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Quantitative risk assessment of human campylobacteriosis associated with thermophilic *Campylobacter* species in chickens

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Abstract

A quantitative risk assessment comprising the elements hazard identification, hazard characterization, exposure assessment, and risk characterization has been prepared to assess the effect of different mitigation strategies on the number of human cases in Denmark associated with thermophilic *Campylobacter* spp. in chickens. To estimate the human exposure to *Campylobacter* from a chicken meal and the number of human cases associated with this exposure, a mathematical risk model was developed. The model details the spread and transfer of *Campylobacter* in chickens from slaughter to consumption and the relationship between ingested dose and the probability of developing campylobacteriosis. Human exposure was estimated in two successive mathematical modules. Module 1 addresses changes in prevalence and numbers of *Campylobacter* on chicken carcasses throughout the processing steps of a slaughterhouse. Module 2 covers the transfer of *Campylobacter* during food handling in private kitchens. The age and sex of consumers were included in this module to introduce variable hygiene levels during food preparation and variable sizes and compositions of meals. Finally, the outcome of the exposure assessment modules was integrated with a Beta–Poisson dose–response model to provide a risk estimate. Simulations designed to predict the effect of different mitigation strategies showed that the incidence of campylobacteriosis associated with consumption of chicken meals could be reduced 30 times by introducing a 2 log reduction of the number of *Campylobacter* on the chicken carcasses. To obtain a similar reduction of the incidence, the flock prevalence should be reduced approximately 30 times or the kitchen hygiene improved approximately 30 times. Cross-contamination from positive to negative flocks during slaughter had almost no effect on the human *Campylobacter* incidence, which indicates that implementation of logistic slaughter will only have a minor influence on the risk. Finally, the simulations showed that people in the age of 18–29 years had the highest risk of developing campylobacteriosis.

Keywords: Quantitative risk assessment; Risk; Monte Carlo simulation; *Campylobacter*; *Campylobacter jejuni*; Chickens; Mitigations

1. Introduction

The Food Safety Risk Analysis used as a tool for the control of biological hazards in foods is becoming internationally accepted. The context was described in

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a report from the FAO/WHO in 1995 (FAO/WHO, 1995). This report states that 'It should be the role of official bodies to use risk analysis to determine realistic and achievable risk levels for food-borne hazards and to base food safety policies on the practical application of the results of these analyses'. Further on, with the implementation of the Agreement on the Application of Sanitary and Phytosanitary Measures (the SPS agreement) (WTO, 1994) an international trade agreement for the first time explicitly recognises that for establishment of rational harmonised regulations and standards for food in international trade, a rigorous scientific process is required. Finally, it has been stated that the elements of the Food Safety Risk Analysis: Risk Management, Risk Assessment, and Risk Communication (FAO/WHO, 1995, 1997) should form the basis when governmental agencies and the industry are establishing Food Safety Objectives (ICMSF, 1998).

In 1997, as a consequence of an increase in the number of registered human enteric infections in Denmark (Anonymous, 1995, 1996, 1997), risk managers at the Danish Veterinary and Food Administration decided to initiate a strategy for the control of pathogenic microorganisms in foods based on the principles of Food Safety Risk Analysis (Anonymous, 1999b). Amongst others, top priority should be given to *Campylobacter* species. A Risk Profile on *Campylobacter* was prepared (http:/www.fdir.dk/publikationer). This recommended that a formal Risk Assessment focusing on thermophilic *Campylobacter* spp. in chicken products should be carried out according to the principles stated by the Codex Alimentarius Commission comprising the elements hazard identification, hazard characterization, exposure assessment, and risk characterization (CAC, 1999). This recommendation was based on the steady increase in the number of registered human cases of campylobacteriosis (Anonymous, 1996, 1997, 1998), the high prevalence of *Campylobacter* in retail chicken products (Anonymous, 1997; 1998) and the fact that several case-control studies had indicated that consuming and/or handling chicken were important risk factors (Norkrans and Svedheim, 1982; Oosterom et al., 1984; Harris et al., 1986; Deming et al., 1987; Kapperud et al., 1992; Ikram, 1994; Schorr et al., 1994; Neal and Slack, 1995).

The objective of this work was therefore to carry out a risk assessment to provide the Danish risk managers with information on the influence of different mitigation strategies on the number of human cases associated with thermophilic *Campylobacter* species in Danish retail chickens using quantitative modelling and Monte Carlo simulation.

2. Risk assessment framework

The present risk assessment includes the steps: (i) hazard identification, which search to identify the risk of campylobacteriosis associated with thermophilic *Campylobacter* in chickens; (ii) hazard characterization, which focuses on evaluating the nature of adverse health effects associated with food-borne *Campylobacter* spp. and how to quantitatively assess the relationship between the magnitude of the food-borne exposure and the likelihood of adverse health effects; (iii) exposure assessment, in which the likelihood and magnitude of exposures to *Campylobacter* as a result of consumption of a chicken meal is estimated; and finally, (iv) risk characterization, which estimates the risk of campylobacteriosis in a given population for a given set of input data.

A risk model, based on a farm to fork approach, was developed to estimate the exposure to *Campylobacter* from chickens and the number of human cases associated with this exposure. The framework of the risk model is seen in Fig. 1. The model details changes in prevalence and number of *Campylobacter* on chickens throughout the production line from slaughter to consumption. Module 1 models the transfer and spread of *Campylobacter* through a chicken slaughterhouse. Module 2 describes the transfer and spread of *Campylobacter* during food handling in private kitchens and the different consumption patterns for different age and sex groups. Output distributions from module 1 were used as input to module 2, and output distributions from module 2 were then integrated with the dose response relationship to estimate the number of human cases for different age and sex groups associated with thermophilic *Campylobacter* species in chickens.

3. Materials and methods

Information and data for the development of the risk model were obtained from Danish surveillance

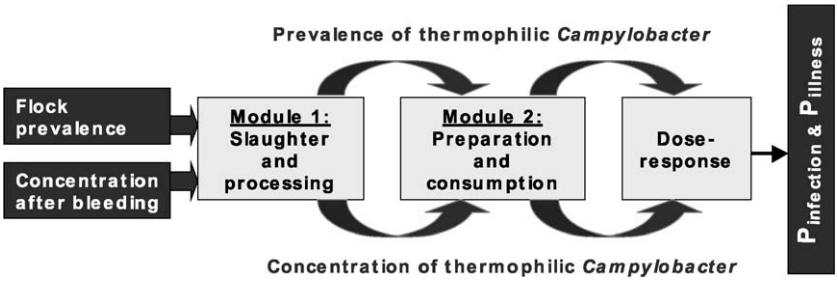

Fig. 1. Framework of the risk model. Concentration represents the number of *Campylobacter* on chickens or in chicken meals.

programs and—when not available—from research studies in other countries. Whenever possible, the data were represented by probabilistic distributions rather than single point estimates, as they were to be integrated in a probabilistic estimation of the risk using Monte Carlo simulation (Vose, 2000). The @RISK software package, version 4.0 (Palisade, USA) in combination with a macro produced in Visual Basic on the Excel platform, Microsoft® Excel 2000 (Microsoft, USA) were used to run the simulations.

