# Health outcome trees for priority foodborne pathogens and conditions for economic costing

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## Executive Summary

Foodborne illness remains a significant cause of morbidity and mortality in Australia. Estimating the economic burden from foodborne illnesses informs the prioritisation of control measures to reduce the burden of illness due contaminated food. An Australian Government Department of Health report of the economic burden of foodborne illness circa 2000 estimated an annual cost of $1.2 billion to the Australian community. This study did not provide estimates for individual pathogens, limiting the ability of the results to be used to prioritise food safety interventions. This report forms part of a project to develop a costing model for the annual cost of food borne illness in Australia circa 2015. The objective of this component is to develop health outcome trees for pathogens and conditions for which cost estimates can be derived, and to provide updated estimates circa 2015.

Using expert opinion relating to the significance of incidence, hospitalisations, deaths, sequel illnesses and preventability for pathogens and agents, we prioritised the following 10 pathogens to develop separate health outcome trees:

- *Salmonella enterica*
- *Campylobacter* spp.
- *Listeria monocytogenes*
- Shiga toxin-producing *Escherichia coli*
- Norovirus
- *Yersinia entercolitica*
- Other pathogenic *Escherichia coli*
- *Salmonella enterica* serotype Typhi
- *Toxoplasma gondii*
- *Shigella* spp.

We developed health outcome trees that include ongoing illness and sequel illnesses that may follow a preceding acute illness. The approach to estimating incidence, the number hospitalisations, and the number of deaths by pathogen followed that of the burden of disease circa 2010 study[1].

In comparison to circa 2010, incidence of many pathogens increased circa 2015, with dramatic increases in the incidence of salmonellosis (58% increase in rate since circa 2010) and campylobacteriosis (25% increase in rate since circa 2010). Consistent with this, we estimated a 32% increase in the rate of hospitalisation for salmonellosis, and a 9% increase in the rate of hospitalisation for campylobacteriosis, and increases in incidence and hospitalisations for Guillain-Barré syndrome, irritable bowel syndrome, and reactive arthritis. Increases in the estimated incidence rate for yersiniosis (eight-fold increase in rate from 2010) were likely driven by the rise of culture-independent testing, and were not reflected in an increase in the number hospitalisations. Health outcome trees for these and other prioritised pathogens will enable costing of interventions and evaluation of measures for foodborne disease control.

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We would also like to thank the following experts for taking part in the survey to prioritise pathogens:

- Joy Gregory
- Phil Haywood
- Gill Hall
- Martyn Kirk
- Ben Polkinghorne
- Michelle Robertson
- Russell Stafford

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## Scope of the Work

This project is undertaken at the National Centre for Epidemiology and Population Health (NCEPH) at the Australian National University for Food Standards Australia New Zealand. It forms one component of a project to construct a whole of government cost model for foodborne illness in Australia that is being overseen by three separate funding agencies (New South Wales Food Authority, Department of Health, Food Standards Australia New Zealand).

The objective of this component is to develop health outcome trees for pathogens and conditions for which cost estimates can be derived. Based on a list of all pathogens that are likely to make a significant or noticeable contribution to the cost of foodborne illness in Australia, this document presents criteria used to prioritise and categorise pathogens for inclusion in a costing model, which includes *Salmonella enterica, Campylobacter* spp., Shiga-toxin-producing *Escherichia coli*, and *Listeria monocytogenes*. A health outcome tree for all gastroenteritis (including that due to unknown cause) is also provided. Full outcome trees for all prioritised pathogens are provided, based on Australian data where possible, including pathogens, their sequelae, and the probabilities of arriving at various health outcome states including GP visits, hospitalisations, fatalities, and where there is no medical consultation. Where ongoing illness is likely, this is also included in the trees. Approaches for non-prioritised pathogens are also provided. The methods used to develop outcome trees are documented, together with descriptions of the data sources and assumptions used to derive the trees. The report is produced in conjunction with Microsoft Excel models that implement the health outcome trees.

# Introduction

In Australia, foodborne illness is a significant cause of morbidity and occasional mortality. Approximately 25% (90% credible interval: 13%-42%) of the 15.9 million episodes of gastroenteritis that occurred in Australia in 2010 were transmitted by contaminated food[1]. This equates to approximately one episode of foodborne gastroenteritis every five years per person. In addition, there were an estimated 5,140 cases of illness not due to gastroenteritis, 35,840 cases of sequel illnesses, 31,920 hospitalisations and 86 deaths due to contaminated food, circa 2010[1 2].

Estimating the economic burden of foodborne illnesses informs the prioritisation of control measures in order to reduce the burden of illness due to contaminated food. There are several components to the cost of foodborne disease, including medical practitioner visits, pathogen tests, antibiotic prescriptions, specialist visits, premature mortality, lost productivity when people ill with gastroenteritis stay home from work, and lost productivity when their carers stay home to look after them.

In 2006, an Australian Government Department of Health study of the economic burden of foodborne illness estimated annual total costs of $1.2 billion[3] in 2000, a significant continuing cost to the Australian community. This study did not provide estimates for individual pathogens, limiting the ability of the results to be used to prioritise food safety interventions, or to calculate the cost-effectiveness of changes to food safety policy through specific case studies.

In this updated foodborne disease costing project, circa 2015, we will use an improved cost-of-illness model for estimating annual economic cost, with estimates of uncertainty, including pathogen-specific estimates for key pathogens such as *Campylobacter* spp. and *S. enterica*, together with costs of pathogens not causing gastroenteritis and chronic sequel illnesses. This report presents criteria used to prioritise and categorise pathogens for inclusion in the costing model, together with the development of health outcome trees, from which cost estimates can be derived. The methods, data sources and assumptions are fully documented, and this report is provided in conjunction with Microsoft Excel models of the health outcome trees.

## Pathogen prioritisation tool

The aim of this tool was to prioritise and categorise pathogens for inclusion in the costing model. Similar tools have been developed in the context of antimicrobial resistance[4], and for prioritisation of diseases for surveillance and research[5]. In these studies, criteria are scored (0, 1, 2 or -1, 0, 1), and then combined in a weighted average to produce a total score. Potential criteria are listed in Appendix Table A1 indicating the weights used for these criteria in the two studies, and whether they were listed in the original scoping discussion at Food Standards Australia New Zealand (FSANZ).

After consultation with FSANZ, we included the following criteria in the prioritisation tool:

- Incidence of disease
- Work and school absenteeism as a result of illness
- Hospitalisations
- Deaths
- Sequel illnesses following acute illness
- Preventability
- Data availability
- Generalisability to other pathogens

The first six criteria were key components in other prioritisation studies, while the last two criteria relate to the availability of relevant Australia data, and the suitability of reference pathogens to be used as a model for other pathogens for which there is little data available. We conducted an online survey of seven experts to capture the first six criteria, and assessed the remaining two criteria within a smaller group of team members. This division of criteria was made because a thorough understanding of data availability and representativeness was needed to assess these later criteria, while the first six were best assessed by experts in foodborne disease.

The prioritisation tool was applied to all 23 potentially foodborne pathogens or agents considered in the 2010 burden of illness study[1]. Pathogens excluded from that study were those acquired only overseas (such as *Vibrio cholera* and *Trichinella spiralis*) and those that cause gastroenteritis but are not proven agents of foodborne disease (such as *Clostridium difficile*). For each pathogen and agent, we summarised estimates of incidence and severity from the circa 2010 study, followed by six questions for the experts to answer, designed around the proposed criteria. An example for non-typhoidal *Salmonella* is provided in Table A2 of the Appendix.

The survey was undertaken by the following seven experts in foodborne illness (see Appendix Table A2 for a summary of experts' qualifications and experience):

- Joy Gregory
- Phil Haywood
- Gill Hall
- Martyn Kirk
- Ben Polkinghorne
- Michelle Robertson
- Russell Stafford

On the basis of responses relating to the first six criteria, Table 1 was produced, with mean rankings for each of the full list of pathogens included in the circa 2010 burden of foodborne illness study. The top 10 pathogens or agents were prioritised for inclusion in the model. Shaded rows in Table 1 indicate these prioritised pathogens.

Of pathogens prioritised by this process, Australian incidence data are available for most pathogens from national or state surveillance, providing good data quality. Data from the 1998 Water Quality Study in Australia[6] was used to estimate incidence of both other pathogenic *E. coli* and norovirus, while seroprevalence data from the United States were used to estimate incidence of *T. gondii*[7]. This study provides rigorously collected Australian cohort data; however it does not capture any recent changes and may not be representative of the Australian population. Estimates of ongoing illness following toxoplasmosis are difficult to obtain due to lack of data. Although there is a lack of recent studies, norovirus acts as a suitable pathogen to generalise to other viruses causing gastroenteritis, most of which also lack recent national data.

| Pathogen | Incidence | Hospitalisations | Deaths | Sequelae | Preventability | Total | Rank |
|---|---|---|---|---|---|---|---|
| Bacteria causing gastroenteritis | | | | | | | |
| *Bacillus cereus* | 2.86 | 2.43 | 2.00 | 0.86 | 0.86 | 9.00 | 16 |
| *Campylobacter* spp. | 8.14 | 7.14 | 6.71 | 5.14 | 6.71 | 33.86 | 2 |
| *Clostridium perfringens* | 4.57 | 3.00 | 1.86 | 1.71 | 2.14 | 13.29 | 11 |
| Shiga toxin-producing *E. coli* | 5.14 | 5.00 | 5.71 | 3.86 | 7.43 | 27.14 | 4 |
| Other pathogenic *E. coli* | 6.29 | 4.14 | 3.29 | 1.14 | 1.71 | 16.57 | 7 |
| *Salmonella enterica* | 7.43 | 6.29 | 6.71 | 7.43 | 6.86 | 34.71 | 1 |
| *S. enterica* ser. Typhi | 3.00 | 3.86 | 4.29 | 1.71 | 3.29 | 16.14 | 8 |
| *Shigella* spp. | 3.29 | 3.43 | 3.86 | 1.14 | 2.86 | 14.57 | 9 |
| *Staphylococcus aureus* | 3.00 | 2.57 | 2.00 | 0.43 | 1.00 | 9.00 | 16 |
| *Vibrio parahaemolyticus* | 2.43 | 2.14 | 2.00 | 0.71 | 1.00 | 8.29 | 21 |
| *Yersinia entercolitica* | 3.86 | 4.00 | 3.57 | 2.14 | 3.29 | 16.86 | 6 |
| Viruses causing gastroenteritis | | | | | | | |
| Adenovirus | 2.71 | 2.29 | 1.86 | 0.57 | 0.71 | 8.14 | 22 |
| Astrovirus | 2.86 | 2.29 | 1.57 | 0.71 | 1.00 | 8.43 | 20 |
| Norovirus | 7.57 | 6.00 | 3.00 | 1.86 | 1.43 | 19.86 | 5 |
| Rotavirus | 2.57 | 2.14 | 2.71 | 0.57 | 1.00 | 9.00 | 16 |
| Sapovirus | 3.14 | 2.86 | 1.29 | 0.71 | 1.29 | 9.29 | 15 |
| Parasites causing gastroenteritis | | | | | | | |
| *Cryptosporidium* spp. | 2.86 | 1.86 | 2.29 | 0.86 | 1.14 | 9.00 | 16 |
| *Giardia lamblia* | 2.86 | 2.57 | 2.57 | 0.71 | 1.43 | 10.14 | 14 |
| Agents not causing gastroenteritis | | | | | | | |
| Hepatitis A | 2.71 | 3.71 | 3.14 | 0.86 | 1.29 | 11.71 | 12 |
| *Listeria monocytogenes* | 4.43 | 4.86 | 7.00 | 7.29 | 4.43 | 28.00 | 3 |
| *Toxoplasma gondii* | 3.57 | 2.71 | 2.57 | 1.86 | 3.71 | 14.43 | 10 |
| Ciguatera | 2.43 | 2.57 | 2.57 | 0.71 | 2.86 | 11.14 | 13 |
| Scombrotoxicosis | 2.86 | 1.71 | 1.71 | 0.86 | 1.00 | 8.14 | 22 |

