

Quantitative Microbial Risk Assessment Model to Estimate Exposure to *Campylobacter* from Consumption of Chicken in the United States

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ABSTRACT (ACADEMIC)

Public health costs of foodborne campylobacteriosis are estimated to cost more than a billion dollars in the United States annually. This pathogen has been primarily associated with chicken production and processing which is a ~\$33 billion industry. To further identify practices that could reduce *Campylobacter* presence, concentration, and persistence in chicken prior to consumption a quantitative microbial risk assessment (QMRA) was conducted for boneless, skinless chicken breast meals prepared and consumed domestically in the contiguous United States. This QMRA model was developed with @RISK software (Palisade Corp., Ithaca, NY) and data inputs can be easily modified and updated. QMRA is a powerful analytic method that can be utilized to model the dynamics between the food pathogen, food commodity, and ingestion. It provides insight into the impacts of the process in its interaction and its surrounding environment. The baseline model determined that consumption of this product resulted in annual means of infections: 328,257, illnesses: 108,174, hospitalizations: 27,754, deaths: 37, cases of *Campylobacter*-associated Guillain-Barré Syndrome (GBS): 1,373, and cases of *Campylobacter*-associated Irritable Bowel Syndrome (IBS): 9,501. The associated annual economic burdens were ~\$192 million for acute campylobacteriosis, ~\$77 million for GBS, and ~\$96 million for IBS.

The effects of targeting and modifying the baseline model's inputs within the farm-to-fork process were studied as follows: Post grow-out (1) prevalence and (2) concentration of *Campylobacter* on chickens at the farm prior to transport, (3) transport crate cleaning frequency prior to loading, (4) temperature storage conditions at post processing/ pre-retail, (5) the frequency of handwashing in the preparation and handling of a chicken meal, and (6) the combination *Campylobacter* mitigation models using the inputs from (2) and (4). Mean yearly illnesses are estimated to decrease by approximately half if on-farm *Campylobacter* prevalence was lowered to 35-50% from the baseline level of 76%. An additional ~50,000 illnesses can be expected if the proportion of home preparers who do not wash their hands increases from 8.3% (baseline) to 20%. The combined effects of reducing on-farm *Campylobacter* concentrations and increasing the proportion of product frozen in-plant have greater impacts on reducing yearly campylobacteriosis and associated costs than either intervention alone.

ABSTRACT (PUBLIC)

Campylobacter is a major foodborne bacterial pathogen that is not well known by the general public, although public health costs are estimated to cost more than a billion dollars in the United States annually. Chicken production is a ~\$33 billion industry that is affected by the contamination of this pathogen. In this study, a quantitative microbial risk assessment (QMRA) was performed to study the impact of *Campylobacter* on chicken meals prepared in the home. Societal and economic burdens were evaluated as well. The models developed provide comprehensive simulations that describe the spread, persistence, and the concentration of *Campylobacter* throughout the farm to fork process in the annual consumption of boneless, skinless chicken breast meals prepared and consumed domestically in the contiguous United States. Additional simulation models were created to compare methods for reducing *Campylobacter* along the food chain that could lead to fewer cases of campylobacteriosis in the United States, or what could happen if there was a breakdown in a hygiene step in the preparation of the chicken meal.

DEDICATION

To my parents, family, and friends – you have lifted me to get to this stage. I love you all. To my wife Jayme – with you by my side, the best is yet to come. I am excited for what the future holds for us.

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INTRODUCTION

Microbial food safety is of great concern to the public. The ramifications involved with foodborne illnesses extoll heavy burdens on public health, food production, retail markets, and healthcare. In times of data analytics, public health officials have a wealth of information and new methodologies that can be employed to aid in the decision-making process. The risk analysis framework can be utilized to assess and manage food safety risks, and to optimize consumer protection from foodborne illnesses. Within this system, natural scientists focus on *risk assessment* about food safety, and social scientists study different aspects of consumer risk perception and the development of effective communication strategies to reduce the impact of potential food hazards. Policy making must consider various facets of the same problem to be effective. Although considerable theoretical and practical advances in both the natural and social sciences, consumer public illnesses due to contaminated foods still represent a considerable threat to public health (FAO-WHO, 2009a; Jaykus et al., 2006).

Risk analysis is comprised of three components – risk management, risk assessment, and risk communication – with the overall scope representing a structured decision-making process. The three components must work in tandem with one another and are complimentary parts of the overall discipline of risk analysis (Bloomfield & Scott, 2013). Microbial risk assessment can be considered a “bottom up” approach in the trial to better understand this relationship, where it allows prediction of health outcomes based on structured models of the risk-generation process (Hoffmann et al., 2007).

The utilization of risk assessment becomes a conduit to provide relevant, quantifiable information to risk managers regarding food safety hazards, public health risks, and technical control options and to identify research needs. For public agencies to target food safety policy on the basis of risk, it is imperative to understand the relationship between the incidence of foodborne illness and consumer exposure to specific foodborne pathogens throughout the food supply (Batz, Doyle et al. 2005). The risk assessment phase links the presence of pathogens in food to public health outcomes, which facilitates regulatory and industry decision making with regards to foodborne disease control (Jaykus et al., 2006). Probabilistic models associated with a microbial risk assessment can be used as a tool to determine the uniformity of different food safety systems, in which the model used in pertinent scenarios have the ability to combine epidemiologic data and inferences with data and assumptions from other information sources in a rigorous, transparent manner to ascribe more completely the potential foodborne microbial hazards and the likely impact of control measures on the risk. Epidemiological and risk assessment approaches are complementary to each other and can be used in tandem to validate and inform each other (FAO-WHO, 2009c; Rosenquist et al., 2003). This information is necessary in to evaluate the probability of illness from a pathogen in each population.

The focus of this project is to facilitate the food safety decision-making process rooted in a quantitative microbial risk analysis in relation to a specific food commodity/microbial hazard combination throughout the farm-to-fork food chain. This risk assessment considers the impact of major control measures applied throughout the farm to consumption model chain in terms of human campylobacteriosis cases following the modular process risk model (MPRM) methodology for food chain quantitative microbial risk assessment (QMRA) (Schaffner, 2008). The risk stemming

from the consumption of boneless, skinless, chicken breast filets contaminated with *Campylobacter* spp. in the contiguous United States in a given year is analyzed. The effects of potential interventions are also measured and compared using the effect on changes to the total annual number of campylobacteriosis illnesses, the associated sequelae, and the economic burden as a metric are estimated. To account for variability and uncertainty @RISK® software (Palisade Corp., Ithaca, NY) is used to simulate risk assessment models and generate results in the form of distributions. As an add-in to Microsoft Excel® (Microsoft Inc., Redmond, WA), the @RISK software performs Monte Carlo simulation to show the distribution of risk assessment outcomes.

