Sequelae of Foodborne Illness Caused by 5 Pathogens, Australia, Circa 2010

Technical Appendix 1

Sequelae Incidence after Bacterial Gastroenteritis: The Sequelae Multiplier

For each sequel, a multiplier was used that estimated the proportion of bacterial gastroenteritis cases that developed into chronic sequelae. This appendix summarizes the relevant studies published during 1995–2012, which we selected for review, as well as the sequelae multipliers that were estimated for Guillain-Barré syndrome (GBS), hemolytic uremic syndrome (HUS), irritable bowel syndrome (IBS), and reactive arthritis (ReA).

GBS

A few studies have quantified the incidence of GBS illness following *Campylobacter* spp. infection by using large cohorts of patients or the literature (online Technical Appendix 1 Table 1). In a population-based cohort study in the United Kingdom, including 2 months of follow-up, 3 cases of GBS occurred among 15,587 *Campylobacter* spp. cases. This yielded a rate of 19.2 cases of GBS per 100,000 cases of campylobacteriosis (1). In Sweden, 0.03% of a cohort of 29,567 persons with laboratory-confirmed *C. jejuni* infection developed GBS illness after 2 months of follow-up, yielding an annual incidence of 30.4 cases of GBS per 100,000 cases (95% CI 13.9–57.8) of *C. jejuni* infection (2). In a literature review, Allos (3) estimated that in the United States, GBS develops in 1 of every 1,058 cases, or 94.5 per 100,000 cases, of *C. jejuni* infection. Baker et al. (4) performed a study of hospital records in New Zealand, which found a rate of 414 cases of GBS per 100,000 *Campylobacter* spp. hospitalizations.

For the sequelae multiplier, a midpoint of 30.4 cases of GBS per 1000,000 cases of campylobacteriosis was taken from the study by McCarthy and Gieseke (2) using a minimum value of 19.2 per 100,000 from the UK study and a maximum value of 94.5 per 100,000 from the study by Allos (3). Although the study by Baker et al. (4) is a valuable one, we excluded it from...
the calculation of our sequelae multiplier because persons hospitalized with *Campylobacter* spp. infection may not be representative of *Campylobacter* spp. cases in the community.

Technical Appendix 1 Table 1. Incidence of GBS after infection with *Campylobacter* spp.*

<table>
<thead>
<tr>
<th>Reference</th>
<th>Study years</th>
<th>Type of study</th>
<th>Country</th>
<th>No. GBS cases/Campylobacter spp. patients</th>
<th>Incidence per 100,000 (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baker et al. (4)</td>
<td>1995–2008</td>
<td>Cohort</td>
<td>New Zealand</td>
<td>35/8,448 hospitalizations</td>
<td>414 (373–459)</td>
</tr>
<tr>
<td>Tam et al. (1)</td>
<td>1991–2001</td>
<td>Cohort</td>
<td>UK</td>
<td>3/15,587 cases</td>
<td>19.2 (17.1–21.5)</td>
</tr>
<tr>
<td>McCarthy and Giesecke (2)</td>
<td>1987–1995</td>
<td>Cohort</td>
<td>Sweden</td>
<td>9/29,563 cases</td>
<td>30.4 (13.9–57.8)</td>
</tr>
<tr>
<td>Allos (3)</td>
<td>1964–1996†</td>
<td>Review and estimation</td>
<td>Global/USA</td>
<td>1/1058 cases</td>
<td>94.5 (2.4–525)</td>
</tr>
</tbody>
</table>

*GBS, Guillain-Barré syndrome.
†Years of reviewed studies.

### HUS

A variety of organisms, drugs and conditions can initiate the symptoms of HUS, but the majority of HUS cases are post-diarrheal—usually caused by Shiga toxin–producing *Escherichia coli* (STEC) (5). In developed communities, STEC is the most commonly implicated organism in HUS (6), and in children, 90% of HUS cases are due to STEC (5). HUS is also associated with *Shigella dysenteriae* serotype 1, particularly in less developed communities (6); however, a recent systematic review was unable to find an adequate number of studies to quantify the association between *S. dysenteriae* serotype 1 and HUS (7). In addition, in a few studies, HUS has been associated with *Clostridium difficile* and *Salmonella enterica* serotype Typhi, but the evidence is limited (8–10). Therefore we estimated food-related HUS cases as a sequel to STEC, which may create an underestimation of HUS if there are food-related HUS cases in Australia from other organisms.

