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Estimated incidence of foodborne illness in New Zealand: Application of overseas models and multipliers

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ESTIMATED INCIDENCE OF FOODBORNE ILLNESS IN NEW ZEALAND: APPLICATION OF OVERSEAS MODELS AND MULTIPLIERS

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the incidence of foodborne disease in New Zealand,
as part of overall contract for scientific services

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SUMMARY

The objective of this project is to use overseas models to estimate the numbers of cases of illness, hospitalisations and deaths due to foodborne agents occurring in a calendar year in New Zealand. The estimates concern illness caused by microbial pathogens only. It should be noted that the applicability of these overseas model to New Zealand is currently uncertain and outputs from this exercise should be viewed as hypothetical. Models were applied to data from the period 2000-2009 and then were repeated, considering only the 2009 year to allow an assessment of changes in the incidence of foodborne illness. For some organisms, two separate overseas models were able to be compared and contrasted.

To produce these estimates, recently published models and results from studies in the United States have been used. These concern 31 major pathogens, as well as foodborne illness caused by "unspecified agents". Unspecified agents were defined as "known agents with insufficient data to estimate agent-specific illness, known agents not yet recognised as causing foodborne illness, substances known to be in food but of unproven pathogenicity, and unknown agents". For the majority of foodborne microbial pathogens the primary outcome of infection is acute gastrointestinal illness (AGI). Consequently, foodborne illness caused by unspecified pathogens is calculated from the incidence of AGI in the community, once the incidence of AGI caused by specified pathogens is estimated and subtracted. Due to data limitations, New Zealand estimates were based on 24 rather than 31 major pathogens.

Application of the US model to the New Zealand situation results in an estimate of approximately 1.4 million cases of illness caused by 24 pathogens per annum, based on data from the period 2000-2009. Of these, just over half a million are estimated to be due to domestically-acquired foodborne transmission. When expressed as a rate, this equates to 129 cases per 1,000 population per annum.

Of the approximately half million cases of domestically acquired foodborne illness, 59% were due to bacteria, 39% due to viruses and only 2% due to parasites. The major contributors being norovirus (39%), *Campylobacter* (34%), *Clostridium perfringens* (12%), *Yersinia enterocolitica* (5%) and non-typhoidal *Salmonella* (4%).

Of the estimated 4,279 hospitalisations due to foodborne illness in New Zealand, 69% were due to viruses, 30% due to bacteria and 1% due to parasites. The pathogens contributing most to hospitalisations due to foodborne illness were norovirus (69%), *Campylobacter* (21%) and non-typhoidal *Salmonella* (4%).

The current study estimated that 17 fatalities would occur in a year due to the 24 major pathogens transmitted by food. Most fatalities (65%) were due to bacteria, with the remainder equally divided between viruses and parasites. The pathogens contributing most to fatalities due to foodborne illness were *Listeria monocytogenes* (35%), norovirus (18%) and *Toxoplasma gondii* (18%).

It was estimated that unspecified agents transmitted by food cause approximately twice as many cases of illness in New Zealand as the 24 known pathogens. Unspecified agents cause more than four times as many hospitalisations as the 24 major pathogens, but only about 40% more fatalities.

For nine of the pathogens (*Clostridium perfringens*, *Campylobacter*, STEC O157, *Salmonella*, *Cryptosporidium*, *Giardia intestinalis*, astrovirus, norovirus and rotavirus) multipliers or population rates from the second Infectious Intestinal Diseases Study (IID2) in Britain were also employed and compared to those based on the US study. Application of the two approaches (US and IID2) to the New Zealand situation generates quite similar estimates for the number of cases of illness due to rotavirus. However, for the other pathogens compared, estimates of illness derived from the US approach were greater by factors ranging from 1.4 to 10.1.

In order to assess the impact of changes in disease incidence during the last decade, all analyses were repeated using New Zealand inputs from the most recent year for which complete data were available (2009) only. This repeat analysis resulted in a 30% decrease in the estimated mean incidence of bacterial disease and an 18% decrease in the estimated total cases of domestically acquired foodborne illness. This is largely due to reductions in notifications for campylobacteriosis in recent years.

1 INTRODUCTION

The objective of this project is to use overseas models to estimate the numbers of cases of illness, hospitalisations and deaths due to foodborne agents occurring in a calendar year in New Zealand. The estimates concern illness caused by microbial pathogens only. It should be noted that the applicability of these overseas model to New Zealand is currently uncertain and outputs from this exercise should be viewed as hypothetical.

To produce these estimates, recently published models and results from studies in the United States (Scallan *et al.*, 2011a; Scallan *et al.*, 2011b) have been used. These concern 31 major pathogens (see Table 11, Appendix 1 for a complete list of these pathogens), as well as foodborne illness caused by "unspecified agents". Unspecified agents were defined as "known agents with insufficient data to estimate agent-specific illness, known agents not yet recognised as causing foodborne illness, substances known to be in food but of unproven pathogenicity, and unknown agents" (Scallan *et al.*, 2011a). For the majority of foodborne microbial pathogens the primary outcome of infection is acute gastrointestinal illness (AGI). Consequently, foodborne illness caused by unspecified pathogens is calculated from the incidence of AGI in the community, once the incidence of AGI caused by specified pathogens is estimated and subtracted.

The estimates in this report represent both an update and an expansion of previous estimates for New Zealand (Lake *et al.*, 2010a). Previous estimates concerned diseases caused by six pathogens only: campylobacteriosis, salmonellosis, listeriosis (invasive; perinatal, and nonperinatal), infection with Shiga toxin-producing *Escherichia coli* (STEC), yersiniosis, and norovirus infection. These estimates used data principally from the years 2000-2005, and have been updated in this report.

In addition, this report provides estimates for as many as possible of the other 25 pathogens addressed by the United States study. For some of these pathogens estimates for New Zealand were not possible (due to lack of data), or not relevant (as data indicated the absence of illness).

Both the United States model and the previous New Zealand estimates took the approach of utilising data from a variety of surveillance sources and scientific studies to provide information on illness incidence at various levels of the reporting "pyramid". The data are scaled up or down by appropriate factors ('multipliers") to complete the estimates at other levels of the pyramid.

Where available, New Zealand data have been used for both the number of cases at levels of the pyramid, and some multipliers. Where multipliers were not available for New Zealand, those derived by the United States model have been used. For nine of the pathogens, multipliers from the recent second Infectious Intestinal Diseases Study (IID2) in Britain have also been employed and compared to those based on the US study (Tam *et al.*, 2011).

2 METHODS

Models were applied using the Excel add-in @Risk (Palisades Corporation). All models were run for 100,000 iterations.

2.1 Application of US Model to New Zealand

The current study followed the published methodology for the US study (Scallan *et al.*, 2011a; Scallan *et al.*, 2011b), including the additional detail in the technical appendices to these papers[1]. Details of where New Zealand specific data were used are given in Appendix 1.

2.1.1 Multipliers

The US model applied two multipliers to observed case numbers, to arrive at an estimate for total community cases. These were:

- Under-reporting multiplier. This was set at one for active surveillance, 1.1-1.3 for passive surveillance and 25.5 for cases from outbreak surveillance.
- Under-diagnosis multiplier. This covers aspects of case presentation to the medical system and aspects related to the sensitivity and specificity of testing methods. Under-diagnosis multipliers were in the range 1.1 (*Mycobacterium bovis*) to 142 (*Vibrio parahaemolyticus*).

Further multipliers were applied to scale the total community cases to:

- Domestically acquired cases
- Foodborne cases

All multipliers are represented by either empirical or parametric distributions, to recognise the degree of uncertainty implicit in them.

Rates of hospitalisation and case-fatality rates were used to estimate the number of hospitalisations and deaths. These estimates were also scaled using multipliers to give domestically acquired foodborne hospitalisations and deaths.

2.2 Application of IID2 Rates and Multipliers to New Zealand

The second Infectious Intestinal Disease (IID2) study in Britain examined a community cohort and a general practitioner (GP) cohort to determine rates of disease and ratios between notified cases and total community cases, and notified cases and GP presenting cases, for disease due to ten enteric pathogens (*Clostridium perfringens*, *Campylobacter*, *Salmonella*, *E.coli* O157, *Cryptosporidium*, *Giardia*, adenovirus, astrovirus, norovirus and rotavirus) (Tam *et al.*, 2011). All of these organisms, except adenovirus, were also included in the US study. IID2 rates and ratios were used to estimate New Zealand incidence for the nine organism also included in the US study.

[1] http://www.cdc.gov/eid/content/17/1/7.htm

The IID2 study makes estimates of the total incidence of certain diseases, but does not present information on rates of hospitalisation or death. Rather than constructing a 'mixed model', IID2 information has only been used to estimate total incidence of disease due to four pathogens specified above for New Zealand.

2.2.1 *Campylobacter*, *Salmonella*, *E.coli* O157, *Cryptosporidium* and *Giardia*

The ratio of notified to total community cases determined in IID2 is exactly equivalent to the product of the under-reporting and the under-diagnosis expansion factors used in the US study for these five organisms. These factors were compared to the US factors and were applied to New Zealand notification data to determine estimates of total illness incidence for these organisms. The IID2 study assumed that rates of disease came from lognormal distributions and derived ratios by dividing the two lognormal distributions under simulation. The quotient of two lognormal distributions is also a lognormal distribution and the reported median, 2.5th and 97.5th percentile ratios were used to reconstruct a lognormal distribution for the ratio, to be used in the current study.

2.2.2 *C.perfringens*, astrovirus, norovirus and rotavirus

C.perfringens, astrovirus, norovirus and rotavirus infections are not individually notifiable diseases in New Zealand or the US and no active or passive surveillance systems are in place to capture information on cases, although some cases in New Zealand are notified under the 'Acute gastroenteritis' category. For these diseases the rates of disease in the community identified in IID2 were applied to the New Zealand population and compared to results of the top down approach of the US study. IID2 assumes that the rates come from a lognormal distribution. The reported mean, 2.5th and 97.5th percentile rates were used to reconstruct a lognormal distribution for use in the current study.

2.3 New Zealand Data Sources

The principal New Zealand data sources used for the estimates in this project were:

- Notifiable disease surveillance data from the EpiSurv database[2], which records the number of cases of a wide range of communicable diseases, some of which may be foodborne. Since 2008, most notified cases have resulted from cases reported by clinical and community laboratories directly. Data on cases were taken from the period 2000-2009, while data on outbreaks were taken from the period 2002-2009;
- Hospitalisation data (2002-2009) from the Data and Statistics Section of the Ministry of Health[3]; and,
- The Acute Gastrointestinal Illness (AGI) study, which included a retrospective survey conducted between February 2006 to January 2007 to establish the incidence of AGI in the New Zealand population (Adlam *et al.*, 2011).

[2] http://www.esr.cri.nz/competencies/Health/Pages/cdSurveillanceactivities.aspx

[3] http://www.moh.govt.nz/moh.nsf/indexmh/dataandstatistics

2.4 Time period

Where the surveillance data used to estimate the incidence of illness were taken from a number of years (either 2000-2009 or 2002-2009), the notification or outbreak case numbers for each year were adjusted to the 2009 population. These case numbers were used as an empirical distribution which was sampled randomly. Consequently, the mean of the annual incidence estimates represents the mean number of notification during the period 2000-2009 or the mean number of outbreak cases in the period 2002-2009. This assumes that the incidence of illness has been stable over the time period from which data were taken. This is apparently not the case for at least two illnesses (campylobacteriosis and STEC infection), as indicated by the number of notified cases, and the effect of using only 2009 data was examined for comparison.