Chapter III describes the effect that selected interventions or scenarios could have on reducing *Campylobacter* prevalence, concentrations, cases of campylobacteriosis and associated public health costs. The scenarios presented in this chapter utilize the baseline model. Modifications are implemented to the baseline model to assay various stepwise alterations within the farm-to-fork process model. The alternative scenarios are developed to represent a broad range within the QMRA system. The effects of modifying the baseline model's inputs within the farm-to-fork process are studied as follows: Post grow-out (1) prevalence and (2) concentration of *Campylobacter* on chickens at the farm prior to transport, 3) transport crate cleaning frequency prior to loading, 4) the storage conditions post processing/ pre-retail, and 5) the frequency of handwashing in the preparation and handling of a chicken meal. Lastly, a combination *Campylobacter* mitigation model is developed using the inputs from a post grow-out intervention model (reducing concentration of *Campylobacter* on the bird prior to transport to the abattoir) and the post processing/ pre-retail model (increase proportion frozen).

CHAPTER I – LITERATURE REVIEW

Part 1 – *Campylobacter* spp. as an Agent of Foodborne Illness (Hazard Characteristics)

A. Clinical Features of Campylobacteriosis

Campylobacter-associated enteritis is the single largest cause for sporadic bacterial gastroenteritis infection world-wide (Gormley et al., 2008). It has been recognized as a definable disease for approximately 40 years with the collaboration between veterinarians and medical doctors, although the bacterium was described as early as 1886 by Theodor Escherich (Escherich, 1886), in a series of articles in the *Münchener Medizinische Wochenschrift* (Alter & Scherer, 2006) (Skirrow, 1977) (Butzler, 2004). *Campylobacters* were classified as vibrios but they were moved to a separate genus in 1963 (Skirrow, 1987). *Campylobacter* (30 taxa), *Arcobacter* (17 taxa), and *Sulfurospirillum* (7 taxa) are now included in the family *Campylobacteraceae* (Butcher & Stintzi, 2017).

Two major species of *Campylobacter* – *C. jejuni* and *C. coli*, are common causes of food-related and environmental morbidity associated with humans, although there appears to be emerging species that also cause human illnesses. *C. jejuni* is by far the most frequent species isolated, and is attributed to 80 – 85% of all campylobacteriosis cases (Gormley et al., 2008). *C. jejuni* is divided into two sub-species: *C. j. jejuni* and *C. j. doylei*. The latter is not commonly associated with human infection and illness. In its initial rise to prominence in the late 1970s, consumption of chicken was suggested to be a contributing factor for *Campylobacter*-associated illness (Batz et al., 2005; N. V. Harris, Weiss, & Nolan, 1986; Skirrow, 1977, 1991). Chicken consumption is considered a major source of campylobacteriosis (Butcher & Stintzi, 2017; FAO-

WHO, 2009c; Gras et al., 2012; Sahin et al., 2015; Skarp, Hänninen, & Rautelin, 2016; Vellinga & Van Loock, 2002; Wilson et al., 2008; Wingstrand et al., 2006).

Asymptomatic infection is common, as it is in other bacterial enteritis. However, *Campylobacter*-associated enteritis patients may exhibit mild to severe symptoms, usually pertaining to gastroenteritis that is clinically indistinguishable from salmonellosis or shigellosis. Abdominal pain may be more acute with campylobacteriosis. Patients are sometimes admitted to hospitals with suspected appendicitis (Skirrow, 1987). The symptoms for *Campylobacter*-related gastroenteritis can easily be misdiagnosed for an infection caused by another pathogen, misdiagnosed as Crohn's disease or ulcerative colitis, or it may often be diagnosed as "unknown causative agent", especially if the stool of the patient is not analyzed for *Campylobacter* spp. (Brooks, Butel, & Morse, 1998; Butzler, 2004; Wall, 2010). Moreover, emerging *Campylobacter*-related myocarditis and pericarditis have been documented (Hessulf et al., 2016; Uzoigwe, 2005; Veerasamy, 2016; Westling & Evengard, 2001).

Common clinical symptoms include watery, often bloody diarrhea (~40% of patients), abdominal pain, fever, headache, malaise, nausea, and fever of approximately 40°C (Epps et al., 2013b). In approximately 50% of patients, diarrhea is preceded by a febrile period with the symptoms mentioned; in patients exhibiting bloody diarrhea, fresh blood, as well as mucus or pus is present, suggesting colorectal inflammation (Butzler, 2004). Vomiting is rare with campylobacteriosis (Brooks et al., 1998; Butzler, 2004). Infections are usually self-limited to a period of five to eight days, rarely lasting longer than 10 days.

Infrequently, extra-intestinal infections or post-infection complications such as reactive arthritis and neurological disorders occur. *C. jejuni* has recently become the most recognized

antecedent cause of Guillain-Barré Syndrome ((GBS), incidence of 1/1000 infections), a polio-like form of paralysis that can result in respiratory and severe neurological dysfunction or death (Alshekhlee et al., 2008; Butzler, 2004; Crushell et al., 2004; Frenzen, 2008; Poropatich, Walker, & Black, 2010; van den Berg et al., 2013). In general, 20 – 40% of GBS cases are believed to be associated with preceding campylobacteriosis (Butzler, 2004; Epps et al., 2013a; Poropatich et al., 2010; Tam et al., 2007; Wall, 2010; Wierzba et al., 2008).

The incubation period is commonly two to six days from infection to illness but can last longer than 10 days. This prolonged incubation period confounds investigations of potential outbreaks because one is usually looking for the source attribution at least a week retroactively. The pathophysiology of *Campylobacter* infection is still somewhat poorly understood, and animal models have proven inadequate in tracking pathogenesis (Epps et al., 2013a; Kaakoush, Mitchell, & Man, 2015). Fecal excretion of *Campylobacter* in confirmed cases is not usually protracted, with ~50% of patients being culturally negative three weeks post-recovery (Skirrow, 1987). The significance lies in that *Campylobacter* transmission is more likely to be of animal origin rather than person-to-person. Symptomless excretors are not considered to be a public health risk even if they handle food.

B. *Campylobacter* spp. Characteristics

The name *Campylobacter* is derived from Greek, meaning ‘curved rod’ (Skirrow, 1987). *Campylobacter* spp. are estimated to be the one of the top two most prevalent causative agents for known bacterial foodborne disease, along with salmonellae (Hendricks et al. 2000; Humphrey et al., 2001). Since its confirmation as a human pathogen in the 1970s, there is still much

uncertainty associated with its behavior, the modes of transmission, and overall characteristics. Some widely-held establishments associated with the bacteria have been questioned in recent research, and it is evident that there is still great uncertainty in its utility of mechanisms for survival, and for the best isolation method to use from various samples ((Hilbert et al., 2010; Skarp et al., 2016; Skirrow, 1991).