Several sources have reported that 3%-7% of sporadic STEC infections develop into HUS (11–14). Australian studies support this estimate range. Vally et al. (15) examined South Australian surveillance data and identified 14 HUS cases and 460 STEC cases, resulting in an estimate of 3% of STEC cases developing into HUS. Sixty percent of HUS case-patients were ≤15 years of age. In addition, in a case–control study in 6 Australian jurisdictions, 113 STEC case-patients were identified, 44 of whom were infected with O157 and 66 who were infected with non-O157 (14). Eight (7%) of all the STEC cases, 1 (2%) case-patient with O157, and 7 (10%) case-patients infected with non-O157 developed HUS (14). Although STEC O157 is more commonly associated with HUS worldwide (6), data on geographic differences in STEC serotypes suggest that in Australia, “non-O157:H7 STEC strains predominate,” and STEC O157:H7 is not as frequently implicated in “diarrhea-associated HUS” (16).
Overseas studies have reported higher proportions of STEC infections developing into HUS. In a cohort study of Argentinian children, aged ≤15 years, 8 (8.6%) of 93 STEC patients developed HUS (17). Through enhanced surveillance in the Netherlands, Van Duynhoven et al. (18) found that HUS developed in 12 of 82 (14.6%) patients. Seventy-five percent of HUS case-patients were ≤15 years (18). With the highest proportion from all reviewed studies, a Swiss linkage study found that HUS developed in 13 (29.5%) of 44 STEC patients, all of whom were ≤15 years of age (19). Several studies on the incidence of HUS after STEC outbreaks have found that ≈20% of STEC cases develop into HUS (20–23). However, Sigmundsdottir et al. found no HUS cases among 9 STEC outbreak patients in Iceland (24) (Technical Appendix 1 Table 2).

A sequelae multiplier proportion of 3% (95% CI 1.7%–5.4%) was chosen, based on the South Australian study by Vally et al. (15). This study was chosen because STEC surveillance in South Australia is more complete than for other Australian states (11) and would therefore give a more representative estimate for Australia than the other available studies.

**Technical Appendix 1 Table 2. Incidence of HUS after STEC**

<table>
<thead>
<tr>
<th>Reference</th>
<th>Study years</th>
<th>Study type</th>
<th>Country</th>
<th>Age of HUS case-patients</th>
<th>No. HUS cases/no. STEC cases</th>
<th>STEC cases developing into HUS, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bradley et al. (20)</td>
<td>2008</td>
<td>Epidemiology investigation and case–control: after an outbreak</td>
<td>USA</td>
<td>Median: 46 y (range 1–88 y), 60% adult</td>
<td>11/56</td>
<td>20</td>
</tr>
<tr>
<td>Lopez et al. (17)</td>
<td>2006</td>
<td>Prospective cohort Case–control: after an outbreak</td>
<td>Argentina</td>
<td>≤15 y</td>
<td>8/93</td>
<td>8.6</td>
</tr>
<tr>
<td>Neil et al. (21)</td>
<td>2009</td>
<td>Case–control: after an outbreak</td>
<td>USA</td>
<td>Not stated</td>
<td>10/57</td>
<td>18</td>
</tr>
<tr>
<td>Vally et al. (15)</td>
<td>1997–2009</td>
<td>Surveillance</td>
<td>Australia</td>
<td>Range: 5–60+, 60% aged ≤15 y</td>
<td>14/460</td>
<td>3</td>
</tr>
<tr>
<td>Frank et al. (22)</td>
<td>2011</td>
<td>Surveillance: after an outbreak</td>
<td>Germany</td>
<td>Median: 42, 88% aged &gt;15 y</td>
<td>845/3816</td>
<td>22</td>
</tr>
<tr>
<td>Kappelli et al. (19)</td>
<td>2000–2009</td>
<td>Linkage</td>
<td>Switzerland</td>
<td>Median: 3.5 y (range 0–15 y)</td>
<td>13/44</td>
<td>29.5</td>
</tr>
<tr>
<td>McPherson et al. (14)</td>
<td>2003–2007</td>
<td>Case–control</td>
<td>Australia</td>
<td>Median: 4 y (range 1–62)</td>
<td>8/113</td>
<td>7</td>
</tr>
<tr>
<td>Sigmundsdottir et al. (24)</td>
<td>2007</td>
<td>Cohort: after an outbreak</td>
<td>Iceland</td>
<td>Not stated</td>
<td>0/9</td>
<td>0</td>
</tr>
<tr>
<td>Rangel et al. (25)</td>
<td>1982–2002</td>
<td>Outbreak surveillance</td>
<td>USA</td>
<td>Not stated</td>
<td>354/8596</td>
<td>4.1</td>
</tr>
<tr>
<td>Jay et al. (23)</td>
<td>1999</td>
<td>Epidemiology investigation and case–control: after an outbreak</td>
<td>USA</td>
<td>Not stated</td>
<td>3/13</td>
<td>23</td>
</tr>
<tr>
<td>Van Duynhoven et al. (18)</td>
<td>1999–2001</td>
<td>Enhanced surveillance</td>
<td>The Netherlands</td>
<td>Range: 0–70 y, 75% aged ≤15 y</td>
<td>12/82</td>
<td>14.6</td>
</tr>
</tbody>
</table>