**Table 1:** Results of pathogen prioritisation, with prioritised pathogens shaded.

## Methods used to develop outcome trees and their purpose

To develop the health outcome trees, broad stages of illness were mapped by pathogen. New cases have an initial acute illness stage that includes diagnosis, and subsequent to this they may recover or progress to ongoing illness, sequelae, or death. The trees do not represent a patient's journey, but various possible health care outcomes in broad categories that provide a framework to identify costs associated with foodborne illness. Arrows in the diagram indicate these stages of illness, but do not reflect pathways for an individual patient, who may, for example, see a GP and later be admitted to hospital. In this report, we estimate the number of domestically acquired illnesses, GP consultations, hospitalisations and deaths associated with each pathogen. Some of these inputs were derived as part of a separate contract for the New South Wales Food Authority to estimate incidence and mortality by age and sex. However, these inputs were adjusted to age categories required for this contract, and included some health outcomes not included in that project.

Additionally, we estimated incidence, GP consultations, hospitalisations, ongoing illnesses and deaths for 4 sequelae illnesses that can follow foodborne illness: Guillain-Barré syndrome (GBS), haemolytic uremic syndrome (HUS), irritable bowel syndrome (IBS), and reactive arthritis (ReA). All these estimates were made for three age categories: <5, 5-65, and 65+ years using data circa 2015 wherever possible.

Following this report, the next stage of the costing report will be to estimate the average number of activities that occur in each outcome category. For example, patients that visit a GP may have various tests, prescription of medication, and time off work. These activities and events can then be costed.

### Data Sources

***Incidence***

Where possible, we used data from a three-year period: 2013-2015. All denominator data were based on the Australian population provided the Australian Bureau of Statistics for each of those years[8]. Estimates relied on disease data obtained from the National Notifiable Disease Surveillance System[9], notifiable disease surveillance at the State and Territory level, the Water Quality Study (WQS) from 1998[6], and the National Gastroenteritis Survey II (NGSII) from 2008-9[10]. The WQS was used for pathogens such as norovirus that are not nationally notifiable. This study is the most recent widespread study of incidence of pathogens such as norovirus and other (non-STEC) pathogenic *E. coli.* Since there are limited local data on *T. gondii,* we applied seroprevalence estimates from a United States study conducted from 1999-2004[7] to Australian population data for 2013-2015. The pathogens and illnesses estimated and the data source used for each is shown in Table 2.

| Pathogen or Illness | Data source | Date |
|---|---|---|
| Total infectious gastroenteritis | NGSII | 2008-2009 |
| Bacteria | | |
| *Campylobacter* spp. | NNDSS | 2013-2015 |
| *Listeria monocytogenes* | NNDSS | 2013-2015 |
| Non-typhoidal *Salmonella* | NNDSS | 2013-2015 |
| *Shigella* spp. | NNDSS | 2013 |
| Shiga toxin-producing *Escherichia coli* | State Surveillance (SA) | 2013-2015 |
| Other pathogenic *Escherichia coli* | NGSII & WQS | 2008, 1998 |
| *Salmonella enterica* serovar Typhi | NNDSS | 2013-2015 |
| *Yersinia enterocolitica* | State Surveillance (NT, Qld, SA, WA) | 2013-2015 |
| Protozoa | | |
| *Toxoplasma gondii* | U.S. Seroprevalence Study | 1999-2004 |
| Viruses | | |
| Norovirus | NGSII & WQS | 2008, 1998 |
| Sequelae | | |
| Guillain-Barré syndrome | NNDSS & Literature | 2013-2015 |
| Haemolytic uraemic syndrome | State Surveillance & Literature | 2013-2015 |
| Irritable bowel syndrome | NNDSS & Literature | 2013-2015 |
| Reactive arthritis | NNDSS, State Surveillance & Literature | 2013-2015 |

**Table 2:** Data sources used for estimating incidence for costing, Australia 2015.

When using the NNDSS or State and Territory surveillance data, notifications where age was unknown were excluded. These were rare – for example, we excluded 0.08% of notifications for *Campylobacter* spp. and 0.16% of notifications for *Salmonella* due to missing age. As *Shigella* spp. notifications diagnosed through culture-independent diagnostic testing (CIDT) were reported differently by States and Territories from 2014, data from 2013 only was used to estimate foodborne incidence for *Shigella* spp. and irritable bowel syndrome arising from *Shigella* spp. Listeriosis during pregnancy can lead to disease in the foetus, which we describe as a congenital case. We used OzFoodNet annual reports over 2001-2011 to estimate the ratio of congenital to non-congenital cases of listeriosis each year, and applied this to national notification numbers between 2013-2015 to estimate the annual number of incident cases of congenital listeriosis.

***General Practice (GP) consultations***

For pathogens causing gastroenteritis, we calculated the proportion of incident cases that consulted a GP using data from the National Gastroenteritis Survey II[10]. For all-cause gastroenteritis, we extracted the proportion of GP consultations per case from this dataset using the case definition for that survey[10], giving a proportion of 0.196 (95% Confidence Interval [CI] 0.156-0.243). This proportion is higher than that reported elsewhere[11], as we included multiple GP consultations for some patients.

Norovirus typically has a short duration of illness of 1-2 days (see Table A4 of the Appendix). Within the NGSII survey, the proportion of cases with illness duration of 1-2 days that

consulted a GP was largely unchanged from the overall proportion, so we used the same proportion of 0.196 (95%CI 0.156-0.243) for norovirus.

For bacterial pathogens causing gastroenteritis, we followed the approach of Hall et al[12]. That is, we calculated the proportion of individuals who consulted a GP by duration of illness, and then calculated an overall proportion as a weighted average using pathogen weights from that study. Applying this approach to our three age groups and using symptom profiles for bacterial pathogens (see Table A4 of the Appendix) showed no clear differences, in line with earlier findings[12], so we used the same proportion for each age group and for all bacterial pathogens, namely 0.367 (95%CI 0.246-0.501).

For pathogens and illnesses that do not cause gastroenteritis, we estimated the proportion of cases that see a GP from the literature, informed by expert opinion. Where there were no new data to inform estimates, we adopted the assumptions of the earlier Abelson study for Australia, which based on the opinion of clinicians and other experts[3]. Table 3 summarises assumptions and relevant literature regarding the number of GP consultations for these illnesses. Where estimates were derived from data, intervals represent 95% confidence intervals, largely those reported in these studies. Where we have used assumptions, we included variation as 95% credible intervals about our point estimate to reflect uncertainty in these assumptions. The choice of 95% intervals here allows direct comparison across pathogens, while later estimates from the models are typically presented as median estimates with 90% credible intervals.

***Sequel illnesses***

We adopted the same assumptions as in our prior work[2] to calculate the proportion of cases of bacterial illness that led to sequel illnesses. We assumed that 0.03% (range 0.0192%-0.0945%) of illnesses due to *Campylobacter* spp. result in GBS, that 3% (95% credible interval 1.7%-5.1%) of cases of Shiga toxin-producing *E. coli* (STEC) result in HUS, that 8.8% (90% credible interval 7.2%-10.4%) of illnesses due to *Campylobacter* spp., *Salmonella*, and *Shigella* spp. result in IBS, and that 7-12% (range 0-26%) of illnesses due to *Campylobacter* spp., *Salmonella*, *Shigella* spp., and *Y. enterocolitica* result in ReA. More detail on the studies underlying these estimates are provided in Ford *et al* and the associated Appendices[2].

| Pathogen or illness | GP visits per case (95% Interval) | References |
|---|---|---|
| Guillain-Barré syndrome | 3.6 (3.56-3.66) | Frenzen (2008) reported 19,728 visits for 5,472 patients (3.6 per patient)[13] |
| Reactive arthritis | 0.80 (0.66-0.89) | Townes (2008) reported 44% visit health provider initially, with 35% visiting a health provider during follow-up.[14] Abelson assumed 20% visited a GP for 4 visits, based on Hannu[15] |
| Haemolytic uraemic syndrome | 3 (1-5) | Abelson assumed 3 visits per patient |
| Irritable bowel syndrome | 4.5 (4.27-4.73) | Abelson assumed 4.5 visits per patient based on BEACH data Flik (2015) estimated around 1-2 additional GP visits post diagnosis based on health insurance claims data from the Netherlands[16] |
| *Listeria monocytogenes* | 2 (1-3) | Abelson assumed 2 visits per patient following acute illness |
| *Salmonella enterica* serovar Typhi | 2 (1-3) | Abelson, Scallan (2015), and Hoffman (2014) did not consider *S. enterica ser.* Typhi As for *Listeria*, we assume 2 visits per patient |
| *Toxoplasma gondii* | 0.2 (0-0.4) | Abelson assumed 0.2 visits per symptomatic case |

**Table 3:** Assumptions concerning GP visits for sequel illnesses and pathogens not causing gastroenteritis. Prior assumptions adopted by Abelson were based on advice from clinicians and other experts[3].

