MICROBIOLOGICAL RISK ASSESSMENT SERIES
11

# RISK ASSESSMENT OF CAMPYLOBACTER SPP. IN BROILER CHICKENS

INTERPRETATIVE SUMMARY

WORLD HEALTH ORGANIZATION FOOD AND AGRICULTURE ORGANIZATION OF THE UNITED NATIONS

2009

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## FOREWORD

The Members of the Food and Agriculture Organization of the United Nations (FAO) and of the World Health Organization (WHO) have expressed concern regarding the level of safety of food at both national and international level. Increasing foodborne disease incidence in recent decades seems, in many countries, to be related to an increase in disease caused by microorganisms in food. This concern has been voiced in meetings of the Governing Bodies of both Organizations and in the Codex Alimentarius Commission. It is not easy to decide whether the suggested increase is real or an artefact of changes in other areas, such as improved disease surveillance or better detection methods for microorganisms in patients and/or foods. However, the important issue is whether new tools or revised and improved actions can contribute to our ability to lower the disease burden and provide safer food. Fortunately, new tools, which can facilitate actions, seem to be on their way.

Over the past decade, Risk Analysis—a process consisting of risk assessment, risk management and risk communication—has emerged as a structured model for improving our food control systems, with the objectives of producing safer food, reducing the numbers of foodborne illnesses and facilitating domestic and international trade in food. Furthermore, we are moving towards a more holistic approach to food safety, where the entire food chain needs to be considered in efforts to produce safer food.

As with any model, tools are needed for the implementation of the risk analysis paradigm. Risk assessment is the science-based component of risk analysis. Science today provides us with in-depth information on life in the world we live in. It has allowed us to accumulate a wealth of knowledge on microscopic organisms, their growth, survival and death, even their genetic make-up. It has given us an understanding of food production, processing and preservation, and of the link between the microscopic and the macroscopic worlds and how we can benefit from, as well as suffer from, these microorganisms. Risk assessment provides us with a framework for organizing all this data and information and to better understand the interaction between microorganisms, foods and human illness. It provides us with the ability to estimate the risk to human health from specific microorganisms in foods and gives us a tool with which we can compare and evaluate different scenarios, as well as identify the types of data necessary for estimating and optimizing mitigating interventions.

Microbiological risk assessment can be considered as a tool that can be used in the management of the risks posed by foodborne pathogens and in the elaboration of standards for food in international trade. However, undertaking a microbiological risk assessment (MRA), particularly quantitative MRA, is recognized as a resource-intensive task requiring a multidisciplinary approach. Nevertheless, foodborne illness is one of the most widespread public health problems, creating social and economic burdens as well as human suffering; it is a concern that all countries need to address. As risk assessment can also be used to justify the introduction of more stringent standards for imported foods, a knowledge of MRA is important for trade purposes, and there is a need to provide countries with the tools for understanding and, if possible, undertaking MRA. This need, combined with that of the Codex Alimentarius for risk-based scientific advice, led FAO and WHO to undertake a programme of activities on MRA at the international level.

The Nutrition and Consumer Protection Division (FAO) and the Department of Food Safety and Zoonoses (WHO) are the lead units responsible for this initiative. The two groups have worked together to develop the area of MRA at the international level for application at both the national and international level. This work has been greatly facilitated by the contribution of people from around the world with expertise in microbiology, mathematical modelling, epidemiology and food technology, to name but a few.

This Microbiological Risk Assessment series provides a range of data and information to those who need to understand or undertake MRA. It comprises risk assessments of particular pathogen-commodity combinations, interpretative summaries of the risk assessments, guidelines for undertaking and using risk assessment, and reports addressing other pertinent aspects of MRA.

We hope that this series will provide a greater insight into MRA, how it is undertaken and how it can be used. We strongly believe that this is an area that should be developed in the international sphere, and the work to date clearly indicates that an international approach and early agreement in this area will strengthen the future potential for use of this tool in all parts of the world, as well as in international standard setting. We would welcome comments and feedback on any of the documents within this series so that we can endeavour to provide member countries, Codex Alimentarius and other users of this material with the information they need to use risk-based tools, with the ultimate objective of ensuring that safe food is available for all consumers.

Ezzeddine Boutrif

Jørgen Schlundt

Nutrition and Consumer Protection Division, FAO Department of Food Safety and Zoonoses WHO – xiii —

This risk assessment has been elaborated over a number of years. A number of national risk assessments that were available or being finalized when this work was initiated in 2001 were used as a basis from which to elaborate this risk assessment. In the course of its development, the risk assessment has been reviewed by two expert consultations—in 2001 and 2002—and been presented in various forums, including the Codex Committee on Food Hygiene and some international scientific conferences and meetings. Finally, it was subjected to peer review in 2006. Comments and feedback received at each of these steps have been taken into account in the finalization of the risk assessment.

In parallel to the elaboration of this risk assessment, research into *Campylobacter* spp. in broiler chickens and related issues has been ongoing, and risk assessment work at a national level has continued in some countries. Taking this into consideration, the recent literature has been reviewed and incorporated into this work as appropriate to ensure that the risk assessment is current at the time of publication in terms of recent developments in scientific knowledge and data.

## ABBREVIATIONS

%	percentage
°C	degree Celsius
CCFH	Codex Committee on Food Hygiene
cfu	colony forming unit
FAO	Food and Agriculture Organization of the United Nations
WHO	World Health Organization

## **1. INTRODUCTION**

The Food and Agriculture Organization (FAO) and the World Health Organization (WHO) assembled an expert drafting group to develop a risk assessment framework for *Campylobacter* spp. in broiler chickens. Initially, the risk management questions posed by the Codex Committee on Food Hygiene (CCFH) were the same as those that were addressed in the risk assessment for *Salmonella* in broiler chickens. The questions requested estimates for: 1) the risk of exposure and illness as a result of exposure to *Campylobacter* from broiler chicken products; and 2) the change in exposure and illness likely to occur following the implementation of different interventions in primary production, processing and food handling.

The initial framework and the document "Draft Hazard Identification, Hazard Characterization and Exposure Assessment of *Campylobacter* spp. in broiler chickens" were presented to the Codex Committee on Food Hygiene (CCFH) for comment in October 2001, in Bangkok, Thailand. In March 2002, work began on completion of the risk characterization portion of the project. The culmination of this work is a risk assessment model and technical document "A Risk Assessment of *Campylobacter* spp. in Broiler Chickens." The model and document include the risk characterization component to complete the risk assessment. Comparisons of risk for a variety of scenarios and mitigation measures were also conducted. The review and analysis of current scientific information and the risk model can be used by FAO and WHO member countries to conduct country-specific risk assessment for *Campylobacter* spp. in chicken products. This interpretative summary provides an overview of the assessment. The complete technical report of the risk assessment is available in volume 12 of the FAO/WHO Microbiological Risk Assessment Series.

## 2. SCOPE OF THE RISK ASSESSMENT

The products considered in this report include fresh intact broilers or whole chickens, and chicken parts. Frozen chicken was also considered. The exposure scenario is limited to consumer home preparation.

In many countries, *Campylobacter jejuni* is isolated as the agent responsible for more than 90% of cases of campylobacteriosis. *Campylobacter coli* is often the second most isolated species. For the most part, when this report is discussing *Campylobacter* spp., especially with respect to human clinical isolates, it is referring to *Campylobacter jejuni*.

The current risk assessment is not intended to describe any one geographical location or system, but rather it was developed and designed to provide relative risk comparisons. It should be understood that the assessment does not provide estimates for the absolute risk attributable to any one system. In order to fully characterize a specific system, the features of that system need to be captured (every system or country is likely to be different to varying degrees), with data specific to that system applied carefully.

## 3. HAZARD IDENTIFICATION

The hazard identification evaluates the scientific data and information and characterizes evidence concerning the sources of *Campylobacter* infection arising from the consumption of chicken in the human population in developed and developing countries. The human incidence, characteristics of the organism, sources of infection, risk factors and characteristics of the infection are covered in this section.

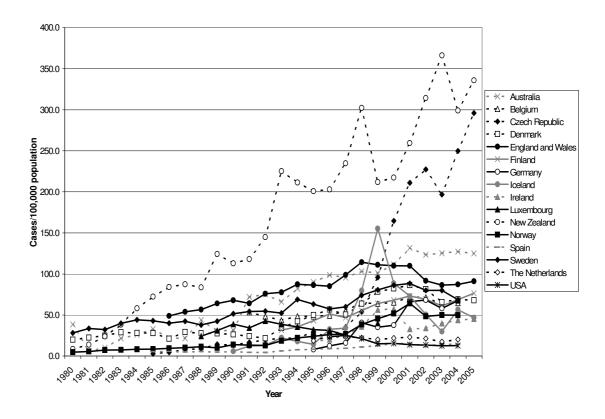
#### 3.1 Human incidence of Campylobacter illness

*Campylobacter* is a leading cause of zoonotic enteric infections in most developed and developing nations (WHO, 2001). The trend for registered human cases per 100 000 inhabitants caused by thermotolerant *Campylobacter* for a number of countries is shown in Figure 1. The reported incidence of *Campylobacter* infections has markedly increased in many developed countries within the last 20-year period. Underreporting of campylobacter infections is an issue in most countries and incidence rates only reflect the number of laboratory-confirmed cases. As a result, the true rate of infection is higher than the number of reported cases, and is estimated to range from 7.6 to 100 times higher (Skirrow, 1991; Kapperud, 1994; Wheeler et al., 1999; Mead et al., 1999; Samuel et al., 2004). Cases are usually caused by *Campylobacter jejuni*, and to a lesser extent by *Campylobacter coli* (Nielsen, Engberg and Madsen, 1997; Wooldridge and Ketley, 1997; Anon., 1999, 2006b; Nadeau, Messier and Quessy, 2001). Most *Campylobacter* infections are classified as sporadic cases, or as part of small, family-related outbreaks, and identified outbreaks are relatively uncommon.

