Food Safety Risk Profile for
Campylobacter species in broiler
(young) chickens

Compiled by the CCFH Working Group on Guidelines for control of Campylobacter and Salmonella spp. in broiler (young bird) chicken meat

June 2007
Some parts of this risk profile are in its present state not representative regarding the global situation due to lack of data from many countries, especially from Latin America and the Caribbean, Africa, the Near East and to a certain extent Asia. It is therefore requested that governments contribute with relevant data to the different sub-headings in the document.
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1 Hazard-food commodity combinations of concern

1.1 Hazards of concern
Campylobacter species, in particular C. jejuni and C. coli.

1.2 The food product of concern
Fresh broiler chicken meat (where fresh is defined according to CCMH); whole chicken carcasses and portions, excluding internal organs.

1.3 Occurrence of the hazard in the food chain
Campylobacter is an important cause of zoonotic enteric infections in most developed and developing nations (WHO, 2000a). The majority of infections seem to be sporadic, i.e. not associated with outbreaks. Most case-control studies have identified poultry products as important risk factors for human sporadic campylobacteriosis (WHO, 2000a).

2 Description of the public health problem

2.1 Description of the hazard

2.1.1 Taxonomy
Thermotolerant Campylobacter spp., the etiological agents of campylobacteriosis, are widespread in nature (Jones, 2001). The principal reservoirs are the alimentary tracts of wild and domesticated birds and mammals. Consequently thermotolerant Campylobacter spp., especially C. jejuni and C. coli, are commonly isolated from water sources, food animals such as poultry, cattle, pigs, and sheep, as well as from cats and dogs (Jones, 2001; FAO/WHO, 2002).

The most important species of Campylobacter are the thermotolerant (i.e. thrives at relatively high temperatures) species: C. jejuni ssp. jejuni, C. coli and C. lari (formerly known as “nalidixic acid resistant thermophilic Campylobacter spp. – NARTC”). Other species which are known to cause human illness are C. upsaliensis, C. fetus ssp. fetus and C. jejuni ssp. Doyley (EFSA, 2005). Most physiological/biochemical, epidemiological and survival information concerns C. jejuni, as this species is the most frequently recovered in human disease.

2.1.2 In vitro growth characteristics
The Campylobacter species are slender, spirally curved rods which are non-sporulating and Gram negative. Campylobacter spp. are relatively inactive biochemically, obtaining their energy from amino acids or tricarboxylic acid cycles intermediates rather than carbohydrates. This makes them difficult to speciate by use of classical biochemical tests (On, 1996), so they are often identified to species level by use of PCR-based methods (Linton et al., 1997; Bolton et al., 2002; On and Jordan, 2003).

Campylobacter are thermotolerant and grow optimally at 42°C. Neither C.jejuni nor C.coli grows below 30.5 or above 45°C. The organism is comparatively slow growing (fastest generation time approximately 1 hour) even under optimum conditions and
does not grow under refrigeration (ESR, 2006). Optimum growth is at a water activity of 0.997 (=0.5% NaCl), minimum aw ≥0.987 (=2.0% NaCl) (ESR, 2006).

Compared with other important foodborne pathogens, such as *Salmonella* species, *Campylobacter* spp. seem ill-equipped to survive outside an animal host (EFSA, 2005). They require a microaerobic atmosphere (ca. 5% oxygen and 10% carbon dioxide) and cannot multiply below about 30°C. However, even at 4°C, low-level metabolic activity can be detected, suggesting that cell integrity is maintained (Park, 2002).

In a review on survival of *Campylobacter* spp. in foods, it was established that they survive poorly in dry or acid conditions, and in sodium chloride above 2% (Jacobs-Reitsma, 2000). Their sensitivity to environmental stresses seems to be confirmed by their lack of genes analogous to those in other bacteria, enabling physiological adaptation to adverse environments - e.g., oxidative stress, osmoregulation, starvation/stationary phase, heat and cold shock (Park, 2002).

There is, however, some indication that they may be capable of other strategies, which explain their survival. A debate among *Campylobacter* researchers is the validity of the presence of viable nonculturable cells (VNC, or viable but nonculturable cells, VBNC) of *Campylobacter* (FSANZ, 2005). Those who support the concept consider that VNC are formed under unfavourable environmental conditions, where campylobacters enter a nonculturable stage i.e. viable cells cannot be detected by a routine culture method. Because VNC are viable *Campylobacter* cells, they may play a role in human campylobacteriosis (Rollins and Colwell, 1986).

### 2.1.3 Temperature effects

*Campylobacter* species are relatively sensitive to heat and irradiation, and so can readily be inactivated during cooking or on the surface of meat by heating at 55°C-60°C for several minutes (ICMSF, 1996). Numbers declined rapidly on sterile meat slices of high and normal pH when incubated at 25°C (Gill and Harris 1982). Freezing rates influence survival more than actual frozen storage. Slow freezing rates are more lethal than rapid freezing because of osmotic stress. Significant reductions in *Campylobacter* numbers were observed when inoculated chicken portions were frozen to –10°C and this effect was attributed to the long freezing time necessary to reach this temperature (19h 40min) (Whyte *et al*., 2005).

### 2.1.4 Virulence

There is a lack of knowledge regarding pathogenicity and virulence of various *Campylobacter* types in humans, especially those linked to post-infection sequelae (EFSA, 2005). The function and role of toxins in pathogenesis is yet to be fully elucidated and remains a topic of debate (ACMSF, 2005). A variety of toxins has been reported, many of which are similar to those found in some other bacteria.

Certain serotypes of *C. jejuni*, particularly Penner Serotype O19 and O41 have been more frequently associated with the Guillain-Barré syndrome (GBS) than other serotypes (AIFST, 2003). Penner Serotype O19 has been associated with GBS in Japanese studies. However, this link was not confirmed in a USA case control study, in which no specific serotypes were associated with GBS (Rees *et al*., 1995).
2.1.5 Resistance to antimicrobial agents

Antimicrobial resistance in Campylobacter spp. is increasing, and most alarming is the increasing resistance to fluoroquinolones (EFSA, 2005). Fluoroquinolone resistant C. jejuni was recognised during the late 1980s in Europe (Nachamkin et al., 2002). Studies from several countries have shown a relationship between approval of fluoroquinolones for use in food producing animals and the development of fluoroquinolone resistance in Campylobacter spp. in animals and humans (EFSA, 2005). As poultry is considered the principal source of campylobacteriosis in industrialised countries, and human-to-human transmission is uncommon, it may be assumed that the contribution from the poultry reservoir plays the leading role in the emergence of fluoroquinolone resistance in Campylobacter spp. (Smith et al., 1999).

