Evira Research Reports 2/2016

# Risk assessment of *Campylobacter* spp. in Finland


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to the experts regarding antimicrobial resistance

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# DESCRIPTION

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| Authors | Manuel González (Evira), Antti Mikkelä (Evira), Pirkko Tuominen (Evira), Jukka Ranta (Evira), Marjaana Hakkinen (Evira), Marja-Liisa Hänninen (UH), Ann-Katrin Llarena (UH) |
| Abstract | *Campylobacter* spp. are among the most common causes of gastrointestinal diseases in EU countries. Between four and five thousand human campylobacteriosis cases are registered each year in Finland, of which the majority are most probably acquired from abroad. The prevalence and concentration of campylobacters in foods are influenced by the whole production chain. Based on retail samples, the average annual prevalence of *Campylobacter* spp. was estimated at 5.5–11.7% (95% CI) in Finnish chicken meat and 1.8–5.9% (95% CI) in turkey meat. No *Campylobacter* spp. were detected from either domestic beef or pork, and their prevalence was estimated to be 0.0–1.2% (95% CI). The mean concentration of *Campylobacter* spp. in contaminated poultry meat was estimated to be low, and the probability of illness per one serving was thus also relatively small. Even so, the assessment implies that thousands of human cases can occur due to meat consumption annually in Finland, with the biggest proportion related to chicken meat. However, the predicted number of cases is affected by many factors with uncertainty, such as the level of cross-contamination, size of serving and total consumption. For a general overview, other campylobacters sources should also be identified and their impact on campylobacteriosis quantified. |
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# KUVAILULEHTI

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| Julkaisija | Elintarviketurvallisuusvirasto Evira |
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| Tiivistelmä | Kampylobakteerit ovat EU-maiden yleisimpiä suolistotulehduksia aiheuttavia bakteereita. Raportoitujen tapausten määrä on Suomessa vuosittain 4 000–5 000 tapausta, joista kuitenkin merkittävä osa on todennäköisesti peräisin ulkomaanmatkoilta. Kampylobakteerien esiintyvyyteen ja pitoisuuteen elintarvikkeissa vaikuttaa koko tuotantoketju. Vähittäismyyntinäytteiden perusteella arvioitu kampylobakteerien keskimääräinen esiintyvyys vuositasolla oli suomalaisessa broilerinlihassa 5,5–11,7 % (95 % CI) ja kalkkunanlihassa 1,8–5,9 % (95 % CI). Kotimaisista sian- ja naudanlihanäytteistä ei todettu kampylobakteereita, ja niissä esiintyvyyden arvioitiin olevan 0,0–1,2 % (95 % CI). Kampylobakteerien keskimääräisen pitoisuuden arvioitiin olevan lihassa matala ja sairastumistodennäköisyyden annosta kohti suhteellisen pieni. Arvion perusteella liha voi kuitenkin aiheuttaa vuosittain tuhansien ihmisten sairastumisen Suomessa, joista suuri osa liittyy broilerinlihaan. Tapausmäärän arvioon vaikuttaa kuitenkin useita epävarmuustekijöitä, kuten ristikontaminaation suuruus, annoskoko ja kulutusmäärä. Kokonaiskuvan saamiseksi myös muut kampylobakteerilähteet tulisi tunnistaa ja arvioida niiden vaikutus tartuntojen määrään. |
| Julkaisuaika | Syyskuu 2016 |
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| Resumé | *Campylobacter* spp. är av de vanligaste orsakerna till mag-tarmsjuka inom EU. Årligen rapporteras mellan fyra och fem tusen humanfall av campylobacterinfektion i Finland, varav majoriteten mest sannolikt har förvärvats utomlands. Hela produktionskedjan inverkar på förekomsten och koncentrationen av *Campylobacter* spp. i livsmedel. På basen av detaljhandels-prover tagna i Finland, uppskattades den årliga genomsnittliga förekomsten av *Campylobacter* spp. till 5,5-11,7 % (95 % CI) i finskt kycklingkött och 1,8-5,9 % (95 % CI) i kalkonkött. *Campylobacter* spp. upptäcktes inte i nötkött eller griskött, men prevalensen i detaljhandelns nöt- och griskött uppskattades till 0,0-1,2 % (95 % CI). Medelkoncentrationen av *Campylobacter* spp. i kontaminerat fjäderfäkött uppskattades vara låg, och sannolikheten för sjukdom per portion därför liten. Enligt riskvärderingen kan kött orsaka tusentals fall av campylobakterios i Finland, en stor del relaterade till kycklingkött. Det uppskattade antalet fall är förknippat med flera osäkerhetsfaktorer såsom graden av korskontaminering, portionens storhet och den konsumerade mängden. För en komplett helhetsbild borde också andra Campylobacter-källor identifieras och deras inverkan på infektionernas mängd uppskattas. |
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# 1 DEFINITIONS AND ABBREVIATIONS

**AFLP**
Amplified fragment length polymorphism, a molecular typing tool based on the PCR method.

**All-in all-out method**
A single age group of animals enter and leave a farm at the same time.

**Antimicrobial Resistance (AMR)**
The ability of a microorganism to multiply or persist in the presence of an increased level of an antimicrobial agent relative to the susceptible counterpart of the same species (CAC/GL 77-2011).

**Antimicrobial Resistance Determinant**
The genetic element(s) encoding the ability of microorganisms to withstand the effects of an antimicrobial agent. They are located either chromosomally or extra-chromosomally and may be associated with mobile genetic elements such as plasmids, integrons or transposons, thereby enabling horizontal transmission from resistant to susceptible strains (CAC/GL 77-2011).

**Bayesian inference, probabilistic inference**
Method of inferring the probable values of unknown quantities by conditioning on observed data, i.e. updating prior distributions to posterior distributions.

**Chicken**
A male or female chicken raised specifically for meat production intended to be slaughtered.

**Chicken (or turkey) slaughter batch**
A group of chickens (or turkeys) that have been raised in the same flock and which are delivered and slaughtered on one single day.

**Chicken carcass**
The body (or carcass) of a chicken collected after slaughter, dressing (plucking and removal of the offal) and chilling prior to any further processing such as freezing, cutting or packaging.

**CAC**
Codex Alimentarius Commission.

**CFU**
Colony Forming Units. CFU/g and CFU/ml represent the number of colony forming bacterial units per gram or ml of sample, respectively.

**CI**
Credible Interval. Bayesian "confidence interval" derived by taking, e.g., the 2.5 and 97.5 percentage points of a distribution for a 95% CI. Thus, the true value has a 95% probability of being within the stated 95% CI.

**Cross-contamination**
Pathogens transferred from one food to another, either by direct contact or by food handlers, contact surfaces or the air (Codex Alimentarius, 2003). Can occur at any step where the product is exposed to the environment, including processing, transportation, retail, catering and in the home. (CAC/GL 61 – 2007)

**Dose–response assessment**
Determination of the relationship between the magnitude of exposure (dose) to a chemical, biological or physical agent and the severity and/or frequency of associated adverse health effects (response). CAC/GL 30-1999. Adopted 1999. Amendments 2012, 2014.

**D-value**
Decimal reduction time, i.e. the time required at a certain temperature to inactivate 90% of the organisms being studied.

**EU**
European Union.

**Evira**
Finnish Food Safety Authority.

**Exposure assessment**
Qualitative and/or quantitative evaluation of the likely intake of biological, chemical and physical agents via food, as well as exposures from other sources if relevant. CAC/GL 30-1999. Adopted 1999. Amendments 2012, 2014.

**FAO**
The Food and Agriculture Organization of the United Nations.

**Flock**
A group of birds reared in the same department having common litter and common feeding and drinking devices.

**Fresh meat**
Meat that apart from refrigeration has not been treated for the purpose of preservation other than through protective packaging and which retains its natural characteristics. CAC/RCP 58-2005

**Hazard**
A biological, chemical or physical agent in, or condition of, food with the potential to cause an adverse health effect.

**Hazard characterization**
Qualitative and/or quantitative evaluation of the nature of the adverse health effects associated with biological, chemical and physical agents that may be present in food. CAC/GL 30-1999. Adopted 1999. Amendments 2012, 2014.

**Hazard identification**
The identification of biological, chemical and physical agents capable of causing adverse health effects and which may be present in a particular food or group of foods. CAC/GL 30-1999. Adopted 1999. Amendments 2012, 2014.

**HACCP**
Hazard Analysis and Critical Control Points.Human case
A person with campylobacteriosis

**kGy**
Kilogray; absorption of one joule of ionizing radiation by one kilogram of matter.

**MAF**
Ministry of Agriculture and Forestry.

**MC**
The Monte Carlo simulation method of generating random numbers from a defined probability distribution (i.e. from a model).

**MCMC**
Markov Chain Monte Carlo sampling. Monte Carlo simulation based on Markov chain sampling techniques.

**MIC**
Minimum Inhibitory Concentration.

**Mkg**
Million kilograms.

**MLST**
Multilocus sequence typing.

**MMM**
The Ministry of Agriculture and Forestry.

**NMKL**
Nordic Committee on Food Analysis.

**Pathogenicity**
The potential capacity of certain species / strains / lineages of microbes to cause disease in humans.

**PCR**
Polymerase chain reaction: a technology in molecular biology to amplify a single or a few copies of a piece of DNA or RNA across several orders of magnitude, generating thousands to millions of copies of a particular DNA or RNA sequence.

**PFGE**
Pulsed-field gel electrophoresis: a technique used for the separation of fragments of a genome / large (DNA) molecules, by applying to a gel matrix an electric field that periodically changes direction.

**Retail batch**
The sale of foodstuffs (here: packaged fresh meat) to ultimate consumers with the same identification information on the package label. One retail batch may consist of foodstuffs produced from one or more slaughter batches.

**Risk**
A function of the probability of an adverse health effect and the severity of that effect, consequential to a hazard(s) in food. CAC/GL 30-1999. Adopted 1999. Amendments 2012, 2014.

**Risk analysis**
A process consisting of three components: risk assessment, risk management and risk communication. CAC/GL 30-1999. Adopted 1999. Amendments 2012, 2014.

**Risk assessment**
A scientifically based process consisting of the following steps: (i) hazard identification, (ii) hazard characterization, (iii) exposure assessment, and (iv) risk characterization. CAC 26th session. Appendix IV. Working Principles for Risk Analysis for Application in the Framework of the Codex Alimentarius.

**Risk characterization**
The process of determining the qualitative and/or quantitative estimation, including attendant uncertainties, of the probability of occurrence and severity of a known or potential adverse health effect in a given population based on hazard identification, hazard characterization and exposure assessment. CAC/GL 30-1999. Adopted 1999. Amendments 2012, 2014.

**Risk communication**
The interactive exchange of information and opinions throughout the risk analysis process concerning risk, risk-related factors and risk perceptions, among risk assessors, risk managers, consumers, industry, the academic community and other interested

parties, including the explanation of risk assessment findings and the basis of risk management decisions. CAC/GL 30-1999. Adopted 1999. Amendments 2012, 2014.

**Risk Estimate**
The qualitative and/or quantitative estimation of risk resulting from risk characterization CAC/GL 30-1999. Adopted 1999. Amendments 2012, 2014.

**Risk management**
The process, distinct from risk assessment, of weighing policy alternatives, in consultation with all interested parties, considering risk assessment and other factors relevant for the health protection of consumers and for the promotion of fair trade practices, and, if needed, selecting appropriate prevention and control options. CAC/GL 30-1999. Adopted 1999. Amendments 2012, 2014.

**Risk profile**
A description of the food safety problem and its context. CAC 26$^{th}$ session. Appendix IV. Working Principles for Risk Analysis for Application in the Framework of the Codex Alimentarius.

**RTE**
Ready-to-eat. Any food that is normally eaten in its raw state or any food handled, processed, mixed, cooked or otherwise prepared into a form that is normally eaten without further listericidal steps. CAC/GL 61 – 2007.

**SCVPH**
The Scientific Committee On Veterinary Measures Relating to Public Health.

**Sensitivity analysis**
A method used to examine the behaviour of a model by measuring the variation in its outputs resulting from changes to its inputs. CAC/GL 30-1999. Adopted 1999. Amendments 2012, 2014.

**Serotype**
A group within a single species of microorganisms, such as bacteria or viruses, that shares distinctive surface structures (antigens) allowing the epidemiological classification of the organisms to the sub-species level.

**ST**
Sequence type, the allelic profile of a bacterial strain, based on the nucleotide sequences of internal fragments of usually seven housekeeping genes.

**THL**
Finnish National Institute for Health and Welfare.

**QMRA**
Quantitative microbiological risk assessment. A computational approach towards quantitative risk estimates.

**Quantitative Risk Assessment**
A risk assessment that provides numerical expressions of risk and an indication of the attendant uncertainties (stated in the 1995 Expert Consultation definition on Risk Analysis). CAC/GL 30-1999. Adopted 1999. Amendments 2012, 2014.

**Qualitative Risk Assessment**
A risk assessment based on data that, while forming an inadequate basis for numerical risk estimations, nonetheless, when conditioned by prior expert knowledge and identification of attendant uncertainties, permits risk ranking or separation into descriptive categories of risk. CAC/GL 30-1999. Adopted 1999. Amendments 2012, 2014.

**Uncertainty analysis**
A method used to estimate the uncertainty associated with model inputs, assumptions and structure/form. CAC/GL 30-1999. Adopted 1999. Amendments 2012, 2014.

**VBNC**
Viable but non-culturable.

**WHO**
World Health Organization.

**WinBUGS/OpenBUGS**
Software with model specification language for computing posterior distributions (i.e. conducting Bayesian inference) using MCMC sampling methods.

**Zoonosis**
Any disease and/or infection which is naturally transmissible directly or indirectly between animals and humans.

# 2 INTRODUCTION

Campylobacters are the most common cause of bacterial enteric infections in industrialized countries. Campylobacters may spread via different vehicles, and warm-blooded animals act as reservoir for pathogenic *Campylobacter* spp. Therefore, they play an explicit role in in the epidemiology of campylobacteriosis.

Foodborne human infections caused by campylobacters have traditionally been linked to poultry meat, although source attribution estimates remain uncertain because of the lack of concurrent information from a comprehensive collection of all potential sources and all human cases (including those not reported). Consumption of poultry other than chicken is minimal in Finland, and risk management is thus targeted at the chicken meat branch. Therefore, the Finnish Ministry of Agriculture and Forestry (MAF) pronounced the Decree on Campylobacters Control of Chickens (10/EEO/2007) based on the Food Law (23/2006), the Animal Disease Law (55/1980) and the Directive on the Monitoring of Zoonoses and Zoonotic Agents (2003/99/EC).

Because of the increasing number of reported human campylobacteriosis cases in Finland, and also because of the growing meat consumption rate, a risk assessment on campylobacteriosis due to meat consumption was conducted. The main goals of the risk assessment in this report were to investigate the prevalence and concentration of campylobacters in fresh chicken, turkey, beef and pork meat available at retail; and to assess the quantitative relative risk these meat types pose to the epidemiology of campylobacteriosis in Finland.

A Finnish risk profile of campylobacters from 2003 (Vahteristo *et al.*, 2003) concluded that further research on possible sources of infection was warranted before a full risk assessment could be produced. This included the need to investigate campylobacters in meat and other sources. Hence, a risk assessment on chicken, turkey, beef and pork meat (2012–2015) was conducted. In addition, the exposure caused by recreational waters in Finland was evaluated to some extent.

It has been acknowledged that a universally applicable general risk assessment model is not feasible due to large differences in underlying situations between countries, and also due to dissimilarities in the amount and type of available data. The availability of data largely defines which parts of the farm-to-fork chain can be modeled and in which way. The risk assessment presented in this report comprised a quantitative assessment of the prevalence and concentration of campylobacters in

fresh meat produced in Finland and sold at retail in packages as sliced or in pieces. Information on the relative risk of different meat types was assessed at the national level. In the study presented here, the focus was on assessing the magnitude of the *Campylobacter* spp. risk that consumers are exposed to from fresh domestic meat available at retail, without including previous steps of the production chain in the assessment.

This assignment was performed in co-operation between the Finnish Food Safety Authority Evira's Risk Assessment Research Unit, the Food and Feed Microbiology Research Unit and the Department of Food Hygiene and Environmental Health of the University of Helsinki. The project was financed by the Ministry of Agriculture and Forestry (MMM 2054/312/2011).