The burden of human *Campylobacter* infections is not known for many developing countries, as national surveillance does not exist. Estimates of incidence in developing countries are from laboratory-based surveillance studies, giving estimates for the general population of from 5 to 20% (Coker et al., 2002). This is quite similar to the general incidence in developed countries. However, available data suggest a significantly higher incidence rate for campylobacteriosis in children. In developing countries, numerous studies have shown *Campylobacter*, and *C. jejuni* in particular, commonly cause infection in children below 5 years of age, and especially in children under the age of one year (Megraud et al., 1990; Calva et al., 1988; Jain et al., 2005; Simango and Nyahanana, 1997; Bodhidatta et al., 2002).

#### 3.2 Sources of illness and risk factors

The sources and incidences of illness differ, sometimes quite dramatically, between developed and developing countries. In developed countries, person-to-person transmission is considered to be infrequent (Altekruse et al., 1999; Ethelberg et al., 2004). In developing countries, in contrast, human carriage may play a larger role in the transmission of infection (Blaser, Taylor and Feldman, 1983). Overall, chicken, poultry and other foods are thought to be the most likely potential sources of infection in developed countries (Blaser and Reller, 1981; Deming et al., 1987; Friedman et al. 2004; Neiman et al., 2003). Most studies have identified handling of raw poultry and the consumption of poultry products as important risk factors, accounting for a variable percentage of cases (Neimann et al., 2003; Wingstrand et al., 2006;



**Figure 1.** The number of registered human cases per 100 000 population caused by *Campylobacter jejuni/coli*. The data presented are those data available to the drafting group up to June 2006.

Sources: CDC-FoodNet; Anonymous, 1999, 2001a, b, c, d, e, f, g, 2002, 2003, 2004, 2005a,b, 2006a; Friedman et al., 2000; Benes, 2001; Kruse, 2001; Georgsson, 2001, pers. comm.

Newell, 1982; Oosterom et al., 1984; Kapperud et al., 1992, 2003; Studahl and Andersson, 2000; Hopkins and Scott, 1983). In addition, cross-contamination of *Campylobacter* from raw chicken to prepared food (Hopkins and Scott, 1983; Kapperud et al., 2003) has been identified as a risk factor. Other food-related risk factors that have repeatedly been identified include consumption of other meat types, and undercooked or barbecued meat (Oosterom et al., 1984; Ikram et al., 1994), raw seafood, drinking untreated surface water (Hopkins, Olmsted and Istre, 1984), unpasteurized milk or dairy products (Neimann et al., 2003; Studahl and Andersson, 2000; Hopkins, Olmsted and Istre, 1984; Harris, Weiss and Nolan, 1986; Schmid et al., 1987). In the USA and New Zealand, consumption of meat cooked outside the home (at restaurants) has also been identified as a risk factor (Ikram et al., 1994; Eberhart-Phillips et al., 1997; Effler et al., 2001; Friedman et al., 2004).

In developing countries, waterborne transmission, direct contact with animals, and environmental sources are thought to be the major routes of human infection (Georges-Courbot et al., 1990; WHO, 2001; Coker et al., 2002). Food sources including chicken, and food production and preparation workers may also serve as important sources for campylobacteriosis (Koulla-Shiro, Loe and Ekoe, 1995; Harvey et al., 2003).

The clinical manifestations of *Campylobacter* infections also differ between developed and developing countries, both in the ages of the affected populations and in the severity of illness. In developed countries, *Campylobacter* enteritis often affects older children and young adults and can be severe, characterized by fever, abdominal cramping and bloody diarrhoea that may require treatment with antimicrobials. In contrast, *Campylobacter* infections in developing countries tend to affect children under one year of age, with more severe symptoms and illness. In older children, the illness and symptoms are often milder. Strain differences could be one explanation for these observed epidemiological differences, with fewer yet more severe infections in developing nations. *C. jejuni* is responsible for a majority of *Campylobacter* infections in both developing and developed countries, although strains such as *C. coli*, *C. lari*, *C. upsaliensis* and *C. hyointestinalis*, and others, may be responsible for a larger proportion of infections in developing countries to developed countries (Georges-Courbot et al. 1986).

#### 3.3 Characteristics of the organism

Bacteria belonging to the genus *Campylobacter* are non-spore forming, oxidase-positive, Gramnegative rods. In general, *Campylobacter* spp. grow at 37°C, but not below 32°C. *C. jejuni* and *C. coli* are distinguished from most other *Campylobacter* spp. by their high optimum growth temperature (42°C). Therefore, one can reasonably assume that *Campylobacter* spp. do not multiply during slaughter, post-processing, transport and refrigerated storage of chicken products. Though *Campylobacter* spp. may persist for prolonged periods in chilled and frozen products, a reduction in the concentration and viability has been recorded after several weeks of storage at 4°C and in frozen poultry after several months (Solow, Cloak and Fratamico, 2003). *C. jejuni* and *C. coli* are rather sensitive to heat (D-values are 0.21 to 2.25 minutes at 55–60°C) (ICMSF, 1996)

*C. jejuni* is predominantly associated with poultry (Tauxe, 1992; Anon., 1998, 1999, 2001a, 2006b; Nadeau, Messier and Quessy, 2001), but has also been isolated from cattle, sheep, goats, dogs and cats (Nielsen, Engberg and Madsen, 1997; Anon., 1999, 2006b). *C. coli* is predominantly found in pigs (Rosef et al., 1983; Nielsen, Engberg and Madsen, 1997; Boes et al., 2005; Jensen et al., 2006), but has also been isolated from poultry, cattle, and sheep (Anon., 1999, 2006b).

### 4. RISK ASSESSMENT MODEL OVERVIEW

The risk assessment, in accordance with CODEX guidelines for conducting microbiological risk assessment, consists of the accumulation of an exposure assessment, hazard characterization and risk characterization. A schematic overview of the framework for the *Campylobacter* risk assessment is shown in Figure 2.

The model was implemented in Microsoft Excel<sup>TM</sup>, together with the add-in @Risk<sup>TM</sup> to provide Monte Carlo simulation capability.

#### 4.1 Exposure assessment

The exposure assessment considers the occurrence and number of *Campylobacter* that may be present in chicken products. As illustrated in Figure 2, the model is based upon a modular structure. The stages from rearing of broilers to the consumption of chicken products are grouped into four main modules, namely: 1. *Farm & Transport*; 2. *Processing*; 3. *Storage*; and 4. *Preparation*. The exposure assessment initially evaluates the frequency and levels of *Campylobacter* on the farm, estimating the probability that a random flock is *Campylobacter*-positive, the within-flock prevalence, and the levels of colonization and contamination of the broilers (internally and externally). Subsequently, the stages of transport, processing, storage and preparation by the consumer are explored, and combined to predict the overall impact that these stages will have upon the contaminating *Campylobacter* load on a random chicken carcass or product to determine the final exposure level.

The *Farm & Transport* module utilizes observational data to estimate the prevalence of *Campylobacter*-positive flocks. A positive flock is defined as a flock that contains one or more birds colonized with *Campylobacter* spp. The within-flock prevalence is related to the rate of transmission within the flock environment and is therefore a time-dependent phenomenon for a positive flock. To estimate within-flock prevalence a dynamic transmission model is used to predict the within-flock prevalence of a positive flock at slaughter (Hartnett et al., 2001). Descriptive models then estimate the extent of external contamination that occurs while the birds are on the farm, and the external contamination that occurs during transportation of the flock to the facility. The level of colonization at the point of slaughter is based upon observational data.

The *Processing* module examines the possible impact that a series of processing stages will have on the level of contamination on a carcass. The model attempts to capture the key elements and stages of processing. Specifically, the model considers the level of contamination on a carcass after scald, de-feathering, evisceration, washing (both with and without chlorine) and chilling (both air and water chilling). For each of the processing stages considered, experimental data has been collected. The impact that the stages will have upon the level of *Campylobacter* contaminating a given product is described mathematically, taking into account the complexity of the process, and the quantity and quality of data available for each stage. The outcome is an estimate of the probability that a random chicken product will be contaminated with *Campylobacter* and a distribution for the number of organisms present.

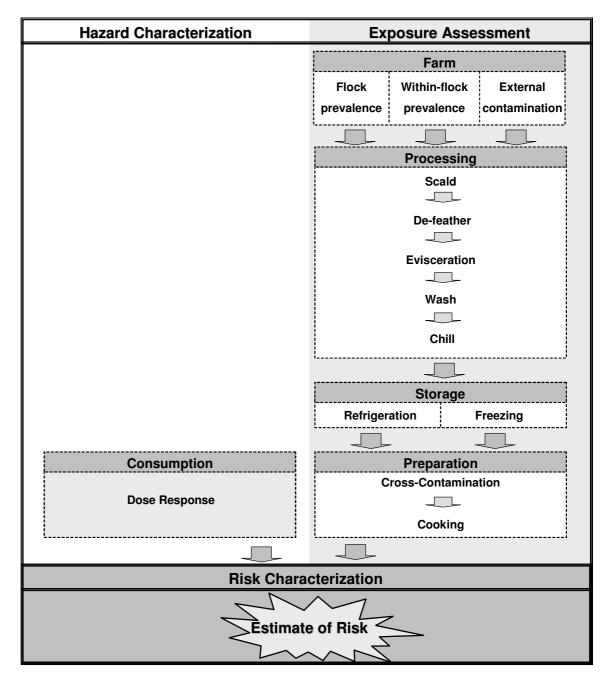


Figure 2. Schematic representation of the risk assessment model for *Campylobacter* spp. in broilers.