*HUS, hemolytic uremic syndrome; STEC, Shiga toxin–producing *Escherichia coli.*

**IBS**

There have been a few systematic reviews and/or meta-analyses on the association between intestinal infection and post-infectious IBS (PI-IBS). A recent review suggests the proportion of persons developing IBS following gastrointestinal infection is 4%–35% (26). In
2010, Haagsma et al. (27) found that 1 year after infection from nontyphoidal \textit{S. enterica} serotypes (hereafter referred to as nontyphoidal \textit{Salmonella} spp.), nontyphoidal \textit{Salmonella} spp., \textit{Shigella} spp., or \textit{Campylobacter} spp., IBS developed in 9\% (95\% CI 7.2–10.7) of patients. Similarly, in a systematic review of 18 studies, Thabane et al. (28) found a pooled incidence of PI-IBS of 10\% (95\% CI 9.4–85.6). Comparably, Halvorson et al. (29) reviewed 8 studies on nontyphoidal \textit{Salmonella} spp., \textit{Shigella} spp., bacterial unspecified, or unspecified, and their association with IBS, and calculated a median prevalence of IBS of 9.8\% (interquartile range 4.0–13.3) in the exposed group and 1.2\% Interquartile rate range 0.04–1.8) in the control group. A review by Smith and Bayles (30) found a mean prevalence of PI-IBS of 15\% from 15 studies, with species of \textit{Campylobacter}, nontyphoidal \textit{Salmonella} spp., and/or \textit{Shigella} as the most common agents of infection.

In the United Kingdom, Neal et al. (31) performed a postal survey and found that 25\% of subjects had persistently altered bowel habits after bacterial gastroenteritis from nontyphoidal \textit{Salmonella} spp., \textit{Shigella} spp., or \textit{Campylobacter} spp.; however, only 7\% met the Rome criteria for new IBS. Also in the United Kingdom, Parry et al. (32) looked at the relationship between IBS and bacterial gastroenteritis from \textit{Campylobacter} spp., nontyphoidal \textit{Salmonella} spp., \textit{Shigella} spp., \textit{E. coli} O157, and \textit{Aeromonas sobria}, and calculated an incidence of new IBS of 16.7\% in the exposed group and 1.9\% in the control group.