## 2.1 Risk management of campylobacters in the food chain

### 2.1.1 Finnish Campylobacter Control Programme

On the European Union level, the Directive on the monitoring of zoonoses and zoonotic agents, the so-called Zoonosis Directive (2003/99/EC), obliges the EU member states to collect relevant and, where applicable, comparable data on zoonoses, zoonotic agents, antimicrobial resistance and food-borne outbreaks.

In Finland, the regulation of campylobacters in poultry was started in 2004 by including the requirement for campylobacters examinations with certain consequences in the MAF Decision on campylobacters control of poultry (3/EEO/2004; Decree 10/EEO/2007 as amended). Finnish slaughterhouses were regulated to implement own-checking systems for *C. jejuni* and *C. coli* in chickens. The decree also requires the Finnish Food Safety Authority (Evira) to prepare a sampling plan for each slaughterhouse in a way that ensures that the demands set in the decree are fulfilled. Between 1 June and 31 October, all chicken slaughter batches must be examined according to a given scheme. During the rest of the year, the sample size is chosen using an expected prevalence of 1% in chicken slaughter batches with 1% accuracy and 95% confidence. The samples must be analysed using a method given by Evira (Evira method no. 3512/5) in approved laboratories, which are to deliver the campylobacters isolates to Evira for confirmation. Evira, for its part, is ordered to report the confirmed positive findings to the laboratory that initially examined the sample, the slaughterhouse, the veterinarian responsible for meat inspection, the owner of the flock, as well as to the official veterinarians of the owner's municipality and the Regional State Administrative Agency.

If the chicken slaughter batches of the same farm repeatedly yield *Campylobacter* spp.-positive results, the owner of the farm has to evaluate the hygiene circumstances and improve the management practices accordingly. The official veterinarian of the municipality must check the changed measures and provide advice in order to rectify the problems. If campylobacters are detected in chicken slaughter batches in two consecutive rearing cycles in a farm, the following batches from this farm are to be slaughtered at the end of the working day at the abattoir.

### 2.1.2 Microbiological Criteria

The European Commission regulates microbiological criteria for foodstuffs in (EC) No. 2073/2005. The Scientific Committee on Veterinary Measures Relating to Public Health (SCVPH), in its opinion on foodborne zoonoses of 12 April 2000, identified *Campylobacter* spp. as one of the public health priorities in Europe needing urgent consideration. The SCVPH also recommended the setting of microbiological criteria only if certain principles are fulfilled (EC discussion paper SANCO/ 1252/2001 Rev. 11). According to the Codex Alimentarius Commission, a microbiological criterion is a risk management metric that indicates the acceptability of a food, or the performance of either a process or a food safety control system following the outcome of sampling and testing for microorganisms, their toxins/metabolites or markers associated with pathogenicity or other traits at a specified point of the food chain (CAC/GL 21 - 1997). However, there are no *Campylobacter* spp.-specific criteria concerning meat or meat products in force, but during recent years the microbiological criteria for campylobacters have been studied (Nauta *et al.*, 2012; Nauta *et al.*, 2015; Ranta *et al.*, 2015; EFSA, 2011). Presently, the European Commission is preparing process hygiene criteria for campylobacters in poultry carcasses.

## 2.2 *Campylobacter* spp. risk assessments conducted in other countries

Several quantitative risk assessments for campylobacters in chicken meat have been developed in recent years to support risk managers in controlling these pathogens (Boysen *et al.*, 2013). The models deal with some or all of the consecutive stages in the chicken meat production chain: primary production, industrial processing, consumer food preparation and the dose–response relationship.

The risk assessments are not only used to assess the incidence of campylobacteriosis due to contaminated chicken meat, but also for analysis of the effect of control measures and the development of proper (microbiological) risk management metrics at different stages in the chicken meat production chain. In 2009, Nauta *et al.* wrote a review paper in which they performed a comparative overview of risk assessment models developed in the United Kingdom, Denmark, the Netherlands, Germany and New Zealand. The introduction of campylobacters to the chicken flock is generally considered to occur via horizontal transmission from the surrounding environment (Jacob-Reitsma *et al.*, 1995; Newell and Fearnley 2003), and once campylobacters are established on the farm, the within-flock prevalence may dramatically increase within a short time (Guerin *et al.*, 2007). The time point of transmission can be close to the slaughter date, increasing uncertainty in the results, when the monitoring programme is based on the testing of chicken flocks one week before slaughter. The detection is therefore sensitive to the testing time and will consequently affect the efficiency of action plans if/when they are based on testing results. Sampling methods also appear to affect on the detection of campylobacters on broiler farms (Søndergaard *et al.*, 2014; Urdaneta *et al.*, 2015).

In general, the action plans followed by the above-mentioned countries were primarily focused on the improvement of biosecurity in primary production, the scheduling of *Campylobacter* spp.-positive flocks at slaughter (Denmark), the reduction of the *Campylobacter* spp. concentration in chicken meat at slaughterhouses by freezing, and reduction of cross-contamination in domestic kitchens through consumer campaigns (Nauta *et al.*, 2008; Havelaar *et al.*, 2007).

All risk assessments compared by Nauta *et al.* 2009, found a negligible effect of logistic slaughter, i.e. the separate processing of positive and negative flocks. Moreover, all these risk assessments concluded that the most effective intervention measures aimed at reducing the *Campylobacter* spp. concentration in meat, rather than reducing the prevalence in the live poultry population. However, the expected effects can vary considerably between EU member states (EFSA, 2011), and it has not been studied how the same interventions would influence the food chain in a country such as Finland, with a low prevalence and different production conditions.

## 2.3 Finnish meat supply and consumption

Finland, similarly to many other developed countries, has undergone a prominent change from an agricultural to an industrialized (and service) society. In the 1960s, husbandry started to concentrate, while the number of operating farms diminished but their size increased. This trend has continued since Finland became a member of the EU in 1995: in 2015, the total number of farms with cattle, swine and poultry was about 25% of that in 1995, whereas the total number of these animals had remained approximately the same (Figure 1) (Tike, 2016). Within the business, the poultry sector has expanded at the expense of the cattle sector, whereas the swine sector has diminished only slightly. The number of large-scale farms has increased, while the number of small- and medium-sized farms has decreased: during 1995–2015, the average number of cattle, swine and chicken expanded roughly from 30 to 80, from 220 to 1 600, and from 4 500 to 30 000 animals per farm annually, respectively. The size of poultry flocks depends on the species: the average number of birds on a turkey farm is 6 000, while the average on a chicken farm is 30 000 birds.

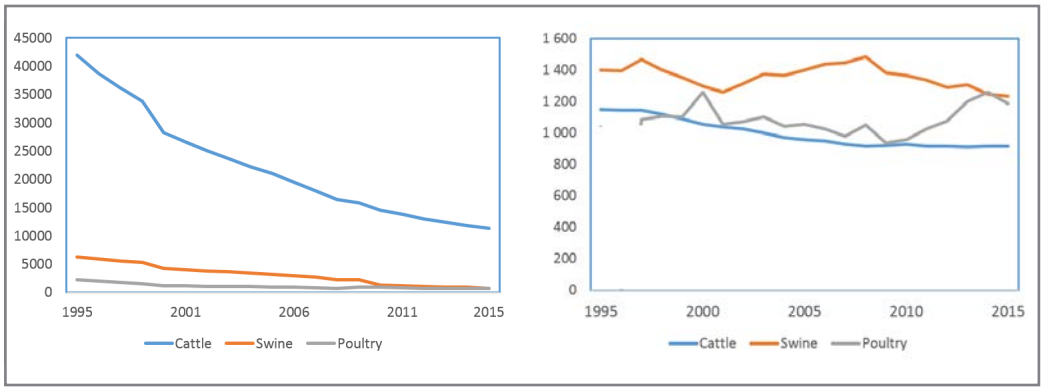


***Figure 1.*** *Number of farms with production animals (left) and number of production animals (right) in Finland during 1995–2015. Number of cattle and swine in thousands and poultry in tens of thousands. Number of poultry missing in 1996 (personal information from Tike 23.5.2016).*

**Production structure**

In Finland, the chicken (or broiler) meat chain is based on imported parents, which are raised in quarantine for about 12 weeks. The 18 week old birds are then moved to premises, where they lay eggs for hatcheries between 25–60 weeks of age. The eggs are hatched for 21 days in hatcheries, from where the chicks are moved to production farms to be raised for 32–39 days. The all-in all-out method is in practice (Siipikarjaliitto, 2015). The premises are cleaned during the empty period of at least one week.

Turkey production is based on imported parent birds. All imported parents enter the farms through quarantine. Young birds are bred for 29 weeks, and then moved to hatching sites where they lay eggs between 30–56 weeks of age. The eggs are hatched in hatcheries for about 28 days before moving the slaughter birds to production farms, from where they are transported to the slaughterhouse after a 14–18-week growing period. Cock and hen turkeys are raised separately. The all-in all-out method is in practice (Siipikarjaliitto, 2015). The premises are cleaned during the empty period of at least one week.

There are about 400 poultry farms in Finland, of which about 200 farms produce about 110 Mkg chicken meat annually (Siipikarjaliitto, 2015). About one hundred turkey farms produce less than 8 Mkg turkey meat.

In Finland there are about 11 000 cattle farms, but only about one quarter of them are identified as meat producers. Thus, most of the beef meat produced in Finland is of dairy origin, where the average number of milking cows is 26 cows per a herd. The cows are slaughtered when their productivity reduces, but calves that are not bred up to milking cows but for meat are raised either on the same farm or collected to be raised on specialized farms. However, the number of beef cattle is increasing, and in 2015 there were almost 60 000 suckler herds in Finland on about 3 000 farms. Beef cattle are grown about for 14–24 months before slaughter, when they may weigh about 500–600 kg (Tike, 2016).

Primary production in the pork chain can roughly be divided into breeding, farrowing, finishing and integrated piggeries, although in reality, pork production has nowadays split into several specialized stages. There are 10–14 piglets in a litter, and they are weaned when they are about five weeks old. When the piglets are two months old and weigh about 20 kg, they are divided into finishing and breeding pigs. The finishing pigs are slaughtered at the age of 4.5 months, when they weigh about 110 kg. There are nowadays about 700 farms with finishing pigs in Finland, the average herd size being about 600 pigs (Tike, 2016).

Total meat production in Finland increased by 25% during 1995–2014, reaching about 380 000 kt (Table 1). The increase in the meat production of turkey, chicken and pork was 519%, 173% and 12%, respectively, while beef production decreased by 14% during the same period of time. In the last ten years of this period (2005-2014), the total meat import increased by 117%, while the total export of meat decreased by 35% (Tike, 2016).

***Table 1.*** *The Finnish production, import and export of meat (kt) in 2008–2014 (Finnish Meat Trade Association 2014, Finland's Poultry Association 2014, Finnish Customs 2014. Information for 2015 not yet available).*

| | 1995 | 2000 | 2005 | 2008 | 2009 | 2010 | 2011 | 2012 | 2013 | 2014 |
|---|---|---|---|---|---|---|---|---|---|---|
| **Total meat** | | | | | | | | | | |
| Production | 306.20 | 327.82 | 375.51 | 398.97 | 382.52 | 382.59 | 387.27 | 381.97 | 387.44 | 383.24 |
| Import | - | 35.61 | 40.95 | 54.0 | 56.30 | 64.60 | 65.40 | 81.40 | 78.10 | 88.89 |
| Export | - | 67.68 | 75.55 | 73.4 | 61.7 | 54.5 | 61.1 | 52.3 | 58.2 | 50.44 |
| [1)]SSR (%) | 92.9 | 91.1 | 98.2 | 99.8 | 96.5 | 93.2 | 92.4 | 90.9 | 92.2 | 91.2 |
| **Pork** | | | | | | | | | | |
| Production | 166.31 | 172.31 | 203.35 | 216.92 | 205.65 | 203.07 | 201.75 | 192.82 | 194.49 | 186.07 |
| Import | 7.69 | 16.06 | 9.93 | 12.64 | 13.09 | 15.82 | 16.44 | 21.07 | 19.47 | 20.23 |
| Export | 6.19 | 16.06 | 36.23 | 50.86 | 41.48 | 33.06 | 37.25 | 26.77 | 30.4 | 25.09 |
| [1)]SSR (%) | 99.8 | 100.8 | 115.8 | 115.9 | 111.7 | 108.3 | 102.6 | 98.8 | 100.2 | 95.3 |
| **Beef** | | | | | | | | | | |
| Production | 95.64 | 90.16 | 84.62 | 80.27 | 81.08 | 82.13 | 82.66 | 80.37 | 80.42 | 82.32 |
| Import | 5.62 | 7.60 | 9.59 | 10.63 | 9.91 | 10.79 | 11.28 | 14.58 | 10.31 | 13.39 |
| Export | 0.70 | 0.44 | 0.94 | 0.89 | 0.84 | 1.42 | 1.12 | 0.53 | 0.99 | 1.38 |
| [1)]SSR (%) | 98.6 | 91.6 | 86.8 | 83.2 | 85.1 | 82.2 | 82.3 | 78.4 | 80.2 | 81.5 |
| **Chicken** | | | | | | | | | | |
| Production | 38.22 | 56.31 | 71.33 | 89.18 | 84.93 | 86.54 | 92.49 | 98.18 | 102.32 | 104.56 |
| Import | - | 1.22 | 3.36 | 3.67 | 3.78 | 3.88 | 3.09 | 3.16 | 3.66 | 4.56 |
| Export | - | 1.97 | 8.64 | 12.87 | 11.77 | 11.62 | 13.52 | 15.47 | 16.18 | 14.17 |
| [1)]SSR (%) | 100.6 | 93.7 | 102.3 | 111.4 | 101.1 | 98.9 | 105.1 | 106.5 | 105.5 | 106.6 |
| **Turkey** | | | | | | | | | | |
| Production | 1.18 | 5.57 | 13.78 | 9.97 | 8.63 | 8.65 | 7.93 | 8.09 | 7.36 | 7.31 |
| Import | - | 1.47 | 1.60 | 1.10 | 1.11 | 1.08 | 1.60 | 1.82 | 1.31 | 1.68 |
| Export | - | 0.16 | 2.53 | 1.17 | 1.20 | 1.52 | 1.65 | 1.59 | 1.81 | 1.52 |
| [1)]SSR (%) | 47.2 | 76.8 | 101.2 | 94.1 | 94.9 | 89.5 | 81.6 | 87.8 | 79.5 | 78.6 |

[1)] SSR = Self-sufficiency rate

## Meat consumption

In Finland, meat production and consumption followed an upward trend from 1995 until 2011, while the total meat consumption per capita in the country increased by almost 18%, reaching a maximum of 77.6 kg in 2011 (Table 2). In 2014, the total amount of meat consumed per capita in Finland was still about 77 kg. Beef and pork consumption has remained quite constant over the last 20 years. Since 1995, the consumption of chicken meat has more than doubled. Turkey meat consumption has varied over the years, but seems to have stabilized during recent years. In 2013, poultry meat consumption exceeded beef consumption for the first time.

Most of the meat consumed in Finland is of domestic origin. As seen in Table 1, self-sufficiency varies from 80% to over 100% depending on the type of meat. About 90% of poultry meat is produced in Finland, and less than 10% of the total domestic poultry production is turkey meat. Other poultry has rather an insignificant role in Finland (Siipikarjaliitto, 2015).