The *Storage* module considers storage under both refrigeration and freezing, as these conditions are known to reduce the numbers of *Campylobacter* on chicken products. For refrigerated storage, data by Koidis and Doyle (1983) are used, and for frozen storage, the experimental studies of Aho and Hirn (1988) were utilized to define time-dependent thermal

death models for *Campylobacter*. Using available data, a relationship between the proportion of organisms remaining and a given amount of time under refrigeration or freezing conditions was estimated to predict contamination levels on a product post-storage.

The *Preparation* module looks at the exposure of individuals to *Campylobacter* from consumer handling and preparation in private kitchens. This is addressed in two parts: (1) cross-contamination from chicken product to other parts of a meal during food handling procedures; and (2) the potential survival of *Campylobacter* in cooked chicken. It should be noted that there is a large degree of uncertainty and variability associated with the food handling procedures. Estimating the risk of infection via cross-contamination and undercooking is a difficult exercise due to the many different possible contamination routes and the considerable diversity in the food handling practises of individuals. Considering the complexity of food handling behaviours, data in the area are available to describe only a very small subset of the behaviours that could lead to exposure.

At present, it is considered infeasible to model distinct kitchen processes into different contamination routes, and to reliably quantify the relative contributions of each route to the overall risk. Therefore, a model previously developed for *Campylobacter* risk assessment has been used (Fazil et al., 2000). Referred to as the "Drip Fluid" model, it is a simplification of the very complex process of cross-contamination.

To estimate the impact of cooking upon contamination levels, four main assumptions are made: (1) that the only cells that will survive during cooking are those within an area that is relatively protected, or insulated, from the heat of the oven; (2) that some fraction of the organisms are located within these protected areas; (3) that the impact of cooking can be characterized by a maximum temperature within these protected areas for a specified duration; and (4) that the variability in consumer practices is responsible for the variation in the maximum temperature reached (due to variation in cooking time). A reduction in cell numbers is calculated based on the assumed time at the final temperature within the protected area. Uncertainty is characterized for each of the numerical assumptions.

#### 4.2 Hazard Characterization

Hazard characterization describes the adverse health effects of a substance, organism or other entity. This component of the risk assessment usually includes a dose-response relationship. This is represented as a probability that a random member of the population will become infected or ill after exposure to a specific number of *Campylobacter* organisms. The types of data that can be used to establish dose-response relationships include animal and human feeding studies, and epidemiological data, such as data from outbreak investigations.

There is insufficient information in the epidemiological literature to allow a dose-response relationship to be derived using epidemiological data. There is one human feeding trial study that was conducted (Black et al. 1988) using just over one hundred healthy young adult volunteers (in the United States of America).

The dose-response data for infection for strain A3249 (Black et al., 1988) have been fitted to dose-response models using maximum likelihood techniques (Medema et al., 1996; Teunis et al., 1996). The exact solution of the Beta-Poisson model has been reported to provide a statistically significant fit to the data (Teunis and Havelaar, 2000). In this risk assessment, the

data for *C. jejuni* A3249 and 81-176 were pooled and fitted to the Beta-Poisson dose-response model.

In order to estimate the probability of illness, the conditional probability of illness following infection is required. The human feeding trial data available did not indicate a clear dose-response relationship for the conditional probability of illness following infection. A conditional probability that is independent of the dose causing infection is applied in this risk assessment.

#### 4.3 Risk Characterization

The risk characterization step of risk assessment integrates the information collected during the hazard identification, exposure assessment and hazard characterization steps to generate estimates of the probability of adverse events that can be predicted to follow the preparation and consumption of chicken. This step links the probability and magnitude of exposure to *Campylobacter* to the probability of illnesses that may occur. The resulting risk is expressed as individual risk, or the risk per serving of chicken. Although this model does not address risk to a specific population, data on amounts of chicken consumed can be incorporated into the model to generate estimates of risk to populations.

As stated earlier, the current risk assessment is not intended to describe any one geographical location or system, but rather it was developed and designed to provide relative risk comparisons. It should be understood that the assessment does not provide estimates for the absolute risk attributable to any one system. In order to fully characterize a specific system, the features of that system need to be captured (every system or country is likely to differ to varying degrees), with data specific to that system applied carefully.

For the purposes of this risk assessment, most of the focus of risk characterization is on scenario analysis. The scenario analysis relies on relative comparisons. Even though the scenario analysis forms the focus of this risk characterization, some basic background information and insight can be gained by looking at a "baseline" model that represents a point of reference. The baseline model in this section is defined as a system with an overall flock prevalence of 80%, in which chickens are water chilled without free chlorine, and sold fresh (refrigerated but not frozen). The model, using this baseline scenario, was run for 10 000 iterations.

Figures 3 and 4 present the results for the estimated change in the levels of contamination on the surface of the chicken, as the chickens progress from the farm to the chilling stage (water chill without chlorine) at the end of the process. The model simulates chickens coming from positive and negative flocks separately. In Figure 3, the change in level of contamination is for chickens coming from test-positive flocks, while in Figure 4 the results are for chickens from test-negative flocks.

In Figures 3 and 4, a contamination level of  $-6 \log_{10}$  cfu indicates no contamination, or 0 cfu (to facilitate incorporating zero on a logarithmic scale). In general, for positive flocks, apart from the initial increase in contamination during the transportation of chickens, the mean level of contamination decreases through the process. Of note, the "min", "max" and other percentiles (10th and 50th percentiles shown) for the levels of contamination are closest together during defeathering, indicating a reduced level of variation in the contamination level. Although the mean level drops at this stage, the defeathering process acts such that contamination is spread from one bird to another in a mixing process so that chickens with low levels of contamination

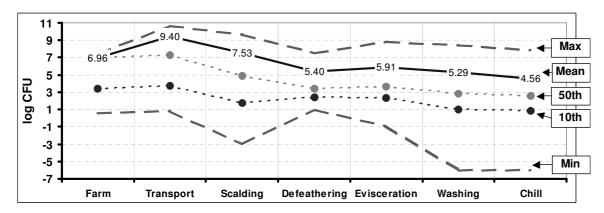


Figure 3. Change in contamination levels on chickens from positive flocks (Baseline model with 80% of flocks positive)

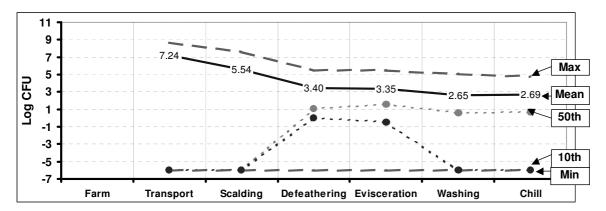


Figure 4. Change in contamination levels on chickens from negative flocks (Baseline model with 80% of flocks positive).

become more contaminated from material drawn off the heavily contaminated carcasses by the de-feathering machinery.

In Figures 3 and 4, a contamination level of  $-6 \log_{10}$  cfu indicates no contamination, or 0 cfu (to facilitate incorporating zero on a logarithmic scale). In general, for positive flocks, apart from the initial increase in contamination during the transportation of chickens, the mean level of contamination decreases through the process. Of note, the "min", "max" and other percentiles (10th and 50th percentiles shown) for the levels of contamination level. Although the mean level drops at this stage, the de-feathering process acts such that contamination is spread from one bird to another in a mixing process so that chickens with low levels of contamination become more contaminated from material drawn off the heavily contaminated carcasses by the de-feathering machinery.

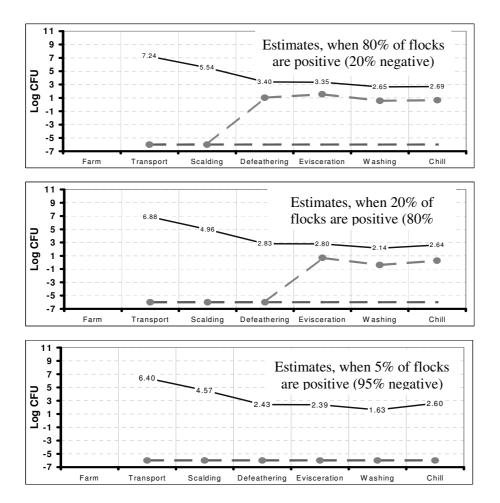
The model simulates a system in which flocks arrive at slaughter in a random fashion, so that a positive flock might arrive first, followed by a negative flock, and then followed by another positive flock. The changes in contamination on birds from positive flocks and on birds from negative flocks as they progress through the process are simulated separately. The separation of positive and negative flocks in the simulation can help to give insight into the differences that exist when previously positive birds enter the system and when previously negative birds enter the system. This feature could be applied to study logistic slaughter (i.e. where test-negative flocks are processed before test-positive flocks to avoid cross-contamination).