It is estimated by modelling that 8% (mean value) of quinolone-resistant Campylobacter cases in humans in the UK are attributable to the food chain (chicken, pig-meat and crops) and 1.3% to the consumption of domestic chicken (Veterinary Laboratories Agency, UK, unpublished). There is large uncertainty about this value. All cases attributable to consumption of UK produced food were attributable to chicken that were prescribed a fluoroquinolone (due to ill-health in the flock). The largest cause of quinolone-resistant Campylobacter illness in humans was foreign travel.

In Norway, the prevalence of fluoroquinolone resistance among C. jejuni isolates from imported and indigenous sporadic human cases of campylobacteriosis and from domestic broilers was assessed (Kruse and Skov Simonsen, 2002). Among the imported human isolates, 60% were resistant to ciprofloxacin compared with 7% of indigenous human isolates. The prevalence of resistance in indigenous human isolates was comparable with the prevalence of resistance in isolates from Norwegian poultry (Kruse et al., 2002). No quinolone preparations are licensed for use in broilers in Norway.

An Australian study of 79 Campylobacter isolates from chicken, found widespread resistance to erythromycin and significant resistance to doxycycline but no resistance to enrofloxacin – a fluoroquinolone antimicrobial chemical (Korolik et al., 1996). Another Australian study (Barton et al., 2001) of antimicrobial resistance of Campylobacter isolated from chickens reported significant resistance to ampicillin, ceftazidime and tetracycline in C. jejuni and C. coli isolates. No fluoroquinolone resistance was detected and there was relatively little resistance to erythromycin or tylosin. In Australia, fluoroquinolones have never been licensed for use in food production animals (FSANZ, 2005).

2.2 Characteristics of the disease in humans

2.2.1 Acute illness

The incubation period for campylobacteriosis ranges from one day to one week and infections usually result in mild to moderate symptoms including diarrhoea (frequently with blood in the faeces), abdominal pain, fever, headache, nausea and/or vomiting (WHO, 2000b). Symptoms, which are usually self-limiting, last from one day to one week, and in up to 20% of cases, for more than a week. Because of the lack of special clinical features, campylobacteriosis is difficult to distinguish from other acute gastrointestinal illnesses.
A particular feature of *Campylobacter* infection is abdominal pain, which may become continuous and sufficiently intense to mimic acute appendicitis (FSANZ, 2005). This is the most frequent reason for admission of *Campylobacter* enteritis patients to hospital (Skirrow and Blaser, 2000).

### 2.2.2 Susceptible populations

More invasive disease such as systemic infections occur in less than 1% of patients with *C. jejuni* infections and are more common in the elderly or very young (EFSA, 2005). Rare manifestations of *C. jejuni* infections include meningitis, endocarditis and septic abortion. Persons with immunoglobulin deficiencies may show prolonged, severe, and recurrent infections. The incidence of campylobacteriosis among HIV-infected patients is higher than in the general population and complications such as recurrent infection and infection with antimicrobial-resistant strains occur more frequently among these patients (Altekruse *et al*., 1995; Slutsker *et al*., 1998).

In developing countries the public health impact of Campylobacter is most dramatic in the very young. Among the pathogens causing pediatric diarrheal diseases, *Campylobacter* is a leading cause –being especially acute during weaning. Children under 1 year of age are highly susceptible to campylobacter infections and may suffer the affects of malnutrition. Overall, children under the age of 4 are at high risk and may suffer several bouts of Campylobacter illness, however in older children and adults the occurrence is significantly lower, but the incidence is still many times higher than that observed for developed nations (FAO/WHO, in press).

### 2.2.3 Dose-response

Volunteer human feeding studies suggested that host susceptibility, foodstuff, virulence and colonization potential of strain, and possibly immunity will influence the infective dose (Black *et al*., 1988; Coleman and Marks, 1998). Moreover, human feeding studies using milk as the vehicle suggested large variations in infective doses for healthy people (Black *et al*., 1988); only 11 out of 50 infected volunteers developed diarrhoea. It appears that clinical disease and infection may not be fully correlated (Teunis *et al*., 2005).

The *Campylobacter* infectious dose is thought to be very low (<500 bacterial cells) (ACMSF, 2005). Moreover, the infective dose appears to be particularly low for children. From the few data available from outbreaks, it has been concluded that 100 cfu or levels of 10 cells of *C. jejuni* per 100 ml in contaminated milk was sufficient to cause disease in children (Riordan *et al*., 1993; Teunis *et al*., 2005).

An overview of reported dose-response models can be found in the risk assessment by FAO and WHO (in press).

### 2.2.4 Annual incidence in humans including differences between age and sex

The reported incidence of *Campylobacter* infections has markedly increased in many developed countries since the 1980s and 1990s (Figure 2.1) (FAO/WHO, in press). In some countries, there has been a steady increase during the last decades. In several countries such as Australia, Denmark, Finland, Ireland, New Zealand, Norway,
Sweden, the Netherlands, and the UK the (reported) incidence rate has exceeded that of *Salmonella* (EFSA, 2006; FAO/WHO, in press).

In a number of countries, the incidence rate has declined the last years probably due to interventions in the poultry production, e.g. in Denmark, Norway, the Netherlands, and the USA (FAO/WHO, in press). In Iceland, a significant decrease in the number of human cases from 1999 to 2000 was attributed to the fact that several interventions and mitigation strategies were introduced both in the primary production and the processing of poultry during this period (Stern *et al.* 2003).

![Figure 2.1. The number of registered human cases per 100,000 population caused by *Campylobacter jejuni/coli*. (FAO/WHO, in press).](image)

The reported incidence rates of *Campylobacter* infections vary widely among countries. In 2004, rates ranged from 12.8 cases per 100,000 inhabitants in USA to 299.1 in New Zealand. Some of the variation may partly be explained by differences in surveillance systems, diagnostic methods and means of reporting, so caution should be used when drawing inferences from the data (FAO/WHO, in press). The burden of human *Campylobacter* infections is not known in many developing countries, as national surveillance does not exist (FAO/WHO, in press). Estimates of incidence in developing countries are from laboratory-based surveillance studies, giving estimates for the general population from 5 to 20% (Coker *et al.*, 2002).