***Ongoing illness***

To allow for costs relating to possible ongoing illness, such as specialist visits, further tests, and rehabilitation, we included a node in trees for *L. monocytogenes*, *T. gondii*, and the four sequel illnesses: GBS, HUS, IBS, and ReA. These additional costs may also include further GP visits or hospitalisations beyond those calculated for an individual without ongoing illness, and will vary by pathogen and sequelae. These are described as "new" ongoing illnesses in trees and tables to clarify that this represents ongoing illness associated with incident cases and will capture all costs associated with ongoing illness over the patient's lifetime. We relied on literature and expert opinion to assess the proportion of cases that are associated with ongoing illness. Table 4 provides a table of our assumptions concerning the proportion of cases requiring longer-term care by illness with references supporting these assumptions. For reactive arthritis, we found little evidence of illness following gastroenteritis persisting over one year, but evidence that illness often persists over three months. We define ongoing illness for this sequel illness as symptoms at three months.

| Illness | Ongoing conditions | Proportion of cases with ongoing illness (95% Confidence Interval) | References |
|---|---|---|---|
| Guillain-Barré syndrome | Permanent disability | <5: 7.5% (6.5-8.5)<br>5-64: 16% (14-18)<br>65+: 49% (47-50) | Extrapolated from Frenzen (2008)[13] |
| Reactive arthritis | Continuing symptoms at three months | 50% (23%-77%) | Leirisalo-Repo (1997)[17]<br>Hannu (2005)[18] |
| Haemolytic uraemic syndrome | Chronic renal failure (at 12 months)<br>End stage renal disease (at 12 months) | 16% (8%-27.7%)<br>4.8% (1%-13.5%) | Elliott (2001)[19]<br>Elliott (2001)[19] |
| Irritable bowel syndrome | Continuing symptoms at 12 months | 42.9% (21.8%-66.0%) | Marshall (2007)[20] |
| Congenital listeriosis | Long-term neurological sequelae | 6.6% (3.4%-10.4%) | de Noordhout (2014)[21] |
| Non-congenital listeriosis | Long-term neurological sequelae | 4.2% (1.2%-7.4%) | de Noordhout (2014)[21] |
| Congenital toxoplasmosis | Chorioretinitis in the first year of life:<br>Chorioretinitis later in life:<br>intracranial calcifications:<br>hydrocephalus:<br>central nervous system abnormalities: | 14% (6.6-25)<br>16.9% (2.9-76)<br>11.4% (5–20.2)<br>1.9% (0.67–4.4)<br>2.7% (0.46–9.6) | Havelaar (2007)[22] |

**Table 4:** Probability of ongoing conditions following foodborne illnesses

***Hospitalisations***

We estimated the number of hospitalisations due to contaminated food by age group for the pathogens and illnesses in Table 1 using separation statistics by principal diagnosis for the financial years 2011-12, 2012-13, and 2013-2014 provided by the Australian Institute of Health and Welfare (AIHW)[23]. Diagnostic codes used were based on the Australian modification of the 10th International Classification of Diseases and are detailed in Technical Appendix 3 of Kirk et al[1] and Technical Appendix 4 of Ford et al[2], with all codes for pathogens causing gastroenteritis (including those for gastroenteritis due to unknown causes) used to estimate hospitalisations from gastroenteritis. The only exception to this was the use of data from 2012-13 only for *Shigella* spp. to ensure consistency with our incidence estimates for that pathogen.

For the circa 2010 study, we used State and Territory data (including principal and additional diagnoses) to estimate total hospitalisations, imputing missing data for some States for some years. As only principal diagnosis data are available online from AIHW, we imputed the number of additional diagnoses for each pathogen based on the percentage of diagnoses that were listed as principal for each pathogen from this State data (Technical Appendix 3)[1]. We cannot directly compare the State and Territory data with AIHW data for the same period, as State and Territory hospitalisation data were provided by calendar year and AIHW data were reported by financial year. To avoid overestimating the number of hospitalisations due to gastroenteritis of unknown cause, we used a conservative assumption when estimating the number of non-principal diagnoses. Using data circa 2010, we compared all diagnoses (principal and non-principal) of gastroenteritis due to unknown cause provided from States and Territories to the number of principal diagnoses of gastroenteritis of unknown cause from the AIHW dataset for the same years. This provided the estimate that the AIHW dataset of principal diagnoses captures 71% of all hospitalisations (principal and non-principal) due to gastroenteritis of unknown origin. We applied this proportion to principal hospitalisations data from the AIHW for 2013-2015 to estimate the total number of hospitalisations for gastroenteritis of unknown origin.

***Deaths***

We estimated the number of deaths due to contaminated food for the three age groups (<5, 5-65, 65+) using data from the Australian Bureau of Statistics on the annual number of deaths from underlying or contributing cause in males and females aged 0-14, 15-64, and 65+ from 2001-2010. Diagnostic codes used were based on the 10$^{th}$ International Classification of Diseases and, like hospitalisations, are detailed in Technical Appendix 3 of Kirk et al[1] and Technical Appendix 4 of Ford et al[2], with all codes for pathogens causing gastroenteritis used to estimate hospitalisations from gastroenteritis. Additionally, we used OzFoodNet annual reports over 2001-2011 to estimate the proportion of congenital listeriosis cases that resulted in neonatal or foetal deaths, and applied this to total listeriosis cases circa 2015 to estimate congenital deaths.

## Estimation Approach

With a few exceptions (discussed below), we used the same estimation approach, distributions and multipliers for each pathogen as were used in the circa 2010 estimation study[1 2], which in turn built on prior work for Australia[24] and the United States[25 26]. We used simulation techniques in @Risk to calculate estimates, using multiple inputs, each with a level of uncertainty. For example, estimates of incidence for pathogens captured by the NNDSS included multipliers to adjust for overseas-acquired cases, for under-reporting, and the proportion of cases that are foodborne. Most multipliers were represented as PERT distributions, while incident cases, hospitalisation and deaths were modelled as discrete distributions. The final output of all calculations was a distribution for the number of cases, GP visits, hospitalisations, ongoing illnesses, or deaths, which we then provide as a median estimate together with a 90% credible interval (CrI). Models were created using these approaches for each age group.

***Incidence***

The incidence of foodborne gastroenteritis was estimated by multiplying the weighted proportion of gastroenteritis for each sex and age group by the Australian population for that sex and age group in 2013-2015 and then multiplying this by the 2010 foodborne proportion (25%; 90% CrI 13%-42%).

For the circa 2010 estimation[1], *T. gondii* estimates were calculated based on a US seroprevalence study[7]. These estimates were adjusted by age and applied to population numbers circa 2015 to produce an estimate for each age group.

In calculating incidence of sequelae circa 2010, we adopted a multiplier approach, building on estimates of incidence of the preceding illnesses. We adjusted this approach for IBS and ReA in children aged less than five. While the Abelson report[3] assumed no cases of these sequelae in children <5, studies suggest that they do occur[27], although they are rare[15 28]. We used the ratio of hospitalisations for IBS and ReA in those aged 5-65 to hospitalisations in those aged 0-4 to adjust our estimates of incidence of these sequelae in children aged 0-4. As our initial sequelae multiplier for IBS was based on studies of adult cases only, there was no need to inflate multipliers for individuals over five to adjust for this change. While the sequelae multiplier for ReA was based on studies that included children, the change in total case numbers due to the adjustment in children aged less than five was sufficiently small that we chose to maintain our original multiplier to allow more direct comparison with the circa 2010 study.

***General Practice (GP) consultations***

For most pathogens causing gastroenteritis, we assumed that a proportion of incident cases consulted their GP, based on data from the NGSII study[10]. For more severe illnesses such as GBS, we allowed for multiple GP visits per case (see Data Sources for more detail).

***Hospitalisations***

Because of the severity of *L. monocytogenes*, *S. enterica* ser. Typhi, GBS, and HUS, all persons with estimated incident cases from contaminated food were assumed to be hospitalised, in line with our previous work[1 2].

***Deaths***

As death data were only available for 2000-2010, we added an additional step to all models estimating foodborne deaths, which multiplied the estimated median rate of foodborne illness for each pathogen or illness by the population for 2013-2015, except for *Shigella* spp. where we adjusted for the 2013 population only, to ensure consistency of our approach for incidence. This adjusted the estimate for changes in population since 2000-2010, although it does not allow us to detect any change in the death rate since 2010. As death data were not available for children aged under five separately, and we had no other information on which to calculate age-specific death rates in children, we assumed one third of deaths in the 0-14 age group occurred in children aged under five, with the remaining two thirds occurring in individuals aged 5-65. The estimation procedure often results in estimates of annual deaths in an age group that were not zero, but were less than one. To capture these rare deaths, we report these estimates as 'fractional' deaths per age group.

## Development of Health Outcome Trees

Outcome trees were produced for each of the prioritised pathogens, drawing on trees developed for the United States[29-31] and those developed by the Foodborne Disease Burden Epidemiology Reference Group (FERG), which was established in 2007 by the World Health Organization to estimate the global burden of foodborne diseases[32]. As before, we note that outcome trees do not reflect a patient's journey, but capture costs of various health outcomes. One slight difference between the approaches of Hoffmann et al[30] and Scallan et al[31] for the United States lies in whether deaths by pathogen are shown to always follow hospitalisation or shown to occur without hospitalisation. Health outcome trees for the FERG costing study do not include hospitalisations[32]. As we have no data to estimate the probability of death for different health seeking behaviours, and we believe that not all deaths follow hospitalisation, we have included death as a possible outcome following other health states without specifying the preceding state, and calculate the probability of death as the ratio of deaths to all incident cases.

Health outcome trees include a node labelled 'No medical care'. We assume that this outcome may include costs associated with self-medication using over-the-counter medications, or costs associated with time away from work, but does not include any costs associated with consultation with a medical practitioner. For conditions that may involve longer-term illness, we include a node for 'ongoing illness' that includes ongoing effects of the illness, allowing for visits to specialists and other health professionals (e.g. physiotherapists).

Sequel illnesses were included in the health outcome trees for relevant pathogens causing the initial illness in order to more easily show the full costs by pathogen. As a consequence of this, the burden due to sequelae, such as IBS and ReA, are included in health outcomes for more than one pathogen. They can be summed to calculate a total by sequel.

For those pathogens not prioritised by the tool, recommendations are made as to the most appropriate tree as a reference.

# Estimates and Health outcome trees for gastroenteritis and prioritised pathogens

## Gastroenteritis

We adopt a health outcome tree for foodborne gastroenteritis due to all causes (Figure 1) that was equivalent to that used by Scallan et al[31] for norovirus, with estimates for the nodes in Table 5. This tree captures gastroenteritis due to unknown causes, a large component of total estimates circa 2010. Note that total GP visits are less than those reported for the NGSII survey[11], since these are for foodborne gastroenteritis only.