In Figure 4, there is no contamination indicated at the farm level because the flock is negative. During transport, however, contamination does occur. The frequency with which contamination occurs at this stage is clearly a function of several factors: the overall prevalence of positive flocks (dictating the probability that a negative flock will be transported in trucks previously used to transport positive flocks); the presence of any refinement in the logistics employed by the industry (e.g. transporting and processing negative flocks first); and the management strategies employed in the industry (e.g. cleaning and disinfection of trucks between loads). Even though the mean level of contamination spikes after transport, the implication is not that all the birds are contaminated. In fact, from the percentiles shown on the figure, it can be seen that the 50th percentile value remains at zero at transportation, indicating that more over 50% of the birds from negative flocks do not become contaminated at that stage. The high mean value is the result of a few birds coming in contact with very high levels of contamination.

The impact of different between-flock prevalence values on the potential for contamination of negative flocks is explored in Figure 5. This figure shows three different hypothetical systems: one in which 80% of the flocks are positive; one in which 20% of the flocks are positive; and one where 5% of the flocks are positive.

The mean level of contamination on flocks that are negative at the farm level spikes from no contamination to a value greater than 0 during transport in all three cases shown. This is because, as long as there are positive flocks in the system, there exists some probability that a negative flock will be transported after a positive flock in a truck in which cross-contamination could occur. Obviously, the probability of this happening decreases as the prevalence of positive flocks in the system decreases.

Although not shown in Figure 5, when the transportation of flocks is purely random, and when the overall flock prevalence is 80%, it was calculated that there is slightly less than a 50% probability that individual birds from negative flocks will become contaminated during transportation. When the overall flock prevalence is 20%, the probability decreases to just under 20%, and when the overall flock prevalence is further reduced to 5%, the probability is approximately 5%. The implementation of management strategies, such as those listed earlier (cleaning, processing negative flocks first, etc.) will have similar effects as far as reducing the probability of negative birds becoming positive due to cross-contamination. However, any of the management strategies would effectively be doing the same thing in different ways and with perhaps different degrees of effectiveness. Employing a combination of strategies may have a synergistic effect.



**Figure 5.** Change in contamination level for birds from negative flocks processed in conjunction with positive flocks (individual birds from positive and negative flocks not mixed, but arrival of positive and negative flocks at processing plant on a random basis)

KEY: Top solid line with values represents mean level, middle curve represents 50th percentile, bottom curve represents minimum value from simulations.

## 5. SCENARIO ANALYSIS

The construction of a simulation model of a system (in this case, characterizing chicken production, processing and consumption) allows users to gain insights into the system that are otherwise unavailable. A key source of insight is the repeated simulation of a variety of scenarios that demonstrate how the system responds to different configurations of inputs and assumptions. Some of these configurations may constitute proposed risk mitigation options. Others may simply be alternative possible realities that must be considered in order to fully understand the system being described.

In the current assessment, several scenarios were constructed that were intended to reflect and inform the formation of potential risk-management strategies. The scenarios could be classified as "general" versus "specific". The "general" scenarios provide insight into potential approaches that might be used to reduce the risk, without defining a specific strategy. The "specific" scenarios are ones that can be interpreted as reflecting the measurement of the impact of a candidate strategy to see how it can be expected to perform. It might also help to determine if there are any complications, caveats or issues that need to be considered in assessing the expected performance of the strategy.

It is important to recognize that, in any of the scenarios presented, the results should be interpreted on a relative basis as opposed to an absolute basis. Specifically, the model does not represent any one geographical location and is purposely intended to provide a generic evaluation of the situation.

# 5.1 Scenario 1: Change in the prevalence of contamination of chickens at the retail stage

The first set of scenarios investigated looked at the effect of altering the prevalence of contaminated chickens at the retail level. These scenarios can be classified as general scenarios, because they do not specify how the prevalence at the retail level is reduced, rather they estimate the effect that could be expected should a strategy be implemented that reduces the prevalence to the levels described. In this scenario, the prevalence level effects are assumed to be measured at the end of processing, or as the products are going to retail sale. The results presented in this scenario capture mitigation strategies anywhere along the food production chain prior to retail that are expected to reduce the prevalence, while having no impact on the levels of *Campylobacter* on positive product.

In order to evaluate the prevalence scenarios, the model was simulated using several different post-process prevalence levels, and the corresponding risk estimates generated. Prevalence levels from 0% to 100% were tested, in 5% intervals, resulting in 21 scenarios. The model was simulated for 5 000 iterations at each prevalence level, and the expected risk from the consumption of refrigerated and frozen chicken was estimated.

Figure 6 shows the estimated expected risk (y-axis) corresponding to a prevalence level (x-axis). The results are based on an assumed contamination level of approximately 4.0  $\log_{10}$  cfu/chicken; however, the relationship applies regardless of the level of contamination.

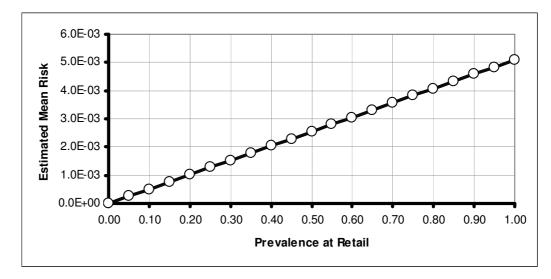


Figure 6. Prevalence at retail versus estimated mean risk-per-serving from consumption of refrigerated chicken.

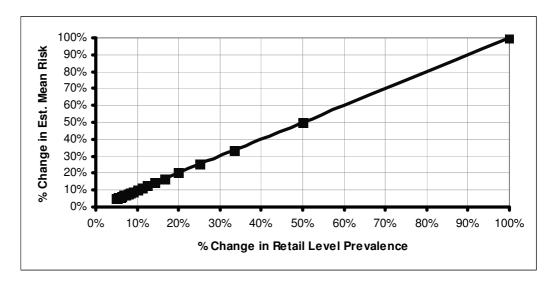


Figure 7. Percent reduction in prevalence versus percent reduction in mean risk.

Figure 7 is a variation on Figure 6, showing the percentage *reduction* in the estimated mean risk that is predicted in response to a specific percentage *reduction* in the prevalence at the retail stage. This sort of reduction in the prevalence of contamination at retail, *where no change in the levels of contamination in positive product is assumed*, may be the result of improved production practices that lead to fewer positive flocks, or test-and-divert strategies that remove contaminated product from the fresh or frozen retail pathway and divert it to thermal processing.

#### 5.2 Scenario 2: Change in the level of contamination

A second risk-management scenario looked at the effect of changes in the contamination level on chickens. Whereas the previous scenario was interested in altering the frequency with which products are contaminated, the current scenario was interested in looking at altering the level of contamination when the product is in fact contaminated. Thus, no change in prevalence is assumed in this scenario.

In order to evaluate the effect that a change in the level of contamination would have on the risk estimates, the model was re-simulated using various contamination levels. The contamination levels in these scenarios are defined to be those that occur at the retail level. As a result, these scenarios could reflect the results of any risk management strategy from farm to the retail level that is able to achieve specified degrees of reduction in contamination levels. The scenarios do not specify what the specific strategy might be or where exactly prior to retail it might be implemented.

Several contamination levels were selected, and the model was re-simulated at each level. The risk associated with each contamination level was then collected and the percent reduction in risk for a corresponding reduction in contamination level was calculated. Figure 8 summarizes the results of the simulations. The horizontal line shows the percent reduction in concentration between successive simulations. Since it is a horizontal line, the interpretation is that the concentration was reduced by a constant percentage for every simulation, approximately 44%, starting at a level of 10 000 000 cfu (7.0 log<sub>10</sub> cfu). The increasing line shows the percent reduction in risk as a result of the corresponding reduction in the contamination level. Note that if the risk were directly proportional to the number of organisms (as is largely the case for prevalence), the two lines would overlap.

Of significance in this figure are the regions above and below the horizontal line. When the percent reduction in mean risk line (the curved line) is below the horizontal line, the implication is that a contamination level decrease in this area does not translate to an equivalent risk reduction level. However, in the region where the percent reduction in mean risk line is above the percent reduction in concentration line, reductions in concentration translate to a greater than equivalent percent risk reduction.

Figure 9 helps to explain the results for this scenario by presenting the information in a slightly different format. In the figure, there are four panels labelled A to D. Each of the four panels has two bars that reflect the initial and final concentration (on the log-scale at left) before and after an intervention strategy, and two points that show the corresponding percent reduction in concentration and the resulting reduction in risk (using the scale to the right). Panel A indicates that when the level of contamination is approximately 6 log<sub>10</sub> cfu, and is reduced by 44%, the risk is reduced by approximately 11%. When the level of contamination on the chickens is lower to start with, the subsequent additional reduction in risk begins to increase. This can be seen in panels B, C and D. In panel B, with a starting contamination level of 3 log<sub>10</sub> cfu, the risk reduction for a 44% concentration reduction is 46%. The risk is reduced by 69% for a 44% concentration reduction when the contamination level is just greater than 2 log<sub>10</sub> cfu (panel C) and 82% when the initial contamination level is approximately 2 log<sub>10</sub> cfu (panel D).

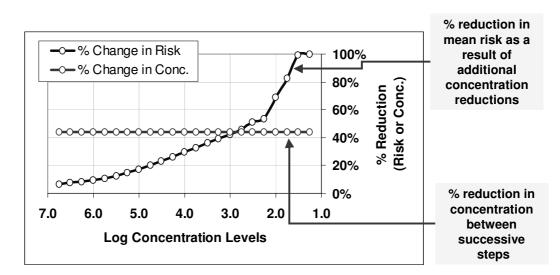


Figure 8. Percent reduction in risk corresponding to percent reduction in concentration for various concentration levels.

