UNIVERSIDADE DE LISBOA

Faculdade de Medicina Veterinária

THE IMPORTANCE OF CONSUMER PHASE MODELS IN QUANTITATIVE MICROBIOLOGICAL RISK ASSESSMENT

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Abstract

The importance of consumer phase models in quantitative microbiological risk assessment

In quantitative microbiological risk assessment (QMRA), the consumer phase model (CPM) describes the part of the food chain from purchase of the food product at retail to the moment of consumption. The large variation in consumer food handling practices and scarce availability of data imply that several simplifying assumptions are made when a CPM is constructed. In the development of a CPM, it is relevant to understand to what extent these models need to include a detailed description of the processes that may result in exposure. The study from Nauta et al. (2009), suggests that "There is no alternative but for a probabilistic approach to risk assessment models of the consumer phase". The purpose of this study is to compare the results given by seven published stochastic CPMs found in the literature for *Campylobacter, Salmonella* and *Listeria monocytogenes*, with two simpler modelling techniques: a constant value "a-factor" (Duarte, Nauta, & Aabo, 2016) and deterministic CPMs, which don't include variation. The modelling techniques are compared by means of absolute risk estimates and relative risk estimates. It was found that the "a-factor" estimates similar absolute risks to the stochastic CPMs, but different relative risks from all the stochastic CPMs. Results also showed that deterministic CPMs estimate different absolute risks from all the stochastic CPMs. Regarding relative risks, it was observed that four in a total of seven deterministic CPMs showed similar results in all the intervention scenarios simulated to the corresponding stochastic CPM. In these four scenarios, deterministic CPMs could be used to assess the effect on the risk of intervention scenarios in the food production chain. It is not clear which situations and assumptions interfere with the results obtained when a deterministic CPM estimates similar or different relative risks from a stochastic CPM. Answering these questions would require more in depth studies about the role and performance of deterministic CPMs in QMRA.

Keywords: *Campylobacter*, consumer phase model, deterministic, food microbiology, *Listeria monocytogenes*, Risk assessment, *Salmonella*, stochastic.

Resumo

A importância de modelos da fase do consumidor em avaliação quantitativa de risco microbiológico

Em avaliação quantitativa de risco microbiológico (AQRM), um modelo da fase do consumidor (MFC), descreve a etapa da cadeia de produção de alimentos desde a compra do produto alimentício até ao momento do seu consumo. Devido à variação considerável existente nas práticas de preparação de alimentos e de escassa disponibilidade de dados nesta fase, na construção de um modelo da fase do consumidor é necessário incluir várias suposições subjectivas no âmbito de simplificar este processo. Na construção de um MFC, é necessário compreender em que medida é que este necessita de incluir descrições detalhadas dos processos que resultam em exposição. O estudo realizado por Nauta et al. (2009) sugere que devem ser sempre usados modelos estocásticos em AQRM para caracterizar a fase do consumidor. O objectivo deste estudo é comparar resultados obtidos por sete modelos estocásticos da fase do consumidor publicados para os microorganismos *Campylobacter, Salmonella* e *Listeria monocytogenes,* com duas técnicas simplificadas ("a-factor" presente no estudo de Duarte, Nauta, & Aabo, (2016) e MFC determinísticos), cujos modelos não incluem variação. As diferentes técnicas são comparadas em termos de risco absoluto e risco relativo.
Verificou-se que a constante "a-factor" estima riscos absolutos semelhantes aos estimados por um modelo estocástico, mas diferentes riscos relativos em todos cenários simulados. Os resultados obtidos também demonstram que os MFC determinísticos estimam riscos absolutos diferentes de todos os modelos estocásticos. Relativamente aos riscos relativos, observou-se que quatro num total de sete MFC determinísticos calcularam resultados semelhantes aos modelos estocásticos correspondentes. Nestes quatro cenários, seria aceitável utilizar MFC determinísticos para estimar o efeito de intervenções na cadeia de produção de alimentos no risco final. Não foi possível esclarecer quais as situações ou suposições que interferem com os resultados obtidos quando um MFC determinístico estima riscos relativos semelhantes ou distintos do modelo estocástico. Dar resposta a estas questões implica a realização de estudos mais aprofundados sobre o papel e desempenho de MFC determinísticos em AQRM.

Palavras-chave: Avaliação de risco, *Campylobacter*, determinístico, estocástico, microbiologia alimentar, modelo da fase do consumidor, *Listeria monocytogenes*, *Salmonella*.

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List of Abbreviations and Symbols

CFU – colony forming units

CSS- cold smoked salmon

CPM- consumer phase model

D^{-1}- per day

QMRA- quantitative microbiological risk assessment

MPD – maximum population density

MRA – microbiological risk assessment

RTE- ready to eat food

RR- relative risks

SD- standard deviation

Internship Report

As part of the Integrated Master's Degree in Veterinary Medicine from the Faculty of Veterinary Medicine, University of Lisbon, I completed two internships with a total duration of six months.

The first internship took place in the Faculty of Veterinary Medicine in the University of Lisbon, from October to January. I was supervised by my co-supervisor, Telmo Nunes, and performed tasks in the areas of epidemiology and food safety risk assessment. During these months, I acquired valuable skills that were essential to prepare for my second internship in Denmark. I performed statistical data analysis in the software "R" and did literature review in food safety risk assessment and in specific consumer phase models, to have more in depth knowledge on the subject before heading to Denmark.

The second internship took place at the National Food Institute, Technical University of Denmark, for three and a half months (from January and April). In this institute work researchers from all over the world, who perform studies in the areas of food safety, toxicology, food microbiology, microbiological risk assessments, risk benefits assessment and nutrition. I had the opportunity to experience a new working environment and interact with highly dedicated scientists on a daily basis. The atmosphere was friendly and people were welcoming, which contributed to make my experience a memorable one.

During my internship, I was supervised by Dr. Maarten Nauta for my Master's dissertation about "the Importance of consumer phase models in Quantitative Microbiological Risk Assessment". With the support from Maarten and due to our weekly meetings, I was able to gain a broad range of skills in a short period of time. I performed literature review and critical analysis on the subject of Quantitative Microbiological Risk Assessment and specifically analysed different consumer phase models in the literature to include in my project. I also learnt advanced risk assessment and modelling techniques by implementing some of the models in Excel spread sheets using Monte Carlo software @risk 5.5 (Palisade). I gained valuable knowledge on interpreting the results obtained from risk assessment modelling techniques accurately, in order to improve food safety.

At the end of my internship in Denmark, I had the opportunity share in more detail the type of work performed during the internship and the results obtained in an oral presentation. This presentation allowed me to practice my oral presentation skills, as well as to think critically and answering questions related to the study conducted.

All these internships, challenges and opportunities contributed for the successful conclusion of this dissertation.

I. Review on microbiological risk assessment

1. Introduction

Zoonotic diseases may be transferred from animals to humans through the production, handling and consumption of contaminated foods, and are considered a significant and widespread global public health threat. In the European Union (EU), over 320,000 human cases are reported each year, but it is likely that the real number is in fact much higher. *Campylobacter spp.*, *Sealmonella* spp., and *Listeria monocytogenes* are important pathogenic microorganisms that cause foodborne diseases in humans. In 2014, *Campylobacter* was the most commonly reported gastrointestinal bacterial pathogen in humans in the European Union (EU), and has been so since 2005. The number of reported confirmed cases of human campylobacteriosis in the EU in 2014 was 236.851. In the same year, *Salmonella* was the second most commonly reported gastrointestinal bacterial pathogen in humans in the EU, with a total of 88.715 confirmed salmonellosis cases reported. Listeriosis has had a statistically significant increase over 2008-2014, with 2.161 confirmed human cases of listeriosis in the EU in 2014 (EFSA-ECDC, 2015).

Assessing risks in the food chain and assuring food safety is a challenging task in the current highly globalized world (Quested et al., 2010). Global changes such as international trade of food and animal feed, international travel and migration, population growth, poverty, aging population and newly emerging pathogens, are important factors that influence incidence and profile of food borne illnesses (Miyagishima & Ka¨ferstein, 2003). International bodies such as World Trade Organization, World Health Organization, Food and Agricultural Organization of the United Nations and the *Codex Alimentarius*, promote the of use scientific-based approaches to prevent food borne illness. This implies an increasing use of risk assessment, a systematic tool used to develop consistent and science-based standards for international trade (Codex Alimentarius Commission, 1999; World Health Organization, 1995).

2. Purposes of microbiological risk assessment

As the production, processing, distribution, marketing, preparation, and consumption of food is a complex and interdependent activity, it is important to stress that food safety is assured through a pragmatic management of a broad array of potential risks. Food safety risks can be controlled by implementation of good hygienic practices (GHPs) and through the hazard analysis of critical control point (HACCP) system. HACCP is a “risk management system based on an evaluation of hazards that are reasonably likely to occur, followed by the

implementation of mitigations to control those hazards to an acceptable level" (Heredia et al., 2008). There are several steps in the food production chain at which control can be applied to prevent or eliminate a food safety hazard or reduce it to an acceptable level. These are called critical control points (CCP), and can only be identified through a deep understanding of the dynamics of microbial composition in food during processing. (CAC, 1997; Heredia et al., 2008; Kilsby and Pugh, 1981).

Microbiological risk assessment (MRA) tools allow to measure the risk of foodborne infection associated to the consumption of contaminated food, by providing a framework to model microbial changes along the food chain. It is stated by (Buchanan & Whiting, 1998), that if MRA is associated with HACCP, has an enormous potential to relate operations in food manufacturing to public health demands.

The information obtained in a MRA is used to support risk-based management decisions, to establish standards for food in international trade, for evaluation of proposed management and intervention strategies, by measuring the risk reduction potential of various risk control options, highlighting data and information gaps and identifying research needs. (Havelaar et al., 2008; Heredia et al., 2008; Lammerding & Paoli, 1997)

2.1 Risk definition and how to measure it

Risk is defined as "a function of the probability of an adverse health effect and the severity of that effect, consequential to a hazard(s) in food" (CAC, 2011). In 1995, the *Codex Alimentarius* defined risk assessment as "the estimation of the severity and likelihood of harm or damage, resulting from exposure to hazardous agents or substances" (Food and Agriculture Organization of the United Nations/WHO, 1995).

In MRA, these hazardous agents and substances represent microorganisms and/or their toxins. The final output of a risk assessment, i.e., the risk, is obtained by combining data and analytical models, that measure the probability of human exposure to pathogenic microorganisms and the degree of human response to that exposure (Heredia et al.,2008).

3. History of microbiological risk assessment

It is known that for long, efforts have been made to assess and measure risks attributed to hazards in the food production chain. As early as in 1983, the use of risk assessment was promoted by the National Research Council (NRC), United States, to support the scientific basis of risk-based decision making. A report entitled "Risk Assessment in the Federal Government: Managing the Process", formalized for the first time the basic concepts of risk assessment (NRC, 1996). In 1995, risk assessment was promoted by the Sanitary and Phytosanitary Agreement and by the Technical Barriers to Trade Agreement to solve

international trade disputes, due to the increasing growth of the global food trade verified in the mid 1990s (WHO, 1995). MRA was one of the strategies used to evaluate food borne illnesses and manage food safety risks. In 1996, a document on principles and guidelines for risk assessment was published by FAO/WHO Joint Expert Consultation for the Codex Committee on Food Hygiene (CCFH) (CCFH, 1996). In the same year, a framework for conducting microbiological risk assessments was also published by the International Life Sciences Institute (ILSI).

The first quantitative microbiological risk assessment (QMRA) of a food chain was published in 1997. It concerned E. coli O157:H7 in hamburgers (Cassin et al., 1998). Since that, several QMRAs have been conducted by international organizations, industry groups and national governments, and nowadays, this tool is consistently used to support free trade and ensure public health. (Schroeder et al., 2007).

4. Risk assessment framework

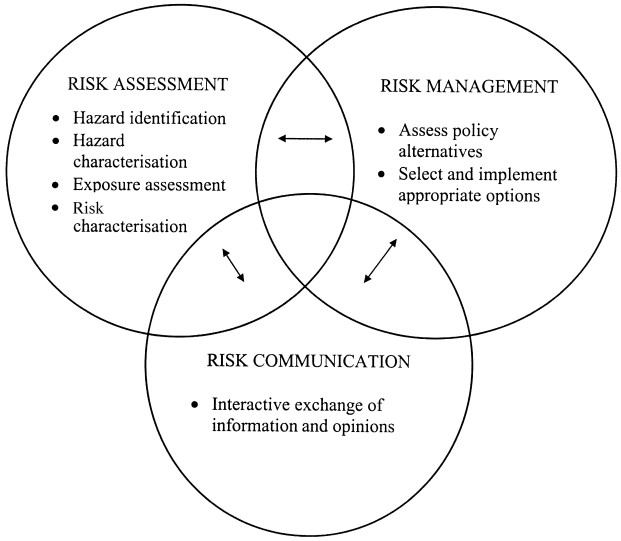
MRA is one of the three components of the risk analysis paradigm, along with risk management and risk communication (Voysey & Brown, 2000). Fig.1 describes the risk analysis framework and the interactions between the three components.

Risk management consists in the analysis of policy alternatives based on the results of risk assessments, and selecting and implementing appropriate control measures (including regulatory measures), if required.

Risk assessment is the scientific evaluation of actual or potential adverse health effects in humans, following exposure to hazards.

Risk communication involves an exchange of information between risk assessors, risk managers, consumers, industry and other interested parties, for subjects regarding risk and risk management processes (CAC, 1999).

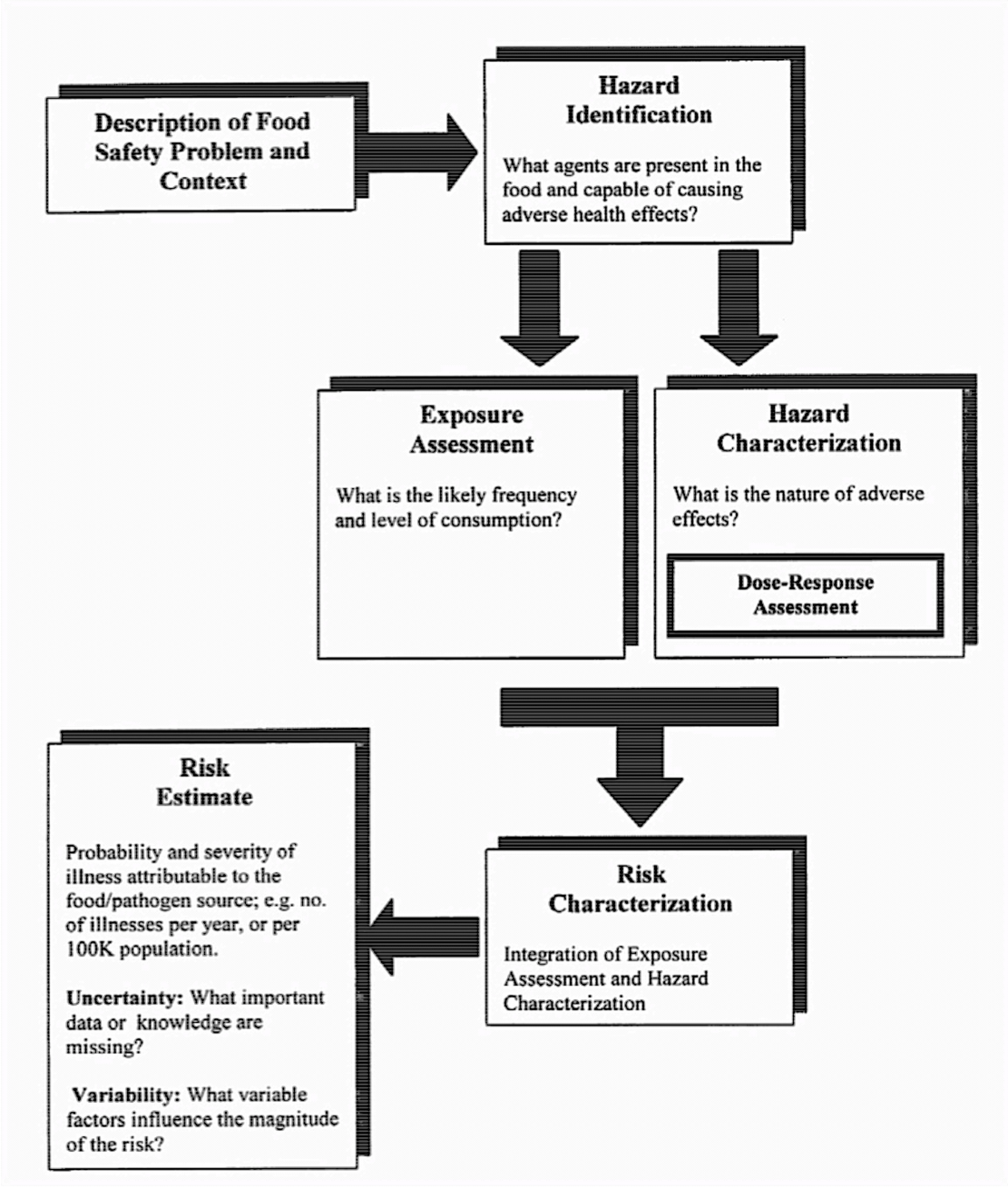
Figure 1: Risk analysis framework (From: Lammerding (1996))



5. Components of microbiological risk assessment:

The four cornerstones of MRA, defined as hazard identification, exposure assessment, hazard characterization (dose–response), and risk characterization (FAO/WHO, 1999) are described in Figure 2.

Figure 2: Steps of microbial food safety risk assessment (From: Lammerding & Fazil (2000)).



5.1 Statement of purpose

Prior to starting a risk assessment, the purpose of the study should be clearly defined. The output form of a risk assessment and possible output alternatives should also be defined. Examples of output forms include: estimate of the prevalence of illness, estimate of annual rate (incidence of human illness per 100,000) or estimate of the rate of human illness and severity per eating occurrence (CAC, 1999).

5.2 Hazard identification

The first step in a formal risk assessment is hazard identification. In a MRA, this activity aims to identify the microorganisms or the microbial toxins that may be present in a specific food and the adverse health effects that can possibly occur due to its presence. (CAC,1999; Center for Disease Control and Prevention, 2005).

The process of hazard identification is predominantly qualitative. To identify hazards, search is conducted on relevant data sources. To obtain information on these hazards, search is conducted in the scientific literature, in databases from the food industry, government agencies or relevant international organizations, and through elicitation of expert opinion. (CAC, 1999).

5.3 Exposure assessment

According to the Codex Alimentarius Comission (1999), this step includes an assessment of the magnitude of actual or anticipated human exposure. In MRA, the exposure assessment might consider the potential extent contamination of food by microorganisms or its toxins, as well as the dietary information. The unit of food that is of interest, i.e., the portion size in most/all cases of acute illness should be specified in this step.

Several factors should be considered when conducting an exposure assessment. The frequency of contamination of foods by the pathogenic agent, its level in foods over time are greatly influenced by intrinsic characteristics of the pathogen, microbiological interactions in the food environment, the initial contamination of the pathogen in the raw material, the type and extent of sanitation and process controls, the methods of processing, packaging, distribution and storage of the foods, and food preparation steps (i.e, cooking and storage). Patterns of food consumption should also be considered in this step. They are determined by the consumers' socio-economic and cultural background, their ethnicity, age (population demographics), regional location, preferences and behaviour. Specific groups, such as infants, children, pregnant women, elderly or immunocompromised individuals, who can be more susceptible to infection or illness than the rest of the population, should also be included in an exposure assessment whenever possible (Gerba et al., 1996). The possibility of food handlers

to act as a source of contamination should also be taken into account, as well as the extent of contact between their hands and the product, and the potential impact of abusive environmental factors like time and temperature (CAC, 1999).

It is important to realize the existing dynamics of the levels of microbial pathogens in food. While levels might be low during food processing due to proper time and temperature controls, they can increase considerably in the following processes of the food chain if these conditions are not controlled (Heredia et al., 2008). Therefore, in the exposure assessment, the transmission of the hazard is often modeled through the food pathway, which includes a series of processes from the source of the raw ingredients (e.g., the farm) to the moment of consumption (Nauta, 2000). Modelling hazard transmission usually involves separating the food pathway into unit operations. These describe the treatments applied to the ingredients during their conversion into food and what is their impact in the hazard. Data is found through direct observation (e.g., surveillance studies measuring the changes in hazards in a production environment), laboratory experimentation (eg. simulation in the laboratory of the processes occurring during manufacturing), or mathematical modelling based on established physicochemical principles (e.g., thermodynamic relationship associated with a heat process) to allow an identification of an input–output relationship for each operation unit (Notermans et al., 1998).

In the exposure assessment, scenarios simulating intervention measures in the food pathway can predict a range of possible exposures. Interventions measures can include the effects of processing (i.e, hygienic design, cleaning and disinfection) but also time/temperature and other conditions of the food history. Food handling, consumption patterns, regulatory controls, and surveillance systems can also be modeled in this step (CAC, 1999).

5.3.1 Predictive microbiology

It is known that numbers of bacteria in food can change at all stages of the food pathway, depending on several factors. The presence, growth, survival, or death of microorganisms in food, can be influenced by the type of food and the way it is handled, stored, and processed. Predictive microbiology tools allow to estimate changes in bacterial numbers (Heredia et al., 2008). The study from McMeekin et al. (1993) describes predictive microbiology as a scientific discipline where microbial behaviour (e.g., growth, survival, inactivation) is predicted as a function of environmental factors. In predictive microbiology, mathematical models are used to predict in a quantitative estimate the increase or decrease in concentrations of microorganisms in food products (Bott, 2014).

Predictive models can be classified as primary or secondary level models, depending on the degree of precision and sensitivity to environmental factors (Whiting & Buchanan, 1994).
Primary models are usually developed in first place to determine the impact of the responses of interest, like the maximum specific growth rate, lag phase duration, or death rate. Afterwards, a secondary model is constructed, which shows the dependence of these factors on environmental conditions. Primary and secondary models can be combined to obtain tertiary models with the use of advanced software packages and expert systems (Buchanan & Whiting, 1998: Ross et al., 2000).
Predictive microbial models allow an estimation of changes in the concentration of the microorganism in the different step of the food pathway, like production, processing and preparation. This information is used by food manufacturers and food safety authorities for developing and evaluating production processes, determining shelf life and setting food safety standards (Bott, 2014; Foegeding, 1997).