***Table 2.*** *Pork, beef, chicken, turkey and total meat consumption annually in Finland (kg/capita) (Tike, 2016).*

| | 1995 | 2000 | 2005 | 2008 | 2009 | 2010 | 2011 | 2012 | 2013 | 2014 | 2015 |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Pork | 33.3 | 33.0 | 33.5 | 35.3 | 34.4 | 34.9 | 36.4 | 36.0 | 35.6 | 34.6 | NA |
| Beef | 19.4 | 19.0 | 18.6 | 18.2 | 17.8 | 18.6 | 18.6 | 18.9 | 18.4 | 18.7 | NA |
| Chicken | 7.6 | 11.6 | 13.3 | 15.1 | 15.7 | 16.3 | 16.3 | 17.0 | 17.8 | 18.5 | 19.8 |
| Turkey | 0.5 | 1.4 | 2.6 | 2.0 | 1.7 | 1.8 | 1.8 | 1.7 | 1.7 | 1.6 | 1.7 |
| **Total** | **65.9** | **69.5** | **73.0** | **75.4** | **74.1** | **76.4** | **77.6** | **77.5** | **77.1** | **76.6** | **NA** |

# 3 RISK ASSESSMENT

*Campylobacter* spp. infections have increased throughout Europe, and since 2005, campylobacteriosis has been the most frequently reported zoonotic disease in humans in the EU-27. A total of 214 268 human cases of campylobacteriosis were reported during 2012, with an EU case-fatality rate of 0.03% (EFSA Journal, 2014). In Finland, during the period from 1995 to 2014, the incidence of campylobacteriosis doubled from 43/100 000 in 1995 to 90/100 000 in 2014. The true incidence of the disease is likely to be much higher than that reported due to passive surveillance, which underestimates the incidence (Olson *et al.*, 2008; Jore *et al.*, 2010). Most of the cases are acquired from abroad. The number of reported cases peaks in July–August, when cases of domestic origin account for a more significant share (THL, 2010; THL, 2014). However, a large proportion of the cases are of unknown origin.

## 3.1 Hazard identification

### 3.1.2 *Campylobacter* spp. taxonomy and general characteristics

*Campylobacter* spp. belong to the epsilonproteobacteria (Cornelius *et al.*, 2012). Three closely related genera, *Campylobacter*, *Arcobacter* and *Sulfospirillum*, are included in the family *Campylobacteraceae* (On, 2001). Bacteria belonging to the genus *Campylobacter* (from the Greek καμπυλος (*kampulos*) = curved and βαχτηρς (*baktron*) = rod) (Sebald and Véron, 1963) are non-spore-forming, oxidase-positive, non-fermenting Gram-negative rods, and a majority of *Campylobacter* spp. species multiply under microaerobic conditions (optimum: 5% $O_2$ and 10% $CO_2$), but not at atmospheric oxygen pressure. The size of the cells ranges between 0.2 to 0.8 µm wide and 0.5 to 5 µm long. Campylobacters are typically motile, with a characteristic corkscrew-like motion that is achieved by means of a single polar unsheathed flagellum at one or both ends of the cell. Some *Campylobacter* spp. species, including *C. jejuni*, adopt a coccal shape when exposed to atmospheric oxygen. Coccal forms may be seen under sub-optimal conditions, and are considered to be a degenerative form (Christensen *et al.*, 2001).

At least twelve out of the 26 so far identified Campylobacter spp. species have been associated with human illness (Table 3). However, the vast majority of infections (95%) are associated with *C. jejuni*, while *C. coli* is responsible for approximately

3–4% of human illnesses (Man, 2011). The thermophilic species *C. jejuni*, *C. coli*, *C. upsaliensis* and *C. lari* share a common feature, the ability to grow at 42 °C, and are therefore referred to as thermophilic campylobacters.

***Table 3.*** *List of valid species and subspecies in the genus Campylobacter (adapted from On 2013, updated by* http://www.bacterio.net/campylobacter.html *and Gilbert et al., 2015).*

| Taxon | Human disease association[1)] |
|---|---|
| *Campylobacter avium* | None as yet |
| *Campylobacter canadensis* | None as yet |
| *Campylobacter coli* | Gastroenteritis |
| *Campylobacter concisus* | Gastroenteritis, periodontitis |
| *Campylobacter cuniculorum* | None as yet |
| *Campylobacter curvus* | Periodontitis, gastroenteritis |
| *Campylobacter corcagiensis* | None as yet |
| *Campylobacter fetus* subsp. *fetus* | Gastroenteritis, septicemia |
| *Campylobacter fetus* subsp. *venerealis* | Septicemia |
| *Campylobacter fetus* subsp. *testudinum* | Bacteremia, diarrhoea (immunocompromised) |
| *Campylobacter gracilis* | Periodontitis |
| *Campylobacter helveticus* | Periodontitis |
| *Campylobacter hominis* | None as yet |
| *Campylobacter hyointestinalis* subsp. *hyointestinalis* | Gastroenteritis |
| *Campylobacter hyointestinalis subsp. lawsonii* | None as yet |
| *Campylobacter iguaniorum* | None as yet |
| *Campylobacter insulaenigrae* | None as yet |
| *Campylobacter jejuni* subsp. *doylei* | Septicemia, gastroenteritis |
| *Campylobacter jejuni* subsp. *jejuni* | Gastroenteritis, Guillain-Barré syndrome |
| *Campylobacter lanienae* | None as yet |
| *Campylobacter lari* subsp. *concheus* | Gastroenteritis |
| *Campylobacter lari* subsp. *lari* | Gastroenteritis, septicemia |
| *Campylobacter mucosalis* | None as yet |
| *Campylobacter peloridis* | Gastroenteritis |
| *Campylobacter rectus* | Periodontitis |
| *Campylobacter showae* | Periodontitis |
| *Campylobacter sputorum* subsp. *bubulus* | Gastroenteritis, abscesses |
| *Campylobacter sputorum* subsp. *sputorum* | |
| *Campylobacter subantarticus* | None as yet |
| *Campylobacter upsaliensis* | Gastroenteritis |
| *Campylobacter ureolyticus* | Gastroenteritis, Crohn's disease |
| *Campylobacter volucris* | None as yet |

[1)] Association with a disease is not necessarily proof of causation

### 3.1.2.1 Growth, survival and inactivation of thermophilic *Campylobacter* spp.

The thermophilic campylobacters, *C. jejuni*, *C. coli*, *C. upsaliensis* and *C. lari*, are distinguished from most other campylobacters by their high optimum growth temperature (42 °C) and their inability to grow below 30.5 °C or above 45 °C (Roberts *et al.*, 1996). Campylobacters multiply slowly, with a generation time of one hour under optimum growth conditions (Hocking, 2003).

Campylobacters can survive in cold water (4 °C) for several weeks, but in warm water only for a few days (25 °C) (Cook and Bolster, 2007; González and Hänninen, 2012). Freezing does not instantly inactivate campylobacters, but may reduce the initial concentration by 1 $\log_{10}$, and subsequently the reduction is gradual during storage (Solow *et al.*, 2003).

Thermophilic *Campylobacter* spp. species are fastidious organisms and sensitive to environmental stress (Table 4). They are not able to multiply outside the intestinal tract. Neither are they able to replicate in food or water, which can, however serve, as infection vectors (Roberts *et al.*, 1996).

***Table 4.*** *Physical limits for the growth of thermophilic campylobacters (ICMSF, 1996).*

| Parameter | Range | Growth Optimum | Growth inhibition |
|---|---|---|---|
| Temperature (°C) | 32–45 | 40–42 | <30.5 & >45 |
| pH | 4.9–9.0 | 6.5–7.5 | <4.9 & >9.0 |
| $O_2$ (%) | - | 3–5 | >15 |
| $CO_2$ (%) | - | 10 | - |
| Water activity | - | 0.997 | < 0.987 |
| NaCl (%) | - | 0.5 | >2 |

The growth of *C. jejuni* and *C. coli* is inhibited at a pH lower than 4.9 and higher than 9, whereas other *Campylobacter* spp. are inactivated at a pH lower than 4. These microorganisms are sensitive to low water activity (their growth is inhibited at $a_w$ < 0.987).

Campylobacters are sensitive to salt concentrations higher than 2% NaCl, which slowly cause their death between 5 and 10 hours. Ascorbic and lactic acids are able to inhibit the growth of these organisms (ESR, 2007).

***Table 5.*** *D-values for Campylobacter spp. (ICMSF, 1996) at temperatures of 50–60 °C.*

| Temperature (°C) | Time (minutes) |
|---|---|
| 50 | 1–6.3 |
| 55 | 0.6–2.3 |
| 60 | 0.2–0.3 |

Campylobacters are also susceptible to radiation, and a 6 $\log_{10}$ reduction is estimated when exposed to 2 kGy. *C. jejuni* and *C. coli* are more sensitive to UV radiation than, for example, *Escherichia coli* (ESR, 2007). However, meat irradiation is not authorised in Finland.

*C. jejuni* can enter a viable but non-culturable (VBNC) state under environmental stress and unfavourable growth conditions that are potentially lethal (Rollins and Colwell, 1986; Moore, 2001; Murphy *et al.*, 2006). However, understanding of the role of the VBNC state of *C. jejuni* in campylobacteriosis is contradictory.

#### 3.1.2.2 Pathogenicity of Campylobacters in humans

A number of virulence factors related to motility, toxin production, adherence and invasion, protein secretion, alteration of host cell signalling pathways, induction of host cell death, evasion of host immune defences, iron acquisition and drug/detergent resistance contribute to the pathogenesis of *C. jejuni* (Hendrixson, 2006; Malik-Kale *et al.*, 2007; Larson *et al.*, 2008). Adhesion and invasion are considered important in the pathogenesis of *C. jejuni*, damaging the colonic epithelial cells and leading to inflammation and diarrhoea. Adhesion to the epithelial surface appears to be mediated by the outer membrane protein CadF (Ziprin *et al.*, 1999) and a number of periplasmic proteins that serve as adhesins (Pei and Blaser, 1993; Pei *et al.*, 1998; Tareen *et al.*, 2013). The bacterial cell surface structures and the flagella play a role in invasion (Guerry 2007; Maue *et al.*, 2013). In addition, genes related to some metabolic functions have been reported in association with hyper-invasive *C. jejuni* strains (Javed *et al.*, 2012). However, the mechanisms are not fully understood and may differ between strains (Baig and Manning, 2014).

### 3.1.3 Methods for isolation and subtyping of thermophilic campylobacters

The presence of thermophilic campylobacters in faecal samples is usually detected by direct plating onto a suitable selective agar, which is incubated at 41.5 °C in microaerobic conditions for 48 h. However, for the detection of campylobacters in food and water, enrichment is needed, as described in the standard methods ISO 10272 (2006), ISO 17995 (2005) and NMKL 119 (2007). These methods use selective enrichment under microaerobic incubation at 41.5 °C, followed by plating on selective agars. Quantitative determination of *Campylobacter* spp. is described in ISO 10272-2 (2006) and NMKL 119 (2007). Typical colonies are examined by morphology, motility, and catalase and oxidase reactivity. Species identification is based on biochemical tests (catalase, hippurate, indoxyl acetate, susceptibility to nalidixic acid and cephalothin). Ongoing revision of the ISO 10272 standard will also include molecular methods (PCR and MALDI-TOF) for confirmation and species identification.

The special characteristics of these organisms, such as high diversity, frequent recombination with the genus, a wide host distribution and the sporadic nature of the disease, complicate the source tracing of campylobacters (Wassenaar and Newell, 2000; Dingle *et al.*, 2001; Strachan *et al.*, 2009). Subtyping beyond the species level is therefore important in collecting information on the relative importance of different

sources in human campylobacters epidemiology, ranging from outbreak investigation and source attribution studies to studies on the population genetics of pathogenic bacteria (Strachan *et al.*, 2009; Skarp *et al.*, 2016). Several typing methods have been developed and applied to study the genetic diversity among mainly *C. jejuni* and *C. coli*, aiding in tracing the sources of infection. Two of the most commonly used subtyping methods are pulsed-field electrophoresis (PFGE) and multilocus sequence typing (MLST).

PFGE is the genotyping method considered as the gold standard to trace the source of campylobacters in outbreak investigations. However, due to the wide genetic variability of these organisms and the high discriminatory power of PFGE, this method it less suitable for long-term epidemiological studies (Engberg *et al.*, 1998; Sails *et al.*, 2003).

MLST is a molecular typing technique that allows examination of the population genetics structure of campylobacters in terms of clonal complexes. MLST utilizes the genetic partial variation of the nucleotide sequence usually in seven housekeeping genes, and was developed by Dingle *et al.* (2001). Unlike PFGE, MLST has been successfully used in long-term epidemiological studies and in deciphering the population structure of campylobacters on a global scale (Dingle *et al.*, 2005; McTavish *et al.*, 2008; de Haan *et al.*, 2010a and b; On, 2013).

### 3.1.4 Epidemiology of *Campylobacter* spp.

The rate of *Campylobacter* spp. infections worldwide has been increasing, exceeding that of salmonellosis (WHO, 2013; THL 2010). Finland and other Nordic countries except Iceland show a higher *Campylobacter* spp. incidence than the average of EU Member States (Table 6), which may partly result from differences in reporting and health-care systems.

Campylobacters can colonize the intestinal tract of a variety of farm animals, including poultry, from which meat and offal can become faecally contaminated during the slaughter process (Ansari-Lari *et al.*, 2011, Keller *et al.*, 2007; Lazou *et al.*, 2014). Contaminated meat may lead to human infection due to improper cooking or due to cross-contamination of ready-to-eat foods by knives, cutting boards or hands (Luber *et al.*, 2006). Poultry meat is considered as a major source in sporadic cases of human campylobacteriosis (Wingstrand *et al.*, 2006; Wilson *et al.*, 2008; Lindmark *et al.*, 2004), whereas most outbreaks have been associated with the consumption of contaminated drinking water (Miller and Mandrell, 2005; Zacheus and Miettinen, 2011) or unpasteurized milk (Lehner *et al.*, 2000; Heuvelink *et al.*, 2009; Davis *et al.*, 2014). Other sources, such as swimming in recreational waters, travelling and contact with pets, have also appeared as risk factors for sporadic campylobacteriosis (Nordic Council of Ministers, 2001; Kapperud *et al.*, 2003; Schönberg *et al.*, 2003).

***Table 6.*** *Registered campylobacteriosis cases in 2008–2012, and incidence in 2012 in Nordic European countries and in the EU (adapted from EFSA, 2014).*

| | Confirmed cases of campylobacteriosis | | | | | Incidence /100 000 |
|---|---|---|---|---|---|---|
| **Country** | **2008** | **2009** | **2010** | **2011** | **2012** | **2012** |
| Denmark | 3 470 | 3 353 | 4 037 | 4 060 | 3 730 | 66.7 |
| Finland | 4 453 | 4 050 | 3 944 | 4 267 | 4 251 | 78.7 |
| Iceland | 98 | 74 | 55 | 123 | 60 | 18.7 |
| Norway | 2 875 | 2 848 | 2 682 | 3 005 | 2 933 | 58.8 |
| Sweden | 7 692 | 7 178 | 8 001 | 8 214 | 7 901 | 83.3 |
| EU total | 190 579 | 201 711 | 215 397 | 223 998 | 214 268 | 55.5 |

Most of the campylobacteriosis cases reported in Finland are sporadic, with no knowledge of the source. Figure 2 illustrates the seasonal distribution of reported campylobacteriosis cases in Finland from 2007 to 2014, showing a seasonal peak of infections during the summer months (Rautelin and Hänninen, 2000; Nylen *et al.*, 2002; Olson, 2008). In Finland, the prevalence of *Campylobacter* spp. in chickens also peaks at the same time (EFSA, 2010).