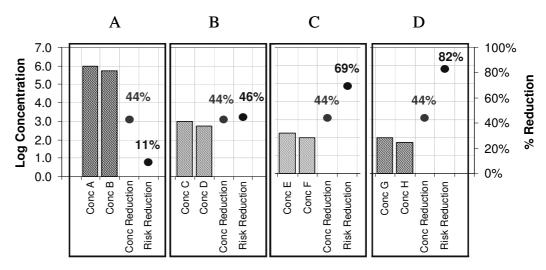


Figure 9. Four representative illustrations of the effect of reducing the contamination level on the reduction in risk.

Scenarios 1 and 2 can be summarized as follows: Any management strategy that alters the prevalence of contaminated chicken at retail would be estimated to have a correspondingly proportional impact on the mean risk. A management strategy that alters the level of contamination has a non-linear relationship (i.e. with significantly varying returns on investment) depending upon the starting level of contamination. If the level of contamination is high, small additional reductions will have only a small effect. If the mean level of contamination is lower, additional contamination level reductions will have a greater than proportional effect on risk. From a management perspective, it will be important to know where

in this continuum the system is located prior to selecting an appropriate strategy. If the level of contamination is reasonably high, reducing the frequency with which chickens are contaminated may yield a relatively greater return on investment. It might be more beneficial in this case to try to reduce the actual number of contaminated chickens entering the system or going to market. However, where the level of contamination is reasonably low, reducing the frequency of contamination may turn out to be less effective than reducing the level of contamination further.

#### 5.3 Scenario 3: Changing between-flock and within-flock prevalence

The third scenario investigated the effect of altering the "between-flock" and "within-flock" prevalences. The between-flock prevalence refers to the prevalence of contaminated flocks at the farm. Altering the between-flock prevalence could be achieved, for instance, by the use of colonization-resistant breeds, if this were a technically feasible option, or through improved biosecurity where that is considered a key determinant of flock contamination.

The within-flock prevalence refers to the prevalence of contaminated birds within a contaminated flock. The within-flock prevalence is usually assumed to eventually converge toward 100% since the contamination will eventually spread throughout the contaminated flock. However, the within-flock prevalence could be less than 100% at the time of slaughter if, for example, biosecurity measures are implemented that delay the introduction of contamination as long as possible. In this case, insufficient contact time among birds occurs, thereby preventing spread of contamination to some fraction of the birds.

To gain insight into the effect of within-flock and between-flock prevalence on the health risk outcome, three scenarios were investigated and compared to a baseline level. Apart from the changes in between-flock and within-flock prevalence, the other parameters of the model were held constant to the baseline situation between simulations. The model was simulated for 10 000 iterations at each scenario and the results are shown in Figure 10. The three scenarios simulated were:

Baseline:	80% between-flock prevalence	100% within-flock prevalence
a 1	10 01 1 1	1000 111 01 1 1

- Strategy 1: 40% between-flock prevalence 100% within-flock prevalence
- Strategy 2: 80% between-flock prevalence 50% within-flock prevalence
- Strategy 3: 40% between-flock prevalence 50% within-flock prevalence

The results presented in Figure 10 are not extremely surprising, indicating trends that we might expect to see. There are some interesting observations that can be made, based upon the results produced, and the mechanisms underlying the process. First, the effect of reducing between-flock prevalence at the farm (Strategy 1) appears to translate to a slightly greater than one-to-one (a 50% between-flock reduction is estimated to produce greater than 50% risk reduction) relationship in risk reduction. Specifically, when the between-flock prevalence is reduced, not only is the probability of a bird from a contaminated flock reaching the consumer reduced, but the probability of a contaminated flock being processed prior to the current one is also reduced, thereby reducing the probability of inter-flock cross-contamination during transport or processing.

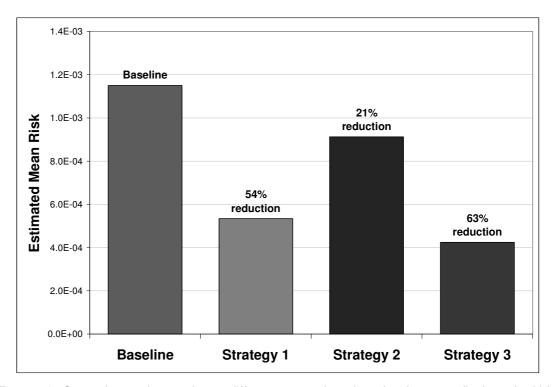


Figure 10. Scenario results: testing 3 different strategies changing between-flock and withinflock prevalence.

Strategy 2 focused on the reduction of within-flock prevalence, while maintaining the overall between-flock prevalence. In this case the assumption is that those flocks that would have been positive are managed in such a way that the number of birds within the positive flock is kept to some value less than 100%. In the current scenario this is set to a value of 50% (half the birds in a positive flock are positive while half are not). The end result of this strategy translates to approximately a 20% reduction in risk. The reduced effectiveness of this strategy is primarily due to the fact that the negative birds from these flocks are being processed in an environment in which they are surrounded by positive birds and thus subject to high probabilities of cross-contamination. As a result, a substantial reduction in the within-flock prevalence would be required (thereby having a substantial impact on cross-contamination probabilities) in order to produce risk reductions of equivalent magnitude.

The final strategy simply illustrates the synergic nature of the individual scenarios. A reduction in both within-flock and between-flock prevalence translates to a greater effect than either of the other two individually. This type of combined reduction (affecting between- and within-flock prevalence) may be a result of biosecurity measures that eliminate the contamination event for some flocks (i.e. reducing the between-flock prevalence), while limiting or otherwise delaying the contamination extent in other flocks (and thereby reducing the within-flock prevalence in positive flocks).

# 5.4 Scenario 4: Changing internal and surface contamination levels before and through processing

The fourth scenario comprised several alternative strategies to reflect a variety of potential changes in the level of contamination before and through processing. The four alternative strategies investigated were:

Strategy 1:	90% reduction in surface contamination level after transport	
Strategy 2:	90% reduction in levels contaminating carcasses at evisceration	
Strategy 3:	90% reduction in surface contamination post-evisceration	
Strategy 4:	90% reduction in initial internal contamination levels (overall reduction in contamination entering the system)	

Strategy 1 is a reduction in the level of surface contamination after transport. In this strategy, there is no reduction in the internal contamination levels. The strategy is such that, after transport, a process is implemented that reduces the amount of contamination on the surface of the birds by 90%. In Strategy 2, the level that contaminates carcasses at evisceration is reduced by 90%. This strategy assumes that the evisceration process is modified, either through new technology, or through some other change in the process, such that the amount of contamination from internal damage or from cross-contamination is reduced by 90%. Strategy 3 can be interpreted to mean that after evisceration, there is a step or process introduced that reduces the level of contamination by 90%. Strategy 3 is similar to strategy 1, but while strategy 1 was done after transport, strategy 3 is conducted further along in the process, after evisceration. Finally, strategy 4 is assumed to reduce the overall contamination level entering the system. In essence, the scenario can be interpreted to be one in which a strategy is employed that affects the levels at which birds are colonized internally at the farm level.

The results of the scenario, shown in Figure 11, with a baseline mean risk level for comparison, show that the impact of altering the contamination level can be quite varied.

In the first strategy, the impact of reducing the contamination level after transport is not as dramatic as some of the other strategies. The 35% reduction in risk is significant, but it is less significant than some of the others because, when simply reducing the contamination level on the surface at this early point in the process, it is possible for contamination through cross-contamination to occur (as a result of viscera damage later in the process). This later contamination essentially undermines the reductions achieved earlier in the process.

The second strategy produced the least significant risk reduction of the four strategies tested. However, the strategy was designed to explore the impact of reducing the level of contamination occurring solely during the evisceration activity. So, a strategy that reduced the amount of contamination coming from the viscera during processing by 90% would reduce the overall risk by 25%. This partly reflects the limited number of organisms on the final carcass that came from viscera during processing, when compared with those which contaminate the external surfaces of carcasses at receipt for slaughter.

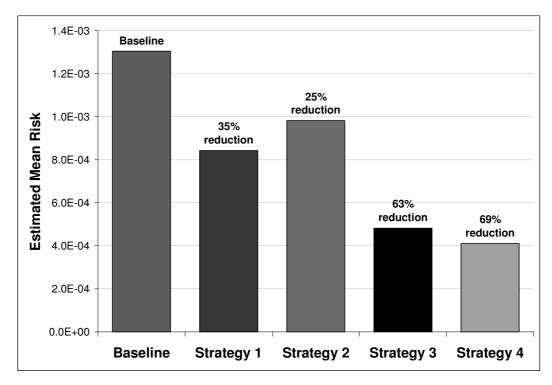


Figure 11. Scenario results: testing strategies that alter the level of contamination

The effect of reducing surface contamination after evisceration, as depicted in Strategy 3, is estimated to have a more significant effect. A 90% reduction in the surface contamination level after evisceration translates to a 63% reduction in the mean risk overall. Unlike the first strategy, in which the surface contamination after transport was reduced by 90%, for Strategy 3 the reduction occurs at evisceration, with fewer subsequent contamination or recontamination stages after that step presenting fewer opportunities to undermine the strategy.

Finally, Strategy 4 reflects an overall reduction in contamination both internally and externally as a result of getting transferred onto the surface of the bird. This strategy could be interpreted to be one that targets the internal contamination level of chickens at the farm level and thus the overall contamination level entering the system. The key to this strategy, that is important for the management of *Campylobacter* in general, is that in order to have the greatest relative impact on the risk by controlling contamination levels, it is essential that either the overall contamination entering the system is reduced, or that a step is introduced late in the process, which decreases the contamination levels. Individual surface contamination reduction steps conducted early in the process will tend to be undermined by subsequent steps in the process.