5.4 Hazard characterization (dose-response)

Hazard characterization describes the severity and duration of adverse effects that may result from the ingestion of a microorganism or its toxin in food, either qualitatively or quantitatively. The severity and duration of adverse health effects can vary a lot within the population after exposure to food borne pathogens, due to several factors. Factors that should be considered in a hazard characterization are the virulence characteristics of the pathogen, the numbers of cells ingested, the general health and immune status of the hosts, and the attributes of the food that alter microbial or host status. In addition, the likelihood that an individual becomes ill due to an exposure to a foodborne pathogen depends on the integration of host, pathogen, and food matrix effects (Buchanan et al., 2000).
Mathematical models have been used to describe dose-response relationships. (Buchanan et al., 2000) The exponential (Table 1, Eq. (1)) and beta-Poisson (Table 1, Eq. (2)), are two of the most used models to describe dose-response relationships. They were initially introduced by (Haas, 1983), and have been used over the years, for different classes of biological agents (Buchanan et al., 1997; Coleman & Marks, 1998; Crockett et al., 1996; Haas, 1983; Medema et al., 1996; Rose & Gerba, 1991).
In the exponential model it is assumed that the probability of a cell causing infection is independent of dose, whereas in the beta-Poisson it is assumed that infectivity is dose dependent.(Haas, 1983).

Expert elicitation could be included in a hazard characterization when a dose-response relationship is not known. These can give insight in considering factors like infectivity and to devise ranking systems that describe severity and/or duration of disease.(CAC, 1999).

Figure 3: Mathematical models (Exponential and Beta-Poisson) that have been used to empirically describe dose-response data for foodborne pathogenic bacteria. (From: Buchanan et al., (2000)).

1. Exponential (Haas, 1983):

$$P_i(d) = 1 - \exp(-rd)$$

where: $P_i(d)$ = probability of infection at dose (d)
d = dose (CFU)
r = model parameter specific for each pathogen

2. Beta-Poisson (Haas, 1983):

$$P_i(d) = 1 - (1 + d/\beta)^{-\alpha}$$

where: $P_i(d)$ = probability of infection at dose (d)
d = dose (CFU)
α = model (infectivity) parameter
β = model (shape) parameter

5.5 Risk characterization

In this step, qualitative or quantitative information obtained from the hazard identification, hazard characterization, and exposure assessment are combined to obtain a risk estimate. This results in a qualitative or quantitative estimate of the probability and severity of adverse health effects in a given population, taking uncertainties in to account and describing them accordingly. Information can be obtained through suitable data or expert judgements. Estimates can be assessed through further comparison with independent epidemiological data that relates hazards to disease prevalence. It is important to stress that the final assessment of the risk will be influenced by all the assumptions made in the previous steps, as well as variability and uncertainty. (CAC, 1999)

5.5.1 Variability and uncertainty

Variability represents the true heterogeneity of the population of subjects considered, and it can be observed and quantified. As it is a consequence of the physical system, it can not be reduced by adding further measurements. Stochasticity and inter-individual variability are two types of variability. Stochasticity occurs when heterogeneity is a consequence of randomness (eg. the result of throwing a dice). Inter-individual variability describes the differences between individuals of a population (eg. the variability of children's heights in school class), which is influenced by genetics, nutrition, other environmental conditions, but also some randomness. Uncertainty is described as a lack of perfect knowledge, meaning that increasing knowledge by implementing further measurements, for example, can help to reduce it (Anderson & Hattis, 1999; Bott, 2014; Murphy, 1998);

In addition to accounting for variability and uncertainty, it is important to assess the influence of the estimates and assumptions used in a risk assessment. In a quantitative risk assessment this is achieved by conducting sensitivity and uncertainty analyses (CAC, 1999).

6. Quantitative risk assessment

Risk assessment can be qualitative or quantitative (CAC, 1999). Qualitative risk assessments are descriptive or categorical treatments of information. Quantitative assessments are mathematical expressions or models that describe the probability of occurrence of an adverse effect. Models are simplified representations of a part of reality (Coleman & Marks, 1998; Alban et al., 2002; Haas et al., 2014; Havelaar et al., 2008). Considering the objectives of the risk assessment and the data available, two types of mathematical models are combined: exposure assessment models and dose-response models (Heredia et al., 2008).

If quantitative information and resources are available, it is preferable to conduct a quantitative risk assessment. If, however, there are limitations regarding data, time and / or other resources, conducting a qualitative risk assessment may be the only option. Qualitative assessments may be used for an initial evaluation of a food safety issue, to evaluate if a specific risk is significant enough and requires more detailed analysis. In a quantitative risk assessment, by combining the likelihood and magnitude of each harm, one is able to quantify the risk of each individual getting ill (Alban et al., 2002; Haas et al., 2014; Nauta & Havelaar, 2008).

Quantitative Microbiological Risk Assessment (QMRA) is used to support several risk management purposes (Nauta et al., 2009). Although it can be used to assess the human health risk associated with the ingestion of microorganisms in food in terms of estimated human

incidence, it is generally not the best tool to use for this purpose. This is explained due to the uncertainty present in the exposure assessment and the dose-response relation. For this purpose, epidemiological data may offer better tools to assess the baseline risk estimate. (Havelaar et al., 2007).

QMRA is also used to assess the effects of intervention measures in the food pathway that aim to reduce risk. If QMRA covers all the food chain, it allows risk managers to compare the evaluation of control measures implemented in all the food chain (Nauta et al., 2009). Intervention measures are assessed considering absolute risks and relative risk reduction. Absolute risk is the risk estimate itself, which will be a lower risk after implementation of an intervention measure. Relative risk is the ratio of the lower incidence estimate and the incidence estimate without intervention. The lower the relative risk, the higher the risk reduction is in an intervention scenario compared to the baseline. As relative risk is associated with less uncertainty than absolute risk, it is considered a more valuable statistic than absolute risk estimates (Nauta et al., 2005b; Duarte et al., 2016).

The efficiency of intervention measures implemented in the food production chain can also be assessed when conducting a QMRA. By incorporating costs of intervention measures, the risk assessor is able to compare the effectiveness of different intervention in reducing human health risks. A balance between costs and benefits of intervention measures can be achieved when costs are incorporated in a QMRA. (Havelaar et al., 2007; Nauta & Havelaar, 2008).

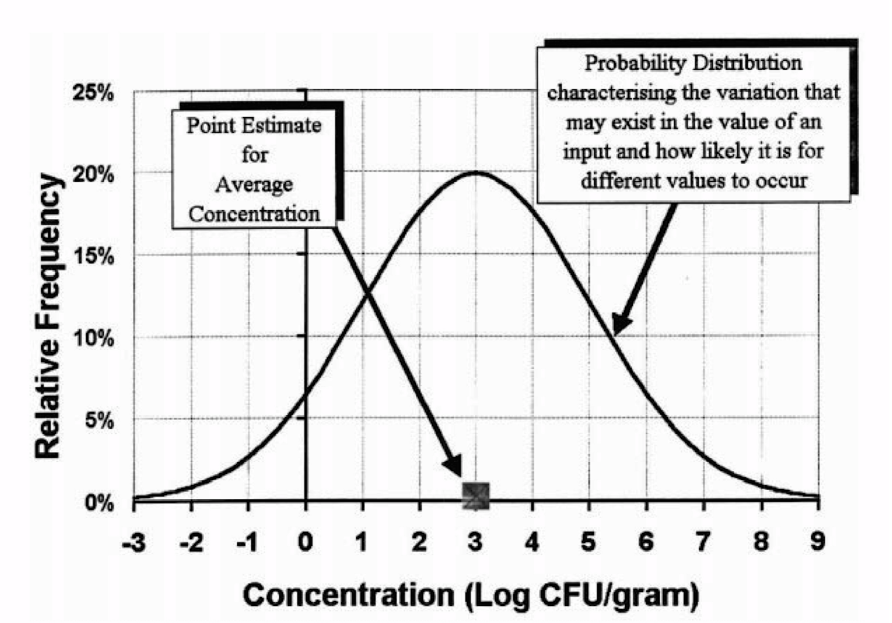
6.1 Stochastic versus deterministic risk assessment

Risk assessment models can be deterministic/'point estimate' or stochastic/'probabilistic'. Deterministic models use single values to describe the inputs that impact the final outcome. Therefore, the risk estimate that they produce is a single value, which can be the average or a worst-case scenario, for example. Probabilistic models use probability distributions to describe the inputs that influence the final outcome. For that reason, the risk estimate produced is a distribution describing a range of values of the risks that an individual or a population might experience. (Heredia et al., 2008; Lammerding & Fazil, 2000). Probability distributions can be attributed based on empirical data, understanding the basic biological phenomena, or on expert opinion elicitation, when there are no other sources of information (Vose, 1998).

Despite the fact that probabilistic models are more complex than deterministic models, they are the preferred method of choice for quantitative risk assessments. This is because variability and uncertainty, described previously in this study, are ignored in a deterministic risk assessment by using a single value to describe the risk (Lammerding & Fazil, 2000). The

necessity of including variability and uncertainty is based on the fact that it is unlikely that microbial risks that affect human health are uniformly distributed and that 'average' episodes or events are likely to cause significant problems (Potter, 1994). Risk management decisions should take into account the extremes of the distributions, the likelihood of occurrence of such events and who might be affected. Figure 4 illustrates the difference between a point-estimate and a probability distribution to describe an input. In this hypothetical example, the graph describes the concentration of a pathogen in a unit of food. The deterministic approach specifies a single value that a parameter could take, while the stochastic approach specifies a range of values that a parameter can take, and how frequently these different values can occur. When a single point value is used to describe a complete data set, it is observed that a considerable amount of information is lost. (Lammerding & Fazil, 2000).

Figure 4: Comparison between a point-estimate and a probability distribution to characterize a data set (From: Lammerding & Fazil (2000)).



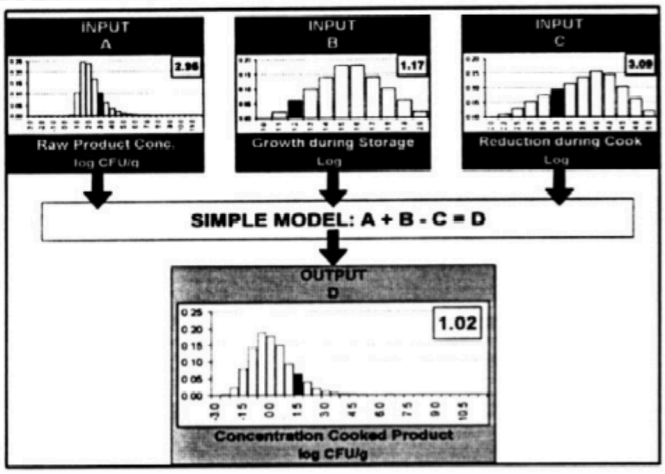
6.1.1 Monte Carlo simulation

Monte Carlo Simulation is an alternative to using analytical techniques to evaluate a risk assessment, as this can be a tedious task, even for a single model. Is it described as a numerical technique, specifically suited for computer applications, which randomly selects a single 'point-estimate' value from each of the probability distributions applied for each input parameter. The selected single values are used to calculate a mathematical solution, which is specified in the risk assessment model. Each result is stored, and the sequence is repeated for a several number of times (iterations). In each iteration a different value is selected, according to the defined probability for each one. Values associated with higher probabilities are more likely to occur, and therefore are selected more frequently. The result of a Monte Carlo analysis is an output described by a frequency distribution of values, which combine the ranges and the frequencies of each input parameters (Lammerding & Fazil, 2000).

Fig. 5 describes a simplified illustration of a Monte Carlo simulation for a hypothetical exposure assessment.

Figure 5: Illustration of Monte Carlo simulation performed to determine the concentration of a pathogen in a food product. There are three inputs: (A) is the concentration of a pathogen in the raw food product, log CFU/g; (B) is the log growth that can occur during transport and storage; and (C) is the log reductions that occur when the product is cooked to various degrees of doneness. (From: Lammerding & Fazil (2000)).

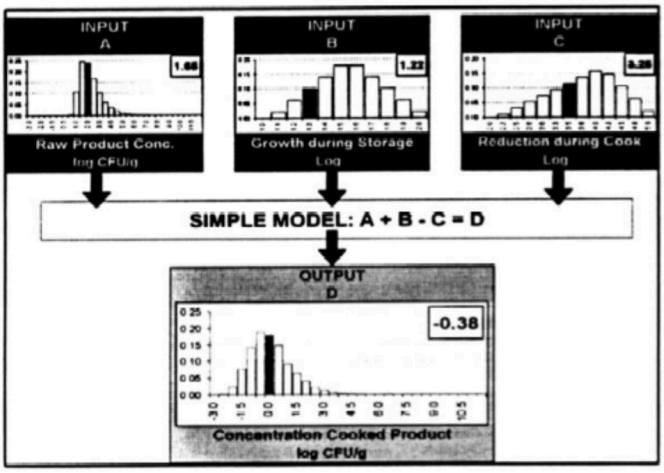
ITERATION 1



In the first iteration, a value is selected from each of the probability distributions for the three inputs (A, B and C). The three values are applied to the mathematical model and the result for the output, D, is calculated and stored.

A = 2.95 log CFU/g (raw product at process)
B = 1.17 log growth (during storage/transport)
C = 3.09 log reduction (during cooking)
Therefore
D = 1.02 log CFU/g (cooked product at home)

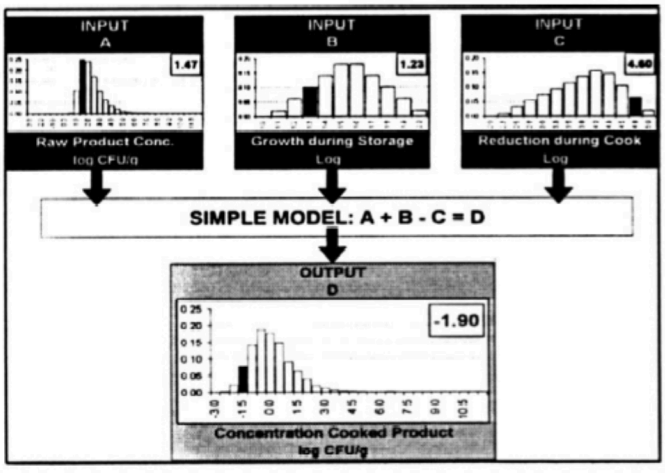
ITERATION 2



In the second iteration, another set of values is randomly selected from the input probability distributions. The values are selected so that values defined as having a high probability of occurring (tall bars) are selected more frequently than those values defined as being possible but rare (short bars).

A = 1.65 log CFU/g (raw product at process)
B = 1.22 log growth (during storage/transport)
C = 3.25 log reduction (during cooking)
Therefore
D = -0.38 log CFU/g (cooked product at home)

ITERATION n



In the next iteration:

A = 1.47 log CFU/g (raw product at process)
B = 1.23 log growth (during storage/transport)
C = 4.60 log reduction (during cooking)
Therefore
D = -1.90 log CFU/g (cooked product at home)

The process is repeated thousands of times. The outputs are combined, resulting in a probability distribution for D, which reflects the range of values that are possible and how likely it is that they occur.

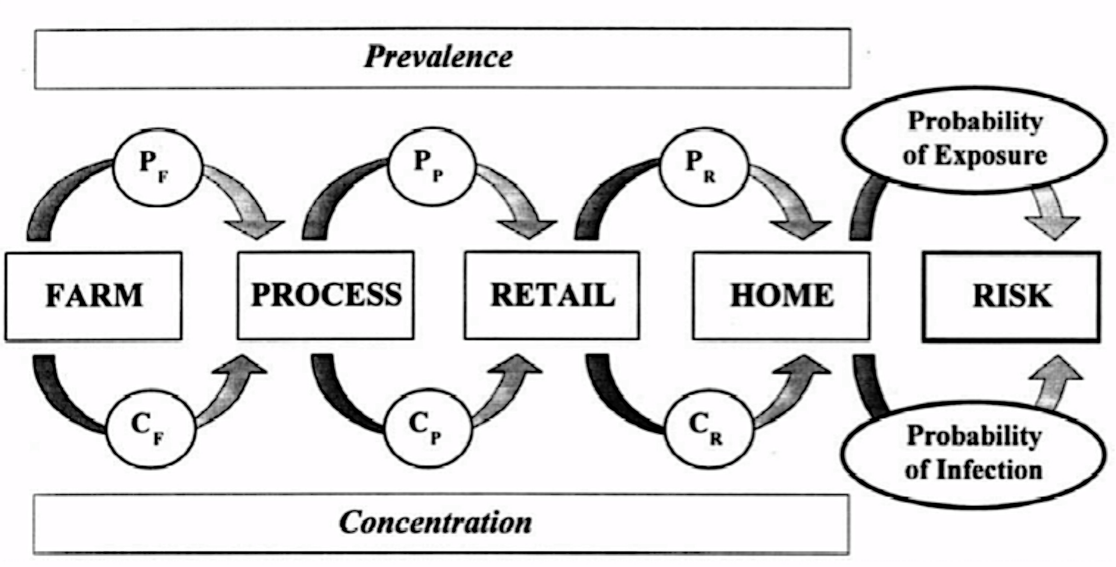
II. The importance of consumer phase models in quantitative microbiological risk assessment

1. Introduction

Risk assessments that consider the influence of various factors in the food pathway, from food production until food consumption and the effect on human health, provide valuable information for risk managers. These have been described as farm-to-fork risk assessments, and have been conducted for different food-pathogen combination (Buchanan et al., 2000; Lammerding & Fazil, 2000; Nauta & Christensen, 2011).

Figure 6 shows the elements of a farm-to-fork risk assessment. The changes in prevalence and concentration of a pathogen are assessed from the farm level through processing and retail to final consumption by the consumer (Lammerding & Fazil, 2000).

Figure 6: Elements of a 'farm-to-fork' risk assessment. Factors that influence or alter the prevalence and/or concentration at the farm (P_F and C_F), during food processing (P_P, C_P), retail storage and handling (P_R, C_R) and in the home (P_H, C_H) are described in the exposure assessment (From: Lammerding & Fazil (2000)).



Some risk assessments start at primary production, while others may start at a later stage of the food chain (Buchanan et al., 2000; Nauta & Christensen, 2011). As described before in this study, according to the *Codex Alimentarius*, all risk assessments should include an exposure assessment. As this step describes the probability of intake of pathogens by consumers (CAC, 1999), it is necessary that risk assessments include models or assumptions that account for the consumer phase. By considering this phase, an essential link between previous phases in the food chain and the dose-response relation is established.

The consumer phase is the part of the food chain following the production and retail, when the consumer transports, stores, prepares and consumes produced food (Nauta & Christensen, 2011). This step is different from all the other steps of the food chain, because it is associated with high variability aspects of human behaviour (Nauta et al., 2009).

Food safety managers have lower interest in this step than in the other stages of the food chain, because in this phase, food can no longer be controlled by food authorities and there is no possibility of enforcing controls by legislation. In this step, proper and safe food handling is the consumer's responsibility, and the only form of control at this point is through education of the population and provision of other types of information (Fischer et al., 2005; Hill et al., 2011).

Representative quantitative data on consumer food handling practices to use in a risk assessment remains scarce. This is due to the difficulty of obtaining unbiased, representative data on human behaviour in the domestic setting in general (Redmond & Griffith, 2003), which restrains research in the consumer phase. Also, because there is high variability in food handling practices, and cultural and social differences between the considered population groups in relation to food preparation practices need to be accounted for (Nauta et al., 2009).

Despite these challenges, a consumer phase model (CPM) needs to be included in a risk assessment, to allow an evaluation of the effectiveness of intervention measures in food production and processing, in terms of human health risk.

To this day, several CPMs have been developed. These may differ substantially in terms of complexity depending on the purpose of the QMRA and the availability of data. Some may only include a few simplifying assumptions, while others may describe in detail the food handling practices, their frequencies of occurrence and use plenty of data sources (Zwietering, 2009).

With the current rapid progress of science and technology, rapid risk management measures of acute public health events are indispensable. These can reduce or prevent disease in affected populations, reduce negative social and economic consequences, and enable implementation of appropriate and timely control measures (WHO, 2012).

This implies that in an acute public health event, it is desired that risk assessment models are built in short period of time, in order to provide a fast scientific ground for further implementation of control measures and more efficient risk management.

An important question to answer in the development of CPMs is to what extent these models need to include a detailed description of the processes that may result in exposure (Nauta & Christensen, 2011). The study from Nauta et al., (2009), suggests that "There is no alternative but for a probabilistic approach to risk assessment models of the consumer phase.". This implies that there is no possibility other than using stochastic CPMs to obtain accurate risk estimates when modelling the consumer phase.

2. Purpose of the project

The purpose of this study is to compare the results given by seven published stochastic consumer phase models found in the literature for *Campylobacter, Salmonella* and *Listeria monocytogenes*, with two simpler modelling techniques, which don't include variation. The modelling techniques are compared by means of absolute risk estimates and relative risk estimates, by simulating the effect on the risk of six hypothetical intervention scenarios in the food production chain.

This project aims to identify particular scenarios in which simpler surrogates that don't include variation estimate similar results to stochastic CPMs. If so, modelling the consumer phase of the exposure assessment stage of a risk assessment would become a faster and simpler process, ideal for use in acute public health disease outbreaks (WHO, 2012; Zwietering, 2009).

The objectives of the study are summarised in the following order below:

- To assess absolute risk estimates and relative risk estimates (by simulation of intervention measures) for different stochastic consumer phase models found in the literature for three pathogens: *Campylobacter, Salmonella* and *L. monocytogenes*.
- To assess absolute risk estimates and relative risk estimates (by considering intervention scenarios) of two simpler approaches that don't include variation: "a-factor" model (Duarte et al., 2016) and deterministic consumer phase models.
- To compare the results in terms of absolute risk estimates and relative risk estimates of the modelling approach using a stochastic CPM, with the approach using an "a-factor" model and a deterministic consumer phase model.

3. Materials and methods

Literature research on Quantitative Microbiological Risk Assessment was conducted in order to obtain published Consumer Phase Models for three microorganisms responsible for food borne zoonoses: *Campylobacter* in broiler chicken (Calistri & Giovannini, 2008; Christensen et al., 2001; Nauta et al., 2008), *Salmonella* in pork (Hill et al., 2011; Messens et al., 2009; Mürmann et al., 2011) and *Listeria monocytogenes* in cold-smoked-salmon (Berjia, 2013; Pouillot & Lubran, 2011). The information was obtained through searching in websites containing large databases of scientific articles and journals, such as Science Direct, Pub-med, Dtu-Find it and Research Gate.

Seven models in total were chosen after the literature research. The simpler models were implemented in Monte Carlo software @Risk 5.5 (Palisade), while the more complex models were provided by my supervisor, Maarten Nauta. These models had already been implemented in the software in a project performed by Mungai, (2015). Dose-Response models specific for each pathogen were also found in the literature.

3.1 Modelling approach using stochastic CPMs

The modelling approach used for all the stochastic CPMs in this study can be divided in two main parts. The first part describes the transport of the food by the consumer, its storage, preparation and consumption. Not all the models describe all these stages. Some models only include the preparation and consumption, while others also include the transport and storage. Each model is described in detail further in this study. The second part includes the dose-response relationship.