Campylobacter spp. are small, curved or spirochete-shaped Gram-negative bacilli that exhibit rapid darting and spinning motions by means of unsheathed, long flagellum at one or both poles of the bacterial cell. They have been described as an “S”, or “gull-wing” shape (Brooks et al., 1998). Skirrow (1987) describes it as such: *The flagellar apparatus is an organ of supreme energy and efficiency, as these bacteria are among the fastest known. Spinning around their long axes like a rifle bullet, they are capable of instantaneous reversal, and this cholera-like darting motion can often be seen in fresh faecal preparations by dark-ground or phase-contrast microscopy, thus permitting a rapid presumptive diagnosis to be made.*

Campylobacter spp. have an optimal growth temperature of 37 – 42°C, and optimal growth is seen on primary plates when grown at 42 – 43°C. The isolation and identification in speciation is predicated by its culture characteristics, where selective media is utilized. Although *C. jejuni* grows well at 36 – 37°C, incubation at 42°C prohibits growth of most of the other bacteria present in feces, thus simplifying the identification of *C. jejuni*. *Campylobacter* spp. cannot grow below 30°C (Jacobs-Reitsma, 2000). It does, however, survive well below its growth temperature and exhibits physiological activity at 4°C (Hazeleger et al., 1998).

Campylobacter spp. have been described as strictly microaerophilic (Skarp et al., 2016; Skirrow, 1977, 1982, 1987). They require 3 – 15% CO₂ and 3 – 5% O₂ for growth as they have a

respiratory metabolism; this results in the production of toxic reactive oxygen species, such as the hydroxyl radical and H₂O₂, that might lead to nucleic acid and protein degradation in the microorganism (Skarp et al., 2016). In water, *C. jejuni* survive well at 20 – 30°C but survive longer at near-refrigeration or refrigeration temperatures, which has also been seen in raw food products such as poultry (Acheson, 2008; Bhaduri & Cottrell, 2004; Solow, Cloak, & Fratamico, 2003).

Although *Campylobacter* spp. are well-adapted in their role as an intestinal pathogen, the bacteria's fastidious nature outside the body – being microaerophilic and thermophilic, as well as its temperature requirement for growth makes *Campylobacter* an unlikely candidate to be a major foodborne pathogen (Alter & Scherer, 2006; Birk et al., 2004; Boronovo, 2006; Buzby, 2001; Davis & Conner, 2007; Hendricks et al., 2000; Josefsen et al., 2002; Kalmokoff et al., 2006; Kudra et al., 2012; Murphy, Carroll, & Jordan, 2006; Park, 2005; Sandberg et al., 2005). Despite these seemingly apparent shortcomings as an ideal foodborne pathogen, a significant number of people suffer from *Campylobacter*-related illnesses annually. The infectious dose has been estimated to be as low as 500 organisms (Black et al., 1988; Medema et al., 1996a, 1996b; Rieu et al., 2007).

C. Clinical and Environmental Sampling and Isolation

Campylobacter identification procedures include motility tests, oxidase tests, and direct detection of *C. jejuni* and *C. coli* antigens in feces by latex agglutination. However, the primary procedures for the diagnosis of suspected *Campylobacter*-associated enteritis involves direct microscopic examination of fresh liquid feces, either with Gram's stain (counterstain with carbol fuchsin) using brightfield microscopy or a wet mount using phase-contrast microscopy and culturing on selective media (Butzler, 2004; Kaakoush et al., 2015).

Campylobacter spp. are differentiated from other standard food and environment bacteria by virtue of their spiral morphology and extremely rapid darting and spinning motions for viable cultures, although the characteristics of viable but non-culturable cells (VBNC) are different (Butcher & Stintzi, 2017). In general, two forms of morphology have been described for *C. jejuni*: culturable, and generally non-culturable coccoid forms. The transition from viable forms to non-viable forms depends on factors such as strain differences, environmental temperature, pH, osmolarity, and medium used for growth, maintenance, and transition times (Josefsen et al., 2002; Lazaro et al., 1999; Park, 2005; Pearson et al., 1993; Tangwatcharin et al., 2006). The reason for the transformation from viable to nonviable remains uncertain, especially the infectivity of nonculturable forms (Lazaro et al., 1999; Murphy et al., 2006; Tangwatcharin et al., 2006). A variety of stressors can result in a complete change in the morphology and physiology of *Campylobacter* spp. – which result in the adoption of a coccoid form and transition to the VBNC state (Tangwatcharin et al., 2006). Modern serology systems are efficient in detecting campylobacteriosis, and blood cultures should be taken if the clinical features and immunocompetence of the patient are indicative. They may also be ordered for certain culture negative (VBNC) cases of suspected *C. jejuni* infection involving reactive arthritis, erythema nodosum, and Guillain-Barré Syndrome.

C. jejuni and *C. coli* can be isolated easily from stool samples by primary plating on selective media and incubation for 48 – 72 h at ~42°C in a microaerophilic atmosphere. Several selective media are in widespread use, such as Skirrow's medium which incorporates vancomycin, polymyxin B, and trimethoprim; while other selective media contain cefoperazone, other antimicrobials, and inhibitory compounds (Brooks et al., 1998). However, the antibiotics in the

selective media and the elevated incubation temperature (42°C) may also inhibit the growth of some of the less well-recognized *Campylobacter* spp. If a *Campylobacter* spp. other than *C. jejuni* is suspected, stool filtration and culture on a non-selective medium, with incubation at 37°C and 42°C should also be used, with another option being the Hippurate hydrolysis test (Butzler, 2004).

When the diagnosis of infection is based exclusively upon culture on selective media, greater than 95% of *Campylobacter* infections would appear to be caused by *C. jejuni* and *C. coli*, and to a minor degree *C. lari*, and *C. upsaliensis*. However, refinements in isolation and identification methods including serotyping, phage typing, pulsed-field gel electrophoresis (PFGE), and genotyping using PCR-based identification methods, have enabled clinicians and researchers to characterize the species and strains further for better clarity of the scope and reach of this human pathogen. Related species such as *C. upsaliensis*, *C. lari*, *C. fetus* subsp. *Fetus*, *C. jejuni* subsp. *Doylei*, *C. concisus*, *Arcobacter butzleri*, and *Arcobacter skirrowii*, have been isolated from human patients with diarrhea (Butzler, 2004; Newell, 2005).

D. Survival of *Campylobacter* in Poultry Production and Processed Products

Unlike salmonellae, multiplication of *Campylobacter* spp. in food or the food environment is unlikely, and focus should be more on survival rather than growth. There are indications that the pathogen may not be as fragile in the environment as once thought, however (Alter & Scherer, 2006; Buswell et al., 1998; Davis & Conner, 2007; Hilbert et al., 2010; Murphy et al., 2006).

Campylobacter have the ability to survive in the farm and processing environments, and also survive cleaning procedures (Peyrat, Soumet, Maris, & Sanders, 2008a, 2008b). The ability of *Campylobacter* to survive the battery of stresses experienced during environmental exposure and

cleaning processes is dependent on the presence and expression of stress response genes (Peyrat et al., 2008a). These properties, along with external factors such as biofilm formation and organic soiling might affect the genetic diversity of the *Campylobacter* population prior to and after cleaning.

