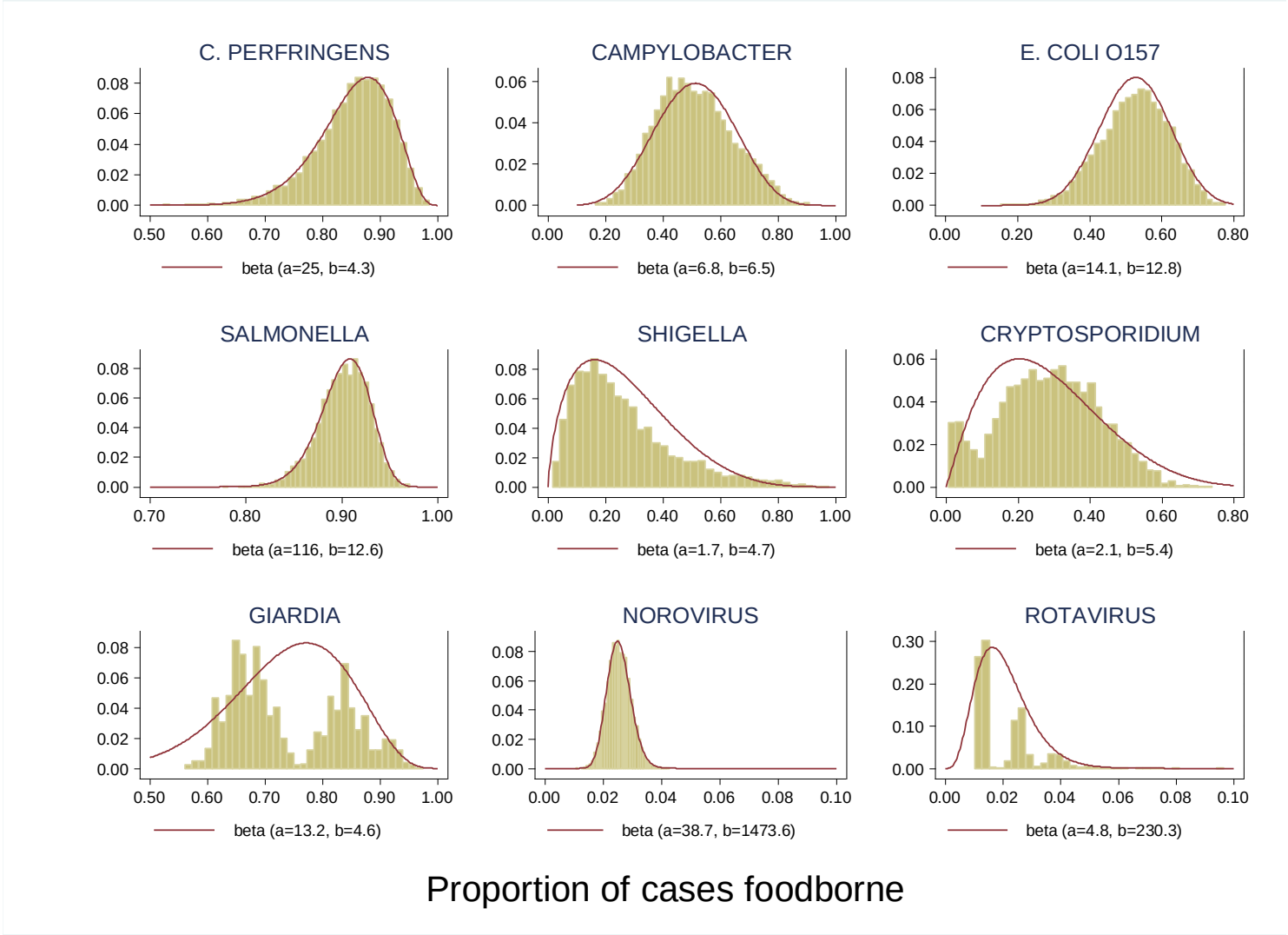
Search terms: *Giardia*/* or Giardiasis/ or *Giardia lamblia*

Flow diagram for locating primary studies of infectious intestinal disease for systematic review



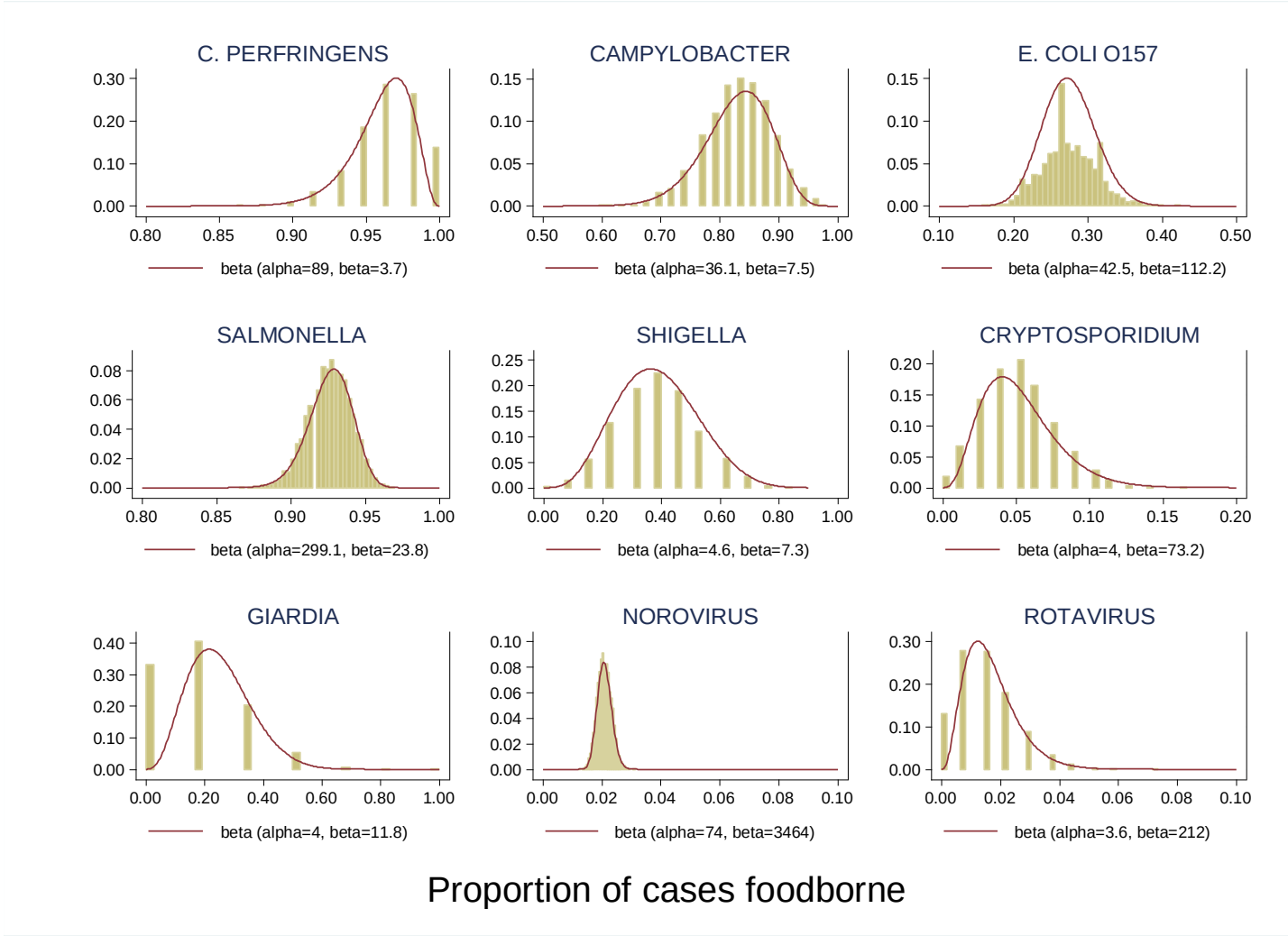
†Web of Science

Appendix 2.1: Bootstrap estimates of the proportion of cases attributable to foodborne transmission with fitted Beta distributions by pathogen, UK outbreak data 2001-08



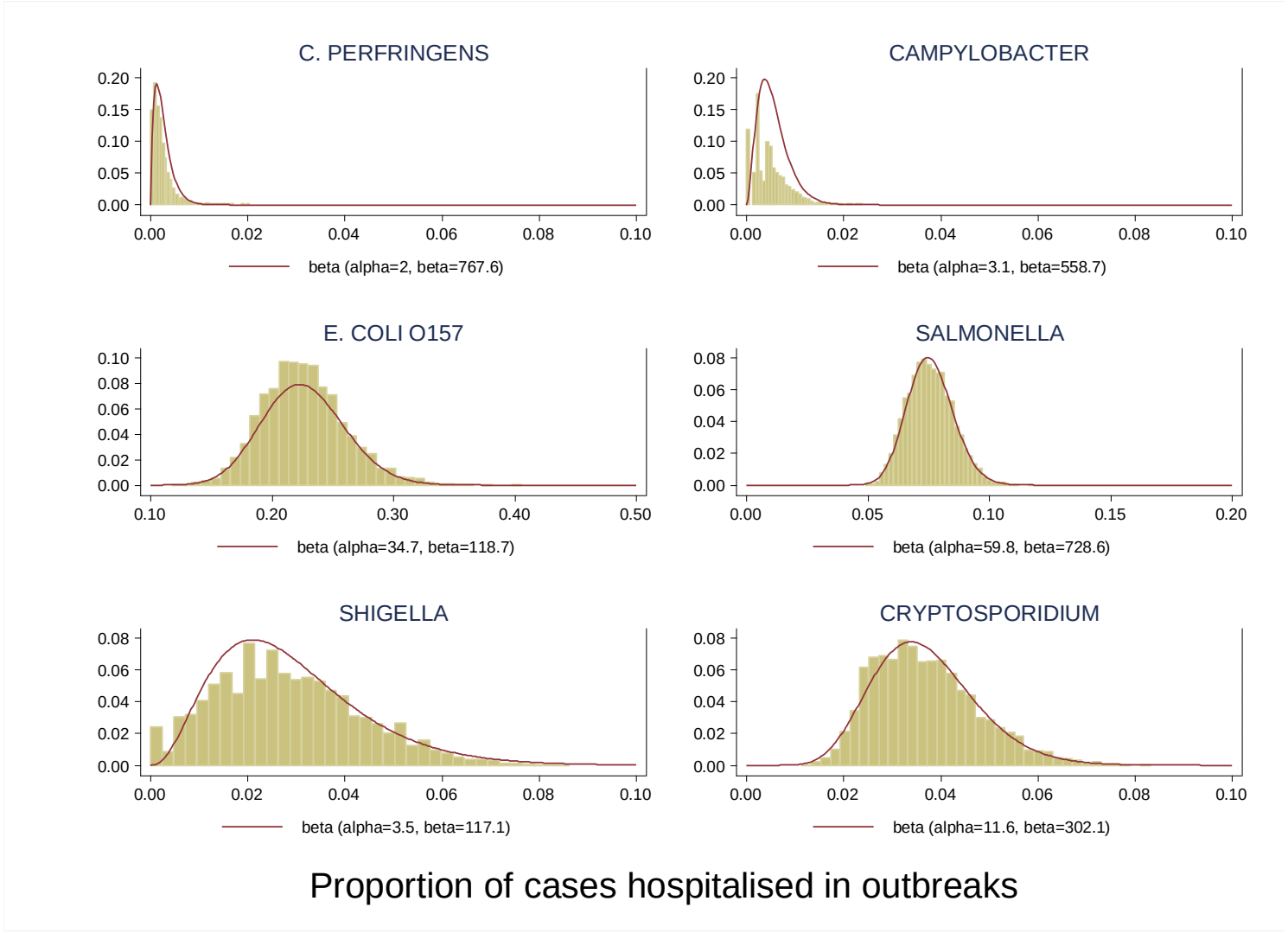
For *Cryptosporidium* and *Giardia*, estimates based on the proportion of outbreaks attributable to foodborne transmission were used in the attribution models (see Appendix 2.2). For astrovirus and sapovirus, no outbreaks involving foodborne transmission were reported

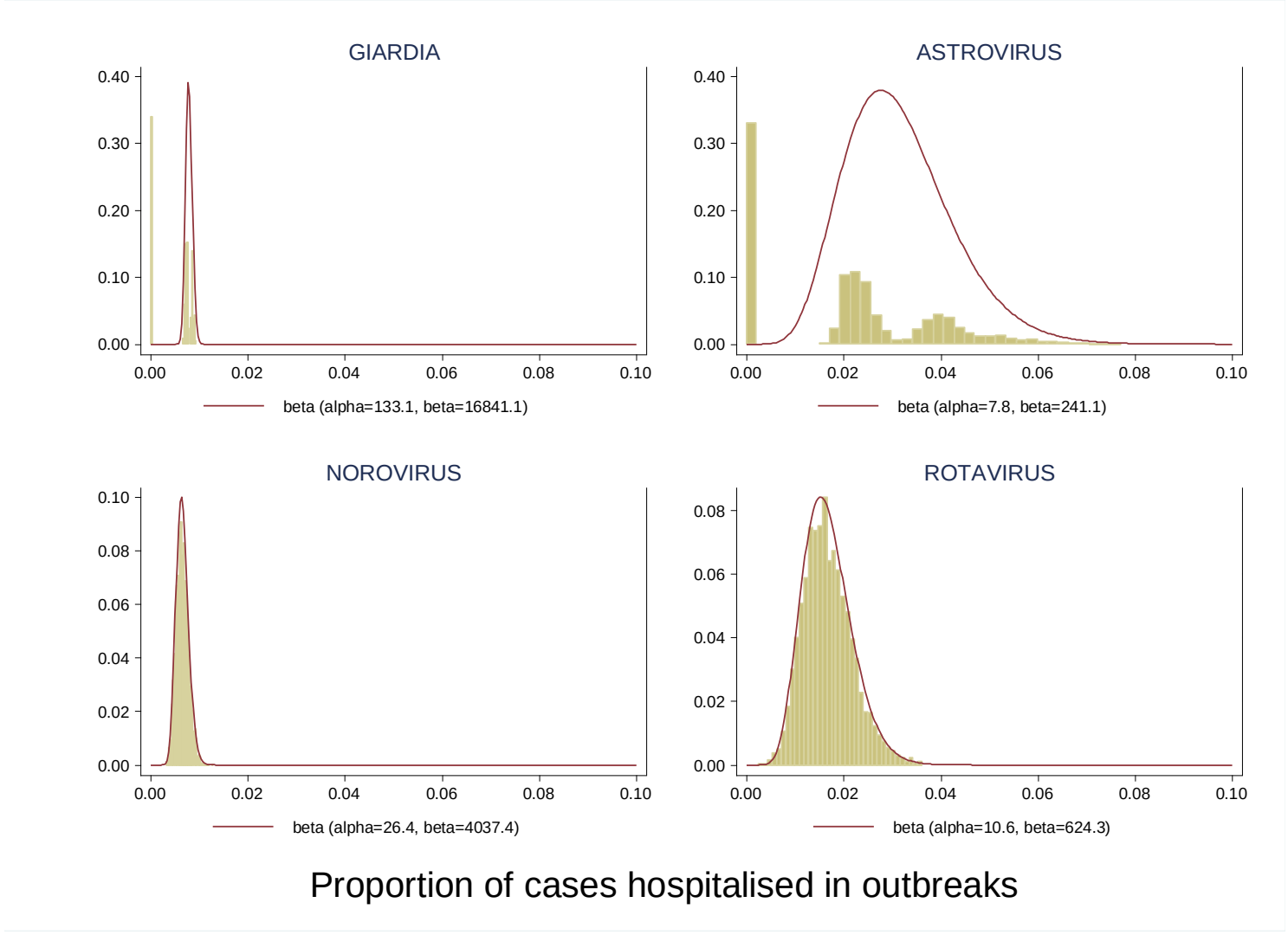
Appendix 2.2: Bootstrap estimates of the proportion of outbreaks attributable to foodborne transmission with fitted Beta distributions by pathogen, UK outbreak data 2001-08



For astrovirus and sapovirus, no outbreaks involving foodborne transmission were reported ..

Appendix 2.3: Bootstrap estimates of the proportion of cases hospitalised with fitted Beta distributions by pathogen, UK outbreak data 2001-08





Proportion of cases hospitalised in outbreaks

Appendix 3.1: Parameters for Model 1

	Incidence					Proportion foodborne				Proportion hospitalised			
Organism	μ_{cp}	σ_{cp}	μ_{gp}	σ_{gp}	**Source**	PF	$a_{\pi p}$	$b_{\pi p}$	**Source**	PH	$a_{\gamma p}$	$b_{\gamma p}$	**Source**
Bacteria													
C. perfringens	-6.50	0.49	-8.34	0.39	A	0.862	25.0	4.3	D	0.0017	2.0	767.6	D
Campylobacter	-4.68	0.22	-6.66	0.18	A	0.501	6.8	6.5	D	0.0046	3.1	558.7	D
E. coli O157 VTEC	-8.11	1.36	-11.51	1.12	A	0.531	14.1	12.8	D	0.2235	34.7	118.7	D
Listeria	--	--	--	--	C	1.000	7.8	3.1	D	--	--	--	H
Salmonella	-7.42	0.71	-8.62	0.46	A	0.904	116.0	12.6	D	0.0751	59.8	728.6	D
Shigella	-9.29	0.97	-9.98	0.27	B	0.222	1.7	4.7	D	0.0260	3.5	117.1	D
Protozoa													
Cryptosporidium	-7.26	0.69	-8.52	0.45	A	0.051	4.0	73.2	D	0.0362	11.6	302.1	D
Giardia	-7.13	0.67	-9.32	0.56	A	0.167	4.0	11.8	D	0.0073	133.1	16,841.1	D
Viruses													
Adenovirus	-4.59	0.21	-7.08	0.28	A	--	4.8	230.3	F	--	10.6	624.3	F
Astrovirus	-5.24	0.29	-7.82	0.37	A	0.000	3.6	437.6	D	0.2222	7.8	241.1	D
Norovirus	-3.06	0.09	-6.18	0.19	A	0.025	38.7	1,473.6	D	0.0064	26.4	4,037.4	D
Rotavirus	-4.37	0.19	-6.60	0.21	A	0.014	4.8	230.3	D	0.0165	10.6	624.3	D
Sapovirus	-3.65	0.13	-6.46	0.19	A	--	38.7	1,473.6	G	--	26.4	4,037.4	G

μ_{cp}: log-transformed (natural logarithm) rate of IID due to pathogen *p*; σ_{cp}: standard error of μ_{cp}; μ_{gp}: log-transformed (natural logarithm) rate of GP consultation due to pathogen *p*; σ_{gp}: standard error of μ_{gp}; $a_{\pi p,}b_{\pi p}$: parameters from a Beta distribution for π_p (the proportion of cases due to pathogen *p* that are attributable to foodborne transmission); $a_{\gamma p}$,$b_{\gamma p}$: parameters for Beta distribution of γ_p (the proportion of cases due to pathogen *p* that are hospitalised)

PF: Proportion foodborne as estimated from outbreak data; PH: Proportion hospitalised as estimated from outbreak data
A: IID2 Study; B: 2009 laboratory reports * IID1 reporting ratio; C: 2009 laboratory reports
D: Outbreak data; F: No outbreak data available, assumed same as rotavirus; G: No outbreak data available, assumed same as norovirus
H: All reported Listeria outbreaks were in hospitals/residential institutions so hospitalisation parameters could not be estimated;