*Campylobacter* can affect any age group but is most often isolated from infants (< 1 year) and young (twenties) adults, with the incidence higher in males (up to 45 years of age) (ESR, 2006). In developing countries, available data suggest an even higher incidence rate of campylobacteriosis in children (FAO/WHO, in press). Case-control community-based studies have provided estimates of 40,000 to 60,000 per 100,000 population for children below 5 years of age, making campylobacteriosis a paediatric disease in developing countries (Coker *et al.*, 2002). Campylobacteriosis contributes, for example, significantly to malnutrition, as campylobacteriosis is particularly acute during the weaning period (WHO, 2000a).
2.2.5 Outcome of exposure

2.2.5.1 Severity of clinical manifestations (e.g., case-fatality rate, rate of hospitalisation)

Approximately 5-13% of people who develop *Campylobacter* enteritis are admitted to hospital (Skirrow and Blaser, 2000; CDC FoodNet, 2002). A fatal outcome of infection with *Campylobacter* spp. is rare and is usually confined to very young or elderly patients or the immuno-compromised suffering from an invasive infection (WHO, 2000b). The proportion of such vulnerable individuals is however increasing in several parts of the world, e.g. USA and the EU (EFSA, 2005; Gerba *et al*., 1996).

FAO/WHO (in press) reports studies from England and Wales, Denmark, USA and New Zealand, where the case-fatality rates ranged from 0.004% to 0.02%.

2.2.5.2 Nature and frequency of long-term complications

Campylobacteriosis has been associated with chronic sequelae that include reactive arthritis, inflammation of the liver and kidney, and Guillain-Barré syndrome (GBS).

Reactive arthritis (incomplete Reiters Syndrome) is a sterile post infectious process, which may affect multiple joints, particularly the knee joint. Pain and incapacitation can last for months or become chronic. Reactive arthritis has been estimated to occur in approximately 1% of patients with campylobacteriosis (FAO/WHO, in press).

GBS is a rare but serious paralytic condition, a demyelating disorder of the peripheral nervous system resulting in weakness, usually symmetrical, of the limbs, weakness of the respiratory muscles and loss of reflexes (areflexia). Estimates suggest that approximately one in every 1000 reported cases of campylobacteriosis leads to Guillain-Barré syndrome (EFSA, 2005; FAO/WHO, in press). Although most GBS patients recover (about 70%), chronic complications and death may occur (Blaser *et al*., 1997). Preceding *Campylobacter* infection appears to be associated with more severe neurological symptoms, slower recovery, and poorer outcome from GBS after one year (Rees *et al*., 1995).

More recently, primary infection with *C. jejuni* has been associated with immunoproliferative small intestinal disease (also known as alpha chain disease), which is a form of lymphoma that arises in small intestinal mucosa-associated lymphoid tissue (Lecuit *et al*., 2004).

2.2.6 Availability and nature of treatment

In the treatment of human campylobacteriosis, fluid replacement is of primary importance. Antimicrobial treatment is usually not indicated for enteritis of moderate severity (EFSA, 2005). In patients who have moderate-to-severe dysentery (diarrhoea with blood), who are elderly, who are presumed to be bacteraemic with chills and systemic symptoms, or who are at increased risk of complications such as immuno-compromised patients, patients with underlying disease, or pregnant women, antimicrobial treatment may be beneficial (Pigrau *et al*., 1997). *Campylobacter* spp. infections can be treated effectively with antimicrobials such as erythromycin, tetracyclines and fluoroquinolones.
2.2.7 Percentage of annual cases attributable to foodborne transmission

So far it has not been possible to quantify the number of *Campylobacter* cases related to specific risk factors, such as food. *Campylobacter* can be transmitted from the principal reservoirs to humans by direct contact with contaminated animals or animal carcasses or indirectly through the ingestion of contaminated food or water. The primary risk in developed countries is considered to be the food related risk factors (FAO/WHO, in press). In developing countries, several environmental sources pose risks for *Campylobacter* infections. Waterborne transmission, direct contact with animals, especially chickens, and animals in cooking areas are thought to be the major routes of human infection (FAO/WHO, in press).

The attributable fraction of risk factors has however been investigated in several cases–control studies. Friedman *et al.* (2004) found that, for people who had not travelled, the largest population attributable fraction (24%) was related to chicken prepared at a restaurant. A Danish case control study indicated that 5-8% were attributed to undercooked poultry, and 15-20% to barbequing poultry meat, beef, and pork (Neiman *et al.*, 2003). In a Dutch study, the identified routes of transmission accounted for 51% of the population attributable risk for *C. jejuni*, and chicken consumption was shown to be the main identified route, 23% (Nauta *et al.*, 2005). From these studies it seems evident that chickens contribute to a large part of the human cases, as there is overlap between subtypes and as handling and eating chicken are common risk factors (FAO/WHO, in press).

Of the 50 outbreaks of campylobacteriosis that took place in England and Wales between 1995 and 1999, 70% were foodborne (Frost *et al.*, 2002). Of these 35 outbreaks, poultry products (13 chicken and 1 duck) were the most commonly identified likely vehicles. The reasons identified as contributing to the outbreaks included cross-contamination (18 outbreaks), inadequate heat treatment (10 outbreaks), and inappropriate storage (7 outbreaks).

2.3 Epidemiology of foodborne disease

2.3.1 Implicated food and factors that influence transmission

The causes of infections with Campylobacter are not clearly understood. In the study of Infectious Intestinal Disease (IID) conducted in England in 1995, one of only two factors identified as significant in elevating the risk of campylobacteriosis was the consumption of chicken at restaurants. The other factor was travel abroad (ACMSF, 2005). Based on case-control studies carried out in the Nordic countries, the most common risk factors have been the consumption of untreated or contaminated drinking water and undercooked poultry meat (EELA, 2003).

In a 1997 case control study in New Zealand, the strongest associations were between campylobacteriosis and undercooked chicken, or consumption of chicken meat in restaurants. There was no association between meats other than poultry and campylobacteriosis (ESR, 2006). In another New Zealand study, there was an increased risk of campylobacteriosis associated with fast foods, and consumption of barbecued chicken (but not chicken cooked by other methods) (Bloomfield and Neal, 1997). An overview of the risk factors related to chicken from New Zealand case control studies can be found in table 2.1.
Table 2.1. New Zealand case control studies containing information on *Campylobacter* in chicken (ESR, 2006).