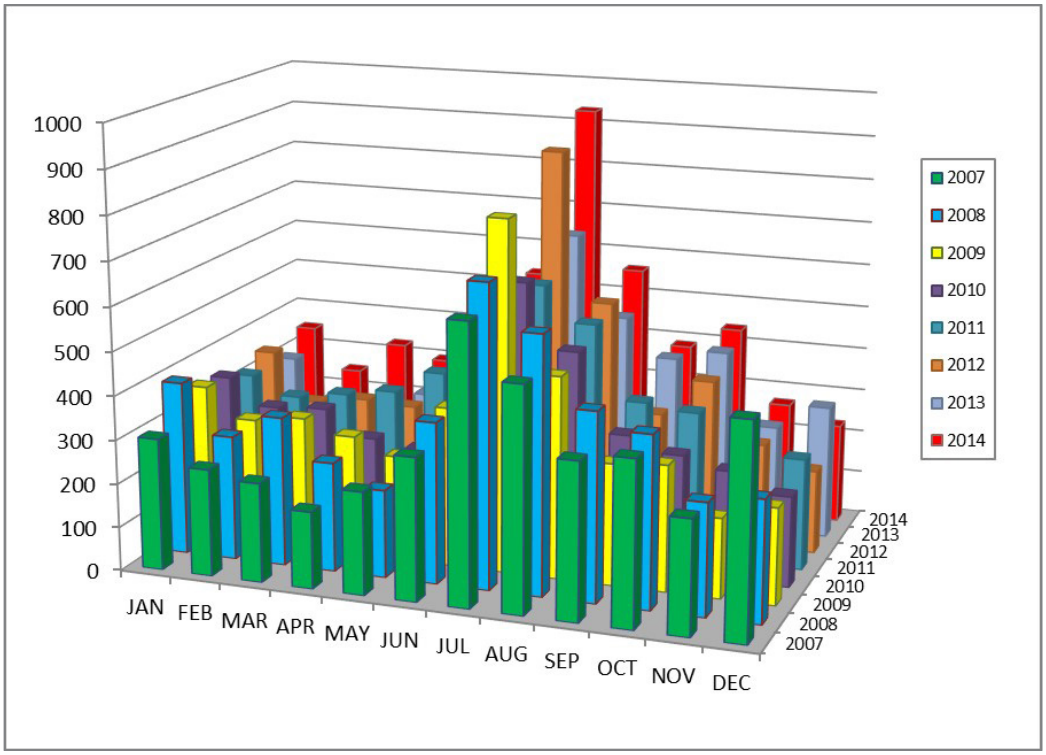


***Figure 2.*** *Number of campylobacteriosis notifications by month, 2007–2014 (THL, 2014).*

Even though campylobacteriosis affects all age groups, the incidence is highest among young adults and lowest among children (aged 5–14 years) and the elderly (≥75 years) (The National Infectious Diseases Register, 2005–2014). Reasons for the high rates of campylobacteriosis among young adults (25 to 29 years) might be increased travel and recreational activity, as well as a tendency to consume high-risk foods (Nakari *et al.*, 2010).

The reported incidence of campylobacteriosis shows a slightly higher rate in males than in females (Table 7). Some of this difference may be due to different habits of food consumption and handling, with men tending to engage in riskier practices (Ekdahl and Andersson, 2004).

***Table 7.*** *Campylobacteriosis incidence in Finland (The National Infectious Diseases Register, 2005-2014).*

| Year | Registered cases | Incidence/100 000 | Men (%) | Women (%) |
|---|---|---|---|---|
| 2005 | 4006 | 76.6 | 53.4 | 46.6 |
| 2006 | 3444 | 65.5 | 54.4 | 45.6 |
| 2007 | 4107 | 78.1 | 51.9 | 48.1 |
| 2008 | 4453 | 84.0 | 52.3 | 47.7 |
| 2009 | 4048 | 77.4 | 52.3 | 47.7 |
| 2010 | 3954 | 73.9 | 52.8 | 47.2 |
| 2011 | 4265 | 79.4 | 53.9 | 46.1 |
| 2012 | 4273 | 79.1 | 54.2 | 45.8 |
| 2013 | 4067 | 74.9 | 52.1 | 47.9 |
| 2014 | 4887 | 90.1 | 54.4 | 45.6 |

About 40-90% of the reported campylobacteriosis cases were linked with travelling abroad during 2005–2014, whereas only 10-20% were considered domestically acquired. According to the statistics, the number of cases originated abroad has slowly increased when those originated in Finland have stayed on the same level. However, around 30-80% of the cases were reported without any information on the subject increasing the uncertainty of the place of origin. *C. jejuni* dominated the analyzed cases over the years showing annual 50-85% sample prevalence. *C. coli* species was detected in 4-7% of the cases while 8-46% of the samples were not analyzed on the species level (THL, 2005–2014).

### 3.1.5 *Campylobacter* spp. outbreaks in Finland

Between 1998 and 2013, 13 waterborne and 20 foodborne campylobacteriosis outbreaks were reported in Finland (Tables 8 and 9). In Finland, the first reported waterborne outbreak occurred in the summer of 1985 (Rautelin *et al.*, 1986), and some large outbreaks have occurred due to the contamination of water supply networks in the country (Kuusi *et al.*, 2004; Kuusi *et al.*, 2005).

***Table 8.*** *Waterborne Campylobacter spp. outbreaks in Finland during 1998–2011.*

| Year | Water supply | n[1)]/N[2)] | Probable mechanism | Reference |
|---|---|---|---|---|
| 1998 | Municipal groundwater supply | 2200/15000 | Cross-connection | Kuusi *et al*. (2005) |
| | Private well | 12/17 | NA | Hatakka and Wihlman (1999) |
| 1999 | Non-community groundwater | 12/NA | Surface water runoff | Hatakka and Halonen (2000) |
| 2000 | Community groundwater | 550[3)]/5500 | Heavy rainfall | Hatakka *et al*. (2001) |
| 2001 | Community groundwater | 50[3)]/700[3)] | Lake infiltration | Hänninen *et al*. (2003) |
| 2001 | Community groundwater | 1000[3)]/18000 | Surface water runoff | Hänninen *et al*. (2003) |
| 2004 | Community groundwater | 5100 | Heavy rainfall | Pitkänen *et al*. (2008) |
| | Private well | 7/14 | Heavy rainfall | Niskanen *et al*. (2005) |
| | Private well | 2/6 | Surface water runoff | Niskanen *et al*. (2005) |
| 2007 | Community groundwater | 9500 | Cross-connection | Laine *et al*. (2010) |
| 2011 | Non-community groundwater | 24 | Contamination | Pihlajasaari *et al*. (2016) |

[1)] n=number of infected persons reported

[2)] N=number of exposed persons

[3)] Approximated

***Table 9.*** *Foodborne Campylobacter spp. outbreaks in Finland since 1998 (The National Zoonosis Centre, 2012).*

| Year | Food | n[1)] | N[2)] | Probable mechanism[4)] | Reference |
|---|---|---|---|---|---|
| 1998 | Chicken salad | 14 | NA | 2 | Hatakka and Wihlman (1999) |
| 1999 | Turkey fillet | 15 | 500 | 1,4 | Hatakka and Halonen (2000) |
| | Raw milk | 5 | 10 | 10 | Hatakka and Halonen (2000) |
| 2000 | Not identified | 5 | NA | 12 | Hatakka *et al.* (2001) |
| 2002 | Chicken salad | 5 | 30[3)] | 1,7,9 | Hatakka *et al.* (2003) |
| | Strawberries | 6 | 25 | 12 | Hatakka *et al.* (2003) |
| 2005 | Not identified | 23 | NA | 12 | Niskanen *et al.* (2006) |
| | Not identified | 14 | NA | 12 | Niskanen *et al.* (2006) |
| 2006 | Not identified | 28 | 421 | 12 | Niskanen *et al.* (2007) |
| 2007 | Salad from garden | 7 | 7 | 12 | Niskanen *et al.* (2010a) |
| | Unpasteurized milk | 4 | 6 | 1 | Niskanen *et al.* (2010a) |
| 2008 | Turkey-vegetable soup | 68 | 500 | 1,2 | Niskanen *et al.* (2010b) |
| | Duck meat | 2 | 2 | 1,2,4 | Niskanen *et al.* (2010b) |

| Year | Food | n[1)] | N[2)] | Probable mechanism[4)] | Reference |
|---|---|---|---|---|---|
| 2010 | Pizza | 5 | NA | 2 | Pihlajasaari *et al.* (2016) |
| | Pigeon meat | 3 | 4 | NA | Pihlajasaari *et al.* (2016) |
| | Not identified | 5 | NA | NA | Pihlajasaari *et al.* (2016) |
| 2012 | Not identified | 22 | 32 | 12 | Pihlajasaari *et al.* (2016) |
| | Duck meat | 3 | 4 | 1,2 | Pihlajasaari *et al.* (2016) |
| | Raw milk | 18 | 62 | 1,4 | Pihlajasaari *et al.* (2016) |
| | Raw milk | 4 | 4 | 1,4 | Pihlajasaari *et al.* (2016) |

[1)] Number of infected persons reported

[2)] Number of exposed persons

[3)] Approximated

[4)] Probable mechanism: 1. contaminated raw material, 2. cross-contamination, 3. insufficient cooling, 4. insufficient heat-treatment, 5. insufficient washing, 6. insufficient premises, 7. faulty storage temperature, 8. faulty distribution temperature, 9. excessive storage time, 10. infected employee, 11. other factor, 12. unknown

### 3.1.6 Sources of thermophilic campylobacters

#### 3.1.6.1 Reservoirs

Campylobacters are widely distributed in the environment. The principal reservoirs are the intestinal tract of wild and domesticated birds and mammals, which are usually symptomless carriers of campylobacters. As the optimum temperature for thermophilic *Campylobacter* spp. coincides with the body temperature of birds rather than mammals, they have been well adapted to the avian gut (Newell and Wagenaar, 2000). The most frequently isolated and examined *Campylobacter* spp. from poultry is *C. jejuni*, but *C. coli* also can be isolated (Van Looveren *et al.*, 2001; Pezzotti *et al.*, 2003). The predominant species in cattle is *C. jejuni*, and that in pigs is *C. coli* (Kramer *et al.*, 2000; Pezzotti *et al.*, 2003; Hartnett *et al.*, 2002). *C. lari* has been found in chickens and seagulls, shellfish, and in fresh and sea water (Leatherbarrow *et al.*, 2007). *C. upsaliensis* is a common inhabitant of dogs and cats (Hald and Madsen, 1997; Steinhauserova *et al.*, 2000; Acke *et al.*, 2009; Andrzejewska *et al.*, 2013), which are also usually symptomless carriers of these organisms.

#### 3.1.6.2 Campylobacters in the food chain

The prevalence of *Campylobacter* spp. in poultry and chickens varies considerably between countries. Finland, Norway and Sweden report a low annual prevalence (5–20%), whereas other European countries have higher prevalences, with up to 90% of chicken flocks colonized (EFSA, 2014).

An EU-wide baseline survey was performed on *Campylobacter* spp. in chicken slaughter batches and carcasses in 2008 (EFSA, 2010). The survey provided reference values, comparable between Member States, in order to consider future microbiological risk management metrics, such as performance objectives along the chicken meat production chain. Many countries displayed a seasonal peak in flock prevalence between June and September. The shape and timing of this peak varies, with northern

European countries having much sharper summer peaks in prevalence compared to the more southern countries. On slaughter carcases, both qualitative and quantitative analyses were performed. At the EU level, the prevalence of *Campylobacter* spp. in chicken slaughter batches as determined from caecal contents was 71.2% and the prevalence of contaminated carcasses was 75.8%. The prevalence of positive slaughter batches varied between EU member states from 2% to 100%, and the prevalence of carcass contamination from 4.9 to 100%. The *Campylobacter* spp. counts in neck and breast skin were below 10 CFU/g in 46% and exceeded 10,000 CFU/g in 5.8% of all samples (EFSA, 2010). The prevalences of *C. jejuni* and *C. coli* in Finnish chicken slaughter batches (n = 411) were 3.9% (95% CI, 3.8–4) and 0 (95% CI, 0–0.9), respectively, and the prevalence of *Campylobacter* spp.-contaminated carcasses was 5.5% (95% CI, 5.4–5.5). The counts of campylobacters were below 10 CFU/g in 97.8% of Finnish chicken neck and breast skin samples.

The *Campylobacter* spp. prevalence in fresh and frozen poultry for human consumption has varied from 7% to 83% in different countries and investigations (Kramer *et al.*, 2000; Shih, 2000). The *Campylobacter* spp. prevalence in meat from other animals different from poultry is lower (Ghafir *et al.*, 2007; Llarena *et al.*, 2014). A possible cause may be the difference in slaughter procedures (Höök, 2005), added to the higher prevalence in living birds.

Because the intestines of dairy cattle are often colonized by *Campylobacter* spp. (Hakkinen *et al.*, 2007; Bianchini *et al.*, 2014), the faecal contamination of raw milk can occur due to lapses in hygiene or failures in the milking process (Schildt *et al.*, 2006). However, most milk is consumed after pasteurization, which destroys campylobacters (Humphrey *et al.*, 2007).

#### 3.1.6.3 Campylobacters in the environment

Campylobacters are subject to various environmental stresses and their survival is affected by extrinsic and intrinsic factors. Faecal contamination of various environmental sources such as soil, and especially water, plays an important role in the transmission cycle of the organism between different hosts, including human patients. Because a wide variety of hosts carry *C. jejuni* and *C. coli* and faecal contamination is common, these bacteria are commonly isolated from natural water bodies, soil and sand (Rodriguez and Araujo, 2010; Jokinen *et al.*, 2011; Hörman *et al.*, 2004). Previous studies have shown that swimming in summer in natural waters could pose a risk of acquiring campylobacteriosis, as was reported, for instance, in a Finnish case-control study (Schönberg-Norio *et al.*, 2004). Since *Campylobacter* spp. are not able to replicate outside the host in the environment, the presence of *Campylobacter* spp. suggests recent faecal contamination (Jones, 2001; Jones, 2005; Snelling *et al.*, 2005).

The key factors affecting the survival of *Campylobacter* spp. in aquatic environments include temperature, UV light and the concentrations of oxygen and nutrients (Thomas *et al.*, 1999b). The survival of *Campylobacter* spp. is favoured by a low temperature,

the absence of sunlight and by low numbers of competing microbiota. Moreover, the viability of *Campylobacter* spp. in water systems is favoured by the ability to form biofilms (Ica *et al.*, 2012) and possibly by free-living amoebae harbouring bacteria intracellularly (Axelsson-Olsson *et al.*, 2005).

The most frequently isolated *Campylobacter* spp. species from surface waters is *C. jejuni* (Thomas *et al.*, 1999a). The contamination of surface water has been associated with discharges of treated wastewater from sewage treatment plants (Bolton *et al.*, 1987), runoff after heavy rains to water supplies, grazing of cattle or sheep on pasture with free access to natural water or from wild animals (e.g. wild birds) defecating directly into water. The isolation of other thermophilic *Campylobacter* spp., such as *C. coli* and *C. lari* (Hokajärvi *et al.*, 2013), is more likely due to agricultural runoff or large flocks of waterfowl (Obiri-Danso and Jones, 1999).

Campylobacters has also been isolated from groundwater after heavy rains and flooding. Several *C. jejuni* outbreaks associated with contaminated groundwater have been reported from Finland, as well as from other countries (Guzman-Herrador *et al.*, 2015). Therefore, intensive grazing on pastures may be a concern if located close to a local groundwater source (Close *et al.*, 2008).

### ***Campylobacter jejuni*** **in swimming water**

In the summer of 2012 (June-September), a total of 50 recreational water samples were collected in three cities in Finland, from which data on human domestically acquired *Campylobacter* spp. infections were also collected. Samples from recreational swimming beaches (12 on lakes and one on a river) were collected by the local public health authorities in association with their official control activities, which are focused on larger swimming sites frequently controlled by authorities (Bathing Water Directive by the EC (2006/7/EC) and the Act on Quality and Control of Bathing Water by the Finnish Ministry of Social affairs and Health (711/2014)).

Water samples from recreational swimming sites (100 ml or 100 ml and 1.5 l) were concentrated by filtration and cultivated after enrichment onto modified charcoal cefoperazone deoxycholate agar (mCCDA) plates. *C. jejuni* isolates from the positive samples (a total of 30 strains) were targeted for MLST typing, as well as whole genome MLST (wgMLST).

A total of 21 STs were found among 30 *C. jejuni* isolates detected from swimming water (Table 10). Four of the STs were found both in human patients and swimming water (ST-45, ST-230, ST-677 and ST-945). Most of the swimming water isolates represented clonal complex CC-45 (33%) or were unassigned (43%). Overlapping STs between water and human strains were found, which indicates that recreational water can be a reservoir for campylobacteriosis, but association of the data with the time of sampling indicated that most swimming water isolates were from the middle of June to the middle of July, while human patient isolates were from a later period (between the middle of July and middle of August) (Kovanen *et al.* 2016).