Studies looking at singular pathogens have also found an association between infectious gastroenteritis outbreaks and IBS. After an outbreak in 2002 in Spain, Mearin et al. (33) noted that before the outbreak, the prevalence of IBS was similar in case-patients and controls (2.9\% vs. 2.3\%); however, 3 months after the outbreak, IBS prevalence in case-patients had increased (9.2\% vs. 1.7\%), and 12 months after the outbreak, prevalence in case-patients remained higher (10.2\% vs. 0.7\%). The cumulative incidence was 7.4\% at 3 months, 10.9\% at 6 months, and 11.6\% at 12 months. In Korea, 12 months after a \textit{Shigella} spp. outbreak, Ji et al. (34) found that IBS had developed in 15 (14.9\%) of 101 case-patients and 6 of 102 (5.9\%) controls. In Canada, 2–3 years after an outbreak of \textit{E. coli} O157:H7 and \textit{Campylobacter} spp., 27.5\% of 904 subjects with self-reported gastroenteritis reported IBS, and 36.2\% of 464 subjects with clinically suspected gastroenteritis reported IBS (35). In a pediatric cohort from the Canadian outbreak, the cumulative incidence of PI-IBS for exposed subjects was 10.5\% vs. a cumulative incidence in controls of 2.5\% (36).
There have been studies on the association of *G. lamblia* with IBS; however, these have produced inconsistent results. While Wensaas et al. (37) found a high prevalence of IBS in exposed patients 2 years after acute giardiasis, Penrose et al. (38) found no linear association between *G. lamblia* and IBS, and a study by D’Anchino et al. (39) concluded that *G. lamblia* infection is a trigger for exacerbating preexisting IBS but could not conclude that *G. lamblia* causes IBS. PI-IBS has also been shown to develop after norovirus. Marshall et al. (40) performed a 2-year study after a norovirus outbreak; of the 89 respondents who reported an acute enteric illness during the outbreak and did not have preexisting IBS, 23.6% reported symptoms consistent with PI-IBS at 3 months versus 3.4% who reported symptoms but remained well during the outbreak. However, at 6, 12, and 24 months, the prevalence of IBS did not differ statistically among exposed and unexposed individuals, suggesting that PI-IBS might be more transient after viral gastroenteritis than it is after bacterial dysentery (40) (Technical Appendix 1 Table 3).

The meta-analysis by Haagsma et al. (27), which suggests that IBS develops in ≈9% (95% CI 7.2%–10.7%) of *Campylobacter* spp., nontyphoidal *Salmonella* spp., and *Shigella* spp. case-patients at 10–12 months of follow-up was chosen as the sequelae multiplier to simulate the plausible proportion of these bacterial pathogens that cause IBS using an alternate PERT distribution. While studies of multiple pathogens have found different rates of PI-IBS depending on etiology, this proportion was chosen for all 3 pathogens because it is a pooled rate that comes from a recent meta-analysis and is similar to PI-IBS rates after bacterial gastroenteritis that were reported in other studies (28,29,41).