4. Hazard identification

Campylobacter is the leading cause of zoonotic enteric human infections in most developed countries (Anonymous, 1999c, 2001b; WHO, 2001). In Denmark, *Campylobacter* surpassed *Salmonella* in 1999, where more than 4000 human cases of campylobacteriosis (78 cases per 100,000 population) were registered (Fig. 2). In Denmark as well as in other developed countries, the human cases are usually caused by *Campylobacter jejuni* (89% in 2000, 93–96% in earlier years) and to a lesser extent by *Campylobacter coli* (11% in 2000, 4–7% in earlier years) (Anonymous, 2001a,b). Most human *Campylobacter* infections are classified as sporadic single cases or part of small family related outbreaks. Identified outbreaks are not common (Friedman et al., 2000a).

The data presented in Fig. 2 reflect the laboratory confirmed cases of *Campylobacter* infections. The true number of cases is considered to be from 7.6 up to 100 times as high as the number of reported cases (Skirrow, 1991; Kapperud, 1994; Wheeler et al., 1999). This means that 30,000–440,000 people in Denmark may have had a *Campylobacter* infection in the year 2000 corresponding to a 'true' incidence of 600–8300 cases per 100,000 population.

The number of human *Campylobacter* infections, in which chickens directly or indirectly are the causative agent, is not known. The high prevalence rates in chicken meat at retail (Anonymous, 2001a,b) and the fact that case-control studies conducted worldwide repeatedly have identified handling raw poultry and eating poultry products as important risk factors for sporadic campylobacteriosis seem to support that chickens play an important role in the transfer of *Campylobacter* to humans (Norkrans and Svedheim, 1982; Hopkins et al., 1984; Oosterom et al., 1984; Harris et al., 1986; Deming et al., 1987; Kapperud et al., 1992; Schorr et al., 1994; Adak et al., 1995; Neal and Slack, 1995; Friedman et al., 2000b; Effler et al., 2001; Neimann, 2001). Other food related risk factors for human campylobacteriosis that have repeatedly been identified include consumption of other meat types, undercooked or barbecued meat, raw seafood, drinking untreated surface water or unpasteurized milk, or dairy products. Eating meat cooked outside the home (at restaurants) and the lack of washing the kitchen cutting board with soap (indicating cross-contamination) have also been identified as risk factors. Other risk factors include exposures when travelling abroad, contacts with pets and farm animals, and recreational activities in nature (Norkrans and Svedheim, 1982; Hopkins et al., 1984; Oosterom et al., 1984; Harris et al., 1986; Deming et al., 1987; Kapperud et al., 1992; Saeed et al., 1993; Schorr et al., 1994; Adak et al., 1995; Neal and Slack, 1995; Friedman et al., 2000b; Effler et al., 2001; Neimann, 2001).

Data from countries where the *Campylobacter* incidence has declined, likely as a result of changes

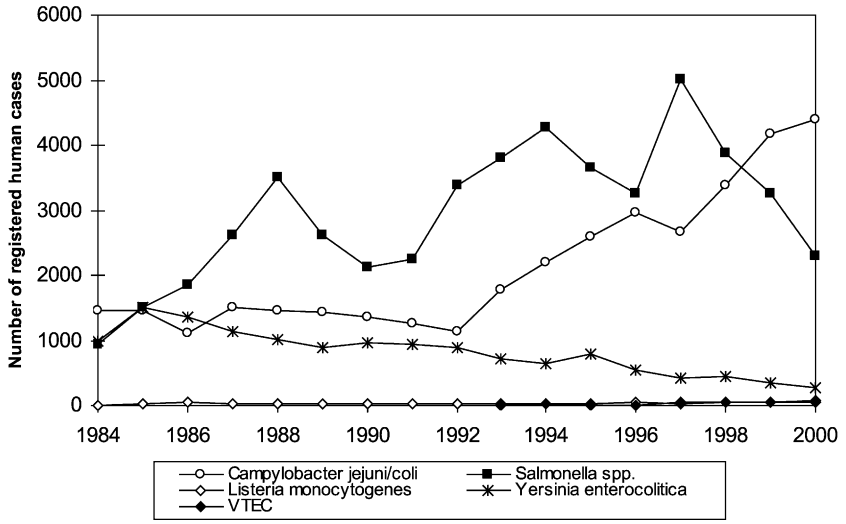

Fig. 2. The number of registered human cases in Denmark caused by the enteric pathogens *C. jejuni*/*C. coli*, *Salmonella* spp., *Yersinia enterocolitica*, *Listeria monocytogenes* and verotoxin producing *Escherichia coli* (VTEC) (Anonymous, 1995, 1996, 1997, 1998, 1999a, 2000, 2001a).

in chicken production or consumption, also supports that eating chickens seems to be an important source of campylobacteriosis. In Belgium, for instance, there was a decrease in the incidence along with the dioxin crisis in June 1999, probably because chicken and other meat products were withdrawn from the shops (Anonymous, 2001b). In Iceland an epidemic of domestically acquired human campylobacteriosis in 1998 and 1999 was controlled by interventions in the poultry production and processing and in education of consumers (Reiersen et al., 2001).

5. Hazard characterization

5.1. Adverse health effects

Enteropathogenic *Campylobacter* may cause an acute enterocolitis, the main symptoms being malaise, fever, severe abdominal pain and watery to bloody diarrhoea. The incubation period varies from 1 to 11 days, typically 1–3 days. In most cases, the diarrhoea is self-limiting and may persist for up to a week (Allos and Blaser, 1995).

Campylobacter infections may be followed by rare but severe non-gastrointestinal sequelae: reactive arthritis, a sterile post infectious process affecting multiple joints, which is often associated with the tissue phenotype HLA-B27 (Peterson, 1994; Allos and Blaser, 1995); the Guillain-Barré syndrome, a demyelating disorder of the peripheral nervous system resulting in weakness, usually symmetrical, of the limbs, weakness of the respiratory muscles and loss of reflexes, that may become chronic or even mortal (Mishu and Blaser, 1993; Mishu et al., 1993; Allos, 1997); and the Miller Fisher Syndrome, a variant of the Guillain–Barré syndrome characterized by opthalmoplegia, ataxia, and areflexia (Othsuka et al., 1988).