3 RESULTS AND DISCUSSION

Of the total number of cases of illness due to a particular microbial organism there is potential for a percentage of the cases to have been acquired while the case was in another country (travel related). There is also potential for microbial organisms to be acquired from a variety of sources (e.g. water, animal contact, infected people) other than food. In order to determine the proportion of the total illness that is domestically acquired and due to food, it is necessary to have an estimate of the percentage of cases that are travel related and the percentage of cases that were acquired from food, rather than another source.

3.1 Use of US Values for the Proportion Travel-related and the Proportion Foodborne

Where New Zealand estimates are available for the proportion of illness due to a particular pathogen that is travel-related and the proportion that is due to consumption of contaminated food these have been used. However, New Zealand estimates are not available for all organisms included in this study and for those other organisms the proportions used in the US study have been applied.

3.1.1 Proportion travel-related

Case report forms for communicable disease investigations in New Zealand often include questions to determine if the case has been overseas during the incubation period of the organism causing the illness. Although this information is not always captured, where the information is completed it allows an estimate of the proportion of travel-related cases to be made for that organism. Table 1 includes the mean estimates of the proportion of New Zealand cases of disease due to 24 major pathogens, that may be travel-related, that were used in the current study. Where it was not possible to derive these estimates from communicable disease investigation data, they were 'borrowed' from the US study (Scallan *et al.*, 2011b). Estimates of the proportion of cases that are travel-related from other countries are included in Table 1 for comparison.

Table 1: Comparison of the proportion travel-related (%) for various pathogens used in the current study to other international estimates

Pathogen	NZ[1]	US[2]	Netherlands[3]	England and Wales[4]
Astrovirus	0*	0		0
Bacillus cereus	<1*	<1	7	0
Brucella spp.	95	16		
Campylobacter spp.	7	20	12	22
Clostridium perfringens	<1*	<1	3	0
Cryptosporidium spp.	8	9	20	5
STEC O157	5	4	12	12
STEC non-O157	5	18	6	12
Giardia intestinalis	24	8	18	21
Hepatitis A virus	50	41	60	
Listeria monocytogenes	8	3	13	0
Mycobacterium bovis	70*	70		
Norovirus	<1*	<1	9	0
Rotavirus	0*	0	9	0
Salmonella, non-typhoidal	18	11	14	12
Salmonella Paratyphi	74			74
Salmonella Typhi	75	67		74
Sapovirus	0*	0		
Shigella spp.	57	15		25
Staphylococcus aureus	<1*	<1	4	0
Toxoplasma gondii	<1*	<1	5	
Vibrio cholerae, toxigenic	100	70		100
Vibrio parahaemolyticus	29	10		
Yersinia enterocolitica	7	7		7

[1] Unless otherwise indicated these values are the mean of annual estimates for 2000-2009 taken from the New Zealand Notifiable Disease Surveillance system (Episurv) as outlined in Appendix 1.
[2] (Scallan *et al.*, 2011b) [3] (Havelaar *et al.*, 2008) [4](Adak *et al.*, 2002)
* Estimates taken from Scallan *et al.*, 2011b

3.1.2 Proportion foodborne

New Zealand-specific information on the proportion of domestically-acquired cases of certain illnesses of microbial origin that were due to food was obtained from an expert consultation (modified Delphi) conducted in 2005 (Lake *et al.*, 2010a). Table 2 includes the mean estimates of the proportion of New Zealand cases of 24 major pathogens that may be acquired from food that were used in the current study. Where it was not possible to derive these estimates from the expert consultation, they were 'borrowed' from the US study (Scallan *et al.*, 2011b). Estimates of the proportion of cases that are foodborne from other countries are included in Table 1 for comparison.

Table 2: Comparison of the mean proportion (%) foodborne for various pathogens used in the current study to other international estimates

Pathogen	NZ[1]	US[2]	Netherlands[3]	Australia[4]	England and Wales[5]
Astrovirus	<1*	<1		10	11
Bacillus cereus	100	100	97	100	100
Brucella spp.	50*	50			
Campylobacter spp.	56	80	48	75	80
Clostridium perfringens	100*	100	94	100	94
Cryptosporidium spp.	8*	8	15	10	6
STEC O157	39	68	45	65	63
STEC non-O157	39	82	45		63
Giardia intestinalis	7*	7	16	5	10
Hepatitis A virus	7*	7	28	10	
Listeria monocytogenes	85	99	79	98	99
Mycobacterium bovis	28	95			
Norovirus	39	26	19	25	11
Rotavirus	<1*	<1	14	2	3
Salmonella, non-typhoidal	60	94	64	87	92
Salmonella Paratyphi	96#				80
Salmonella Typhi	96*	96			80
Sapovirus	<1*	<1			
Shigella spp.	31*	31		10	8
Staphylococcus aureus	100*	100	91	100	96
Toxoplasma gondii	31	50	59		
Vibrio cholerae, toxigenic	100*	100			90
Vibrio parahaemolyticus	89	86		71	
Yersinia enterocolitica	56	90		75	90

[1] Unless otherwise stated these estimates were derived from a New Zealand expert consultation (Lake *et al.*, 2010a)
[2] (Scallan *et al.*, 2011b) [3] (Havelaar *et al.*, 2008) [4] (Hall and Kirk, 2005) [5] (Adak *et al.*, 2002)
* Estimates taken from Scallan *et al.*, 2011b
# Assumed to be the same as for *Salmonella* Typhi

For a number of organisms the New Zealand expert estimates of the proportion of cases that will be due to foodborne transmission are lower than most overseas estimates. The main exception to this observation is for norovirus, where the New Zealand estimate of the proportion foodborne is higher than any overseas estimate.

3.2 Estimated Incidence of Illness due to Major Pathogens using US Study Approach

Information was available to determine the incidence of foodborne illness in New Zealand due to 24 major pathogens, compared to 31 pathogens in the US study. Estimates of the incidence of domestically acquired foodborne illness due to these pathogens, based on data from 2000-2009, is given in Table 3. Estimates of the number of associated hospitalisations and deaths are included in Table 4. For further information on all aspects of these tables see Appendix 1.

Table 3: Estimated annual number of cases of domestically acquired foodborne illness caused by 24 pathogens, New Zealand[1]

Pathogen	Laboratory confirmed cases, mean[2]	Multipliers, mean		Total community cases, mean (90% CI)	Proportion travel-related (%)	Domestically acquired cases, mean (90% CI)	Proportion foodborne (%)	Domestically acquired, foodborne cases, mean (90% CI)
		Under-reporting	Under-diagnosis					
Bacteria								
Bacillus cereus	15	25.5	29.3	11,281 (0-42,441)	<1	11,243 (0-42,297)	96	10,833 (0-40,652)
Brucella spp.	1	1.0	15.2	19 (0-62)	100	0 (0-0)	50	0 (0-0)
Campylobacter spp.	12,090	1.0	30.3	363,490 (184,508-551,434)	7	338,767 (171,964-514,211)	56	190,092 (93,748-297,938)
Clostridium perfringens	88	25.5	29.3	65,442 (9,839-191,877)	<1	64,999 (9,805-191,229)	100	64,989 (9,806-191,031)
STEC O157	103	1.0	26.1	2,718 (1,526-4,587)	5	2,578 (1,447-4,351)	39	1,018 (545-1,755)
STEC non-O157	5	1.0	106.8	560 (94-1,056)	5	531 (89-1,002)	39	210 (35-408)
Listeria monocytogenes	24	1.0	2.1	50 (37-63)	8	46 (34-58)	85	39 (29-50)
Mycobacterium bovis	8	1.0	1.1	8 (5-14)	70	3 (1-4)	28	1 (0-1)
Salmonella spp., non-typhoidal	1,600	1.0	29.3	46,618 (27,992-79,598)	19	37,923 (22,692-64,791)	60	22,570 (13,218-38,827)
Salmonella Paratyphi	25	1.0	13.3	337 (171-606)	73	90 (44-164)	96	86 (42-159)
Salmonella Typhi	32	1.0	13.3	428 (206-802)	75	107 (49-203)	96	102 (47-196)
Shigella spp.	133	1.0	33.3	4,405 (2,804-6,773)	57	1,912 (1,182-2,986)	31	596 (354-950)
Staphylococcus aureus	7	25.5	29.3	5,205 (0-18,464)	<1	5,183 (0-18,404)	100	5,182 (0-18,296)
Vibrio cholerae, toxigenic	1	1.0	33.1	29 (0-110)	100	0 (0-0)	100	0 (0-0)
Vibrio parahaemolyticus	2	25.5	29.3	1,685 (0-9,645)	29	1,182 (0-6,738)	89	1,049 (0-5,979)

Pathogen	Laboratory confirmed cases, mean[2]	Multipliers, mean		Total community cases, mean (90% CI)	Proportion travel-related (%)	Domestically acquired cases, mean (90% CI)	Proportion foodborne (%)	Domestically acquired, foodborne cases, mean (90% CI)
		Under-reporting	Under-diagnosis					
Yersinia enterocolitica	466	1.0	122.8	56,660 (40,561-77,223)	7	52,888 (37,842-72,123)	56	29,715 (20,276-41,841)
Total bacteria				**558,935 (349,786-787,163)**		**517,452 (320,730-734,380)**		**326,482 (195,584-492,022)**
Parasites								
Cryptosporidium spp.	905	1.0	98.6	88,198 (56,940-134,394)	8	81,423 (52,520-124,098)	8	6,786 (4,112-10,696)
Giardia intestinalis	1,593	1.0	46.3	74,815 (52,002-101,659)	24	56,823 (39,290-77,573)	7	4,072 (2,638-5,862)
Toxoplasma gondii		1.0	NA	2,509 (1,992-3,086)	<1	2,501 (1,985-3,075)	31	783 (559-1,041)
Total parasites				**165,522 (125,300-217,363)**		**140,747 (105,697-187,068)**		**11,641 (8,458-15,861)**
Viruses								
Astrovirus		NA	NA	45,826 (34,828-56,825)	<1	45,819 (34,822-56,815)	<1	229 (82-395)
Hepatitis A virus	79	1.0	9.1	832 (399-1,486)	50	416 (196-754)	7	31 (12-64)
Norovirus		NA	NA	559,719 (366,304-781,213)	<1	557,851 (365,113-778,678)	39	218,701 (137,967-315,565)
Rotavirus		NA	NA	45,826 (34,828-56,825)	<1	45,819 (34,822-56,815)	<1	229 (82-395)
Sapovirus		NA	NA	45,826 (34,828-56,825)	<1	45,819 (34,822-56,815)	<1	229 (82-395)
Total viruses				**698,029 (503,670-920,309)**		**695,724 (502,022-917,255)**		**219,419 (138,698-316,261)**
Total pathogens				**1,422,486 (1,126,295-1,740,262)**		**1,353,923 (1,066,792-1,663,366)**		**557,542 (398,779-746,074)**

90% CI = 90th percentile credible interval

[1] See Appendix 1 for further details of calculations

[2] Case numbers are based on the years 2000-2009 for notifications or 2002-2009 for outbreaks. Mid-year population estimates were used to adjust all case numbers to their 2009 equivalents

Table 4: Estimated annual number of domestically acquired foodborne hospitalisations and deaths caused by 24 pathogens, New Zealand