<table>
<thead>
<tr>
<th>Risk/Protective factor</th>
<th>Odds ratio (CI)</th>
<th>Reference, year</th>
</tr>
</thead>
<tbody>
<tr>
<td>Eating undercooked poultry (risk)</td>
<td>4.94 (1.03, 23.62)</td>
<td>Ikram <em>et al.</em>, 1994</td>
</tr>
<tr>
<td>Poultry eaten at a friend’s house (risk)</td>
<td>3.18 (1.0, 10.73)</td>
<td>Ikram <em>et al.</em>, 1994</td>
</tr>
<tr>
<td>Consuming fresh chicken (as opposed to frozen)</td>
<td>1.8 (0.82, 3.82)</td>
<td>Ikram <em>et al.</em>, 1994</td>
</tr>
<tr>
<td>Eating poultry at home (protective)</td>
<td>0.36 (0.14, 0.9)</td>
<td>Ikram <em>et al.</em>, 1994</td>
</tr>
<tr>
<td>Freezing fresh chicken before consuming (protective)</td>
<td>0.58 (0.18, 1.83)</td>
<td>Ikram <em>et al.</em>, 1994</td>
</tr>
<tr>
<td>Buying frozen chicken (protective)</td>
<td>0.71 (0.34, 1.31)</td>
<td>Ikram <em>et al.</em>, 1994</td>
</tr>
<tr>
<td>Recent consumption of raw and undercooked chicken (risk)</td>
<td>4.52 (2.88, 7.10)</td>
<td>Eberhart-Phillips <em>et al.</em>, 1997</td>
</tr>
<tr>
<td>Chicken eaten in restaurants (risk)</td>
<td>3.85 (2.52, 5.88)</td>
<td>Eberhart-Phillips <em>et al.</em>, 1997</td>
</tr>
<tr>
<td>Chicken purchased frozen (protective)</td>
<td>0.61 (0.48, 0.77)</td>
<td>Eberhart-Phillips <em>et al.</em>, 1997</td>
</tr>
<tr>
<td>Chicken baked or roasted (protective)</td>
<td>0.75 (0.60, 0.94)</td>
<td>Eberhart-Phillips <em>et al.</em>, 1997</td>
</tr>
</tbody>
</table>

CI = confidence interval

In England and Wales, from 1996 to 2000, campylobacteriosis caused 80 deaths of an estimated 687 deaths per year from indigenous foodborne disease. The most important cause of indigenous foodborne disease was contaminated chicken. Chicken consumption accounted for more disease, deaths, and healthcare usage than any other food type (Adak *et al.*, 2005).

There is strong evidence to suggest the most significant food source of human campylobacteriosis is chicken, and a reduction of the levels of *Campylobacter* on chicken would be expected to lead to a reduction in the number of human *Campylobacter* cases (ACMSF, 2005). This has also been demonstrated in countries where interventions have been implemented in the broiler production or where poultry has been withdrawn from the market. In Belgium after the dioxin crisis in 1999, when all poultry meat and eggs were withdrawn from the market, campylobacteriosis cases were reduced by 40% (Vellinga and Van Lock, 2002). Another example is in Iceland, where introduction of fresh poultry meat on the market in the 1990s was followed by a dramatic increase in the incidence of human campylobacteriosis. By introducing strict control measures during 2000 including monitoring of all flocks and freezing of contaminated carcasses, the annual human incidence was reduced by 70%, which documents that poultry was a major determinant for human campylobacteriosis on Iceland in the late 1990s (Stern *et al.*, 2003).

There is also strong epidemiological evidence confirming the transmission of *Campylobacter* from raw poultry meat or other raw meat to ready-to-eat food products such as salad or fruits via cross-contamination in retail and domestic food preparation (ESR, 2006; FSA, 2005; Luber and Bartelt, 2007). See also section 3.2.5 Consumer handling.
2.3.2 Frequency and characteristics of foodborne sporadic cases

In many countries campylobacteriosis accounts for only a small proportion of total reported outbreaks (0.5 to 6%) (EELA, 2003; ESR, 2006). The disease is often regarded as occurring mostly in sporadic cases and not in outbreaks. It has been claimed that it is due to the fact that *Campylobacter* do not multiply in air or at room temperature, so poor food handling is less likely to result in multiplication and consequent spread of the organism. In addition, the relatively long incubation period means that outbreaks are less likely to be recognised and reported (Frost, 2001).

In Denmark, Neimann *et al.* (2003) identified the following risk factors of sporadic campylobacteriosis: consumption of undercooked poultry, consumption of red meat at a barbecue, consumption of grapes and drinking unpasteurized milk. Foreign travel was also found to be a significant risk factor.

2.3.3 Epidemiological data from outbreak investigations

Most incidents of infection with *Campylobacter* do not form part of outbreaks (ACMSF, 2005). In UK only 0.4% cases between 1995 and 1999 were outbreak-associated and of the 2,374 general outbreaks of infectious intestinal disease where an aetiological agent was identified, *Campylobacter* accounted for only 50 (2%) (Frost *et al.*, 2002). However, outbreaks do occasionally occur. Large campylobacteriosis outbreaks have often been associated with contaminated drinking water or drinking raw or contaminated milk (Friedman *et al.*, 2000; FSAI, 2002), and occasionally with contaminated poultry meat (Pearson *et al.*, 2000).

2.4 Regional and seasonal influences on the incidence of foodborne illness due to the hazard

USA and northern Europe experience a peak in notifications of campylobacteriosis during the summer months of June, July and August (ACMSF, 2005; EELA, 2003; EFSA, 2005), while peak notifications in Australia and New Zealand are observed in late Spring to early Summer (October to December) (ESR, 2006; FSANZ, 2005). In England and Wales increased campylobacteriosis was associated with increased temperature rather than the season *per se*, especially in children under 5 (Louis *et al.*, 2005).

2.5 Economic impact or burden of the disease

2.5.1 Medical, hospital costs

Although the incidence of sequelae is comparatively low in terms of disease burden measured as disease adjusted life years (DALYs), campylobacteriosis sequelae are important (EFSA, 2005). Havelaar *et al.* (2000) estimated the Dutch health burden from campylobacteriosis to be 1400 DALYs, of which acute gastroenteritis were 440 DALYs and residual symptoms of Guillain-Barré syndrome 340 DALYs, while the rest was linked to increased mortality.