Appendix 3.2: Parameters for Model 2

	Proportion foodborne						Proportion hospitalised					
	Binomial likelihood			Uniform prior			Binomial likelihood			Beta prior		
Organism	f_p	o_p	**Source**	$u_{\pi p}$	$v_{\pi p}$	**Source**	h_p	m_p	**Source**	$a_{\gamma p}$	$b_{\gamma p}$	**Source**
Bacteria												
C. perfringens	1,691	1,964	D	0.761	1.000	Table 1	2	1,120	D	1.6	277.1	J
Campylobacter	373	761	D	0.420	0.800	Table 1	2	424	D	3.5	2,119.3	J
E. coli O157 VTEC	564	1,041	D	0.400	0.760	Table 1	197	877	D	1.0	1.0	K
Listeria	6	8	D	0.690	1.000	Table 1	--	--	H	1.0	1.0	K
Salmonella	7,128	7,892	D	0.550	0.950	Table 1	419	5,527	D	1.2	75.3	J
Shigella	65	310	D	0.082	0.310	Table 1	4	153	D	0.9	7.1	J
Protozoa												
Cryptosporidium	4	65	D	0.000	0.120	Table 1	31	836	D	1.2	99.1	J
Giardia	1	7	D	0.050	0.300	Table 1	1	137	D	1.2	150.4	J
Viruses												
Adenovirus[1]	30	2,338	F	0.000	0.100		20	1,211	F	3.1	1,819.8	J
Astrovirus	2	285	D	0.005	0.107	Table 1	2	88	D	2.5	1,252.6	J
Norovirus	1500	58,855	D	0.000	0.390	Table 1	80	12,333	D	3.2	6,124.2	J
Rotavirus	30	2,338	D	0.005	0.130	Table 1	20	1,211	D	3.6	1,295.6	J
Sapovirus[1]	1500	58,855	G	--	--		80	12,333	G	3.9	3,072.6	J

Incidence parameters are the same as those for Model 1 (see Appendix 3.1); [1] Estimates for these two pathogens could not be calculated from this model because of the lack of published data to inform prior parameters

f_p: Cases involved in foodborne outbreaks; o_p: All cases involved in outbreaks; $u_{\pi p}$,$v_{\pi p}$: Lower and upper bounds of uniform prior distribution for π_p (the proportion of cases due to pathogen *p* attributable to foodborne transmission); h_p: outbreak cases due to pathogen *p* hospitalised; m_p: all outbreak cases; $a_{\gamma p}$,$b_{\gamma p}$: Parameters from Beta prior distribution for γ_p (the proportion of cases due to pathogen *p* that are hospitalised)

D: Outbreak data; E: No outbreak data available, assumed same as *Listeria*; F: No outbreak data available, assumed same as rotavirus; G: No outbreak data available, assumed same as norovirus

H: All reported Listeria outbreaks were in hospitals/residential institutions so hospitalisation parameters could not be estimated; I: No outbreak data, assumed same as *Campylobacter*

J: IID1 and IID2 GP Presentation Studies; K: Non-informative Beta distribution used

Appendix 3.3: Parameters for Model 3

	Proportion foodborne						Proportion hospitalised					
	Binomial likelihood			Uniform prior			Binomial likelihood			Beta prior		
Organism	f_p	o_p	Source	$u_{\pi p}$	$v_{\pi p}$	Source	h_p	m_p	Source	$a_{\gamma p}$	$b_{\gamma p}$	Source
Campylobacter	373	761	D	0.110	1.000	Table 2	2	424	D	3.5	2,119.3	J
E. coli O157 VTEC	564	1,041	D	0.090	0.642	Table 2	197	877	D	1.0	1.0	K
Listeria	6	8	D	0.180	1.000	Table 2	--	--	H	1.0	1.0	K
Salmonella	7,128	7,892	D	0.090	1.000	Table 2	419	5,527	D	1.2	75.3	J

Incidence parameters are the same as those for Model 1 (see Appendix 3.1)

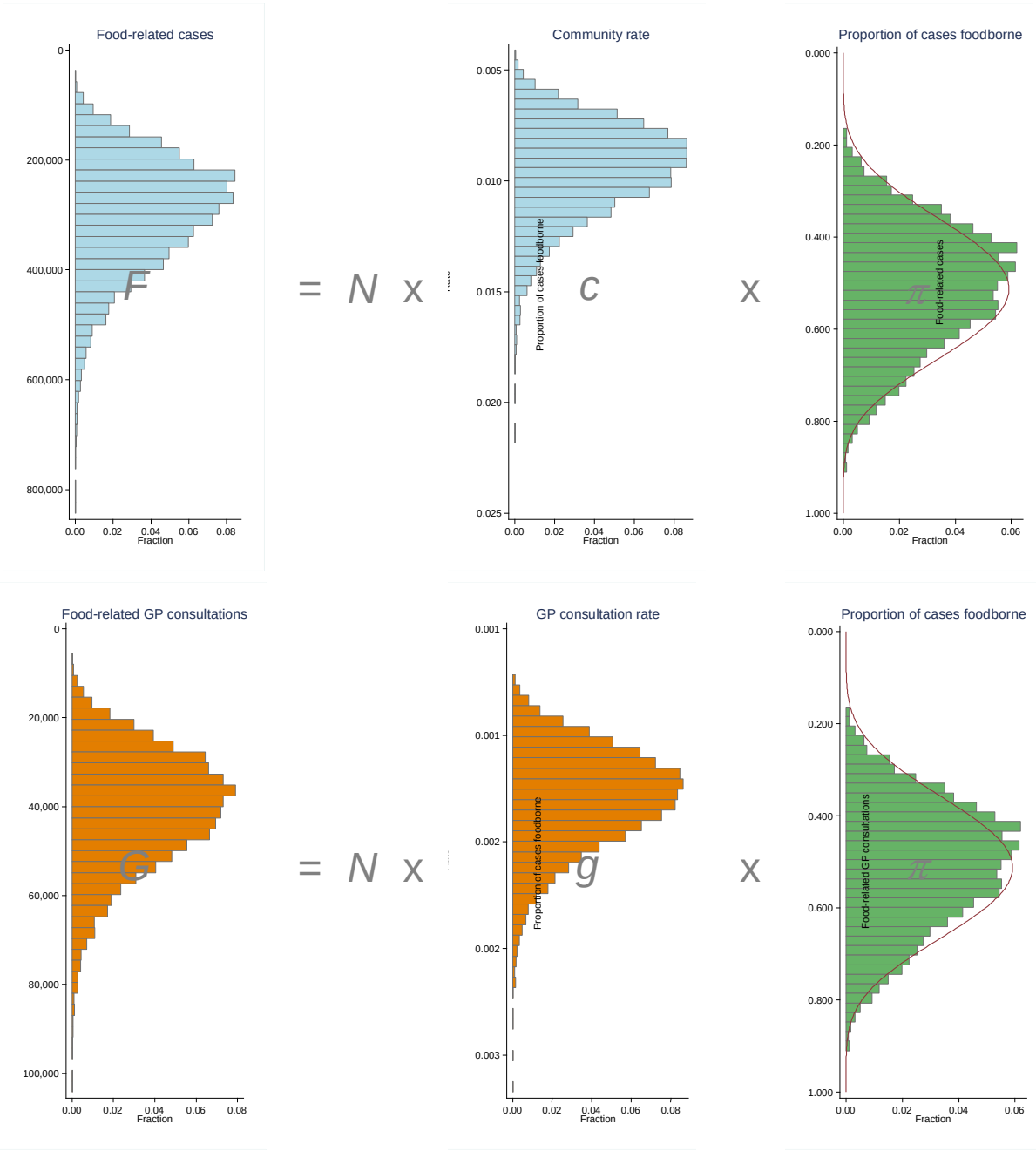
D: Outbreak data

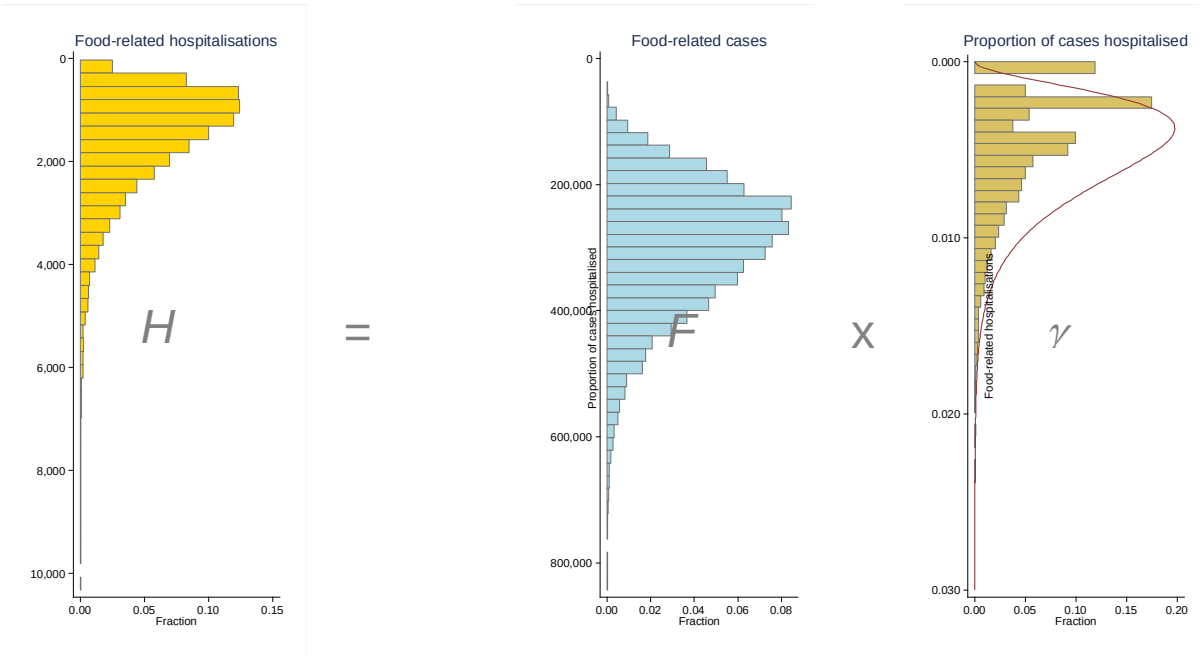
H: All reported Listeria outbreaks were in hospitals/residential institutions so hospitalisation parameters could not be estimated

J: IID1 and IID2 GP Presentation Studies; K: Non-informative Beta distribution used

Appendix 4: Monte Carlo approach using *Campylobacter* as an example

The Monte Carlo simulation approach relies on defining adequate distributions for each of the model parameters. Parameter values from these distributions are then sampled at random and used to calculate outcome values. The outcomes from 100,000 simulations are used to derive expected distributions of the number of cases, GP consultations and hospital admissions attributable to foodborne transmission. The median and central 95% of the resulting distributions represent the point estimates and 95% credible intervals. This approach is illustrated graphically below:





N = UK population estimate for 2009

Appendix 5: Proportion of foodborne illness attributed to specific food commodities (scaled estimates from published food attribution studies)

Table A5a: C. perfringens

Author	Greig	Havelaar
Food Commodity		
Seafood	2.00	6.40
Dairy	0.40	4.20
Eggs	0.10	3.20
Unspecified red meat	4.80	0.10
Game	0.10	0.10
Beef and lamb	38.9	50.9
Pork	6.50	8.50
Poultry	24.1	7.40
Grains and beans	0.10	3.20
Oils and sugars	0.10	0.10
Produce	2.80	7.40
Complex and other	20.1	8.50
Total (%)	100	100

Table A5b: Campylobacter

Author	Davidson	Greig	Hoffman	Pires1*	Pires2*	Havelaar
Food Commodity						
Seafood	0.90	2.60	0.80	3.00	5.80	7.60
Dairy	9.50	34.60	7.80	8.10	13.00	9.80
Eggs	4.90	1.60	2.60	2.00	3.80	3.30
Unspecified red meat	1.50	2.10	0.90	33.0	36.4	0.10
Game	1.90	0.10	2.00	0.10	0.10	0.10
Beef and lamb	7.70	4.70	4.40	0.10	0.10	4.30
Pork	4.80	0.50	4.40	8.60	2.60	5.40
Poultry	61.3	34.5	71.7	43.1	35.8	58.5
Grains and beans	0.10	0.50	0.10	0.40	0.70	2.20
Oils and sugars	0.10	0.10	0.10	0.80	0.90	0.10
Produce	6.30	4.70	5.20	0.60	0.90	5.40
Complex and other	1.10	14.10	0.10	0.10	0.10	3.30
Total (%)	100	100	100	100	100	100

Pires 1 comprises estimates based on the percentage of outbreaks attributed to different food commodities; Pires 2 comprises estimates based on the percentage of cases in outbreaks attributed to different food commodities

Table A5c: E. coli O157

Author	Davidson	Greig	Hoffman	Havelaar
Food Commodity				
Seafood	0.30	0.50	0.10	3.80
Dairy	5.70	10.2	4.10	8.80
Eggs	0.50	0.10	0.10	2.50
Unspecified red meat	2.40	7.20	1.90	0.10
Game	2.60	0.10	3.30	0.10
Beef and lamb	54.5	46.1	69.8	55.5
Pork	1.50	0.50	0.60	7.60
Poultry	0.30	1.40	0.90	3.80
Grains and beans	0.10	1.00	0.10	3.80
Oils and sugars	0.10	0.10	0.10	0.10
Produce	29.2	20.4	18.9	8.80
Complex and other	2.70	12.3	0.10	5.00
Total (%)	100	100	100	100

Table A5d: Listeria

Author	Davidson	Greig	Hoffman	Little	Havelaar
Food Commodity					
Seafood	5.70	11.2	7.10	19.2	19.1
Dairy	25.7	41.3	23.6	2.10	26.5
Eggs	0.10	0.10	0.30	0.10	4.20
Unspecified red meat	48.6	13.1	54.0	3.60	0.10
Game	0.60	0.10	0.30	0.10	0.10
Beef and lamb	2.10	5.70	1.60	15.6	11.7
Pork	2.40	11.2	1.30	5.50	9.50
Poultry	2.30	9.50	2.70	13.5	7.40
Grains and beans	0.10	0.10	0.20	0.10	6.40
Oils and sugars	0.10	0.10	0.10	0.10	0.10
Produce	8.00	1.90	8.70	6.00	8.50
Complex and other	4.30	5.70	0.10	34.1	6.40
Total (%)	100	100	100	100	100

Table A5e: Salmonella spp

Author	Davidson	Greig (SE)*	Greig (Other)*	Hald	Hoffman	Pires1*	Pires2*	Havelaar
Food Commodity								
Seafood	1.60	4.20	2.60	0.10	2.00	2.40	2.80	4.40
Dairy	6.70	6.40	6.30	0.10	7.40	5.40	5.50	7.70
Eggs	19.1	43.4	13.8	62.4	22.1	79.1	79.6	24.1
Unspecified red meat	4.60	0.70	3.50	0.10	1.90	0.10	0.10	0.10
Game	1.50	0.10	0.10	0.10	1.60	0.10	0.10	0.10
Beef and lamb	5.40	5.10	9.70	3.70	11.1	0.70	0.40	14.2
Pork	6.90	1.50	5.70	16.8	5.80	1.80	0.90	15.3
Poultry	33.0	11.6	18.4	16.3	35.6	5.60	6.40	16.4
Grains and beans	2.10	12.1	3.40	0.10	0.30	0.10	0.40	4.40
Oils and sugars	0.10	0.10	0.10	0.10	0.10	1.30	2.20	0.10
Produce	17.0	3.60	21.3	0.10	11.9	3.40	1.50	6.60
Complex and other	1.90	11.2	15.0	0.10	0.10	0.10	0.10	6.60
Total (%)	100	100	100	100	100	100	100	100

*Greig (SE) comprises estimates for *Salmonella* Enteritidis; Greig (Other) comprises estimates for other *Salmonella* types; Pires 1 comprises estimates based on the percentage of of outbreaks attributed to different food commodities; Pires 2 comprises estimates based on the percentage of cases in outbreaks attributed to different food commodities

Table A5f Shigella

Author	Davidson	Greig	Hoffman
Food Commodity			
Seafood	13.7	9.80	8.20
Dairy	7.30	14.8	3.60
Eggs	1.10	0.10	0.90
Unspecified red meat	5.10	0.10	9.80
Game	1.50	0.10	0.80
Beef and lamb	6.50	6.10	3.20
Pork	2.90	2.40	3.20
Poultry	3.10	6.10	5.10
Grains and beans	2.20	0.10	2.00
Oils and sugars	0.10	0.10	0.10
Produce	44.6	29.5	62.8
Complex and other	12.0	30.7	0.10
Total (%)	100	100	100

Table A5g: Cryptosporidium

Author	Davidson	Hoffman	Havelaar
Food Commodity			
Seafood	3.00	8.40	24.1
Dairy	6.40	6.40	9.90
Eggs	0.10	0.30	3.30
Unspecified red meat	0.50	1.50	0.10
Game	4.20	5.90	0.10
Beef and lamb	18.1	8.10	28.5
Pork	3.80	2.20	4.40
Poultry	1.00	1.30	3.30
Grains and beans	0.10	0.30	0.10
Oils and sugars	0.10	0.10	0.10
Produce	47.2	65.3	23.0
Complex and other	15.5	0.10	3.30
Total (%)	100	100	100

Table A5h: *Giardia*

Author	Havelaar
Food Commodity	
Seafood	15.2
Dairy	9.40
Eggs	0.10
Unspecified red meat	0.10
Game	0.10
Beef and lamb	23.4
Pork	5.90
Poultry	3.50
Grains and beans	0.10
Oils and sugars	0.10
Produce	38.6
Complex and other	3.50
Total (%)	100