#### 5.5 Scenario 5: Risk mitigation impacts from freezing of fresh chicken.

The fifth scenario investigates the effects of freezing as a risk mitigation strategy. In this scenario, comparisons were made between fresh product stored refrigerated for up to 9 days, and product held frozen prior to consumption for up to 6 weeks. The risk associated with each

of these strategies was estimated for a range of contamination levels so that the effects of each strategy could be measured as a function of the concentration. The results of this scenario are shown in Figure 12.

Freezing has been found to have an inactivation effect on *Campylobacter* reducing the level of contamination. As a result, it is estimated that the chickens that are frozen have a lower risk than those that are sold and stored refrigerated. From Figure 12, it can be seen that an equivalent level of risk can be maintained for more heavily contaminated product that is frozen in comparison with a less contaminated product that is held under refrigeration. For instance, the mean risk for refrigerated chicken at a mean contamination level of  $4.5 \log_{10}$  cfu is approximately the same as for frozen chicken at a mean contamination level of approximately 5.25 log<sub>10</sub> cfu at the time of freezing. While the results in general are expected, the scenario analysis facilitates at least an approximate quantification of the magnitude of the impact of freezing.

The second part of the current scenario was to explore in more detail the potential complications that could arise as a result of a freezing strategy. The scenario explores a mechanism whereby the consumers' subsequent preparation practices could have a significant impact on the effectiveness of a risk-reduction strategy associated with freezing. The specific mechanism explored is where a product that is frozen experiences *reduced cooking effectiveness* due to insufficient thawing, resulting in a lower final cooking temperature being attained in parts of the chicken. Two scenarios of diminished cooking effectiveness, due to freezing, were explored:

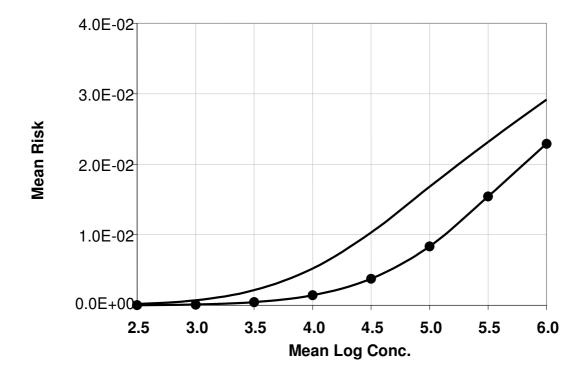
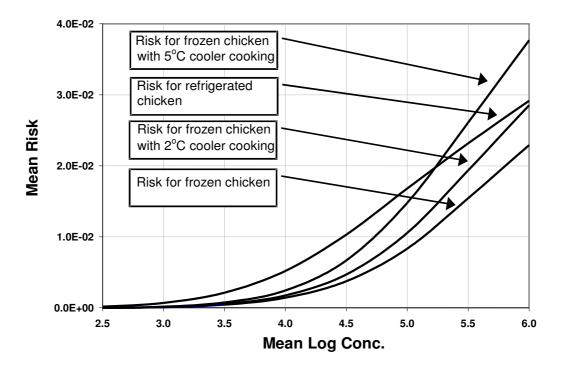


Figure 12. Risk estimates for frozen (-•-) and refrigerated (----) chicken as a function of contamination level.

- Effect 1: Final cooking temperature is 2°C cooler in cold spots for frozen chicken.
- Effect 2: Final cooking temperature is 5°C cooler in cold spots for frozen chicken

Figure 13 shows the results of the original part of the scenario together with the modifications to the scenario that are based on reduced cooking effectiveness. When the effect of freezing is assumed to lower the cooking effectiveness by reducing the final cooking temperature in the cooler spots of the chicken by 2°C, the relative benefit of freezing begins to diminish. As the level of contamination increases, the balance between freezing reductions and impact on cooking effectiveness begins to diminish. At a mean contamination level of approximately 6  $\log_{10}$  cfu, the risk from fresh refrigerated chicken is estimated to be the same as that of frozen chicken subjected to lower cooking effectiveness. When the effect of freezing reduces the temperature in the cooler spots of the chicken by  $5^{\circ}$ C, the mean risk is estimated to start out lower. As the contamination level increases, the risk also increases, until eventually it is estimated that the risk from frozen chicken could in fact turn out to be greater than the risk from refrigerated chicken. In other words, in this scenario, the reduced likelihood of survival of organisms in frozen product is counterbalanced by their increased likelihood of survival during cooking. When the level of contamination on chicken is greater than approximately  $5.25 \log_{10}$  cfu, the risk from frozen chicken exceeds that of fresh-refrigerated chicken when a significant cooking effect from freezing is included. This scenario does not provide a definitive conclusion regarding the impact of freezing. It does however demonstrate the complexity of the interactions that can occur in suggesting changes to the ways in which the product is stored.



**Figure 13.** Potential complications in risk reduction as a result of pursuing a freezing strategy, as a function of contamination level.

## 6. SUMMARY AND KEY FINDINGS

This risk assessment has been prepared in response to a request for advice from the Codex Committee on Food Hygiene (CCFH). The risk assessment has adopted the standard terminology of the Codex Alimentarius Commission guidelines for microbiological risk assessment. It is structured in the stages of hazard identification, exposure assessment, hazard characterization, and risk characterization. An overall schematic of the risk assessment model is provided in Figure 2.

The risk assessment consists of a technical report entitled "A Risk Assessment of *Campylobacter* spp. in Broiler Chickens" and a computational model implemented in Microsoft Excel and @Risk (a spreadsheet 'add-in' used to implement Monte Carlo simulation algorithms). This document is a summary of the technical report.

The risk assessment model is a mathematical description (including Monte Carlo simulation) of a number of phenomena that, through a complex web of interactions, contribute to the level of risk associated with consuming broiler chicken products contaminated with *Campylobacter*. This assessment is a result of merging models developed prior to this assessment in Canada, the United Kingdom and Denmark. These risk assessments were carried out for different reasons and their individual structures and emphases varied accordingly.

#### 6.1 Model Use and Adaptation

The authors do not advocate the exact structure of the model as the *preferred* model for all decisions. The questions posed by CCFH were sufficiently vague that the process was best served by the development of a model that contained the major elements from the three 'parent' risk assessments, but did not focus on detailed measurement of the risk reduction impact of any specific risk management strategy.

In some cases, the model elements are quite complex, while in others they are relatively simple. These model elements could be customized or replaced by other elements as required for a particular decision context. As an example, the model contains a module that provides detailed computation of the spread of *Campylobacter* colonization within a commercial broiler flock. This module, and other complex elements in the model, can be customized to a particular situation or, with due consideration, replaced by a much simplified model element that serves the same functional role in the simulation (e.g. employing a single fixed number, such as 80%, as an estimate of the prevalence of *Campylobacter* within positive flocks). In a similar fashion, the very simple current module for exposure via cross-contamination in the home could be replaced by an alternative simple module, or a highly complex sub-model that characterizes cross-contamination behaviours, pathways and frequencies in considerable detail, if the user had access to the data and expertise required to establish such a cross-contamination model.

The risk assessment model does not describe any particular closed system (e.g. the production and consumption of poultry in a specific country). Rather, it provides a series of generic production, processing and handling elements that could be adapted or customized to describe a variety of different situations. The model is best viewed as providing a model infrastructure that can be applied by FAO and WHO member countries, including CCFH risk

management working groups, to support a wide variety of risk-management decisions. This is evident from the breadth and generality of the scenarios that were analysed as part of the risk characterization.

At present, the model can be used in two distinct modes. The first mode is the generation of insight into the complex mechanics of the propagation of the risk of campylobacteriosis through the production, processing and consumer handling subsystems. The development of this insight might be considered a prerequisite to risk-management strategy development. Risk assessors and risk managers could employ the model as a test bed for ideas on how the system might be managed and how the system reacts to various changes, intentional or otherwise. The use of the model to generate insight into the drivers of risk is limited only by the imagination and resources of the user in adapting and applying the model to uncover new relationships.

The second mode of use would be to characterize very specific risk-mitigation options. The infrastructure to do this is available in the current model. Strategies designed to affect any number of model elements can be evaluated quantitatively by comparing the baseline scenario with the risk estimates generated by including the changed model elements caused by the risk-management options. Examples of elements that might be modified by risk-management strategies include: between-flock prevalence, within-flock prevalence, surface contamination during transport, decontamination through scalding, inactivation effectiveness in chilling water, home storage, or cooking behaviours.

As an example, if a risk manager were to impose a requirement for the freezing of any product that comes from a flock that has tested positive, the impact of this policy could be measured in comparison with a baseline where freezing of broilers is not related to the status of the flock. An additional model element could be added to take into account the sensitivity of the flock testing or other relevant parameters. Accordingly, the logic that imposes the freezing requirement on test-positive lots would need to be added to the risk assessment. As a result of such adaptations, the model would gradually accumulate a richer description of implemented risk-management options.