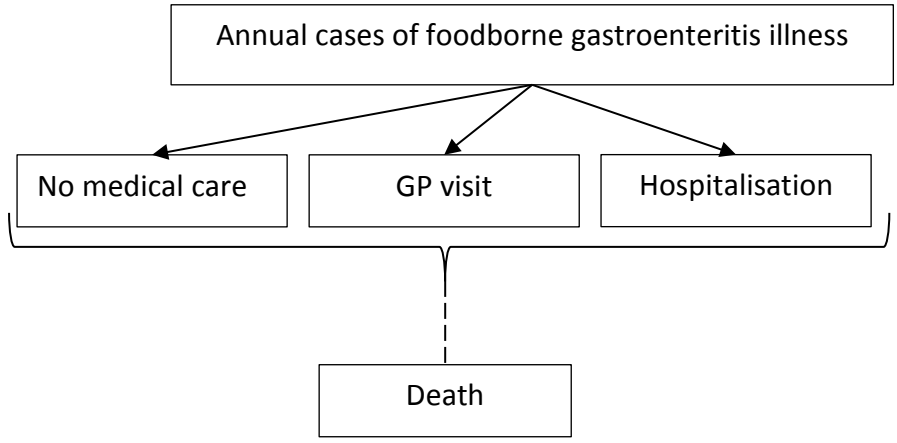


**Figure 1:** Health outcome tree for gastroenteritis due to all causes; dashed lines indicate that death may follow the preceding states

| | Age group | Incidence | GP visits | Hospitalisations | Deaths |
|---|---|---|---|---|---|
| Gastroenteritis | <5 | 602,255 (331,270-992,508) | 117,252 (62,975-200,599) | 5,928 (5,344-6,514) | 0.93 (0.71-1.22) |
| | 5-65 | 3,664,943 (2,915,558-4,579,189) | 716,990 (530,656-965,403) | 26,445 (25,577-27,340) | 8.9 (7.1-11.1) |
| | 65+ | 135,607 (71,942-230,774) | 13,328 (12,659-14,016) | 13,328 (12,659-14,017) | 46 (35-59) |
| **TOTAL** | | 4,427,598 (3,607,276-5,404,157) | 868,929 (669,169-1,125,255) | 45,705 (44,442-46,997) | 56 (45-69) |

**Table 5:** Estimates of the annual incidence, GP visits, hospitalisations and deaths for foodborne gastroenteritis with 90% credible intervals, Australia circa 2015

## *Campylobacter*

We use a health outcome tree (Figure 2) for *Campylobacter* spp. that includes the sequel illnesses GBS (as in Batz et al[29] and Scallan et al[31]), as well as ReA and IBS. We assumed there were no deaths due to ReA. We assumed GP visits for GBS occurred in the context of ongoing illness. Node estimates are provided in Table 6.

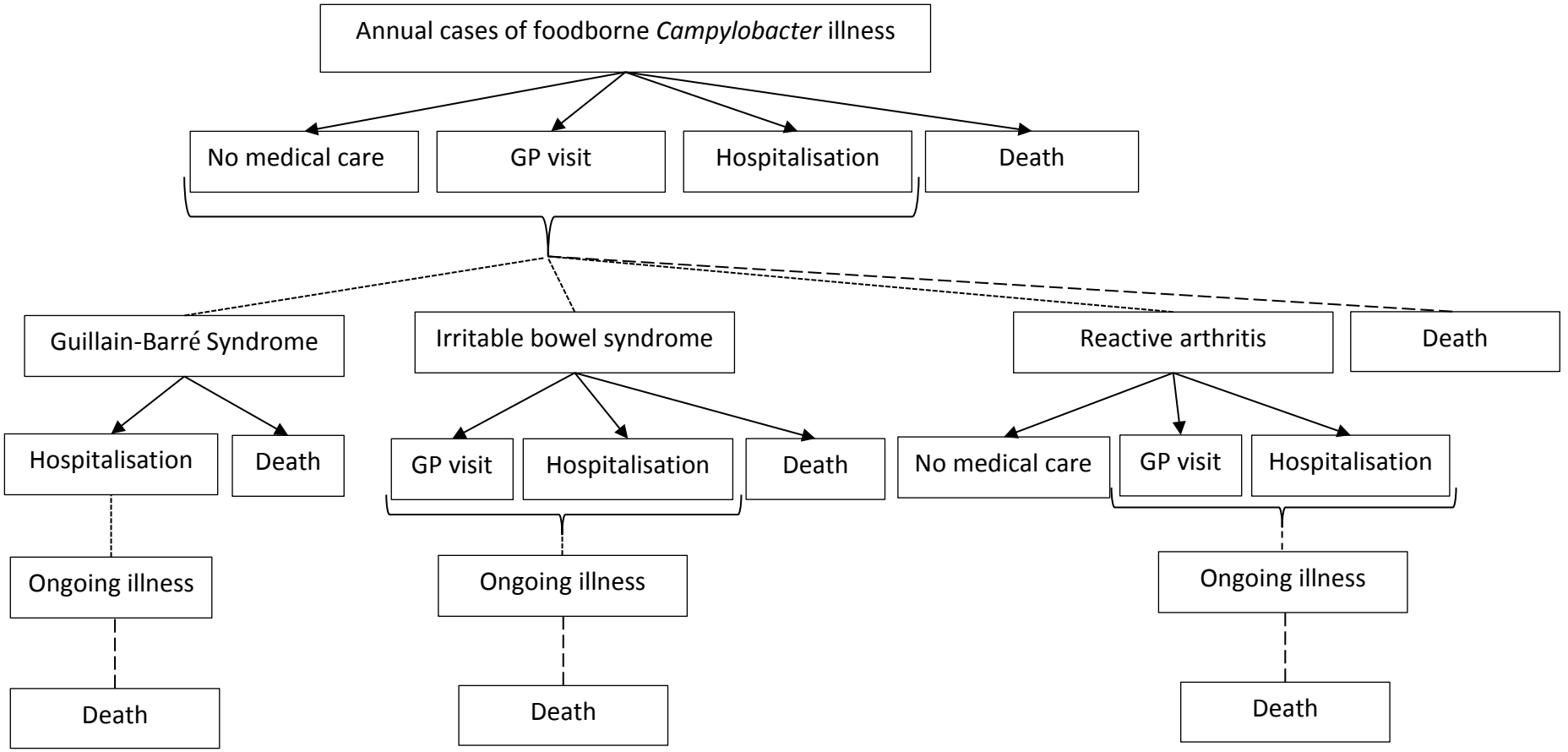


**Figure 2:** Health outcome tree for *Campylobacter* spp.; dotted lines indicate the potential for these sequel illnesses to follow acute illness, and for ongoing illness to result from sequelae, while dashed lines indicate that death may follow the preceding state

| | Age group | Incidence | GP visits | Hospitalisations | New ongoing illness | Deaths |
|---|---|---|---|---|---|---|
| *Campylobacter* | <5 | 22,609 (15,140-33,391) | 8,270 (5,240-12,968) | 160 (119-205) | - | 0.22 (0.16-0.29) |
| | 5-65 | 154,658 (136,782-174,294) | 56,915 (43,576-71,596) | 1,734 (1,585-1,891) | - | 1.35 (1.03-1.70) |
| | 65+ | 38,750 (31,731-47,154) | 14,211 (10,518-18,935) | 1,112 (980-1,255) | - | 2.05 (1.55-2.60) |
| **TOTAL** | | 217,023 (195,432-240,047) | 79,635 (61,051-101,247) | 3,009 (2,800-3,224) | - | 3.6 (3.0-4.3) |
| Guillain-Barré | <5 | 9 (5-15) | 31 (18-54) | 9 (5-15) | 0.64 (0.36-1.14) | 0 |
| | 5-65 | 60 (51-72) | 218 (184-258) | 60 (51-72) | 9.6 (7.8-11.9) | 1.2 (0.7-1.8) |
| | 65+ | 15 (12-20) | 54 (42-72) | 15 (12-20) | 54 (42-72) | 6.1 (3.4-9.3) |
| Irritable Bowel | <5 | 1.58 (1.03-2.39) | 7 (5-11) | 0.63 (0-1.26) | 3.26 (1.52-5.96) | 0 (0-0) |
| | 5-65 | 12,964 (11,391-14,823) | 58,307 (50,750-67,290) | 797 (689-900) | 25,268 (12,717-39,946) | 0.13 (0.09-0.18) |
| | 65+ | 3,408 (2,755-4,189) | 15,341 (12,336-18,957) | 155 (125-191) | 6,587 (3,283-10,720) | 1.24 (0.86-1.76) |
| Reactive arthritis | <5 | 1,133 (897-1,418) | 895 (668-1,160) | 4.1 (1.8-6.8) | 445 (224-720) | - |
| | 5-65 | 12,100 (9,616-15,168) | 9,559 (7,215-12,447) | 48.5 (39.0-58.3) | 4,732 (2,411-7,680) | - |
| | 65+ | 3,006 (2076-4,286) | 2,365 (1,581-3,488) | 5.4 (3.4-7.5) | 1,170 (566-2,055) | - |

**Table 6:** Estimates of the annual incidence, GP visits, hospitalisations, ongoing illnesses, and deaths due to foodborne *Campylobacter* spp. and its sequelae with 90% credible intervals, Australia circa 2015

### *Listeria monocytogenes*

The tree distinguishes between congenital and adult cases of invasive listeriosis (Figure 3), with data in Table 7. This outcome tree is consistent with that of Hoffmann et al[30], Scallan et al[31] and FERG[32]. We assume GP visits occur in the context of ongoing illness.

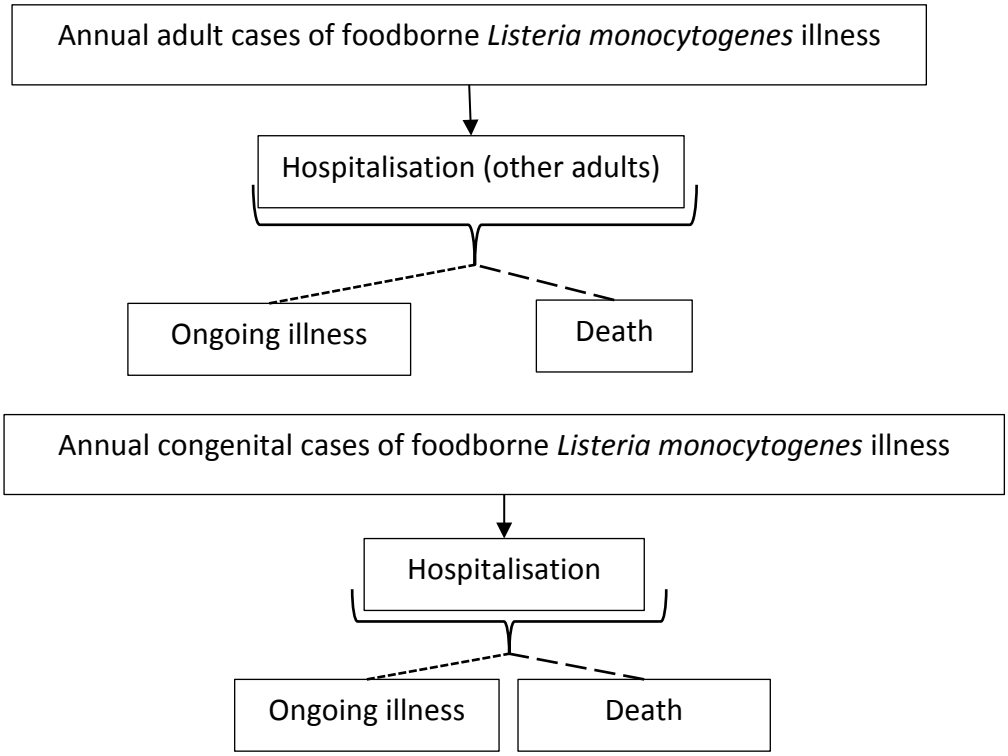


**Figure 3:** Health outcome tree for *Listeria monocytogenes*; dotted lines indicate potential for ongoing illness following hospitalisation, while dashed lines indicate that death may follow hospitalisation

| | Age group | Incidence | GP visits | Hospitalisations | New ongoing illness | Deaths |
|---|---|---|---|---|---|---|
| *Listeria monocytogenes* | congenital | 7 (0-24) | 15.6 (3.9-36.6) | 7 (0-24) | 0.52 (0.13-1.23) | 2 (0-8) |
| | 0-4 | 9 (5-17) | 17.6 (6.9-38.9) | 9 (5-17) | 0.36 (0.09-0.91) | 0 (0-0) |
| | 5-65 | 52 (37-69) | 102 (48-172) | 52 (37-69) | 2.14 (0.6-4.2) | 3.3 (2.5-4.0) |
| | 65+ | 89 (71-110) | 176 (85-283) | 89 (71-110) | 3.69 (1.04-6.91) | 11 (8-13) |
| **TOTAL** | | 160 (133-187) | 318 (216-432) | 160 (133-187) | 7 (4-10) | 17 (13-20) |