Development of antimicrobial resistance, such as the emergence of fluoroquinolone-resistant *C. jejuni* in humans, may compromise treatment of patients in severe cases where drug treatment is required (Piddock, 1995, 1999). In severe cases the drug of choice is usually erytromycin, though fluoroquinolones such as ciprofloxacin and norfloxacin are also used (Blaser et al., 1983).

5.2. Dose–response relationship

Only few studies describing the human response to a known dose of *Campylobacter* exist. In one experi-

ment, a dose of 500 organisms ingested with milk caused illness in one volunteer (Robinson, 1981). In another experiment involving 111 healthy young adults from Baltimore, doses ranging from 800 to 20,000,000 organisms caused diarrhoeal illness (Black et al., 1988). Rates of infection increased with dose, but development of illness did not show a clear dose relation. In an outbreak at a restaurant, the number of *C. jejuni* in the causative chicken meal was estimated to range from 53 to 750 *C. jejuni* per gram (Rosenfield et al., 1985). These few investigations indicate that the infective dose of *C. jejuni* may be relatively low.

The data generated by Black et al. (1988) have formed the basis of a dose–response model, which translates the number of organisms an individual is exposed to, into an estimate of the individual's probability of acquiring infection and illness. This estimate is dependent on (i) the numbers of organisms ingested, (ii) the probability of each individual organism to survive and infect the host once it is ingested, and (iii) the probability that the host become ill once infected. The estimate is also influenced by the virulence and the colonization potential of the ingested strain, the vehicle with which it is ingested (Black et al., 1988), and the susceptibility of the individual, e.g. immune status, age, and stomach contents (Coleman and Marks, 1998).

With respect to dose–response models, Haas (1983) used a stochastic Beta–Poisson model to describe the probability of infection (not illness) as a function of the ingested dose. In this model, it is assumed that the microorganisms in the ingested vehicle are randomly Poisson distributed and that each individual organism will have the same probability (p) of causing infection, where p is Beta(α, β) distributed. The Beta distribution reflects the uncertainty and the variability between individual humans on the probability of an organism to cause infection. Based on data from a human feeding trial by Black et al. (1988), Medema et al. (1996) calculated the maximum likelihood estimates for α and β ($\alpha = 0.145$ and $\beta = 7.59$) assuming that the dose–response relationship could be described by the Beta–Poisson model.

In the present model, the exact number of organisms ingested (n) is estimated for each iteration. Therefore, the probability (P_{inf}) that at least one of the n organisms will infect the host can be determined as $P_{inf} = 1 - (1 - p)^n$. Assuming that each individual organism will have the same probability (p) of infection in a given individual person, the assumption that the probability p may vary from person to person was included in the formula. When this formula was integrated with the maximum likelihood estimates (α, β) (Medema et al., 1996) the probability of infection could be expressed as $P_{inf} = 1 - (1 - \text{Beta}(\alpha, \beta))^n$.

From the feeding trial data presented by Black et al. (1988), *Campylobacter* infection was not always followed by illness. Only 11 of the 50 infected volunteers showed symptoms of illness. Additionally, development of illness did not show a clear dose relation. In the present dose–response model, it was assumed that the dose ingested and the probability of illness are two uncoupled processes. Thus, independently of the dose ingested, if a person becomes infected, there is a certain probability (in this case, 22%) that the person will become ill. The uncertainty on the probability of illness was described by a Beta distribution.

6. Exposure assessment

Two successive mathematical models (module 1 and module 2) were developed to estimate the likelihood and magnitude of exposures to *Campylobacter* as a result of consumption of a chicken meal. These detailed the prevalence and the number of *Campylobacter* on chickens throughout the production line from slaughter to consumption and the consumption patterns of different age and sex groups. No growth models were included in the exposure assessment, as thermophilic *Campylobacter* species do not multiply below 32 °C (ICMSF, 1996).

6.1. Module 1 (slaughter and processing)

Module 1 (Fig. 3) describes the impact of the processes scalding, defeathering, evisceration, and washing and chilling on the prevalence of *Campylobacter* contaminated broilers and on the number of *Campylobacter* on either chilled or frozen whole carcasses. Only contamination of the exterior skin surface was considered.

As input data in the module, the *Campylobacter* flock prevalence, the flock sizes, and the chronology

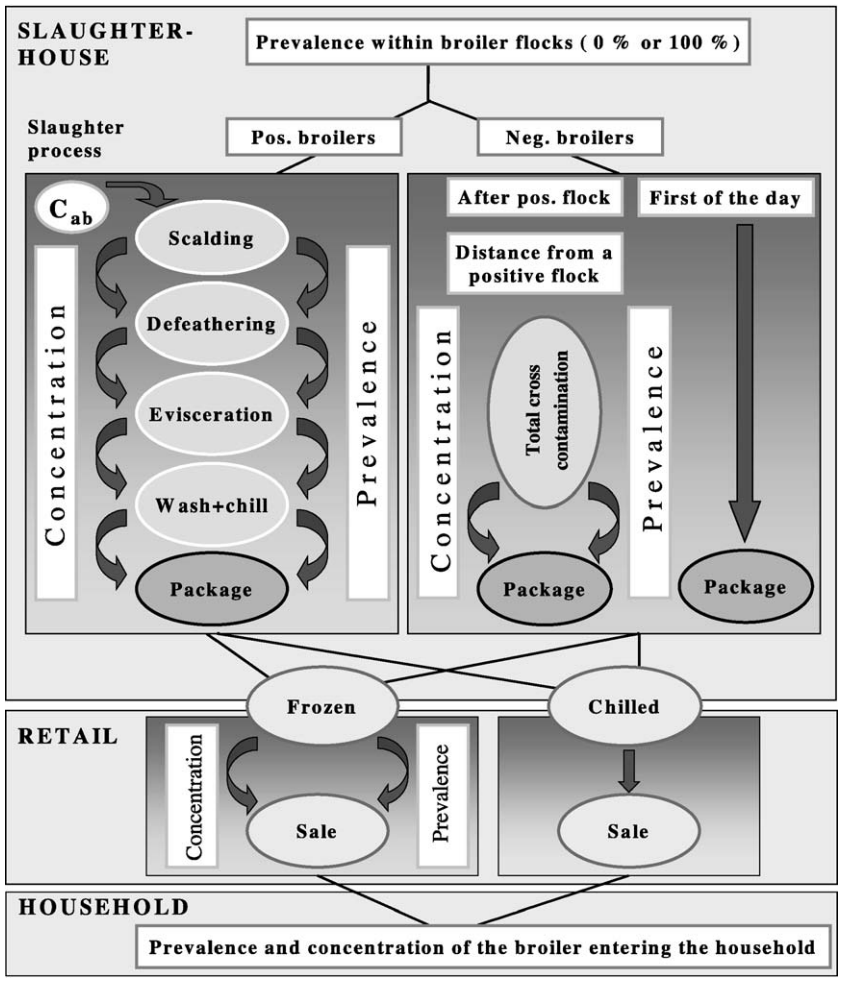

Fig. 3. Framework of module 1 addressing changes in prevalence and number of *Campylobacter* on broiler carcasses throughout the processing line. Concentration represents the number of *Campylobacter* on carcasses.