#### 6.2 Key findings

The risk assessment provides a tool for discovery and measurement of complex relationships determining the risk of illness upon preparation and consumption of chicken products contaminated by *Campylobacter jejuni*. The five scenarios described above and in more detail in the main report constitute a sample of a large array of potential scenarios that can be studied and evaluated. These scenarios led to the following findings:

- Reduction in retail prevalence of positive chicken products has a roughly proportional effect in risk reduction.
- Reduction in the contamination level of positive chicken products has a somewhat more complex relationship with the estimate of risk. For highly contaminated products, moderate reductions in the contamination level have relatively mild effects. As the contamination level is further reduced, further reductions have increasing relative impact and eventually yield significant relative risk reductions.
- Between-flock prevalence is roughly proportional to the risk of illness. The presence of cross-contamination between flocks complicates this slightly due to the risk that

negative flocks become contaminated by positive flocks during transport and in the slaughter plant.

- Reduction in within-flock prevalence clearly reduces the overall estimate of risk, but with a less-than-proportional rate due to the presence of cross-contamination in the slaughter process, which increases the within-flock prevalence for carcasses during processing.
- A number of scenarios were compared wherein the contamination levels in the processing environment were reduced. The analysis indicates the greatest benefit from reduced total load of *Campylobacter* in the intestinal tract of birds (thereby reducing the total load on the system). In addition, the benefits of reductions in levels of contamination that take place early in the processing stages can be undermined by cross-contaminating processes later in the processing environment.
- Freezing of poultry will inactivate *Campylobacter* slowly over time. This has been suggested and implemented as a risk mitigation measure, particularly for positive flocks, in some countries. The scenario includes the potential that freezing may present countervailing risks through reduced effectiveness of cooking. An example is provided where the net effect of freezing is to increase risk when both the reduction in numbers and the reduction in the effect of cooking are considered. This is an example of the complex relationships that can exist and which can be examined using such models to facilitate more informed risk-management decision-making.

These findings represent a sample of the analyses that countries might conduct by adapting the modelling approaches conducted in this assessment.

## 7. REFERENCES

- Aho, M. & Hirn, J. 1998. Prevalence of campylobacteria in the Finnish broiler chicken chain from producer to the consumer. *Acta Veterinaria Scandinavica*, 29: 451–462.
- Altekruse, S.F., Stern, N.J., Fields, P.I. & Swerdlow, D.L. 1999. *Campylobacter jejuni* An emerging foodborne pathogen. *Emerging Infectious Diseases*, 5: 28–35.
- Anon[ymous]. 1998. Trends and sources of zoonotic agents in animals, feeding stuff, food and man in the European Union in 1996. Part 1. Community Reference Laboratory on the Epidemiology of Zoonoses, BgVV, Berlin, Germany.
- Anon. 1999. Trends and sources of zoonotic agents in animals, feedstuffs, food and man in the European Union in 1997. Part 1. Document No. VI/8495/98 – Rev. 2 of the European Commission, Community Reference Laboratory on the Epidemiology of Zoonoses, BgVV, Berlin, Germany.
- Anon. 2001a. Trends and sources of zoonotic agents in animals, feeding stuff, food and man in the European Union and Norway in 1999. Part 1. Document No. SANCO/1069/2001 of the European Commission, Community Reference Laboratory on the Epidemiology of Zoonoses, BgVV, Berlin, Germany.
- Anon. 2001b. Annual report on zoonoses in Denmark, 2000. Ministry of Food, Agriculture and Fisheries, Denmark.
- Anon. 2001c. National surveillance data November 2000. *New Zealand Public Health Report*, 8(1): 6. Provided in response to the FAO/WHO call for data.
- Anon. 2001d. Data available on *Campylobacter* in Finland. University of Helsinki, Faculty of Veterinary Medicine, National Institute of Food Research Institute. Provided in response to the FAO/WHO call for data.
- Anon. 2001e. Campylobacter data from Hungary. Provided in response to the FAO/WHO call for data.
- Anon. 2001f. Notifiable and other diseases in New Zealand. Annual Report 2000. Population and Environmental Health Group, Institute of Environmental Science and Research Limited, New Zealand.
- Anon. 2001g. Food Safety Authority of Ireland Annual Report 2000. Food Safety Authority of Ireland, Dublin, Ireland.
- Anon. 2002. Food Safety Authority of Ireland Annual Report 2001. Food Safety Authority of Ireland, Dublin, Ireland.
- Anon. 2003. Food Safety Authority of Ireland Annual Report 2002. Food Safety Authority of Ireland, Dublin, Ireland.
- Anon. 2004. Food Safety Authority of Ireland Annual Report 2003. Food Safety Authority of Ireland, Dublin, Ireland.
- Anon. 2005a. Enter-net annual report 2004; surveillance of enteric pathogens in Europe and beyond. Enter-net surveillance hub, HPA, Centre for Infections, Colindale, London, UK.
- Anon. 2005b. Food Safety Authority of Ireland Annual Report 2004. Food Safety Authority of Ireland, Dublin, Ireland.
- Anon. 2006a. Notifiable and other diseases in New Zealand. Annual Report 2005. Population and Environmental Health Group, Institute of Environmental Science and Research Limited, New Zealand.
- Anon. 2006b. Trend and sources of zoonoses, zoonotic agents and antimicrobial resistance in the European Union in 2004. European Food Safety Authority, Italy.
- Benes, C. 2001. National Institute of Public Health, Centre of Epidemiology and Microbiology, Czech Republic. Provided in response to the FAO/WHO call for data.
- Black, R.E., Levine, M.M., Clements, M.L., Hughes, T.P. & Blaser, M. 1998. Experimental *Campylobacter jejuni* infection in humans. *Journal of Infectious Diseases*, 157: 472–479.

- Blaser, M.J. & Reller, L.B. 1981. Campylobacter enteritis. New England Journal of Medicine, 305: 1444–1452.
- Blaser, M.J., Taylor, D.N. & Feldman, R.A. 1983. Epidemiology of *Campylobacter jejuni* infections. *Epidemiologic Reviews*, 5: 157–176.
- Bodhidatta, L., Vithayasai N., Eimpokalarp B., Pitarangsi C., Serichantalergs O. & Isenbarger D.W. 2002. Bacterial enteric pathogens in children with acute dysentery in Thailand: increasing importance of quinolone-resistant *Campylobacter*. *Southeast Asian Journal of Tropical Medicine and Public Health*, 33: 752–757.
- Boes, J., Nersting J., Nielsen E.M., Kranker S., Enoe C., Wachmann H.C. & Baggesen D.L. 2005. Prevalence and diversity of *Campylobacter jejuni* in pig herds on farms with and without cattle or poultry. *Journal of Food Protection*, 68: 722–727.
- Calva, J.J., Ruiz-Palacios, G.M., Lopez-Vidal, A.B. & Bojahil, R. 1988. Cohort study of intestinal infection with *Campylobacter* in Mexican children. *Lancet*, 1: 503–506.
- CDC-FoodNet Suveillance results. No date. Centers for Disease Control and Prevention, National Center for Infectious Diseases, Division of Bacterial and Mycitic Diseases, Foodborne and Diarrheal Diseases Branch, http://www.cdc.gov/ncidod/dbmd/foodnet/
- Coker, A.O., Isokpehi, R.D., Thomas, B.N., Amisu, K.O. & Obi, C.L. 2002. Human campylobacteriosis in developing countries. *Emerging Infectious Diseases*, 8: 237–244.
- Deming, M.S., Tauxe, R.V., Blake, P.A., Blake, S.E., Dixon, S.E., Fowler, B.S., Jones, T.S., Lockamy, E.A., Patton, C.M., Sikes, R.O. 1987. *Campylobacter* enteritis at a university from eating chickens and from cats. *American Journal of Epidemiology*, 126: 526–534.
- Eberhart-Phillips, J., Walker, N., Garrett, N., Bell, D., Sinclair, D., Rainger, W. & Bates, M. 1997. Campylobacteriosis in New Zealand: results of a case-control study. *Journal of Epidemiology and Community Health*, 51: 686–691.
- Effler, P., Ieong, M.-C., Kimura, A., Nakata, M., Burr, R., Cremer, E. & Slutsker, L. 2001. Sporadic *Campylobacter* infections in Hawaii: Associations with prior antibiotic use and commercially prepared chicken. *Journal of Infectious Disease*, 183: 1152–1155.
- Ethelberg, S., Olsen, K.E.P., Gerner-Smidt, P. & Molbak, K. 2004. Household outbreaks among cultureconfirmed cases of bacterial gastrointestinal disease. *American Journal of Epidemiology*, 159: 406–412.
- Fazil, A., Lowman, R., Stern, N. & Lammerding, A. 2000. A quantitative risk assessment model for *Campylobacter jejuni* in fresh chicken. Provided in response to the FAO/WHO call for data.
- Friedman, C.R., Neimann, J, Wegener, H.C. & Tauxe, R.V. 2000. Epidemiology of *Campylobacter jejuni* infections in the United States and other industrialized nations. pp. 121–138, *in:* I. Nachamkin and M.J. Blaser (editors). *Campylobacter*. American Society for Microbiology, Washington, DC, USA.
- Friedman, C.R., Hoekstra, R.M., Samuel, M., Marcus, R., Bender, J., Shiferaw, B., Reddy, S., Ahuja, S.D., Helfrick, D.L., Hardnett, F., Carter, M., Anderson, B. & Tauxe, R.V. 2004. Risk factors for sporadic *Campylobacter* infection in the United States: A case-control study in FoodNet sites. *Clinical Infectious Diseases*, 38(Suppl. 3): S285-296.
- Georges-Courbot, M.C., Baya, C., Beraud, A.M., Meunier, D.M.Y. & Georges, A.J. 1986. Distribution and serotypes of *Campylobacter jejuni* and *Campylobacter coli* in enteric *Campylobacter* strains isolated from children in the Central African Republic. *Journal of Clinical Microbiology*, 23: 592–594.
- Georges-Courbot, M.C., Cassel-Beraud, A.M., Gouandjika, I., Monges, J. & Georges, A.J. 1990. A cohort study of enteric *Campylobacter* infection in children from birth to two years in Bangui (Central African Republic). *Transactions of the Royal Society of Tropical Medicine and Hygiene*, 84: 122–125.
- Georgsson, F. 2001. Personal communication. Provided in response to the FAO/WHO call for data.
- Harris, N.V., Weiss, N.S. & Nolan, C.M. 1986. The role of poultry and meats in the etiology of *Campylobacter jejuni/coli* enteritis. *American Journal of Public Health*, 76: 407–411.
- Hartnett, E., Kelly, L., Newell, D., Wooldridge, M. & Gettinby, G. 2001. A quantitative risk assessment for the occurrence of *Campylobacter* in chickens at slaughter. *Epidemiology and Infection*, 127(2): 195–206.