Table A5i: Norovirus

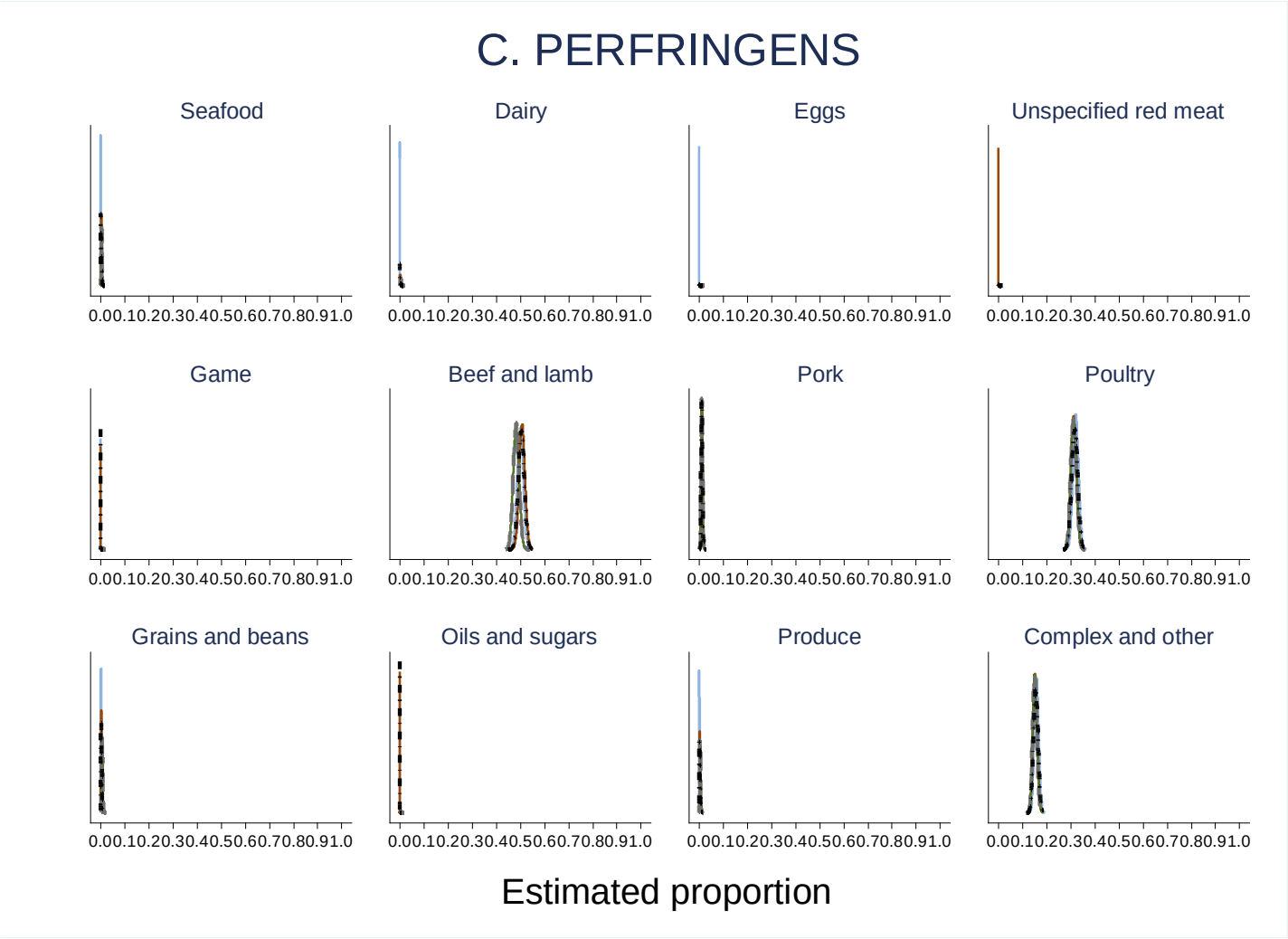
Author	Davidson	Hoffman	Havelaar
Food Commodity			
Seafood	35.7	35.6	34.7
Dairy	2.50	3.00	4.30
Eggs	0.90	1.10	4.30
Unspecified red meat	9.60	9.80	0.10
Game	0.30	0.60	0.10
Beef and lamb	2.70	1.50	6.50
Pork	2.30	1.50	6.50
Poultry	2.20	1.60	6.50
Grains and beans	4.30	6.10	10.8
Oils and sugars	0.10	0.10	0.10
Produce	31.5	39.0	15.2
Complex and other	7.80	0.10	10.8
Total (%)	100	100	100

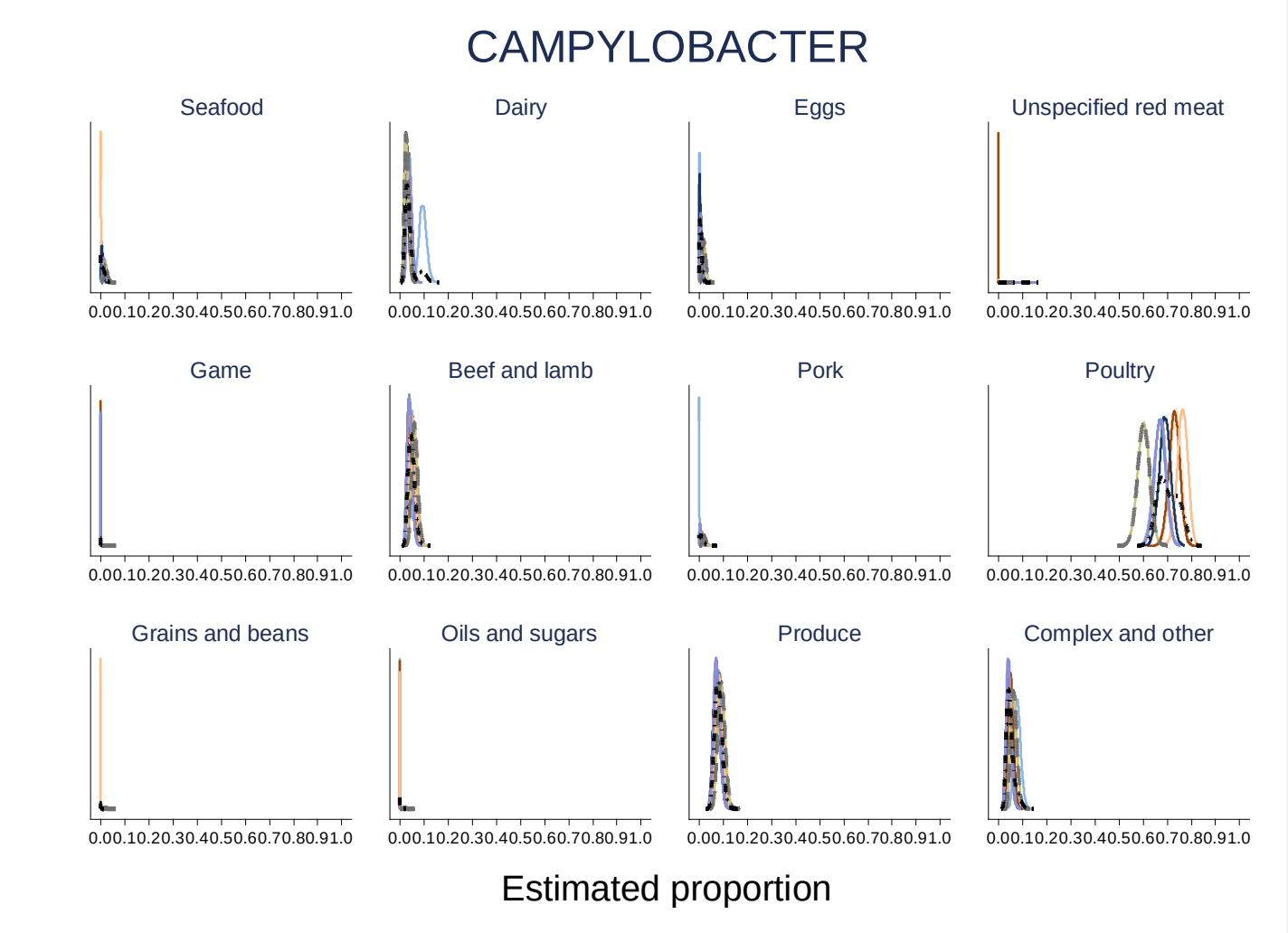
Table A5j: Rotavirus

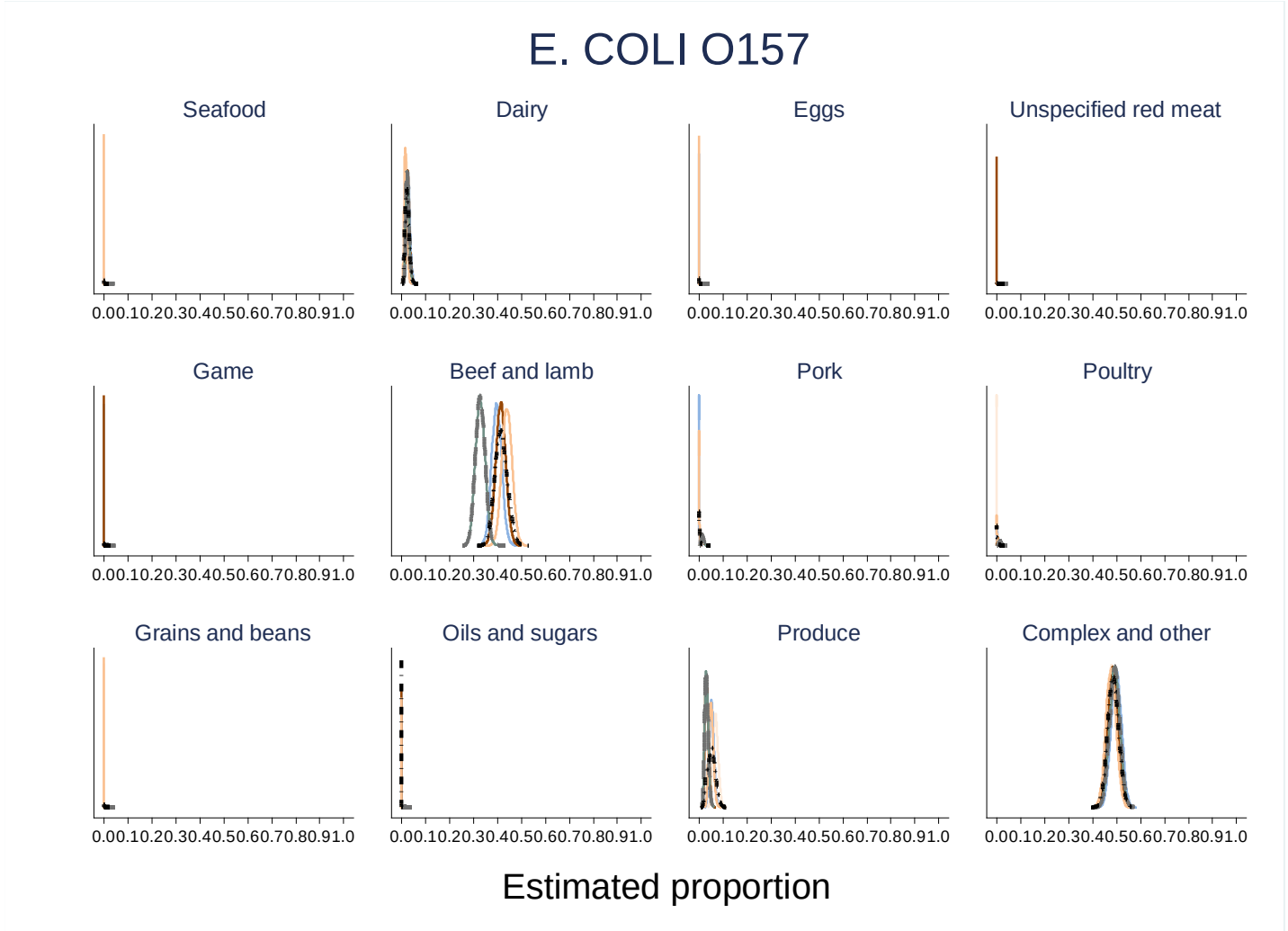
Author	Havelaar
Food Commodity	
Seafood	31.5
Dairy	3.30
Eggs	0.10
Unspecified red meat	0.10
Game	0.10
Beef and lamb	0.10
Pork	5.00
Poultry	0.10
Grains and beans	11.6
Oils and sugars	0.10
Produce	39.8
Complex and other	8.30
Total (%)	100

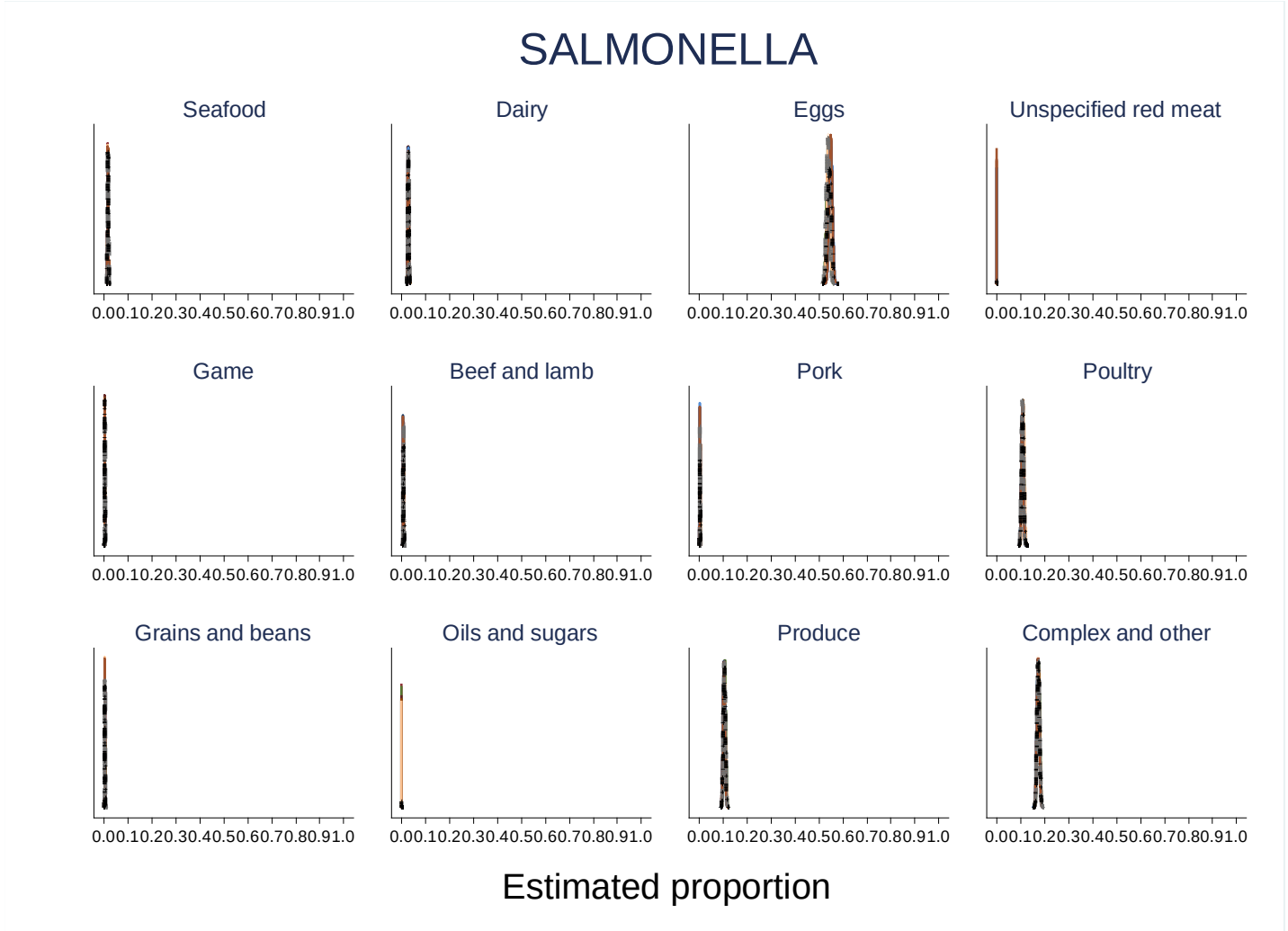
Appendix 6.1: Density plots of posterior distributions for the proportion of foodborne illness cases attributable to each food commodity by pathogen. Models based on Bayesian modelling approach

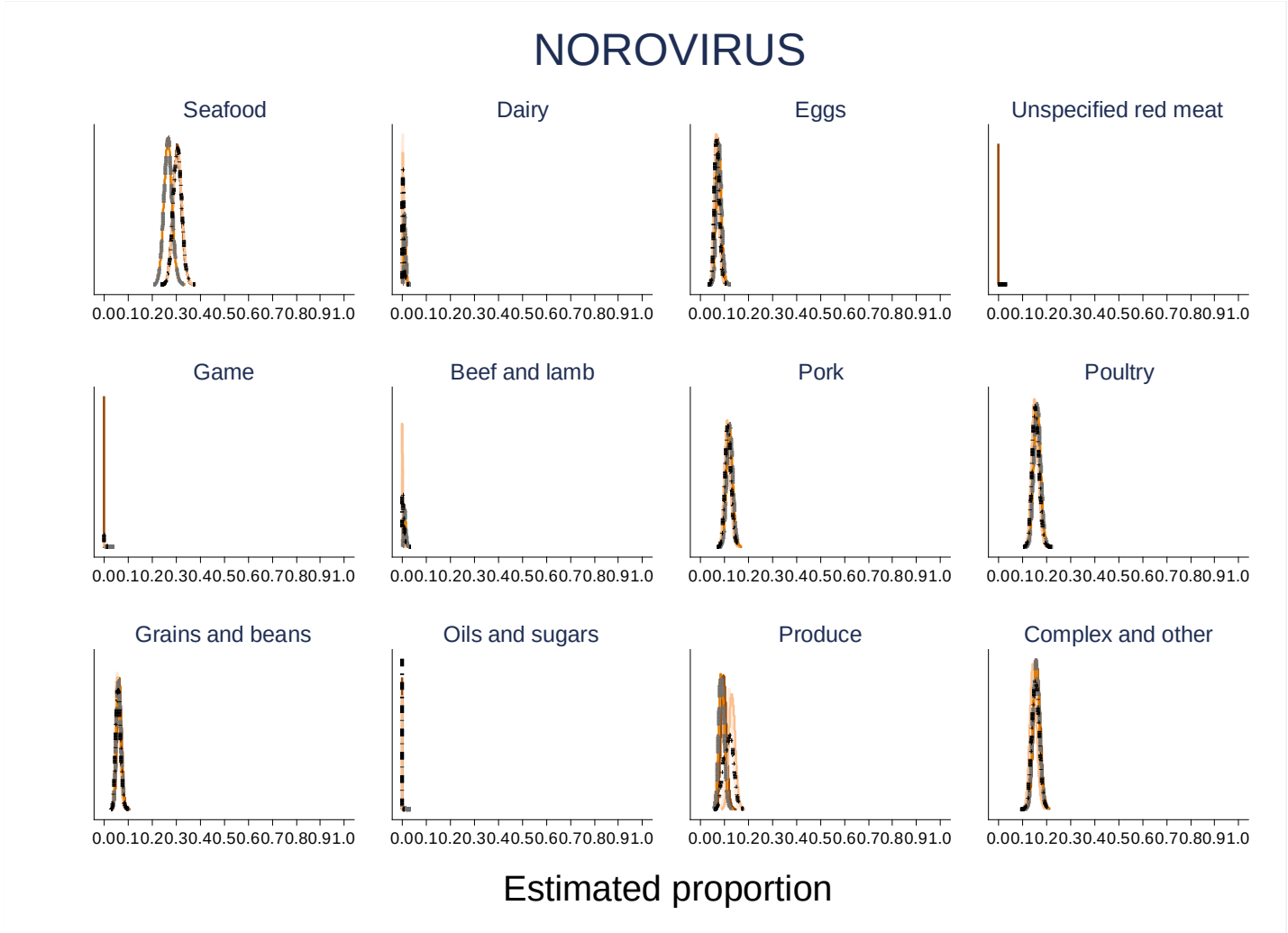
Appendix 6.1: Density plots of posterior distributions for the proportion of foodborne illness cases attributable to each food commodity by pathogen. Models based on Bayesian modelling approach. Each colour corresponds to a model with a different set of priors based on published food attribution studies. The grey line corresponds to a model with a vague prior that assumes the probability of transmission from all commodities to be equal; the dashed black line corresponds to the density of the combined distributions across all models (excluding the model with a vague prior)


C. PERFRINGENS
Seafood
Dairy
Eggs
Unspecified red meat
Game
Beef and lamb
Pork
Poultry
Grains and beans
Oils and sugars
Produce
Complex and other
0.00.10.20.30.40.50.60.70.80.91.0
Estimated proportion