An Australian study suggests that approximately 13.3% of *Campylobacter* enteritis patients are hospitalised, and remained in hospital for 3 nights per person (median) (Hall, 2003).

The economic burden due to *Campylobacter* infection is large. In the United States, the annual estimated cost is around US$4.3 billion (Buzby and Roberts, 1997) and in
the Netherlands 21 million EUR (ESR, 2006). The average cost of a case of acute *Campylobacter* infection (excluding longterm sequelae) in England in 1995 was estimated to be £315 and the total cost for foodborne *Campylobacter* infections in 2000 over £113 million (ACMSF, 2005). In Sweden, Sundström (2007) estimated the “true” number of yearly campylobacteriosis cases to 80,227 (based on 7,789 registered cases). The associated costs would then be 48 million USD and approximately 60% of the these could be attributed to production losses when employees are absent due to their own illness.

The costs associated with two waterborne campylobacteriosis outbreaks in Finland were estimated by Kangas (2004). The cost for those who did not visit a doctor but were unable to work and took medicines (79.4% of those with symptoms) were 90 EUR per case. Those who in addition visited a doctor (17.4%) costed 550 EUR and those who were hospitalized (3.2%) costed 1,965 EUR. The mean cost per case was 289 EUR.

### 2.5.2 Working days lost due to illness, etc.

An Australian study indicates that 84% of people developing *Campylobacter* enteritis miss 5 days per person (median) from work/school/recreational/holiday activities (Hall, 2003).

### 3 Food Production, processing, distribution and consumption

#### 3.1 Characteristics of the commodity that is involved and that may impact on risk management

The term ‘broiler chicken meat’ principally concerns either whole carcases, or parts of the carcase or boned out meat of the species *Gallus gallus*. The water activity ($a_w$) of poultry meat is about 0.98 to 0.99. The pH of chicken breast muscle is 5.7 to 5.9, while that of leg muscle is 6.4 to 6.7. Both poultry muscle and skin are excellent substrates for supporting the survival and growth of a wide variety of microorganisms (ICMSF, 2005).

Whole or individual parts of birds may be packaged raw for direct sale. Where the birds are portioned, they are generally cut into a number of pieces and packages, for example placed on porous food trays (open cell, expanded polystyrene) and covered with a plastic film. Most frozen poultry is packaged in plastic bags clipped at the end and then frozen in high-velocity freezers. Before freezing, poultry may be injected with various salts, flavourings and oils in order to increase the juiciness of the meat.

#### 3.2 Description of the farm to table continuum

The prevalence of *Campylobacter* sp. in food is affected by its prevalence in the production farms and in the animals. The contamination of the final product is also dependent on procedures carried out in abattoirs and during the various stages of the food production chain. The farm to table continuum could be schematically be described as below.
1. Manage grandparent flocks
2. Eggs to hatchery
3. Hatchery
4. Receive day-old chicks to Parent flocks
5. Manage parent flocks
6. Eggs to hatchery
7. Hatchery
8. Receive day-old chicks at grower sheds
9. Grow broiler chickens
10. Depopulate
11. Transport to slaughterhouse

12. Receive at slaughterhouse
13. Ante-mortem inspection
14. Slaughter
15. Dress
16. Post-mortem inspection
17. Chill carcass (air, spray or immersion)

18. Pack whole carcass or portion carcass or other (added value)
19. Chill or freeze
20. Storage

21. Transport
22. Wholesale premises
23. Transport
24. Retail premises

25. Transport
26. Consumer

3.2.1 Primary production
Broilers are commonly colonized by *Campylobacter* spp., being symptomless intestinal carriers of the organism. The reported proportion of positive flocks (the flock prevalence) within the EU varied between countries, ranging from 5% to more than 90% (EC, 2003). In Switzerland, for example, 23% of the sampled broiler farms were positive for *Campylobacter* in 2005 (Anon., 2006).

In the Netherlands as well as in Sweden, the variability of the prevalence of infection between flocks was large, while within flocks it was small (Nauta and Havelaar, in manuscript; Hansson *et al*., in press), *i.e.* flocks were often found to be either negative or positive for *Campylobacter* and in a positive flock, the majority of birds were infected. In the Swedish study, the contamination level in the surrounding environment at the farm was similar for both positive and negative flocks, which highlights the importance of biosecurity as a means to reduce the prevalence (Hansson *et al*., in press).
The risk assessment performed within the Dutch CARMA-project showed that human cases are predominantly associated with flocks with high concentrations of Campylobacter. As a consequence, the strong correlation between levels of Campylobacter on meat originating from birds reared in the same flock suggests that the scheduling of flocks with high levels of Campylobacter from fresh meat production (e.g. to the processing industry) may be an effective tool for risk reduction (Nauta and Havelaar, in manuscript).

The sources of Campylobacter is not well known. There is a debate on whether vertical transmission can occur at all (ACMSF, 2005). Callicot et al. (2006) did not find any evidence of this when studying Campylobacter isolates from grandparent flocks and its progeny in Iceland, and concluded that if occurring, does not seem to be a significant transmission route.

Keener et al. (2004) identified that horizontal transmission of Campylobacter mainly occurs through contaminated water, litter, insects, rodents, and wild birds and by farm workers via their boots. In a study in Iceland, risk factors for a flock being positive for Campylobacter spp. increased with age and flock size. Additionally, vertical ventilation systems were strongly associated with positive flocks (Barrios et al., 2006).

A qualitative estimate of the importance of risk factors resulting in an increased probability of poultry carrying Campylobacter, is shown in Table 3.1 (FSANZ, 2005).

<table>
<thead>
<tr>
<th>Biosecurity</th>
<th>Campylobacter</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vertical transmission from breeder flocks</td>
<td>Campylobacter</td>
</tr>
<tr>
<td>Positive chicks</td>
<td>Campylobacter</td>
</tr>
<tr>
<td>Previously positive flocks</td>
<td>Campylobacter</td>
</tr>
<tr>
<td>Litter/Insects</td>
<td>Campylobacter</td>
</tr>
<tr>
<td>Contaminated Feed</td>
<td>Campylobacter</td>
</tr>
<tr>
<td>Age of birds</td>
<td>Campylobacter</td>
</tr>
</tbody>
</table>

Reducing the proportion of Campylobacter infected poultry flocks can be achieved by the application of strict biosecurity measures (EFSA, 2005).