Certain strains of *Campylobacter* can take a very long time to repair cellular damage and regenerate from its VBNC state (Humphrey, 2001). It is within most established protocols to incubate growth media for only 48 hours when samples are examined for *Campylobacter* spp (Butcher & Stintzi, 2017). However, this may result in false negatives.

Protective Factors

Hermans, et al. (Hermans, Van Deun, et al., 2011) suggests that *C. jejuni* occupies a protected niche in the ceca of the infected broiler, making the pathogen less susceptible to the bactericidal effects of the cinnamon-oil ingredient trans-cinnamaldehyde observed *in-vitro*, yielding no reduction in *Campylobacter* counts in the broiler cecum. This was the case for both *in-vivo* models utilized in the study. It was concluded that these observations put the possible use of in-feed plant-derived antimicrobial compounds to effectively control caecal *C. jejuni* colonization in poultry in question. There was a marked discrepancy between in-vitro and in-vivo activity of the active ingredient tested, where neither in a classic in-vivo trial nor in the caecal loop experiment was trans-cinnamaldehyde able to reduce caecal numbers of *C. jejuni* strain KC 40, despite marked *in-vitro* reduction using the same compound. However, the authors noted in the review that the probability of the bacterium to reach and colonize the broiler cecum could be reduced by supplementing medium-chain fatty acids to the broiler feed or even completely prevented with a combination of formic acid and sorbate.

Biofilm Production

Campylobacter spp. can form and multiply in biofilms on a variety of surfaces (Joshua et al., 2006). *Campylobacter* spp. have been described as having the ability to maintain its viability in mixed biofilms even under conditions that inhibit growth. Survival was evident underneath the biofilm even with low-nutrient media and unfavorable atmospheric conditions for one week at 10°C (Alter & Scherer, 2006). The surface coverage of the biofilms formed was correlated with the survival rate of the pathogen, where an increase in formation of viable but non-culturable (VBNC) cells was noted by Trachoo and Frank (Trachoo & Frank, 2002), however Kalmokoff et al. (Kalmokoff et al., 2006) did not. They observed coccoid forms, but also reported that some cells retained their natural spiral shape. Protein formation of *Campylobacter* spp. may provide a clue into the biofilm formation. In the same experiment, they identified differences in protein expression profiles in bioforming bacteria and planktonic bacteria – proteins involved in motility (*flaA* and *flab*), the filament cap (*fliD*) and basal body (*flgG* and *flgG*) and the chemotactic protein (*cheA*) exhibited higher expression levels in biofilms. Also, proteins involved in the general stress response (*GroEL* and *GroES*), adhesion (*flaC* and *peb1*) and proteins involved in biosynthesis and energy generation showed higher expression. Initial findings that *Campylobacter* spp. form biofilms under stress is of significant interest for its ability to preserve itself under duress, however, the persistence in the environment is still very speculative (Joshua et al., 2006).

Commensalism

A study conducted by Hilbert, et al. (2010) may give insight into the survival mechanism of *Campylobacter* spp. on the surface of poultry. The team demonstrated that *C. jejuni* could withstand conditions of atmospheric oxygen tension and maintain viability when the pathogen

was co-cultured with *Pseudomonas* spp., a primary spoilage organism in poultry that sometimes causes nosocomial infections among humans. A combination of the two organisms showed a prolonged survival of *C. jejuni* in atmospheric conditions up to 48 hours, whereas a mixture of other common spoilage organisms yielded survival of *C. jejuni* in the environment for no longer than 18 hours. Moreover, metabolic commensalism regarding oxygen depletion is known from bacterial communities building up biofilms, where ancillary bacteria creates a microenvironment that optimizes survival of anaerobic obligate and anaerobic/microaerophilic bacteria by locally reducing oxygen levels (Hilbert et al., 2010). The authors state that microbial commensalism was diverse in *C. jejuni* isolates from different sources; *Campylobacter* isolates from chicken meat and humans showed greater ability to survive than fecal isolates from broilers in a co-mixture with *P. putida*. In observations under scanning electron microscopy, the development of fiber-like structures intertwining *C. jejuni* and *P. putida* cells suggests metabolic commensalism between the two organisms.

Through quorum sensing, *C. jejuni* may sense the environment for low oxygen tension and actively flagellate to the site, and interact using flagella apparatus and motility (among other mechanisms) with strictly aerobic bacteria (Finlay & Esteban, 2009). This would lead to finding a microenvironment that enhances their survival on the surface of food products (Gurry, 2007). These mechanisms may point to a possible survival mechanism for *C. jejuni* on chicken meat, and thus provide an explanation of the differences of strain prevalence at different stages of poultry growing, processing, retail, and consumption.

Internalization of Campylobacter spp. and Surface Contamination

In broiler carcasses, *Campylobacter* has been associated with the chicken skin as well as the overall surface of the carcass (Oyarzabal, 2005). Removal of the skin in the processing pathway can reduce counts of this pathogen, it does not, however, have an effect on the numbers of *Campylobacter* on the internal surfaces of the carcass (Berrang et al., 2002).

Undercooking poultry is commonly recognized as a direct risk to the consumer due to the significance of surface contamination. *Campylobacter* found within the muscle, however, is not regarded in microbial risk assessments. *Campylobacter* spp. has been isolated from deep muscle tissues of chickens sampled either in the factory or from retail outlets (Berndtson, Tivemo, & Engvall, 1992; Humphrey et al., 2001; Lubert & Bartelt, 2007; Phillips, 1995). There is the possibility that internalization of *Campylobacter* spp. in the deep tissue of poultry may pose a greater direct risk to public health than surface contamination in issues of undercooking (Berrang et al., 2002; Humphrey et al., 2001; Phillips, 1995).

In a study from Lubert and Bartelt (Lubert & Bartelt, 2007), approximately 20% of fillets harbor *Campylobacter* spp. within the deep tissue, a region where some of the bacteria has the potential to survive if undercooking were to occur. The concentration of *Campylobacter* spp. in the breast meat was $-1.54 \log \text{CFU/fillet (146 g)}$. All samples identified with internal contamination in this study, also carried *Campylobacter* on their surface. However, in a study conducted by Phillips (Phillips, 1995), internalization of *Campylobacter* could occur without surface contamination, as two out of three surface-contaminated samples harbored *Campylobacter* in the flesh of chickens. The author reiterated the case that internalized pathogens pose a greater health hazard to consumers than undercooking. In a 1992 study of Berndtson et al. (Berndtson et al.,

1992) as reviewed by Luber and Bartelt (Luber & Bartelt, 2007), the investigators analyzed the prevalence of *Campylobacter* spp. in Swedish broiler chicken breast and thigh muscles and found that only 3% of the tested samples were positive for the pathogen. Several factors may play into the uncertainty of internal contamination, including: 1) developments and sensitivities in sampling and detection, 2) diligence for testing for internalization, 3) different processing steps and interventions used in lowering the bacterial count throughout processing, such as country-to-country methodologies and practices, 4) different usage of packaging methods such as MAP, vacuum, CO₂ enhanced packaging, and 5) adaptability and protein expressions in different *Campylobacter* serotypes.