**Table 7:** Estimates of the annual incidence, GP visits, hospitalisations, ongoing illnesses and deaths due to foodborne *Listeria monocytogenes* with 90% credible intervals, Australia circa 2015

## Norovirus

We adopt the health outcome tree used by Scallan et al[31] for norovirus (Figure 4), with estimates for the nodes in Table 8.

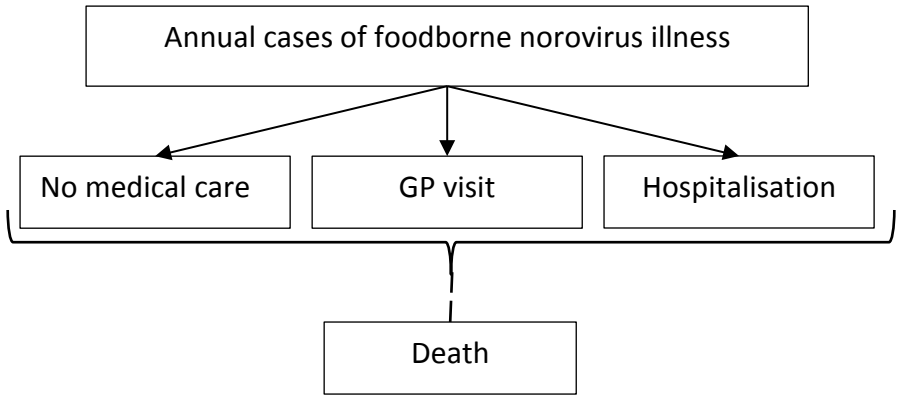


**Figure 4:** Health outcome tree for norovirus; dashed lines indicate that death may follow the preceding states

| | Age group | Incidence | GP visits | Hospitalisations | Deaths |
|---|---|---|---|---|---|
| Norovirus | <5 | 57,986 (12,740-126,688) | 11,312 (2,452-25,599) | 116 (31-256) | 0 (0-0) |
| | 5-65 | 234,732 (64,835-491,271) | 45,958 (12,527-99,551) | 87 (23-207) | 0.17 (0.05-0.38) |
| | 65+ | 8,631 (2,305-19,559) | 1,691 (446-3,915) | 100 (25-250) | 0.56 (0.15-1.19) |
| **TOTAL** | | 307,997 (125,564) | 59,933 (24,856-114,334) | 329 (165-535) | 0.75 (0.3-1.4) |

**Table 8:** Estimates of the annual incidence, GP visits, hospitalisations and deaths for foodborne norovirus with 90% credible intervals, Australia circa 2015

## *Salmonella*

We use a health outcome tree (Figure 5) for *Salmonella* that includes reactive arthritis and irritable bowel syndrome as sequel illnesses. Estimates for these nodes are shown in Table 9. As before, we assumed there were no deaths due to ReA.

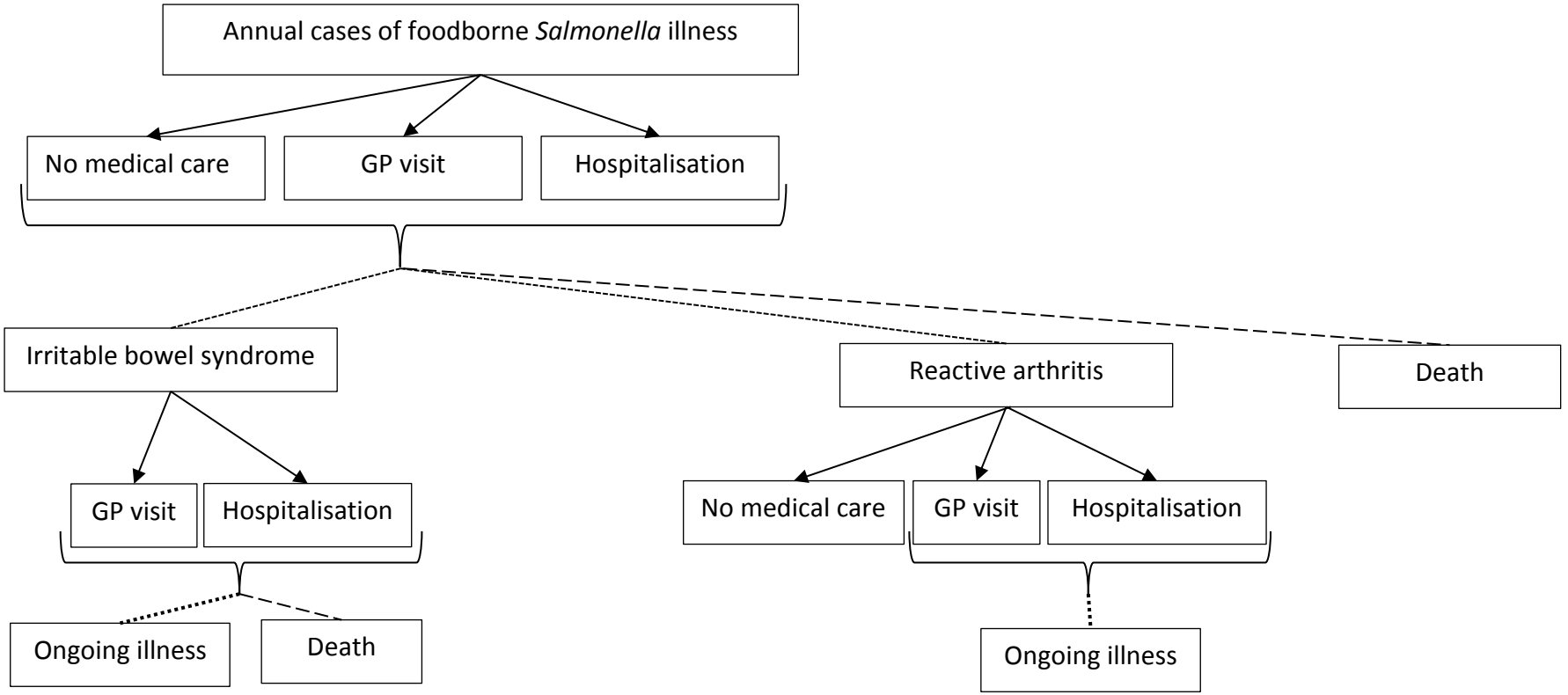


**Figure 5:** Health outcome tree for *Salmonella*; dotted lines indicate potential sequelae following acute illness, and potential ongoing illness following GP consultation or hospitalisation for a sequel illness, while dashed lines indicate that death may follow the preceding state

| | Age group | Incidence | GP visits | Hospitalisations | New ongoing illness | Deaths |
|---|---|---|---|---|---|---|
| *Salmonella* | <5 | 15,200 (9,750-23,450) | 5,556 (3,420-9,030) | 535 (386-720) | - | 0.5 (0.3-0.6) |
| | 5-65 | 44,288 (38,830-50,758) | 16,267 (12,524-20,804) | 1,354 (1,212-1,506) | - | 3.5 (2.7-4.5) |
| | 65+ | 8,045 (6,507-9,947) | 2,955 (2,168-3,982) | 508 (428-604) | - | 9.0 (6.5-11.6) |
| **TOTAL** | | 68,073 (59,562-78,090) | 25,063 (19,135-31,747) | 2,406 (2,174-2,651) | - | 13 (10-16) |
| Irritable Bowel syndrome | <5 | 1.06 (0.69-1.63) | 5 (3-7) | 0.2 (0-0.4) | 2.1 (0.96-3.94) | 0 (0-0) |
| | 5-65 | 3,895 (3,425-4,475) | 17,535 (15,226-20,310) | 256 (224-289) | 7609 (3848-12,041) | 0 (0-0.1) |
| | 65+ | 705 (575-875) | 3,173 (2,572-3,965) | 50 (40-62) | 1,374 (682-2,252) | 0.4 (0.3-0.6) |
| Reactive arthritis | <5 | 370 (292-461) | 292 (220-379) | 1.5 (0.6-2.4) | 145 (73-234) | - |
| | 5-65 | 3,930 (3,180-4,950) | 3,106 (2,360-4,053) | 17.2 (13.8-20.6) | 1,549 (788-2,516) | - |
| | 65+ | 715 (485-1,011) | 561 (371-820) | 1.9 (1.2-2.6) | 275 (134-492) | - |

**Table 9:** Estimates of the annual incidence, GP visits, hospitalisations, ongoing illnesses, and deaths due to foodborne *Salmonella* and its sequelae with 90% credible intervals, Australia circa 2015

## *Shigella*

We adapt the health outcome tree used by Hoffmann et al[30] for *Shigella* spp.(Figure 6) to include reactive arthritis and irritable bowel syndrome as potential sequel illnesses, assuming as before that there are no deaths due to reactive arthritis. Estimates for the nodes are provided in Table 10.