***Table 10.*** *Sequence types and number of human and swimming water isolates in 2012.*

| | | Source | | | | Source | |
|---|---|---|---|---|---|---|---|
| CC | ST | Human stool[1)] | Water[2)] | CC | ST | Human stool[1)] | Water[2)] |
| 21 | 19 | 2 | | UA | 951 | 1 | |
| 21 | 50 | | 1 | UA | 1 030 | 1 | |
| 22 | 22 | | 1 | UA | 1 080 | 2 | |
| 45 | 11 | 1 | | UA | 1 607 | | 1 |
| 45 | 45 | 20 | 7 | UA | 1 286 | | 1 |
| 45 | 230 | 13 | 2 | UA | 1 294 | | 1 |
| 45 | 538 | 1 | | UA | 1 367 | 2 | |
| 45 | 2 219 | | 1 | UA | 2 068 | 1 | |
| 61 | 61 | 2 | | UA | 3 322 | | 1 |
| 283 | 267 | 20 | | UA | 4 881 | | 1 |
| 283 | 383 | 1 | | UA | 6 513 | | 1 |
| 677 | 677 | 18 | 1 | UA | 6 515 | | 1 |
| 677 | 794 | 2 | | UA | 6 516 | | 1 |
| 677 | 6 514 | | 1 | UA | 6 517 | | 2 |
| 692 | 991 | 1 | | UA | 6 518 | | 1 |
| 952 | 3 492 | 1 | | UA | 6 519 | | 2 |
| 952 | 4 582 | | 1 | UA | 6 591 | 1 | |
| 952 | 4 871 | | 1 | UA | 6 626 | 1 | |
| 952 | 5 987 | 1 | | UA | 7 007 | 1 | |
| 1 287 | 945 | 1 | 1 | | | | |
| 1 332 | 1 276 | 1 | | | | | |

[1)] 95 human isolates were included

[2)] 30 swimming water isolates were included

### 3.1.6.4 Source attribution

Source attribution can be exclusively based on typing data alone, e.g. when assessing both food sources and non-food sources (e.g. recreational waters, pets). Other data that include exposure, such as consumption data, can be accounted when assessing relative exposures between food sources. Naturally, consumption data do not apply to non-food sources. Therefore, differences also arise due to the different types of data used in the assessments (Skarp *et al.* 2016). Various source attribution approaches have been summarized by Pires (2013), and some source attribution studies for *Campylobacter* spp. have been reported by Wilson *et al.* (2008), Hakkinen *et al.* (2009), de Haan *et al.* (2010a and b), Ranta *et al.* (2011) and de Haan *et al.* (2012).

In Finland, during a seasonal peak, 34% of the human *Campylobacter* spp. isolates had an overlapping sero/PFGE genotype pattern with those of chickens (Kärenlampi *et al.*, 2003; Hakkinen *et al.*, 2009). In Denmark, the greatest overlap was found between human and chicken isolates, whereas wildlife carried different serotypes (Petersen *et al.*, 2001). A study performed in Scotland linked *C. jejuni* isolates from ruminants and *C. jejuni* isolates obtained from children younger than five years of age who were living in a rural area (Strachan *et al.*, 2009).

Source attribution studies for campylobacters have found food to be associated with about 30–80% of human cases, based on various levels of typing information (Table 11). However, considerable uncertainty remains. This is partly due to lack of systematic data collection from all relevant sources at overlapping calendar times (Smid *et al.*, 2013; Sheppard *et al.*, 2010), and changes in available typing information and in exposure patterns over time, as well as genuine differences between countries. Advanced subtyping methods provide a more accurate tool in order to identify the potential sources of infection, as well as to estimate their relative importance to the burden of campylobacteriosis. However, source attribution based on subtyping data alone does not account for differences in magnitudes in population-level exposures between sources of infection and the uncertainties in population prevalence due to small numbers of samples. The overlap of subtypes between clinical isolates and those of potential sources of infection may indicate a clonal relationship.

***Table 11.*** *Proportion of campylobacteriosis cases attributed to food in different countries.*

| Country | Proportion (%) | References |
|---|---|---|
| USA | 80 | Mead *et al*. (1999) |
| UK | 80 | Adak *et al*. (2002) |
| Netherlands | 30–80 | Van Duynhoven *et al*. (2002) |
| France | 80 | Anonymous (2004) |
| Australia | 75 | Hall *et al*. (2005) |
| Netherlands | 42 | Havelaar *et al*. (2008) |

Data on STs of temporally concurrent *Campylobacter* spp. isolates from multiple reservoirs and humans are usually rare. Earlier published Finnish data contain STs of domestic human cases from the years 1996, 1999, 2002, 2003, 2006 and 2012 (n = 513), of bovine samples from 2003 (n = 102), and of chicken samples from 1999, 2003, 2004, 2006, 2007, 2008 and 2012 (n = 331), as well as 4 turkey samples from 2003 (Llarena *et al.*, 2015; de Haan *et al.*, 2010a and b; Kärenlampi *et al.*, 2007; Kovanen *et al.*, 2014). Additionally, positive turkey meat samples collected during 2013–2014 in this project were typed for MLST (n = 28). These resulted in the following STs: 11 (n = 3), 45 (n = 4), 583 (n = 6), 670 (n = 1), 883 (n = 5), 945 (n = 3), 1 326 (n = 5) and 1 701 (n = 1).

As bacterial types evolve over time, it is difficult to compare human isolates, e.g. using PFGE, with those found in reservoirs over long and/or non-overlapping periods of time. Nevertheless, some genotypes (hence STs) in a reservoir can be persistent over long time spans, so that the MLST method is useful for this purpose. For instance, ST-45 CC and ST-45 appear to be very stable in the Finnish chicken population (Llarena *et al.*, 2015). Many of the human isolates may be a result of travelling, but the human cases subtyped in Finnish studies have all been confirmed domestic cases. The source-specific number of isolates per year or month is generally rather small, in spite of the very systematic sampling of, for instance, chickens in Finland. The other potential sources of infection remain much less frequently sampled, leading to even smaller number of isolates, which hampers the source attributions between the sources.

Published historical STs and those extracted from the new turkey isolates sampled in the quantitative microbiological risk assessment (QMRA) project presented in this report were compared. The comparisons for ST distributions are presented in Figure 3. Altogether, there were 160 different STs in total and 74 different STs in human isolates. The distribution of STs in human isolates shares some similarity with the distribution of chicken isolates, and less so with bovine and turkey. ST-45 was the most common type in human and chicken isolates, and one of the most common in turkey, but not so in bovine isolates (Figure 3).

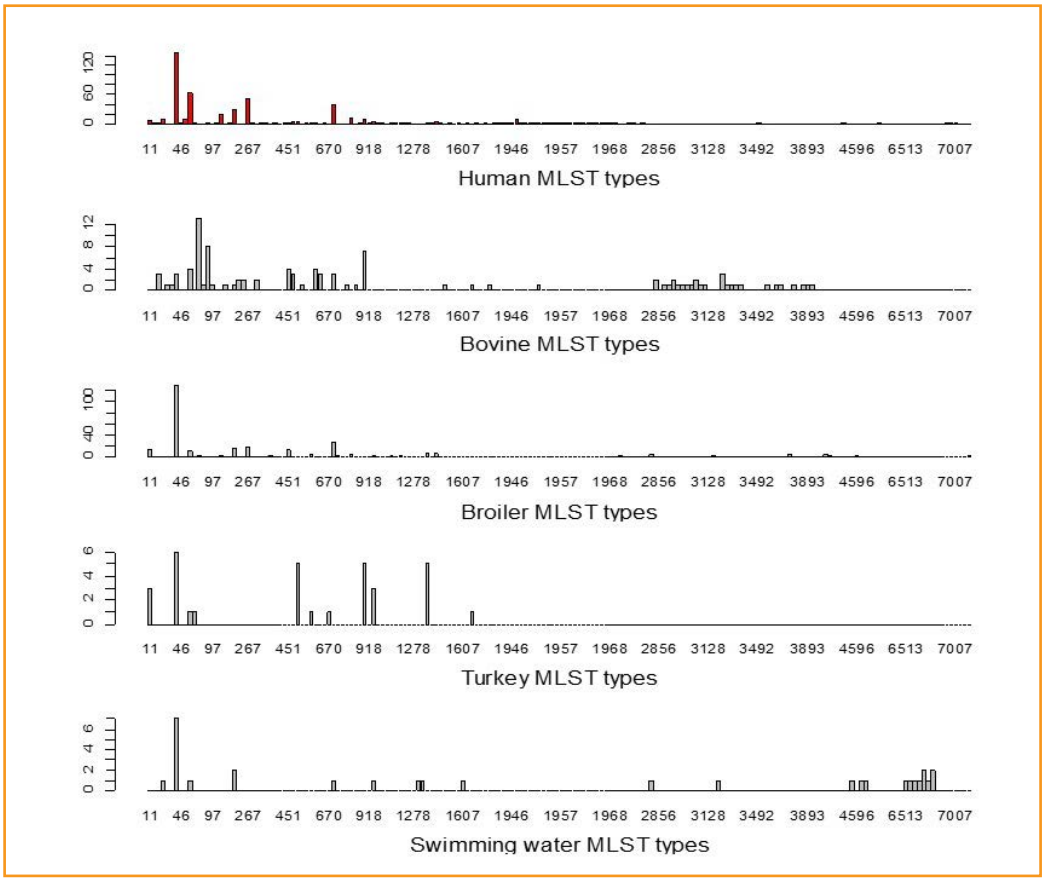


***Figure 3.*** *STs of published historical MLST isolates (humans, bovine, chicken, swimming water) and new isolates from turkey meat samples from 2013–2014.*

## 3.2 Hazard characterization

### 3.2.1 Campylobacteriosis

WHO categorizes campylobacters as a group 2 pathogen, defining it as a pathogen that causes moderate individual risk but low community risk, i.e. a pathogen that can cause human or animal disease but is unlikely to be a serious hazard to the community, livestock, the environment or workers on a farm, in a slaughterhouse or in the laboratory. The infection may cause serious symptoms, but with effective treatment and preventative measures, the adverse effects and spread of infection can be limited (WHO, 2004).

*Campylobacter* enteritis is an acute diarrhoeal disease. The incubation period, from the time of exposure to onset of symptoms, is from two to five days, with a range of one to ten days (Horn and Lake, 2013). The most common symptoms of *Campylobacter* spp. infection include fever, diarrhoea, abdominal cramps and malaise as the major symptoms. In addition, nausea, headache and muscle pain can occur. Symptoms usually last for 3–6 days. The vast majority of cases are mild, but abdominal pain can sometimes be so strong that it has been misdiagnosed as appendicitis, leading to unnecessary appendectomy (Vaidya *et al.*, 2014; Moore *et al.*, 2005). Excretion of the organism in stools last on average from 2 to 3 weeks. Most cases of campylobacteriosis are, however, self-limiting. Specific treatment is not usually necessary, except balancing dehydration caused by diarrhoea. However, in severe cases, antibiotics are needed.

Fatality resulting from campylobacteriosis is rare in developed countries, and it causes the most severe consequences in small children, the elderly or the immunosuppressed. In developing countries, where *C. jejuni* infection is hyperendemic, the decreasing case-to-infection ratio with age suggests the acquisition of immunity (Randremanana *et al.*, 2014). In 2014, the EU case fatality rate was 0.03% (EFSA 2015). In Finland, no fatal campylobacteriosis cases have been reported.

In some cases, campylobacteriosis is followed by severe sequelae, such as irritable bowel disease (Riddle *et al.*, 2012), reactive arthritis (Carter and Hudson, 2009) and Guillain-Barré syndrome (GBS) (Young and Mansfield, 2005; Jacobs *et al.*, 2008). The prevalence of reactive arthritis (ReA) has been estimated between 1% and 7% (Pope *et al.*, 2007). In a Finnish case-control study, ReA occurred in 7% of patients with *Campylobacter* spp. infection (Hannu *et al.*, 2002; Hannu *et al.*, 2004; Schönberg-Norio *et al.*, 2010).

### 3.2.2 Antimicrobial resistance

Antimicrobial resistance is the capacity of a microorganism to resist the growth inhibitory or killing activity of an antimicrobial beyond the normal susceptibility of the specific bacterial species. Microbiological (epidemiological) resistance means a reduced susceptibility of bacteria to antimicrobials above a breakpoint that is defined by the upper limit of normal susceptibility of the concerned species.

In bacterial cells, genes or mutations encoding resistance to antimicrobials may be present on the chromosome or on mobile genetic elements, or both (EFSA, 2015a). Bacteria can be resistant to antimicrobials by using several mechanisms: enzymatic degradation of antimicrobials, antimicrobial target modification, changing the bacterial cell permeability and alternative pathways to escape the activity (Verraes *et al.*, 2013).

Human campylobacteriosis usually clears of its own accord without treatment. If antimicrobial treatment is needed, the most common drugs used are macrolides, such as erythromycin, and fluoroquinolones, such as ciprofloxacin (Wieczorek and Osek, 2013). Since the 1990s, increasing resistance of campylobacters to antimicrobials, especially to fluoroquinolones, has been reported in isolates from both animals and

humans. The development of resistance to fluoroquinolones among campylobacters has occurred concurrently with the extensive use of these antimicrobials in food production animals (Luangtongkum *et al*., 2009). Fluoroquinolone resistance in campylobacters has limited their usefulness as a drug of choice in the treatment of human infection in many countries. Similarly, resistance to macrolides is increasing in several *Campylobacter* spp. isolates, particularly in *C. coli*; however, erythromycin resistance in human isolates is still relatively low. Furthermore, gentamicin also remains effective against campylobacters, although it would normally be considered only for serious *Campylobacter* spp. infections. In the EU, the occurrence of resistance to ciprofloxacin among human *Campylobacter* spp. isolates is also high. In some countries, the resistance to fluoroquinolones in *C. jejuni* and *C. coli* is at such a high level that these antimicrobials can no longer be considered an option for the treatment of human campylobacteriosis. Among *Campylobacter* spp. isolates from food production animals, the levels of resistance for ciprofloxacin, nalidixic acid and tetracyclines are also generally high. However, variation between countries in the resistance of campylobacters from humans and food production animals is large (EFSA, 2015b).

An analysis of possible relationships between the consumption of antimicrobial agents and the occurrence of antimicrobial resistance in humans and food-producing animals (EFSA 2015a) observed no associations between the consumption of fluoroquinolones in food-producing animals and the occurrence of resistance in *Campylobacter* spp. from human infections, whereas positive associations were seen in consumption of macrolides and tetracyclines in food-producing animals and the occurrence of resistance in *Campylobacter* spp. from human cases. The report states that the occurrence of resistance in *Campylobacter* spp. from humans may be influenced by resistance in *Campylobacter* spp. from food-producing animals, as undercooked chicken and contaminated ready-to-eat food have been identified as the most common sources of human campylobacteriosis in the EU (EFSA Biohaz Panel, 2010a) and campylobacters are infrequently transmitted between humans.

In Finland, antimicrobial resistance in zoonotic and indicator bacteria is monitored in the FINRES-Vet programme, as required by the Decision 2013/652/EC. In this programme, the antimicrobial susceptibilities of *Campylobacter* spp. isolates from chickens, pigs and cattle are tested.

During recent years, fluoroquinolone resistance has been moderate in bovine *C. jejuni* and porcine *C. coli*. In *C. jejuni* from chickens, fluoroquinolone resistance increased from 0% in 2013 to 25% in 2014. The reason of this rise is not known, as fluoroquinolones are not used in chicken production in Finland. An actual need for antimicrobials in chicken production is rare and treatments are used very seldom. In *C. coli* from pigs, resistance was also detected to streptomycin (37%) and erythromycin (2.3%) in 2013, and in bovine *C. jejuni* to streptomycin (13.9%) and tetracycline (2.8%) in 2012. Table 12 indicates the resistance levels of campylobacters from different animal species.

***Table 12.*** *Occurrence of resistance (R%) to selected antimicrobials in Campylobacter spp. isolates.*

| Antibiotic | Chickens (*C. jejuni*, 2014) n = 88 | Cattle (*C. jejuni*, 2012) n = 72 | Pigs (*C. coli*, 2013) n = 131 |
|---|---|---|---|
| Erythromycin | 0 | 0 | 2.3 |
| Gentamicin | 0 | 0 | 0 |
| Nalidixic acid | 25.0 | 13.9 | 19.1 |
| Ciprofloxacin | 25.0 | 13.9 | 18.3 |
| Streptomycin | 0 | 13.9 | 37.4 |
| Tetracycline | 17.0 | 2.8 | 0 |

According to the results of the FINRES-Vet programme, resistance to antimicrobials among campylobacters from food production animals in Finland is still relatively rare, but for an unknown reason, fluoroquinolone resistance in chicken *C. jejuni* has significantly increased.