Investigations in the distribution of *Campylobacter* spp. have revealed that the pathogen is spread unevenly on and within poultry (Luber & Bartelt, 2007; Phillips, 1995). Phillips (Phillips, 1995) provides an explanation to the non-correlation between isolation of organisms in different sections of the chickens by the isolation procedures used. The standard isolation method in testing for *Campylobacter* spp. may not isolate damaged organisms which may, be able to cause illness when ingested. Overall, not much data exist on the internalization of *Campylobacter* spp. on the tissue of chicken meat (Berndtson et al., 1992; Luber & Bartelt, 2007).

E. Virulence and Toxin Production

Campylobacteriosis can occur in the infected patient by at least three mechanisms: 1) intestinal colonization by ingested cells and production of bacterial cytotoxin, 2) bacterial invasion of intestinal cells of the jejunum, ileum, and colon, and 3) extraintestinal translocation, in which the organisms cross the intestinal epithelium and migrate via the lymphatic system to various

extraintestinal sites. These possible mechanisms are not mutually exclusive, and *Campylobacter* may make use of any combination of these mechanisms depending on the host status and attributes of the infection strain (Zheng et al., 2006).

The acidic environment of the human intestinal tract can influence *Campylobacter* survival and pathogenesis. Murphy et al. (Murphy et al., 2006) reviewed that HCl does not have a significant effect on the survival of *Campylobacter* spp. After 30 minutes in pH 4.42 – 5.35 in yogurt, the pathogen was still able to be recovered. HCl has little inhibitory effect at pH values above 5.0. Gastric acid is a potent barrier against ingested pathogens and is primarily a pH-HCl-dependent mechanism. Due to the reported sensitivity to *Campylobacter* spp. to acidic conditions, it has been suggested that ingesting the pathogen with buffers such as milk or with water that aid rapid wash through could reduce the oral infective dose. That acid-sensitive organisms can survive *in vitro* when inoculated onto the surface of certain food surfaces, combined with their survival in an acidified broth environment is a good indicator of the ability of pathogens to survive extreme acid conditions when ingested with certain food sources. An adaptive tolerance response (ATR) induces a survival mechanism in *Campylobacter* spp. that leads to tolerance of the susceptibility to unfavorable conditions (Murphy et al., 2006).

Tam et al. (Tam et al., 2009), reported that use of acid-suppressing medications was a major risk factor in *Campylobacter* enteritis. Chicken-related exposures accounted for 41% of adult cases and the recent use of acid-suppressing medications increased the risk for illness three-fold, accounting for 10% of *Campylobacter*-associated illness in this case-control study conducted in England during 2005 – 2006. They reviewed the work of Neal and Slack (Neal & Slack, 1997), which was in agreement with their finding.

In human campylobacteriosis, invasion in and translocation across the epithelial cell barrier of the intestine are important steps for causing enteritis. Despite the well-established importance of *Campylobacter* as a human pathogen, specific mechanisms of virulence in campylobacteriosis are still not fully understood. Both adhesion and invasion may be important in the pathogenesis of the disease and enterotoxin and cytotoxic factors have been described in strains of *C. jejuni* (Dasti et al., 2010; Johnson & Lior, 1988; Zheng et al., 2006). *Campylobacter* spp. produce various cytotoxins called cytolethal distending toxins (CDT). These are the only verified *Campylobacter* toxins identified, so far (Dasti et al., 2010). Symptoms associated with human campylobacteriosis have not been definitive attributable to the toxin, although it may truly be a virulence factor (Wassenaar, 2011). CDT were found to induce cell distension in studies performed *in vitro* using cells lines such as HeLa, Chinese Hamster Ovary (CHO), and Caco-2 cells which is characterized by elongation, swelling, and eventual cell death (McFarland & Neill, 1992). In studies of CDT *in vivo*, CDT causes inflammation (production of interleukin (IL-8), recruitment of dendritic cells, macrophages, and neutrophils) to the infection site in the intestine (Epps et al., 2013a; McFarland & Neill, 1992). In contrast, the host response in chickens does not promote inflammation of the intestinal epithelium. Likewise, CDT induce the production of neutralizing antibodies only in man, but not in chickens, indicating host-specific recognition of *C. jejuni* antigens (Dasti et al., 2010).

F. Chronic Sequelae from Campylobacteriosis

Acute gastroenteritis from *Campylobacter* infection has established itself as a major foodborne societal burden. Secondary illnesses experienced by the patient after the initial onset of campylobacteriosis is also coming into light as a significant attribute to the overall cost of illness

(direct medical expenditures and secondary expenditures in loss of productivity) and disease burden as measured by quality of life, although the extent of what types of chronic sequelae developed and the full impact of known sequelae is limited. In this section, a few notable chronic sequelae associated with campylobacteriosis are discussed in further detail.

Irritable Bowel Syndrome

Irritable bowel syndrome (IBS) is a common ailment of the gastrointestinal tract with a multifactorial etiology and no reliable biomarker (Ballou & Keefer, 2017). It is a chronic functional gastrointestinal (GI) ailment characterized by abdominal distress and altered bowel habits. IBS stands in the top 10 reasons patients consult primary care physicians, the most commonly diagnosed disease by gastroenterologists, and accounts for approximately 12% of total conditions diagnosed by primary-care physicians (Ballou & Keefer, 2017). Scallan et al. (Scallan et al., 2015) reported that among an estimated 845,000 annual cases of domestically acquired foodborne campylobacteriosis, it is expected that there would be 74,000 cases of post-infectious IBS.

In diagnosis, the Rome III diagnostic criteria represents the gold-standard for the diagnosis of IBS and include symptoms of recurrent abdominal pain/discomfort for ≥ 3 days/ month over the past three months that are associated with ≥ 2 of the following: improvement with defecation, onset associated with a change in the stool frequency, or onset associated with a change stool form (Ballou & Keefer, 2017; Drossman, 2006). IBS is classified into subtypes based on the predominant stool pattern, including IBS with constipation (IBS-C), IBS with diarrhea (IBS-D), and IBS with mixed bowel patterns of constipation and diarrhea (IBS-M). IBS-D estimated to account for $\sim 33\%$ of cases (Ballou & Keefer, 2017).

Approximately 10 – 20% of adults in the United States suffer from IBS (Buono et al., 2017a, 2017b; Nellesen, Yee, Chawla, Lewis, & Carson, 2013). The disease is often seen more frequently in women, with 60 – 75% of the attributable cases being female (Nellesen et al., 2013). Of the total number of cases, approximately 6 – 17% develop IBS following acute gastroenteritis (Spiller & Lam, 2012). Post-infectious IBS predominantly manifests itself in the form of IBS-D, followed by IBS-M (International Foundation for Functional Gastrointestinal Disorders, 2016).