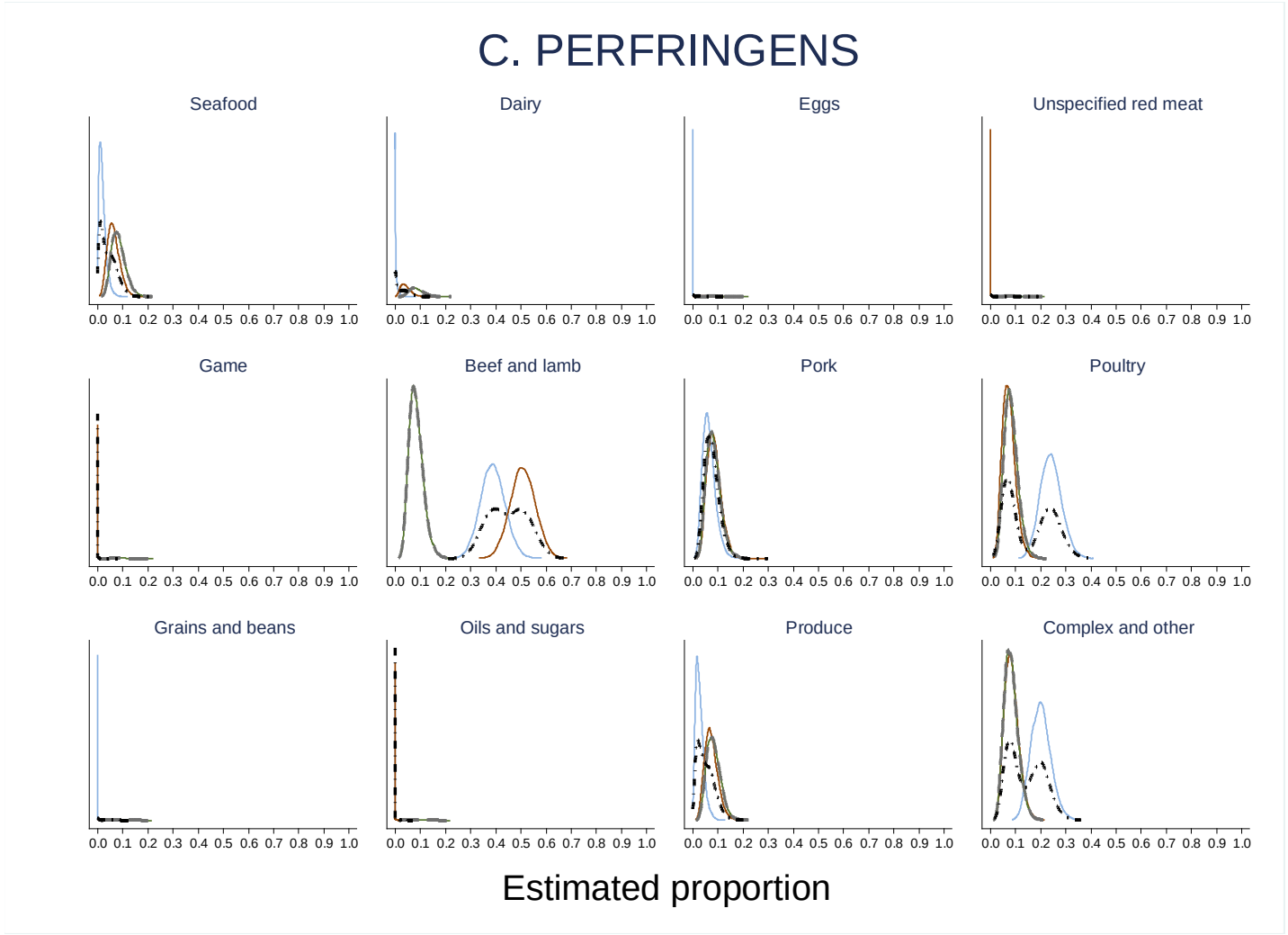

CAMPYLOBACTER
Seafood
Dairy
Eggs
Unspecified red meat
Game
Beef and lamb
Pork
Poultry
Grains and beans
Oils and sugars
Produce
Complex and other
0.00.10.20.30.40.50.60.70.80.91.0
Estimated proportion

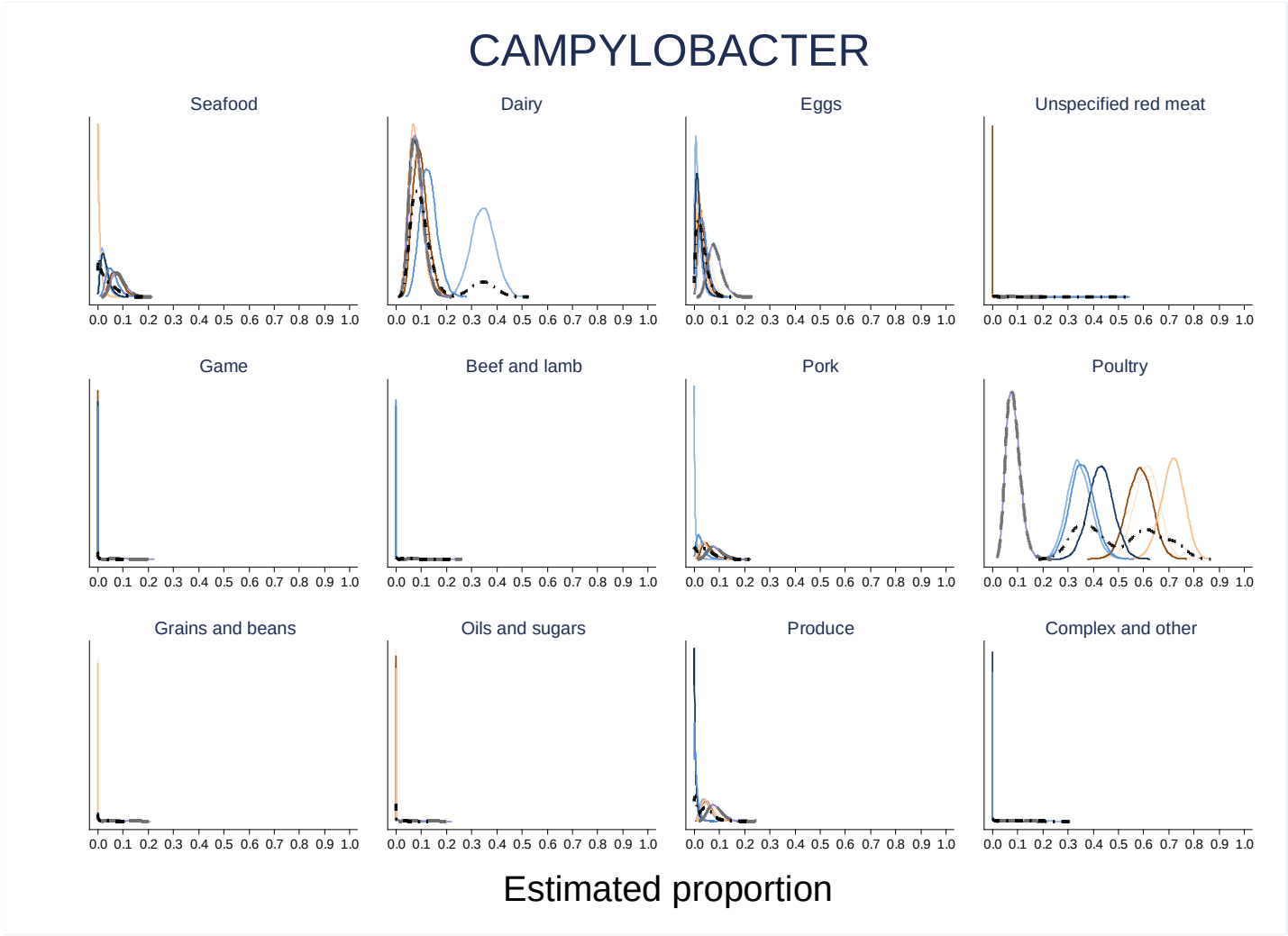

E. COLI O157
Seafood
Dairy
Eggs
Unspecified red meat
Game
Beef and lamb
Pork
Poultry
Grains and beans
Oils and sugars
Produce
Complex and other
0.00.10.20.30.40.50.60.70.80.91.0
Estimated proportion

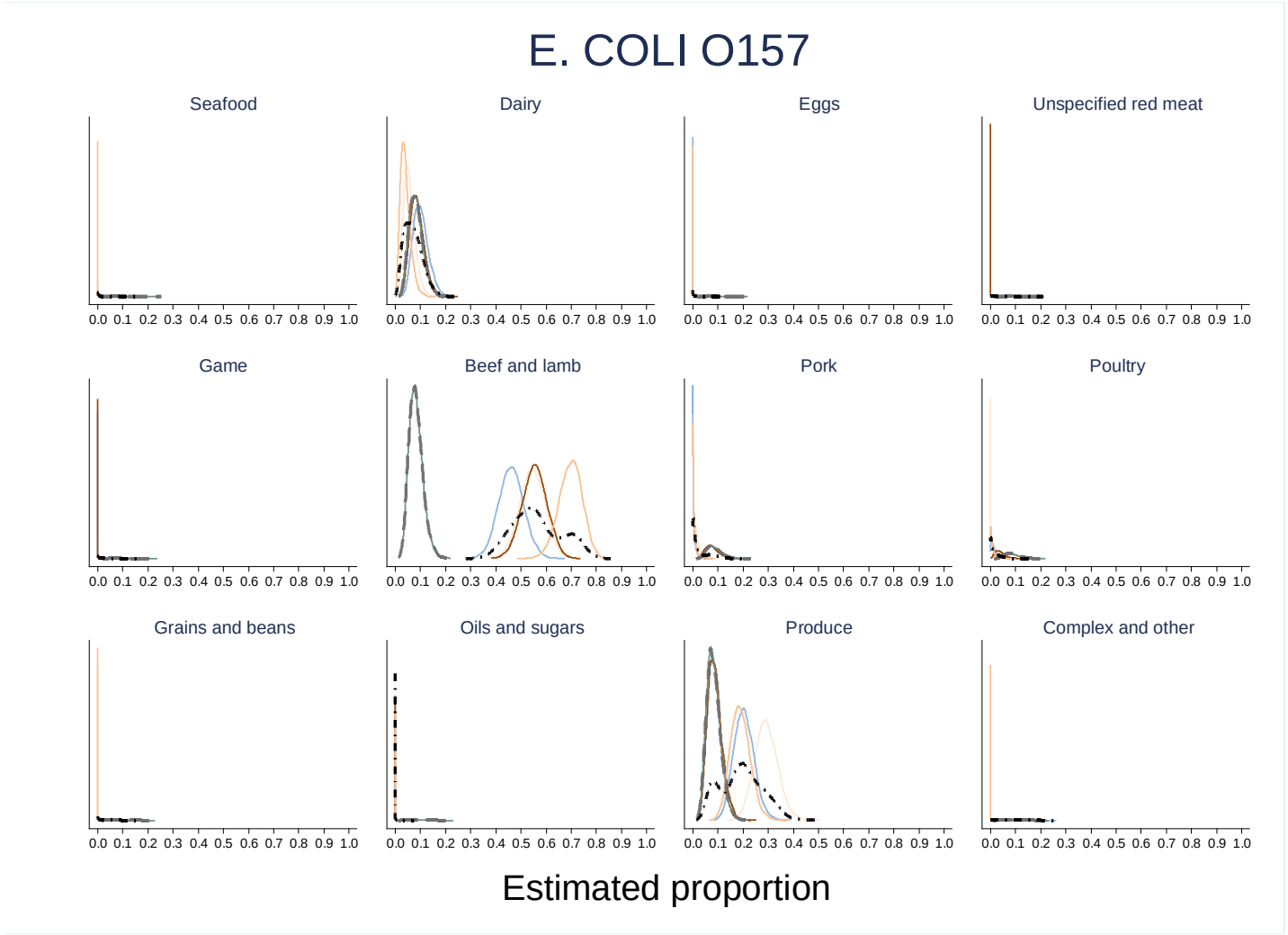

SALMONELLA
Seafood
Dairy
Eggs
Unspecified red meat
Game
Beef and lamb
Pork
Poultry
Grains and beans
Oils and sugars
Produce
Complex and other
0.00.10.20.30.40.50.60.70.80.91.0
Estimated proportion

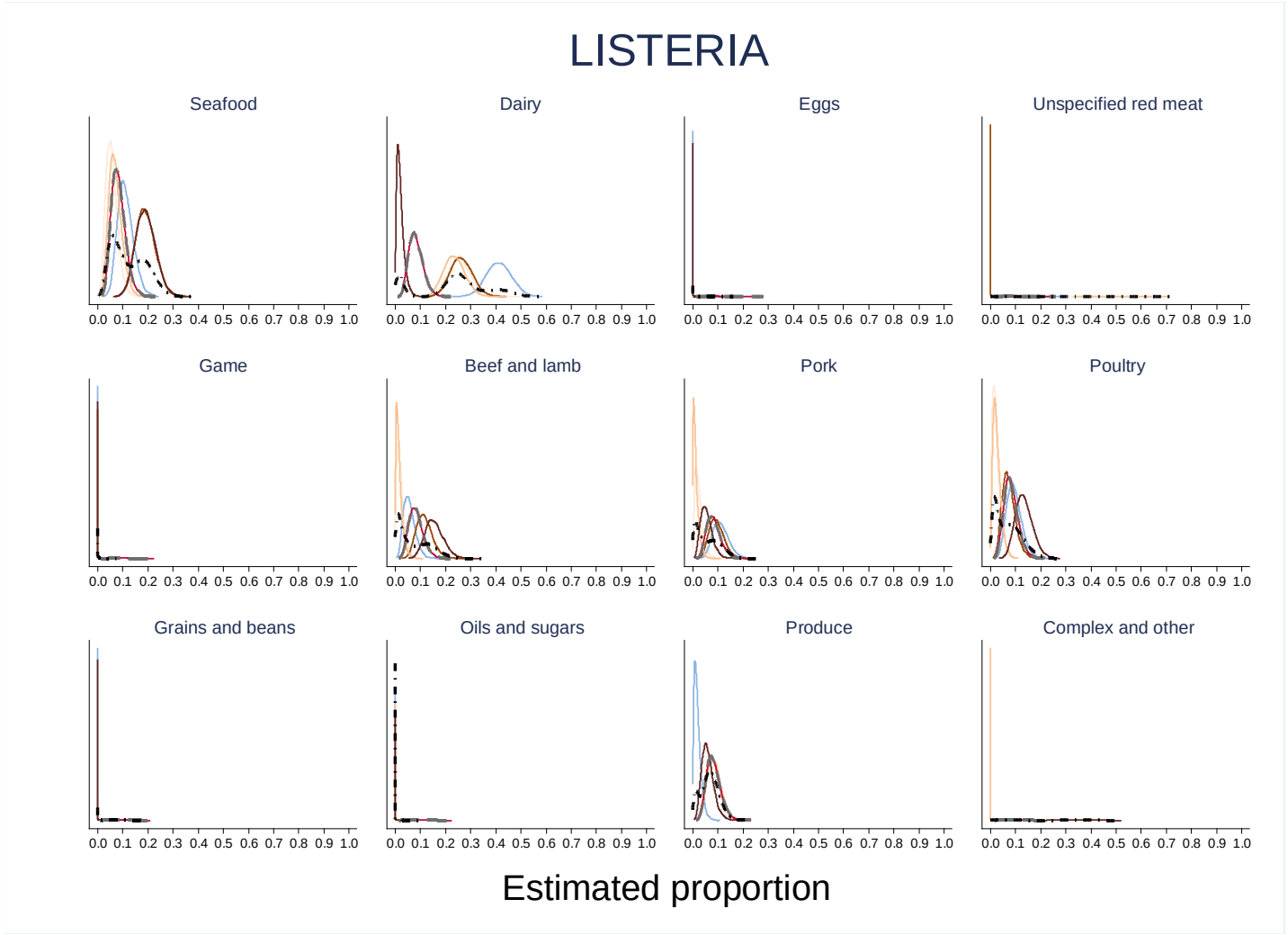

NOROVIRUS
Seafood
Dairy
Eggs
Unspecified red meat
Game
Beef and lamb
Pork
Poultry
Grains and beans
Oils and sugars
Produce
Complex and other
0.00.10.20.30.40.50.60.70.80.91.0
Estimated proportion

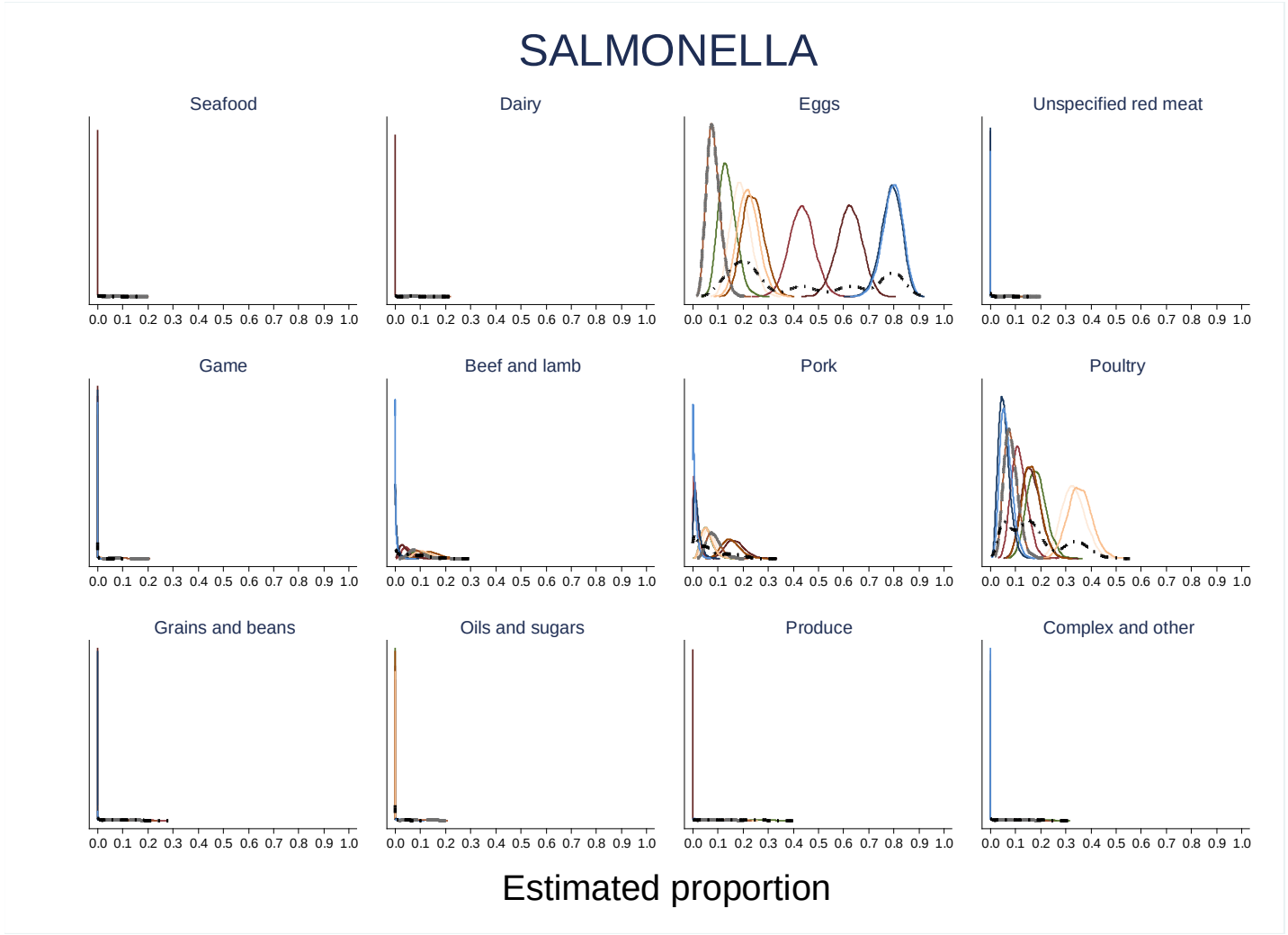
Appendix 6.2: Density plots of posterior distributions for the proportion of foodborne illness cases attributable to each food commodity by pathogen. Models based on prior information from published food attribution studies only, without the use of outbreak data. The grey line corresponds to a model with a vague prior that assumes the probability of transmission from all commodities to be equal; Each colour corresponds to a model with a different set of priors based on published food attribution studies. The dashed black line corresponds to the density of the combined distributions across all models (excluding the model with a vague prior)