of broiler flocks slaughtered at a Danish slaughterhouse in the period from February 1998 to January 1999 were used. The flock prevalence data were based on a pooled faecal sample of 10 broilers from each flock slaughtered. It was assumed that either none or all birds in a flock were infected with *Campylobacter* at arrival to the slaughterhouse. This assumption was made since it has been shown that the time from initial infection to a full-blown infection of all broilers in a flock occurs within a few days (Berndtson, 1996), indicating that the probability that a flock will contain both positive and negative birds at slaughter is low.

As input data to describe the number of *Campylobacter* on the carcasses at the beginning of the slaughter line, the concentration data generated after bleeding by Mead et al. (1995) were used.

The levels of carcass contamination on broilers from *Campylobacter* positive flocks will inevitably change through slaughtering. Depending on the processing step, the number of *Campylobacter* on a carcass may increase or decrease and in rare cases the broiler carcasses may even become *Campylobacter* negative, because all *Campylobacter* organisms have been removed. Data generated by Izat et al. (1988) and Oosterom et al. (1983b) formed the basis

of the distributions developed to model changes in the number of *Campylobacter* on carcasses through the processing steps: scalding, defeathering, evisceration, and washing and chilling. The changes in the number of *Campylobacter* were modelled as an additive process in the logarithmic scale.

In addition to the changes in the number of *Campylobacter* on the carcasses, the different slaughter processes could also contribute to cross-contamination between birds within a flock and between flocks slaughtered successively. This has indirectly been demonstrated in studies investigating the *Campylobacter* contamination of slaughter equipment (Oosterom et al., 1983a). Cross-contamination from *Campylobacter* positive to *Campylobacter* negative flocks was implemented in module 1 as a worst case scenario, assuming that the first carcass in a negative flock would obtain a number of *Campylobacter* after processing similar to the number of *Campylobacter* on the previous positive carcass after washing and chilling. It was then assumed that the *Campylobacter* cells would be diluted out of the slaughter process as a function of the number of negative carcasses slaughtered. To describe the number of carcasses that needs to be slaughtered before the number of *Campylobacter* on a bird is reduced 50%, a half-concentration-constant was defined (B_{half}). As this constant is currently unknown, the effect of cross-contamination was analysed for five different values ($B_{half}=0$, 300, 1000, 3000, and 6000). In the general risk simulations B_{half} was set to 1000. The within flock cross-contamination in *Campylobacter* positive flocks was not specifically considered in the present model.

After processing, the carcasses for sale on the Danish marked are either stored as chilled (approximately 26%) or frozen products (approximately 74%). While chilled storage (at 4 °C) does not seem to affect the number of *Campylobacter* considerably (Blankenship and Craven, 1982; Oosterom et al., 1983b; Yogasundram and Shane, 1986), the number of *Campylobacter* will be reduced due to freezing at −20 °C (approximately 0.5–2.5 log units) (Hänninen, 1981; Oosterom et al., 1983b; Yogasundram and Shane, 1986). In the model the reduction due to freezing was described by a uniform distribution with a minimum of 0.5 and a maximum of 1.5. No further changes in the number of *Campylobacter* during transport and storage were considered in the model. The ratio of chilled compared to frozen chicken products sold in retail stores was included.

6.2. Module 2 (preparation and consumption)

The transfer of *Campylobacter* from a *Campylobacter* contaminated chicken to the consumer may occur through several contamination routes. Humans may become infected by direct contact, i.e. by licking on hands that have been in contact with a chicken or, indirectly, by consuming an undercooked chicken meal or a food item, e.g. salad or prepared chicken, which has been cross-contaminated during handling or preparation of a raw chicken. It is not known to which extend each of these processes contributes to the overall transfer of *Campylobacter* from chickens to consumers. Since *Campylobacter* is rather sensitive to heat (Blankenship and Craven, 1982; Humphrey and Lanning, 1987), the transfer of *Campylobacter* to humans due to undercooking is assumed to be a rather insignificant event. To simplify the process, only the transfer caused by cross-contamination via unwashed cutting boards was included in the module (Fig. 4), as this pathway was assumed to be the most important route of transfer. Hence, module 2 in the risk model quantifies the transfer of *Campylobacter* from a contaminated raw chicken to preparation surfaces and subsequently from these surfaces to ready-to-eat food (salad and prepared chicken). It was assumed that washing the cutting boards, immediately after handling of the raw chicken, would eliminate the risk of cross-contamination. In contrast, if the cutting boards were not washed, there would be a risk of transferring *Campylobacter*.

Several studies have shown that the extent of kitchen hygiene (safe/unsafe food handling) depends on age and sex (Altekruse et al., 1995; AIM Nielsen og Levnedsmiddelstyrelsen, 1997; Jay et al., 1999). Therefore, the hygiene level and the number of people within different age and sex groups, who prepare meals, were included in the module. Data from an American telephone survey (Yang et al., 1998) comprising approximately 15,000 persons in seven different states were used to describe the hygiene level during food handling. The interviewed people, divided in different age and sex groups,