The total annual cost of IBS in the United States is estimated to be between \$1.7 and \$10 billion in direct medical costs, excluding prescription and over-the-counter medicine, and up to \$20 billion dollars in indirect medical costs (Nellesen et al., 2013). Nellesen et al. (Nellesen et al., 2013) estimates that in the U.S. the direct cost per-patient for IBS ranged from \$2,269 to \$12,007 (2012 USD) annually, while the indirect cost ranged from \$1,356 to \$9,933 (2012 USD) annually. The distribution health care costs vary widely, particularly outpatient costs (12.7% to >50% of total costs), inpatient costs (6.2% to 40.8%), and cost of medicine (5.9% to 46.6%). They make no distinction to the subtype of IBS in their cost of illness estimations in their findings, although most valuations are derived from IBS-C.

Ballou and Keefer's estimation specifies the mean all-cause health care cost of IBS to IBS-D was \$13,038 (standard deviation of \$24,645) per case, per year (Ballou & Keefer, 2017; Buono et al., 2017a). In this attribution, approximately half (50.3%) was allotted to outpatient services such as diagnostic tests and laboratory or radiology services. The remaining costs were attributed to prescriptions (19.5%), inpatient admissions (13.6%), emergency room visits (8.5%), and visits to the doctor (8.1%).

Medical treatment associated with IBS are more or less suboptimal and illness burden is high (Ballou & Keefer, 2017; Buono et al., 2017a, 2017b). In discussing indirect costs, along with physiological nature of gastrointestinal distress, IBS sufferers are more likely to miss work, experience decreased productivity, and use many healthcare resources (Ballou & Keefer, 2017). IBS-related cases account for 1.5 – 2.7 million physician visits annually (Nellesen et al., 2013). Moreover, due to the chronic stress associated with the disease, approximately 40 – 70% of individuals with IBS meet the criteria for a psychiatric disorder, exhibiting comorbid depression, anxiety, and/or panic disorders (Ballou & Keefer, 2017). The disease attributes to impairment and or avoidance of daily activities in the patient.

Reactive Arthritis

The term “reactive arthritis” (ReA) is the phenomenon of inflammation in joints and other tissues following enteric infection. This term was first coined in 1969 by Ahvonen and colleagues to describe arthritis that occurs during or after an infection of another body site, without evidence of initial infection of the joint – also termed sterile arthritis (Townes, 2010). Multiple joints can be affected, but it is predominantly a lower-limb arthritis especially in the knee, with symptoms of pain and incapacitation usually resolving completely within several months of onset (Nielsen et al., 2010; Pope et al., 2007).

ReA typically develops within four weeks following a gastroenteritis or urogenital infection with obligate or facultative intracellular bacteria, such as *Campylobacter*, *Salmonella*, *Shigella*, *Yersinia*, *Clostridium difficile*, *Chlamydia*, and to a far lesser extent, *E. coli* O157:H7 (Pope et al., 2007). In *Campylobacter*-associated ReA most symptoms last less than 6 months. Acute

gastroenteritis may lead to a chronic inflammatory state with elevated C-reactive protein and erythrocyte sedimentation rate (Nielsen et al., 2010).

At the onset of ReA, the symptom of the triggering infection has subsided and the diagnostic tests using stool samples are usually negative; the causative agent is confirmed via a serological analysis, although the organism cannot be always identified (Nielsen et al., 2010; Pope et al., 2007). To provide clarity to the ambiguous nomenclature of ReA, most rheumatologists apply the designation of ReA only to infection caused by *Campylobacter*, *Salmonella*, *Shigella*, *Yersinia*, or *Chlamydia* species and that sterile arthritis that follow other infections to be labeled as “postinfectious arthritis” (Townes, 2010).

The prevalence of acute ReA has been estimated between 1 – 7% post-campylobacteriosis, with it being described in 0.7 – 24% of cases (Nielsen et al., 2010; Pope et al., 2007). Scallan et al. (Scallan et al., 2015) estimates *Campylobacter*-associated ReA at 7%, basing this estimation on a Finnish population study that diagnosed ReA by physical examination. The incidence among case-patients were then compared and matched with controls. The estimates of those cases seeking medical care was set at 44%, based on a U.S. study (Schonberg-Norio et al., 2010). Pope et al. (Pope et al., 2007) estimated that the annual incidence of ReA after bowel or urogenital infection is 30 to 40 per 100,000 cases. In Finland, the annual incidence was reviewed to be 4.3 per 100,000 cases. The most affected age group of ReA is young adults with equal incidence shared between males and females. Like other foodborne pathogens associated with ReA, the sequela seems to be a rare complication of *Campylobacter* infection among children (Pope et al., 2007).

In 2003, the United States spent \$322.8 billion on medical costs related to rheumatological conditions (Townes, 2010). Due to the ambiguity of ReA in diagnosis, very few studies directly

mention the cost associated per case of ReA (Scharff, 2010). The study estimated the cost per case value of \$3,742, with a total societal cost at \$7.9 billion annually for *Campylobacter*-related cases. The estimation is based on the sum of medical costs and monetized QALY losses (productivity losses in the USDA model). In formulating the estimation, the author notes the values very conservative, lower bound estimate as it does not include arthritis symptoms persisting longer than 25 weeks past the resolution of the acute foodborne illness due to the lack of reliable data on these chronic conditions (Roberts, 2013).

As Townes (Townes, 2010) states, “ReA is a concept, not a well-defined disease.” To date, there is great uncertainty surrounding the overall disease burden of ReA, and the full extent of it cannot be put into focus without greater stringency on the definition, clinical spectrum, and objective classification criteria of the sequelae. Although this sequela may be a significant source of economic and social burden associated with post-*Campylobacter* infection, the true scope remains very uncertain.

Guillain-Barré Syndrome

Longer-term sequelae associated with campylobacteriosis include reactive arthritis, Reiters Syndrome, and Guillain-Barré Syndrome (GBS) (Crushell et al., 2004; Havelaar et al., 2007; Mangen et al., 2004). Guillain-Barré Syndrome is an autoimmune disease of acute progressive neuropathy that is characterized by paralysis, pain, muscular weakness, and distal sensory loss. It is characterized by the demyelination of motor and sensory nerves or deterioration of axonal nerves of the peripheral nervous system. The nerve damage can lead to muscle weakness, paralysis, and death. GBS has become the most frequent cause of acute flaccid paralysis since the elimination of

poliomyelitis in the world (Buzby & Roberts, 1997; Crushell et al., 2004; Epps et al., 2013a; Tam et al., 2007).