Pathogen	Mean Hospitalisation rate[1] (%)	Total hospitalised cases, mean (90%CI)	Domestically acquired, foodborne hospitalised cases, mean (90%CI)	Mean Fatality rate[1] (%)	Total fatalities, mean (90%CI)	Domestically acquired, foodborne fatalities, mean (90%CI)
Bacteria						
Bacillus cereus	0.4	3 (0-19)	3 (0-18)	0	0 (0-0)	0 (0-0)
Brucella spp.	73.2	2 (0-7)	0 (0-0)	0.9	0 (0-0)	0 (0-0)
Campylobacter spp.	7.2	1,735 (832-2,763)	908 (423-1,488)	<0.1	2 (0-9)	1 (0-4)
Clostridium perfringens	0.6	26 (2-92)	26 (2-92)	<0.1	2 (0-10)	2 (0-10)
STEC O157	16.7	34 (8-118)	13 (3-45)	<0.1	0 (0-1)	0 (0-1)
STEC non-O157	16.7	1 (0-4)	0 (0-1)	<0.1	0 (0-0)	0 (0-0)
Listeria monocytogenes	100	52 (32-76)	41 (25-60)	14.9	7 (2-13)	6 (2-11)
Mycobacterium bovis	58.9	5 (3-8)	0 (0-1)	1.4	0 (0-0)	0 (0-0)
Salmonella spp., non-typhoidal	12.3	394 (212-698)	191 (101-340)	0.1	3 (0-11)	1 (0-5)
Salmonella Paratyphi	24.9	13 (3-31)	3 (1-8)	0	0 (0-0)	0 (0-0)
Salmonella Typhi	78.9	51 (27-86)	12 (6-21)	0	0 (0-0)	0 (0-0)
Shigella spp.	23.6	63 (25-135)	8 (3-19)	0	0 (0-0)	0 (0-0)
Staphylococcus aureus	6.4	23 (0-90)	23 (0-90)	<0.1	0 (0-1)	0 (0-1)
Vibrio cholerae, toxigenic	45.8	1 (0-4)	0 (0-0)	0	0 (0-0)	0 (0-0)
Vibrio parahaemolyticus	13.7	16 (0-99)	10 (0-62)	0.9	1 (0-6)	1 (0-4)
Yersinia enterocolitica	8.4	78 (35-126)	41 (18-68)	<0.1	0 (0-2)	0 (0-1)
Total bacteria		**2,497 (1,540-3,569)**	**1,279 (765-1,882)**		**15 (5-31)**	**11 (3-22)**
Parasites						
Cryptosporidium spp.	4.3	77 (41-125)	6 (3-10)	0	0 (0-0)	0 (0-0)
Giardia intestinalis	3.4	107 (56-197)	6 (3-11)	0	0 (0-0)	0 (0-0)
Toxoplasma gondii	2.6	129 (79-189)	40 (23-62)	0.2	9 (6-13)	3 (2-4)
Total parasites		**313 (226-434)**	**52 (34-74)**		**9 (6-13)**	**3 (2-4)**
Viruses						
Astrovirus	0.4	255 (203-314)	1 (0-2)	<0.1	0 (0-0)	0 (0-0)
Hepatitis A virus	56.7	89 (34-183)	3 (1-7)	0	0 (0-0)	0 (0-0)
Norovirus	1.3	7,516 (2,337-15,530)	2,938 (890-6,118)	<0.1	8 (5-12)	3 (2-5)
Rotavirus	1.7	1,049 (844-1,261)	5 (2-9)	<0.1	0 (0-1)	0 (0-0)
Sapovirus	0.4	255 (203-314)	1 (0-2)	<0.1	0 (0-0)	0 (0-0)
Total viruses		**9,164 (3,982-17,167)**	**2,948 (911-6,128)**		**9 (6-12)**	**3 (2-5)**
Total pathogens		**11,974 (6,668-20,051)**	**4,279 (2,142-7,501)**		**33 (22-50)**	**17 (9-28)**

90% CI = 90th percentile credible interval

[1] Hospitalisation and fatality rates refer to the proportion (%) of incident cases who are hospitalised and or die as a result of a particular disease of microbial origins

3.2.1 Incidence of foodborne illness

Application of the US model to the New Zealand situation results in an estimate of approximately 1.4 million cases of illness caused by 24 pathogens per annum. This estimate is based on on the mean number of notifications for the years 2000-2009 or the mean number of outbreak cases for the years 2002-2009, scaled to the New Zealand population in 2009. Of these, just over half a million are estimated to be due to domestic foodborne transmission. When expressed as a rate, this equates to 129 cases per 1,000 population per annum. The equivalent rate for the US population is 31 cases per 1,000 population per annum. The US incidence estimates were mainly based on surveillance data from the years 2000-2008, scaled to the US population in 2006. This difference in population rate is not surprising, as New Zealand notified rates of many of the relevant diseases, particularly campylobacteriosis, are higher than in the US (Centers for Disease Control and Prevention, 2010; Population and Environmental Health Group (ESR), 2010). However, it is uncertain whether these differences in reported disease rates are due to actual differences or due to differences in surveillance systems. For example, the US FoodNet system, that provided much of the data for their incidence of foodborne illness estimates, only collects information in ten US states, while New Zealand's EpiSurv system has full national coverage.

Of the approximately half million cases of foodborne illness, 59% were due to bacteria, 39% due to viruses and only 2% due to parasites. This is quite different to the US situation, where 59% of foodborne illnesses were estimated to be due to viruses, with 39% due to bacteria (Scallan *et al.*, 2011b). This is in spite of the fact that New Zealand estimates of the proportion of norovirus cases that are due to foodborne transmission is higher than the US estimate (39% compared to 26%, see Table 2).

The pathogens contributing most to the estimated incidence of foodborne illness in New Zealand were norovirus (39%), *Campylobacter* (34%), *Clostridium perfringens* (12%), *Yersinia enterocolitica* (5%) and non-typhoidal *Salmonella* (4%).

3.2.2 Hospitalisations

The current exercise estimated that approximately 4,000 people will be hospitalised in New Zealand each year due to foodborne illness. When expressed as a population rate (99 per 100,000) this is also higher than the equivalent US figure (19 per 100,000) (Scallan *et al.*, 2011b).

Of the hospitalisations due to foodborne illness in New Zealand, 69% were due to viruses, 30% due to bacteria and 1% due to parasites. Again, this is different to the pattern seen in the US study, in which 64% of hospitalisations were due to bacteria and 27% due to viruses.

These differences may be due to a single modelling decision. For both studies, the hospitalisation rate for norovirus was assumed to be the same as the rate of hospitalisation for general acute gastrointestinal illness. For the current study, this figure was taken from the national AGI study (Adlam *et al.*, 2011) and yielded an estimate of 1.3%. The US study based their estimate of the hospitalisation rate for acute gastrointestinal illness on hospital discharge data and yielded an estimate of 0.03% (Scallan *et al.*, 2011b). Adoption of the much lower US hospitalisation rate would result in a decrease in the estimated number of

norovirus-related hospitalisation from approximately 7,500 to about 170 and a decrease in the domestically acquired foodborne norovirus hospitalisations from approximately 2,900 to about 66.

The pathogens contributing most to hospitalisations due to foodborne illness were norovirus (69%), *Campylobacter* (21%) and non-typhoidal *Salmonella* (4%).

3.2.3 Deaths

The current study estimates that 17 fatalities would occur in a year due to 24 pathogens transmitted by food. When expressed as a population rate (3.9 per million) this figure is very similar to the equivalent estimate for the US population (4.5 per million) (Scallan *et al.*, 2011b).

For New Zealand, most fatalities (65%) were due to bacteria, with the remainder equally divided between viruses and parasites.

The pathogens contributing most to fatalities due to foodborne illness were *Listeria monocytogenes* (35%), norovirus (18%) and *Toxoplasma gondii* (18%).

3.3 Estimated Incidence of Illness due to Unspecified Pathogens

Estimates of the number of episodes of AGI due to unspecified foodborne pathogens have been derived from data on the incidence of AGI in the community, after deducting the incidence of AGI caused by specified pathogens. Results are shown in Table 5.

Table 5: Estimated annual number of episodes of domestically acquired foodborne illness, hospitalisations and deaths caused by 24 major pathogens and unspecified agents transmitted through food, New Zealand

Cause	Illnesses		Hospitalisations		Deaths	
	Mean (90%CI)	%	Mean (90%CI)	%	Mean (90%CI)	%
Major known pathogens	557,542 (398,779-746,074)	29	4,279 (2,142-7,501)	19	17 (9-28)	41
Unspecified pathogens	1,368,421 (965,838-1,874,466)	71	18,397 (5,181-37,803)	81	24 (11-42)	59
Total	1,925,963 (1,518,836-2,423,013)	100	22,676 (7,531-44,723)	100	41 (27-59)	100

90%CI = 90th percentile credible interval

The results presented in Table 5 suggest that unspecified pathogens transmitted by food cause more than twice as many cases of illness in New Zealand as the 24 known pathogens for which incidence estimates were calculated. When expressed as a population rate (317 per 1,000) this figure is much higher than the equivalent US estimate (128 per 1,000) (Scallan *et al.*, 2011a). However, this is not unexpected, as these calculations draw heavily on national estimates of general acute gastrointestinal illness. The estimated rate of acute gastrointestinal illness in New Zealand (Adlam *et al.*, 2011) is estimated to be about twice that used in the US study (Scallan *et al.*, 2011a).

Unspecified pathogens cause more than four times as many hospitalisations as 24 major pathogens, but only about 40% more fatalities.

3.4 Estimated Incidence of Illness due to Selected Pathogens using IID2 Multipliers

Table 6 shows the different multipliers (*Campylobacter* spp., STEC O157, *Salmonella* spp., *Cryptosporidium* spp. and *Giardia intestinalis*) and population rates (astrovirus, norovirus and rotavirus) derived from the US study and the IID2 study. For *Clostridium perfringens* a US multiplier was used, while an IID2 population rate was used. The IID2 study also reports a multiplier for the ratio of community cases to notified cases (median = 2,519). However, in both the UK and New Zealand cases of *C. perfringens* infection are only notified when foodborne transmission is suspected and very few cases are notified in New Zealand) (1-6 cases per annum during the period 2006-2010). Table 6 also shows the corresponding estimates of the incidence of illness from these organisms in New Zealand.

Table 6: Comparison of illness rates and case multipliers between the US study and the British IID2 study and associated estimates of illness incidence

Pathogen	Factor type	Factor value		Estimate of incidence in New Zealand, mean (90% CI)	
		US, mean	IID2, mean*	US	IID2
Clostridium perfringens	US = under-reporting/under-diagnosis IID2 = population rate	747.2	0.0015	65,442 (9,839-191,877)	6,774 (2,514-14,161)
Campylobacter spp.	Under-reporting/under-diagnosis	30.3	9.5	363,490 (184,508-551,434)	115,260 (55,426-186,270)
STEC O157	Under-reporting/under-diagnosis	26.1	18.4	2,718 (1,526-4,587)	1,895 (77-7,114)
Salmonella spp., non-typhoidal	Under-reporting/under-diagnosis	29.3	6.0	46,618 (27,992-79,598)	9,543 (2,144-24,803)
Cryptosporidium spp.	Under-reporting/under-diagnosis	98.6	10.4	88,198 (56,940-134,394)	9,417 (2,234-23,716)
Giardia intestinalis	Under-reporting/under-diagnosis	46.3	17.2	74,815 (52,002-101,659)	27,361 (7,553-64,520)
Astrovirus	Population rate	0.011	0.0053	45,826 (34,828-56,825)	23,792 (14,153-36,934)
Norovirus	Population rate	0.122	0.047	559,719 (366,304-781,213)	203,735 (173,830-236,710)
Rotavirus	Population rate	0.011	0.0127	45,826 (34,828-56,825)	55,570 (39,865-74,744)

90%CI = 90th percentile credible interval

* The IID2 study reports median values for the ratio of community to national surveillance cases. For consistency with the US study, mean values of the reconstructed lognormal distributions for the ratios are reported here.