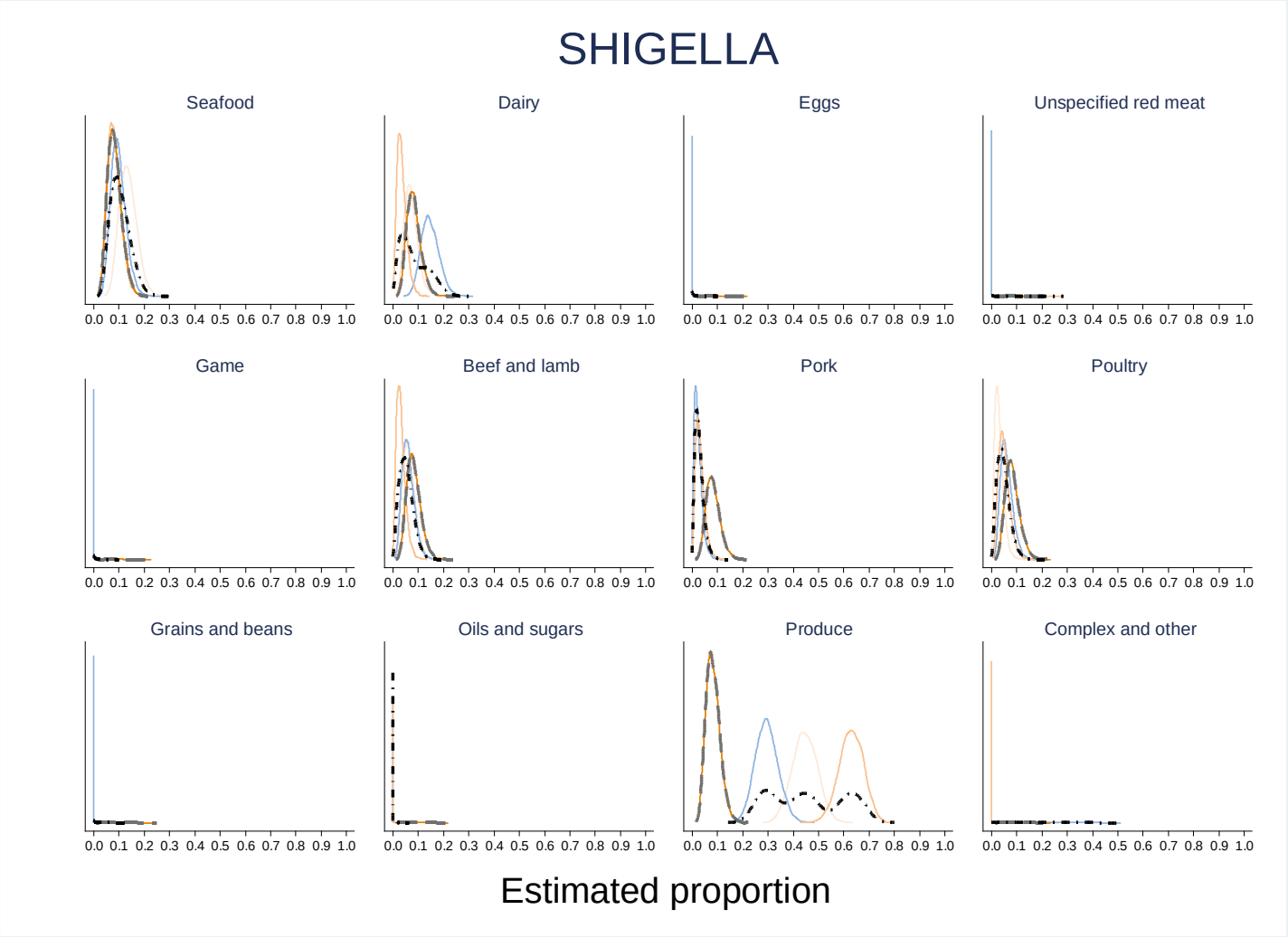

C. PERFRINGENS
Seafood
Dairy
Eggs
Unspecified red meat
Game
Beef and lamb
Pork
Poultry
Grains and beans
Oils and sugars
Produce
Complex and other
0.0 0.1 0.2 0.3 0.4 0.5 0.6 0.7 0.8 0.9 1.0
Estimated proportion

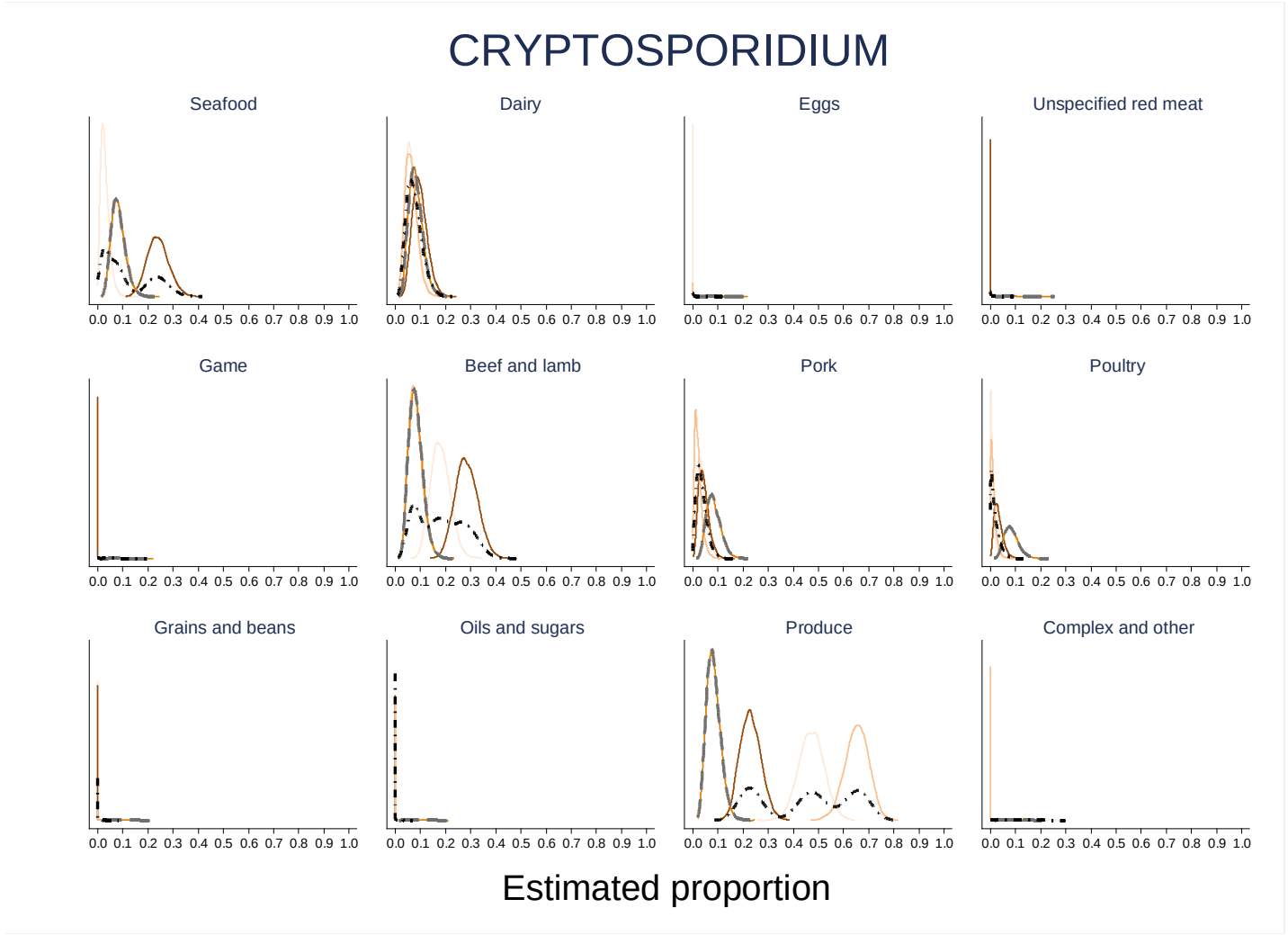

CAMPYLOBACTER
Seafood
Dairy
Eggs
Unspecified red meat
Game
Beef and lamb
Pork
Poultry
Grains and beans
Oils and sugars
Produce
Complex and other
0.0 0.1 0.2 0.3 0.4 0.5 0.6 0.7 0.8 0.9 1.0
Estimated proportion

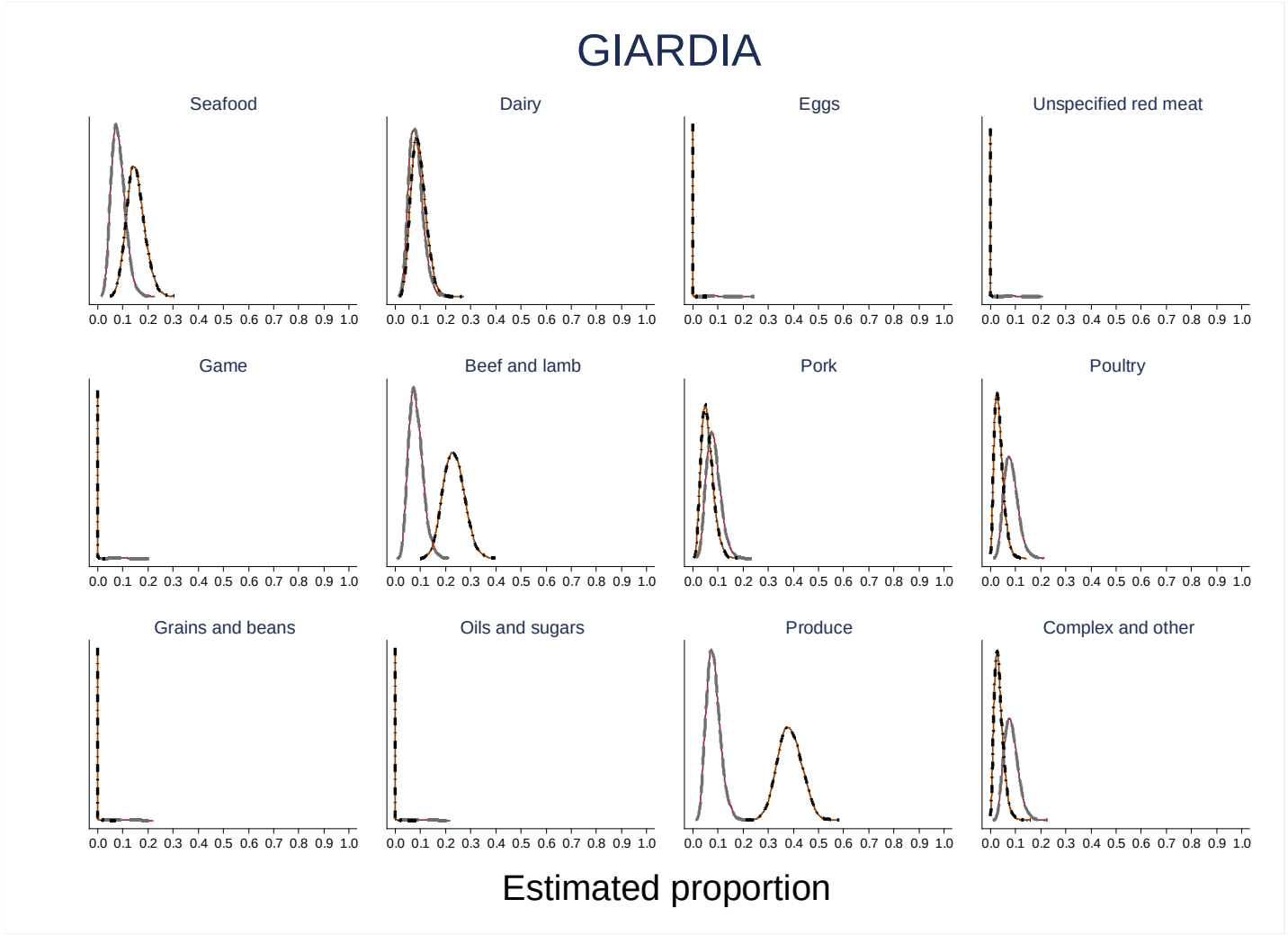

E. COLI O157
Seafood
Dairy
Eggs
Unspecified red meat
Game
Beef and lamb
Pork
Poultry
Grains and beans
Oils and sugars
Produce
Complex and other
0.0 0.1 0.2 0.3 0.4 0.5 0.6 0.7 0.8 0.9 1.0
Estimated proportion

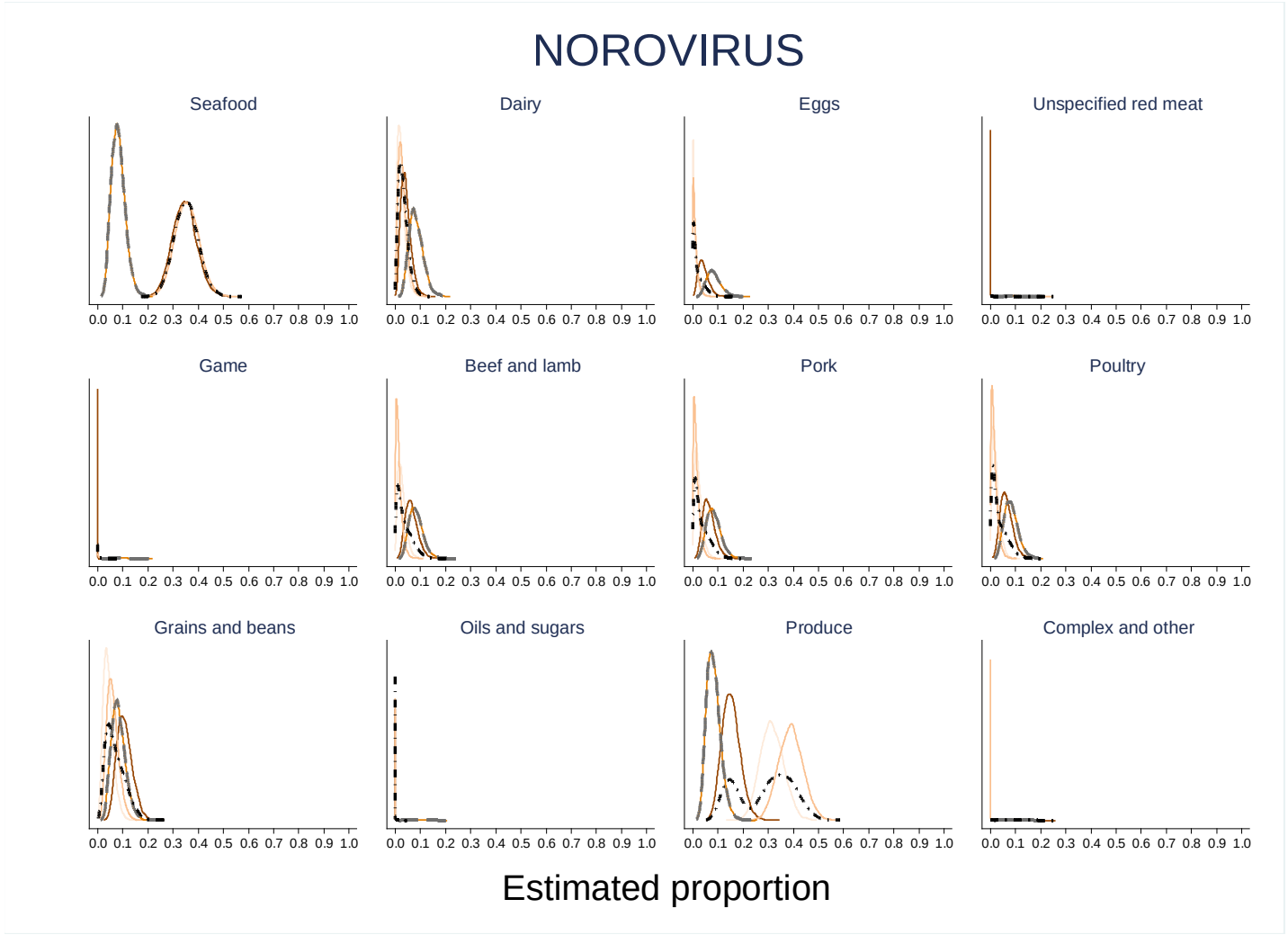

LISTERIA
Seafood
Dairy
Eggs
Unspecified red meat
Game
Beef and lamb
Pork
Poultry
Grains and beans
Oils and sugars
Produce
Complex and other
0.0 0.1 0.2 0.3 0.4 0.5 0.6 0.7 0.8 0.9 1.0
Estimated proportion

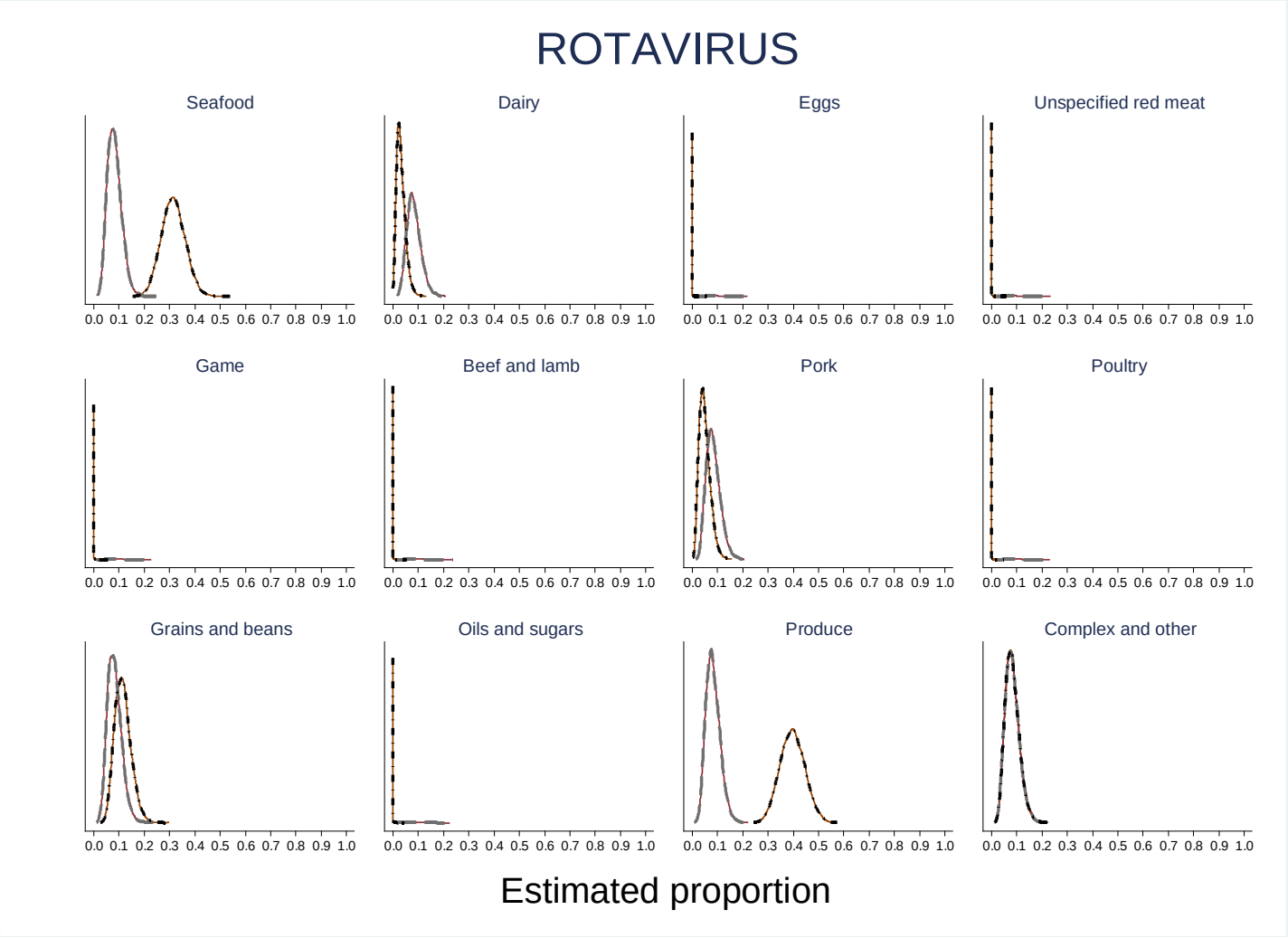

SALMONELLA
Seafood
Dairy
Eggs
Unspecified red meat
Game
Beef and lamb
Pork
Poultry
Grains and beans
Oils and sugars
Produce
Complex and other
0.0 0.1 0.2 0.3 0.4 0.5 0.6 0.7 0.8 0.9 1.0
Estimated proportion