There are three major sub-forms of GBS that affect the peripheral nervous system. The major sub-forms of GBS are: 1) acute inflammatory demyelinating polyneuropathy characterized by the demyelinating of peripheral nerves, 2) acute motor axonal neuropathy characterized by degeneration of axonal components of peripheral nerves, and 3) Miller-Fischer syndrome characterized by areflexia, and ophthalmoplegia (Epps et al., 2013a).

Many medical researchers believe that GBS is a reaction by a person's immune system responding to fight off several potential triggers, such as a gastrointestinal or respiratory infection (Alshekhlee et al., 2008; McCarthy & Giesecke, 2001). One trigger associated with GBS is an onset of post-*Campylobacter* infection. An estimated 20 – 50% of patients with Guillain-Barré Syndrome had become infected with *Campylobacter* 1 – 3 weeks prior to the onset of GBS symptoms (Buzby, Allos, & Roberts, 1997; Crushell et al., 2004) (Nyati & Nyati, 2013; Poropatich et al., 2010; Tam et al., 2007; Wierzba et al., 2008). Scharff (Scharff, 2010) estimated the cost per case is \$2,165, with a total societal cost at \$4.5 billion annually for *Campylobacter*-related cases. In the United States in the late 1990s, it was estimated that of the 2,658 to 9,575 cases annually, 532 to 3,830 of the patients had a preceding *Campylobacter* infection (Buzby & Roberts, 1997). Conversely, it is estimated that approximately 1 in 1,000 cases of campylobacteriosis will result in GBS (Butzler, 2004; Crushell et al., 2004; Mangen et al., 2004). Similarly, in the Netherlands, it is approximated that of the average annual campylobacteriosis cases (~79,000), there would also be 59 cases of GBS/year attributed to the illness (1 in 1338) (Mangen et al., 2004). Ellström et al. (Ellström et al., 2014) reported that the virulence traits of GBS associated with *Campylobacter* infection include

the ability of some *C. jejuni* strains to incorporate sialic acids in the lipooligosaccharide of the cell wall, resulting in structures mimicking human gangliosides.

About 20% of cases of GBS require mechanical ventilation due to paralysis. Of those, it is estimated that 63% of patients improve to resume work, 27% suffer permanent or prolonged disability to prevent reentry into the workforce and 10% die. Of the 80% of cases that do not require mechanical ventilation, approximately 81% resume work and 19% experience prolonged or permanent disability. No mortality is expected in non-ventilated cases (Buzby & Roberts, 1997; Poropatich et al., 2010).

The overall mortality rate for GBS is reported to be 3% to 13% (Alshekhlee et al., 2008; van den Berg et al., 2013), with the most frequently described cause of death being attributed to respiratory failure, pneumonia, cardiac arrest, and autonomic dysfunction. The in-hospital death rate as per the 2007 national statistics on outcomes of hospitalized patients suffering from Guillain-Barré Syndrome by HCUPnet of the Dept. of Health and Human Services (Agency for Healthcare Research and Quality, 2009) is in agreement with the death rate posed by Buzby and Roberts (Buzby & Roberts, 1997). Of the 6,346 patients discharged with acute infectious polyneuritis (ICD-9-CM 357.0), there were 130 in-hospital deaths reported (2.04% with an SE of 0.41%) in 2007.

Part 2 – U.S. Production and Consumption of Chicken

A. U.S. Production

In the United States, broiler (chickens that are bred and raised specifically for meat consumption) production plays a significant portion of the meats and poultry sector. In 2013, the U.S. production of live-weight broilers was 37.83 billion pounds, and production has increased incrementally, with the 2017 forecast to be around 41.53 billion pounds (United States Department of Agriculture, 2017). The U.S. exports for the time for broilers ranged from ~7.4 billion pounds in 2013 to a forecast of ~6.9 billion pounds for 2017 (United States Department of Agriculture, 2017).

B. International and National Valuation

The United States, China, Brazil, and the European Union make up approximately 73 percent of the world's broiler production, with the U.S. holding about 27 percent of global production (Harris, 2008). The United States ranks second in global broiler export, holding about 1/3 share of the total in the world poultry trade (as of 2011), while the top exporter, Brazil, accounts for 40% of total world exports on poultry meat (Harris, 2008; Johnson, 2015). The United States exports about 20% of chicken and about 12% of turkey produced in the nation (U.S. Poultry and Egg Association, 2016b).

The value of poultry and poultry parts (broilers, eggs, turkeys, chickens) in 2014 was \$48.3 billion (U.S. Dept. of Agriculture, 2015). In the breakdown of the valuation, broilers made up 68%, eggs 21%, turkeys 11%, and <1% from chickens of the proportion. The value of broilers produced

during 2014 was \$32.7 billion, which was an increase of 6% from 2013 (U.S. Dept. of Agriculture, 2015). Excluding broilers, the value of sales from chickens during 2014 was \$966 million, a jump of \$87.9 million from 2013.

The annual total economic impact of the poultry industry was estimated at around \$470 billion (U.S. Poultry and Egg Association, 2016b). A direct impact of \$188.8 billion is associated with 374,100 jobs nationally. From farming to manufacturing, to transportation, and retailing, the poultry industry has an impact of \$281 billion (2014) (U.S. Poultry and Egg Association, 2016b). Moreover, \$32.9 billion in taxes (\$11.7 billion state and \$21.2 billion federal) is associated with the industry.

C. Value of Poultry Industry to Virginia

The poultry industry is a major agricultural commodity in Virginia, and it is home to eight major poultry processing plants. In 2014, the economic activity of this industry was responsible for \$13.8 billion (U.S. Poultry and Egg Association, 2016a). Of that, the chicken industry contributed the majority the impact, creating more than \$10.5 billion to the Commonwealth's economy, which created or supported around 39,000 total jobs (Virginia Poultry Federation, 2016). The industry is also a sizable tax stream, where it paid about \$678 million in federal taxes and \$386 million in state and local taxes.

Broiler chicken production value for 2014 was \$917,917,000, and turkeys accounted for \$325,987,000 of the total for the Commonwealth (U.S. Dept. of Agriculture, 2015). In terms of production, Virginia ranked 8th nationally in broiler chicken production, where 262,000,000 heads (1,441,000,000 lbs.) were produced in 2014 and ranked 5th nationally in turkey production, where

more than 16,800,000 heads (443,520 lbs.) were produced in the same year (Virginia Poultry Federation, 2016; U.S. Dept. of Agriculture, 2015). More than 800 chicken farms and greater than 300 turkey farms are in place in the Commonwealth to sustain the industry.

D. Potential Economic Problems of Poultry Production

The industry faces enormous and complex challenges, which include the rising cost of feed ingredients, concern of bird health, environmental impact of grow-out and processing, pandemic diseases, and volatility of consumer preference (Harris, 2008). Consumer preference will continue to define the landscape of the industry, with consumption of certain parts of the chicken being preferred over another (U.S. Chicken Council, 2012) as well as uncertainty involving the methodology of grow-out of chickens (i.e. antibiotic usage) (Harris, 2008). Consumers are also raising concerns about the welfare and treatment of the birds including stunning, slaughter and handling methods, and processing (Harris, 2008). The conveyance of how the industry is using due diligence to food safety issues is also of concern.