Application of the two approaches (US and IID2) to the New Zealand situation generates quite similar estimates for the number of cases of illness due to rotavirus. However, for the other pathogens compared, estimates of illness vary by factors ranging from 1.4 (STEC O157) to 10.1 (*C.perfringens*). While it is not possible to say which estimate is more plausible, it should be noted that the IID2 study measured actual rates of illness in the community, while the US study draws on observed data, but incorporates it into a theoretical model.

It should be noted that the IID2 study only provides multipliers for estimating the incidence of illness and does not provide corresponding multipliers for hospitalisations and deaths.

3.5 Estimated Incidence of Illness due to Major Pathogens Based on Most Recent Complete Year of Data (2009)

The approach outlined in Appendix 1 and used to generate the figures in Tables 3 and 4 assumes that the rate of a particular disease is largely stable over the period from which data are used (2000-2009 for notifications). For illness due to two bacteria (*Campylobacter* and STEC) in New Zealand this is unlikely to be true. Notified cases of campylobacteriosis decreased significantly in 2007 and 2008, while reported STEC cases have generally increased over the last decade. Notifications for some other diseases have also changed across the decade (2000-2009). For example, the number of notified salmonellosis cases decreased by about a third through the decade considered. Table 7 and 8 represent a repeat of the analysis summarised in Table 3 and 4, but using only New Zealand data from the 2009 year, wherever possible. New Zealand data for 2009 used include notification and outbreak case numbers, proportions travel related, proportions hospitalised and proportions of deaths.

While estimated mean domestically acquired foodborne case numbers for illness due to parasites and viruses are largely the same as estimates based on 2000-2009 data, the estimate of the number of cases of bacterial illness decreases by 30% when estimates are based only on 2009 data. Most of this reduction in estimated bacterial illness is due to decreases in estimates of *Campylobacter*-associated cases. The estimated mean domestically acquired foodborne cases of illness due to all 24 pathogens reduced by 18% when data from the 2009 year was used compared to using data from 2000-2009.

Basing estimates on 2009 notifications reduces the contribution of *Campylobacter* to total domestically acquired foodborne cases from 34% to 25% and for domestically acquired foodborne hospitalisations from 21% to 15%.

The US study also recognised evidence for trends in notifications for hepatitis A virus, *Staphylococcus aureus* and *Vibrio* spp. This was dealt with in a similar manner to that outlined above; use of the notifications from the index year only (2006 for the US study). Uncertainty around this figure was generated by fitting a simple linear regression to the notifications for 2000-2007 and bootstrapping on the regression residuals, scaled for uncertainty of the linear fit, plus the constant predicted mean counts[4].

[4] http://www.cdc.gov/EID/content/17/1/7-Techapp2.pdf

Table 7: Estimated annual number of cases of domestically acquired foodborne illness caused by 24 pathogens, New Zealand, 2009[1]

Pathogen	Laboratory confirmed cases[2]	Multipliers, mean		Total community cases, mean (90% CI)	Proportion travel-related (%)	Domestically acquired cases, mean (90% CI)	Proportion foodborne (%)	Domestically acquired, foodborne cases, mean (90% CI)
		Under-reporting	Under-diagnosis					
Bacteria								
Bacillus cereus	0	25.5	29.3	0 (0-0)	<1	0 (0-0)	96	0 (0-0)
Brucella spp.	0	1.0	15.2	0 (0-0)	100	0 (0-0)	50	0 (0-0)
Campylobacter spp.	7,176	1.0	30.3	215,703 (164,807-278,433)	7	200,528 (153,213-258,845)	56	112,529 (80,144-151,834)
Clostridium perfringens	88	25.5	29.3	65,333 (21,356-137,401)	<1	65,116 (21,287-136,963)	100	65,105 (21,284-136,928)
STEC O157	135	1.0	26.1	3,561 (2,223-5,614)	5	3,378 (2,108-5,325)	39	1,333 (789-2,157)
STEC non-O157	8	1.0	106.8	841 (594-1,157)	5	798 (564-1,097)	39	315 (209-452)
Listeria monocytogenes	28	1.0	2.1	58 (49-68)	9	52 (44-62)	85	44 (37-53)
Mycobacterium bovis	6	1.0	1.1	7 (6-7)	70	2 (2-2)	28	1 (0-1)
Salmonella spp., non-typhoidal	1,129	1.0	29.3	32,882 (24,429-43,214)	16	27,476 (20,412-36,108)	60	16,353 (11,792-21,935)
Salmonella Paratyphi	25	1.0	13.3	334 (191-570)	72	93 (54-160)	96	90 (51-154)
Salmonella Typhi	35	1.0	13.3	467 (268-798)	82	82 (47-141)	96	79 (45-135)
Shigella spp.	119	1.0	33.3	3,948 (2,977-5,150)	65	1,384 (1,044-1,806)	31	431 (307-586)
Staphylococcus aureus	0	25.5	29.3	0 (0-0)	<1	0 (0-0)	100	0 (0-0)
Vibrio cholerae, toxigenic	0	1.0	33.1	0 (0-0)	100	0 (0-0)	100	0 (0-0)
Vibrio parahaemolyticus	7	25.5	29.3	5,200 (1,692-10,980)	29	3,632 (1,163-7,713)	89	3,221 (1,031-6,849)

Pathogen	Laboratory confirmed cases[2]	Multipliers, mean		Total community cases, mean (90% CI)	Proportion travel-related (%)	Domestically acquired cases, mean (90% CI)	Proportion foodborne (%)	Domestically acquired, foodborne cases, mean (90% CI)
		Under-reporting	Under-diagnosis					
Yersinia enterocolitica	431	1.0	122.8	52,437 (38,961-69,836)	7	48,786 (36,249-64,974)	56	27,409 (19,319-37,722)
Total bacteria				**380,771 (303,034-478,279)**		**351,327 (277,275-444,766)**		**226,910 (165,857-309,435)**
Parasites								
Cryptosporidium spp.	854	1.0	98.6	83,174 (62,030-110,560)	8	75,989 (56,671-101,008)	8	6,332 (4,332-8,948)
Giardia intestinalis	1,640	1.0	46.3	77,035 (59,876-99,402)	24	63,161 (49,092-81,500)	7	4,527 (3,188-6,233)
Toxoplasma gondii		1.0	NA	2,509 (1,992-3,086)	<1	2,501 (1,985-3,075)	31	783 (559-1,041)
Total parasites				**162,718 (133,941-196,881)**		**141,651 (116,451-171,632)**		**11,642 (9,118-14,694)**
Viruses								
Astrovirus		NA	NA	45,826 (34,828-56,825)	<1	45,819 (34,822-56,815)	<1	229 (82-395)
Hepatitis A virus	44	1.0	9.1	465 (354-608)	75	116 (88-152)	7	9 (5-14)
Norovirus		NA	NA	559,719 (366,304-781,213)	<1	557,851 (365,113-778,678)	39	218,701 (137,967-315,565)
Rotavirus		NA	NA	45,826 (34,828-56,825)	<1	45,819 (34,822-56,815)	<1	229 (82-395)
Sapovirus		NA	NA	45,826 (34,828-56,825)	<1	45,819 (34,822-56,815)	<1	229 (82-395)
Total viruses				**697.662 (503,000-920,289)**		**695,424 (501,470-917,323)**		**219,397 (138,407-316,069)**
Total pathogens				**1,241,151 (1,023,987-1,482,464)**		**1,188,402 (974,196-1,427,017)**		**457,949 (351,209-581,502)**

90% CI = 90th percentile credible interval

[1] See Appendix 1 for further details of calculations

[2] Case numbers are based on 2009 notifications and outbreaks

Table 8: Estimated annual number of domestically acquired foodborne hospitalisations and deaths caused by 24 pathogens, New Zealand, 2009

Pathogen	Mean Hospitalisation rate[1] (%)	Total hospitalised cases, mean (90%CI)	Domestically acquired, foodborne hospitalised cases, mean (90%CI)	Mean Fatality rate[1] (%)	Total fatalities, mean (90%CI)	Domestically acquired, foodborne fatalities, mean (90%CI)
Bacteria						
Bacillus cereus	NA	0 (0-0)	0 (0-0)	NA	0 (0-0)	0 (0-0)
Brucella spp.	NA	0 (0-0)	0 (0-0)	NA	0 (0-0)	0 (0-0)
Campylobacter spp.	8.0	1,148 (791-1,505)	599 (393-824)	0.0	0 (0-0)	0 (0-0)
Clostridium perfringens	0.6	26 (3-83)	26 (3-83)	0.0	0 (0-0)	0 (0-0)
STEC O157	4.9	13 (9-17)	5 (3-7)	0.7	2 (1-2)	1 (0-1)
STEC non-O157	4.9	0 (0-0)	0 (0-0)	0.7	0 (0-0)	0 (0-0)
Listeria monocytogenes	100	56 (39-73)	43 (30-57)	14.3	8 (6-10)	6 (4-8)
Mycobacterium bovis	62.8	4 (4-4)	0 (0-0)	1.0	0 (0-0)	0 (0-0)
Salmonella spp., non-typhoidal	14.0	316 (218-414)	157 (106-211)	0.1	2 (1-3)	1 (1-1)
Salmonella Paratyphi	12.0	6 (4-8)	2 (1-2)	0.0	0 (0-0)	0 (0-0)
Salmonella Typhi	74.3	52 (36-68)	9 (6-12)	0.0	0 (0-0)	0 (0-0)
Shigella spp.	16.0	38 (26-50)	4 (3-6)	0.0	0 (0-0)	0 (0-0)
Staphylococcus aureus	NA	0 (0-0)	0 (0-0)	NA	0 (0-1)	0 (0-0)
Vibrio cholerae, toxigenic	NA	0 (0-0)	0 (0-0)	NA	0 (0-0)	0 (0-0)
Vibrio parahaemolyticus	0.0	0 (0-0)	0 (0-0)	0.0	0 (0-0)	0 (0-0)
Yersinia enterocolitica	10.7	92 (64-121)	48 (32-66)	0.0	0 (0-0)	0 (0-0)
Total bacteria		**1,751 (1,377-2,128)**	**893 (675-1,131)**		**12 (6-25)**	**8 (3-18)**
Parasites						
Cryptosporidium spp.	2.7	46 (32-60)	4 (2-5)	0.0	0 (0-0)	0 (0-0)
Giardia intestinalis	2.7	89 (61-116)	5 (3-7)	0.0	0 (0-0)	0 (0-0)
Toxoplasma gondii	2.6	129 (79-189)	40 (23-62)	0.2	9 (6-13)	3 (2-4)
Total parasites		**264 (204-331)**	**49 (32-71)**		**9 (6-13)**	**3 (2-4)**
Viruses						
Astrovirus	0.4	255 (203-314)	1 (0-2)	<0.1	0 (0-0)	0 (0-0)
Hepatitis A virus	54.5	48 (33-63)	1 (0-1)	0.0	0 (0-0)	0 (0-0)
Norovirus	1.3	7,516 (2,337-15,530)	2,938 (890-6,118)	<0.1	8 (5-12)	3 (2-5)
Rotavirus	1.7	1,049 (844-1,261)	5 (2-9)	<0.1	0 (0-1)	0 (0-0)
Sapovirus	0.4	255 (203-314)	1 (0-2)	<0.1	0 (0-0)	0 (0-0)
Total viruses		**9,123 (3,928-17,067)**	**2,946 (904-6,142)**		**9 (6-12)**	**3 (2-5)**
Total pathogens		**11,138 (5,929-19,102)**	**3,888 (1,830-7,093)**		**30 (21-44)**	**14 (8-24)**

90% CI = 90th percentile credible interval NA = not applicable, due to no incident cases

[1] Hospitalisation and fatality rates refer to the proportion (%) of incident cases who are hospitalised and or die as a result of a particular disease of microbial origins

3.6 Estimated Incidence of Illness due to Unspecified Pathogens Based on Most Recent Complete Year of Data (2009)

Due to the methodology used to estimate the number of cases of domestically acquired foodborne illness due to unspecified pathogens, a reduction in the number of cases due to known major pathogens will increase the estimated number of cases due to unspecified pathogens. Table 9 repeats Table 5, but using data only from the 2009 year, wherever possible.