<table>
<thead>
<tr>
<th>Reference</th>
<th>Year of publication</th>
<th>Study years</th>
<th>Country</th>
<th>Study type</th>
<th>Foodborne pathogen</th>
<th>IBS patients after infectious gastroenteritis, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Koh et al. (41)</td>
<td>2012</td>
<td>2008–2010</td>
<td>Korea</td>
<td>Prospective cohort</td>
<td>Nontyphoidal <em>Salmonella</em> spp., <em>Shigella</em> spp., STEC O157, <em>Vibrio cholerae</em>, <em>Giardia lamblia</em></td>
<td>9.2% at 3 mo†, 12.3% at 6 mo†</td>
</tr>
<tr>
<td>Wensaas et al. (37)</td>
<td>2012</td>
<td>2007–2008</td>
<td>Norway</td>
<td>Historic cohort</td>
<td><em>Giardia lamblia</em></td>
<td>46.1% at 3 y</td>
</tr>
<tr>
<td>Schwille-Kiuntke et al. (26)</td>
<td>2011</td>
<td>-</td>
<td>Global</td>
<td>Systematic review</td>
<td><em>Campylobacter</em> spp., <em>Escherichia coli</em>, <em>G. lamblia</em>, norovirus, nontyphoidal <em>Salmonella</em> spp., <em>Shigella</em> spp., <em>Trichinella britovi</em>, bacterial, viral, and parasitic gastroenteritis and travelers’ diarrhea</td>
<td>4%–36% Incidence range</td>
</tr>
<tr>
<td>Thabane et al. (36)</td>
<td>2010</td>
<td>2002–2008</td>
<td>Canada</td>
<td>Outbreak study</td>
<td><em>E. coli</em> O157:H7, <em>Campylobacter</em> spp.</td>
<td>10.5%†</td>
</tr>
<tr>
<td>Haagsma et al. (26)</td>
<td>2010</td>
<td>-</td>
<td>The Netherlands</td>
<td>Meta-analysis</td>
<td><em>Campylobacter</em> spp., nontyphoidal <em>Salmonella</em> spp.</td>
<td>9% (95% CI 7.2–10.7)</td>
</tr>
</tbody>
</table>
The causes of ReA are ambiguous because no formal definition or agreed-upon diagnostic criteria exist (42,43). Although the primary focus of the infection is usually through the gut or urogenital track, ReA has also been associated with respiratory pathogens (42). The classical gastrointestinal microbes resulting in ReA are *Yersinia enterocolitica*, nontyphoidal *Salmonella* spp., *Shigella* spp., and *Campylobacter* spp (43). and most agree that the term “ReA” should be applied only to infection caused by these gastrointestinal pathogens and *Chlamydia* spp (43); however, nonclassical ReA forms have been associated by a variety of other bacteria, including *Brucella* and *Staphylococcus*, and many authors have applied the term ReA for arthritis after infection with *C. difficile*, *Cryptosporidium*, *Giardia lamblia*, *E. coli*, and *Strongyloides* spp (43,44). With the majority of the literature focusing on the 4 classical gastrointestinal pathogens as triggers for ReA, we chose to use these to estimate the incidence of ReA due to contaminated food. If other enteric pathogens are in fact associated with ReA, our estimates of foodborne ReA may be conservative.
We were unable to find any published systematic reviews that report a global incidence rate for ReA after infection with the bacterial pathogens *Campylobacter* spp., nontyphoidal *Salmonella* spp., *Shigella* spp., and *Y. enterocolitica*. Because there are no diagnostic criteria for ReA, the case definition and the resulting incidences vary (42). The literature suggests that the incidence of ReA as a sequel to bacterial gastroenteritis varies by the enteric pathogen. For each of the bacterial enteric pathogens that precede ReA, we compiled papers that reported the proportion of cases that developed into ReA published in 2000 or later where all enteric cases were confirmed by a laboratory (Technical Appendix 1 Table 4). Because there is still quite a bit of variation in incidence in studies by pathogen, the median and range for *Campylobacter* spp., nontyphoidal *Salmonella* spp., *Shigella* spp., and *Y. enterocolitica* from the studies in Technical Appendix 1 Table 4 were calculated for the sequelae multiplier and used to simulate a distribution of the plausible proportion of cases that result in this sequel using an alternate PERT or PERT distribution, respectively. From the literature, we assume that 7% (range 2.8%-16%) of foodborne *Campylobacter* spp., 8.5% (range 0%-26%) of foodborne nontyphoidal *Salmonella* spp., 9.7% (range 1.2%-9.8%) of foodborne *Shigella* spp., and 12% (range 0%-23.1%) of foodborne *Y. enterocolitica* result in ReA. These distributions were then applied to the estimates of domestically acquired foodborne cases for each of the preceding bacterial pathogens.