### 3.2.3 Dose–response relationship

The current data on *Campylobacter* spp. virulence indicate a low infectious dose compared with many other bacterial pathogens, suggesting that even a small number of bacteria can cause human infection. In addition, the dose–response relationship depends on the type of ingested food and virulence of the *Campylobacter* spp. strain. There is a general lack of studies on the human response to known doses of *Campylobacter* spp., but existing data indicate that the number of *Campylobacter* spp. cells able to infect 50% of the exposed population may be relatively small (Robinson, 1981). *Campylobacter* spp. infection has been induced with a minimum dose of 500–800 up to 10 000 cells (Black *et al.*, 1988; Robinson, 1981), indicating that only a relatively small number of bacteria in a piece of food stored in a refrigerator may cause illness. Differences in infectious doses can be attributed to several factors, such as the type of contaminated food consumed and the health status of the exposed person. According to a study by Chrystal *et al.* (2008), counts of campylobacters detected in whole bird carcasses may vary from less than 2.6 $\log_{10}$ CFU per carcass to more than 6.8 $\log_{10}$ per carcass, making a small drop of raw meat juice sufficient to provide an infective dose for humans.

Data from human trials (Black *et al.*, 1988; Tribble *et al.*, 2010) indicate that *Campylobacter* spp. infection correlates proportionally with the ingested dose and gradually reaches saturation. However, the probability of illness may not be a monotonic function of the ingested dose. The probability of infection increased from approximately 50% at 800 cells to approximately 100% at 1 x $10^8$ cells. In contrast, the probability of illness was approximately 20% at 800 cells, rising to approximately 55% at 9 x $10^4$ cells, and declining to 0% at 1 x $10^8$ cells. In other words, infection occurs in proportion to the dose, but the development of illness does not show a direct correlation with the dose changes (Teunis *et al.*, 2005). According to a volunteer study (Black *et al.*, 1988), one should put into perspective the conclusion that the

illness risk may be small, and possibly lowest at high doses: while this may still be true for adult subjects with a history of campylobacteriosis, children may have a high risk of becoming ill, possibly due to a lack of protective immunity (Teunis *et al.*, 2005; Tribble *et al.*, 2010).

Exponential and beta-Poisson models are those that can best describe the dose–response relationship in the absence of a definitely infective dose (e.g. <500 cells), assuming that a single cell can generate infection, according to FAO / WHO (2009).

The exponential model assumes that all cells have the same probability, *r*, of generating infection in all potential hosts and the dose (i.e. number of cells) follows a Poisson distribution, with a mean of *E*(*d*) organisms per serving (FAO / WHO, 2009).

$$P(\text{inf} \mid r, E(d)) = 1 - e^{(-rE(d))} = 1 - P(\text{zero infective cells}) = 1 - \text{Poisson}(0 \mid rE(d))$$

Here, *P*(inf) corresponds to the probability of infection, *r* is the probability that a single cell can generate infection and *E*(*d*) is the average of microorganisms in each serving.

The beta-Poisson model is based on an assumption where *r* follows a beta(α, β) distribution (variability between hosts). Conditionally on a given actual number of cells, *d*, the infection probability is then

$$P(\text{inf} \mid \alpha, \beta, d) = 1 - \Gamma(\alpha + \beta)\Gamma(\beta + d) / (\Gamma(\beta)\Gamma(\alpha + \beta + d))$$

This model was used in the present quantitative risk assessment, with parameters α = 0.145, β = 7.59. By integration over possible values of d (when *d* has Poisson(E(*d*)) distribution), this would lead to an expression that is often approximated (if β>>1, β>>α) as (FAO / WHO, 2009)

$$P(\text{inf} \mid \alpha, \beta, E(d)) = 1 - \left(1 + \frac{E(d)}{\beta}\right)^{-\alpha}$$

In the equation, *P*(inf) corresponds to the probability of infection, *E*(*d*) is the mean ingested dose, and α and β are parameters of the beta distribution. To supplement the dose–response model, the relationship between infection and the development of disease also needs to be established. The data set regarding to the probability of illness given infection (*P*(ill|inf)) was obtained from the literature (Black *et al.*, 1988; Nauta *et al.*, 2007). Based on this data set, about 33% (95% CI: 23.6–42.8%) of infections led to illness.

The dose $d$ in the meal results from the serving size $w_s$ (grams) and initial bacterial concentration (CFU/g) in fresh meat, accounting cross-contamination via kitchen utensils. The cross-contamination information was adopted from earlier work (Nauta *et al.*, 2012) based on experimental results on chicken salad preparation, but the concentrations were predicted based on retail sample data (2012–2014) presented in this report.

## 3.3 Exposure assessment

### 3.3.1 Production chain

Figure 4 presents a general overview of the farm-to-fork process of meat production and the possible stages of *Campylobacter* spp. contamination. The key events in the farm-to-fork chain are suitable targets for prevention and control in order to reduce foodborne campylobacteriosis. In addition to the presented steps, there are several other stages both within and between the steps that need consideration.

#### 3.3.1.1 Primary production

The EC set the farm-to-table concept as one of its basic principles in combating food safety issues when preparing the Food Law and establishing the European Food Safety Authority (EC press release IP/00/12790, 8.11.2000). Measures at the farm level have been regarded as an effective way to reduce *Campylobacter* spp. contamination in the rest of the food chain (Lin, 2009; Hald *et al.*, 2008; Bahrndorff *et al.*, 2013). However, the efficacy of measures at the farm level can vary depending on the local conditions (EFSA Biohaz Panel, 2011). Reduction of the *Campylobacter* spp. load in poultry has been estimated to lead to a significant reduction in the incidence of human campylobacteriosis in those countries where the *Campylobacter* spp. prevalence in poultry is high (EFSA Biohaz Panel, 2011).

**Poultry**

In broiler production, vertical transmission of campylobacters from an infected breeder flock has been suggested (Cox *et al.*, 2012), but horizontal transmission from the environment is considered more likely (Jacobs-Reitsma, 1997; Callicott *et al.*, 2006). Once introduced, *Campylobacter* spp. colonization spreads rapidly throughout the flock. Up to 80–100% of the birds of one flock may become infected within 3–7 days after initial exposure (Guerin *et al.*, 2007). Hence, virtually all birds in a positive flock carry campylobacters in their intestinal tract. Due to the longer breeding period compared to chickens, turkey flocks may have a higher risk of being colonized by campylobacters than chicken flocks.

Contamination of poultry flocks by campylobacters at the farm level is multifactorial, and there is insufficient data to identify the relative importance of one factor compared with another. There are several pathways by which poultry can become contaminated with campylobacters, but the age of the birds and environmental contamination

appear to be the most important risk factors on farms. Campylobacters have been recovered from the boots, equipment and vehicles (crates, modules and forklift trucks) used in the shed. Thinning is commonly used in many European countries and may be a major risk factor for the introduction of *C. jejuni* into the chicken shed (Hansson *et al.*, 2010; Torralbo *et al.*, 2014), but it is not in use in Finland. The potential risk of infection during transport to slaughter from *Campylobacter* spp.-positive crates has also been highlighted (Hansson *et al.*, 2005).

**Other production animals**

Campylobacters are the most common zoonotic pathogens isolated from healthy cattle (Madden *et al.*, 2007; Milnes *et al.*, 2008). Cattle are usually symptomless carriers of *Campylobacter* spp. (Stanley *et al.*, 1998). The shedding of the organism can vary between individual animals, which can be persistent or intermittent shedders (Kwan *et al.*, 2008; Hakkinen and Hänninen, 2009). A Finnish survey determined the prevalence of thermophilic *Campylobacter* spp. in bovine rectal faecal samples (n = 952) from 12 slaughterhouses from January to December 2003. The total prevalence of *Campylobacter* spp. in faecal samples was 31.1%. *C. jejuni*, the most common species, was present in 19.5% of the samples (Hakkinen *et al.*, 2007). In a Finnish study on pathogens in bulk tank milk, no campylobacters were detected (Ruusunen *et al.*, 2013). However, faecal contamination of milk was observed, which suggests the possibility of *Campylobacter* spp. contamination, as these organisms are present in cattle.

The significance of *Campylobacter* spp. colonization of dairy and beef cattle relates not only to the potential for contamination of milk at the farm and the carcass during slaughter, but also surface and sub-surface water during the disposal of abattoir effluents and animal slurries to land (Hänninen *et al.*, 2003; Stanley and Jones, 2003). Furthermore, several studies have reported that the prevalence of campylobacters in cattle is highest at pasture (Hänninen *et al.*, 1998; Stanley *et al.*, 1998; Grove-White *et al.*, 2010; Duncan *et al.*, 2014).

*Campylobacter* spp. colonization of sheep has been reported in a few studies (Oporto *et al.*, 2007; Grove-White *et al.*, 2010), and the shedding of campylobacters appears to correlate with grazing (Jones et al., 1999), showing the highest rates due to stress as a result of lambing, weaning and moving to new pastures (Stanley and Jones, 2003). The dominant species in sheep seems to be *C. jejuni*, while *C. coli* is less common. Pigs, however, have been demonstrated to be an important reservoir for *C. coli* (Moore *et al.*, 2002; Nielsen *et al.*, 1997; Oporto *et al.*, 2007; Denis *et al.*, 2011), and spread the contamination in the farm environment in a similar way as described for cattle.

### 3.3.1.2 Secondary production

*Campylobacter* spp. prevalence and concentration are influenced during the different stages of the slaughter process (Figure 4). During the process, meat can be contaminated by intestinal contents from the animal itself, by employees of the food production enterprise, or from the processing environment.

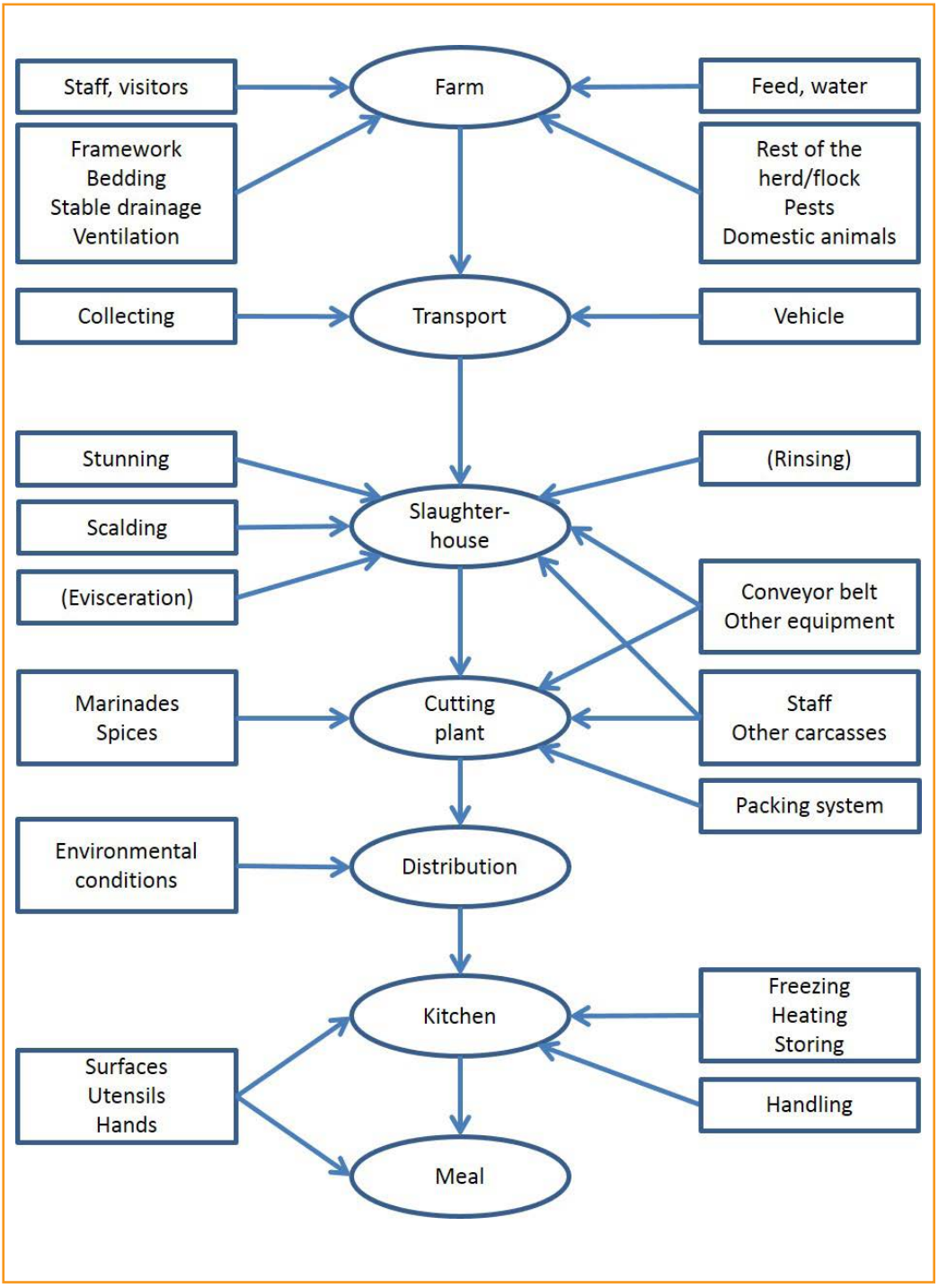


***Figure 4.*** *A simplified chart of possible routes for Campylobacter spp. infection in relation to meat production and food preparation. Cross-contamination due to handling may affect meat at every stage.*

**Poultry**

The chicken cecum is a highly developed organ located at the junction of the large and small intestines. A cecum can be colonized to a high level of up to $10^{10}$ CFU/g of caecal contents by *C. jejuni* (Achen *et al.*, 1998; Friis *et al.*, 2010). Campylobacters appear to survive the slaughter procedures, such as scalding, evisceration and immersion chilling, which allows the contamination of carcasses. The bacterial counts on carcass surfaces may be as high as approximately 7 $\log_{10}$ (Stern and Pretanik, 2006; Nauta *et al.*, 2007). However, in the EU baseline survey, the counts on Finnish chicken carcasses did not exceed 500 CFU/g, and on most of the positive carcasses were under the detection level.

The scalding tank water is an important vehicle for cross-contamination both within a slaughter batch and between batches, especially if a *Campylobacter* spp.-negative batch is slaughtered after a positive batch. Equipment that can reduce faecal leakage has been estimated to decrease the consumer risk by up to 80%, and in addition, using 2.5% lactic acid or 10% trisodium phosphate in the scalding water has shown a maximum of 1 $\log_{10}$ unit decrease in the counts (Havelaar *et al.*, 2007). Thorough washing and cooling after scalding further reduces the level of contamination (Guerin *et al.*, 2010; Rosenquist *et al.*, 2006).

During defeathering, the feather follicles in the skin are opened and allow the *Campylobacter* spp. cells move inside them, which may reduce the wash-off effect of washing of the carcass surface (Cason *et al.*, 2004). Ruptures of the viscera during evisceration may lead to extensive faecal contamination of a carcass (Rosenquist *et al.*, 2006). The FAO/WHO expert group (2003), in a risk assessment of campylobacters in chicken, concluded that reducing surface contamination after evisceration can have a significant impact in reducing the risk of exposure.

A *Campylobacter* spp.-positive slaughter batch may contaminate the equipment and spread bacteria into air, which can constitute a significant occupational risk of campylobacteriosis for people working on the slaughtering line (Wilson, 2004; Allen *et al.*, 2007).

Chemical decontamination of meat and poultry carcasses is not permitted by EU regulations at any stage of production and processing of carcasses, primal cuts and final products (Bolder, 1997; Koutsoumanis *et al.*, 2006). The situation differs markedly from that in the United States, where decontamination systems are approved by the Food Safety and Inspection Service of the U.S. Department of Agriculture (USDA) if certain criteria are fulfilled (Sofos *et al.*, 1999; Sofos, 2002; Sofos, 2005).