- Overview of the modelling approach:

3.1.1.1 Input distributions

The inputs applied for each CPM are described in this step. The relevant parameters are specified, as well as their corresponding notation and distribution.

3.1.1.2 Consumer phase model description

In this step, the consumer phase model is briefly described and its formulas and diagrams are provided.

3.1.1.3. Dose-response model

The dose-response model applied for each pathogen is described and the corresponding formula provided.

The final output after implementation of the Dose-Response Model is the probability of illness of each individual after exposure to a certain dose of contaminated food with different pathogens.

3.1.1 *Campylobacter* CPMs

Three CPMs for *Campylobacter* in chicken meat were selected from a study performed by Nauta and Christensen (2011): Nauta CPM (Nauta et al., 2008), Christensen CPM (Christensen et al., 2001) and Calistri CPM (Calistri & Giovannini, 2008). A recent review of the performance of these models is given in a study by Chapman et al., (2016). The models describe the transfer and survival of *Campylobacter* in raw chicken meat, from retail purchase to meal consumption, with *Campylobacter* originating from that meat.

Human exposure to *Campylobacter* can occur as a result of undercooking or cross-contamination during meal preparation. It is largely accepted that all *Campylobacter* are inactivated through heating of the meat and that proper hygiene measures can prevent cross-contamination. An episode of undercooking is assumed to be unlikely and may be especially important when whole carcasses are considered (FAO/WHO, 2009).

In all *Campylobacter* CPMs described, cross-contamination is considered to be the dominant route of exposure, which does not necessarily occur via the meat itself but via a ready-to-eat (RTE) food item (e.g. salad)

For this reason, at the point of exposure, *Campylobacter* may be in the prepared meat itself, but more likely in a RTE food at side dish, like a salad prepared on the same cutting board as the raw chicken meat (Nauta et al., 2009).

3.1.1.1- Input distribution

According to the approach used by (Nauta & Christensen, 2011), the input distributions (initial concentration, prevalence and portion size) are assumed to be the same for all the considered *Campylobacter* CPMs. The same dose-response model was also applied for the three CPMs. By using this approach, the difference of the risk estimates is expected to be only attributable to differences inherent to the models, which allows further comparison of the results.

The input distributions are described as:

- Concentration of *Campylobacter* at retail, C_{ret} (in cfu/g), is defined by a normal distribution of the logs (mean= 1.5, standard deviation= 1.2).
- With prevalence p_{prev} of 0.25, log C_{ret} is sampled from a normal distribution with mean= 1.5 and standard deviation= 1.2, otherwise C_{ret} is 0 cfu/g.
- Portion sizes, $w_{c,}$ are sampled from a lognormal distribution with mean= 189 g, standard deviation=127, and maximum portion size of 1 kg.

The number of *Campylobacter* in cfus on one portion of consumed meat, $N_{portion}$, is defined by the Poisson distribution described below:

$$N_{portion} \sim Poisson\ (C_{ret} \times W_c) \quad (1)$$

where ~ represents "is a sample from," so $N_{portion}$ varies with different portions.

3.1.1.2- Consumer phase models

The purpose of each CPM is to describe the probability distribution of ingested doses consequential to the distribution of $N_{portion}$ for a large set of meat portions. Empirical distributions to model uncertainty and variability are used to characterize the parameters in all the CPMs related to *Campylobater* described below. All the parameters of the CPMs are described in more detail in Annex II.

- Nauta CPM

The study from Nauta et al (2008) relied on other studies (De Jong et al., 2008; Van Asselt et al., 2008) to use *Lactobaccili* as a tracer for *Campylobacter* in an observational study. The study considered bacterial transfer from inoculated chicken breast fillets to salads in ready-to-eat chicken salad prepared at home by consumer volunteers. From this study, a data set describing the variability of transfer rates from raw meat to salad was obtained, and an empirical distribution was used for the transfer rates p_{tr} (described in Appendix in Nauta & Christensen (2011)). If p_{tr} is variable and sampled from this distribution, this allows:

$$d \sim Binomial\left[N_{portion}, p_{tr}\right]. \quad (2)$$

where ~ represents "is a sample from," so $N_{portion}$ varies with different portions, and p_{tr} represents the probability of a single cfu from the portion ending up in the dose.

- Christensen CPM

This model is a simplified version of the CPM by (Christensen et al., 2001), which still gives very similar results to the original version. It describes the transfer of bacteria from raw chicken to equipment (board or knife), and from equipment to cooked chicken or accompanying salad.

$$d \sim Binomial\ [N_{portion}, tCE, (tEC \times fCC\ + (1 - tEC \times fCC) \times tES \times fCS)] \qquad (3)$$

where,

- The transfer rate chicken to equipment tCE, is defined by ten to the power of minus Pert distribution, with minimum value=1; most likely value= 2 and maximum value= 6. ~10^−Pert(1,2,6)
- The transfer rate equipment to chicken, tEC is defined by ten to the power of minus Pert distribution, with minimum value=1; most likely value= 2 and maximum value= 6. ~10^−Pert(1,2,6)
- The frequency of chicken to chicken contamination, fCC is considered to be =1.
- The transfer rate equipment to salad, tES is defined by defined by ten to the power of minus Pert distribution, with minimum value=1; most likely value= 2 and maximum value= 6. ~10^−Pert(1,2,6)
- The frequency of chicken to salad contamination, fCS is considered to be =1.

- Calistri CPM

For this model, Calistri and Giovaninni (2008) use the same transfer rate data as the Brynestad CPM (Luber et al., 2006), but derived different empirical distributions. It describes the transfer of bacteria from raw chicken to equipment (board or knife) and to hands, and from hands and equipment to cooked chicken or accompanying salad:

$$d \sim Binomial\left[N_{portion}, tCE \times tER \times fCE + (1 - tCE \times tER \times fCE)\ tCH \times tHR \times fCH\right], \qquad (4)$$

where:

- The transfer rates tCE, tER, tCH and tHR are defined by empirical distributions. Variables are summarized in Table 4 in Calistri and Giovaninni (2008).
- Chicken to environment contamination fCE, is defined by a Bernoulli distribution that returns =1 with probability p_{CE} = 0.124, otherwise returns = 0.
- Chicken to hand contamination fCH, is defined by a Bernoulli distribution that returns =1 with probability p_{CH}= 0.259, otherwise returns = 0.

The output from Nauta, Christensen and Calistri CPMs return the probability distribution of the number of cfu's ingested (i.e. the dose) consequential to the distribution of $N_{portion}$ over a large set of meat portions.

3.1.1.3- Dose-response model

For *Campylobacter*, a Beta-Poisson model was used to describe the probability of infection from an ingested dose *d* (WHO/FAO, 2009):

$$p(infection|dose) = 1 - \left(1 + \frac{d}{\beta}\right)^{-\alpha} \quad (5)$$

where α = 0.145 and β= 7.59.

As the classic Beta-Poisson model describes the response in terms of probability of infection, and the desired output is the probability of illness Q_{ill} , a standard multiplier of 0.33 is used, based in studies from Black et al. (1988) and from WHO/FAO (2009). Subsequently, the mean of the probability of illness was considered for comparison of the results.
After implementation of each CPM and the dose-response model, the final outputs obtained are the probabilities of illness for each individual after exposure to a certain dose *d*, the number of cfus of *Campylobacter* in contaminated salad.

3.1.2- *Salmonella* CPMs

3.1.2.1- Input Distribution

Following a similar approach used for *Campylobacter* CPMs, the same inputs (initial concentration, prevalence) and dose-response model were applied to all *Salmonella* models, to assure that the risk estimates are only attributable to differences in the models. The inputs applied to all the models were obtained from Messens et al. (2009), while the dose-response model used was obtained from FAO/WHO Risk assessments of *Salmonella* in eggs and broiler chickens (WHO/FAO, 2002).

The input distributions are described as:

- Concentration of *Salmonella* at retail (log cfu/g), is defined by a Normal distribution (mean= 1.4; standard deviation= 0.7)
- Prevalence, p is = 0.12
- Weight of portion, is defined by a Normal distribution with mean= 93 and SD=14.83

3.1.2.2- Consumer phase models

- Bollaerts CPM:

Bollaerts CPM is part of a QMRA conducted to evaluate the risk of human salmonellosis through household consumption of fresh minced pork meat in Belgium (Messens et al., 2009). It is included in module 6 of the exposure assessment, where the process of preparing meat in households is simulated, considering that the meal partially consists of a portion of minced pork meat and another food item. It is assumed that pork meat is cooked in all situations, while the other food item is sometimes consumed raw.

To account for variability and uncertainty, empirical distributions are used to describe the growth of *Salmonella* during transport and storage at home, cross contamination during meal preparation of a ready to eat meal (from meat to the other food product via cook's hands or carving board), and microbial inactivation during cooking.

It is assumed that cross-contamination to another food item occurs via the cook's hands (step 7 on Fig. 7) or via the carving board used to manipulate the minced meat (step 8 on Fig. 7).

Cross-contamination via the carving board is firstly modelled as transfer of *Salmonella* from the meat after manipulation by hands to the board, and from the board to another food item

(step 9 on Fig. 7). After food handling, the number of *Salmonella* present on the minced meat is described in step 5 of the diagram in Figure 7.

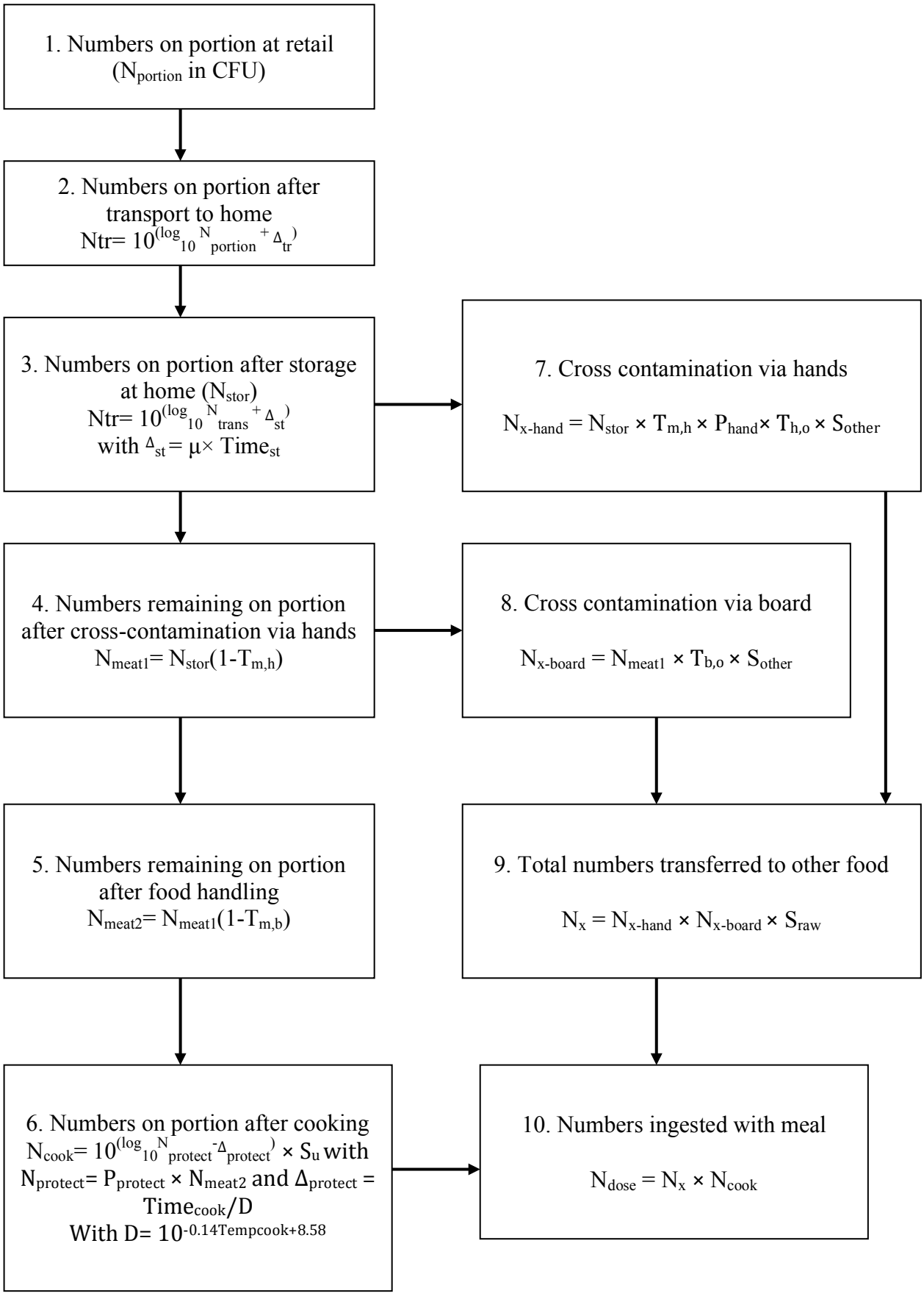
The effect of undercooking is modelled in this CPM, and it is assumed that only a proportion of *Salmonella* cells in the protected area will survive to the cooking process (step 6 in Fig. 7).

The final output of the model is the number of *Salmonella* ingested at the moment of consumption, which includes the sum of the number of *Salmonella* in the minced meat that survived cooking and the number transferred to another food item consumed raw (step 10 in Fig. 7).

Bollaert's CPM output gives the number of *Salmonella* on the minced pork meat which survived the cooking process, as well as the number transferred to another food item that is consumed raw.

All the parameters of the CPM are described in more detail in Annex II.

Figure 7: Schematic representation of Bollaerts CPM. See details in Table 10 in Annex II. (Adapted from: Messens et al., (2009) and Mungai (2015)).



- EFSA CPM

EFSA CPM is part of a full risk assessment model for *Salmonella* in the pork production chain in selected European Union member states (Hill et al., 2011). This CPM can also be found in a more recent study by Swart et al., (2016). In this risk assessment three types of pork meat are considered: minced pork, pork cuts and fermented sausages. For the purpose of this study, the only model considered was for pork cuts.

Empirical distributions are used to model variability and uncertainty related to the processes of transport, storage and meal preparation of 10,000 portions of pork cuts.

The growth of *Salmonella* in transport and storage (at home) is modeled by using time and temperature parameters (Baranyi's dynamic growth model).

The preparation of pork cuts is modelled in the most part as a cross-contamination process, from pork products to ready-to-eat food (eg. salads or bread). Transmission of *Salmonella* can occur within the household from raw meat juice present in surfaces, equipment or personnel carrying the bacteria.

The parameters below are considered to describe cross-contamination:

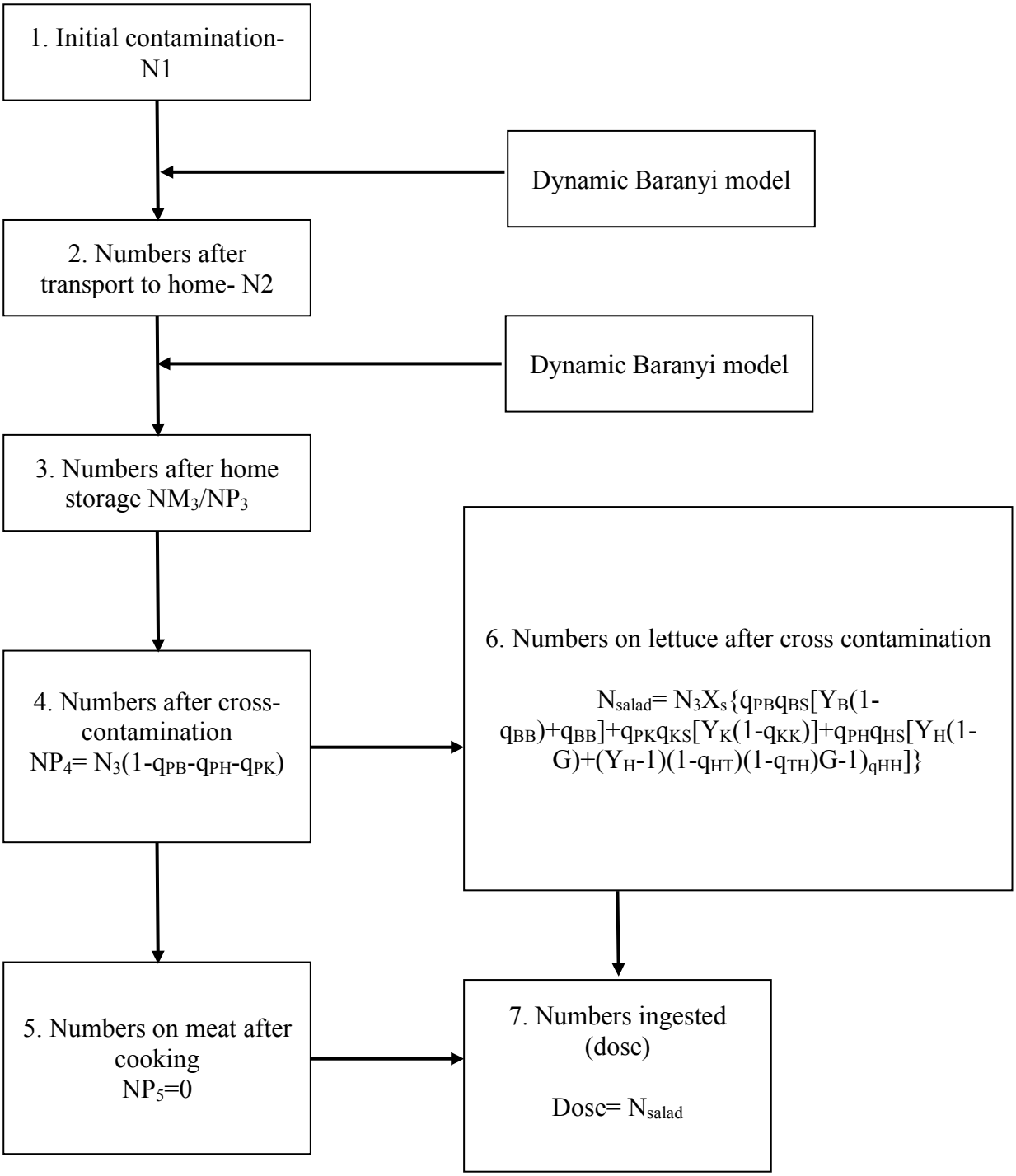
- Transfer between pork cuts, knife, cutting board and hands through cutting.
- Cross-contamination between hands and tap, through washing of the board.
- Cross-contamination between hands and tap , through washing the knife
- Cross-contamination between hands and tap, through washing hands.
- Cutting the salad

Inadequate cooking is not considered in this CPM due the assumption that the heating will destroy all *Salmonella* cells, since they are present exclusively on the outside of the product (step 5 in Fig. 8).

EFSA's CPM output is the number of cfus of *Salmonella* in each portion of pork cuts at the point of consumption (step 7 in Fig. 8).

All the parameters of the CPMs are described in more detail in Annex II.

Figure 8: Schematic representation of EFSA CPM. See details in Table 11 in Annex II. (Adapted from: Hill et al., (2011) and Mungai (2015)).



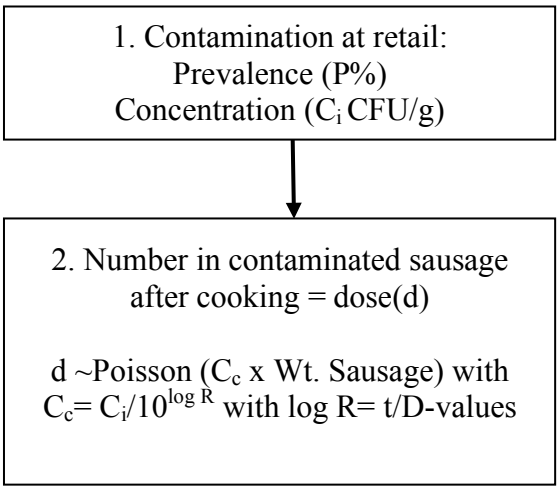
- Murmann CPM

This CPM is part of a quantitative microbiological risk assessment to estimate the risk of *Salmonella* infection through the consumption of fresh pork sausages prepared at barbecues in Porto alegre, Brazil (Mürmann et al., 2011). In this study, the thermal effect of cooking during meal preparation is modelled by using time-temperature profiles measured during the project. Empirical distributions are used to account for variability and uncertainty related to the cooking time (min), internal temperature (ºC), and Log cycle reduction, except for the D-values, which were calculated deterministically.

Murmann's CPM output is the ingested number of *Salmonella* cells in one contaminated cooked pork sausage (i.e the dose).

All the parameters of the CPMs are described in more detail in Annex II.

Figure 9: Schematic representation of Murmann CPM. See details in Table 12 in Annex II (Adapted from: Mürmann et al., (2011) and Mungai (2015).



3.1.2.3. Dose-response model

The Beta-Poisson dose response model used for the three *Salmonella* CPMs (Equation 5) was the dose-response model used for the FAO/WHO (2002) risk assessments of *Salmonella* in eggs and broiler chickens, with $\alpha = 0.1324$ and $\beta = 51.45$. These parameters were estimated from outbreaks with several serovars and were considered to be more appropriate than parameters from feeding trials with single serovars. As it is obtained from outbreak data, the resulting Beta Poisson model can be interpreted to describe the probability of illness from a certain dose. (WHO/FAO, 2002). Subsequently, the mean of the probability of illness was considered for comparison results.

The outputs of the Beta Poisson dose-response model are the mean probabilities of illness after consumption of a certain dose of fresh minced pork meat in Belgium for Bollaerts CPM, pork cuts for EFSA CPM and fresh pork sausages for Murmann CPM.

3.1.3- *Listeria monocytogenes* CPM

A consumer phase model was obtained from the exposure assessment of a study performed by Berjia (2013), a risk-benefit assessment of cold-smoked salmon (CSS). In this project, the risk of *Listeria monocytogenes* is evaluated against the benefits of the intake of omega-3 fatty acids in Denmark. The model described is deterministic, including parameters described by fixed values, which don't account for variability and uncertainty. As the purpose of this project is to use stochastic models that act as a basis for comparison with simpler alternatives -"a-factor" and deterministic CPMs- the original version of the model will only be described later in this study. In this step, changes were made to the model with the purpose of adding variability to it, in order to obtain a stochastic model. Stochastic growth models, primary and secondary, were obtained from Pouillot & Lubran (2011) and combined with Berjia CPM. In addition, the inputs storage temperature, storage time and growth rate were substituted to variable values through implementation of distributions.