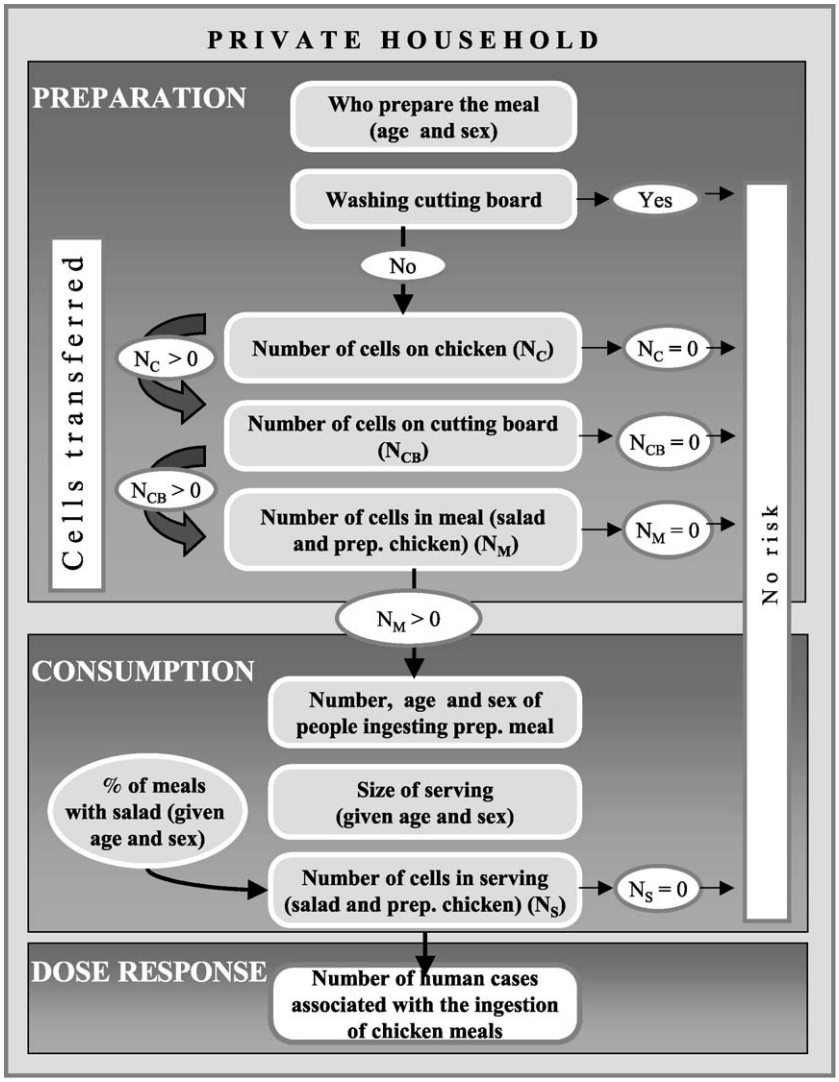

Fig. 4. Framework of module 2 covering the transfer and spread of *Campylobacter* during food handling in private kitchens and various consumption patterns for different age and sex groups. N_C, number on carcasses; N_{CB}, number on cutting board; N_M, number in prepared meal; N_S, number in servings.

reported whether or not they usually washed the cutting boards with soap or bleach after contact with raw meat. In the module, persons preparing meals were divided into six age and sex groups: women 18–29 years, 30–65 years, and above 65 years; and men 18–29 years, 30–65 years and above 65 years. The number of males and females preparing meals in each of these age groups in Denmark was obtained by combining data from a Danish Dietary Survey (Andersen et al., 1996) with demographic data from Statistics Denmark.

Several factors may influence the number of *Campylobacter* transferred from a raw chicken to a cutting board and further to a prepared meal. Such factors include: the amount of drip fluid, the contact area between the raw chicken and the cutting board, the time lag between placing the raw chicken and the prepared chicken on the cutting board, etc. The data

availability on this subject is rather limited. Two studies describe transfer of bacteria (*E. aerogenes*) via surfaces (Zhao et al., 1998; Chen et al., 2001). The results of Zhao et al. were used as a guide to produce distributions describing the level of cross-contamination with *Campylobacter* from raw chicken to the cutting board and from the cutting board to the salad and/or back to the prepared chicken.

The number of ingested *Campylobacter* organisms depends on the frequency of consuming chicken and the frequency of eating salad with a chicken meal as well as the amounts eaten. Such data were obtained from a Danish Dietary Survey (Andersen et al., 1996).

The preparation and consumption of chicken or chicken/salad were linked to information on family compositions, which included data on numbers of adults and children in households in Denmark and their age and sex. These data were obtained from Statistics Denmark.

Finally, the consumption data and the family compositions were linked to the persons, who prepare meals, to determine the total number of people being exposed to *Campylobacter* by eating chicken meals and to define the distribution of exposed people within different age and sex groups.

7. Risk characterisation (results and discussion)

In the risk characterization part, the estimated exposure was integrated with the dose–response model to provide a risk estimate and to determine the influence of different mitigation strategies on this risk estimate.

7.1. Risk estimate

The number of human cases of campylobacteriosis associated with the consumption of chicken meals was estimated to approximately one case out of 14,300 chicken servings. As the number of servings including chicken ingested in Denmark was estimated to approximately 201 million per year, based on data from Statistics Denmark and the Danish Dietary Survey (Andersen et al., 1996), the expected number of cases associated with the consumption of *Campylobacter* contaminated chicken meals prepared in private kitchens was estimated to be approximately 14,000 per year (95% confidence interval; from 7753 to 20,942 cases). Compared to the 4386 registered human cases in 2000 (Anonymous, 2001a) or an assumed actual number of cases of 30,000–440,000 (se above) the estimated number of human cases arising from eating chicken seems to be a realistic result. Despite the fact that there may be other routes of infection via chicken as well as other sources of infection than chickens, the estimate indicates that cross-contamination during food handling in private kitchens from *Campylobacter* contaminated chickens seems to be an important route of exposure to *Campylobacter*.

The simulations also showed that especially young men (aged 18–29 years) and to some extent young women (aged 18–29 years) were at higher risk and that males and females above the age of 65 were at lower risk than other groups (Fig. 5). This is in relatively good agreement with the actual frequencies of campylobacteriosis registered by the Statens Seruminstitut (Anonymous, 2001a).

7.2. Influence of mitigation strategies

Assuming that chickens contribute to human campylobacteriosis in Denmark, the developed risk model was used as a tool to run simulations designed to estimate the influence of the following mitigation strategies: (1) a reduction of the flock prevalence (a reduction of the number of *Campylobacter* positive broiler flocks); (2) a reduction of the number of *Campylobacter* on the chicken carcasses; (3) a reduction of cross-contamination from *Campylobacter* positive to *Campylobacter* negative flocks during slaughter; and (4) a reduction of the occurrence of cross-contamination during food handling. The influence of these strategies on (i) the fraction of *Campylobacter* positive chickens leaving the slaughterhouse; (ii) the number of *Campylobacter* on these birds; and (iii) the incidence of campylobacteriosis were simulated.