- Harvey, S.A., Winch, P.J., Leontsini, E., Torres Gayoso, C., Lopez Romero, S., Gilman, R.H. & Oberhelman, R.A. 2003. Domestic poultry-raising practices in a Peruvian shantytown: implications for control of *Campylobacter jejuni*-associated diarrhea. *Acta Tropica*, 86: 41–54.
- Hopkins, R.S. & Scott, A.S. 1983. Handling raw chicken as a source for sporadic *Campylobacter jejuni* infections. *Journal of Infectious Diseases*, 148: 770.
- Hopkins, R.S., Olmsted, R. & Istre, G.R. 1984. Endemic Campylobacter jejuni infection in Colorado: identified risk factors. American Journal of Public Health, 74: 249–250.
- ICMSF. 1996. *Micro-organisms in Foods V. Characteristics of Microbial Pathogens*. Blackie Academic & Professional, London, UK. pp. 45–65.
- Ikram, R., Chambers, S., Mitchell, P., Brieseman, M.A. & Ikam, O.H. 1994. A case control study to determine risk factors for *Campylobacter* infection in Christchurch in the summer of 1992–3. *New Zealand Medical Journal*, 107: 430–432.
- Jain, D., Sinha, S., Prasad, K.N. & Pandey, C.M. 2005. Campylobacter species and drug resistance in a north Indian rural community. Transactions of the Royal Society for Tropical Medicine and Hygiene, 99: 207– 214.
- Jensen, A.N., Dalsgaard, A., Baggesen, D.L. & Nielsen, E.M. 2006. The occurrence and characterization of *Campylobacter jejuni* and *C. coli* in organic pigs and their outdoor environment. *Veterinary Microbiology*, 116: 96–105.
- Kapperud, G. 1994. Campylobacter infections. Epidemiology, risk factors and preventive measures (in Norwegian). Tidskr. Nor. Lægeforen., 114: 795–799.
- Kapperud, G., Skjerve, E., Bean, N.H., Ostroff, S.M. & Lassen, J. 1992. Risk factors for sporadic *Campylobacter* infections: Results of a case-control study in Southeastern Norway. *Journal of Clinical Microbiology*, 30: 3117–3121.
- Kapperud, G., Espeland, G., Wahl, E., Walde, A., Herikstad, H., Gustavsen, S., Tveit, I., Natas, O., Bevanger, L. & Digranes, A. 2003. Factors associated with increased and decreased risk of *Campylobacter* infection: a prospective case-control study in Norway. *American Journal of Epidemiology*, 158: 234–242.
- Koidis, P. & Doyle, M.P. 1983. Survival of *Campylobacter jejuni* in fresh and heated red meat. *Journal of Food Protection*, 46(9): 771–774.
- Koulla-Shiro, S., Loe, C. & Ekoe, T. 1995. Prevalence of *Campylobacter* enteritis in children from Yaounde (Cameroon). *Central African Journal of Medicine*, 41: 91–94.
- Kruse, H. 2001. *Campylobacter* and campylobacteriosis in Norway. Provided in response to the FAO/WHO call for data.
- Mead, P.S., Slutsker, L., Dietz, V., McCaig, L.F., Bresee, J.S., Shapiro, C., Griffin, P.M. & Tauxe, R.V. 1999. Food-related illness and death in the United States. *Emerging Infectious Diseases*, 5: 607–625
- Medema, G.J., Teunis, P.F.M., Havelaar, A.H. & Haas, C.N. 1996. Assessment of the dose-response relationship of *Campylobacter jejuni*. *International Journal of Food Microbiology*, 30: 101–111.
- Megraud, F., Boudraa, G., Bessaoud, K., Bensid, S., Dabis, F., Soltana, R. & Touhami, M. 1990. Incidence of *Campylobacter* infection in infants in western Algeria and the possible protective role of breast feeding. *Epidemiology and Infection*, 105: 73–78.
- Nadeau, E., Messier, S.& Quessy, S. 2001. Prevalence and comparison of genetic profiles of thermotolerant *Campylobacter* strains isolated from poultry and sporadic cases of campylobacteriosis in humans. Provided in response to the FAO/WHO call for data.
- Neimann, J., Engberg, J., Molbak, K. & Wegener, H.C. 2003. A case-control study of risk factors for sporadic campylobacter infections in Denmark. *Epidemiology and Infection*, 130: 353–366.
- Newell, D.G. 1982. *Campylobacter* enteritis: epidemiology and clinical data from recent isolations in the region of Freiburg, West Germany. MTP Press, Lancaster.

- Nielsen, E.M., Engberg, J. & Madsen, M. 1997. Distribution of serotypes of *Campylobacter jejuni* and *Campylobacter coli* from Danish patients, poultry, cattle, and swine. *FEMS Immunology and Medical Microbiology*, 19: 47–56.
- Oosterom, J., den Uyl, C.H., Bänffer, J.R.J. & Huisman, J. 1984. Epidemiological investigations on *Campylobacter jejuni* in households with primary infection. *Journal of Hygiene (London)*, 92: 325–332.
- Rosef, O., Gondrosen, B., Kapperud, G. & Underdal, B. 1983. Isolation and characterization of *Campylobacter jejuni* and *Campylobacter coli* from domestic and wild animals in Norway. *Applied Environmental Microbiology*, 46: 855–859.
- Samuel, M.C., Vugia, D.J., Shallow, S., Marcus, R., Segler, S., McGivern, T., Kassenborg, H., Reilly, K, Kennedy, M., Angulo, F. & Tauxe, R.V. 2004. Epidemiology of sporadic *Campylobacter* infection in the United States and declining trend in incidence, FoodNet 1996–1999. *Clinical Infectious Diseases*, 38 (Suppl 3): S165–S174.
- Schmid, G.P., Schaefer, R.E., Plikaytis, B.D., Schaefer, J.R., Bryner, J.H., Wintermeyer, L.A. & Kaufmann, A.F. 1987. A one-year study of endemic campylobacteriosis in a midwestern city: association with consumption of raw milk. *Journal of Infectious Diseases*, 156: 218–222.
- Simango, C. & Nyahanana, M. 1997. *Campylobacter* enteritis in children in an urban community. *Central African Journal of Medicine*, 43: 172–175.
- Skirrow, M.B. 1991. Epidemiology of *Campylobacter* enteritis. *International Journal of Food Microbiology*, 12: 9–16.
- Solow, B.T., Cloak, O.M. & Fratamico, P.M. 2003. Effect of temperature on viability of *Campylobacter jejuni* and *Campylobacter coli* on raw chicken or pork skin. *Journal of Food Protection*, 66: 2023–2031.
- Studahl, A. & Andersson, Y. 2000. Risk factors for indigenous *campylobacter* infection: a Swedish case-control study. *Epidemiology and Infection*, 125: 269–275.
- Tauxe, R.V. 1992. Epidemiology of *Campylobacter jejuni* infections in the United States and other industrialized nations. pp. 9–19, *in:* I. Nachamkin, M.J. Blaser and L.S. Tompkins (editors). *Campylobacter jejuni*: Current status and future trends. American Society for Microbiology, Washington DC, USA.
- Teunis. P.F.M., van der Heijden, O.G., van der Giessen, J.W.B. & Havelaar, A.H. 1996. The dose-response relation in human volunteers for gastro-intestinal pathogens. RIVM report # 284550002.
- Teunis, P.F.M. & Havelaar, A.H. 2000. The beta Poisson dose-response model is not a single hit model. *Risk Analysis*, 20: 513–520.
- Wheeler, J.G., Sethi, D., Cowden, J.M., Wall, P.G., Rodrigues, L.C., Tompkins, D.S., Hudson, M.J. & Roderick, P.J. 1999. Study of infectious intestinal disease in England: rates in the community, presenting to general practice and reported to national surveillance. *British Medical Journal*, 318: 1046–1050.
- WHO [World Health Organization]. 2001. The increasing incidence of human campylobacteriosis. Report and proceedings of a WHO consultation of experts, Copenhagen, Denmark, 21–25 November 2000.
- Wingstrand, A., Neimann, J., Engberg, J., Nielsen, E.M., Gerner-Smidt, P., Wegener, H.C. & Molbak, K. 2006. Fresh chicken as main risk factor for campylobacteriosis, Denmark. *Emerging Infectious Diseases*, 12: 280–285.
- Wooldridge, K.G. & Ketley, J.M. 1997. *Campylobacter* host cell interactions. *Trends* in *Microbiology*, 5: 96–102.

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