SHIGELLA
Seafood
Dairy
Eggs
Unspecified red meat
Game
Beef and lamb
Pork
Poultry
Grains and beans
Oils and sugars
Produce
Complex and other
0.0 0.1 0.2 0.3 0.4 0.5 0.6 0.7 0.8 0.9 1.0
Estimated proportion

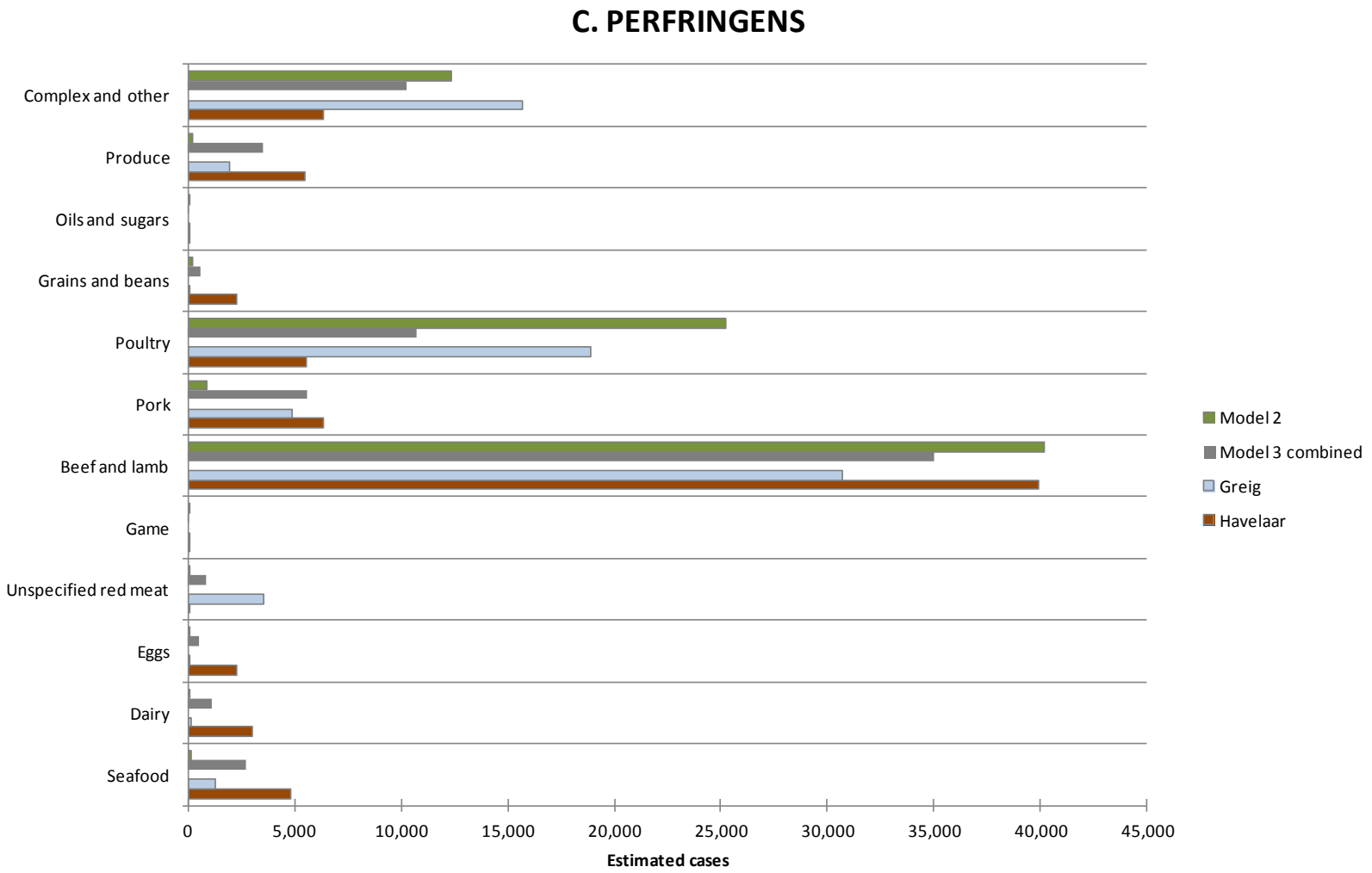

CRYPTOSPORIDIUM
Seafood
Dairy
Eggs
Unspecified red meat
Game
Beef and lamb
Pork
Poultry
Grains and beans
Oils and sugars
Produce
Complex and other
0.0 0.1 0.2 0.3 0.4 0.5 0.6 0.7 0.8 0.9 1.0
Estimated proportion

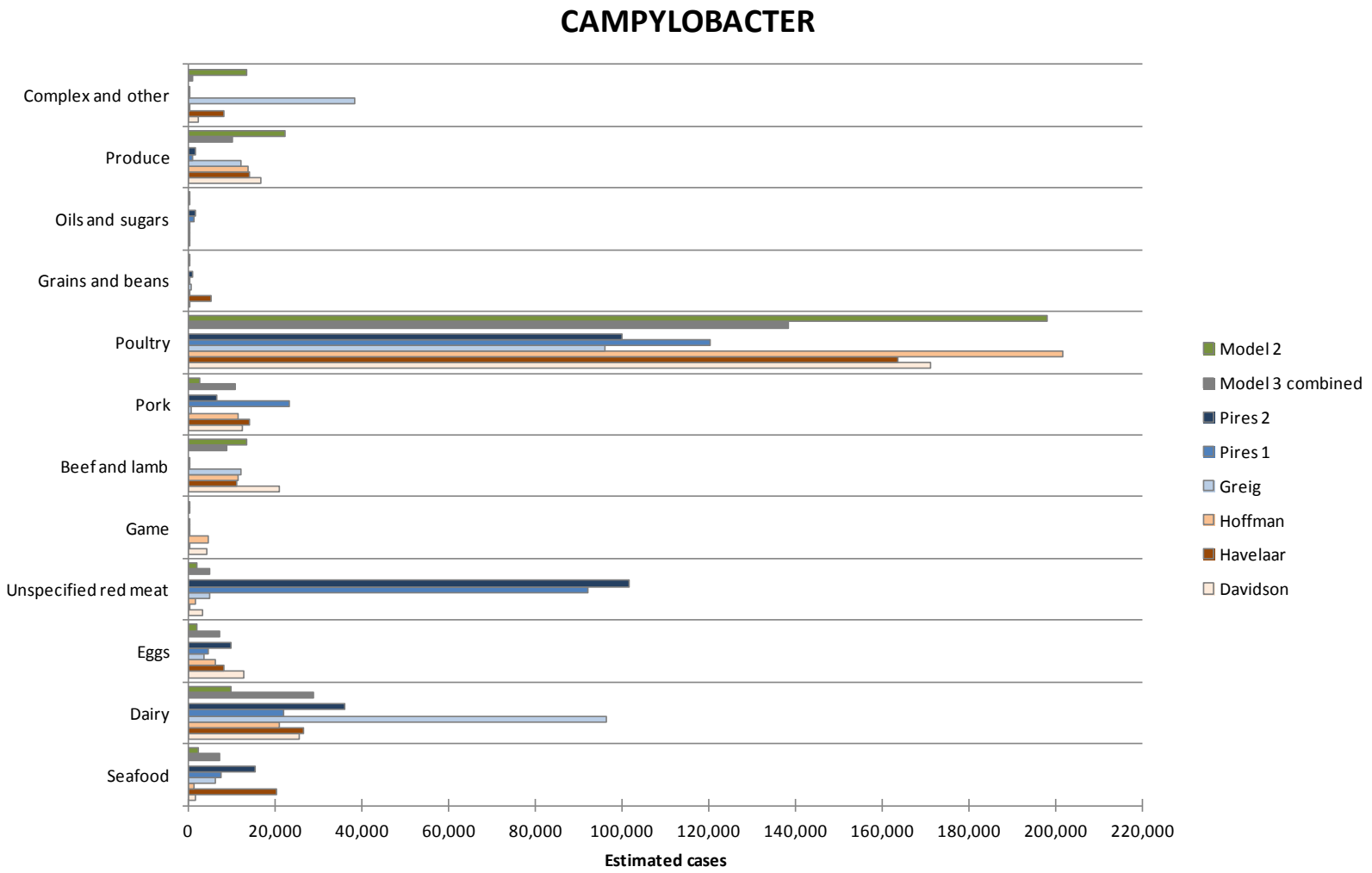

GIARDIA
Seafood
Dairy
Eggs
Unspecified red meat
Game
Beef and lamb
Pork
Poultry
Grains and beans
Oils and sugars
Produce
Complex and other
0.0 0.1 0.2 0.3 0.4 0.5 0.6 0.7 0.8 0.9 1.0
Estimated proportion

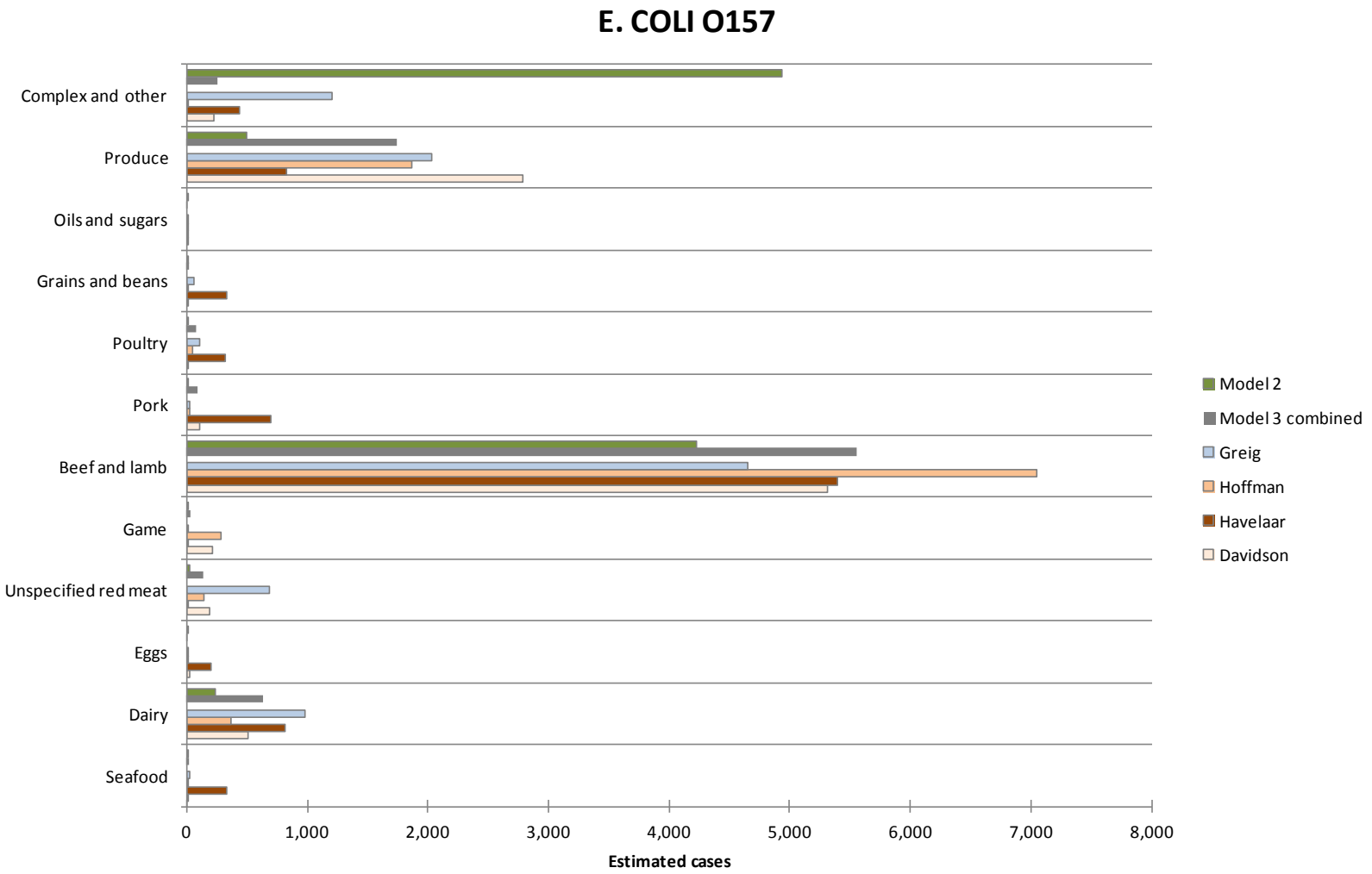

NOROVIRUS
Seafood
Dairy
Eggs
Unspecified red meat
Game
Beef and lamb
Pork
Poultry
Grains and beans
Oils and sugars
Produce
Complex and other
0.0 0.1 0.2 0.3 0.4 0.5 0.6 0.7 0.8 0.9 1.0
Estimated proportion

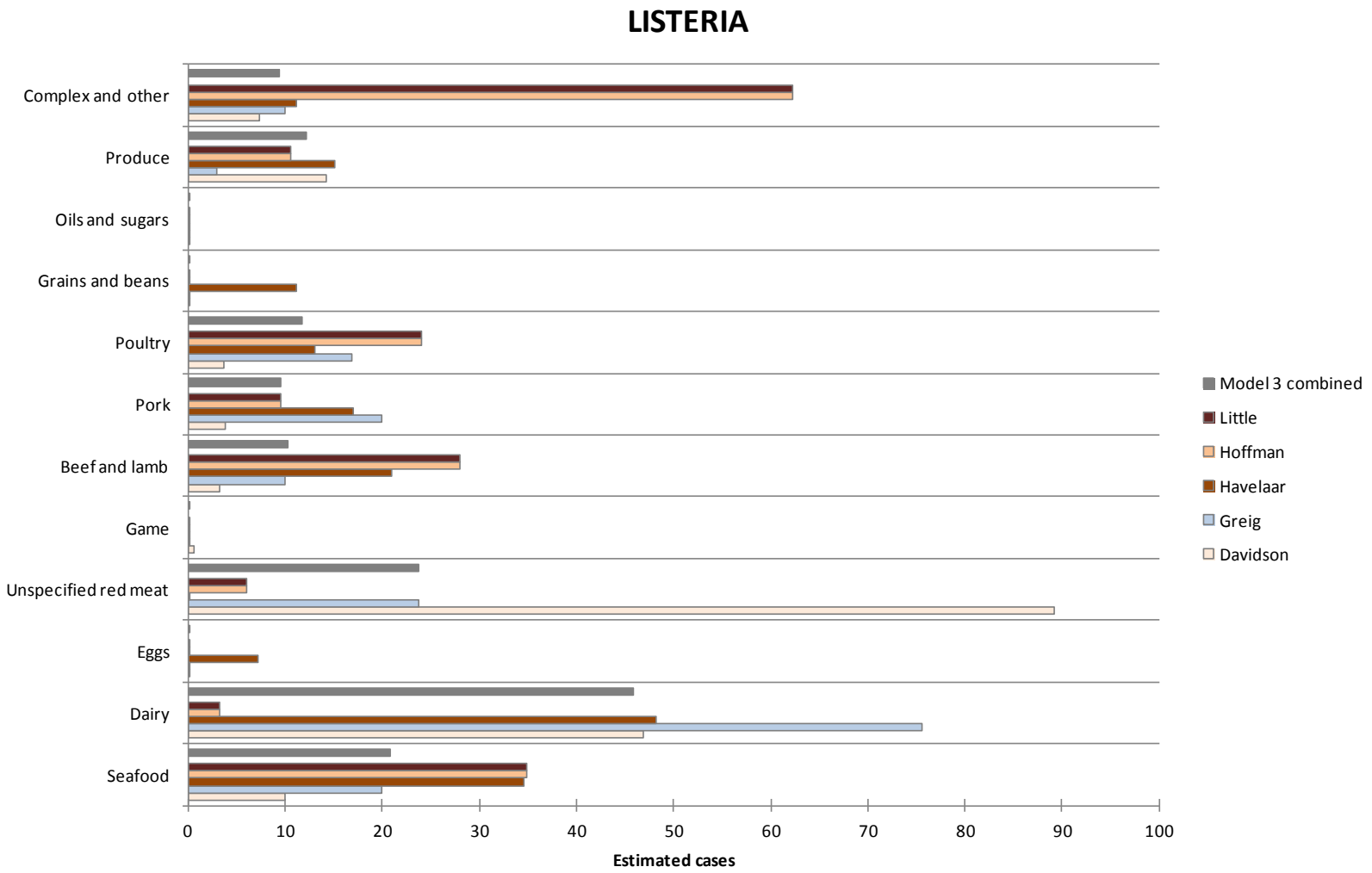

ROTAVIRUS
Seafood
Dairy
Eggs
Unspecified red meat
Game
Beef and lamb
Pork
Poultry
Grains and beans
Oils and sugars
Produce
Complex and other
0.0 0.1 0.2 0.3 0.4 0.5 0.6 0.7 0.8 0.9 1.0
Estimated proportion