7.2.1. Reduction of flock prevalence

The simulations showed a linear relationship between the flock prevalence and the fraction of positive chickens leaving the slaughterhouse (Fig. 6A), and between the flock prevalence and the inci-

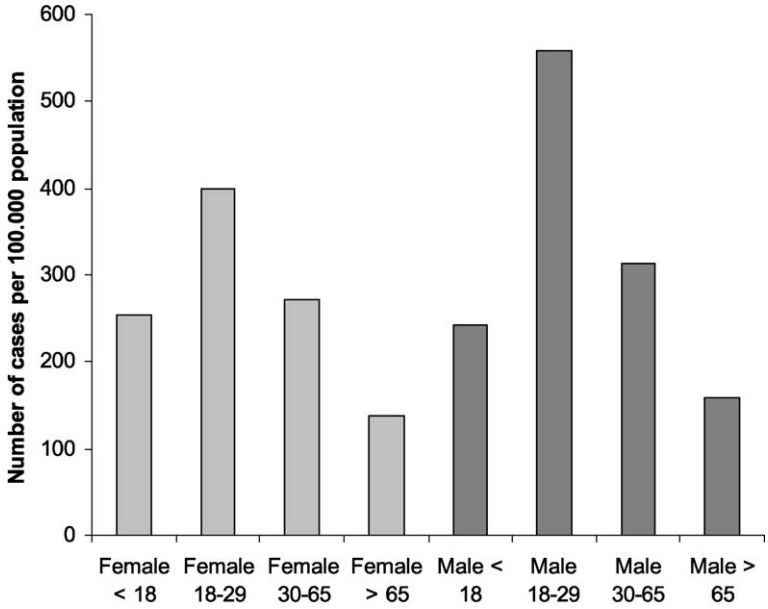

Fig. 5. Estimated values of the incidence of campylobacteriosis associated with the consumption of a chicken meal for different age and sex groups.

dence of campylobacteriosis (Fig. 6C). A minor increase in the number of *Campylobacter* on the positive chickens was seen with increased flock prevalence (Fig. 6B). This could be explained by the fact that cross-contamination from positive to negative flocks is relatively more predominant at low flock prevalences and that the number of *Campylobacter* on these contaminated chickens is lower than the average of the already positive birds. Therefore, the average number of *Campylobacter* on all positive chickens will be lower for low flock prevalences compared to high flock prevalences.

The simulations indicated that if the flock prevalence was reduced for example two times then the number of cases associated with consumption of chicken meat would also be reduced approximately two times. This is because there is a one-to-one relationship between the two parameters.

Several countries have implemented or are at the point of implementing strategies to reduce the number of *Campylobacter* contaminated broiler flocks. Until now establishment of "strict hygienic barriers" or "biosecurity zones" at each poultry house seems to be the only preventive option shown to work in practice (Kapperud et al., 1993; Humphrey et al., 1993; Berndtson et al., 1996; Reiersen et al., 2001). Other proposed tools to prevent flock contamination are vaccination and competitive exclusion, which however, need further research since the data published so far are inconclusive (Stern, 1994; Widders et al., 1996).

7.2.2. Reduction of the number of Campylobacter on chicken carcasses

The simulations showed that a relatively large reduction of the number of *Campylobacter* on the chickens, for example a reduction of 3 $\log_{10}$ CFU/chicken (e.g. from the simulated mean level = level 0 to level −3, Fig. 7A), only lead to a minor reduction (less than 10%) in the fraction of positive chickens leaving the slaughterhouse (Fig. 7A). On the contrary, the number of *Campylobacter* on the positive chickens was significantly reduced (Fig. 7B), which was expected since a step had been implemented in the risk model to remove a certain fraction of *Campylobacter* from the carcasses somewhere in the slaughter process. The minor effect on the number of positive carcasses at the end of slaughter even after introduction of a decrease of 3 log units demonstrate the need for quantitative detection methods, as the effect of

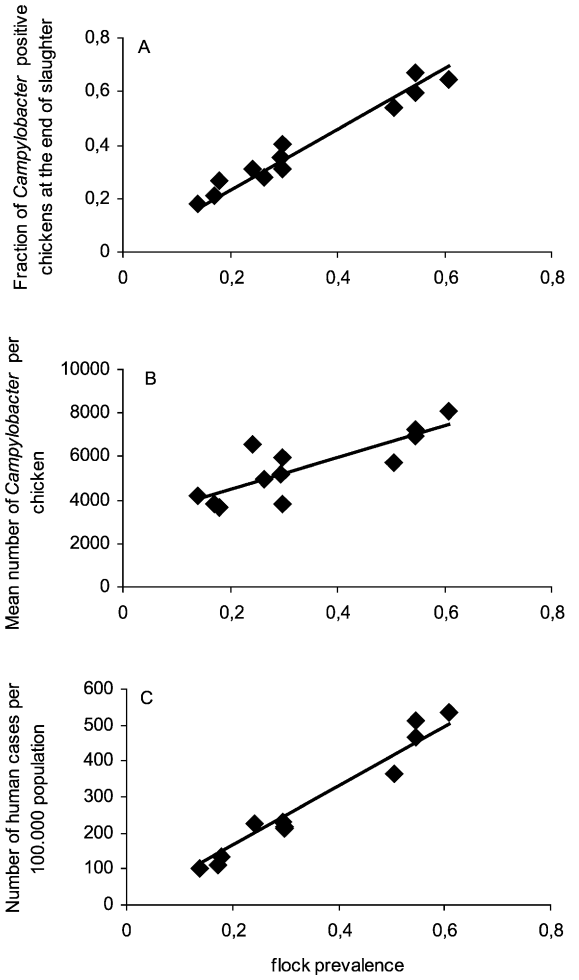

Fig. 6. Relationship between the flock prevalence and (A) the fraction of positive chickens leaving the slaughterhouse, (B) the number of *Campylobacter* on the positive chickens, and (C) the incidence of campylobacteriosis. The scattering of the simulation sampling points around the fitted line is due to the stochastic nature of the input data used in the model, i.e. they are true data obtained from a slaughter program.

such a decrease in the number of *Campylobacter* would not have been detected by qualitative methods.

The incidence of campylobacteriosis related to consumption of a chicken meal was reduced significantly by reducing the number of *Campylobacter* on the carcasses (Fig. 7C). Even though such a reduction had almost no influence on the fraction of positive chickens (Fig. 7A), a reduction of the number of *Campylobacter* on the positive chickens of for example a factor 100 (2 log units), resulted in a reduction of the number of human cases of approximately a factor 30 (from a simulated incidence of, e.g. 300 to 10 per 100,000 population) (Fig. 7C).