Table 9: Estimated annual number of episodes of domestically acquired foodborne illness, hospitalisations and deaths caused by 24 major pathogens and unspecified agents transmitted through food, New Zealand, 2009

Cause	Illnesses		Hospitalisations		Deaths	
	Mean (90%CI)	%	Mean (90%CI)	%	Mean (90%CI)	%
Major known pathogens	457,949 (351,209-581,502)	24	3,888 (1,830-7,093)	17	14 (8-24)	35
Unspecified pathogens	1,441,664 (1,025,000-1,961,784)	76	18,680 (5,438-38,260)	83	26 (13-43)	65
Total	1,899,613 (1,480,396-2,415,878)	100	22,568 (7,438-44,716)	100	40 (27-58)	100

90%CI = 90th percentile credible interval

As expected, the contribution of 24 major pathogens to the total burden of illness decreased from 29 to 24%, while their contribution to hospitalisations decrease from 19 to 17% and their contribution to fatalities decreased from 41 to 35%. The estimated total burden of disease (major pathogens plus unspecified pathogens) decreases by only 1%. Note that the rate of AGI used in this calculation dates from 2006, not 2009, and no attempt has been made to adjust this estimate.

3.7 Estimated Incidence of Illness due to Selected Pathogens using IID2 Multipliers Based on Most Recent Complete Year of Data (2009)

Table 10 shows the different multipliers (*Campylobacter* spp., STEC O157, *Salmonella* spp., *Cryptosporidium* spp. and *Giardia intestinalis*) derived from the US study and the IID2 study and the corresponding estimates of the incidence of illness from these organisms in New Zealand, based only on 2009 notification data. This represents an update of Table 6. As estimates for *C.perfringens*, astrovirus, norovirus and rotavirus are based on population rates, rather than on notifications, consideration of only the 2009 year will have no impact on the estimated incidence of illness due to these organisms and these data are not repeated in Table 10.

Table 10: Comparison of illness rates and case multipliers between the US study and the British IID2 study and associated estimates of illness incidence for New Zealand, 2009

Pathogen	Factor type	Factor value		Estimate of incidence in New Zealand, mean (90% CI)	
		US, mean	IID2, mean	US	IID2
Campylobacter spp.	Under-reporting/ under-diagnosis	30.3	9.5	215,703 (164,807-278,433)	68,412 (46,202-96,323)
STEC O157	Under-reporting/ under-diagnosis	26.1	18.4	3,561 (2,223-5,614)	2,485 (105-9,212)
Salmonella spp., non-typhoidal	Under-reporting/ under-diagnosis	29.3	6.0	32,882 (24,429-43,214)	6,734 (1,690-16,533)
Cryptosporidium spp.	Under-reporting/ under-diagnosis	98.6	10.4	83,174 (62,030-110,560)	8,882 (2,235-21,787)
Giardia intestinalis	Under-reporting/ under-diagnosis	46.3	17.2	77,035 (59,876-99,402)	28,164 (8,024-65,700)

90%CI = 90th percentile credible interval

* The IID2 study reports median values for the ratio of community to national surveillance cases. For consistency with the US study, mean values of the reconstructed lognormal distributions for the ratios are reported here.

4 GENERAL COMMENTS

The US model and the IID2 study represent two very different approaches to solving the same problem, that of extrapolating from disease notifications to the total burden of illness in the population. The IID2 represents a largely empirical approach, while the US study represents a largely theoretical approach. Both of these approaches will include aspects that are country-specific and their direct application to the New Zealand situation needs to be viewed with caution. For instance, it is unknown whether an individual with gastrointestinal illness in New Zealand is more or less likely to present to the medical system (and potentially become a notification) than an equivalent individual in the British population. Similarly, it is unknown whether factors embodied in the US under-diagnosis multipliers (the proportion of severe cases, the probability of severe and non-severe cases presenting to the medical system, sensitivity and specificity of the laboratory testing system) are applicable to New Zealand.

Similarly, the uncertainty around incidence estimates derived from the IID2 approach are empirically determined, while uncertainty quantified in the US study includes empirical uncertainty in observed data and some theoretical or speculative uncertainty elements. In particular, the US decision to double the number of hospitalisations and deaths to account for under-diagnosis does not appear to be based on any observed evidence. The extensive use of the Pert distribution to quantify uncertainty in the US model may also be questioned. The Pert distribution is primarily used for modelling expert opinion (Vose, 2008), but is used in the US model to represent uncertainty in data from a range of sources. This point is acknowledged by the authors of the US study and the decision to use this distribution so extensively appears to have been a matter of practicality.

In both the New Zealand and US surveillance systems, disease due to some pathogens is notifiable, while for other pathogens cases only come to the attention of the surveillance system when the pathogen is the causal agent in an outbreak. The US study considered data for pathogens causing both disease that is notifiable and causing outbreaks. Ratios of total laboratory confirmed to outbreak-related laboratory confirmed cases were determined for 11 pathogens. The median of these ratios was then used as the under-reporting multiplier for pathogens only reported due to their involvement in outbreaks (*B.cereus, C. perfringens,* enterotoxigenic *E. coli, S. aureus* and group A *Streptococcus* in the US study). While this approach is novel and useful, it assumes that the balance between outbreak and sporadic cases is similar for the organisms to which this approach was applied (*B. cereus, C. perfringens, S. aureus, V. parahaemolyticus* in the current study). The base data used to determine the under-reporting multiplier for outbreak cases in the US Study[5] clearly demonstrates that the ratio of reported outbreak cases to laboratory confirmed cases can vary hugely from pathogen to pathogen. For example, the ratio for *Yersinia enterocolitica* was 381, suggesting mainly sporadic cases, while for STEC O157 the ratio was 5.4, suggesting a higher proportion of cases associated with outbreaks.

Use of outbreak cases as the index for estimating the incidence of disease due to some organisms results in application of very large multipliers to very small numbers of cases, with little consideration of the aetiology of disease due to the particular organism. For these reasons, it is likely that this is one of the weaker aspects of the US model and of our

[5] http://www.cdc.gov/eid/content/17/1/7-Techapp4.pdf

application of that model. It is interesting to note that the largest disparity observed between disease incidence based on the US model and incidence based on IID2 was for *C. perfringens*, where the US model applied the outbreak case approach.

Values used for the proportion of incident cases that are due to foodborne transmission have the potential to impact significantly on final estimates of the number of domestically acquired foodborne cases, hospitalisations and deaths. New Zealand estimates of these proportions in this report are either based on local expert consultation or are 'borrowed' from the US study. While some of these proportions are quite similar to estimates made for other countries (those for the Netherlands are also based on an expert consultation), some differ quite markedly. It is not possible to say whether these differences reflect true differences in the epidemiology of the diseases in different countries or merely differences in opinion.

There appears to be potential to validate the proportion of campylobacteriosis in New Zealand that is foodborne by reference to recent attribution studies, based on molecular sub-typing (French and Marshall, 2010). However, this attribution study apportions human cases to sources, while the current study apportions cases to a foodborne transmission route. For example, cases who are infected with *Campylobacter* from direct contact with *Campylobacter*-colonised cattle and cases who are infected after consuming contaminated beef will be attributed to the same source (ruminants), but not the same transmission route. The study of French and Marshall (2010) clearly identifies a decrease over time in the proportion of human cases attributable to poultry and an increase in the proportion attributable to cattle and sheep. However, the proportion of the reduced incidence attributable to the foodborne transmission route may not have changed.

If the proportion of campylobacteriosis cases attributable to poultry is used as a surrogate for the proportion foodborne, then in 2008/2009 in the Manawatu, a median of 50.4% of human cases were attributed to poultry sources (chicken, duck, turkey and spent hen), while in 2009/2010 this proportion was 53.2% (French and Marshall, 2010). These values are similar to the overall proportion foodborne figure used in the current study (56%).

Application of the US model to the New Zealand situation would be improved by availability of New Zealand specific values for a number of the inputs to the model, specifically:

- More recent estimates of the proportion of illness due to specific pathogens that is due to foodborne transmission and extensive of the range of estimates to cover all organisms included in this report;
- Better information on total case numbers of disease due to pathogens that are not individually notifiable, but are potentially foodborne (*B. cereus, C. perfringens, S. aureus, V. parahaemolyticus*); and
- Pathogen-specific New Zealand information on under-reporting and under-diagnosis multipliers or the elements used to calculate these multipliers.

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APPENDIX 1 DETAILS OF THE APPLICATION OF THE SCALLAN (US) MODEL TO THE ESTIMATION OF THE INCIDENCE OF FOODBORNE ILLNESS IN NEW ZEALAND

The current project aims to replicate the approach taken by a recent US study to estimate the annual incidence of foodborne illness in New Zealand. In the following notes the terms 'US study' and 'Scallan model' refers to this exercise. The US study determined the incidence of foodborne illness due to 31 major pathogens and the illness due to 'unspecified pathogens'. Both of these aspects will be addressed for New Zealand.

The following notes identify areas where US data will be replaced by New Zealand data. However, unless specifically stated all aspects of the Scallan model will be applied.

Major Pathogens

The Scallan model uses two main approaches to estimate the incidence of disease associated with potentially foodborne microbial hazards:

- Scaling up from surveillance data. Scaling up involves application of two 'expansion factors'; an under-reporting factor and an underdiagnosis factor, as described in Section 2.1.
- Scaling down from total population or at-risk population subgroups, by application of rates of disease in those populations (norovirus, rotavirus, sapovirus, *Toxoplasma gondii*).

For different organisms, the scaling up was applied to either active surveillance, passive surveillance or outbreak surveillance data. The full list of organisms and the techniques applied are listed in Table 11.