**Cattle and swine**

Various studies have reported *Campylobacter* spp. contamination rates of 0 to 25% in bovine carcasses at slaughter before chilling and 1.5 to 3% after chilling (Grau, 1988; Beach *et al.*, 2002; Minihan *et al.*, 2004; Bohaychuk *et al.*, 2011). In a Finnish slaughterhouse survey on campylobacters in cattle faeces and carcasses, the contamination level of carcasses before chilling was 3.5%, whereas the prevalence in cattle was 31% (Hakkinen *et al.*, 2007). Contrary to poultry carcasses, on which campylobacters are protected in folds and crevices of the skin (Corry and Atabay, 2001), drying of the carcass surface along with exposure to atmospheric oxygen

during chilling decreases the survival of campylobacters on bovine carcasses and red meat (Grau *et al*., 1988). Similarly, chilling reduces the occurrence of campylobacters on pig carcasses and the contamination rates are low (Nesbakken *et al*., 2008; Bohaychuk *et al*., 2011).

### 3.3.2 Exposure from the environment

Campylobacters are common in warm-blooded animals and birds, which spread the bacteria to environment. Campylobacters can survive in the aquatic environment for extended time periods (González and Hänninen, 2012). They are frequently detected in natural waters, such as lakes, streams and rivers (Jones, 2001). Due to the low infective dose for *Campylobacter* spp. infection, contaminated surface water may cause infection through swimming. Swimming in natural sources of waters has been identified to be an independently associated risk factor for sporadic *Campylobacter* spp. infection (Koenraad *et al*., 1997; Schönberg-Norio *et al*., 2004). However, in contrast to the study of Schönberg-Norio *et al*. (2004), a Norwegian study (Kapperud *et al*., 2003) associated swimming in the sea, lakes and swimming pools with a reduced risk of *Campylobacter* spp. infection.

It is difficult to identify infections resulting from the environment without more detailed background information on the cases, or without proper microbial typings, and more data would be needed for the traceback. Finnish environmental samples (outdoor swimming water) have revealed a variety of STs that can also be found in other sources of infection (Kovanen *et al*. 2016).

### 3.3.3 QMRA: campylobacters in fresh meat samples

#### 3.3.3.1 Materials and methods

Serving sizes ($w_s$) were obtained from the FINN DIET (2012) survey (chicken and turkey meat: mean = 100 g, sd = 73 g, pork and beef: mean = 76 g, sd = 49 g). This serving size represents the total amount of each meat type consumed per day by an adult male. Due to the lack of accurate information, it was taken that there is no difference in serving sizes between fresh meats and other meats (meat products). According to the expert opinion, from 20% to 30% of the total sale of domestic meats ($f$) is sold as fresh in Finland. Based on the total domestic amounts (see Table 1) consumed per year ($v$ = production-export), this makes approximately, $n_s = v \cdot f \cdot 1000 / w_s$ consumed domestic fresh meat servings per year. In this formula, the parameter $w_s$ denotes the size of a random portion.

The sampling of retail meat packages was conducted during 2012–2014 in the Helsinki area. In total, 1 981 samples in 754 batches were collected and analysed for campylobacters at Evira (Table 13). Fresh meat from chicken, turkey, pork and beef were chosen for this risk assessment, because they were considered as potential sources of foodborne campylobacteriosis in Finland. The meat samples that were examined for the risk assessment were chosen to be comparable with each other, and sampled shortly before the intended time of consumption. Hence, packages of

domestic chicken meat, turkey meat, beef and pork as slices or in pieces were sampled from retail shops, carried to the laboratory as a consumer would have carried them home, and stored as a consumer would have done.

***Table 13.*** *Number of samples, retail batches and positive isolates in retail meat in 2012–2014.*

| | Positive samples | Total samples | Positive batches | Total batches |
|---|---|---|---|---|
| Chicken | 76 | 608 | 31 | 226 |
| Turkey | 32 | 558 | 17 | 185 |
| Pork | 0 | 414 | 0 | 169 |
| Beef | 0 | 401 | 0 | 174 |
| **Total** | **108** | **1 981** | **48** | **754** |

All the samples were examined for *Campylobacter* spp. before their expiration date, usually within three days from the time of purchase. The examination of thermophilic *Campylobacter* spp. was performed using accredited modified NMKL 119:2007 and ISO 10272-2:2006. The collected samples were either strips (chicken, beef and pork) or fillets (turkey). The samples from turkey meat were fillets, because no unseasoned strips were available at grocery stores. The samples were allocated to the domestic meats only. The weight of one sample varied from 250 g to 500 g, and all samples were without skin. The samples were rinsed with the same amount (weight (g)/ volume (ml)) of buffered peptone water, and 25 ml of this (corresponding to 25 g of meat) was examined by enrichment. Quantification was performed from a serial dilution of the rinsing fluid.

The samples were picked up from several supermarkets in the Helsinki area. However, these samples represented food companies whose share of the total market is over 90% in Finland, and they distribute their products all over the country. The data collection was carried out during the years 2012–2014. The samples were gathered around the year in order to assess the seasonal variation in the prevalence of *Campylobacter* spp. These samples were classified according to their retail batch numbers, which indicated the origin of the meat product. One retail batch may consist of products from one or more flocks or herds. The number of samples per retail batch varied from 1 to 16, which is only a small proportion of the total size of one batch.

The sample results from chicken and turkey meat are presented in Table 14. A total of 108 samples were positive. The observed concentrations are roughly presented in Figure 5. These samples were obtained from 31 chicken meat retail batches and 17 turkey meat retail batches. No campylobacters were detected from pork (414 samples from 169 retail batches) or from beef (401 samples from 174 retail batches).

Three enumeration results were above 10 CFU/g. The highest observed concentration was 38 CFU/g in chicken meat and 0.5 CFU/g in turkey meat. The numbers of positive samples in 31 (detected) contaminated chicken meat retail batches varied from 1 to 6 and the total numbers of samples taken from these batches varied between 1 and 8. The corresponding numbers for 17 (detected) contaminated turkey meat retail batches were from 1 to 5 positive samples and from 2 to 16 samples per batch.

***Table 14.*** *Results (2012–2014) for Campylobacter spp. in chicken and turkey fresh meat at the retail level.*

| Year | Month | Chicken | | | | Turkey | | | |
|---|---|---|---|---|---|---|---|---|---|
| | | Positive samples | Ana-lysed samples | Positive batches | Batches sampled | Positive samples | Ana-lysed samples | Positive batches | Batches samp-led |
| **2012** | November | 0 | 7 | 0 | 4 | 0 | 4 | 0 | 4 |
| | December | 0 | 20 | 0 | 14 | 0 | 15 | 0 | 6 |
| | **Total 2012** | **0** | **27** | **0** | **18** | **0** | **19** | **0** | **10** |
| **2013** | January | 0 | 22 | 0 | 8 | 0 | 21 | 0 | 10 |
| | February | 6 | 38 | 2 | 17 | 0 | 30 | 0 | 10 |
| | March | 6 | 36 | 2 | 13 | 1 | 23 | 1 | 9 |
| | April | 0 | 41 | 0 | 20 | 0 | 30 | 0 | 14 |
| | May | 0 | 28 | 0 | 16 | 0 | 24 | 0 | 7 |
| | June | 0 | 20 | 0 | 9 | 0 | 18 | 0 | 6 |
| | July | 6 | 36 | 5 | 15 | 8 | 39 | 4 | 15 |
| | August | 11 | 45 | 4 | 12 | 1 | 39 | 1 | 15 |
| | September | 3 | 45 | 1 | 17 | 0 | 44 | 0 | 16 |
| | October | 4 | 45 | 1 | 18 | 5 | 41 | 1 | 13 |
| | November | 3 | 40 | 2 | 11 | 0 | 40 | 0 | 16 |
| | December | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| | **Total 2013** | **39** | **396** | **17** | **156** | **15** | **349** | **7** | **130** |
| **2014** | January | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| | February | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| | March | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| | April | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| | May | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| | June | 3 | 45 | 2 | 13 | 2 | 46 | 1 | 11 |
| | July | 24 | 80 | 8 | 24 | 7 | 84 | 4 | 19 |
| | August | 10 | 60 | 4 | 15 | 8 | 60 | 5 | 15 |
| | September | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| | October | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| | November | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| | December | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| | **Total 2014** | **37** | **185** | **14** | **52** | **17** | **190** | **10** | **45** |
| | **TOTAL (2012–2014)** | **76** | **608** | **31** | **226** | **32** | **558** | **17** | **185** |

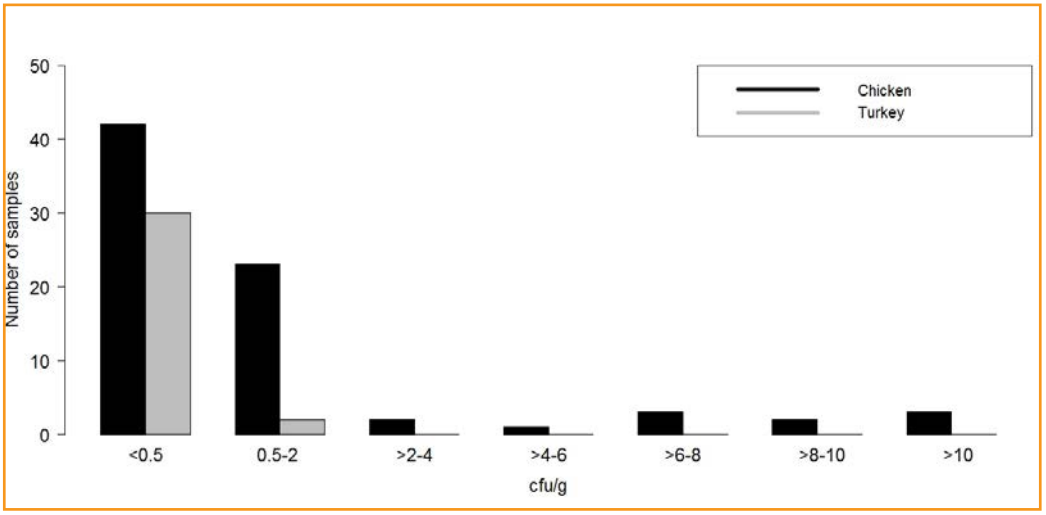


***Figure 5**. Positive concentrations obtained from 76 chicken meat samples and 32 turkey meat samples. These samples originated from 31 chicken meat retail batches and 17 turkey meat retail batches.*

A Bayesian prevalence–concentration model was developed to analyse the Finnish retail data from 2012–2014 for the specific fresh meats in order to obtain population estimates and make predictions as inputs for the consequent QMRA model, including the dose–response model. The combined prevalence–concentration model allowed an estimation of the overall prevalence in retail products, as well as the distribution of concentrations for positive samples. The dose–response model was adopted from the existing literature (Haas, 2002; Nauta *et al.*, 2007), but applied with Finnish consumption data.

Prevalence was modelled with a binomial model describing the batch prevalence and within-batch prevalence for the retail batches of chicken and turkey. The model takes into account that samples taken from the same retail batch are correlated due to their common origin. Seasonal changes in monthly prevalences were also accounted for using a Markovian time series. A simplified version of the prevalence model was applied to beef and pork, because no positive observations were found in these meat types. The simplified model does not include a time series for seasonal changes, and neither is it possible to separately estimate the batch prevalence and the within-batch prevalence for retail batches. However, the proportion of meat units with *Campylobacter* spp. contamination can be estimated.

The $\log_{10}$ concentrations in retail samples were modelled with a normal distribution, hierarchically accounting for variation between and within retail batches. The hierarchical Bayesian model was separately applied for chicken and turkey.

### 3.3.3.2 Results

The estimated monthly percentage of retail batches containing one or more contaminated units is presented in Tables 15 and 16. The retail batch prevalence was at its highest in the summer. The peak was reached in July, when approximately one-

third of both chicken and turkey meat retail batches were contaminated. In total, the retail batch prevalence was slightly higher in chicken meat than in turkey meat. In these contaminated retail batches, the estimated percentage of meat units containing *Campylobacter* spp. was 59.5% (95% CI: 49.8–68.8%) in chicken meat batches and 28.6% (95% CI: 18.3–40.0%) in turkey meat batches.

***Table 15.*** *Mean, median and 95% credible interval for the prevalence of Campylobacter spp. in chicken meat retail batches.*

| Month | Mean | Median | CI 2.5% | CI 97.5% |
|---|---|---|---|---|
| January | 8.6% | 7.1% | 0.2% | 26.3% |
| February | 11.9% | 10.7% | 2.7% | 28.2% |
| March | 11.9% | 10.5% | 2.7% | 28.3% |
| April | 7.1% | 6.2% | 0.5% | 19.1% |
| May | 7.8% | 6.8% | 0.5% | 20.9% |
| June | 14.6% | 13.7% | 3.4% | 30.8% |
| July | 33.3% | 32.7% | 18.9% | 50.7% |
| August | 29.0% | 28.3% | 15.2% | 46.5% |
| September | 13.8% | 13.0% | 2.9% | 29.6% |
| October | 11.0% | 10.2% | 2.2% | 24.6% |
| November | 11.0% | 10.0% | 2.3% | 25.4% |
| December | 7.8% | 6.3% | 0.1% | 23.8% |

***Table 16.*** *Mean, median and 95% credible interval for the prevalence of Campylobacter spp. in turkey meat retail batches.*

| Month | Mean | Median | CI 2.5% | CI 97.5% |
|---|---|---|---|---|
| January | 5.3% | 2.7% | 0.0% | 25.6% |
| February | 5.6% | 3.6% | 0.0% | 21.9% |
| March | 9.4% | 6.7% | 0.4% | 34.4% |
| April | 7.1% | 5.0% | 0.1% | 25.1% |
| May | 8.8% | 6.5% | 0.2% | 30.1% |
| June | 14.7% | 12.9% | 1.7% | 38.2% |
| July | 33.2% | 32.1% | 15.0% | 58.1% |
| August | 29.9% | 28.6% | 12.5% | 55.6% |
| September | 9.9% | 7.8% | 0.3% | 30.1% |
| October | 9.1% | 7.0% | 0.5% | 28.9% |
| November | 4.9% | 2.9% | 0.0% | 20.0% |
| December | 5.5% | 2.4% | 0.0% | 27.9% |

The average prevalence of *Campylobacter* spp. in retail meats within a year was estimated to be 8.3% (95% CI: 5.5–11.7%) in chicken and 3.4% (95% CI: 1.8–5.9%) in turkey meat. The retail meat prevalence was significantly higher in chicken meat than in turkey meat, because both the batch prevalence and the within-batch prevalence were estimated to be greater in chicken meat compared to turkey meat. No positive

results were found in beef or pork. The estimated 95% credible interval for the percentage of retail meats with contamination was from 0.0 to 1.2% in both beef and pork.

A high proportion of positive samples were below the limit of determination. Statistically, these were interpreted as left-censored data, which contributed to the parameter estimation accordingly. Based on all retail sample data, posterior predictive distributions were computed for the initial $\log_{10}$ concentration in a random sample to be purchased by a consumer, and for the final bacterial count in the meal. The posterior predictive distributions for the concentration of *Campylobacter* spp. in retail poultry meats are presented in Figure 6. These distributions include the uncertainty related to the unknown model parameters, as well as the variability between different contaminated meat units. The predicted concentration was considerably higher in contaminated chicken meat than in contaminated turkey meat, as can be seen in Figure 6. The predicted mean concentration was 1.96 CFU/g in contaminated chicken meat and 0.27 CFU/g in contaminated turkey meat. The predicted concentration for turkey meat is very low, because all observed concentrations were equal to or less than 0.5 CFU/g. The concentration could not be estimated for beef and pork, because no positive concentrations were detected in these meat types.