A reduction of the number of *Campylobacter* on chickens may be obtained by the introduction of different techniques during processing. It is well known that for example freezing of the meat leads to a drop in the concentration of approximately 2 log units (Hänninen, 1981; Oosterom et al., 1983b;

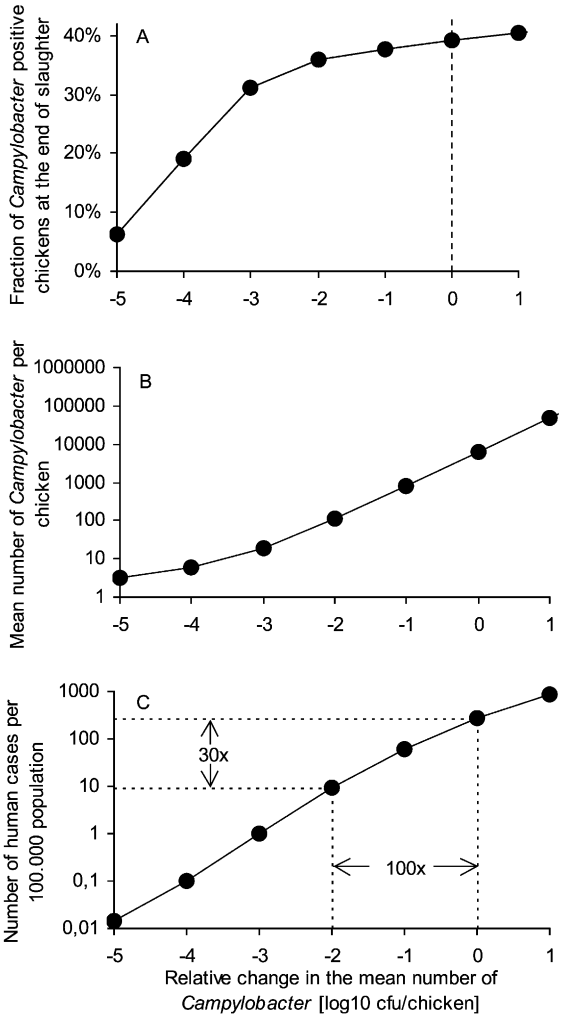

Fig. 7. Relationship between the number of *Campylobacter* on contaminated chickens and (A) the fraction of positive chickens leaving the slaughterhouse, (B) the number of *Campylobacter* on the positive chickens, and (C) the incidence of campylobacteriosis.

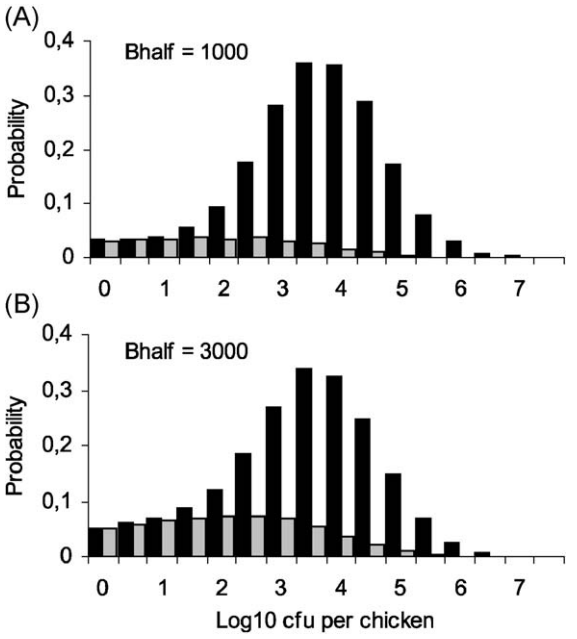

Fig. 8. Simulated distributions for the number of *Campylobacter* on *Campylobacter* contaminated chickens originating from *Campylobacter* positive flocks (black bars) and cross-contaminated chickens originating from *Campylobacter* negative flocks (grey bars) at the end of the slaughter processes for a B_{half} value of 1000 (A) and 3000 (B).

Yogasundram and Shane, 1986). Other techniques that might have a positive effect on removal or inactivation of *Campylobacter* are increased scalding temperature, improved evisceration techniques (to avoid faecal contamination of the meat), the use of more water throughout the entire slaughter line, forced air-chilling, and the introduction of disinfectants. Also methods to prevent cross-contamination from faecal material to the exterior of the broilers during transport (or at the farms) might be ways to reduce the final number of *Campylobacter* on the chickens. At present the information available is not sufficient to suggest which technique or techniques are the most efficient.

7.2.3. Reduction of cross-contamination between flocks at slaughter

It was speculated if implementation of logistic slaughter, i.e. a routine where all negative flocks are slaughtered prior to the positive flocks on a day, could influence the risk estimate. The situation where no cross-contamination occurs (B_{half} equal to zero) was compared to the effect of different levels of cross-contamination.

The simulations showed that for B_{half} values of 1000 and 3000, the mean number of *Campylobacter* on cross-contaminated, originally *Campylobacter* negative chickens was significantly lower than the average number on the birds originating from *Campylobacter* positive flocks at the end of the slaughter processes (Fig. 8).

The simulations also showed that although cross-contamination between flocks may result in more positive chickens leaving the slaughterhouse, the change in the incidence of campylobacteriosis was rather limited (Fig. 9). Even for high values of B_{half} ($B_{half} = 6000$) the increase in the incidence of campylobacteriosis due to cross-contamination between flocks during slaughter was only approximately 16% (a factor of 1.16). It was therefore concluded that introduction of logistic slaughter only seems to have a rather limited effect on the number of human cases compared to for example the introduction of procedures to reduce the number of *Campylobacter* on the chickens.

The relatively limited effect may be explained by the fact that although the number of positive chickens increased with increasing degree of cross-contamination, the number of *Campylobacter* on these cross-contaminated chickens was low compared to the

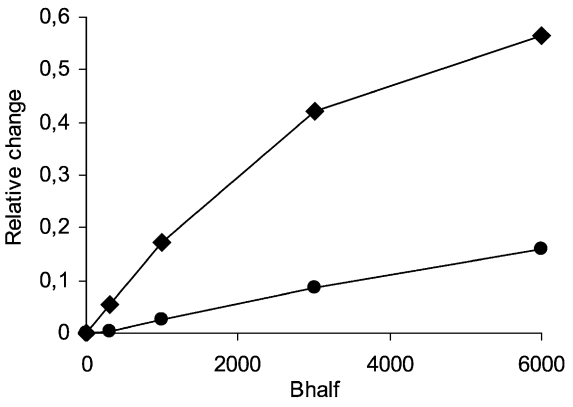

Fig. 9. Relative change in the number of *Campylobacter* positive chickens at the end of slaughter (♦) and in the incidence of campylobacteriosis (●) for different degrees of cross-contamination from positive to negative flocks (B_{half}).

number on the birds originating from *Campylobacter* positive flocks. The probability of transferring *Campylobacter* from cross-contaminated chickens to ready-to-eat food during food handling will therefore also be low and, hence, also the probability of exposure and illness.

7.2.4. Reduction of cross-contamination during food handling

Education of consumers to obtain a reduction of cross-contamination during food handling was included in the model by changing the number of people who do not wash their cutting board during food handling.