3.1.3.1- Input distribution

The inputs for Berjia CPM are described below:

- In the study of (Berjia, 2013), the initial concentrations of *L. monocytogenes* in cold smoked salmon are {0.5: 1.5: 2.5: 3.5} and their prevalences {0.28: 0.05: 0.01: 0} (Jørgensen and Huss 1998). As stated previously, the input of the initial concentration was modified in order to add variability to the model. It is now defined by a Normal distribution with mean=1,2189 log cfu/g and standard deviation= 0.8 (ILSI 2010). The parameters used to define the Normal Distribution (i.e. the mean and standard deviation) were obtained using different methods. The mean was calculated by adding the products of the initial concentrations and their respective prevalences, stated in (Jørgensen and Huss 1998). The value of standard deviation (SD) ($\log_{10}$)= 0,8 was chosen in this situation based on ILSI (2010), as it is usually used as a default value to describe the standard deviation of a batch, when no better data or more specific information on a batch is available.
- The portion size ingested is a fixed value of 23 grams.

3.1.3.2 Consumer phase models

- Berjia CPM (adapted)

Storage time, storage temperature, growth rate and lag-time are modeled in this CPM. Storage temperature (T_i) was obtained from Table A5.3 in p. 260 (WHO, 2004). Duration of the storage is described by a normal distribution with mean=14 days (Berjia, 2013) and a standard deviation of 3,5 (Expert opinion). Growth rate (μ) was obtained from (Pouillot & Lubran, 2011), consisting of a primary and secondary models.

The primary growth model is the model #4 in the study by Pouillot and Lubran (2011), with alternative point estimates for μ_{ref} = 6.19 (d^{-1}) and T_{min} = -1.18 C:

$$X_{end,i} = min\left(X_{0,i} + \frac{\mu_i}{log_e(10)} \times \max\left(t_{i\,i} - \lambda_i, 0\right), MPD_i\right) \quad (6)$$

The inputs for this three-phase linear model (Buchanan et al., 1997) are described below:

- $X_{0;i}$ ($\log_{10}$ cfu/g) is the concentration of *L. monocytogenes* in the CSS at the beginning of the storage; μ_i is the specific growth rate of L. *monocytogenes* per day (d^{-1}); t_i the duration of the storage; λi= 0 is the lag time (d); MPD_i the maximum population density ($\log_{10}$ cfu/g).
- The model number 4 (FDA/FSIS, 2003) used no lag=0 days, a constant MPD_i= 7.27 $\log_{10}$ cfu/g) (Delignette-Muller et al., 2006), and a square root model (Ratkowsky et al., 1982) as a secondary model for μ,

$$\mu_i = \mu_{ref} \times \left(\frac{T_i - T_{min}}{T_{ref} - T_{min}}\right)^2 \quad (7)$$

with a constant m_{ref} =6.19 d^-1(FDA/FSIS, 2003) for T_{ref} = 25 C (Delignette-Muller et al., 2006) and a constant T_{min}= -1.18 C (FDA/FSIS, 2003).

All the parameters of the CPMs are described in more detail in Annex II.

3.1.3.3- Dose response model

An exponential dose-response model is used to estimate the probability of infection by *L. monocytogenes* (FAO/WHO 2004):

$$Pinf = 1 - e^{-rD} \quad (8)$$

where,

- P_{inf} is the probability of severe illness, D is the number of *L. monocytogenes* consumed, and
  r is the parameter that defines the dose–response relation for the population being considered.
- For healthy population, *r* is 2.37×10^{-14} (FAO/WHO 2004).
- For susceptible population, *r* is 1.06×10^{-12} (FAO/WHO 2004).

The susceptible group is, on average, ~40 times more susceptible than the 'healthy' population. The result is not the probability of infection, but the probability of severe illness.
The final output is the probability of severe illness caused by *L. monocytogenes* after consumption of cold smoked salmon (CSS).

3.2 Modelling approach using a surrogate "a-factor"

A surrogate for a CPM used in a study about the effect of carcass decontamination on the risk for consumers was found in the literature (Duarte et al., 2016). In this study, during processing after slaughter (i.e. particularly during cooking), the number of *Salmonella* is assumed to decrease according to a reducing factor *a*. The "a-factor" summarises the effect of transfer, growth, cross contamination and survival of *Salmonella* in pork meat in a constant value.
Taking into account that the process of building a stochastic model for the consumer phase can be complex and time consuming, using a fixed value as a surrogate would allow simpler and faster results, but one doesn't know how valid it is. Although this approach has only been used for *Salmonella* in the literature, it was possible to implement it to the other pathogen's consumer phase models as well.

3.2.1 Modelling approach

The modelling approach using the "a factor" in this study can be divided in two main parts. The first part describes the transfer, growth, cross contamination and survival, summarised in a constant value. The second part includes the dose-response relationship.

- Overview of the modelling approach:

3.2.1.1 Input distributions

The inputs applied for each "a-factor" remain the same as the ones used for the stochastic models, including variation.

3.2.1.2 "a-factor"

In this step, the "a-factor" summarises the effect of transfer, growth, cross contamination and survival in a constant value. One "a-factor" correspondent to each stochastic CPM is calculated.

3.2.1.3 Dose-response model

The dose-response modesl applied for each pathogen remain the same as the ones used for the stochastic models.

The final output after implementation of the dose-response model is the probability of illness of each individual after exposure to a certain dose of contaminated food with different pathogens.

3.2.2 Calculating the reducing "a-factor"

In the study by Duarte et al. (2016), the number of *Salmonella* is assumed to decrease during the processing after slaughter (i.e. particularly during cooking) by a reducing factor *a*. The ingested dose (D) per serving is a product of the multiplication of the "a-factor" (*a*) and the concentration at retail (C_{retail}) of each pathogen.

For *Campylobacter*, obtaining an "a-factor" surrogate for each CPM is calculated by solving Equations (9) and (10):

$$Risk = \int \left(1 - \left(1 + \frac{a \times C_{retail}}{\beta}\right)^{-\alpha}\right) f(C_{retail}) d(C_{retail}) \quad (10)$$

$$Risk = \int \left(1 - \left(1 + \frac{D}{\beta}\right)^{-\alpha}\right) f(D) d(D) \quad (9)$$

Equation (9) includes the Beta Poisson dose-response model for the probability of illness (*Risk*) from the distribution of the doses ingested *D*, with $\alpha = 0.145$ and $\beta = 7.59$ (WHO/FAO, 2009). Equation (9) is used to calculate the variable *Risk*, based on the distribution of doses *D* obtained from the stochastic CPM.

By assuming that the distribution of doses *D* is defined by the multiplication of the "a-factor" by the distribution of concentrations at retail, $D = a \times C_{retail}$, it follows that Equation (10) must be true.

In Equation (10), the values of the distribution of concentrations at retail $f(C_{retail})$, alpha α and beta β, and *Risk* are known. In the study performed by Duarte et al. (2016) for *Salmonella*, the *Risk* was obtained from epidemiological data. In this particular study, the *Risk* is the value of the absolute risk estimate obtained from the risk assessments conducted with the stochastic CPMs for *Campylobacter*, in Equation (9) (values are described in Table 2).

Then the "a-factor" is the only unknown value, which can be found by using the Excel Solver add-in (values described in Table 1).

Table 1: Details of the inputs used to estimate the "a-factor" for Nauta, Christensen and Calistri CPMs.

Inputs	**Nauta CPM**	**Christensen CPM**	**Calistri CPM**
Risk	0.0039	0.0040	0.0022
C_{retail} **– Normal distribution (** μ, σ)	μ=1.5; σ=1.2 cfu/g	μ=1.5; σ=1.2 cfu/g	μ=1.5; σ=1.2 cfu/g
β	7.59	7.59	7.59
α	0.145	0.145	0.145

Table 2: Description of the values of each "a-factor" obtained for Nauta, Christensen and Calistri CPMs.

	Nauta CPM	**Christensen CPM**	**Calistri CPM**
"a-factor" value	0.00213	0.00222	0.00085

After *a* is obtained, the absolute risk estimate, *Risk,* for a baseline scenario is calculated through the Equation (11), where *p* represents the prevalence of *Campylobacter* (0.25) described in the stochastic CPMs (Nauta & Christensen, 2011). Alternative risks for identical intervention scenarios used for stochastic CPMs were also calculated, as well as the relative risk of each intervention scenario.

$$Risk = \int \left(1 - \left(1 + \frac{a \times Cretail}{\beta}\right)^{-\alpha}\right) f(Cretail) d(Cretail) \times p \qquad (11)$$

For *Salmonella*, obtaining an "a factor" surrogate for each CPM is calculated by solving the Equations (9) and (10).

Equation (9) includes the Beta Poisson dose-response model for the probability of illness (*Risk*) from the distribution of the doses ingested *D*, with α = 0.1324 and β= 51.45 (WHO/FAO, 2002). This equation is used to calculate the variable *Risk*, based on the distribution of doses *D,* obtained from the stochastic CPM. By assuming that the distribution of doses *D* is defined by the multiplication of the "a-factor" by the distribution of concentrations at retail, $D = a \times C_{retail}$, it follows that Equation (10) must be true.

In Equation (10), the values of the distribution of concentrations at retail $f(C_{retail})$, alpha *α* and beta *β*, and *Risk* are known. In the study performed by Duarte et al. (2016) for *Salmonella*, the *Risk* was obtained from epidemiological data. In this particular study, the *Risk* is the value of the absolute risk estimate obtained from the risk assessments conducted with the stochastic CPMs for *Salmonella*, in Equation (9) (values are described in Table 3).

Then "a-factor" is the only unknown value, which can be found by using the Excel Solver add-in (values are described in Table 4).

Once obtaining *a*, the absolute risk estimate, *Risk*, for a baseline scenario was calculated through the Equation (11), where *p* represents the prevalence of *Salmonella* (0.12) described in the CPM by Messens et al., (2009). Alternative risks for identical intervention scenarios used for stochastic CPMs were also calculated, as well as the relative risk of each intervention scenario.

Table 3: Details of the inputs used to estimate the "a-factor" for Bollaerts, EFSA and Murmann CPMs.

Inputs	Bollaerts CPM	EFSA CPM	Murmann CPM
Risk	0.0001	0.0013	0.0003
C_{retail} – **Normal distribution (** μ, σ)	μ=1.4; σ=0.7 cfu/g	μ=1.4; σ=0.7 cfu/g	μ=1.4; σ=0.7 cfu/g
β	51.45	51.45	51.45
α	-0.1324	-0.1324	-0.1324

Table 4: Description of the values of each "a-factor" obtained for Bollaerts, EFSA and Murmann CPMs.

	Bollaerts CPM	EFSA CPM	Murmann CPM
"a" factor value	0.00474	0.0657	0.0122

For *L. monocytogenes*, obtaining an "a-factor" surrogate for the CPM by Berjia (2013) is calculated by solving Equations (12) and (13):

$$Risk = \int \left(1 - e^{(-r \times D)}\right) f(D) d(D) \tag{12}$$

$$Risk = \int \left(1 - e^{(-r \times \alpha \times Cretail)}\right) f(Cretail) d(Cretail) \tag{13}$$

Equation (12) includes the exponential dose-response model for the probability of illness (*Risk*) from the distribution of the doses ingested *D*, with $r = 2.37 \times 10^{-14}$ and $r = 1.06 \times 10^{-12}$ for healthy and susceptible population, respectively (Ross et al., 2009).

Equation (12) is used to calculate the variable *Risk*, based on the distribution of doses *D,* obtained from the stochastic CPM. By assuming that the distribution of doses *D* is defined by the multiplication of the "a-factor" by the distribution of concentrations at retail, $D = a \; x \; C_{retail}$, it follows that Equation (13) must be true.

In Equation (13), the values of the distribution of concentrations at retail $f(C_{retail})$, *r*, and *Risk* are known. In the study performed by Duarte et al. (2016) for *Salmonella*, the *Risk* was obtained from epidemiological data. In this particular study, the *Risk* is the value of the absolute risk estimate obtained from the risk assessments conducted with the stochastic CPMs for *L. monocytogenes*, in Equation (13) (values described in Table 5).

Then "a-factor" is the only unknown value, which can be found by using the Excel Solver add-in (values described in Table 6).

Table 5: Details of the inputs used to estimate the "a-factor" for Berjia CPM (adapted).

Inputs	**Berjia CPM (adapted) Healthy Population**	**Berjia CPM (adapted) Susceptible Population**
Risk	3.56249^{-7}	1.59303^{-5}
C_{retail} **– Normal distribution (** μ, σ)	μ=16.55; σ=0.8 cfu/g	μ=16.55; σ=0.8 cfu/g
***r*- Healthy Population**	$r = 2.37 \times 10^{-14}$	$r = 2.37 \times 10^{-14}$
***r*- Susceptible Population**	$r = 1.06 \times 10^{-12}$	$r = 1.06 \times 10^{-12}$

Table 6: Description of the values of each "a-factor" obtained for Berjia CPM (adapted).

	Berjia CPM (adapted) Healthy Population	**Berjia CPM (adapted) Susceptible Population**
"a" factor value	432513	432682

Once obtaining *a*, the absolute risk estimate, *Risk*, for a baseline scenario was calculated through the Equation (14), where *p* represents the prevalence of *L. monocytogenes* (0.385) described in the CPM by Berjia (2013). Alternative risks for identical intervention scenarios used for the stochastic CPM were also calculated, as well as the relative risk of each intervention scenario.

$$Risk = \int \left(1 - e^{(-r \times \alpha \times Cretail}\right) f(Cretail) d(Cretail) \times p \tag{14}$$

3.3 Modelling approach using deterministic CPMs

As the results obtained by using a single fixed value *a* proved to be significantly different from the results of the stochastic CPMs in all the scenarios considered, another approach was adopted. This alternative consisted in using a deterministic CPM, and would allow to simplify and speed-up the process of building a CPM, when compared to a stochastic CPM.

The purpose of this second approach is to evaluate the performance of a deterministic CPM in estimating absolute risks and relative risks of intervention scenarios compared to stochastic CPMs. For *Campylobacter* and *Salmonella*, the deterministic CPMs were obtained by modifying all the original stochastic CPMs found in the literature by eliminating all the existing variation. This was achieved by substituting all the parameters described by distributions in the CPMs by fixed values, which would be their means. For *Listeria*, a deterministic CPM was found in the literature by Berjia (2013), and was used for this purpose.

It is important to stress that the parameters used in the deterministic CPMs and the stochastic CPMs described previously in this study are the same (except for Berjia CPM). The difference between them remains solely in using fixed values for the inputs in the deterministic models and distributions for the inputs in the stochastic models. For this reason, these models are not described in detail in this step, but can be found in Annex II.

3.3.1 Modelling approach

The modelling approach used for all the deterministic CPMs in this study can be divided in two main parts. The first part describes the transport of the food by the consumer, its storage, preparation and consumption. Not all the models describe all these stages. Some models only include the preparation and consumption, while others also include the transport and storage. Each model is described in detail further in this study, in Annex II. The second part includes the dose-response relationship.

- Overview of the modelling approach:

- Input distributions

The inputs applied for each CPM were the same as the ones used for the stochastic CPMs, including variation.

- Consumer phase model description

The details of the deterministic CPMs are described in Annex II.

- Dose response model

The dose-response model applied for each pathogen is the same as the one used for the correspondent stochastic model.

The final output after implementation of the dose-response model is the probability of illness of each individual after exposure to a certain dose of contaminated food with different pathogens.

4. Models performance analysis

4.1 Absolute risk estimates

The absolute risk estimates, or absolute mean probabilities of illness, were obtained for all the modelling approaches after running 100,000 iterations in Monte Carlo software @Risk 5.5 (Palisade).

4.2 Relative risk estimates

The effect on the risk of six hypothetical intervention scenarios in the food production chain was evaluated by means of relative risk, assuming as a baseline a scenario without intervention (Duarte et al., 2016).

Six intervention scenarios simulate changes in the concentration of the pathogen at retail (mean and standard deviation) and changes in the pathogens prevalence. It is important to stress that the control measures are assumed to be implemented somewhere along the food production chain, at primary production or during industrial processing. Therefore, they do not affect the CPM itself (Nauta & Christensen, 2011). Some scenarios are more realistic than others, however, the purpose of this project is solely to compare the performance of different stochastic CPMs with simpler approaches that don't include variation in a diverse range of scenarios.

Each scenario representing the potential effect of one or more control measures is described below:

- Scenario 1: 0,5 log reduction in the mean of the concentration at retail.
- Scenario 2: 1 log reduction in the mean of the concentration at retail.
- Scenario 3: 0,5 decrease in the standard deviation of the concentration at retail.
- Scenario 4: 0,5 increase in the standard deviation of the concentration at retail.
- Scenario 5: Decreasing the prevalence at retail by 10%.
- Scenario 6: Increasing the prevalence at retail by 50%.

Scenarios 1 and 2 represent practical control measures that are believed to affect the mean concentrations of the pathogen, like for example, decontamination of broiler meat during industrial processing to reduce *Campylobacter* (Gellynck et al., 2008; Havelaar et al., 2007).

Scenario 3 simulates, for example, implementation of protocols where heavily contaminated meat products are diverted from the fresh broiler meat production chain to reduce

Campylobacter, which results in a decrease of the standard deviation (Nauta et al., 2009; Nauta & Havelaar, 2008).

Scenario 4 represents, for example, a situation of protocols where less control samples are taken, which results in more variation in the concentration of the pathogen in food, and increases the standard deviation (Mungai, 2015).

Scenario 5 simulates, for example, actions such as logistical slaughter and/or processing that would reduce cross contamination between contaminated and non-contaminated lots/batches with *Salmonella*. (Mungai, 2015).

Scenario 6 represents, for example, implementation of protocol with irregular environmental testing for *L. monocytogenes* within the ready-to-eat food industry (Tompkin & Scott, 1999).

Absolute risk estimates for risk reduction scenarios were obtained by implementing the models in @risk software using 10,000 iterations. For easier comparison of results, the relative risk (*RR*) of each intervention scenario was calculated using Equation (15), by dividing the alternative risk by the predefined baseline risk:

$$RR = \frac{Q_{ill}^{*}}{Q_{ill}} \tag{15}$$

Where Q_{ill} * is the absolute risk estimate of each intervention scenario and Q_{ill} is the absolute risk estimate. The lower the relative risk, the higher the risk reduction in scenario compared to the baseline.

4.3 A comparison of the absolute risk estimates

The performance of the simpler models was first evaluated in terms of absolute risk estimates (probability of illness per meal ingested), for further comparison with the ones obtained by stochastic CPMs. Even when a stochastic CPM is used, this parameter holds in general large uncertainty. This can be cause of the uncertainty about the CPM itself, the uncertainty of the dose-response and the uncertainty of the concentration at retail. Nevertheless, the purpose of this project is merely to compare the performance of two simpler models that don't include variation with stochastic CPMs.

4.3.1 “a-factor” versus stochastic

In all seven models, the absolute risk estimates calculated using an “a-factor” were very similar to the ones estimated by the corresponding stochastic CPMs.

4.3.2 Deterministic versus stochastic

Deterministic CPMs give mixed results in the different models from the three pathogens. For *Campylobacter* models, deterministic CPMs estimate higher absolute risks than both the “a-factor” and the stochastic CPMs (Fig. 10). For *Salmonella* models, Deterministic CPMs estimate very similar absolute risks when compared to the stochastic CPMs and the “a-factor” (Fig. 11). On the other hand, in Berjia model the Deterministic CPM estimates lower absolute risks than the stochastic CPM and the “a-factor” (Fig. 12).

Figure 10: Illustration of the absolute risks (probability of illness per meal ingested) obtained using three different modelling approaches for *Campylobacter*: stochastic CPM (eg. Nauta STO), “a-factor” surrogate (eg. Nauta “a”), and deterministic CPM (eg. Nauta DET).

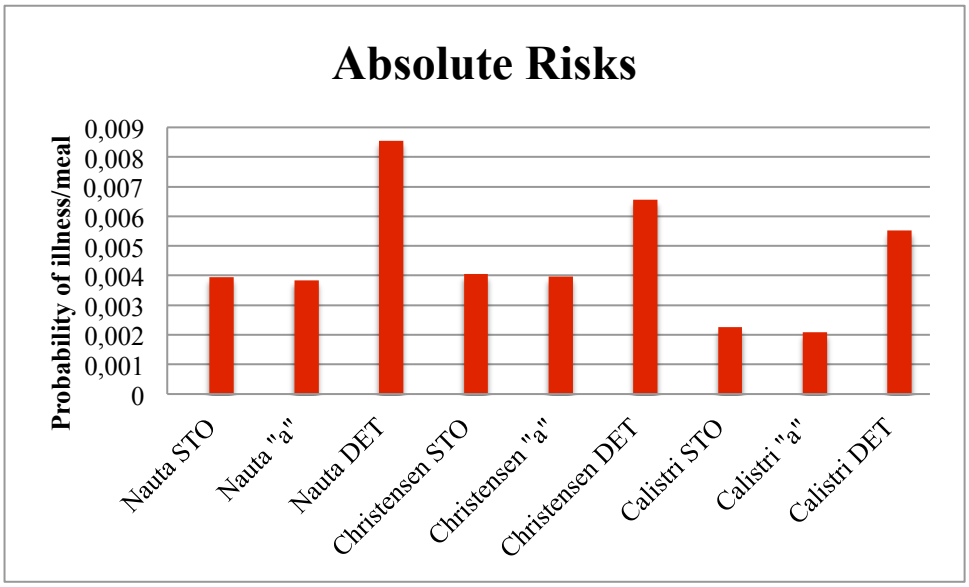


Figure 11: Illustration of the absolute risks (probability of illness per meal ingested) obtained using three different modelling approaches for *Salmonella*: stochastic CPM (eg. EFSAa STO), "a-factor" surrogate (eg. EFSA "a"), and deterministic CPM (eg. EFSA DET).