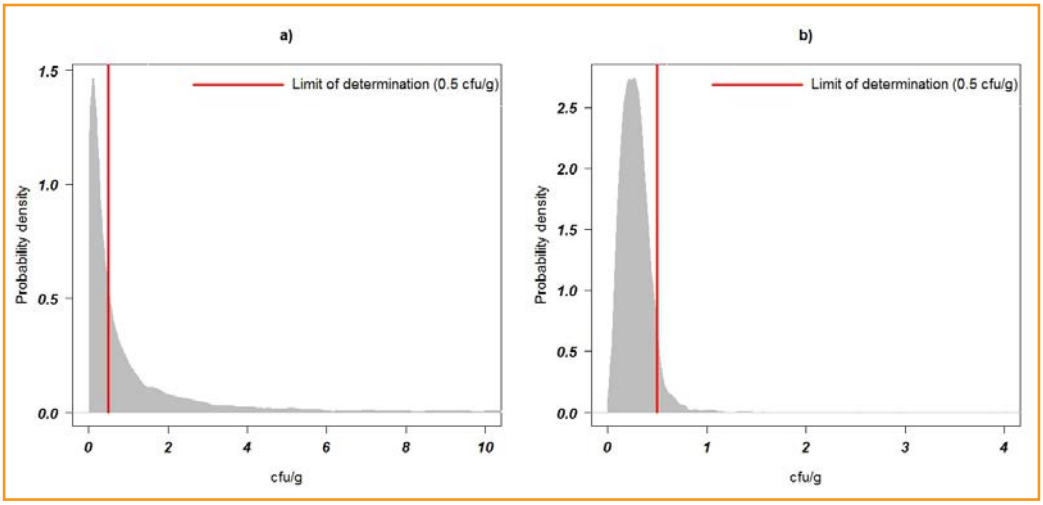


***Figure 6.*** *Posterior predictive distribution for the concentration of Campylobacter spp. in contaminated a) chicken meat and b) turkey meat at retail.*

## 3.4 Risk characterization

### 3.4.1 QMRA: risk estimate

The main goal of the risk assessment presented in this report was to quantify the risk of human campylobacteriosis caused by the consumption of meat and to collect information on environmental sources. The risk due to meat consumption depends on the estimated number of contaminated servings, but also on the level of contamination per such serving and the serving size. The level of contamination depends on the initial contamination of the raw material and the steps of the production chain. However, in this study, the data directly provided information on the exposure near the expected time of food preparation, because the samples were collected from retail shops.

Fresh meat is typically prepared by directly placing it in an oven, frying pan or grill. In Finland, it is also common to buy poultry meat as spiced, cut and packed in disposable packages. It was assumed that the meat itself is thoroughly cooked, which eliminates all bacteria. However, due to kitchen cross-contamination, campylobacters may indirectly enter the meal, even though the level of cross-contamination can be expected to be small. There is a lack of specific measured data on the effect of kitchen hygiene on campylobacters in Finland. The amount of cross-contamination was estimated based on a previous study by Nauta *et al*. (2012). Their investigation described cross-contamination in the kitchen, based on experimental data with volunteers preparing a chicken salad cross-contaminated by raw meat via utensils.

The risk per serving is the probability of illness when consuming a random serving. This depends on the probability ($q$) of the serving resulting from a contaminated meat purchase, and the probability of the number of bacteria ($d$) in the final meal. The parametric expression could be interpreted as the population fraction of cases of illness from all consumed servings if all the unknown parameters were given. The uncertainty of the parameters is accounted for by integration over a distribution of parameters. The contamination probability ($q$) (uncertain fraction) and bacterial counts ($d$) (variable number) are simulated from the posterior predictive distribution, based on retail samples and model assumptions:

$$P(\text{illness} \mid \text{retail samples \& assumptions}) = \int_0^1 \sum_{d=0}^{\infty} qP(\text{illness} \mid d)P(d, q \mid \text{data})\, dq$$

For beef and pork, the probability of illness could not be estimated due to the absence of positive samples. The probability of illness due to a single contaminated serving is presented in Table 17.

***Table 17.*** *Morbidity (ill) and infection (inf) per single contaminated serving.*

| | Mean ill/inf | CI 2.5% ill/inf | CI 97.5% ill/inf |
|---|---|---|---|
| Chicken meat 'as fresh' | 7.77/23.58% | 0.48/1.50% | 18.24/52.53% |
| Chicken meat as cooked + cc[1)] | 0.05/0.15% | 0.00/0.00% | 0.35/1.05% |
| Turkey meat 'as fresh' | 5.61/17.2% | 0.95/2.95% | 11.39/32.43% |
| Turkey meat as cooked + cc[1)] | 0.01/0.02% | 0.00/0.00% | 0.04/0.11% |

[1)] Cross-contamination to salad

The probability of illness due to any random meal was estimated to evaluate the risk at the population level. According to the results, around 40 per $10^6$ chicken meat servings led to disease due to cross-contamination. Hypothetically, if the meat had been eaten as raw, approximately 6 400 per $10^6$ portions would have caused illness in humans. For turkey meat, the corresponding results were 3 per $10^6$ and 2 000 per $10^6$.

The predicted number of disease cases would then be described by binomial distribution Bin($p$, $n_s$), where the probability of illness depends on the ingested random dose $d$ according to a chosen dose–response model (see section 3.2.3) for some number of servings $n_s$.

The relative risks (calculated for all servings, not just contaminated) of each meat type are presented in Tables 18 and 19. For beef and pork, distributions of concentrations could not be estimated due to the absence of positive samples. However, scenarios for beef and pork were evaluated based on assumed concentrations taken to be similar to those detected from the chicken and turkey samples, together with the estimated (low) prevalence. However, the concentrations for beef and pork are more likely overestimates, because the survival of campylobacters on cattle and swine carcasses is limited by drying and exposure to oxygen, in contrast to poultry carcasses with their protecting skin (Grau *et al.*, 1988; Corry and Atabay, 2001; Nesbakken *et al.*, 2008).

The results indicate that chicken meat caused relatively the highest infection risk and a larger number of human cases than any of the other meat types (Table 18). Turkey meat caused a significantly greater risk than beef and pork, because the estimated percentage of foods with contamination is significantly higher (Table 18). However, turkey meat is likely to have the smallest share of the total disease burden when the total consumption is taken into account (Table 19).

***Table 18.*** *Mean and 95% credible interval for the relative proportions of each meat type from the total number human cases they cause. Total consumption is not taken into account in this scenario.*

| | Mean | CI 2.5% | CI 97.5% |
|---|---|---|---|
| Chicken meat | 68.6% | 8.3% | 99.1% |
| Turkey meat | 22.6% | 0.6% | 79.0% |
| Beef meat | 4.4% | 0.0% | 33.0% |
| Pork meat | 4.4% | 0.0% | 33.1% |
| **Total** | **100 %** | | |

***Table 19.*** *Mean and 95% credible interval for the relative proportion of each meat type from the total number of human cases they cause. Total consumption is taken into account.*

| | Mean | CI 2.5% | CI 97.5% |
|---|---|---|---|
| Chicken meat | 82.0% | 14.6% | 99.8% |
| Turkey meat | 2.9% | 0.0% | 18.1% |
| Beef meat | 5.4% | 0.0% | 38.9% |
| Pork meat | 9.7% | 0.0% | 62.4% |
| **Total** | **100 %** | | |

The probability of illness due to the cross-contamination per one contaminated serving is very low (Table 17). The cross-contamination may still cause a considerable number of disease cases, because hundreds of millions of servings ($n_s$) are consumed every year. The predicted number of human campylobacteriosis cases due to fresh meat includes considerable uncertainty. Hence, the predictive distribution has a very long tail, and the posterior mean is much higher than the posterior median (Table 20).

***Table 20.*** *Mean and 95% credible interval for the predicted total number of human cases due to salad (or uncooked or RTE foods) that is cross-contaminated from fresh meat. Information on cross-contamination experiments by Nauta et al. (2007) was exploited in the prediction.*

| Source of cross-contamination | Mean | Median | CI 2.5% | CI 97.5% |
|---|---|---|---|---|
| Chicken fresh meat | 8 700 | 1 690 | 40 | 60 890 |
| Turkey fresh meat | 40 | 20 | 0 | 150 |
| Beef fresh meat | 150 | 20 | 0 | 2 000 |
| Pork fresh meat | 310 | 40 | 0 | 2 200 |
| **Total** | **9 200** | **2 150** | **90** | **60 980** |

The number of confirmed human campylobacteriosis cases in Finland was 4 059 in 2013 and 4887 in 2014 (THL, 2016). According to the National Register Infectious Diseases Register (2013-2014), half of the registered cases (50.0% in 2013 and 50.3% in 2014) were acquired from abroad, the place of origin was unknown in 38.0% of the cases in 2013 and 32.8% in 2014, and the remaining 12.0% and 16.9% of cases were of domestic origin. However, it is assumed that only about one-tenth of the true cases are registered (STM, 1997; EFSA, 2014). Thus, the actual number of domestic human campylobacteriosis cases would then be from about 5 000 to 20 000 per year.

The results indicate that chicken fresh meat may be responsible for a significant proportion of the human cases in Finland. The predicted number of human cases due to all meat types was estimated to be around 2 150 (posterior median). However, this estimate is affected by many uncertain factors such as the amount of cross-contamination, serving size, and total consumption. Hence, the 95% credible interval for the predicted number of human cases due to all meat types is very wide, from 90 to 60 980 cases.

### 3.4.2 Sensitivity and uncertainty analysis

In the analysis of prevalence and concentration at retail, the sensitivity to different prior distributions in the Bayesian model was assessed. The analysis demonstrated that the results are not substantially sensitive to the alternative prior distributions in the prevalence–concentration model. In addition to the log-normal model for concentrations, a gamma distribution was explored. The main difference in predictions between these two models was that the gamma model led to a predictive distribution that has a very long tail compared to the log-normal model. The log-normal model was better supported by the data compared to the gamma model. Hierarchical and non-hierarchical models were also compared. The results revealed that the hierarchical model was much better supported by the data than the non-hierarchical model. Due to the large proportion of negative samples and most positive samples being below the limit of determination, separate analysis of each meat type may be sensitive to the few measured concentrations. Therefore, an evidence synthesis model was applied by assuming a hierarchical model with common variance components for both chicken and turkey meat.

The information on cross-contamination was based on the meal preparation in domestic kitchens. However, a significant proportion of the fresh meats are prepared in the catering industry. According to the literature, 34% of the Finnish population uses catering services daily (STM, 2010). As no data are available on the differences in cross-contamination at home and in catering, it was considered similar in both premises. Professional kitchens use convenience foods, which reduces the possibility of cross-contamination. On the other hand, they handle different food items at the same time, and the quantities handled are larger, which can increase cross-contamination. Finnish home kitchens usually use meat already packed in portions that are ready for the pan or oven without cutting or other handling. The results based on retail sampling may not be applicable to professional kitchens as such, because they often use imported meat, and the retail samples were of domestic origin.

### 3.4.3 Assumptions and limitations

The turkey fresh meat sampled at retail consisted of fillets (large pieces), whereas the other fresh meat samples were strips (small pieces). This may have an effect on the survival of campylobacters, which is not possible to estimate. On the other hand, risk estimates for turkey strips may not be relevant, as such products are not available at retail. The estimation of the risk of human campylobacteriosis due to beef and pork consumption contained rough assumptions. Without data on concentration levels in pork and beef, we can only make scenarios assuming concentrations similar to those in chicken and turkey. In this risk assessment, all the samples were of fresh meat, because it was considered to cause the highest infection risk for humans. However, meat preparations (marinated, salted etc.) and products cannot be ruled out as a source of campylobacters for humans. Their share of the sales is 70–80%, but no data on the contamination level are available.

As the meat samples were collected within the Helsinki area, not all retail branches in the whole Finland were represented. Nevertheless, the samples represented major meat companies corresponding to the majority of the Finnish meat market. Since the model is based on retail sample data, the results are not vulnerable to assumptions that would be needed when predicting the concentrations at retail starting from earlier stages of the food chain, e.g. slaughter sample data.

The final consumer risk estimate greatly depends on kitchen cross-contamination. As there have been no studies on Finnish kitchen practices, the amount of cross-contamination was based on foreign studies that may not represent Finnish food preparation. A large proportion of the servings may nevertheless be prepared with sufficiently good hygiene. There were no comparable estimates for the catering industry (restaurants, schools, hospitals, canteens) either that could be quantified in the model.

The total number of servings is likewise uncertain and based on total annual consumption amounts divided by the typical serving size. A fundamental limitation of the predictive model is that it only applies to foods for which a plausible serving size can be assigned. This may represent only the average serving size of healthy adults, but those of other population groups can vary significantly. Furthermore, the dose–

response function may not describe all population groups, particularly the sensitive groups. Finally, other routes of infection do not involve foods. Hence, within the same predictive model, direct comparison is not possible for exposures via, for instance, swimming waters and pets, which lack a working definition and data on exposure.

Source attribution models are under development for a comparison of all sources of infection. These require genetic subtyping methods and extensive data from several potential sources, not only from foods but also from the environment. However, source attribution based on typing data alone cannot account for differences in the magnitudes of population-level exposures or uncertainties in type-specific population prevalences due to small sample sizes. Therefore, unifying approaches for risk assessment are still needed.

# 4 CONCLUSIONS AND RECOMMENDATIONS

**Conclusions**

The results suggest that chicken, turkey, beef and pork fresh meat may cause about one-third of all domestic human campylobacteriosis cases. Based on this study, most of these cases can be related to the consumption of chicken meat. However, the predicted number of human cases due to meat consumption includes considerable uncertainty. Among the main factors that cause uncertainty is the level of kitchen cross-contamination.

The average annual estimate for *Campylobacter* spp. prevalence in fresh domestic chicken meat was about 8%, which is relatively low. Self-sufficiency of chicken meat is nowadays high in Finland, but the decrease of self-sufficiency and/or increase of imports from countries with higher prevalence would cause significant raise in human campylobacteriosis incidence. For instance, 50% retail-prevalence would lead over the six-fold (95% CI: 4.4-9.1) increase in fresh chicken meat related human campylobacteriosis cases in Finland.

According to the results, fresh chicken meat causes a much higher *Campylobacter* spp. infection risk for humans than the other meat types. Fresh turkey meat causes a significantly higher infection risk for humans than beef or pork. However, the consumption volume of turkey meat is very small in Finland compared to other meat types. Hence, in total, fresh turkey meat probably causes fewer cases than any other fresh meat type.

Although the prevalence of *Campylobacter* spp. in fresh chicken meat at retail was low, it was significantly higher compared to other fresh meat types. The risk of *Campylobacter* spp. infection acquired from poultry fresh meat appears greater during the late summer, as the prevalence of campylobacters in poultry fresh meat peaked in July to August. The results show that the prevalence of *Campylobacter* spp. is most likely very low in fresh beef and pork.

The concentration of campylobacters was below the limit of determination (0.5 CFU/g) in most positive samples. Hence, proper methods for statistical analysis of the censored data are crucial to produce a predictive distribution for the concentration. Due to the lack of positive observations, the *Campylobacter* spp. concentration in contaminated fresh beef and pork is still unknown.

The within-batch *Campylobacter* spp. prevalence was estimated to be much higher than the between-batch prevalence at retail. Therefore, the clustering of samples is important to take into account when assessing exposure from poultry.

The between-batch variance in *Campylobacter* spp. concentrations was estimated to be significantly greater than the within-batch variance at the retail level.

The results suggest that the probability of illness per single serving, due to cross-contamination of salad or other uncooked food from fresh meat, is relatively low.

This risk assessment did not take into account meat preparations and products, such as salted, marinated and cooked food items.

Recreational water can be one source of campylobacters. However, representative sampling of environmental sources is challenging, and more targeted studies are needed to elucidate the routes of *Campylobacter* spp. infections.

The sampling frequency should be much higher than was possible in our study, and it should preferably include several samplings per day to increase the possibility of finding more isolates for comparison.

The contamination of water is a random event and the survival of bacteria during warm weather and exposure to sunlight (UV) is short. More targeted studies are needed on the connection between water contamination and infection of patients.

**Recommendations**

Special attention should be paid to kitchen hygiene. This is obviously one of the most important factors to reduce the number of foodborne *Campylobacter* spp. infections. Based on this study, the number of human cases could be up to a thousand-fold higher if all bacteria carried by fresh meat are ingested.

Simple guidelines such as the 'five keys to safer food' launched by WHO are also effective against campylobacters sprawl in the kitchen environment (1. keep clean, 2. separate raw and cooked, 3. cook thoroughly, 4. keep food at safe temperatures, 5. use safe water and raw materials), and are additionally worth distributing in countries with a high level of hygiene.

Generally accepted good hygiene practices should be followed throughout the food chain to reduce the load of campylobacters.

Information campaigns on safe eating and drinking habits should be targeted at Finnish travellers in order to prevent foodborne campylobacteriosis being acquired from abroad.

Data on the response of different susceptible populations are required for more specific exposure assessment.

More information and genotyping data on campylobacters strains from potential sources – whether food or other sources – are needed in order to provide more accurate risk assessments.

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