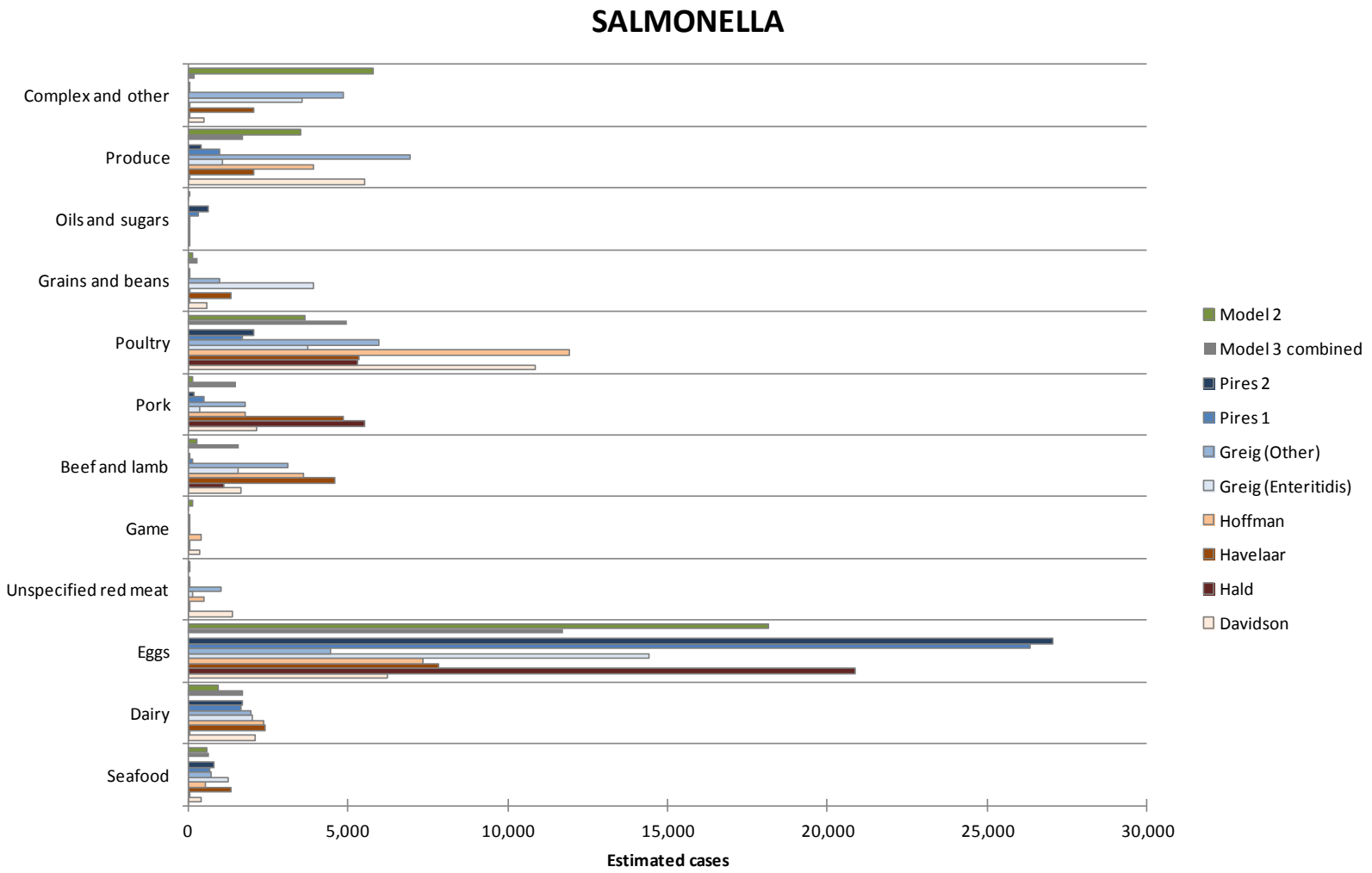
Appendix 7: Median estimates for the proportion of foodborne illness cases by attributable to each food commodity. Results from models with priors from individual studies, combined posteriors across all studies using the Bayesian approach (green), and combined posteriors across all studies using only prior information (without outbreak data)

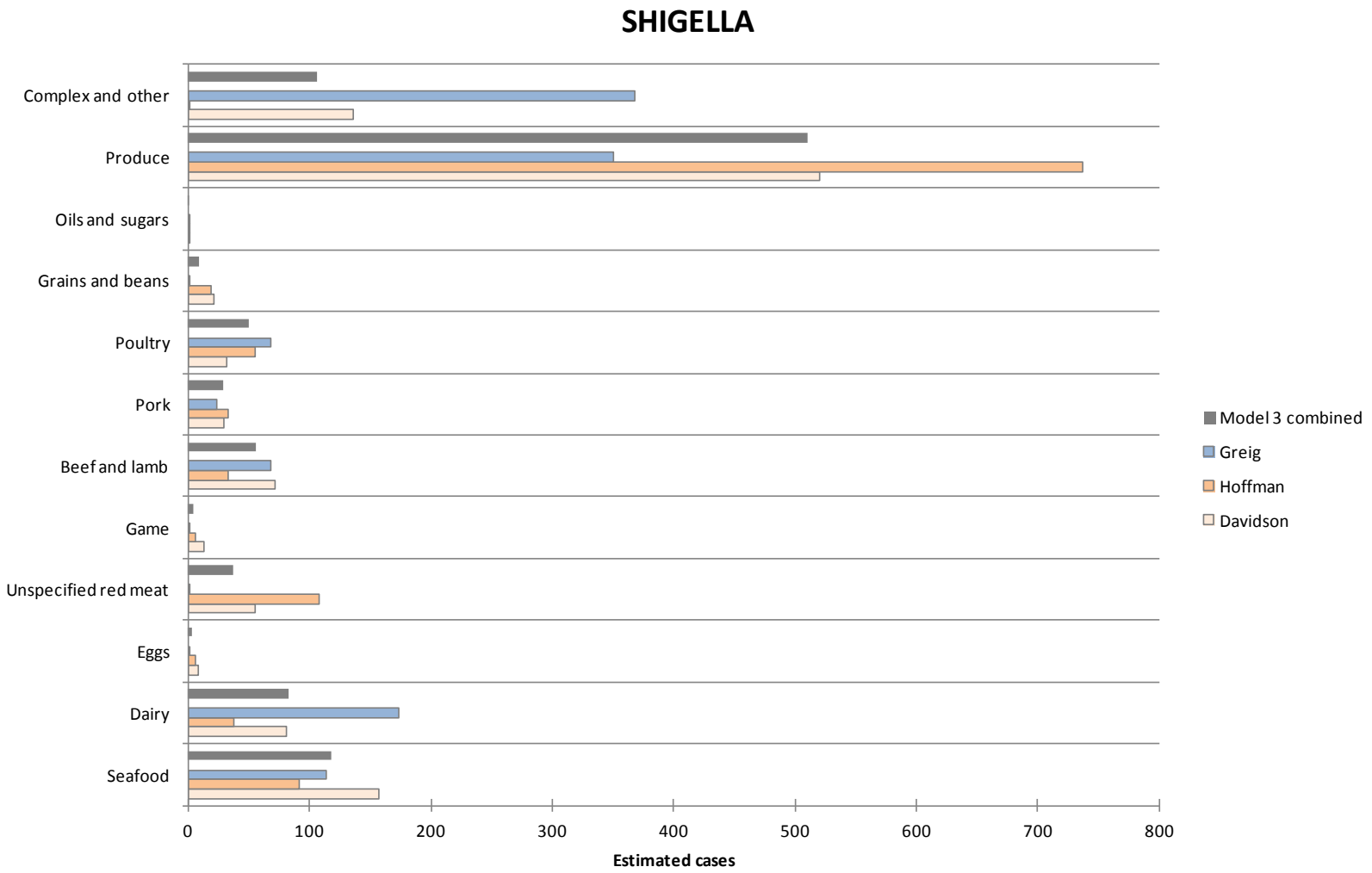

C. PERFRINGENS
Complex and other
Produce
Oils and sugars
Grains and beans
Poultry
Pork
Beef and lamb
Game
Unspecified red meat
Eggs
Dairy
Seafood
0
5,000
10,000
15,000
20,000
25,000
30,000
35,000
40,000
45,000
Estimated cases
Model 2
Model 3 combined
Greig
Havelaar

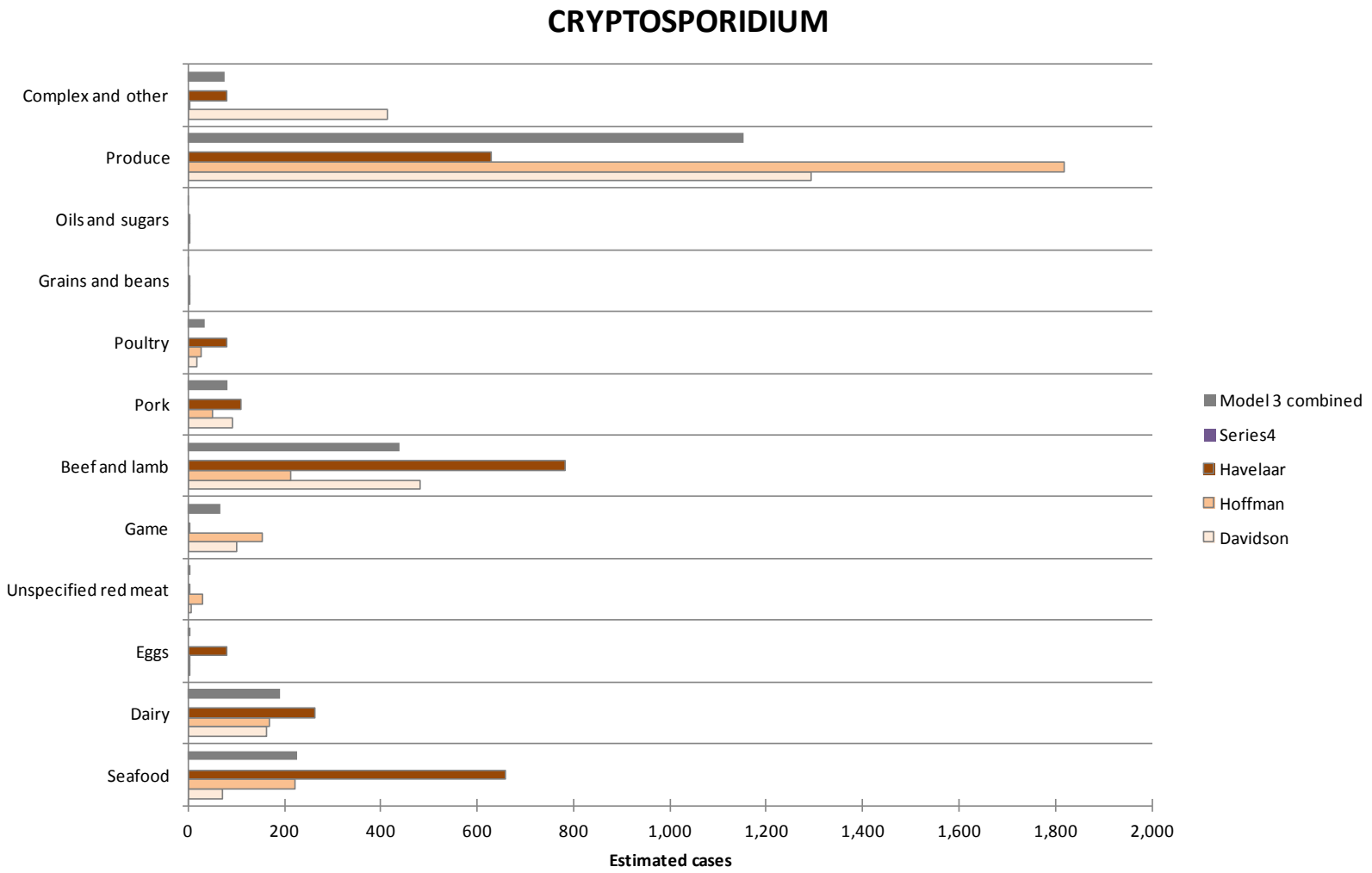

CAMPYLOBACTER
Complex and other
Produce
Oils and sugars
Grains and beans
Poultry
Pork
Beef and lamb
Game
Unspecified red meat
Eggs
Dairy
Seafood
0
20,000
40,000
60,000
80,000
100,000
120,000
140,000
160,000
180,000
200,000
220,000
Estimated cases
Model 2
Model 3 combined
Pires 2
Pires 1
Greig
Hoffman
Havelaar
Davidson

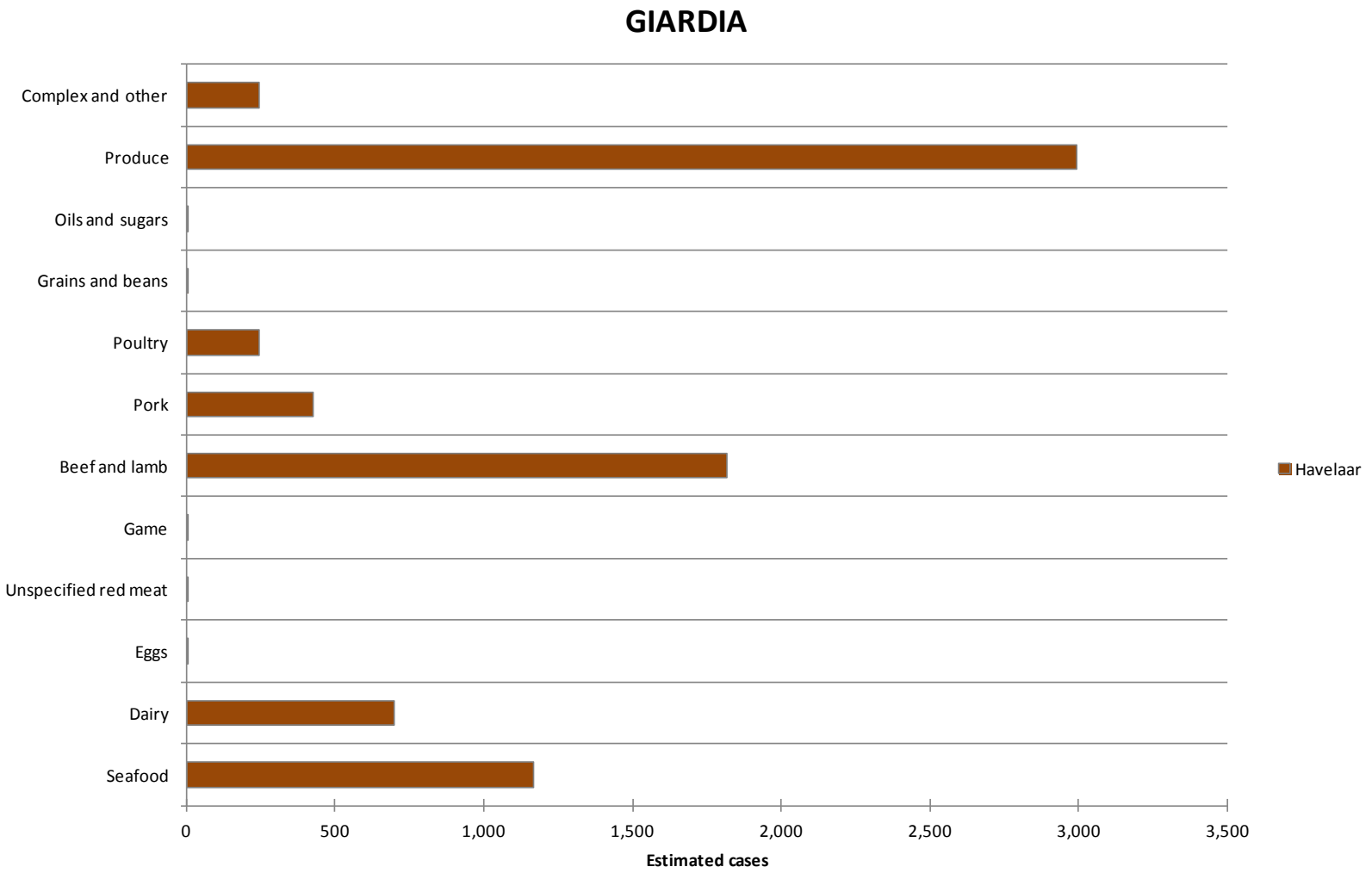

E. COLI O157
Complex and other
Produce
Oils and sugars
Grains and beans
Poultry
Pork
Beef and lamb
Game
Unspecified red meat
Eggs
Dairy
Seafood
0
1,000
2,000
3,000
4,000
5,000
6,000
7,000
8,000
Estimated cases
Model 2
Model 3 combined
Greig
Hoffman
Havelaar
Davidson

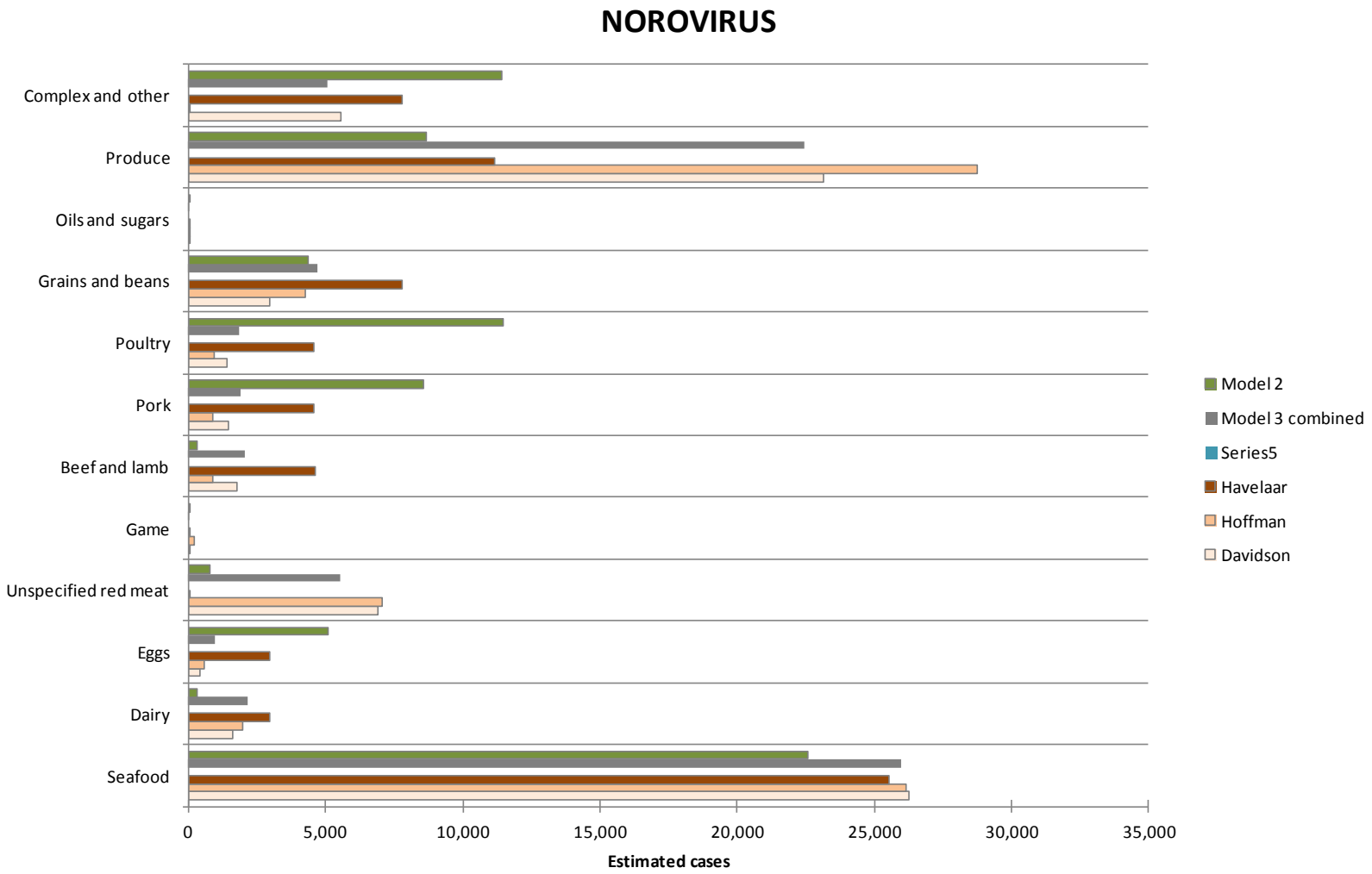

LISTERIA
Complex and other
Produce
Oils and sugars
Grains and beans
Poultry
Pork
Beef and lamb
Game
Unspecified red meat
Eggs
Dairy
Seafood
0
10
20
30
40
50
60
70
80
90
100
Estimated cases
Model 3 combined
Little
Hoffman
Havelaar
Greig
Davidson