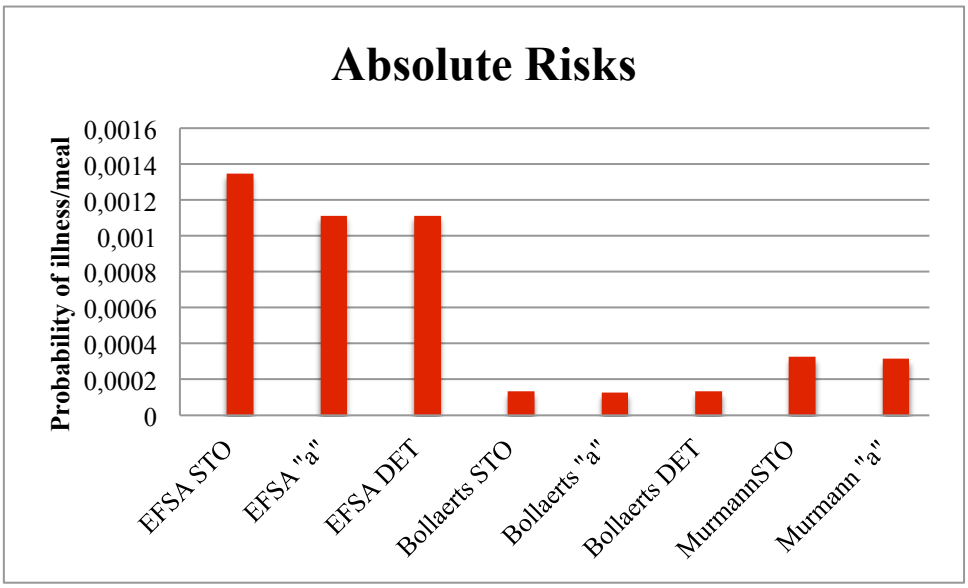
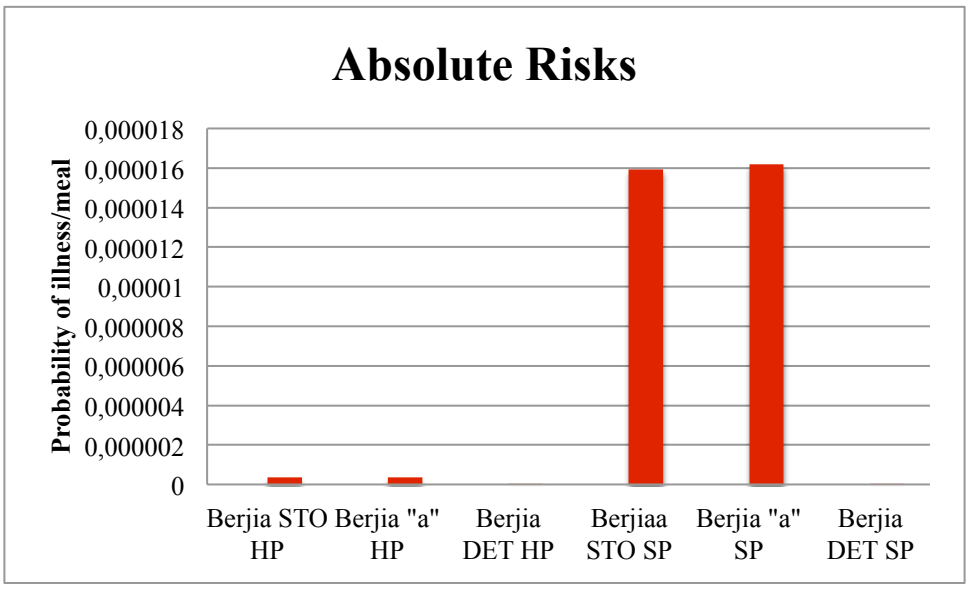


Figure 12: Illustration of the absolute risks (probability of illness per meal ingested) obtained using three different modelling approaches for *L. monocytogenes*: stochastic CPM (eg. Berjia STO), "a-factor" surrogate (eg. Berjia "a"), and deterministic CPM (eg. Berjia DET).



4.4 A comparison of the relative risk estimates

Comparing the performance of the simpler models with the stochastic CPMs in terms of relative risks is very useful to estimate the impact of control measures in the food production chain. There is also lower level of uncertainty than the absolute risk estimates because the uncertainties will be cancelled out when the absolute risk estimates are divided in Equation (15).

4.4.1 *Campylobacter* CPMs

Figures 13, 14, 15 and 16, show the relative risks estimated by all *Campylobacter* stochastic CPM´s, surrogates "a factor" and deterministic CPMs, after simulating the implementation of an intervention scenario of 0.5 and 1 log reduction in the mean of the initial concentration and 0,5 reduction and increase in the standards deviation of the initial concentration.

4.4.1.1 "a-factor" versus stochastic CPMs

For Nauta, Christensen and Calistri CPMs:

Figures 13 and 14 show the relative risk estimated by all *Campylobacter* stochastic CPM´s, surrogates "a factor" and deterministic CPMs, after simulating the implementation of an intervention scenario of 0.5 and 1 log reduction in the mean of the initial concentration.

In a scenario of a 0.5 log reduction in the mean of the initial concentration, we observe (in Fig. 13) that using surrogates "a factor" results in a lower relative risk estimates (or higher risk reduction) compared to using stochastic CPMs. The results show that if a surrogate "a factor" is used to calculate the risk of this intervention scenario, the risk will be underestimated when compared to using a stochastic CPM. Similar results for an intervention scenario of 1 mean log reduction are illustrated in Fig. (14). It was found that using surrogates "a factor" will result in lower RR estimates (higher risk reduction) when compared to using stochastic CPMs, meaning the risk will be underestimated if an "a factor" is used.

To investigate the effect of changes in the variation, a 0.5 reduction and a 0.5 increase in the standard deviation of the initial concentration scenarios were considered.

The results illustrated in Fig. (15) show that for a 0.5 reduction in the SD, "a factor" surrogates estimate lower relative risks (or a higher risk reduction) when compared to a stochastic CPMs. On the other hand, if we increase the variation by increasing the standard deviation by 0.5, using surrogates "a factor" will estimate higher relative risks (Fig.16).

4.4.1.2 Deterministic CPMs versus stochastic CPMs

For Nauta, Christensen and Calistri CPMs:

When Deterministic CPMs are used, in a scenario of a 0.5 log reduction in the mean of the initial concentration we obtain very similar relative risk estimates as the ones given by stochastic CPM (Fig. 13). For a 1 mean log reduction in the initial concentration scenario the relative risk estimates are still very similar between the deterministic and stochastic CPMs (Fig. 14).

Whenever a Deterministic CPM is used either in a scenario of 0.5 increase or 0.5 decrease in the SD, we obtain very similar relative risks to the stochastic CPMs (Figs. 15 and 16).

Figure 13: Illustration of the relative risks obtained by simulation of an intervention scenario of 0.5 log reduction in the mean of the concentration at retail of *Campylobacter*. Results were calculated using three different modelling approaches: Stochastic CPM (eg. Nauta STO), "a-factor" surrogate (eg. Nauta "a"), and Deterministic CPM (eg. Nauta DET).

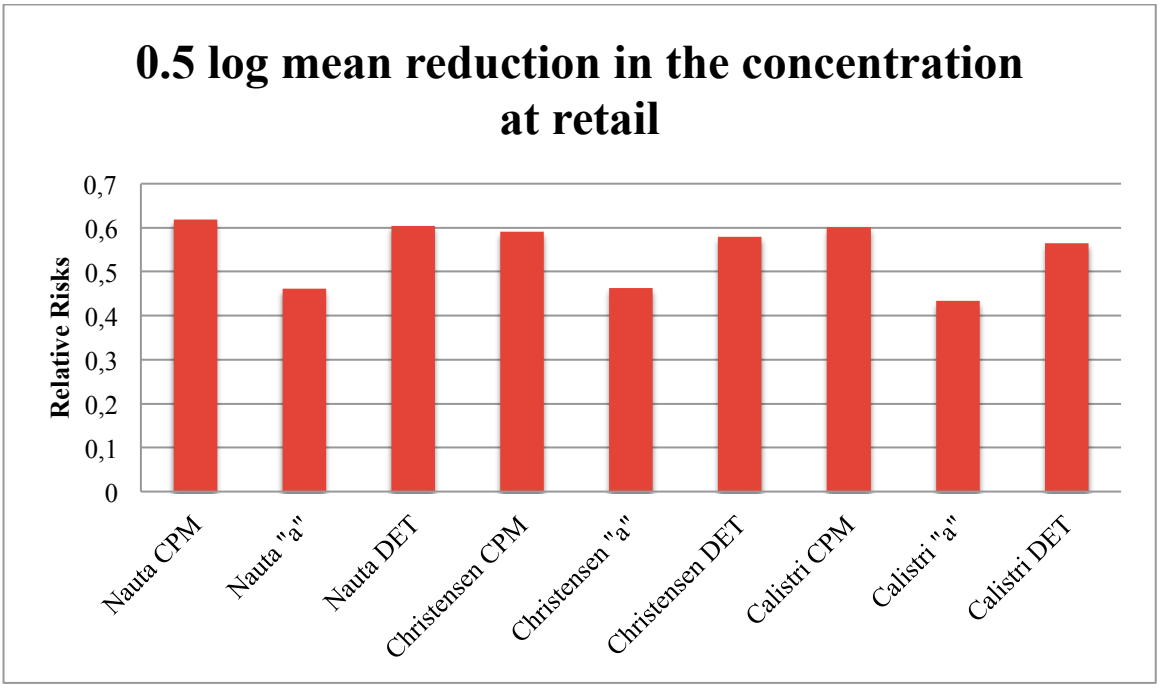


Figure 14: Illustration of the relative risks obtained by simulation of an intervention scenario of 1 log reduction in the mean of the concentration at retail of *Campylobacter*. Results were calculated using three different modelling approaches: Stochastic CPM (eg. Nauta STO), "a-factor" surrogate (eg. Nauta "a"), and Deterministic CPM (eg. Nauta DET).

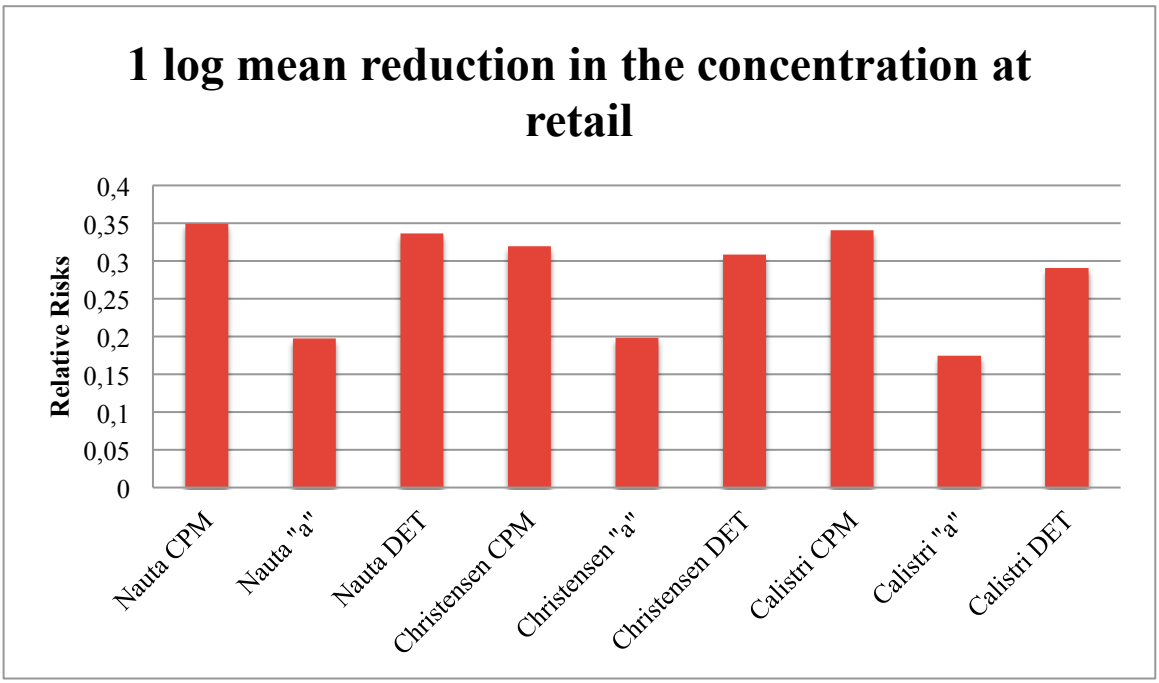


Figure 15: Illustration of the relative risks obtained by simulation of an intervention scenario of 0.5 reduction in the standard deviation of the concentration at retail of *Campylobacter*. Results were calculated using three different modelling approaches: Stochastic CPM (eg. Nauta STO), "a-factor" surrogate (eg. Nauta "a"), and Deterministic CPM (eg. Nauta DET).

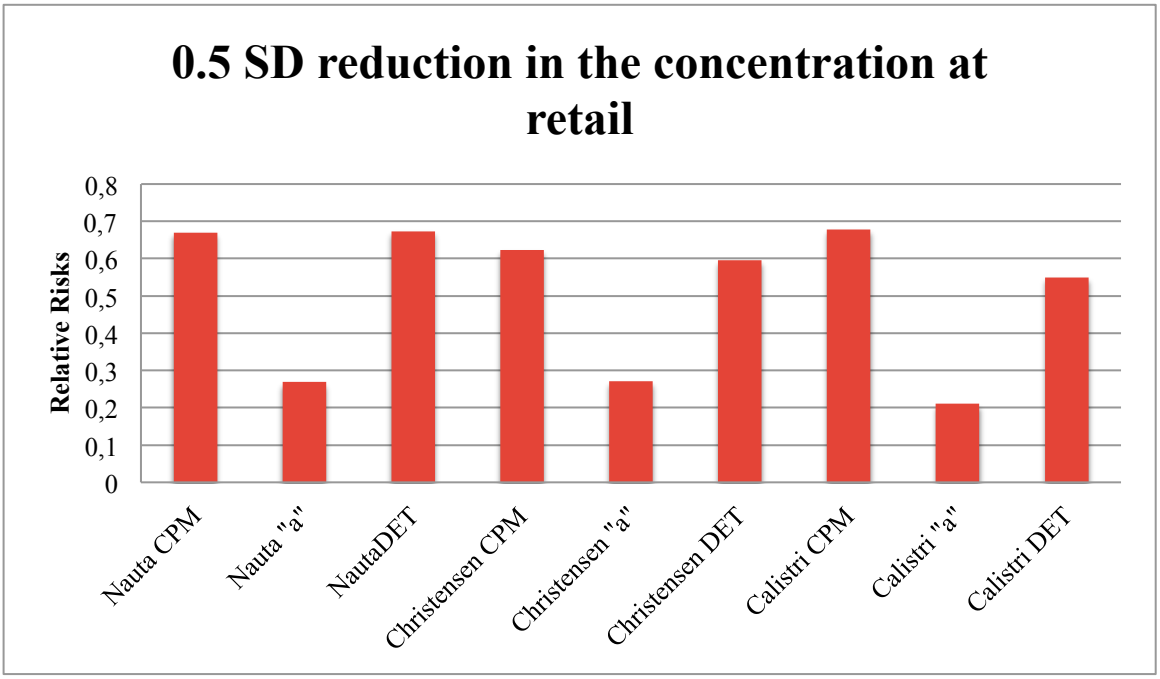
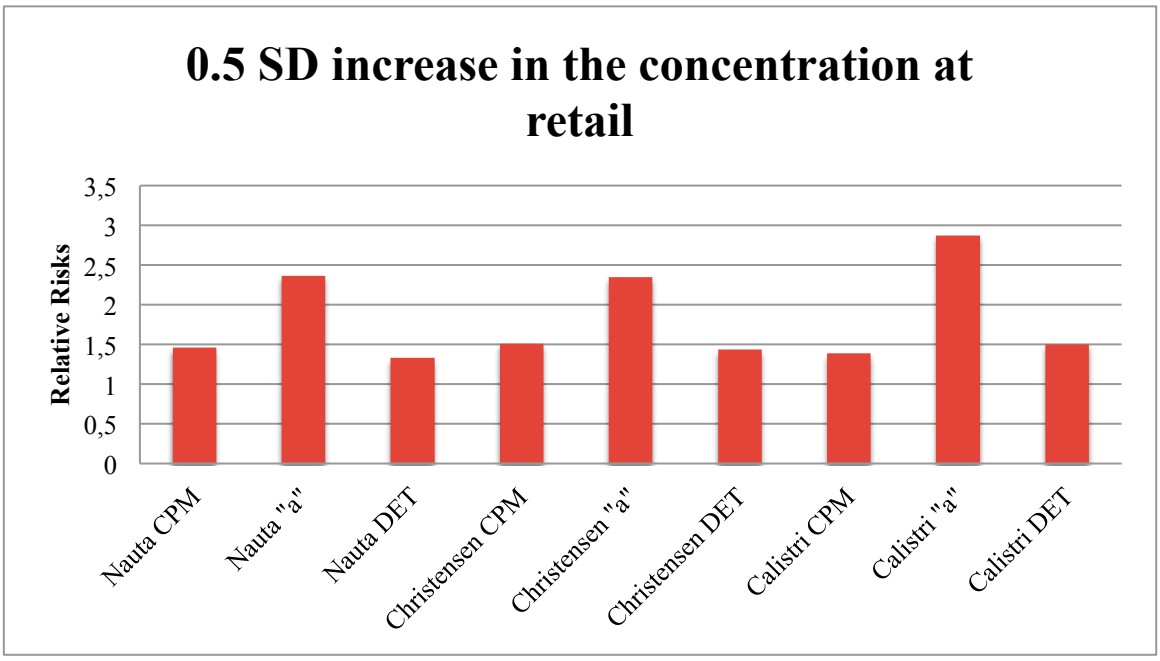


Figure 16: Illustration of the relative risks obtained by simulation of an intervention scenario of increasing by 0.5 the standard deviation of the concentration at retail of *Campylobacter*. Results were calculated using three different modelling approaches: Stochastic CPM (eg. Nauta STO), "a-factor" surrogate (eg. Nauta "a"), and Deterministic CPM (eg. Nauta DET).



4.4.2 *Salmonella* CPMs

In Figures 17, 18, 19, 20 we observe the relative risk estimated by all *Salmonella* stochastic CPM´s, surrogates "a-factor", and deterministic CPMs, after simulating the implementation intervention scenarios of 0.5 and 1 log reduction in the mean of the initial concentration, and 0,5 reduction and increase in the standard deviation of the initial concentration.

4.4.2.1 "a-factor" versus stochastic CPMs

Considering Bollaerts, EFSA and Murmann CPMs:

For a scenario of 0.5 log reduction in the mean of the initial concentration, our results show that "a factor" surrogates estimate lower relative risks (or higher risk reduction) compared to stochastic CPMs (Fig. 17). For an intervention scenario of 1 mean log reduction (Fig. 18) a similar scenario as the 0.5 log reduction is found: using a surrogate "a-factor" will result in lower RR estimates (higher risk reduction) when compared to using stochastic CPMs.

In addition, intervention scenarios that change the variation were simulated: 0.5 reduction and 0.5 increase in the standard deviation of the initial concentration.

For a 0.5 decrease in the SD, "a factor" surrogates estimate lower relative risks (higher risk reduction) when compared to a stochastic CPMs (Fig. 19). However, when increasing the standard deviation by 0.5, "a factor" surrogates estimates higher relative risks (Fig. 20).

4.4.2.2 Deterministic CPMs versus stochastic CPMs

In a 0.5 log reduction in the mean of the initial concentration scenario, Bollaerts deterministic CPMs estimates lower relative risks (close to the "a-factor" results) than Bollaerts stochastic CPMs. EFSA deterministic CPM, however, in this intervention scenario estimates very similar relative risks to EFSA stochastic CPM (Fig.17). When a 1 log reduction in the mean of the initial concentration is performed: Bollaerts deterministic CPM gives lower relative risk estimates than the corresponding stochastic CPM, whereas EFSA's deterministic CPM results are very similar to the original EFSA's stochastic CPM (Fig.18).

When changes in the variation are performed, for a 0.5 reduction in the standard deviation, Bollaerts deterministic CPM estimates lower relative risks than Bollaerts stochastic CPM. EFSA's deterministic CPM, on the other hand, estimates very similar relative risk estimates to EFSA's stochastic CPM (Fig. 19). When increasing the standard deviation of the initial concentration by 0.5, Bollaerts deterministic CPM will estimate higher relative risks than Bollaerts stochastic CPM, while EFSA's deterministic CPM will estimate very similar relative risks to EFSA's stochastic CPM (Fig. 20).

Figure 17: Illustration of the relative risks obtained by simulation of an intervention scenario of 0.5 log reduction in the mean of the concentration at retail of *Salmonella*. Results were calculated using three different modelling approaches: stochastic CPM (eg. EFSA STO), "a-factor" surrogate (eg. EFSA "a"), and deterministic CPM (eg. EFSA DET).

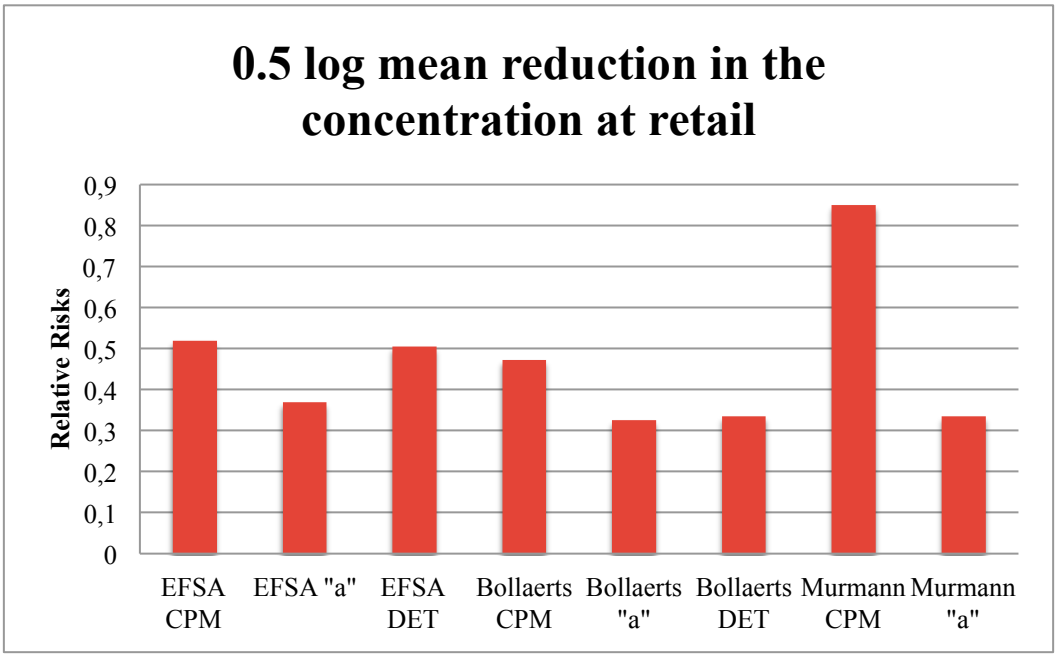


Figure 18: Illustration of the relative risks obtained by simulation of an intervention scenario of 1 log reduction in the mean of the concentration at retail of *Salmonella*. Results were calculated using three different modelling approaches: stochastic CPM (eg. EFSA STO), "a-factor" surrogate (eg. EFSA "a"), and deterministic CPM (eg. EFSA DET).