Table 11: Major pathogens and modelling approaches used in the US study of the incidence of foodborne illness

Pathogens for which laboratory-confirmed illnesses were scaled up to estimate the total number of illnesses		
Active surveillance	***Passive surveillance***	***Outbreak surveillance***
• *Campylobacter* spp. • *Cryptosporidium* spp. • *Cyclospora cayetanensis* • *Escherichia coli*, shiga toxin-producing (STEC) O157 • *Escherichia coli*, shiga toxin-producing (STEC) non-O157 • *Listeria monocytogenes* • *Salmonella*, non-typhoidal • *Salmonella* Typhi • *Shigella* spp. • *Yersinia enterocolitica*	• *Brucella* spp. • *Clostridium botulinum* • *Giardia intestinalis* • Hepatitis A virus • *Mycobacterium bovis* • *Trichinella* spp. • *Vibrio cholerae*, toxigenic • *Vibrio parahaemolyticus* • *Vibrio vulnificus* • *Vibrio* spp., other	• *Bacillus cereus* • *Clostridium perfringens* • *Escherichia coli*, enterotoxigenic (ETEC) • *Escherichia coli*, Diarrheagenic other than STEC and ETEC • *Staphylococcus aureus* • *Streptococcus* spp., Group A
Pathogens for which populations were scaled down to estimate the total number of illnesses		
• Astrovirus • Sapovirus	• Norovirus • *Toxoplasma gondii*	• Rotavirus

Standard rates of hospitalisation and death, from surveillance data, are applied to total case estimates to estimate the number of hospitalised cases and the number of fatalities.

Factors are then applied to estimates of incident cases, hospitalisations and fatalities to estimate the domestically acquired proportion and the foodborne proportion.

Surveillance Data

The US study sampled actual surveillance data (case numbers, outbreak-related case numbers) from a range of years (2000-2007 for CDC outbreak, COVIS and NNDSS data, 2005-2008 for FoodNet data, 2004-2007 for Tuberculosis Surveillance data). Case numbers were adjusted using population estimate ratios to standardise on the 2006 population. The 2006 population estimate was also used for estimates derived from scaling down.

The current study standardised New Zealand estimates on latest complete year (2009) using mid-year population estimates from Statistics NZ[6]. Notifiable disease case numbers for 2000-2009 were adjusted to 2009 mid-year population and outbreak case numbers for 2002-2009 were adjusted to 2009 mid-year population. This approach results in mean incidence estimates that are based on the mean number of notification or outbreak cases in the year range, after adjustment to their 2009 equivalents.

Under-reporting factors

The US under-reporting factor is set at 1 for active surveillance systems, 1.1-1.3 for passive surveillance systems, and an uncertainty distribution with mean 25.5 for data from outbreak surveillance systems.

Since 2008 New Zealand laboratories have been required to report notifiable disease cases to Medical Officers of Health. However, for diseases where comparative data are available (salmonellosis and shigellosis) there has been good agreement between laboratory-confirmed cases and notifications over the last ten years. For the purposes of the current study, New Zealand notifiable disease data was treated as active surveillance data (under-reporting factor = 1). Outbreak-related case numbers were scaled using the US factor. An initial analysis of New Zealand outbreak cases versus notifications for 11 pathogens suggests that this is not unreasonable. A more detailed analysis of salmonellosis cases in New Zealand found a ratio of 11 between salmonellosis notifications and notified outbreak-related cases (King *et al.*, 2011). The equivalent ratio for the US was 15.9.

Underdiagnosis factors

The underdiagnosis factors used in the US study were calculated by estimating (pert distributions) the proportion of cases that will be severe/mild, the proportion of severe/mild cases that will seek medical care, the proportion of medical care seeking cases who will have a specimen submitted, the proportion of specimens that will be analysed for the particular pathogen and the sensitivity of the test method.

[6] http://www.stats.govt.nz/browse_for_stats/population/estimates_and_projections/national-pop-estimates.aspx

While there are likely to be some differences between the New Zealand and US situations, the US factors were used unadjusted.

The equivalent to the under-diagnosis factor from previous New Zealand incidence estimates included New Zealand-specific (but not pathogen-specific) information on GP requested submission of specimens (Sarfati *et al.*, 1997). However, the largest component of the scaling factors came from various overseas studies (IID1, various Dutch studies, etc.). The components of the US under-diagnosis factors were considered and no robust New Zealand pathogen-specific data could be identified to be substituted.

Proportion hospitalised

The US study used the same data sources used for case number data to determine a proportion hospitalised for each year. These data were then sampled as an empirical distribution.

Wherever possible, New Zealand proportions of hospitalised cases were calculated in the manner used to calculate this proportion for the 'Annual Report Concerning Foodborne Disease in New Zealand' (Lim *et al.*, 2010; Williman *et al.*, 2009). These are calculated by dividing the number of hospital admissions for a particular year associated with a particular pathogen by the number of notifications for that pathogen. Data were available to calculate these proportions for the years 2002-2009. Hospital admissions were used in preference to indications of hospitalisation in the notifiable diseases database (Episurv), as information from the latter source is often incomplete. The exception to this was for tuberculosis due to *M. bovis*. For this organism the number of hospital admissions was much greater than the number of notifications and the rate of hospitalisation for this organism was calculated using information from the notifiable disease surveillance system only.

For organisms where the New Zealand incidence was calculated from outbreak-related cases there are often no reports of outbreak-related cases being hospitalised. It was assumed that this was due to the small number of cases involved. For these organisms, data on the proportion of cases hospitalised was taken from the US study.

Proportion died

The US study used the same data sources used for case number data to determine a proportion of fatalities for each year. These data were then sampled as an empirical distribution.

New Zealand data on case fatality was taken from the national notifiable disease surveillance system (Episurv). This data source was used in preference to New Zealand mortality data due to the time lag before release of the New Zealand mortality data (the most recent data are available for 2007). This approach allows calculation of case fatality rates (percentages) for notifiable diseases for each year and for these organisms the New Zealand approach will be the same as the US approach. Proportions were calculated for the years 2000-2009.

For two notifiable diseases (brucellosis and cholera) there were a very small number of cases notified and no deaths. For cholera, the US COVIS system also registered no deaths for the period 2000-2007 and a zero value was used for the cholera death rate. For brucellosis, the US study used a mean case fatality rate of 0.9%. This rate was applied to the New Zealand study.

For organisms where the New Zealand incidence was calculated from outbreak-related cases there are often no reports of outbreak-related fatalities. It was assumed that this was due to the small number of cases involved. For these organisms, data on the proportion of fatal cases was taken from the US study.

Underdiagnosis factor for hospitalisations/deaths

The US study assumed that a proportion of hospitalisations and deaths due to the organisms of interest would be incorrectly diagnosed. An arbitrary factor of two was used to scale up estimates of hospitalised and fatal cases. This was not applied in situations where cases numbers were derived by scaling down.

The US scaling factor was applied to hospitalisation and fatalities for all calculations involving scaling up in the current study.

Proportion travel-related (or proportion domestically acquired)

The US study derived an estimate of the proportion travel-related cases from surveillance data or epidemiological investigations. The derived proportion was used as the modal/most likely value for a pert distribution with the minima and maxima being a 50% relative increase/decrease on this value on an odds scale.

For notified diseases in New Zealand risk factor information is collected. This often includes information on whether the case had travelled overseas during the incubation period of the organism. These data allow an estimate of the proportion travel-related cases to be determined for each surveillance year. Where this was possible for a particular pathogen, a proportion was calculated based on aggregated data for the years 2003-2009 and these values used as the modal value for a pert distribution, as for the US study. Due to changes in case report forms travel information was not available in all years for all organisms and in some cases data were from a narrower range of years.

For several organisms this information is not available:

- Brucellosis. Surveillance summaries have included a comment that "there is no evidence of locally-acquired brucellosis since 1998". To allow for some uncertainty in this statement, the proportion travel related was represented by a pert distribution with modal value 1.0 and minimum and maximum values of 0.99 and 1.0. This contrasts with the US situation where only 16% of cases were believed to be travel-related.
- Tuberculosis from *Mycobacterium bovis*. The expression 'travel-related' tends to have different meaning with respect to tuberculosis and the estimate used in the US study (70%) derives from an epidemiological study in which 70% of a case series of *M. bovis* tuberculosis cases were born outside the US (Winters *et al.*, 2005). Analysis

of the latest three years of New Zealand surveillance data found that, on average, 71% of cases were either new cases born outside New Zealand or reactivated cases with an original diagnosis outside New Zealand. On this basis, the US estimate for travel-related cases will be used for the current study. However, it is worth noting that an analysis of tuberculosis due to *M. bovis* for the period 1996-2003 found that only 33% of *M. bovis* cases were born outside New Zealand, compared to 70% for *M. tuberculosis* (Baker *et al.*, 2003).

- Cholera. All cholera cases reported in New Zealand during the period 2000-2009 (n = 8) reported overseas travel during the incubation period for the organism.

For organisms where estimates were based on outbreak-related cases, no information on the proportion travel-related was usually available. In most cases, the proportions used in the US study were applied, which generally assumed that most cases were domestically acquired. A recently updated Risk Profile found that of the 32 confirmed *V. parahaemolyticus* notified cases between 2000 and 2010, 12 were suspected to be acquired overseas (including five cases associated with seafood privately imported from the Pacific Islands and consumed in New Zealand) (Lake *et al.*, 2010b). Of the 29 cases reported from 6 outbreaks over the same period, 18 cases were suspected of being caused by seafood privately imported from the Pacific Islands, while the remaining 11 cases were acquired overseas. Thus the proportion related to overseas travel was 7/32 notifications and 11/29 outbreak cases. These data were used to define a beta distribution for the proportion of New Zealand *V. parahaemolyticus* infection cases that were travel-related.

For organisms for which the calculations are top down, the proportion travel-related was taken from the US study.

Proportion foodborne

US estimates for this proportion were derived from a range of epidemiological studies.

The New Zealand expert consultation conducted in 2005 derived estimates of the proportion foodborne for a number of organisms (Cressey and Lake, 2005). Where these estimates are available they were used.

Where the organism had not been included in the New Zealand expert consultation US estimates of the proportion foodborne were used.

Organisms excluded

No New Zealand information was available for several organisms included in the US study, for which incidence was calculated by scaling up. In other words, there were no credible New Zealand figures to scale up from. These were:

- *Clostridium botulinum*. No cases notified in New Zealand since 1985.
- *Cyclospora cayetanensis.* No information on infections in New Zealand.
- *Escherichia coli*, enterotoxigenic (ETEC). Only one potential ETEC outbreak has been reported in New Zealand, in 1981.
- *Escherichia coli*, diarrheagenic other than STEC and ETEC. The US study assumes that incidence of disease due to these organisms will be the same as the incidence of

disease due to ETEC. As no New Zealand estimate of disease due to ETEC has been made, none will be included for these organisms.

- *Streptococcus* spp., Group A. One outbreak caused by group A *Streptococcus* has been reported in New Zealand during the period 2002-2009. This was classed as a non-enteric outbreak and the outbreak was believed to have occurred through person to person transmission.
- *Trichinella* spp. No cases of trichinellosis have been reported in New Zealand since 2001.
- *Vibrio vulnificus.* No cases or outbreaks of disease due to *V. vulnificus* have been reported in New Zealand during the period 2000-2009. Only three cases of *V. vulnificus* infection have been recorded New Zealand. Two of these cases presented at a Whakatane hospital with wound infections (Wright, 1991). The third case was recorded by Rotorua hospital in 1989 (McCoubrey, 1996). The patient in this case died from septicaemia, but further details are not available
- *Vibrio* spp., other. Two cases of *V. mimicus* have been reported (one in 2009 and one in 2010), but this is not considered to be sufficient basis for an analysis.