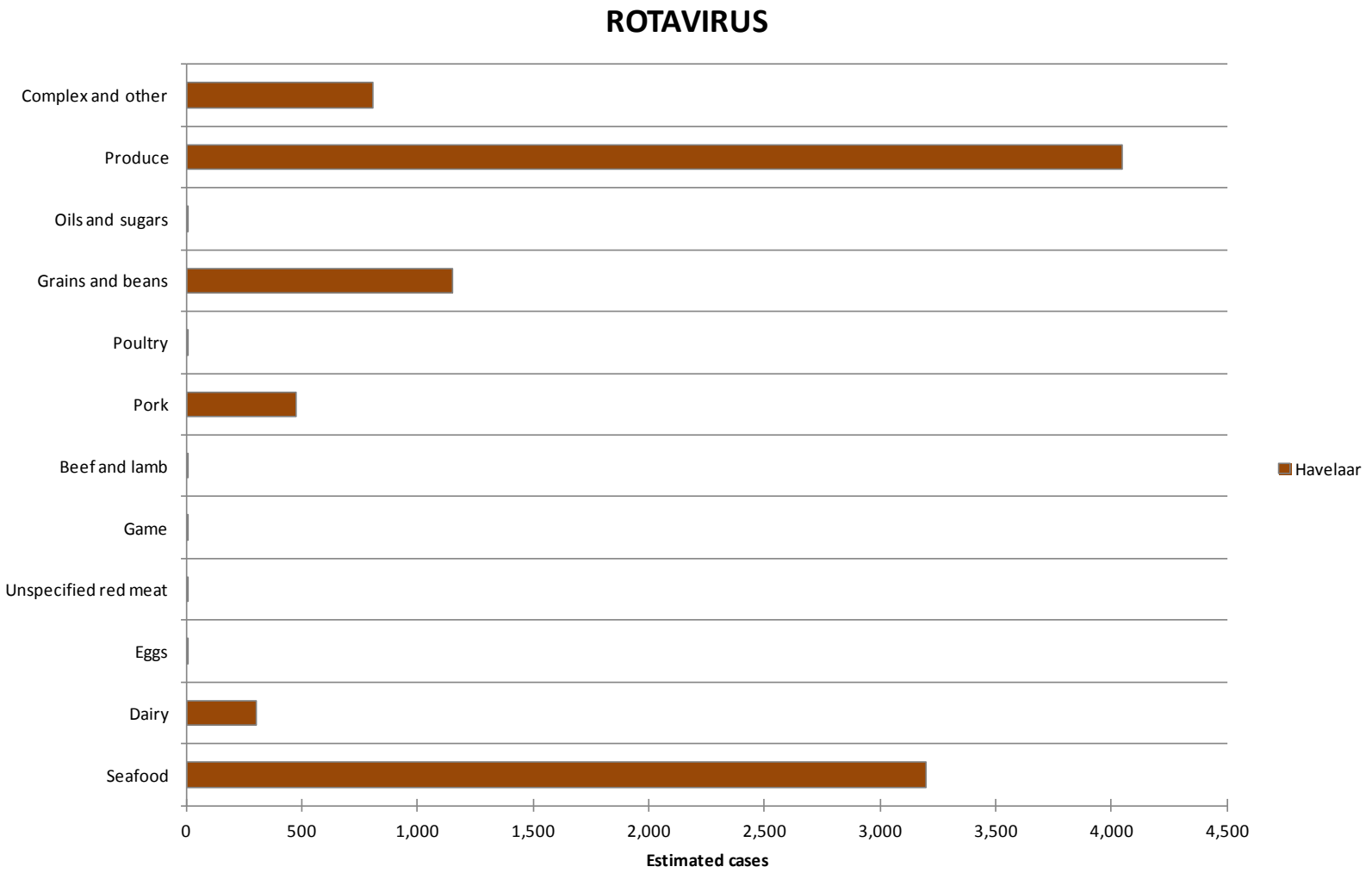

SALMONELLA
Complex and other
Produce
Oils and sugars
Grains and beans
Poultry
Pork
Beef and lamb
Game
Unspecified red meat
Eggs
Dairy
Seafood
0
5,000
10,000
15,000
20,000
25,000
30,000
Estimated cases
Model 2
Model 3 combined
Pires 2
Pires 1
Greig (Other)
Greig (Enteritidis)
Hoffman
Havelaar
Hald
Davidson


SHIGELLA
Complex and other
Produce
Oils and sugars
Grains and beans
Poultry
Pork
Beef and lamb
Game
Unspecified red meat
Eggs
Dairy
Seafood
0
100
200
300
400
500
600
700
800
Estimated cases
Model 3 combined
Greig
Hoffman
Davidson


CRYPTOSPORIDIUM
Complex and other
Produce
Oils and sugars
Grains and beans
Poultry
Pork
Beef and lamb
Game
Unspecified red meat
Eggs
Dairy
Seafood
0
200
400
600
800
1,000
1,200
1,400
1,600
1,800
2,000
Estimated cases
Model 3 combined
Series4
Havelaar
Hoffman
Davidson


GIARDIA
Complex and other
Produce
Oils and sugars
Grains and beans
Poultry
Pork
Beef and lamb
Game
Unspecified red meat
Eggs
Dairy
Seafood
0
500
1,000
1,500
2,000
2,500
3,000
3,500
Estimated cases
Havelaar


NOROVIRUS
Complex and other
Produce
Oils and sugars
Grains and beans
Poultry
Pork
Beef and lamb
Game
Unspecified red meat
Eggs
Dairy
Seafood
0
5,000
10,000
15,000
20,000
25,000
30,000
35,000
Estimated cases
Model 2
Model 3 combined
Series5
Havelaar
Hoffman
Davidson


ROTAVIRUS
Complex and other
Produce
Oils and sugars
Grains and beans
Poultry
Pork
Beef and lamb
Game
Unspecified red meat
Eggs
Dairy
Seafood
0
500
1,000
1,500
2,000
2,500
3,000
3,500
4,000
4,500
Estimated cases
Havelaar

Appendix 8.1: Estimates of food commodity-specific attribution: Comparison of estimated cases, GP consultations and hospitalisations between models using Dirichlet priors from published studies and models using a vague Dirichlet prior for the proportion of cases attributable to each food commodity. Note that these estimates are based on five organisms for which sufficient data were available from reported outbreaks and published studies (*C. perfringens*, *Campylobacter*, *E. coli* O157, *Salmonella* and norovirus).

A8.1a: FOODBORNE CASES

	Priors from published studies			Vague prior		
	Estimated cases			Estimated cases		
Food commodity	Mean[1]	Median[2]	95% CrI	Mean[1]	Median[2]	95% CrI
Seafood	26,533	26,226	(20,377 - 34,601)	27,563	27,239	(21,348 - 35,502)
Dairy	14,664	12,240	(5,932 - 37,328)	11,633	11,110	(6,115 - 20,170)
Eggs	30,883	25,828	(11,567 - 81,743)	35,831	30,968	(15,863 - 86,115)
Unspecified red meat	12,583	3,130	(175 - 39,142)	7,885	7,450	(3,863 - 14,207)
Game	730	351	(60 - 3,269)	8,024	7,609	(4,008 - 14,434)
Beef and lamb	70,849	63,339	(31,977 - 147,376)	70,945	64,967	(35,359 - 141,222)
Pork	13,016	12,596	(8,543 - 19,825)	16,978	16,627	(12,027 - 23,987)
Poultry	248,050	242,919	(165,634 - 359,700)	218,483	213,847	(147,261 - 313,761)
Grains and beans	5,415	5,247	(3,530 - 8,230)	11,884	11,500	(7,588 - 18,387)
Oils and sugars	353	91	(0 - 2,125)	7,906	7,454	(3,866 - 14,223)
Produce	38,054	36,897	(24,865 - 57,705)	39,108	38,158	(25,466 - 58,883)
Complex and other	60,105	53,732	(34,687 - 121,017)	64,366	58,311	(38,813 - 127,631)
Total[3]	521,235	506,782	(365,163 - 747,472)	520,603	507,897	(365,033 - 749,072)

[1]Mean estimate from posterior distributions; [2]Median estimate from posterior distributions; [3]Median totals are obtained by summing over posterior distributions of all food commodities and obtaining the median of the resulting distribution. They are therefore not the sum of individual medians

A8.1b: GP CONSULTATIONS

	Priors from published studies			Vague prior		
	Estimated GP consultations			Estimated GP consultations		
Food commodity	Mean[1]	Median[2]	95% CrI	Mean[1]	Median[2]	95% CrI
Seafood	1,628	1,573	(1,001 - 2,512)	2,006	1,965	(1,355 - 2,904)
Dairy	2,052	1,742	(911 - 5,011)	1,591	1,537	(893 - 2,595)
Eggs	6,652	6,048	(2,719 - 14,213)	7,164	6,602	(3,312 - 14,322)
Unspecified red meat	1,648	318	(20 - 5,187)	968	919	(461 - 1,750)
Game	109	60	(18 - 452)	1,006	957	(499 - 1,807)
Beef and lamb	9,289	8,823	(4,985 - 16,519)	9,470	8,967	(5,342 - 16,295)
Pork	1,011	955	(500 - 1,856)	1,473	1,426	(913 - 2,284)
Poultry	33,893	33,456	(24,106 - 46,562)	29,774	29,363	(21,418 - 40,331)
Grains and beans	353	326	(197 - 675)	1,198	1,152	(675 - 1,979)
Oils and sugars	47	12	(0 - 291)	970	924	(462 - 1,753)
Produce	4,812	4,703	(3,098 - 7,135)	5,211	5,104	(3,448 - 7,631)
Complex and other	6,908	6,713	(4,373 - 10,619)	7,428	7,245	(5,034 - 10,912)
Total[3]	68,404	67,359	(49,931 - 92,892)	68,259	67,228	(50,122 - 92,042)

[1]Mean estimate from posterior distributions; [2]Median estimate from posterior distributions; [3]Median totals are obtained by summing over posterior distributions of all food commodities and obtaining the median of the resulting distribution. They are therefore not the sum of individual medians

A8.1c: HOSPITALISATIONS

	Priors from published studies			Vague prior		
	Estimated hospitalisations			Estimated hospitalisations		
Food commodity	Mean[1]	Median[2]	95% CrI	Mean[1]	Median[2]	95% CrI
Seafood	177	162	(108 - 333)	251	202	(121 - 692)
Dairy	266	179	(60 - 936)	273	184	(61 - 1,020)
Eggs	1,783	1,400	(371 - 5,652)	1,858	1,471	(432 - 5,623)
Unspecified red meat	62	37	(2 - 251)	113	58	(19 - 546)
Game	30	16	(3 - 134)	123	70	(25 - 562)
Beef and lamb	2,611	1,151	(205 - 13,539)	2,123	953	(194 - 11,397)
Pork	88	68	(42 - 259)	161	108	(59 - 584)
Poultry	929	863	(429 - 1,827)	935	857	(429 - 1,899)
Grains and beans	47	36	(21 - 141)	139	87	(41 - 571)
Oils and sugars	2	1	(0 - 11)	111	58	(19 - 532)
Produce	734	543	(206 - 2,243)	614	491	(189 - 1,758)
Complex and other	3,532	1,853	(477 - 16,555)	3,591	1,903	(500 - 17,210)
Total[3]	10,261	6,921	(2,522 - 37,402)	10,292	7,027	(2570 - 37,893)

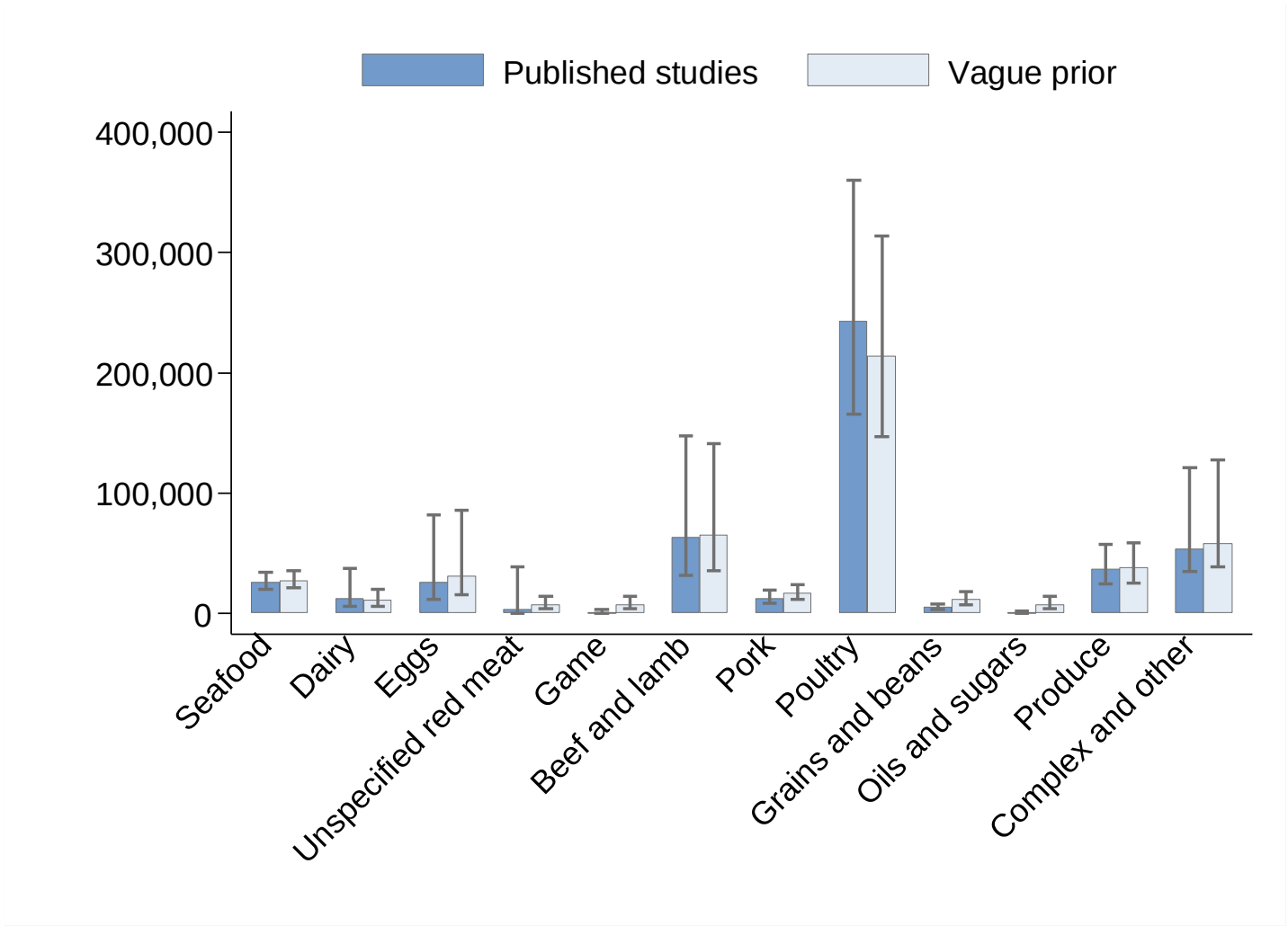
[1]Mean estimate from posterior distributions; [2]Median estimate from posterior distributions; [3]Median totals are obtained by summing over posterior distributions of all food commodities and obtaining the median of the resulting distribution. They are therefore not the sum of individual medians

Appendix 8.2: Estimates of food commodity-specific attribution: Bar graphs comparing estimated cases, GP consultations and hospitalisations between models using Dirichlet priors from published studies and models using a vague Dirichlet prior for the proportion of cases attributable to each food commodity. Note that these estimates are based on five organisms for which sufficient data were available from reported outbreaks and published studies (*C. perfringens, Campylobacter, E. coli* O157*, Salmonella* and norovirus).

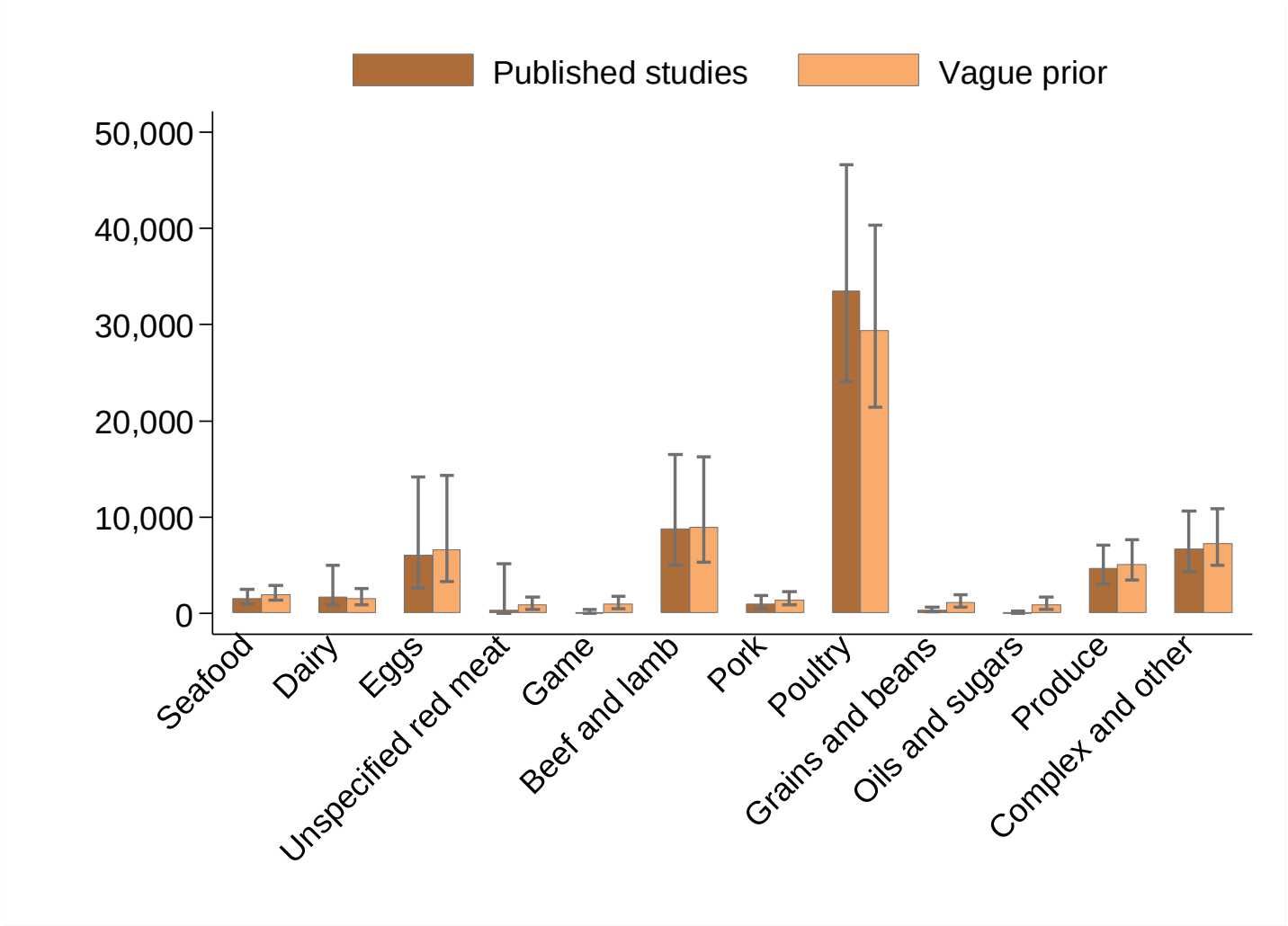
Both types of models use a Bayesian approach, combining data from reported outbreaks on the proportion of outbreak cases attributable to different food commodities with prior information in the form of a Dirichlet distribution. The two types of models differ in that in the first, the Dirichlet prior is informed by values obtained from published studies. This prior information has more influence on the results where data are sparse. In the second type of model, the Dirichlet prior is non-informative (vague); it regards prior information about the importance of different food commodities as irrelevant so that only outbreak data contribute to estimation.

The two sets of models produce broadly similar results for foodborne cases, GP consultations and hospitalisations. Estimates based on models with vague priors tend to give greater importance (higher estimates) for food commodities that contribute fewer cases (unspecified red meat, pork, oils and sugars, grains and beans).

A8.2a: FOODBORNE CASES



A8.2b: GP CONSULTATIONS



A8.2c: HOSPITALISATIONS