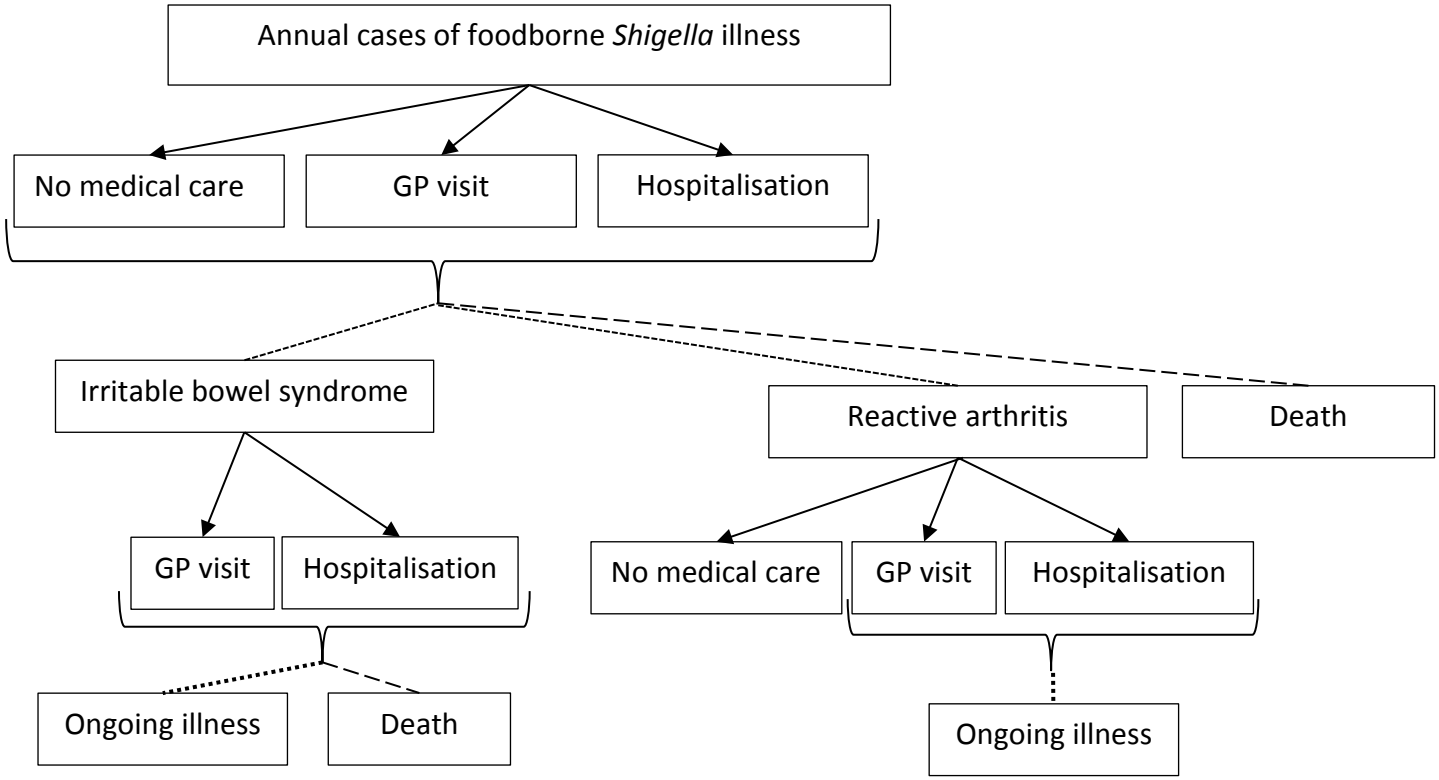


**Figure 6:** Health outcome tree for *Shigella* spp.; dotted lines indicate potential sequelae following acute illness, and ongoing illness following GP visit or hospitalisation for reactive arthritis, while dashed lines indicate that death may follow the preceding state

| | Age group | Incidence | GP visits | Hospitalisations | New ongoing illness | Deaths |
|---|---|---|---|---|---|---|
| *Shigella* | <5 | 66 (33-123) | 24 (12-47) | 7 (3-12) | - | 0.01 (0-0.02) |
| | 5-65 | 258 (208-316) | 95 (69-127) | 16 (13-17) | - | 0.03 (0.01-0.05) |
| | 65+ | 15 (10-22) | 6 (4-9) | 2 (1-3) | - | 0 (0-0) |
| **TOTAL** | | 344 (278-420) | 126 (90-170) | 26 (21-32) | - | 0.04 (0.02-0.07) |
| Reactive arthritis | <5 | 5 (3-10) | 4 (2-8) | 0.006 (0.003-0.009) | 2 (1-5) | - |
| | 5-65 | 21 (17-26) | 17 (13-22) | 0.003 (0.002-0.004) | 8 (4-14) | - |
| | 65+ | 1.24 (0.82-1.89) | 0.98 (0.63-1.52) | 0.008 (0.005-0.01) | 0.5 (0.2-0.9) | - |
| Irritable Bowel syndrome | <5 | 6 (3-11) | 27 (13-50) | 0 (0-0) | 11.1 (2.5-24.9) | 0 (0-0) |
| | 5-65 | 23 (18-28) | 103 (80-127) | 1.05 (0.92-1.19) | 44 (22-73) | 0 (0-0) |
| | 65+ | 1.32 (0.89-1.97) | 6 (4-9) | 0.16 (0.1-0.24) | 2.6 (1.2-4.8) | 0 (0-0) |

**Table 10:** Estimates of the annual incidence, GP visits, hospitalisations, ongoing illnesses, and deaths due foodborne *Shigella* spp. and its sequelae with 90% credible intervals, Australia circa 2013

## Shiga toxin-producing *Escherichia coli* (STEC)

We use a health outcome tree (Figure 7) for Shiga toxin-producing *E. coli* (STEC) that includes sequel illness due to haemolytic uraemic syndrome (HUS) and is similar to those of Scallan et al[31] and Hoffmann et al[30]. Estimates for these nodes are shown in Table 11. We assume GP visits for HUS occur in the context of ongoing illness.

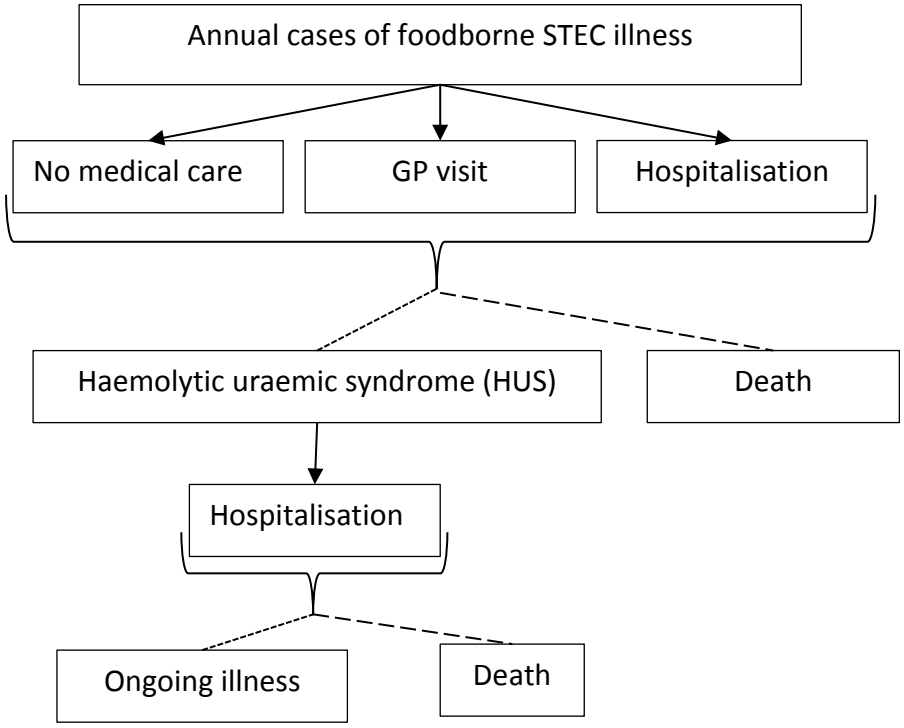


**Figure 7:** Health outcome tree for Shiga toxin-producing *Escherichia coli* (STEC); dotted lines indicate potential sequel illness of haemolytic uraemic syndrome (HUS) following acute illness and potential ongoing illness following hospitalisation for HUS, while dashed lines indicate that death may follow the preceding states

| | Age group | Incidence | GP visits | Hospitalisations | New ongoing illness | Deaths |
|---|---|---|---|---|---|---|
| STEC | <5 | 313 (145-667) | 115 (52-251) | 0 (0-2) | - | 0 (0-0) |
| | 5-65 | 1,893 (1,290-2,725) | 691 (446-1,060) | 4 (0-8) | - | 0 (0-0) |
| | 65+ | 716 (418-1,116) | 262 (147-429) | 0 (0-2) | - | 0 (0-0) |
| **TOTAL** | | 2,978 (2,218-3,914) | 1,095 (758-1,542) | 4 (1-9) | - | 0 (0-0) |
| HUS | <5 | 10 (4-22) | 27 (7-77) | 10 (4-22) | 1.5 (0.5-4.2) | 0 (0-0) |
| | 5-65 | 59 (39-87) | 171 (55-336) | 59 (39-87) | 9.3 (4.2-18.7) | 0.94 (0.63-1.33) |
| | 65+ | 22 (12-36) | 63 (19-138) | 22 (12-36) | 3.4 (1.5-7.6) | 1 (0-1) |

**Table 11:** Estimates of the annual incidence, GP visits, hospitalisations, ongoing illnesses, and deaths due to foodborne STEC and its sequelae with 90% credible intervals, Australia circa 2015

## Other (non Shiga toxin-producing) pathogenic *Escherichia coli* (*E. coli*)

The health outcome tree for other (non-STEC) pathogenic *E. coli* is shown in Figure 8, with estimates for the nodes in Table 12. Neither Scallan et al[31] nor Hoffmann et al[30] include health outcome trees for non-STEC pathogenic *E. coli*. The FERG study includes both incidence and deaths due to diarrhoeal disease due EPEC and ETEC, but no ongoing illness or GP visits associated with the illness[32].