These organisms were excluded from the current study.

Salmonella Paratyphi

The US study included disease due to *Salmonella* Paratyphi with non-typhoidal *Salmonella* spp.. *S.* Paratyphi is notifiable in New Zealand and much of the information necessary to calculate incidence, etc. for this organism is available. It was considered that the aetiology of paratyphoid fever in New Zealand was more closely aligned to typhoid fever than salmonellosis.

S. Paratyphi was treated separately in the current study. Where New Zealand specific information was lacking (e.g. underdiagnosis factor, proportion foodborne) information from the US study for *S.* Typhi was used.

Organism specific details

The organism specific notes below identify aspects in which the modelling of disease incidence in New Zealand differed from that in the US and provides more detail of the generic aspects identified above.

Astrovirus

All US factors were applied to New Zealand population estimates.

Bacillus cereus

Incidence estimates were based on New Zealand outbreak cases numbers for years 2002-2009. No outbreak-related hospitalisations or deaths due to *B. cereus* were reported in New Zealand in the period 2002-2009. This is probably due to the small number of outbreak-related cases in New Zealand (n = 0-51 per annum). Rates of hospitalisation (mean = 0.4%) and death (mean = 0.0%) were taken from the US study. An estimate of the proportion of

travel-related cases was taken from the US study (mean = <1%), while an estimate of proportion foodborne was taken from the 2005 New Zealand expert consultation (Cressey and Lake, 2005).

Brucella spp.

Incidence estimates were based on New Zealand notifiable disease data. However, it should be noted that only 12 cases of brucellosis have been notified in New Zealand during the years 2000-2009. The proportion hospitalised was represented by an empirical distribution of the proportion hospitalised in each year that brucellosis cases were notified in New Zealand. The mean of this distribution is 0.73, compared to a mean of 0.55 used for the US study. No deaths were reported from brucellosis in New Zealand during the surveillance period used. The proportion used in the US study (mean = 0.9%) was applied.

Surveillance summaries have included a comment that "there is no evidence of locally-acquired brucellosis since 1998". To allow for some uncertainty in this statement, the proportion travel related was represented by a pert distribution with modal value 1.0 and minimum and maximum values of 0.999 and 1.0. This contrasts with the US situation where only 16% of cases were believed to be travel-related.

An estimate of the proportion foodborne was taken from the US study (mean = 50%).

Campylobacter spp.

Incidence estimates were based on New Zealand notified cases numbers for years 2002-2009. The proportion hospitalised was calculated from New Zealand hospital discharges and notifications for the years 2002-2009 and represented by an empirical distribution (mean = 7.2%). The proportion of deaths was taken from New Zealand notifiable disease data for the years 2000-2009 and represented by an empirical distribution (mean = 0.008%). The proportion travel-related was taken from New Zealand surveillance summary reports for the period 2003-2009 and represented by a pert distribution (modal value = 6.8%). An estimate of proportion foodborne was taken from the 2005 New Zealand expert consultation (Cressey and Lake, 2005).

Clostridium botulinum

No botulism cases have been notified in New Zealand since 1985, providing no base figures to scale up from. No estimate was made for this organism.

Clostridium perfringens

Incidence estimates were based on New Zealand outbreak case numbers for years 2002-2009. No outbreak-related hospitalisations or deaths due to *C. perfringens* were reported in New Zealand in the period 2002-2009. This is probably due to the small number of outbreak-related cases in New Zealand (n =38-215 per annum). Rates of hospitalisation (mean = 0.6%) and death (mean = 0.04%) were taken from the US study. Estimates of the proportion of travel-related cases (virtually none) and the proportion foodborne (virtually all) were also taken from the US study.

Cryptosporidium spp.

Incidence estimates were based on New Zealand notifications for years 2000-2009. The proportion hospitalised was calculated from New Zealand hospital discharges and notifications for the years 2002-2009 and represented by an empirical distribution (mean = 4.3%). The proportion of deaths was taken from New Zealand surveillance reports for the years 2000-2009 and represented by an empirical distribution (mean = 0.00%; no deaths). The US study included an average 0.3% case fatality rate, but the relatively high number of notifications in New Zealand (600-1200 per annum) and the lack of any reported fatalities suggest that this figure is not applicable to New Zealand.

The proportion travel-related was taken from New Zealand surveillance summary reports for the period 2006-2009 and represented by a pert distribution (modal value = 7.7%). An estimate of proportion foodborne was taken from the US study (mean = 8%).

Cyclospora cayetanensis

No information on cases of this parasite in New Zealand was found. While it is likely that occasional cases will occur in New Zealand, they will almost certainly be travel-related. No estimate was made for this organism.

Escherichia coli, enterotoxigenic (ETEC)

The US study estimated ETEC cases by scaling up from outbreak-related cases. Only one potential ETEC outbreak has been reported in New Zealand, in 1981 (Bettelheim and Reeve, 1982). No estimate was made for this group of organisms.

Escherichia coli, shiga toxin-producing (VTEC/STEC) O157

In the US study STEC O157 was treated separately to other STEC types (non-O157 STECs). At some US surveillance sites, similar numbers of O157 and non-O157 cases are reported. In New Zealand, typing data allows the proportion of VTEC/STEC cases due to O157 to be determined for the period 2004-2009.

The proportion hospitalised was calculated from New Zealand hospital discharges and notifications for all VTEC/STEC for the years 2006-2009 and represented by an empirical distribution (mean = 16.7%). It should be noted that this figure is substantially lower than the figure for the proportion of hospitalisations for VTEC/STEC O157 in the US study (mean = 46.2%).

The proportion of deaths was taken from New Zealand surveillance reports for the years 2000-2009 for all VTEC/STEC and represented by an empirical distribution (mean = 0.07%). The US study included an average 0.5% case fatality rate.

The proportion travel-related was taken from New Zealand surveillance summary reports for the period 2006-2009 and represented by a pert distribution (modal value = 5.0%). An

estimate of proportion foodborne was taken from the 2005 New Zealand expert consultation and represents all VTEC/STEC (Cressey and Lake, 2005).

Escherichia coli, shiga toxin-producing (VTEC/STEC) non-O157

In New Zealand, typing data allows the proportion of VTEC/STEC cases due to serotypes other than O157 to be determined for the period 2004-2009.

The US study used differing figures for O157 and non-O157 STECs for the proportions hospitalised, dead, travel-related and foodborne. There are no New Zealand data to support a differing aetiology for O157 and non-O157 STECs and these proportions have been kept the same for both classes of organisms. The US study also included differential underdiagnosis factors (higher for non-O157 STECs) and this is plausible for New Zealand, as few clinical laboratories currently test for non-O157 STECs (King *et al.*, 2007). The US underdiagnosis factors have been adopted for the current estimates of New Zealand incidence.

Escherichia coli, diarrheagenic other than STEC and ETEC

The US study assumes that incidence of disease due to these organisms will be the same as the incidence of disease due to ETEC. As no New Zealand estimate of disease due to ETEC has been made, none will be included for these organisms.

Giardia intestinalis

Incidence estimates were based on New Zealand notifications for the years 2000-2009. The proportion hospitalised was calculated from New Zealand hospital discharges and notifications for the years 2002-2009 and represented by an empirical distribution (mean = 3.4%). The proportion of deaths was examined for New Zealand surveillance reports for the years 2000-2009. No fatalities due to giardiasis were reported and a zero value was applied for the fatality rate. The US study included an average 0.1% case fatality rate, but the relatively high number of notifications in New Zealand (1200-1700 per annum) and the lack of any reported fatalities suggest that a zero death rate should be used for the current estimation process.

The proportion travel-related was taken from New Zealand surveillance summary reports for the period 2005-2009 and represented by a pert distribution (modal value = 23.9%). An estimate of proportion foodborne was taken from the US study (mean = 7%).

Hepatitis A virus

Incidence estimates were based on New Zealand notifications for the years 2000-2009. The proportion hospitalised was calculated from New Zealand hospital discharges and notifications for the years 2002-2009 and represented by an empirical distribution (mean = 56.6%). The proportion of deaths was examined for New Zealand surveillance reports for the years 2000-2009. No fatalities due to hepatitis A were reported and a zero value was applied for the fatality rate. While the US study included a lower rate of hospitalisation (mean = 31.5%), it included an average 2.4% case fatality rate. While it seems plausible that the

occasional fatality due to hepatitis A virus will occur, the US case fatality rate appears unrealistically high for the New Zealand situation.

The proportion travel-related was taken from New Zealand surveillance summary reports for the period 2003-2009 and represented by a pert distribution (modal value = 52.3%). An estimate of proportion foodborne was taken from the US study (mean = 7%).

Listeria monocytogenes

Incidence estimates were based on New Zealand notifications for the years 2000-2009. The proportion hospitalised was calculated from New Zealand hospital discharges and notifications for the years 2002-2009 and represented by an empirical distribution (mean = 109%; more than one hospitalisation for some cases in the same year). The proportion of deaths was taken from New Zealand surveillance reports for the years 2000-2009 and represented by an empirical distribution (mean = 14.9%). This case fatality rate is very similar to that used in the US study, which included an average 15.9% case fatality rate.

The proportion travel-related was taken from New Zealand surveillance summary reports for the period 2007-2009 and represented by a pert distribution (modal value = 8.0%). An estimate of proportion foodborne was taken from the 2005 New Zealand expert consultation and is very similar to the US estimate (Cressey and Lake, 2005).

Mycobacterium bovis

Case numbers for tuberculosis notifications were taken from New Zealand notification system for years 2000-2009 and combined with estimates of the proportion of notifications that are due to *M. bovis* from tuberculosis surveillance reports for the period 2000-2009[7] (mean = 2.1%).

The course of tuberculosis means that cases may be admitted to hospital on multiple occasions over an extended period of time. Therefore, a calculation of hospital admissions divided by notifications may overestimate the proportion cases that are hospitalised. For this reason, the proportion hospitalised was based on national surveillance data, as these estimates of hospitalised proportions were based on the same case reports. The hospitalisation rate for all tuberculosis cases was used for those cases due to *M. bovis* and was taken from the years 2003-2009 and represented by an empirical distribution (mean = 58.9%)

The proportion of deaths was taken from New Zealand surveillance reports for the years 2000-2009 for tuberculosis and represented by an empirical distribution (mean = 1.4%). While the hospitalisation rate for New Zealand is very similar to that used in the US study (mean = 55%), the New Zealand case fatality rate is much lower than that used in the US study (4.7%).

Due to the long time course of tuberculosis, the expression ‘travel-related’ tends to have a different meaning with respect to tuberculosis. The estimate used in the US study (70%) derive from an epidemiological study in which 70% of a case series of *M. bovis* tuberculosis

[7] http://www.surv.esr.cri.nz/surveillance/AnnualTBReports.php

cases were born outside the US (Winters *et al.*, 2005). These cases are classified as travel-related. Analysis of the latest three years of New Zealand surveillance data found that, on average, 71% of cases were either new cases born outside New Zealand or reactivated cases with an original diagnosis outside New Zealand. On this basis, the US estimate for travel-related cases was used for the current study. However, it is worth noting that an analysis of tuberculosis due to *M. bovis* for the period 1996-2003 found that only 33% of *M. bovis* cases were born outside New Zealand, compared to 70% for *M. tuberculosis* (Baker *et al.*, 2003).