### 3.2.2 Transport of live poultry

Poultry can become contaminated by Campylobacter during transportation. Contamination can occur directly via faecal material from other birds in the flock or indirectly from transport crates contaminated with faecal material. In this way negative flocks can become externally contaminated during transport, but it is unlikely that these flocks will be colonised during transport, unless the transport time is lengthy (FSANZ, 2005). Numbers on contaminated live birds (feathers) can increase 10-fold during transport (Keener et al, 2004).

Commonly used methods for washing and disinfecting crates have been found to be inefficient and Campylobacter has been detected on washed crates (Jacobs-Reitsma
and Bolder, 1998; Slader et al., 2002; Hanson et al., 2005). Corry et al. (2002) found that the three main reasons for transport crate contamination were inadequate cleaning, resulting in residual faecal soiling; disinfectant concentration and temperature of disinfectant too low; and that contaminated recycled flume water was used to soak the crates.

3.2.3 Primary processing

Processing converts live birds into poultry carcasses and poultry meat and in doing so exposes the meat to contamination from the outside of the bird, potentially the intestinal contents of the bird and the processing environment. Processing can be divided into a number of stages. Published studies on the effect of these stages on both the level and prevalence of Campylobacter on chicken carcasses are often conflicting, indicating a large amount of variability associated with each process. Table 3.2 highlights the typical effect of processing factors on the numbers of Campylobacter on chicken carcasses. It is recognised that individual plants or companies may perform these tasks differently and to different levels of hygiene (FSANZ, 2005).

<table>
<thead>
<tr>
<th>Process stage</th>
<th>Comments</th>
<th>Reduce</th>
<th>Minimal</th>
<th>Increase</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stun/Skill</td>
<td></td>
<td></td>
<td>√</td>
<td></td>
</tr>
<tr>
<td>Scald – low temperature</td>
<td>Some reduction due to removal of Campylobacter</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Scald – high temperature</td>
<td>Reduction due to death of Campylobacter and physical removal</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>De-feathering</td>
<td>Cross-contamination</td>
<td></td>
<td></td>
<td>√</td>
</tr>
<tr>
<td>Effective Washing</td>
<td>Physical removal of bacteria</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Evisceration</td>
<td>Contamination from intestinal contents</td>
<td></td>
<td></td>
<td>√</td>
</tr>
<tr>
<td>Washing</td>
<td>Physical removal</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chilling – immersion (low organic build-up and up to 50 ppm free chlorine)</td>
<td>Some removal, cross-contamination</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chilling – immersion (insufficient free chlorine and excessive organic build-up)</td>
<td>Some removal, cross-contamination</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chilling – air</td>
<td>Death from desiccation</td>
<td></td>
<td></td>
<td>√</td>
</tr>
<tr>
<td>Portioning</td>
<td>No growth, some cross-contamination</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

The general trend is that prevalence and levels of Campylobacter contamination fall during poultry processing (Mead et al., 1995).

Together with evisceration, transportation of birds from farm to slaughter site results in an increase in both the prevalence and levels of Campylobacter contamination on birds. Both immersion chilling and air chilling have been found to have a minimal effect in reducing the prevalence of Campylobacter on poultry carcasses. Immersion chilling may physically remove some Campylobacter by washing but is offset by
cross contamination between carcasses. Unlike immersion chilling, air chilling does not have a washing effect, but avoids cross contamination between carcasses (FSANZ, 2005).

A number of processing aids have been used to reduce *Campylobacter* contamination on poultry carcasses, including hot water, chlorine, organic acids (acetic acid, lactic acid, citric acid and succinic acid), chlorine dioxide, trisodium phosphate and acidified sodium chlorite (Keener *et al*., 2004).

Mangen *et al*. (2005) estimated that the most cost-effective intervention measures for controlling *Campylobacter* in the chicken meat chain were reduction of faeces leakage in the slaughter line, decontamination of the carcass by dipping in a chemical solution, and a combination of both. Phage therapy might be another cost-effective intervention measure, depending on assumed costs/chicken. However, these interventions will not eliminate all *Campylobacter* cases attributed to chicken meat.

In a risk assessment in Denmark, it was concluded that cross-contamination from positive to negative flocks during slaughter had almost no effect on the human *Campylobacter* incidence, which indicates that implementation of logistic slaughter would only have a minor influence on the risk (Rosenquist *et al*., 2003).

### 3.2.4 Secondary processing, distribution and retail sale of product

*Campylobacter* does not grow in the presence of air, therefore temperature and time of transport and storage have negligible effect on the growth of Campylobacter. Frozen storage, however, leads to a reduction of *Campylobacter* levels (FSANZ, 2005).

Over a seven month study period in South Wales, New Zealand, raw chicken samples (whole, breast with skin and pieces) were purchased from supermarkets and butchers shops. *Campylobacter* was present in 75% (n = 175) of supermarket (often pre-packaged) chicken and 59% (n = 125) of butcher’s shop chicken, which was often loose and packaged at point of sale. Overall, whole chickens were most frequently positive, followed by breast meat and then chicken pieces. Differences in handling practices were observed and on visual assessment of the pre-packaged poultry from supermarkets, 90% contained trapped surface moisture on the inside of the packaging. This micro-environment may be conducive to the survival of the organism (Harrison *et al*., 2001).

Raw poultry is frequently contaminated. A prevalence of 89% in retail minced/diced chicken samples in New Zealand has been demonstrated, (Wong *et al*., 2006) while retail cooked chicken is rarely contaminated (0.07% based on a 1995 New Zealand survey).

In the UK, Campylobacter were isolated from 68% of retail chicken (FSA, 2005). A high level of Campylobacter was enumerated from chicken skin (mean of $7.4 \times 10^4$ cfu g$^{-1}$ of skin).

In studies in Germany 100 fresh retail chicken breast fillets (skinless and deboned) were analysed by means of a rinse sample for surface and 55 fillets for internal pathogen contamination using 10 g meat and a most probable number technique.
Prevalence was 87% on the surface and 20% in the deep tissue. The mean number of Campylobacter on the surface of the fillets was 1903 CFU, with a median of 537 CFU and a maximum of 38905 CFU. Campylobacter counts inside the tissue were <1 CFU/g meat. In addition, the influence of the type of package on the occurrence of the pathogen was investigated. Data provide an indication of less favourable conditions for survival of the pathogen on chicken meat packed under a modified atmosphere of carbon dioxide in nitrogen, in comparison with ambient air or vacuumed packages. (Luber and Bartelt, 2007).