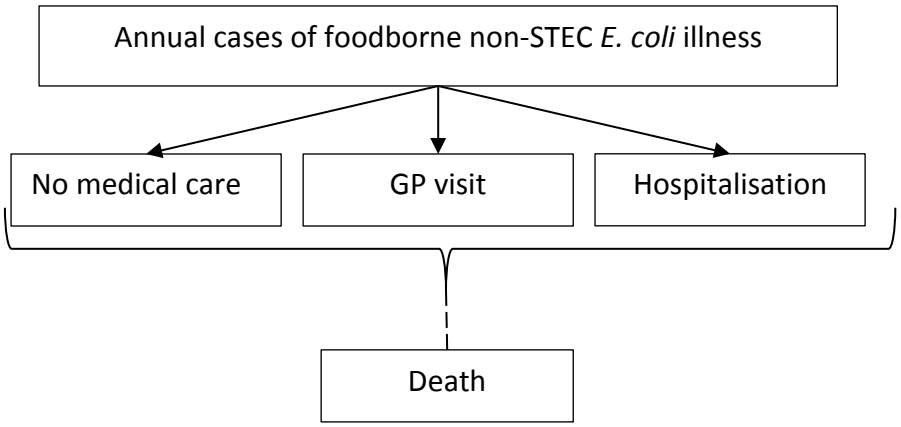


**Figure 8:** Health outcome tree for other (non-STEC) pathogenic *E. coli*; dashed lines indicate that death may follow the preceding states

| | Age group | Incidence | GP visits | Hospitalisations | Deaths |
|---|---|---|---|---|---|
| *E. coli* | <5 | 32,508 (8,188-108,376) | 14,265 (5,598-28,000) | 1 (0-5) | 0 (0-0) |
| | 5-65 | 230,335 (77,985-576,913) | 84,314 (27,841-216,695) | 15 (5-40) | 0 (0-0) |
| | 65+ | 8,411 (2,738-23,192) | 3,083 (991-8,732) | 12 (4-33) | 0 (0-0) |
| **TOTAL** | | 281,987 (127,219-626,934) | 102,327 (44,675-240,503) | 31 (14-61) | 0 (0-0) |

**Table 12:** Estimates of the annual incidence, GP visits, hospitalisations and deaths due to foodborne other (non-STEC) pathogenic *E. coli* with 90% credible intervals, Australia circa 2015

## *Toxoplasma gondii*

The outcome trees presented in Hoffmann et al[30], Scallan et al[31] and FERG[32] distinguish between congenital and adult cases. Due to insufficient data applicable to Australia, we do not explicitly model congenital cases (Figure 9). Data are provided in Table 13.

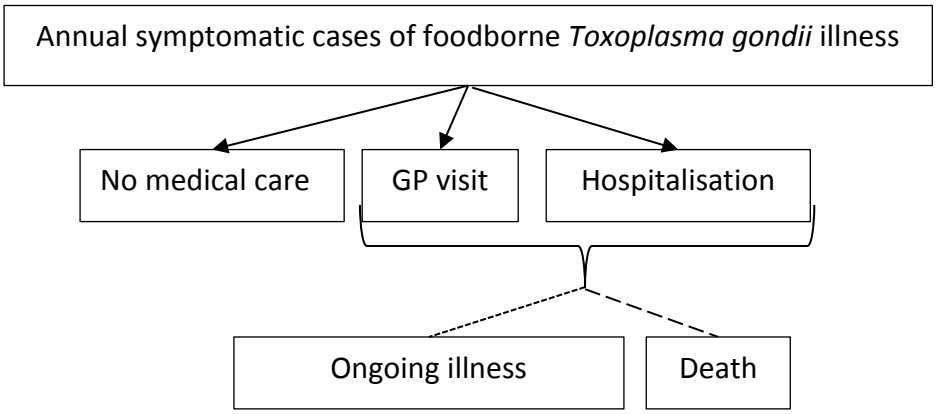


**Figure 9:** Health outcome trees for *Toxoplasma gondii*; dotted lines indicate the potential for ongoing illness to follow health care; dashed lines indicate that death may follow the preceding states

| | Age group | Incidence | GP visits | Hospitalisations | New ongoing illness | Deaths |
|---|---|---|---|---|---|---|
| *Toxoplasmosis* | 0-4 | 463 (249-755) | 86 (0-221) | 0 (0-0) | 2 (1-4) | 0.02 (0.01-0.04) |
| | 5-65 | 2,290 (1,700-3,016) | 452 (0-978) | 22 (13-33) | 11 (6-18) | 1.09 (0.57-1.76) |
| | 65+ | 181 (129-241) | 35 (0-78) | 3 (0-7) | 0.9 (0.5-1.4) | 0.16 (0.09-0.26) |
| **TOTAL** | | 2,944 (2,299-3,716) | 583 (172-1,065) | 25 (16-37) | 15 (10-21) | 1.3 (0.75-2.0) |

**Table 13:** Estimates of the annual incidence, GP visits, hospitalisations, ongoing illnesses and deaths due to foodborne *Toxoplasma gondii* with 90% credible intervals, Australia circa 2015

## *Salmonella enterica* serovar Typhi

Neither Scallan et al[31], nor Hoffman et al[30] develop health outcome trees for *S. enterica* ser. Typhi. The FERG outcome trees for *S. enterica* ser. Typhi consider incidence, deaths, and cysts and liver abscesses[32]. In line with our earlier work[1], we assume that all incident cases are hospitalised (Figure 10), with data provided in Table 14, and assume GP visits occur to this group following hospitalisation.

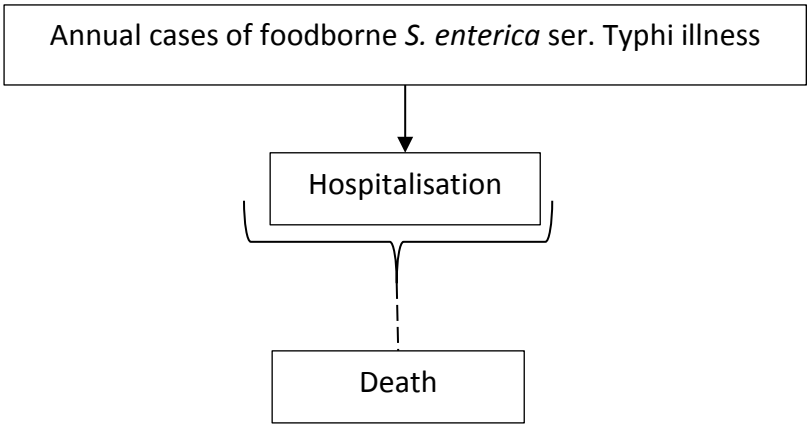


**Figure 10:** Health outcome tree for *S. enterica* ser. Typhi; dashed lines indicate that death may follow hospitalisation

| | Age group | Incidence | Hospitalisations | GP visits | Deaths |
|---|---|---|---|---|---|
| *S. enterica* ser. Typhi | <5 | 3 (1-5) | 3 (1-5) | 5 (3-9) | 0 (0-0) |
| | 5-65 | 17 (13-22) | 17 (13-22) | 34 (22-51) | 0.03 (0.02-0.05) |
| | 65+ | 0.07 (0-0.35) | 0.07 (0-0.35) | 0.19 (0.04-0.45) | 0.04 (0.02-0.06) |
| **TOTAL** | | 20 (16-25) | 20 (16-25) | 40 (24-60) | 0.08 (0.05-0.11) |

**Table 14:** Estimates of the annual incidence, GP visits, hospitalisations and deaths due to foodborne *S. enterica* ser. Typhi with 90% credible intervals, Australia circa 2015

### *Yersinia enterocolitica*

Our health outcome tree for *Yersinia enterocolitica* is similar to that of Hoffmann et al[30], but also includes potential sequelae due to ReA (Figure 11) assuming as before that there are no deaths due to ReA. Estimates for the nodes are provided in Table 15.

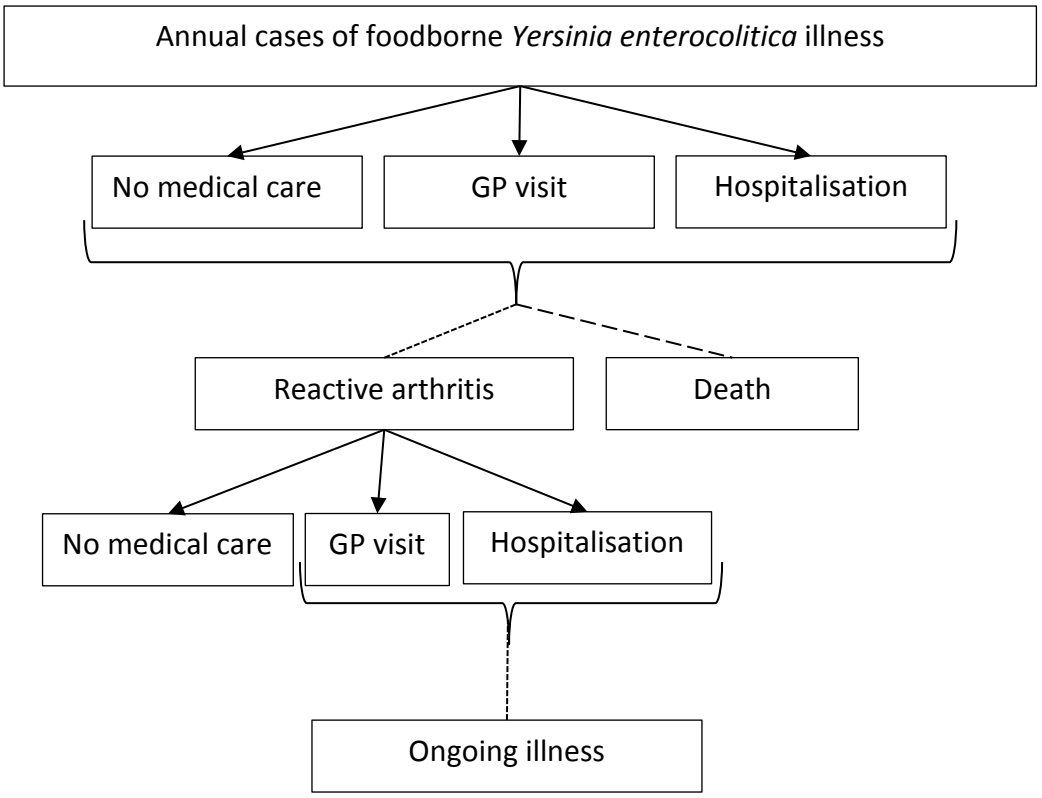


**Figure 11:** Health outcome tree for *Yersinia enterocolitica*; dotted lines indicate potential sequelae following acute illness, and potential for ongoing illness to follow health care for reactive arthritis, while dashed lines indicate that death may follow the preceding states

| | Age group | Incidence | GP visits | Hospitalisations | New ongoing illness | Deaths |
|---|---|---|---|---|---|---|
| *Yersinia* | <5 | 1,462 (639-2,614) | 533 (227-1,003) | 9 (0-18) | - | 0 (0-0) |
| | 5-65 | 5,882 (4,444-7,449) | 2,148 (1,510-2,955) | 21 (11-33) | - | 0.31 (0.19-0.45) |
| | 65+ | 3,353 (2,039-4,899) | 1,223 (714-1,902) | 5 (1-11) | - | 0 (0-0) |
| **TOTAL** | | 10,813 (8,534-13,167) | 3,942 (2,893-5,228) | 36 (21-52) | - | 0.31 (0.19-0.45) |
| Reactive arthritis | <5 | 48 (34-64) | 83 (17-171) | 0.18 (0.08-0.31) | 19 (9-32) | - |
| | 5-65 | 518 (390-652) | 406 (297-535) | 2.2 (1.8-2.6) | 2001 (102-331) | - |
| | 65+ | 151 (111-204) | 120 (84-166) | 0.24 (0.15-0.33) | 59 (29-100) | - |

**Table 15:** Estimates of the annual incidence, GP visits, hospitalisations, ongoing illnesses, and deaths due to foodborne *Yersinia* spp. and its sequelae with 90% credible intervals, Australia circa 2015

## Health Outcome Trees and recommendations for non-prioritised pathogens

There are several pathogens that were not prioritised by experts. We describe health outcome trees for pathogens causing gastroenteritis and other pathogens separately.