An estimate of proportion foodborne (most likely 28%) was taken from the 2005 New Zealand expert consultation (Cressey and Lake, 2005). The US study used a mean estimate of 95% foodborne for tuberculosis due to *M. bovis.*

Norovirus

The US estimation of norovirus cases uses the overall rate of acute gastrointestinal illness and apportions a mean of 11% of this disease to norovirus. Data from the New Zealand AGI study conducted in 2006-2007 was used to apply this approach to the New Zealand population (Adlam *et al.*, 2011). It should be noted that the US studies used for this calculation reported approximately half the rate of AGI in the community to the New Zealand study (0.6 illnesses per person per annum for the US, compared to 1.1 for New Zealand).

The rate of hospitalisation was also taken from the New Zealand AGI study (3/296 = 1.0%). This is higher than the figure used in the US study (modal value 0.3%), based on hospital discharge data.

The New Zealand AGI study did not provide any information for calculation of a case fatality rate. Analysis of New Zealand norovirus outbreak cases does provide estimates of case fatality (mean = 0.12% for 2003-2009). However, outbreak analysis is likely to only capture more serious cases of disease and this proportion of fatalities is unlikely to be valid at a population level. The US study described the fatality rate by a pert distribution, with modal value 0.000026 (0.0026%). This appears plausible and was used for the current study.

The proportion travel-related was taken from the US study, while the proportion foodborne was taken from the 2005 New Zealand expert consultation (Cressey and Lake, 2005).

Rotavirus

The incidence of rotavirus infection in the US was estimated on the basis that 75% of children experience an episode of clinical illness due to rotavirus by 5 years of age. All US factors were applied to New Zealand population estimates.

***Salmonella enterica,* non-typhoidal serotypes**

The US study includes *Salmonella* Paratyphi with normal enteric *Salmonella*. However, the aetiology of these *Salmonella* types appears to be quite distinct in New Zealand, with *Salmonella* Paratyphi having more in common with *Salmonella* Typhi[8]. In the current study *Salmonella* Paratyphi has been treated as a separate disease-causing agent.

[8] http://www.cph.co.nz/Files/DiseaseReport05.pdf

For non-typhoidal *Salmonella*, case numbers were taken from New Zealand notification system for years 2000-2009. The proportion hospitalised was calculated from New Zealand hospital discharges and notifications for the years 2002-2009 and represented by an empirical distribution (mean = 12.3%). The proportion of deaths was taken from New Zealand surveillance reports for the years 2000-2009 and represented by an empirical distribution (mean = 0.09%).

The proportion travel-related was taken from New Zealand surveillance summary reports for the period 2003-2009 and represented by a pert distribution (modal value = 18.4%). The proportion foodborne was taken from the 2005 New Zealand expert consultation (Cressey and Lake, 2005).

Salmonella Paratyphi

For *Salmonella* Paratyphi, case numbers were taken from New Zealand notification system for years 2000-2009. The proportion hospitalised was calculated from New Zealand hospital discharges and notifications for the years 2002-2009 and represented by an empirical distribution (mean = 24.9%). The proportion of deaths was examined for New Zealand surveillance reports for the years 2000-2009. No fatalities due to *S.* Paratyphi were reported and a zero value was applied for the fatality rate.

The proportion travel-related was taken from New Zealand surveillance summary reports for the period 2003-2009 and represented by a pert distribution (modal value = 73.5%). For the proportion foodborne, the distribution used in the US study for *Salmonella* Typhi was used (mean = 96%).

The *Salmonella* Typhi expansion factor for underdiagnosis from the US study was used for *Salmonella* Paratyphi.

Salmonella Typhi

For *Salmonella* Typhi, case numbers were taken from New Zealand notification system for years 2000-2009. The proportion hospitalised was calculated from New Zealand hospital discharges and notifications for the years 2002-2009 and represented by an empirical distribution (mean = 78.9%). The proportion of deaths was examined for New Zealand surveillance reports for the years 2000-2009. No fatalities due to *S.* Typhi were reported and a zero value was applied for the fatality rate. These proportions are almost identical to those used in the US study (75.7% and 0%, respectively).

The proportion travel-related was taken from New Zealand surveillance summary reports for the period 2003-2009 and represented by a pert distribution (modal value = 76.4%). For the proportion foodborne, the distribution used in the US study was used (mean = 96%).

Sapovirus

As with rotavirus, the incidence of sapovirus infection in the US was estimated on the basis that 75% of children experience an episode of clinical illness by 5 years of age. All US factors were applied to New Zealand population estimates.

Shigella spp.

Case numbers were taken from New Zealand notification system for years 2000-2009. The proportion hospitalised was calculated from New Zealand hospital discharges and notifications for the years 2002-2009 and represented by an empirical distribution (mean = 23.6%). The proportion of deaths was examined for New Zealand surveillance reports for the years 2000-2009. No fatalities due to shigellosis were reported and a zero value was applied for the fatality rate. The hospitalisation rate is similar to that used in the US study (mean = 20.2%), but a 0.1% case fatality rate was used in the US study.

The proportion travel-related was taken from New Zealand surveillance summary reports for the period 2003-2009 and represented by a pert distribution (modal value = 57.8%). The proportion foodborne was taken from the US study (mean = 31%).

Staphylococcus aureus

Incidence estimates were based on New Zealand outbreak case numbers for the years 2002-2009. Hospitalisations due to *Staphylococcus aureus* outbreak cases were only reported in one year and the rate for that year was quite high (36.4%). No *Staphylococcus aureus* outbreak associated deaths were reported in the period 2002-2009. This is probably due to the small number of outbreak-related cases in New Zealand. Rates of hospitalisation (mean = 6.4%) and death (mean = 0.04%) were taken from the US study. Estimates of the proportion of travel-related cases (virtually none) and the proportion foodborne (virtually all) were taken from the US study.

Streptococcus spp., Group A

Estimates for the incidence of disease due to this organism in the US were based on scaling up from numbers of outbreak-related cases. One outbreak caused by group A *Streptococcus* has been reported in New Zealand during the period 2002-2009. This was classed as a non-enteric outbreak and the outbreak was believed to have occurred through person to person transmission. No estimate was made for this group of organisms.

Toxoplasma gondii

Incidence of disease due to this parasite was calculated by an approach involving scaling down from the total population. All US factors were applied to New Zealand population estimates.

Trichinella spp.

No cases of trichinellosis have been reported in New Zealand since 2001. No estimate was made for this organism.

Vibrio cholerae, toxigenic

The US maintains a passive surveillance system for disease due to infection with *Vibrio* species (COVIS). In New Zealand cholera is a notifiable disease.

Case numbers were taken from New Zealand notification system for years 2000-2009. However, it should be noted that only eight cases of cholera have been notified in New Zealand during this period. The proportion hospitalised was calculated from New Zealand hospital discharges and notifications for the years 2002-2009 and represented by an empirical distribution (mean = 45.8%). The proportion of deaths was taken from New Zealand surveillance reports for the years 2000-2009 and represented by an empirical distribution (mean = 0.0%; no deaths). The hospitalisation and death rates are similar to that used in the US study (mean = 43.1% and 0.0%, respectively).

All cholera cases reported in New Zealand during the period 2000-2009 (n = 8) reported overseas travel during the incubation period for the organism and it was assumed that 100% of New Zealand cases would be travel related. The proportion foodborne was taken from the US study (mean = 100%).

Vibrio vulnificus

The US maintains a passive surveillance system for disease due to infection with *Vibrio* species (COVIS).

No cases or outbreaks of disease due to *V. vulnificus* have been reported in New Zealand during the period 2000-2009 (Lake *et al.*, 2010b). Only three cases of *V. vulnificus* infection have been recorded New Zealand. Two of these cases presented at a Whakatane hospital with wound infections (Wright, 1991). The third case was recorded by Rotorua hospital in 1989. The patient in this case died from septicaemia, but further details are not available (McCoubrey, 1996).

No estimate was made for this organism.

Vibrio parahaemolyticus

The US maintains a passive surveillance system for disease due to infection with *Vibrio* species (COVIS).

For New Zealand, the incidence of disease due to infection with *V. parahaemolyticus* was estimated by scaling up from outbreak-related cases, using the methodology for this approach included in the US study.

New Zealand outbreak data for the years 2002-2009 were reviewed. Outbreak cases were only reported in 2007 (n = 11 cases) and 2009 (n = 7 cases). Hospitalisations due to *V. parahaemolyticus* were modelled as a uniform distribution with the limits being the rates that occurred in 2007 and 2009 (27.3% and 0.0%, respectively). No *V. parahaemolyticus* outbreak associated deaths were reported in New Zealand in the period 2002-2009. This is probably due to the small number of outbreak-related cases in New Zealand. Case fatality rates (mean = 0.9%) were taken from the US study.

A recent New Zealand study concluded that 7/32 (21.9%) confirmed *V. parahaemolyticus* infection cases may be travel-related, while 11/29 (37.9%) of outbreak-related cases were travel-related (Lake *et al.*, 2010b). It should be noted that the difference in outbreak cases between the current study (18) and Lake *et al.* (29) is due to the latter study including data from 1999-2000. These data were used to define a beta distribution for the proportion of New Zealand *V. parahaemolyticus* infection cases that were travel-related. The proportion foodborne was taken from the 2005 New Zealand expert consultation.

***Vibrio* spp., other**

No New Zealand information was available on infection with *Vibrio* species, other than *V. parahaemolyticus, V. cholerae* and *V. vulnificus.* No estimate was made for this group of organisms.

Yersinia enterocolitica

Case numbers were taken from New Zealand notification system for years 2000-2009. The proportion hospitalised was calculated from New Zealand hospital discharges and notifications for the years 2002-2009 and represented by an empirical distribution (mean = 8.3%). The proportion of deaths was taken from New Zealand surveillance reports for the years 2000-2009 and represented by an empirical distribution (mean = 0.02%).

The proportion travel-related was taken from New Zealand surveillance summary reports for the period 2004-2009 and represented by a pert distribution (modal value = 5.9%). The proportion foodborne was taken from the 2005 New Zealand expert consultation.

Unspecified Pathogens

The incidence of foodborne illness due to ‘unspecified pathogens’ was determined in the US study by subtracting the incidence of 24 pathogens which may cause acute gastroenteritis from the total burden of acute gastrointestinal illness and then applying weighted mean estimates of the proportion domestically acquired and the proportion foodborne for the 24 specific pathogens.

This approach was followed to derive an equivalent estimate for New Zealand. Estimates of the total burden of acute gastrointestinal illness were derived from the New Zealand AGI study (Adlam *et al.*, 2011).

Proportions domestically acquired and proportions foodborne were derived in the following manner:

- Run model for 24 major pathogens for 100,000 iterations.
- At each iteration, calculate to separate overall percentages of cases, hospitalisation and deaths that were domestically acquired and the separate overall percentages of cases, hospitalisations and deaths that were foodborne among those that were domestically acquired.
- Fit a pert distribution (four parameter) to the simulated values for each of these percentages.
- Increase the variance parameter of the four parameter pert distribution by a factor of two.
- Run a simulation applying these pert distributions to the difference between total cases, hospitalisations and deaths due to acute gastrointestinal illness and the total cases, hospitalisations and death due to the specified major pathogens.