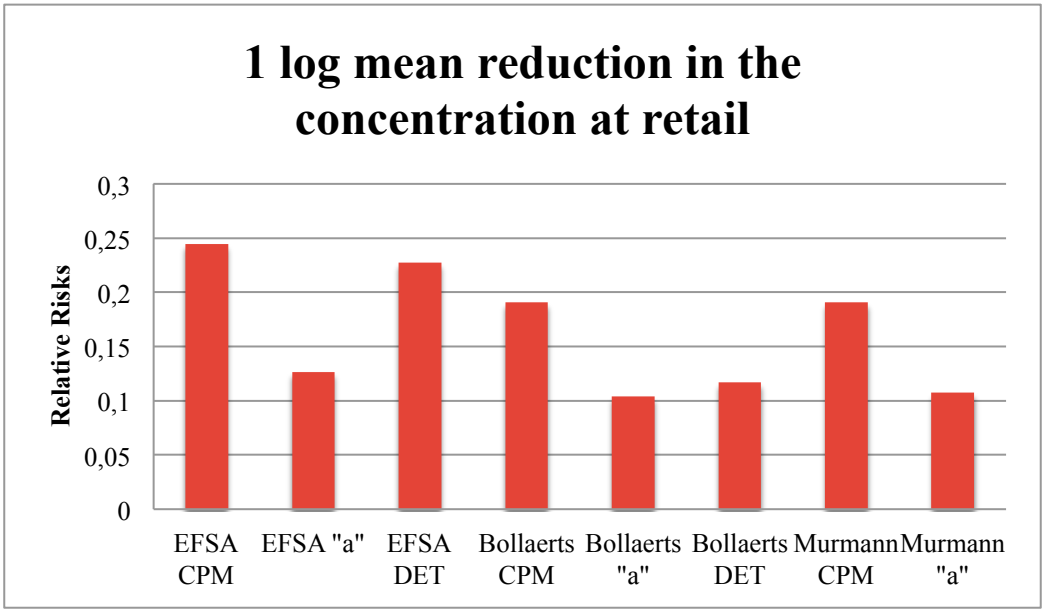


Figure 19: Illustration of the relative risks obtained by simulation of an intervention scenario of 0.5 reduction in the standard deviation (SD) of the concentration at retail of *Salmonella.* Results were calculated using three different modelling approaches: stochastic CPM (eg. EFSA STO), "a-factor" surrogate (eg. EFSA "a"), and deterministic CPM (eg. EFSA DET).

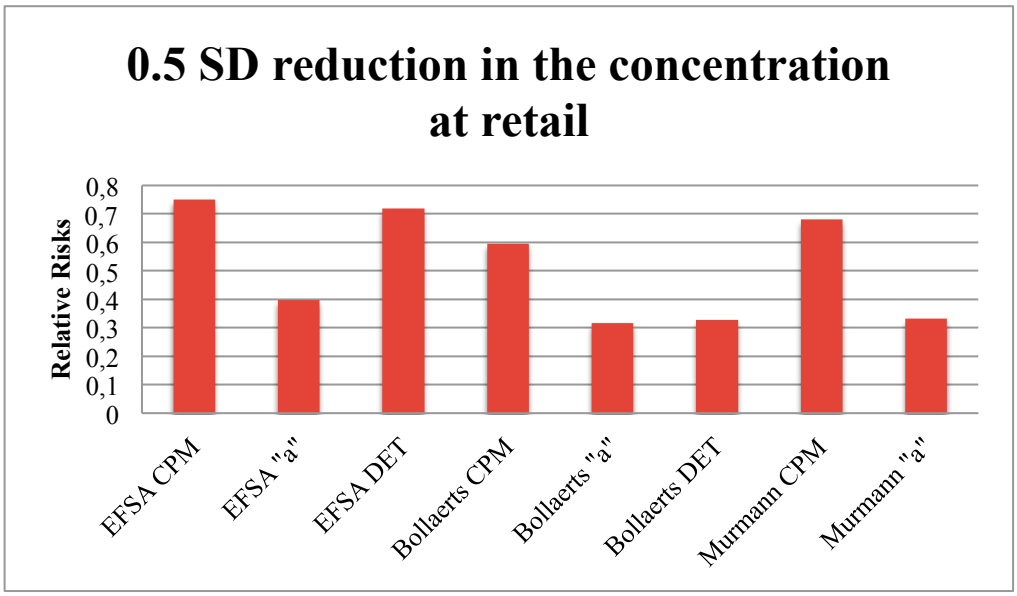
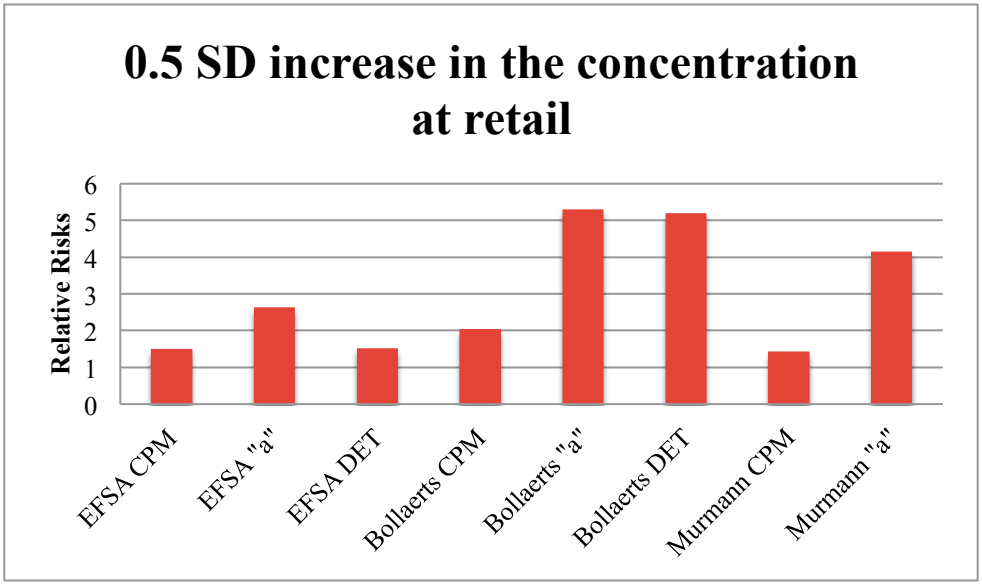


Figure 20: Illustration of the relative risks obtained by simulation of an intervention scenario of an increase by 0.5 of the standard deviation (SD) of the concentration at retail of *Salmonella.* Results were calculated using three different modelling approaches: stochastic CPM (eg. EFSA STO), "a-factor" surrogate (eg. EFSA "a"), and deterministic CPM (eg. EFSA DET).



4.4.3 *Listeria monocytogenes* CPMs

Figs 21, 22, 23 and 24 show the relative risks estimated by *L. monocytogenes* stochastic CPM´s and surrogates "a-factor", after simulating the implementation of intervention scenarios of 0.5 and 1 log reduction in the mean of the initial concentration, as well as a 0.5 reduction and increase in the standard deviation.

4.4.3.1 "a-factor" and deterministic CPMs versus stochastic CPMs

In Fig. 21, we verify that for the healthy population, as well as susceptible population, "a-factors" and deterministic CPMs estimate lower relative risks (or higher risk reduction) compared to using stochastic CPMs in a scenario of a 0.5 mean log reduction in the initial concentration. As for a 1 mean log reduction intervention scenario (Fig. 22), similar results as 0.5 log reduction are found: using surrogates "a factor" and deterministic CPMs (considering both healthy and susceptible population) will estimate lower relative risks (higher risk reduction) compared with using stochastic CPMs.

For changes in the variation, intervention scenarios such as 0.5 reduction and 0.5 increase in the standard deviation of the initial concentration were performed. It is found that for a 0.5 reduction in the SD, "a factor" surrogates and deterministic CPMs (for healthy and susceptible population) will give lower relative risk estimates (higher risk reduction) when compared to a stochastic CPMs (Fig. 22), while when increasing the standard deviation by 0.5, both "a factor" surrogates and deterministic CPMs will estimate higher relative risk estimates (Fig. 23).

Figure 21: Illustration of the relative risks obtained by simulation of an intervention scenario of a 0.5 log reduction in the mean of the concentration at retail of *L. monocytogenes*. Results were calculated using three different modelling approaches: stochastic CPM (eg. Berjia STO HP), "a-factor" surrogate (eg. Berjia "a" HP), and deterministic CPM (Berjia DET HP). HP- healthy population; SP- susceptible population.

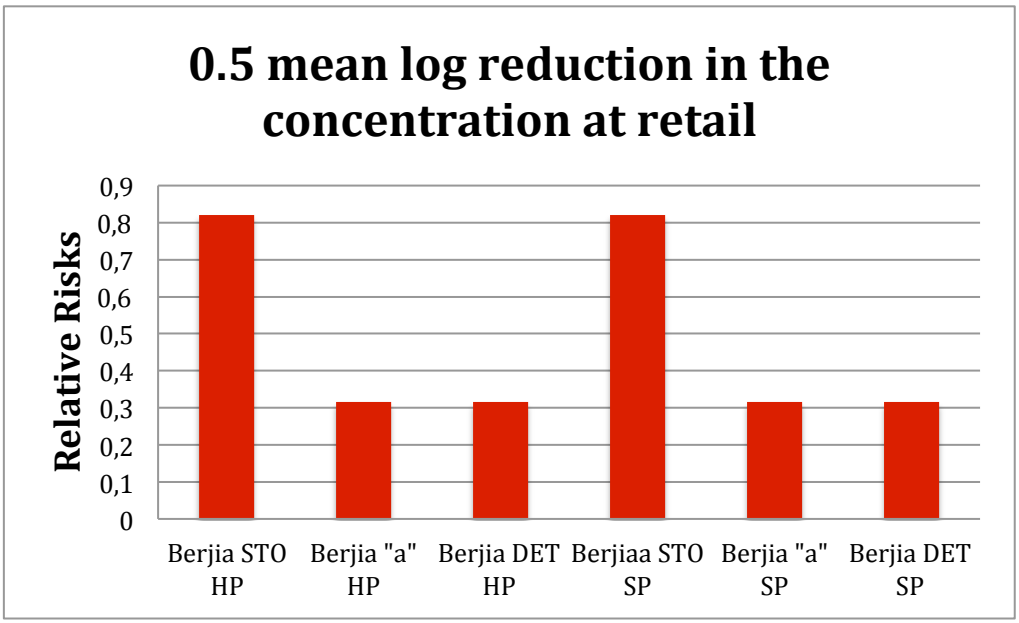


Figure 22: Illustration of the relative risks obtained by simulation of an intervention scenario of a 1 log reduction in the mean of the concentration at retail of *L. monocytogenes*. Results were calculated using three different modelling approaches: stochastic CPM (eg. Berjia STO HP), "a-factor" surrogate (eg. Berjia "a" HP), and deterministic CPM (Berjia DET HP). HP- healthy population; SP- susceptible population.

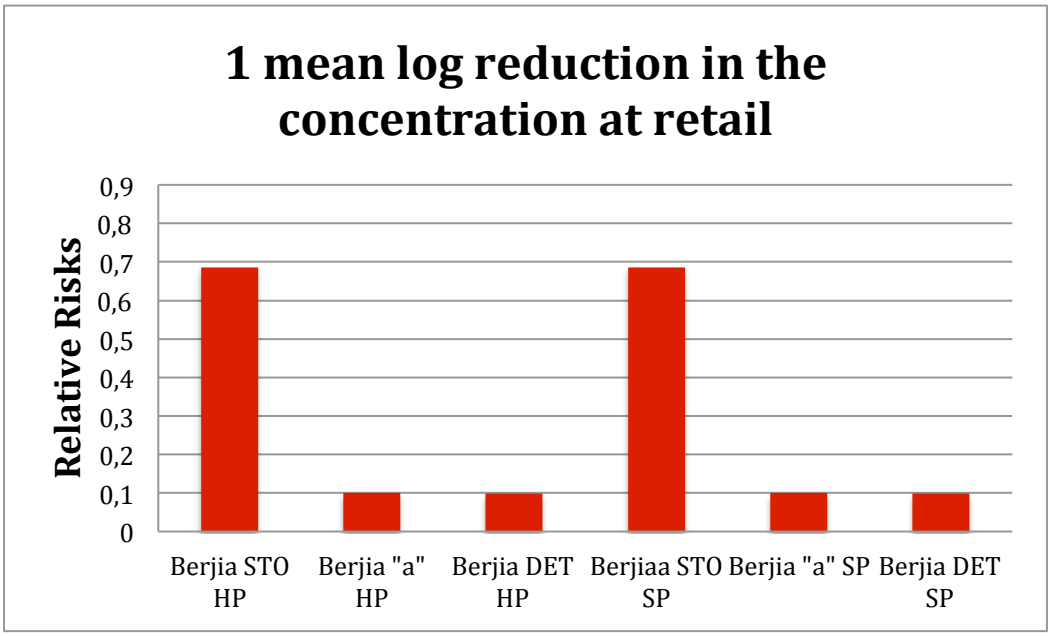


Figure 23: Illustration of the relative risks obtained by simulation of an intervention scenario of a 0.5 reduction in the standard deviation (SD) of the concentration at retail of *L. monocytogenes.* Results were calculated using three different modelling approaches: stochastic CPM (eg. Berjia STO HP), "a-factor" surrogate (eg. Berjia "a" HP), and deterministic CPM (Berjia DET HP). HP- healthy population; SP- susceptible population.

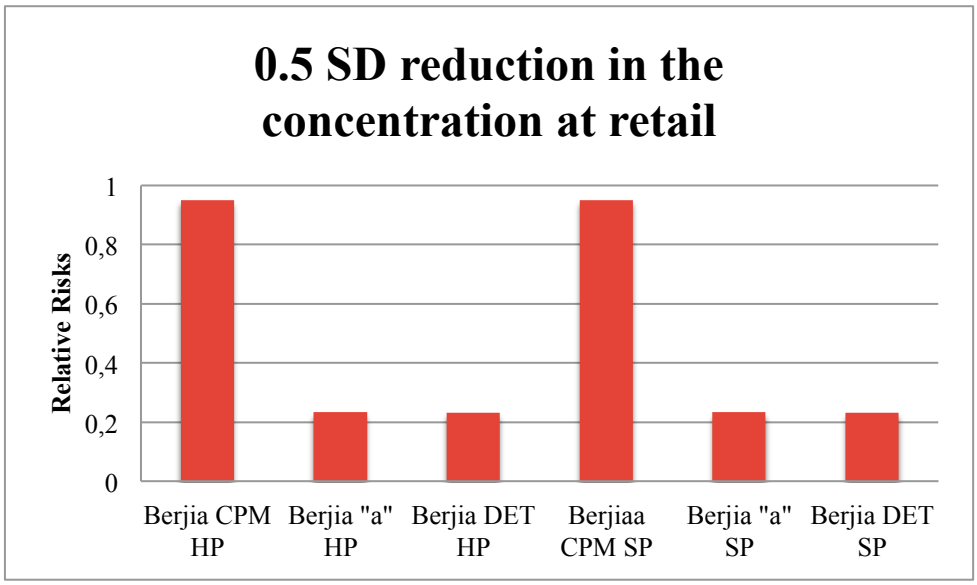
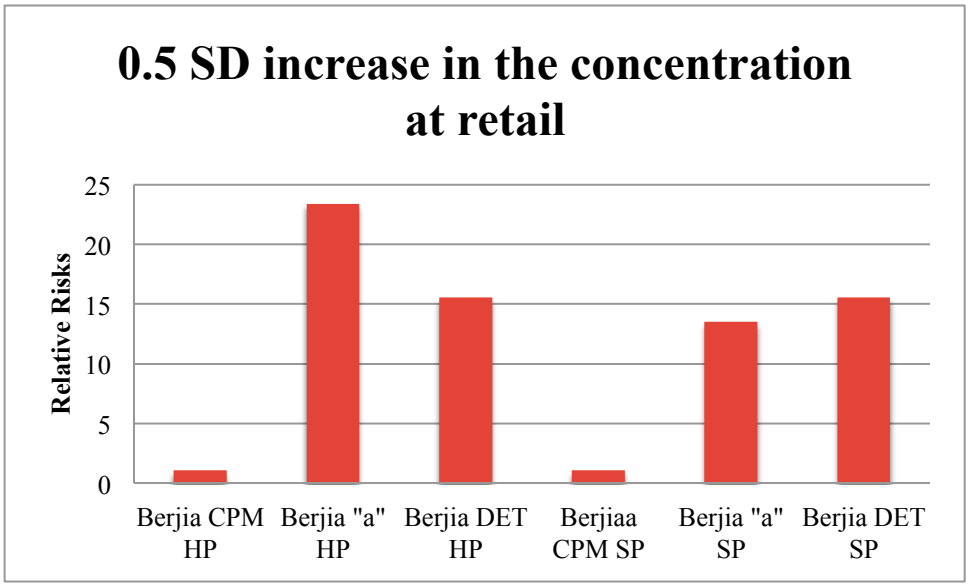


Figure 24: Illustration of the relative risks obtained by simulation of an intervention scenario of an increase of the standard deviation (SD) by 0.5 in the concentration at retail of *L. monocytogenes.* Results were calculated using three different modelling approaches: stochastic CPM (eg. Berjia STO HP), "a-factor" surrogate (eg. Berjia "a" HP), and deterministic CPM (Berjia DET HP).
HP- healthy population; SP- susceptible population.



4.4.4 Changes in the prevalence

As illustrated in Figs. 25 to 30 in Annex IV, it is observed that in intervention scenarios where changes in the prevalence occur (10% decrease or 50% increase), a stochastic CPM, a surrogate "a factor" or a deterministic CPM, will estimate very similar relative risks. These results show that intervention scenarios that change the prevalence of *Campylobacter*, *Salmonella* and *Listeria* don't interfere with the relative risk estimates given by a stochastic CPM, a surrogate or a deterministic given that the prevalence is not calculated inside the consumer phase, but only further in the process of risk assessment. If the prevalence of pathogens in contaminated food is changed, the change in the probability of illness is proportional to the changes in the prevalence for all CPMs, assuming that there was no interaction between foods with different prevalences in the consumer phase. Using a CPM to evaluate the effect of control measure that affect exclusively the prevalence becomes irrelevant (Nauta & Christensen, 2011).

4.5 Overview of the results

Tables 7 and 8 provide a general overview of the results obtained by comparing the performance of the "a-factor" and deterministic CPM with the stochastic CPM in term of of (almost) equal (=), increased (+) or decreased absolute risk.

Table 7: Overview of the results: comparison of the performance of the results obtained by using an "a-factor" and deterministic CPM with a stochastic CPM in terms of (almost) equal (=), increased (+) or decreased absolute risk.

	"a-factor"	**Deterministic CPM**
Nauta CPM	**(=)**	**(+)**
Christensen CPM	**(=)**	**(+)**
Calistri CPM	**(=)**	**(+)**
Bollaerts CPM	**(=)**	**(=)**
EFSA CPM	**(=)**	**(=)**
Murmann CPM	**(=)**	**---**
Berjia CPM HP	**(=)**	**(-)**
Berjia CPM SP	**(=)**	**(-)**

Table 8: Overview of the results: comparison of the performance of the results obtained by using an "a-factor" and deterministic CPM with a stochastic CPM in terms of (almost) equal (=), increased (+) or decreased relative risk of four intervention scenarios.

Intervention Measures	CPMs	"a-factor"	Deterministic CPM
0.5 mean log reduction in the concentration at retail	Nauta CPM	(-)	(=)
	Christensen CPM	(-)	(=)
	Calistri CPM	(-)	(=)
	Bollaerts CPM	(-)	(-)
	EFSA CPM	(-)	(=)
	Murmann CPM	(-)	---
	Berjia CPM HP	(-)	(-)
	Berjia CPM SP	(-)	(-)
1 mean log reduction in the concentration at retail	Nauta CPM	(-)	(=)
	Christensen CPM	(-)	(=)
	Calistri CPM	(-)	(=)
	Bollaerts CPM	(-)	(-)
	EFSA CPM	(-)	(=)
	Murmann CPM	(-)	---
	Berjia CPM HP	(-)	(-)
	Berjia CPM SP	(-)	(-)
0.5 reduction of the standard deviation in the concentration at retail	Nauta CPM	(-)	(=)
	Christensen CPM	(-)	(=)
	Calistri CPM	(-)	(=)
	Bollaerts CPM	(-)	(-)
	EFSA CPM	(-)	(=)
	Murmann CPM	(-)	---
	Berjia CPM HP	(-)	(-)
	Berjia CPM SP	(-)	(-)
0.5 increase of the standard deviation in the concentration at retail	Nauta CPM	(+)	(=)
	Christensen CPM	(+)	(=)
	Calistri CPM	(+)	(=)
	Bollaerts CPM	(+)	(-)
	EFSA CPM	(+)	(=)
	Murmann CPM	(+)	---
	Berjia CPM HP	(-)	(+)
	Berjia CPM SP	(-)	(+)

III. Discussion

1. Purpose of the study

In quantitative microbiological risk assessment (QMRA), the consumer phase model (CPM) describes the part of the food chain from retail purchase of the food product to the moment of consumption. The large variation in consumer food handling practices and scarce availability of data imply that several subjective and simplifying assumptions are made when a CPM is constructed.

In the development of a CPM, it is relevant to understand to what extent these models need to include a detailed description of the processes that may result in exposure. It has been stated that "There is no alternative but for a probabilistic approach to risk assessment models of the consumer phase." (Nauta et al., 2009). The purpose of this project was to obtain stochastic CPMs from the literature to act as a baseline model with which two simpler modelling approaches will be compared to. The simpler approaches chosen are a surrogate "a-factor" found in a study from (Duarte et al., 2016) and deterministic CPMs. These don't include variation and would be ideal to use in acute public health food borne disease outbreaks, when fast responses and actions are needed. Stochastic CPMs, on the other hand, frequently involve complex and lengthy building processes (WHO, 2012; Zwietering, 2009).

It was decided to choose stochastic CPMs from different pathogens (*Campylobacter, Salmonella* and *Listeria*) and foods, with different levels of complexity, in favour of obtaining an independent and broad range of results. *Campylobacter* models are for broiler chicken, *Salmonella* models are for pork meat and *L. monocytogenes* model is for a ready-to-eat food (cold smoked salmon).

The performance of the CPMs was evaluated in terms of absolute risk estimates and relative risk estimates of various intervention scenarios.

2. "a-factor" versus stochastic CPMs

A surrogate for a CPM used in a study about the effect of carcass decontamination on the risk for consumers was found in the literature, a constant value expressed by the factor *a*. This constant assumes that the survival of *Salmonella* in pork meat, from the carcass to the consumed serving, is identical between servings.