From the simulations it was obvious that an improvement of the hygiene level in private kitchens (by washing the cutting board) could reduce the incidence of campylobacteriosis (Fig. 10). There was a linear one-to-one relationship between the occurrence of not washing the cutting board and the number of human cases. This means that efforts, directed into improving the frequency of washing the cutting board by for example a factor 2, would result in a reduction of the incidence of campylobacteriosis by a factor 2.

The consumer model has been restricted only to focus on the effect of not washing the cutting board. Certainly, there are other ways in which *Campylobacter* can be transferred from the raw chicken to the final meal. However, it was assumed that the event not washing the cutting board was an important route of cross-contamination in private kitchens. In addition, the process was mathematically similar to many other possible transfer routes during food preparation. Therefore, when measuring the effect of relative changes it may not be necessary to look at all the possible routes of transfer. Thus, a relative change in the number of people washing cutting board may be extended also to represent an example of a general change in hygiene habits.

Several surveys on consumer habits show that many people prepare their food in a way which supports the transfer of microorganisms such as for example *Campylobacter* to ready-to-eat items (Williamson et al., 1992; Altekruse et al., 1995; AIM Nielsen og Levnedsmiddelstyrelsen, 1997; Worsfold and Griffith, 1997; Daniels, 1998; Griffith et al., 1998; Yang et al., 1998; CASA, 1999; Jay et al., 1999). Improving the food hygiene by education of consumers was one of the strategies that were implemented on Iceland to reduce the *Campylobacter* incidence (Reiersen et al., 2001). The obtained decrease in the human cases could, however, not be linked to separate interventions/mitigations implemented.

7.3. Limitations

The risk model only considers the risk of campylobacteriosis for the normal population in Denmark associated with chilled or frozen, whole chickens of Danish origin, which have been prepared in a private home.

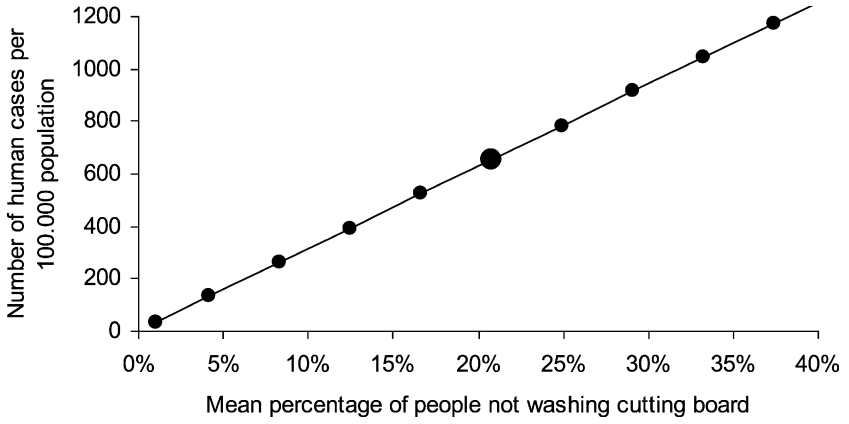

Fig. 10. Relationship between the mean percentage of people not washing the cutting board and the incidence of campylobacteriosis.

The estimated values of risk rely on the information and data used and the assumptions taken throughout the entire risk model. Not all assumptions have been confirmed/validated. The data, which describe the slaughter processes, and the consumer behaviour are very sparse and may not reflect the actual situation in Denmark. Several detailed processes along the production line and in the kitchen have been simplified, and the dose–response relationship is based on only one study describing the response in young American volunteers to two strains of *C. jejuni*. Therefore, with the current state of knowledge, a model like the one presented cannot be used to generate true risk estimates. As emphasized previously, the objective of this risk assessment was not to produce a risk estimate, but to provide the Danish risk managers with information of the relative importance of different simulated mitigation strategies in chicken production, processing and preparation. We believe that as long as the underlying mathematical assumptions are true, good estimates on relative changes can be made independently of the validity of the risk estimates. Thus, the model can be used to compare the relative effect of different mitigation or intervention strategies and to compare the relative risks for different age and sex groups.

8. Conclusions

Four different mitigation strategies to reduce the incidence of campylobacteriosis associated with the consumption of chicken meals have been compared by running Monte Carlo simulations on a quantitative risk model developed to detail the probability of exposure to *Campylobacter* and the likelihood of campylobacteriosis associated with this exposure.

The simulations indicated that the incidence of campylobacteriosis associated with consumption of chicken meals could be reduced 30 times by introducing a 2-log reduction of the number of *Campylobacter* on the chicken carcasses. To obtain a similar reduction of the incidence of campylobacteriosis, the flock prevalence should be reduced approximately 30 times (e.g. from 60% to 2%) or the kitchen hygiene improved approximately 30 times (e.g. from 21% not washing the cutting board to 0.7%). An efficient risk management option may therefore be to introduce methods to reduce the number of *Campylobacter* on the carcasses somewhere from farm to retail. One way to reduce the concentration could for example be freezing the carcasses. Eliminating cross-contamination from positive to negative flocks during slaughter had almost no effect on the human incidence, which indicates that logistic slaughter has a minor influence on the risk. Finally, the simulations showed that people in the age of 18–29 years had the highest risk of campylobacteriosis associated with the consumption of chicken meals.

In conclusion, the presented risk model provide the Danish risk managers with valuable information on which mitigation strategies in the ‘farm to fork’ chain that would be important to consider in relation to reducing the transmission of *Campylobacter* to humans. It should be emphasised, though, that the simulated results are completely dependent on the information and data used and the assumptions taken during the development of the risk model and that the model needs to be validated and further improved when new information becomes available. It should also be considered whether other modelling approaches than the farm to fork approach would give more reliable results. Research in this area is ongoing. However, other approaches might not allow for the same level of insight in the actual slaughter and food handling procedures.

The strategy to reduce the number of *Campylobacter* on the chicken carcasses has been welcomed by the Danish risk managers as a supplement to the ongoing work at farm level to reduce the flock prevalence. In cooperation with the Danish poultry association and the industry, the risk managers discuss options to reduce the number of *Campylobacter* on the chicken meat during processing. As an example, the reducing effect of different chilling and scalding techniques are being tested. Another option, which has been discussed, is to direct *Campylobacter* negative flocks to the production of chilled chicken meat. It should be noted though that at present the options to be implemented have not yet been chosen by the risk managers.

As described in the risk management procedure, the effect of the upcoming management options will be monitored, e.g. by parameters such as the human incidence of campylobacteriosis as well as the prev-

alence and the concentration of *Campylobacter* in the chicken products.

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