There have also been reports of frequent Campylobacter contamination on the external packaging of chicken. In UK, contamination levels of 3-8% have been found (Bolton et al., 1999; FSA, 2005; Health Protection Agency, 2004).

A survey of 300 retail packs of fresh chilled poultry products in New Zealand showed that 72 (24%) packs were externally contaminated with C. jejuni (Wong et al., 2004). Offal samples had the highest rate of external contamination (52%) followed by whole chickens (34%) and chicken portions (14.5%). Of the 250 packs of whole or portioned chicken meat sampled, 21 were positive but with low C. jejuni counts of <6 MPN/pack, 22 packs recorded counts in the range of 6-190 MPN/pack, and 3 samples recorded 480-2200 MPN/pack.

The high prevalence of contamination in raw retail chicken and to a lesser extent on the exterior of the packaging introduces the risk of cross contamination during purchase, transport and handling in the service industries and domestic settings (ESR, 2006).

3.2.5 Consumer handling

Although considered thermotolerant, Campylobacter are sensitive to temperatures above their optimal growth range and are readily inactivated by pasteurisation treatment and domestic cooking process. For example, cooking or heating at 55-60 °C for several minutes readily destroys Campylobacter (ICMSF, 1996). The D value for C. jejuni at 50 °C is 0.88-1.63 minutes (Forsythe, 2000).

However, considering the high prevalence of Campylobacter contamination in raw chicken and on the exterior of the packaging, there is a significant risk for cross-contamination in domestic or industrial kitchens. Cross-contamination in the home can occur either directly to other ready-to-eat foods or indirectly via food contact surfaces, dish-cloths, hands etc. In German experiments it was found that the mean transfer rates of Campylobacter from contaminated chicken legs and filets to hands were 3-4% (Luber et al., 2006). The transfer from legs to the plate (0.3%) was significantly smaller than that to cutting boards and knife (1.1%). Average transfer rates from hands or kitchen utensils to ready-to-eat foods ranged from 2.9 to 27.5%.

In the UK, studies have been made on Campylobacter contamination in homes and catering establishments (FSA, 2005). In homes, 8% of cooked chicken samples and 7% of ready-to-eat food items served with the meals (salad) and other cooked ingredients within the meal, were contaminated with Campylobacter. The corresponding figures for catering establishments were 6% and 8%, respectively. Assessment of contact surfaces indicated that up to 20% of various surfaces in consumers kitchens were contaminated with Campylobacter. In catering
establishments surface contamination was slightly less pronounced. CHEF tests (enzyme tests) showed that 83% of chicken was adequately cooked by consumers, and that stir-fry and barbecuing methods are most likely to result in inadequate cooking. In meals prepared by catering establishments all chickens were shown to be adequately cooked.

Given the high numbers of the pathogen on the chicken meat surface in comparison with low levels of internal contamination, cross-contamination during the preparation of contaminated chicken can be a more important pathway for consumers’ exposure to Campylobacter than the consumption of undercooked meat (Luber and Bartelt, 2007).

Brynestad et al. (in press) developed a retail product to consumer model to examine how chicken prepared in German homes exposes the consumer to Campylobacter spp. and the level of resulting illness. The model shows that reducing the Campylobacter load on the chicken may result in a greater reduction in the incidence of human illness than reducing prevalence of contaminated products.

3.3 Summary of current risk management practices

3.3.1 Primary production

In some countries control programmes in primary production have been implemented in order to reduce the prevalence of Campylobacter-positive flocks sent to slaughter (FSA 2003; 2006; Hansson et al., in press).

In the UK, the overall objective of the Campylobacter control strategy is to significantly reduce the presence of Campylobacter in UK produced chicken on retail sale (FSA, 2003; 2006). The goal is to achieve a 50% reduction by 2010. The main focus has been action on farms, activities being based around a campaign to improve biosecurity on intensive chicken farms (housed birds). The UK Industry has been closely involved in development of the strategy and in its implementation, including participation in Campylobacter research. UK Food Safety Agency stresses that industry involvement has been crucial in ensuring delivery of a practical approach to Campylobacter control.

In Sweden, a voluntary surveillance program for Campylobacter in broilers was initiated by the Swedish Poultry Meat Association in 1991. This program was revised in 2001 with a focus on reducing the annual incidence of Campylobacter-positive slaughter batches. By 2005 the annual incidence of Campylobacter-positive slaughter batches had decreased from 20% in 2002 to 13%. The strategy behind the reduction has been, and continues to be, improved biosecurity. During the study period about 40% of producers seldom delivered Campylobacter-positive batches (<10% positive batches/year) and some of producers consistently delivered Campylobacter-negative flocks to slaughter (Hansson et al., in press).

In Norway an Action Plan Against Campylobacter in Broilers has been introduced. Besides surveillance at farm, slaughterhouse level and retail, focus is on doing follow ups on identified Campylobacter-positive farms, comprising standardised consultations and the introduction of measures to reduce flock infection (NZC, 2007).

Implementation of biosecurity measures based on evaluation of their effectiveness are part of the Campylobacter risk management strategy in New Zealand (NZFSA, 2006).
3.3.2 Processing
In Iceland in 2000, carcasses from flocks which tested positive on the farms at 4 weeks of age were subsequently frozen prior to distribution (Stern et al., 2003). This gave a clear effect in the percentage of broiler carcass rinses positive for *Campylobacter*, from 62% in 1999 down to 15% in 2000. There was also a drop in domestic human cases, from a peak level of 116/100,000 in 1999 to 33/100,000 in 2000. Stern et al. (2003) however propose that several factors, such as public education, enhanced on-farm biological security measures, carcass freezing and other unidentified factors, such as variations in weather, could have contributed to the large reduction in poultry-borne campylobacteriosis.

In Denmark the efficacy of post processing heat treatment (75°C for 15 seconds) is being investigated (ACMSF, 2005). Since *Campylobacter* is thought to be particularly sensitive to freezing, work is also in hand on the effects of freezing at minus 18°C for 10 days. Current work suggests that these treatments bring about a 1.95 and 1.6 - log reduction respectively in *Campylobacter* contamination levels.

Some of the major poultry producers in New Zealand have introduced the use of leak proof packaging (ESR, 2006). This is intended to prevent chicken juice leakage and potential cross contamination from the exterior of the package. This may contribute to the overall reduction of *Campylobacter* infection in the community.