<table>
<thead>
<tr>
<th>Reference</th>
<th>Study years</th>
<th>Study type</th>
<th>Country</th>
<th>ReA cases/gastroenteritis cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Schonberg-Norio et al. (45)</td>
<td>2002</td>
<td>Cross sectional</td>
<td>Finland</td>
<td>8/201 (4.0%)</td>
</tr>
<tr>
<td>Doorduyn et al. (46)</td>
<td>2005</td>
<td>Case–control</td>
<td>The Netherlands</td>
<td>20/434 (4.6%)</td>
</tr>
<tr>
<td>Townes et al. (47)</td>
<td>2002–2004</td>
<td>Cohort</td>
<td>USA</td>
<td>302/2384 (12.7%)</td>
</tr>
<tr>
<td>Schiellerup et al. (48)</td>
<td>2002–2003</td>
<td>Case–case comparison</td>
<td>Denmark</td>
<td>131/1003 (13.1%)</td>
</tr>
<tr>
<td>Pope et al. (49)</td>
<td>1966–2006</td>
<td>Review</td>
<td>Europe</td>
<td>1%–5%</td>
</tr>
<tr>
<td>Rees et al. (50)</td>
<td>1998–1999</td>
<td>Cohort</td>
<td>USA</td>
<td>9/324 (2.8%)</td>
</tr>
<tr>
<td>Hannu (51)</td>
<td>1997–1998</td>
<td>Cohort</td>
<td>Finland</td>
<td>45/609 (7.4%)</td>
</tr>
<tr>
<td>Loch and Krogfelt (52)</td>
<td>1997–1999</td>
<td>Cohort</td>
<td>Denmark</td>
<td>27/173 (15.6%)</td>
</tr>
<tr>
<td>Arnedo-Pena et al. (53)</td>
<td>2005</td>
<td>Outbreak study</td>
<td>Spain</td>
<td>6/67 (9%)</td>
</tr>
<tr>
<td>Doorduyn et al. (46)</td>
<td>2005</td>
<td>Case–control</td>
<td>The Netherlands</td>
<td>8/181 (4.4%)</td>
</tr>
<tr>
<td>Townes et al. (47)</td>
<td>2002–2004</td>
<td>Cohort</td>
<td>USA</td>
<td>204/1356 (15.0%)</td>
</tr>
<tr>
<td>Schiellerup et al. (48)</td>
<td>2002–2003</td>
<td>Case–case comparison</td>
<td>Denmark</td>
<td>104/619 (16.6%)</td>
</tr>
<tr>
<td>Lee et al. (54)</td>
<td>1999</td>
<td>Outbreak study</td>
<td>Australia</td>
<td>38/261 (14.6%)</td>
</tr>
<tr>
<td>Rees et al. (50)</td>
<td>1998–1999</td>
<td>Cohort</td>
<td>USA</td>
<td>2/100 (2.0%)</td>
</tr>
<tr>
<td>Buxton et al. (55)</td>
<td>1999–2000</td>
<td>Case–control</td>
<td>Canada</td>
<td>17/66 (25.7%)</td>
</tr>
<tr>
<td>Hannu et al. (56)</td>
<td>1999</td>
<td>Outbreak study</td>
<td>Finland</td>
<td>5/63 (7.9%)</td>
</tr>
<tr>
<td>Rudwaleit et al. (57)</td>
<td>1998</td>
<td>Outbreak study</td>
<td>Germany</td>
<td>0/286 (0%)</td>
</tr>
<tr>
<td>Urfer et al. (58)</td>
<td>1993</td>
<td>Outbreak study</td>
<td>Switzerland</td>
<td>1/156 (0.6%)</td>
</tr>
<tr>
<td>Townes et al. (47)</td>
<td>2002–2004</td>
<td>Cohort</td>
<td>USA</td>
<td>29/299 (9.7%)</td>
</tr>
<tr>
<td>Schiellerup et al. (48)</td>
<td>2002–2003</td>
<td>Case–case comparison</td>
<td>Denmark</td>
<td>10/102 (9.8%)</td>
</tr>
<tr>
<td>Rees et al. (50)</td>
<td>1998–1999</td>
<td>Cohort</td>
<td>USA</td>
<td>1/81 (1.2%)</td>
</tr>
<tr>
<td>Reference</td>
<td>Study years</td>
<td>Study type</td>
<td>Country</td>
<td>ReA cases/gastroenteritis cases</td>
</tr>
<tr>
<td>-------------------</td>
<td>-------------</td>
<td>-------------------------</td>
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<td>--------------------------------</td>
</tr>
<tr>
<td>Huovinen et al.</td>
<td>2006</td>
<td>Case–control</td>
<td>Finland</td>
<td>11/248 (4.4%)</td>
</tr>
<tr>
<td>(59)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Townes et al.</td>
<td>2002–2004</td>
<td>Cohort</td>
<td>USA</td>
<td>5/35 (14.3%)</td>
</tr>
<tr>
<td>(47)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Schiellerup et al.</td>
<td>2002–2003</td>
<td>Case–case comparison</td>
<td>Denmark</td>
<td>21/91 (23.1%)</td>
</tr>
<tr>
<td>(48)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rees et al.</td>
<td>1998–1999</td>
<td>Cohort</td>
<td>USA</td>
<td>0/8 (0%)</td>
</tr>
<tr>
<td>(50)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hannu et al.</td>
<td>1998</td>
<td>Outbreak study</td>
<td>Finland</td>
<td>4/33 (12.1%)</td>
</tr>
<tr>
<td>(60)</td>
<td></td>
<td></td>
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</tr>
</tbody>
</table>

*Incidence of ReA after Campylobacter spp. infection: median 7%, range 2.8%–16%; after Salmonella spp. infection: median 8.5%, range 0%–26%; after Shigella spp. infection: median 9.7%, range 1.2%–9.8%; after Yersinia enterocolitica infection: median 12%, range 0%–23.1%. ReA, reactive arthritis. Nontyphoidal Salmonella spp., nontyphoidal S. enterica serotypes.

**References**