By using this surrogate, it is not necessary to make additional assumptions on the survival of the pathogen. However, it is stated in the study that this assumption is unlikely to be correct (Duarte et al., 2016). This is based on the fact that studies for *Campylobacter* on broiler meat have shown that there is variation present in transfer and survival during the consumer phase, and therefore it is essential to consider it in a risk assessment (Duarte et al., 2016; Nauta &

Christensen, 2011; Nauta et al., 2009). Besides these statements, the performance of the "a-factor" had never been evaluated and compared to the performance of stochastic consumer phase model.

In this project, the "a-factor" was implemented in the software @Risk and absolute risk estimates and relative risks were calculated. It is important to stress that the relative risks were calculated considering the same intervention scenarios as the stochastic CPMs, in order to accurately compare the results. The same dose-response model was also applied. Besides the fact that the "a-factor" had only been used for *Salmonella* in the literature, it was easy to also use it for consumer phase models of the other pathogens considered in this study.

2.1 A comparison of the absolute risk estimates

In section 4 (Models performance analysis), one is able to compare the absolute risk estimates of each "a-factor" with the correspondent stochastic consumer phase model in all the models for the three pathogens.

We observe that in all the seven models considered, the "a-factor" estimates absolute risks very similar to the stochastic CPM in the literature. Since the inputs used for solving the integral to obtain the "a-factor" were the absolute risk estimates given by each stochastic CPM, the concentration at retail of the pathogen, the specific values of the parameters for the dose-response model and the distribution of the doses (number of CFUs of the pathogen) ingested, it was expected that the absolute risk estimates obtained by using the "a-factor" would be the same as the results obtained by using stochastic CPMs. In this case, they are not identical due to the Monte Carlo Simulation performed in Monte Carlo software @Risk 5.5 (Palisade).

Besides obtaining very similar results, it is known that the uncertainty in absolute risk estimates is in both cases large: there is intrinsic uncertainty in the CPM and in the "a-factor", as well as uncertainty in the distribution of each pathogen concentration at retail, and dose-response model, which is still varying.

2.2 A comparison of the relative risk estimates

To estimate the effect of intervention scenarios in the food production chain, the relative risks of six different intervention scenarios were calculated. Relative risks are often used to assess the effects of interventions or control measures in the food production chain. These become more relevant when evaluating the performance of CPMs and "a-factor", because they hold a smaller level of uncertainty than absolute risk estimates because the uncertainties are partly cancelled out when there is division of the absolute risk estimates. (Duarte et al., 2016).

The results of the relative risks show that in intervention scenarios of 0.5 and 1 log reduction on the mean of the initial concentration, the "a-factor" estimates lower relative risks than all seven stochastic CPMs considered. This means that the risk of an intervention scenario is underestimated, and consequently the effect of the control measure in terms of risk reduction is overestimated: the control measure seems to work better than it actually does. This goes in agreement with what has been stated before in studies for *Campylobacter* in broiler meat: there is variation in transfer and survival in the consumer phase, and it needs to be taken into account. (Duarte et al., 2016; Nauta & Christensen, 2011; Nauta et al., 2009).

For an intervention scenario that aimed to simulate a decrease in the variation, a reduction of the standard deviation of the initial concentration by 0.5 was performed. The "a-factor" estimates lower relative risks than the stochastic correspondent CPM in all the models for the three pathogens. In the seven models considered, if a surrogate "a-factor" is used in place of a stochastic CPM to calculate the risk of an intervention scenario of a 0.5 reduction in the standard deviation, the risk will be underestimated. On the other hand, if an "a-factor" is used to assess the effect of an intervention scenario which simulates an increase in variation (by raising the standard deviation of the initial concentration by 0.5), it will estimate higher relative risks than the original stochastic CPM (i.e. overestimates the risk of an intervention scenario). It was also found that when increasing the variation, the difference between the results estimated by the "a-factor" and the corresponding consumer phase model is higher than when the variation is reduced, which leads us to believe that in this scenario one should consider to use a stochastic CPM.

3. Deterministic CPMs versus stochastic CPMs

Since the relative risk estimates obtained with an "a-factor" were very consistent, and were found to be significantly different from all the relative risks given by a stochastic CPMs in all intervention scenarios considered, it was decided to take a second approach and observe how it performed. This approach consisted in modifying the original version of stochastic CPMs, in order to make them deterministic CPMs. This was achieved by changing all the parameters that were described by probabilistic distributions in stochastic CPMs to fixed values, obtained by calculating the mean of each distribution inside a parameter. Annex II contains tables including the seven CPMs considered in this project, showing side by side the stochastic and deterministic version of the models, and the differences in the variables using distributions (in the stochastic CPM) or fixed values (in the deterministic CPM). This approach could still simplify the process of building a stochastic CPM.

The performance of each deterministic CPM was evaluated in the same terms as the "a-factor", through calculation of the absolute risk estimates and the relative risks of each intervention scenario. It is important to stress that, as with the "a-factor", the input concentration of the model is still variable, and the dose-response models used are the same as the stochastic CPMs.

3.1 A comparison of the absolute risk estimates

Table 7 provides an overview of the results of the absolute risk estimates and compares performance of the results obtained by using an "a-factor" and deterministic CPM with a stochastic CPM in terms of (almost) equal (=), increased (+) or decreased absolute risk.

The absolute risk estimates obtained by using a deterministic CPM were different from the stochastic CPMs for *Campylobacter*, *Salmonella* and *L. monocytogenes*. *Campylobacter* deterministic CPMs all estimated higher absolute risks than each corresponding deterministic version. For *Salmonella* CPMs (EFSA and Bollaerts), the deterministic version estimated very similar absolute risk estimates with the stochastic CPM. For *L. monocytogenes* models (Bejia STO and Berjia DET for healthy and susceptible population), the deterministic version gave lower absolute risk estimates than the stochastic. As these were quite mixed results, no conclusions were taken on the performance of deterministic CPMs to estimate absolute risks. Occasionally, they estimate similar results, but one doesn't know in which situations that occurs and what are the parameters that influence these results.

3.2 A comparison of the relative risk estimates

Relative risk estimates were also calculated in order to assess the effect of intervention scenarios in the food production chain. Table 8 provides an overview of the results of the relative risk estimates and compares the performance of the results obtained by using an "a-factor" and deterministic CPM with a stochastic CPM in terms of (almost) equal (=), increased (+) or decreased relative risk of four intervention scenarios.

Campylobacter deterministic CPMs estimate very similar relative risks to the stochastic CPMs in all the considered intervention scenarios (0.5 and 1 log reduction of the mean of the initial concentration, 0.5 increase and decrease of the standard deviation, 10% decrease and 50% increase of the prevalence).

Salmonella deterministic CPMs however, didn't perform similarly to the *Campylobacter* CPMs. EFSA deterministic CPM estimates very similar relative risks to stochastic CPM in all intervention scenarios. Bollaerts deterministic CPM, on the other hand, estimates lower relative risks than Bollaerts stochastic CPM in intervention scenarios of 0.5 and 1 log reduction in the mean of the initial concentration and reduction of the standard deviation of

the initial concentration by 0.5. When there is an increase of the standard deviation by 0.5, Bollaerts deterministic CPM estimates higher relative risks than the stochastic CPM. This might be due to the fact that Bollaerts CPM was the most complex model used in this project, contained more variables and variability was included in most steps by including distributions. As in all these steps distributions were substituted by a fixed value (their mean), there might have been an excessive simplification to the point were the deterministic CPM gives similar results as when using a single constant value like the "a-factor". Murmann stochastic CPM was not possible to convert in to a deterministic CPM. The step "cooking time (min)" (see. Annex II) was defined by a Pert distribution with values 15, 20, 30 being the minimum, most likely and maximum, respectively. If one used the mean of this distribution as a fixed value to describe the cooking time, the final absolute risk estimates would be zero. For this reason, this deterministic CPM was not evaluated in this part of the project because to obtain a positive value for absolute risk estimates one would have to use a higher value than the mean to obtain a positive absolute risk, which wouldn't be consistent with the approach taken to all the other CPMs.

Regarding *L. monocytogenes*, Berjia deterministic CPMs (for healthy and susceptible population) estimate relative risks with the same pattern observed in Bollaerts models. In intervention scenarios of 0.5 and 1 log reduction in the mean of the initial concentration and reduction of the standard deviation of the initial concentration by 0.5, deterministic CPMs estimate lower relative risks than the stochastic CPMs. When there is an increase in the standard deviation deterministic CPMs estimate higher relative risks than the stochastic CPM. As the deterministic CPM estimates similar results as the "a factor", the same explanation used in Bollaerts models can apply to this situation: Berjia deterministic CPM was simplified to the point where considering a model with various steps that are fixed values and using a single constant value makes almost no difference at all.

For intervention scenarios considering changes in the prevalence, all the modelling approaches estimated similar relative risks. These results go accordingly with what has already been said in the literature: If there is a change in the prevalence of pathogens in contaminated food, the change in the probability of illness is proportional to the change in prevalence for all CPMs, if we assume that there was no interaction between foods with different prevalences in the consumer phase. It becomes thus irrelevant to use a CPM when assessing the effect of control measures in the food production chain that affect exclusively the prevalence (Nauta & Christensen, 2011).

In a general overview, four in a total of seven deterministic CPMs showed similar relative risks in all the intervention scenarios considered to the corresponding stochastic CPM. In

these four situations, deterministic CPMs could be used to assess the effect of intervention scenarios in the food production chain.

4. Modelling limitations

One should always keep in mind that models are always a simplification of reality, and they usually contain a large amount of assumptions, simplifications and abstractions (Nauta, 2009). The assumptions considered and made when building a CPM, whether it is stochastic or deterministic, will have great impact on the outcome of the risk assessment. In situations when one needs to decide which CPM to use to achieve specific objectives, it is of great interest to understand what is the effect of the assumptions made when building a deterministic or stochastic CPM (Nauta, 2009).

IV. Conclusion

This project was developed with the purpose of assessing how simpler modelling techniques that don't include variation compare to stochastic modelling techniques, to account for the consumer phase of a QMRA. These different techniques were compared in terms of absolute risk estimates and relative risk estimates.

Seven consumer phase models were found in the literature for the pathogens *Campylobacter, Salmonella* and *Listeria monocytogenes*, for the food products broiler chicken, pork meat and cold smoked salmon, respectively. These models are in general more complicated to build, as they include variation and are therefore stochastic.

The simpler modelling approaches consist of a constant value designated by "a-factor", presented in a study about the effect of carcass decontamination on the risk for consumers (Duarte, Nauta, & Aabo, 2016), and six deterministic consumer phase models.

Results showed that the constant "a-factor" approach provides similar absolute risk estimates as the stochastic CPMs, but different relative risks in all the intervention scenarios performed. These results demonstrate that the results of (Duarte, Nauta, & Aabo, 2016) may have been influenced by using an "a-factor" instead of a stochastic CPM to account for the consumer phase.

It was also found that all deterministic CPMs provide different absolute risk estimates, but when relative risks were calculated, some estimate similar results to stochastic CPMs while others do not. It is not clear which situations and assumptions interfere with the results obtained when a deterministic CPM estimates similar or different relative risks from a stochastic CPM.

By undertaking this project, we were able to assess the performance of the "a-factor" in terms of absolute and relative risks, as the results obtained were very consistent. On the other hand, the results of the performance of deterministic CPMs were not as consistent. Due to the fact that this project was only developed during three and a half months, there was not enough time to understand what criteria should be considered when choosing a fixed value instead of a distribution in a deterministic CPM in order to obtain similar relative risks than stochastic CPMs.

At the end of this study, some questions arose: Why do deterministic CPMs estimate similar relative risks to stochastic CPMs in some situations and others don't? What are the best criteria to use when choosing a fixed value to replace a distribution in a deterministic CPM in order to obtain accurate relative risks? What are the factors that influence the results when deterministic CPMs give similar relative risks to "a factor" constant or to stochastic CPMs?

Answering these questions would require more in depth studies about the role and performance of deterministic CPMs in QMRA.

Regarding CPMs in general, efforts should be put in more attention and studies, besides the lack of interest given to these by risk managers. There are still a lot of limitations regarding the consumer phase that deserve more concern: the lack of available data for research studies, the high variability in consumer practices between different cultures and individually, and the lack of control of this phase by professionals. As well as increasing research studies in these subjects, education of the population about food borne illnesses, safe transportation, storage, preparation and cooking practices is also vital for reducing food borne illnesses (Fischer et al., 2005; Redmond & Griffith, 2003).

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V. Annexes

Annex I

List of the distributions used and their meanings (Adapted from: Vose (2008))

1. Bernoulli- The Bernoulli distribution is a Binomial distribution with n = 1. It returns a 1 with probability p and a zero otherwise. It is very useful to model a risk event that may or may not occur.
2. Binomial- The Binomial distribution models the number of successes from n independent trials where there is a probability p of success in each trial. This distribution assumes that the probability p does not change the more trials are performed.
3. Beta- The Beta distribution is often used to describe the uncertainty about a probability in a binomial process, given a number of trials n have been made with a number of recorded successes s. In this situations, α is set to the value (s + x) and β is set to (n - s + y), where Beta(x, y) is the prior.
4. Cumulative- The Cumulative distribution includes a minimum, maximum, $\{x_i\}$,$\{P_i\}$ values, where $\{x_i\}$ is an array of *x*-values with cumulative probabilities $\{P_i\}$ and where the distribution falls between the minimum and maximum.
5. Discrete- The Discrete distribution is a general type of function used to describe a variable that can take one of several explicit discrete values $\{x_i\}$ and where a probability weight $\{p_i\}$ is assigned to each value.
6. Lognormal- The Lognormal distribution is useful for modelling naturally occurring variables that are the product of a number of other naturally occurring variables. Central Limit Theorem shows that the product of a large number of independent random variables is Lognormally distributed.
7. Normal- The normal distribution is a probability distribution that associates the normal random variable *X* with a cumulative probability . The graph of the normal distribution depends on the mean and the standard deviation. It is used to model a naturally occurring variable (for example, the height of adult European males), for the distribution of errors in statistical theory and for approximation of uncertainty distribution.

8. PERT- The PERT (or BetaPERT) distribution is a version of the Beta distribution and requires the same three parameters as the Triangular distribution, namely minimum (a), most likely (b) and maximum (c). It is used exclusively for modelling expert estimates, where one is given the expert's minimum, most likely and maximum guesses. It is a direct alternative to a Triangular distribution.
9. Poisson- The Poisson (λ) distribution models the number of occurrences of an event in a given time with an expected rate of λ events when the time between successive events follows a Poisson process.
10. Uniform- A Uniform distribution assigns equal probability to all values between its minimum and maximum.

Annex II

Table 9: Description of the values and distributions used in Nauta stochastic CPM and Nauta deterministic CPM

Step	Relevant parameter(s) description	Notation	Value/distribution - Stochastic	Value/distribution- Deterministic
Contamination at retail	Concentration of *Campylobacter* at retail (cfu/g)	C_{ret}	IF(*p*prev=1; 10^~Normal(1.5, 1.2)),0))	IF(*p*prev=1; 10^~Normal(1.5,1.2)) ,0)
	Prevalence at retail	p_{prev}	~Bernoulli(pprev)) with p_{prev}= 0.25	0.25
	Portion sizes	w_C	~LogNormal(189, 127)	~LogNormal(189, 127)
	Number of *Campylobacter* on one portion of consumed chicken meat (cfus)	$N_{portion}$	~ Poisson($C_{ret} \times w_C$)	~ Poisson($C_{ret} \times w_C$)
Cross-contamination	Variability of transfer rates from raw meat to salad	p_{tr}	See Appendix in Nauta & Christensen (2011)	0.000514

Table 10: Description of the values and distributions used in Christensen stochastic CPM and Christensen deterministic CPM

Step	**Relevant parameter(s) description**	**Notation**	**Value/distribution- Stochastic**	**Value/distribution- Deterministic**
Contamination at retail	Concentration of *Campylobacter* at retail (cfu/g)	C_{ret}	IF(*p*prev=1; 10^~Normal(1.5,1.2)),0)	IF(*p*prev=1; 10^~Normal(1.5, 1.2)),0)
	Prevalence at retail	p_{prev}	~Bernoulli (p_{prev}) with p_{prev}= 0.25	0.25
	Portion sizes	w_C	~LogNormal(189, 127)	~LogNormal(189, 127)
	Number of *Campylobacter* on one portion of consumed chicken meat (cfus)	$N_{portion}$	~ Poisson($C_{ret} \times w_C$)	~ Poisson($C_{ret} \times w_C$)
Cross-contamination	Transfer rate chicken to equipment	*t*CE	$\sim 10^{-Pert(1,2,6)}$	0.0117
	Transfer rate equipment to chicken	*t*EC	$\sim 10^{-Pert(1,2,6)}$	0.0117
	Frequency of chicken to chicken contamination	*f*CC	= 1	= 1
	Transfer rate equipment to salad	*t*ES	$\sim 10^{-Pert(1,2,6)}$	0.0017
	Frequency of chicken to salad contamination	*f*CS	= 1	= 1

Table 11: Description of the values and distributions used in Calistri stochastic CPM and Calistri deterministic CPM

Step	Relevant parameter(s) description	Notation	Value/distribution- Stochastic	Value/distribution- Deterministic
Contamination at retail	Concentration of *Campylobacter* at retail (cfu/g)	C_{ret}	IF(*p*prev=1; 10^~Normal(1.5,1.2)),0)	IF(*p*prev=1; 10^~Normal(1.5, 1.2)),0)
	Prevalence at retail	p_{prev}	~Bernoulli (pprev) with p_{prev}= 0.25	0.25
	Portion sizes	w_C	~LogNormal(189, 127)	~LogNormal(189, 127)
	Number of *Campylobacter* on one portion of consumed chicken meat (cfus)	$N_{portion}$	~ Poisson($C_{ret} \times w_C$)	~ Poisson($C_{ret} \times w_C$)
Cross-contamination	Transfer from meat to kitchenware	*t*CE	See Table 4 in Calistri & Giovannini (2008)	0,0096
	Transfer from kitchenware to meat	*t*ER	See Table 4 in Calistri & Giovannini (2008)	0.0871
	Transfer from meat to hands	*tCH*	See Table 4 in Calistri & Giovannini (2008)	0.0167
	Transfer from hands to meat or ready-to-eat food	*t*HR	See Table 4 in Calistri & Giovannini (2008)	0.0192
	Chicken to environment contamination	fCE	~Bernoulli p_{CE} with probability p_{CE} = 0.124	0.124
	Chicken to hand contamination	fCH	~Bernoulli (p_{CH}) with probability p_{CH} = 0.259	0.259

Step	Relevant parameter(s) description	Notation	Value/distribution- Stochastic	Value/distribution- Deterministic
	Probability of chicken to environment contamination	*p*CE	0.124	0.124
	Probability of chicken to hand contamination	pCH	0.259	0.259

Table 12: Description of the values and distributions used in Bollaerts stochastic CPM and Bollaerts deterministic CPM.

Step	Relevant Parameter(s) description	Notation	Value/distribution-Stochastic	Value/distribution-Deterministic
Contamination at retail	Concentration of *Salmonella* at retail (log CFU/g)	N_{meat}	Normal(1.4,0.7)	Normal(1.4,0.7)
	Prevalence at retail	p	0.12	0.12
	Weight of a portion minced pork meat (g)	$W_{portion}$	Normal(93, 14, 83^2)	Normal(93, 14, 83^2)
	Numbers in portion (CFU)	$N_{portion}$	$10^{\text{Wportion x Nmeat}}$	$10^{\text{Wportion x Nmeat}}$
Numbers on portion after transport	Temperature of portion at retail (°C)	$Temp_{meat}$	Normal(3.14, 7.78)|(−2, 15)	5.3753
	External temperature (°C)	$Temp_{ex}$	~f = πf1+(1−π)f2 with π =0.64, f1 ~Normal(6.7,17.9) and f2 ~Normal(20.1, 33.0)	11.5271
	Maximal possible change in temperature (°C)	Δ_{max}	$=\text{Temp}_{ex}-\text{Temp}_{meat}$	$=\text{Temp}_{ex}-\text{Temp}_{meat}$
	Maximal change larger than 0 (no =0, yes=1)	S	$I(\Delta_{max}>0)$	$I(\Delta_{max}>0)$
	Change in temperature (°C)	Δ	=Normal(3.72,2.82)|(0 , Δ_{maxk}) × S	2.3512
	Temperature of portion at end of transport (°C)	$Temp_{end}$	$\text{Temp}_{meat} + \Delta$	$\text{Temp}_{meat} + \Delta$
	Transport time (in 15 minutes)	$Time_{tr}$	~ Discrete(v;w) with v = [1, 2, 3, 4, 5, 6, 7, 8, 16] w = [0.005, 0.05, 0.18, 0.25, . . . 0.22, 0.16, 0.07, 0.03, 0.035]	4.995
	Salt concentration of minced	$NaCl$	~Uniform(1.12, 1.75)	1.4350

	meat (%)			
	Temperature ($^{\circ}$C) of portion after transport time l, $l \in [0, Time_{tr}]$	$Temp_l$	$= Temp_{meat} + \frac{Time_l}{Time_{tr}}(Temp_{end} - Temp_{meat})$	$= Temp_{meat} + \frac{Time_l}{Time_{tr}}(Temp_{end} - Temp_{meat})$
	Total log growth during transport integrated out over transport time	Δ_{tr}	$=\int\Delta(\mu_l, Time_l)^{dTime_l}$ with $\mu_l = f(NaCl, Temp_l)$	$=\int\Delta(\mu_l, Time_l)^{dTime_{kl}}$ with $\mu = f(NaCl, Temp_l)$
	Numbers on portion after transport to home (CFU)	N_{trans}	$=10^{(\log_{10} N_{portion} + \Delta_{tr})}$	$=10^{(\log_{10} N_{portion} + \Delta_{tr})}$
Numbers on portion after storage	Temperature (ºC) of portion during storage at home	$Temp_{st}$	~Normal(7, 2.97^2)	6.9982
	Time (hours) of storage at home	$Time_{st}$	~Betapert(0, 2, 5)	2.1667
	Total log growth during storage	Δ_{st}	$= \mu \times Time_{st}$	$= \mu \times Time_{st}$
	Growth rate during storage	μ	$= f(NaCl, Temp_{st})$	$= f(NaCl, Temp_{st})$
	Numbers on portion (CFU) after storage at home	N_{stor}	$=10^{(\log 10 (N_{trans} + \Delta_{st})}$	$=10^{(\log 10 (N_{trans} + \Delta_{st})}$
Cross contamination by hand	Number on other food due to cross-contamination via hands	$N_{X\text{-}hand}$	$=N_{stor} \times T_{m,h} \times P_{hand}$ $T_{h,o} \times S_{other}$	$=N_{stor} \times T_{m,h} \times P_{hand}$ $T_{h,o} \times S_{other}$
	Proportion transferred from meat to hand	$T_{m,h}$	~Beta(1.78, 41.10)	0.415
	Proportion persisting on hands after (not) washing	P_{hand}	~Discrete(1, K; π_h, 1-πh) with K~Beta(0.24, 6.67)	0.1699
	Proportion transferred from hand to other food	$T_{h,o}$	~Beta(0.6, 2.3)	0.2069
	Handling meat before other food (no=0, yes=1)	S_{other}	~Bernoulli(π) with $\pi \sim$ Uniform [0.5 − 0.1, 0.5 + 0.1]	0.5