Pathogens causing gastroenteritis that were included in the 2010 burden of disease study[1] but not prioritised by experts for development of specific outcome trees are:

- Adenovirus
- Astrovirus
- *Bacillus cereus*
- *Clostridium perfringens*
- *Cryptosporidium*
- *Giardia lamblia*
- Rotavirus
- Sapovirus
- *Staphylococcus aureus*
- *Vibrio parahaemolyticus*

Combined, these pathogens are estimated to account for an estimated one death and 241 hospitalisations each year circa 2010[1]. We propose the following health outcome tree for these pathogens causing gastroenteritis (Figure 12) which has the same structure to that for norovirus. This tree is consistent with the health outcome tree for *C. perfringens* produced by Scallan et al[31], and the trees produced for *C. perfringens*, *Cryptosporidium*, and *V. parahaemolyticus* by Hoffmann et al[30] except for the inclusion of diarrhoea relapse for *Cryptosporidium* by Hoffmann[30]. Neither of these studies considered the other pathogens listed above. The FERG study included health outcome trees for *B. cereus*, *Cryptosporidium*, *C. perfringens*, *Giardia* spp., and *S. aureus*, however these trees included either incidence only (*B. cereus*, *Giardia* spp.), or incidence and death only (*S. aureus*, *C. perfringens*, *Cryptosporidium*)[32].

Although health outcome trees and estimates of health states for these pathogens, and that of gastroenteritis due to unknown causes are not directly calculated here, the estimates for gastroenteritis includes these pathogens, so that total costs can be calculated.

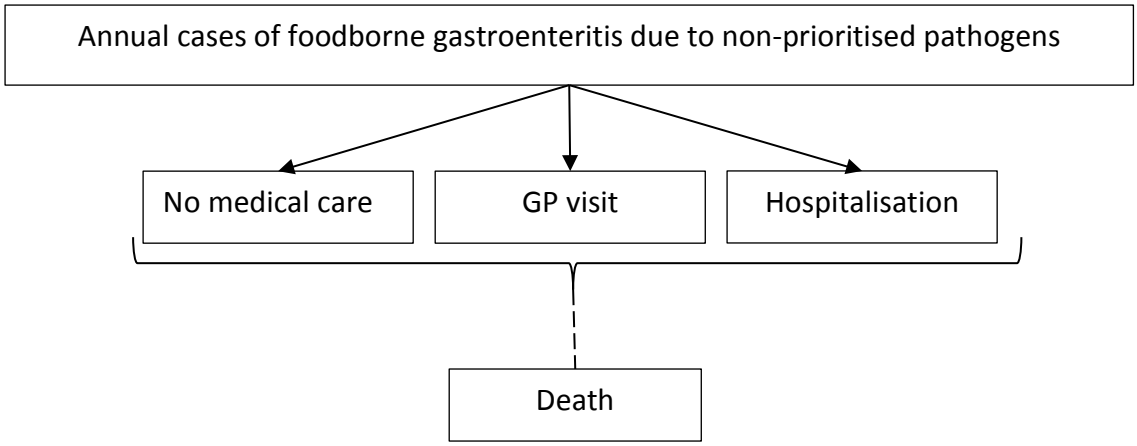


**Figure 12:** Proposed health outcome tree for non-prioritised pathogens causing gastroenteritis; dashed lines indicate that death may follow the preceding states

The remaining identified pathogens (not causing gastroenteritis) that were not prioritised by experts are:

- Hepatitis A
- Ciguatera
- Scombrotoxicosis

Hepatitis A was not considered by either Scallan et al[31] or Hoffmann et al[29] in their costing of foodborne illness. The FERG study includes both incidence and deaths due to Hepatitis A[32]. We propose a health outcome tree for Hepatitis A that is consistent with that for other non-prioritised pathogens (Figure 13), and recommend similar trees for Ciguatera and Scombrotoxicosis. Table 16 and Table 17 provide estimates of incidence, hospitalisations and deaths for Hepatitis A and Ciguatera by age group.

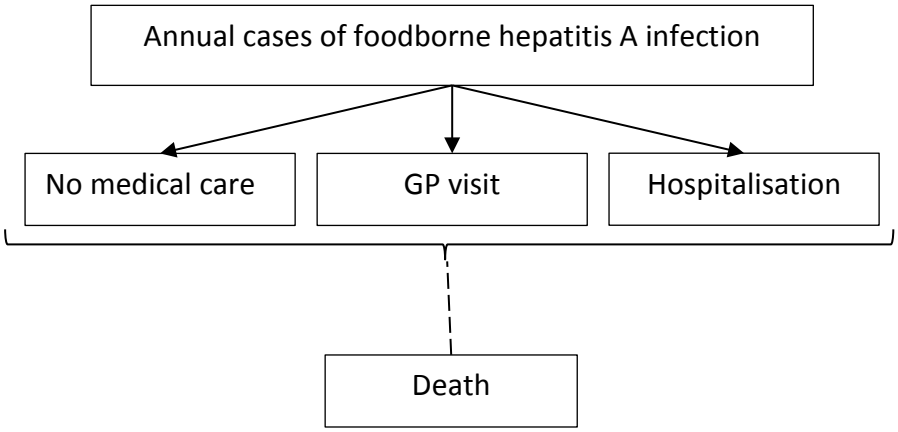


**Figure 13:** Proposed health outcome tree for hepatitis A; dashed lines indicate that death may follow the preceding states

| | Age group | Incidence | Hospitalisations | Deaths |
|---|---|---|---|---|
| Hepatitis A | <5 | 1.9 (0.8-4.2) | 0.5 (0.2-1.2) | 0.02 (0.01-0.04) |
| | 5-65 | 26.7 (21.0-33.8) | 15.8 (11.7-21.3) | 0.17 (0.10-0.31) |
| | 65+ | 1.0 (0.5-1.8) | 1.5 (0.6-2.7) | 0.66 (0.34-1.17) |
| **TOTAL** | | 29.9 (23.9-37.2) | 18.0 (13.7-23.6) | 0.87 (0.52-1.38) |

**Table 16:** Estimates of the burden of disease due to Hepatitis A with 90% credible intervals, Australia circa 2015

| | Age group | Incidence | Hospitalisations | Deaths |
|---|---|---|---|---|
| Ciguatera | <5 | 0 | 0 | 0 |
| | 5-65 | 230 (148-327) | 41 (25-58) | 0 |
| | 65+ | 20 (6-45) | 17 (9-28) | 0 |
| **TOTAL** | | 253 (168-351) | 59 (41-79) | 0 |

**Table 17:** Estimates of the burden of disease due to Ciguatera with 90% credible intervals, Australia circa 2015

## Discussion

This report provides health outcome trees for gastroenteritis due to all causes (including unknown) and 10 pathogens prioritised for economic costing by foodborne disease experts. The top three prioritised pathogens identified in this process were *Salmonella*, *Campylobacter* spp., and *L. monocytogenes*, which are estimated to be the leading causes of death, and three of the four leading causes of hospitalisation circa 2015. *Salmonella* and *Campylobacter* also contribute significantly to the burden of foodborne disease through sequel illnesses.

Comparison of findings circa 2015 with those circa 2010 shows increases in the incidence and hospitalisation rate for both *Salmonella* and *Campylobacter*, together with related increases in GBS, ReA, and IBS. In contrast, incidence and hospitalisation rates for *L. monocytogenes* circa 2015 have decreased when compared with circa 2010. There are several possible reasons for the increasing incidence of *Salmonella* and *Campylobacter*, including increasing disease due to foodborne sources, such as chicken and eggs, and changes in testing practices, including increasing numbers of tests and changing sensitivity of tests in pathology laboratories.

The health outcome trees proposed here are similar to those proposed in United States and global studies of foodborne illness, and the approach to develop estimates for disease states is consistent with that of the 2010 Australian foodborne burden of disease study. Where there have been differences in data sources, approach or assumptions with 2010 or the earlier costing study for Australia, these differences are documented. Note that the outcome trees are not intended to represent a patient's journey through health states or health care, but rather provide estimates of health states to capture costs due to various health outcomes.

There are some limitations of this study. The approach used to prioritise pathogens was based on a single survey of seven experts, and was not formally structured as an expert elicitation. Nevertheless, findings across experts were fairly consistent – all experts ranked *Salmonella*, *Campylobacter*, norovirus, and *L. monocytogenes* in their top ten prioritised pathogens. We were not able to obtain updated data on deaths for this study, and so have assumed death rates circa 2015 are unchanged from those circa 2010. For pathogens such as *Salmonella* and *Campylobacter* spp., which have seen increases in rates of incidence and hospitalisation, this may result in an underestimate of total deaths due to these two pathogens.

We were also not able to obtain data on hospitalisations where a foodborne pathogen was coded as an additional cause of hospitalisation. We attempted to adjust for this using conservative assumptions based on data from States and Territories circa 2010 that included both primary and additional diagnoses, however, this may have resulted in some errors in the estimates of hospitalisations. Due to lack of new data for norovirus, non-STEC

pathogenic *E. coli*, and lack of any Australian data for *T. gondii*, estimates of incidence use the same data as our 2010 study, although numbers have been adjusted for increases in population size.

Changes in testing and reporting practices make it challenging to interpret estimates for *Shigella* spp. and *Y. enterocolitica*. In the case of *Shigella* spp., we chose to restrict data to 2013 only to avoid differences in reporting practices by State and Territory, while the greater than eight-fold increase in estimates of incidence for *Y. enterocolitica* is likely to be driven by increased use and sensitivity of culture-independent testing in 2014 and 2015. Some of this increase in yersiniosis is likely to be a true reflection of the incidence, as previous culturing practices in laboratories has not appropriately selected for this pathogen.

## Conclusions

In this report, we developed health outcome trees and estimates of the burden of disease of prioritised foodborne pathogens, agents and sequelae in Australia with updated data for 2015. We identified increases in the incidence and hospitalisations due to gastroenteritis from all causes, salmonellosis and campylobacteriosis since 2010. Increases in estimates for some illnesses, such as yersiniosis, are likely due to changes in pathology testing of faecal specimens using more sensitive tests. In this report, we used current surveillance and hospitalisation data where possible, but there are gaps for some pathogens and some outcomes. In future efforts it will be important to seek new information sources to ensure that estimates reflect contemporary disease incidence and outcomes. The outcome trees and estimates in this report will form a key component in costing foodborne disease in Australia circa 2015.

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