The UK Food Safety Agency are discussing the feasibility for testing and scheduling of flocks, so that negative flocks can be slaughtered before positive ones, or directing positive flocks for heat treatment, freezing, or the use of other techniques which will reduce the level of *Campylobacter*.

3.3.3 Food service and the home
Consumer education campaigns exist in many countries, however little information is available regarding their efficiency.

In Denmark, a company markets *Campylobacter*-free chickens, for which Danish consumers are prepared to pay a price premium (ACMSF, 2005). Danish legislation covering *Campylobacter*-free status requires that “the flock shall be controlled to give a 95% guarantee that less than 1% of birds are infected with Campylobacter” (tested on farms and processing plants).

4 Other Risk Profile Elements

4.1 The extent of international trade of the food commodity
In 2005 approximately 71 million tonnes of chicken meat were produced globally (http://faostat.fao.org). Out of this, 11.1 million tonnes were exported. This compares with 12.2 million tonnes of beef and 11.6 million tonnes of pig meat. The ten leading exporters of chicken meat were Brazil (26%), the USA (24%), the Netherlands (9%), China (8%), France (7%), Belgium (4%), United Kingdom (3%), Germany (3%), Thailand (3%) and Poland (2%).

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4.2 Existence of regional/international trade agreements and how they may affect the public health impact with respect to the specific hazard/commodity combination(s)

Some issues have arisen regarding Campylobacter and trade, but no existing trade agreements are known.

4.3 Public perceptions of the problem and the risk

In Denmark, a study was performed on consumers’ choices of food products. Consumers were in general willing to pay a premium for campylobacter-free chicken (Christensen et al., 2006). On average, consumers were willing to pay an extra 15 DKK (2 EUR) for a chicken being Campylobacter-free.

In New Zealand, consumer organisations give attention to the risks with Campylobacter in chicken and give advice on proper consumer handling in stores and homes in order to reduce the risk (http://www.consumer.org.nz/newsitem.asp?docid=2654&category=News&topic=Fresh%20chicken%20full%20of%20campylobacter). The Green Party in New Zealand is also engaged in demanding actions against Campylobacter. These include e.g. requirements on testing of Campylobacter before slaughter, routine sampling on poultry products and heat treatment of poultry manure before being spread on farm land (http://www.greens.org.nz/food-revolution/diseasefree.asp).

5 Available Information and Major Knowledge Gaps

5.1 Existing national MRAs on the hazard/commodity combination(s)

Australia: Scientific Assessment of the Public Health and Safety of Poultry Meat in Australia, Food Standards Australia New Zealand, November 2005.


The Netherlands: CARMA project (Campylobacter Risk Management and Assessment), 2000 – 2004 Research, Risk Model Farm to Fork, Effects of interventions on risk covering the whole food chain.


5.2 *Areas where major absences of information exist that could hamper MRM activities including, if warranted, the conduct of an MRA*

There is a lack of knowledge regarding many aspects of *Campylobacter* in broiler meat products and the relation to human campylobacteriosis. These data gaps hamper not only risk management activities but also risk assessments on *Campylobacter* in broilers. The FAO/WHO risk assessment of *Campylobacter* spp. in broiler chickens (FAO/WHO, in press) identified the following data gaps:

5.2.1 *Hazard Identification*

- National surveillance data on the number of *Campylobacter* infections per 100,000 inhabitants in developing countries.
- Survey data on the load of *Campylobacter* in chicken products in developing countries.
- The risk factors for campylobacteriosis in developed and developing countries.

5.2.2 *Exposure assessment*

5.2.2.1 *On-Farm*

- Evidence regarding the routes of *Campylobacter* infection of broilers.
- Survey data on the prevalence of flocks, both within-flock and between flocks
- Data on the probability and level of contamination of a bird during transport.
- Studies on the dynamics of within-flock transmission.

5.2.2.2 *Processing*

- Prevalence and enumeration data for poultry before and after various processing steps such as scalding, defeathering, evisceration, washing and chilling
- Prevalence and enumeration data comparing various methods of chilling – air chilling, water chilling, water chilling with chlorine, etc.
- Data describing the actual cross-contamination between positive and negative flocks and within positive flocks during the different slaughter processes.
• Prevalence and enumeration data comparing different scalding temperatures and different packaging methods.
Data on the relationship between the concentration on neck skin samples and the concentration on the whole chicken in order to calculate a conversion factor.

• Data on the microbial implications of carcass de-boning.

5.2.2.3 Post-processing and Consumer Handling
• Survey data and direct observational data on consumer practices in preparation and handling of chicken that especially detail the number of times transfer of *Campylobacter* could occur during handling and preparation.
• Research data detailing amounts of *Campylobacter* that are transferred to and from surfaces during preparation of chicken and the meal.
• Survey data and direct observational data on preparation and handling practices of chicken in restaurants and other retail establishments.
• Additional data on cooking of chicken that addresses areas of the chicken where *Campylobacter* may be protected from heat.

5.2.3 Hazard characterization
• Data on strain variability regarding virulence/pathogenicity.
• Studies on the mechanisms of infectivity, virulence/pathogenicity
• While it would be helpful to have additional human volunteer studies, the ethical issues around this type of experiment mean that this cannot be done. However non-human primate studies might be an option including:
  o Studies with other strains of *Campylobacter* jejuni ranging from $10^1$ to $10^9$ organisms.
  o Studies that focus on lower doses of the organisms from 1 to $10^3$ organisms.
• Epidemiological data available from outbreak studies that have enumerated the number of *Campylobacter* in suspected food items and includes information on attack rates, illnesses, etc.
• Additional epidemiological data on susceptible sub-populations including immuno-compromised, children under the age of 5, elderly, etc
• Additional epidemiological data on susceptibility of children under 1 year of age.
• Data describing the impact of immunity.
• Studies on the true number of human infections and other sequelae caused by *Campylobacter*, incl. GBS etc.
• Studies on the true number of human infections caused by *Campylobacter* from different sources e.g. chicken products.

Moreover, a harmonisation of laboratory methods would assist in the comparison of test results.
6 References


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Veterinary Laboratory Agency, UK. (unpublished) Assessment of, relative to other pathways, the contribution made by the food chain to the problem of quinolone resistance in microorganisms causing human infections. Centre for Epidemiology and Risk Analysis (CERA), Veterinary Laboratories Agency, UK.