	Numbers remaining on portion after cross-contamination via hands	N_{meat1}	$=(1-T_{m,h}) \times N_{stor}$	$=(1-T_{m,h}) \times N_{stor}$
Probability that other board is used	Probability that other board is used	$N_{X\text{-}board}$	$=N_{board2} \times T_{b,o} \times S_{other}$	$=N_{board2} \times T_{b,o} \times S_{other}$
	Probability that other board is used	S_b	~Bernoulli(π) with π ~ Uniform [0.1 − 0.05, 0.1 + 0.05]	~Bernoulli(π) with π ~ Uniform [0.1 − 0.05, 0.1 + 0.05]
	Probability that other board is used	$T_{m,b}$	$\frac{1}{100}10^{\kappa}$ with κ ~N(0.171,0.16^2)	0.01485
	Probability that other board is used	π_{b1}	~Beta(2820, 159)	0.6191
	Probability that same board is used and washed	π_{b2}	~Beta(2913, 66)	0.3809
	Probability that same board is used and not washed	π_{b3}	$=1 - \pi_{b0} - \pi_{b1}$	0.0316
	Numbers remaining on board after board manipulation: (1) other board, (2) same board washed, (3) same board not washed	N_{board2}	~Discrete(0,κ,N_{board1} ;π_{b0}, π_{b1}, π_{b2} with $\kappa=10^{(\log 10 Nboard1-\Delta)}$ with Δ~Beta(1, 4.5, 7)	6.4072
	Proportion transferred from board to other food	$T_{b,o}$	$\frac{1}{100}10^{\kappa}$ with κ ~N(1.46,0.3^2)	0.2946

	Numbers remaining on portion after food handling	N_{meat2}	$= N_{meat1} \times (1 - T_{m,b})$	$= N_{meat1} \times (1 - T_{m,b})$
Cooking	Proportion protected area	$P_{protect}$	~Uniform(0, 0.1)	0.0499
	Numbers in the protected area (CFU)	$N_{protect}$	$=P_{protect} \times N_{meat2}$	$=P_{protect} \times N_{meat2}$
	Probability of undercooking	π_u	~Betapert(0.05, 0.10, 0.2)	0.1008
	Undercooking (no=0, yes=1)	S_u	~Bern(π_u)	0.111
	Exposure temperature (ºC) of protected area in case of undercooking	$Temp_{cook}$	~Betapert(60, 65, 70)	64.9999
	Exposure time (minutes) of protected area in case of undercooking	$Time_{cook}$	~Betapert(0.5, 1, 1.5)	1.0000
	Numbers of portion after cooking	N_{cook}	$10^{(\log_{10} N_{protect} - \Delta_{protect})} \times S_u$ with $\Delta_{protect} = Time_{cook}/D$ with $D = 10^{-0.14Temp_{cook}+8.58}$	$10^{(\log_{10} N_{protect} - \Delta_{protect})} \times S_u$ with $\Delta_{protect} = Time_{cook}/D$ with $D = 10^{-0.14Temp_{cook}+8.58}$
Numbers ingested when consuming meal (CFU)		N_{dose}	$= N_X + N_{cook}$	$= N_X + N_{cook}$

Table 13: Description of the values and distributions used in EFSA stochastic CPM and EFSA deterministic CPM.

Step	Relevant Parameter(s) description	Notation	Value/distribution-Stochastic	Value/distribution-Deterministic
Contamination at retail	Concentration of *Salmonella* at retail (log CFU/g)	N_{meat}	Normal(1.4,0.7)	Normal(1.4,0.7)
	Prevalence at retail	p	0.12	0.12
	Transport time from retail to home (min)	t_{tr}	G([0,30,50,120],[096,.02,.02])	29.9867
	Temperature during transport (ºC)	T_{tr}	G([-2,0,2,4,6,8,10],[0.003,0.023,0.135,0.242,0.253,0.344])	5.5142
	Storage time (h)	t_{st}	G([16,72,104,29,13,3,0,11,0,0,0,0,3,0],[0.25,0.5,1,2,3,4,5,6,7,8,9,10,11,12,14])	22.0676
	Refrigerator temperature (ºC)	T_{st}	G([0,1,2,3,4,5,6,7,8,9,10,11,12],[0.01,0.02,0.05,0.09,0.11,0.17,0.22,0.15,0.12,0.04,0.01,0.01])	3.4344
	Probability of not washing hands	p_H	0.14	0.14
	Probability of not washing knife	p_K	0.038	0.038
	Probability of not washing board	P_B	0.27	0.27
	Probability of preparing salad	P_s	0.3	0.3
	Survival rate on the knife	tKK	0.0	0.0

	Survival rate on the board	*tBB*	0.02	0.02
	Transfer from pork cuts to board	*tPB*	0.03	0.03
	Transfer from board to salad	*tBS*	0.26	0.26
	Transfer form pork cuts to knife	*tPK*	0.05	0.05
	Transfer from knife to salad	*tKS*	0.58	0.58
	Transfer from pork cuts to hands	*tPH*	0.08	0.08
	Transfer from hands to salad	*tHS*	0.02	0.02
	Transfer from tap to hands	*tTH*	0.023	0.023
	Survival on hands	*tHH*	0.006	0.006
	Transfer from hands to tap	*tHT*	0.002	0.002

Table 14: Description of the values distributions used in Murmann stochastic CPM.

Step	Relevant Parameter(s) description	Notation	Value/distribution
Contamination at retail	Concentration of *Salmonella* at retail (log CFU/g)	N_{meat}	Normal(1.4,0.7)
	Prevalence at retail	p	0.12
Concentration in sausage after cooking	Per gram concentration (cfu $g^{-1)}$	C_c	$C_i/10^{\log R}$
	D- value (min)	D	$10^{(10.122-0.151T)}$
	Internal temp (ºC)	T	10.51+Normal(3.43, 0.19) × t
	Cooking time (min)	t	Betapert (15, 20, 30)
Numbers at consumption	Weight of one sausage (g)	Wt. sausage	63.5

Table 15: Description of the values and distributions used in the adapted stochastic CPM from Berjia.

Step		Relevant Parameter(s) description	Notation	Value/distribution
Contamination at retail		Prevalence of *Listeria monocytogenes* in cold smoked salmon	P	38.50%
Growth rate (logNt)	**Primary Growth Model**	Concentration of L. *monocytogenes* in CSS at the beginning of the storage (log 10 cfu/g)	$X_{0;i}$	~Normal (1,219; 0.8)
		specific growth rate of L. *monocytogenes* (d^{-1})	μ_i	Equation (7)
		Duration of the storage (d),	t_i	~Normal(14; 3,5)
		Lag time (d)	λ_i	0
		Maximum population density (log_{10} cfu/ g)	MPD	7.27
	Secondary Growth Model	Constant	$M_{ref}(d^{-1})$	6.19
		Constant	T_{ref}	25ºC
		Constant	T_{min}	-1.18 ºC
		Storage temperature	T_i	Annex (Cumulative distribution)

Dose of *Listeria* ingested (cfu/g)		Portion size (Fish intake)	F_{intake}	23g
		Dose of *Listeria* ingested (cfu/g)	$D_{Listeria}$	$F_{intake} \times N_t$

Table 16: Description of the values and distributions used in Berjia deterministic CPM.

Step	Relevant Parameter(s) description	Notation	Value/distribution
Contamination at retail	Prevalence of *Listeria monocytogenes* in cold smoked salmon	P	38.50%
	Concentration of L. *monocytogenes* in CSS at the beginning of the storage (cfu/g)	N_0	10^(~Normal (1,219; 0.8))
Storage, preparation and consumption	Growth rate of L. *monocytogenes* (log cfu/d)	μ	0.113
	Duration of the storage (days),	t	14
	Lag time (d) (WHO/FAO, 2004)	λ	0.167
	Storage temperature	Ti	5ºC
	Concentration of *Listeria* after storage	logNt	N_0+ 0.113 *(t-λ)
	Portion size (Fish intake)	F_{intake}	23g
	Dose of *Listeria* ingested (cfu/g)	$D_{Listeria}$	$F_{intake} * N_t$

Annex III

Table 17: Results of the relative risks obtained after simulation of six hypothetical intervention scenarios in the food production chain, for *Campylobacter* CPMs.

"STO" refers to stochastic CPM, "a" refers to using a surrogate "a-factor" (Duarte et al., 2016), DET refers to using a deterministic CPM. "SD" refers to standard deviation. "(C_{retail})" refers to the concentration of *Campylobacter* at retail.

Relative Risk Estimates	Nauta STO	Nauta "a"	Nauta DET	Christensen STO	Christensen "a "	Christensen DET	Calistri STO	Calistri "a"	Calistri DET
0,5 mean log reduction (C_{retail})	0.609	0.462	0.5980	0.564	0.464	0.5730	0.584	0.435	0.561
1 mean log reduction (C_{retail})	0.348	0.197	0.331	0.311	0.199	0.304	0.346	0.175	0.288
0,5 reduction SD (C_{retail})	0.664	0.269	0.672	0.600	0.272	0.597	0.689	0.2111	0.550
0,5 increase SD (C_{retail})	1.424	2.367	1.329	1.454	2.345	1.430	1.380	2.870	1.506

Table 18: Results of the relative risks obtained after simulation of six hypothetical intervention scenarios in the food production chain, for *Salmonella* CPMs. "STO" refers to stochastic CPM, "a" refers to using a surrogate "a-factor" (Duarte et al., 2016), DET refers to using a deterministic CPM. "SD" refers to standard deviation. "(C_{retail})" refers to the concentration of *Salmonella* at retail.

Relative Risk Estimates	Bollaerts STO	Bollaerts "a"	Bollaerts DET	EFSA STO	EFSA "a"	EFSA DET	Murmann STO	Murmann "a"
0,5 mean log reduction (C_{retail})	0.493	0.326	0.334	0.554	0.396	0.506	0.850	0.335
1 mean log reduction (C_{retail})	0.180	0.103	0.117	0.250	0.126	0.221	0.180	0.108
0,5 decrease in SD (C_{retail})	0.654	0.317	0.327	0.720	0.398	0.662	0.68	0.332
0,5 increase in SD (C_{retail})	2.00	5.300	5.211	1.361	2.635	1.360	1.43	4.161

Table 19: Results of the relative risks obtained after simulation of six hypothetical intervention scenarios in the food production chain, for *L. monocytogenes* CPMs. "STO" refers to stochastic CPM, "a" refers to using a surrogate "a-factor" (Duarte et al., 2016), DET refers to using a deterministic CPM.
"HP" refers to healthy population, and "SP" refers to susceptible population. "SD" refers to standard deviation. "(C_{retail})" refers to the concentration of *L. monocytogenes* at retail.

Relative Risk Estimates	Berjia HP STO	Berjia HP "a"	Berjia HP DET	Berjia SP STO	Berjia SP "a "	Berjia SP DET
0,5 mean log reduction (C_{retail})	0.80	0.3171	0.3165	0.80	0.3172	0.3165
1 mean log reduction (C_{retail})	0.66	0.1002	0.1001	0.66	0.1002	0.1001
0,5 decrease in SD (C_{retail})	0.950	0.236	0.235	0.950	0.236	0.235
0,5 increase in SD (C_{retail})	1.052	18.670	15.406	1.053	14.245	15.402

Annex IV

Figure 25: Illustration of the relative risks obtained by simulation of an intervention scenario of a reduction of the prevalence *Campylobacter* at retail by 10%. Results were calculated using three different modelling approaches: stochastic CPM (eg. Nauta STO), "a-factor" surrogate (eg. Nauta "a"), and deterministic CPM (Nauta DET).

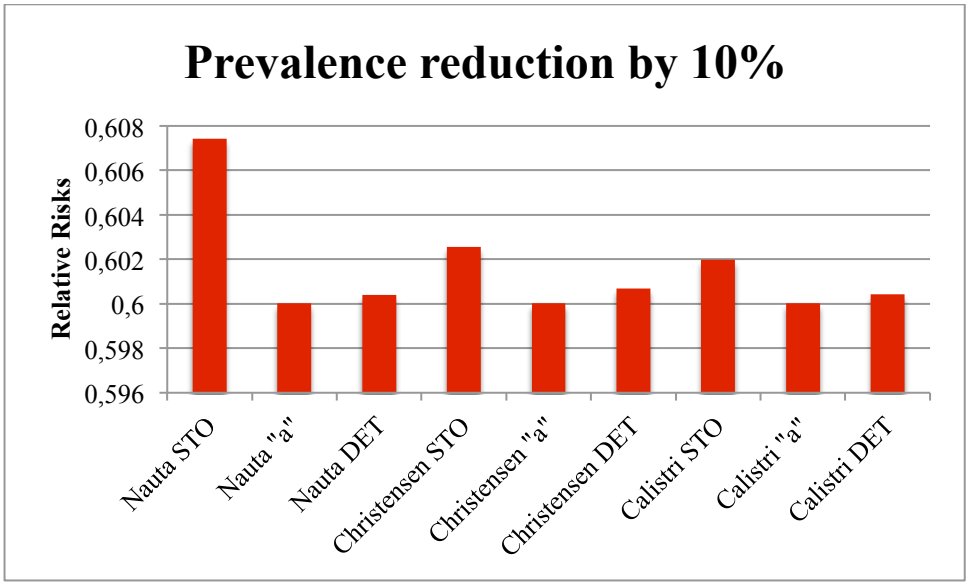

Figure 26: Illustration of the relative risks obtained by simulation of an intervention scenario of an increase of the prevalence of *Campylobacter* at retail by 50%. Results were calculated using three different modelling approaches: Stochastic CPM (eg. Nauta STO), "a-factor" surrogate (eg. Nauta "a"), and Deterministic CPM (Nauta DET).

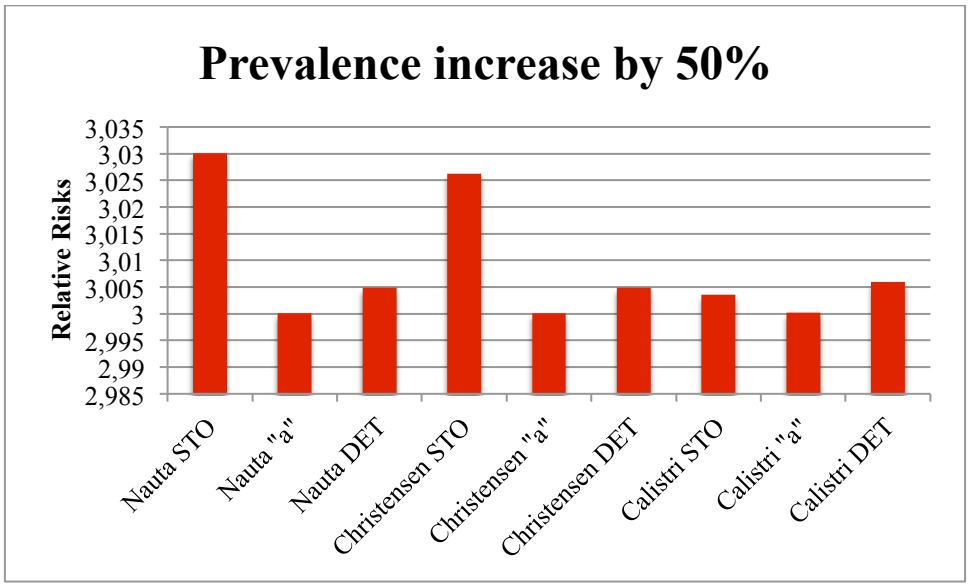

Figure 27: Illustration of the relative risks obtained by simulation of an intervention scenario of a reduction of the prevalence of *Salmonella* at retail by 10%. Results were calculated using three different modelling approaches: stochastic CPM (eg. EFSA STO), "a-factor" surrogate (eg. Nauta "a"), and deterministic CPM (EFSA DET).

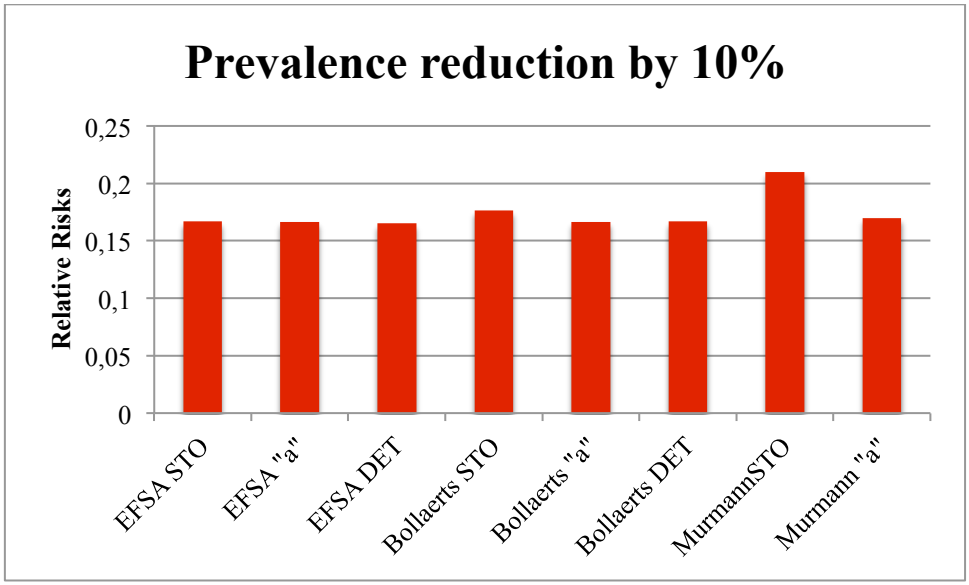

Figure 28: Illustration of the relative risks obtained by simulation of an intervention scenario of an increase of the prevalence of *Salmonella* at retail by 50%. Results were calculated using three different modelling approaches: stochastic CPM (eg. EFSA STO), "a-factor" surrogate (eg. Nauta "a"), and deterministic CPM (EFSA DET).

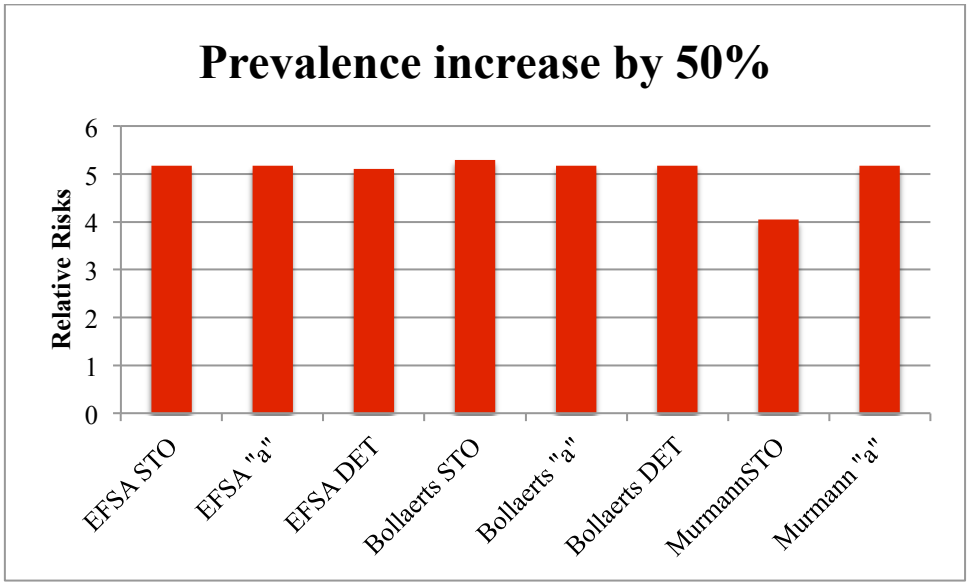

Figure 29: Illustration of the relative risks obtained by simulation of an intervention scenario of a reduction of the prevalence of *Listeria monocytogenes* at retail of by 10%. Results were calculated using three different modelling approaches: stochastic CPM (eg. Berjia STO HP), "a-factor" surrogate (eg. Berjia "a" HP), and deterministic CPM (Berjia DET HP). "HP" refers to healthy population; "SP" refers to susceptible population.

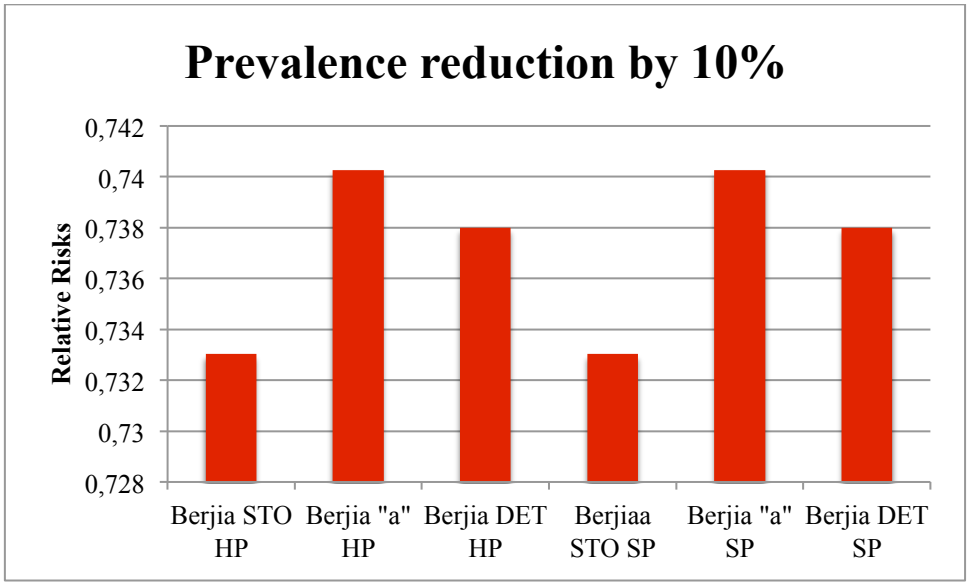

Figure 30: Illustration of the relative risks obtained by simulation of an intervention scenario of an increase of the prevalence of *Listeria monocytogenes* at retail by 50%. Results were calculated using three different modelling approaches: stochastic CPM (eg. Berjia STO HP), "a-factor" surrogate (eg. Berjia "a" HP), and deterministic CPM (Berjia DET HP). "HP" refers to healthy population; "SP" refers to susceptible population.