E. U.S. Consumption of Chicken

The availability and low-cost of broilers and broiler products have led to a gradual annual increase in consumption of these food commodities in the United States since the 1960s. Per capita consumption of total chicken (broilers and other chickens – retail weight) increased from 28.0 lbs. in 1960, to 66.2 lbs. in 1990, to an estimated 108.1 lbs. (91.3 lbs. broilers) for 2017 (U.S. Chicken Council, 2017). U.S. consumption of poultry meat surpasses that of beef or pork. Specifically, for broiler consumption, which this risk assessment focuses on, the per capita

boneless retail weight (meat consumption) for 2015 was 62.3 lbs. (U.S. Dept. of Agriculture ERS, 2016).

Whether in or out of the home, chicken meals have become a major staple in the diet of U.S. consumers. In a survey conducted by the National Chicken Council (U.S. Chicken Council, 2012), the average number of meals or snacks that contained chicken eaten by the respondents was 5.2 in the prior two weeks. The survey described that the average 2012 rate for consuming chickens purchased at a supermarket (presumably at home) was 3.4 times in a two-week period. Moreover, the frequency of eating fresh/frozen, boneless and skinless chicken breast meat/breast tenders that was cooked at home during a four-week period was 3.8 (U.S. Chicken Council, 2012). Chicken consumption did not differ significantly by gender.

Of those that purchased boneless and skinless chicken breasts for consumption at home, 41% of respondents noted they buy only when on sale at featured lower price and stock up to put in home freezer, 25% responded that they buy as needed without too much concern for price, and 19% responded that they buy only when on sale at a featured lowered price but do not stock up to put in home freezer, and 6% of those surveyed stated that they only purchased skinless/boneless chicken breasts for special occasions, such as gathering of extended family and friends, outdoor grilling, food for special gathering outside of home, and similar occasions (U.S. Chicken Council, 2012). Price followed by convenience were the top two reasons consumers would eat boneless and skinless chicken breast/breast tenders more often.

F. On-Farm Contamination of Broilers with *Campylobacter*

In a typical grow-out house, the birds are reared on litter, with feed and water liberally

available. While on the growing farm, the birds may be exposed to a variety of potential sources of *Campylobacter*. These may include insects, rodents, other farm animals, and wild birds (Ellis-Iversen et al., 2012; Tudor et al., 2015). Unless rigorous control of hygiene is enforced, farm staff may also introduce *Campylobacters* from external sources (Ellis-Iversen et al., 2012).

Contaminated drinkers on the grow-out farms have been found to be associated with *Campylobacter*-positive poultry flocks. These drinkers were only contaminated after the birds were colonized (Ellis-Iversen et al., 2012). Drinkers may harbor bacteria and may be contacted by multiple birds. The contaminated water drinkers would then act as a distributor of bacteria within a flock rather than an introduction vehicle.

The contamination of the shed entrance and anteroom is a likely step in the pathway between the flock and a potential persistent or incoming bacterial reservoir on the farm. Contamination of this area can occur in both directions as this source is one of the first areas contaminated after the flock is infected, and on occasions, found to be contaminated before the flock. The main doors and anteroom act as critical barriers into the shed, and special attention is warranted in the sanitation program. Partial depopulation or thinning (the practice of creating space in houses as the birds grow by culling a subpopulation of the flock) also poses as a risk factor for *Campylobacter* colonization of flocks due to breaches of the shed's biosecurity barrier (FAO-WHO, 2009c).

Although catching crews and their equipment were often found to be contaminated with *Campylobacter* prior to their arrival on the farm, catchers and their crew or other vehicles coming onto the farm were not significantly associated with *Campylobacter* (+) flocks. Ellis-Iversen, et al. (Ellis-Iversen et al., 2012) explain that a correlation may not have been made due to a large

proportion of flocks are already positive upon the arrival of the catching teams, thus not exposing to confounding risk.

In industrialized countries, *Campylobacter* is highly prevalent among broiler flocks, averaging 60 – 80% of birds being colonized with the bacterium at the slaughter age (Herman et al., 2003; Hermans, Van Deun, et al., 2011; Rasschaert, Houf, & De Zutter, 2007). The concentration levels are reported to be around 7 to 9 log₁₀ CFU/g in the cecum of an infected broiler (Bahrndorff et al., 2015). They also noted that the level of *Campylobacter* was slightly lower than in contents, with a geometric mean count decreasing from 7.4 log₁₀ CFU/g on day 4 to 6.9 log₁₀ CFU/g at day 12 post-infection, and that there was a significant correlation between the level of the pathogen in individual fecal and caecal samples before slaughter. Kaakoush et al. (Kaakoush et al., 2015) reported that *C. jejuni* strains survive in chicken feces for up to 6 days after excretion, and that, chicken feces may be a potential source of transmission to other chickens at the farm, during transport, as well as to the environment and humans when poultry manure is used as fertilizer. Giombelli and Gloria (Giombelli & Gloria, 2014) concluded that flocks of chickens infected with *Campylobacter* are 30 times more likely to produce carcasses contaminated with high prevalence and counts of *Campylobacter*.

Manifestation of Campylobacteriosis on Broilers

Campylobacteriosis in broilers is mostly commensal but it may be exhibited as a chronic, contagious bacterial disease. It has been referred to as *avian virbrionic hepatitis*, where it manifests into liver disease, unevenness, and increased culls in young growing flocks, and reduced egg production in older birds (Bell & Weaver, 2002). Clinical exhibitions of symptoms in chickens is not commonly observed but subclinical carriage is common in flocks (Awad et al., 2015).

The dynamics of *Campylobacter* infection is not yet fully understood in broilers, however, chicken lineage and time of colonization may influence variability in colonization of the intestines of birds (Berrang et al., 2004; Hazards, 2011; Hermans, Van Deun, et al., 2011; Newell & Fearnley, 2003; Stas, Jordan, & Woldenhiwet, 1999). However, there seems to be a causal link between fecal samples at the farm level and caecal load at the time of slaughter (Bahrndorff et al., 2015).

Feed Withdrawal

The duration of holding time during transport as well as the time the birds are kept before processing may play a role in the level of *Campylobacter* shedding. Feed withdrawal is the process of removing poultry access to feed several hours prior to loading and transport to the abattoir. This is intended to mitigate intestinal contents from contaminating carcasses during processing, as birds will frequently defecate onto both the transport crate surfaces and onto other birds (Wesley et al., 2009). The time allotted allows time for the feed in the digestive tract to pass through and be voided. Feed is generally removed approximately 8 – 12 hours prior to processing, during this time the birds remain in the broiler house with access to water for as long as possible prior to catching. In proper implementation, during the time the birds are caught and caged, feed passage slows dramatically, which optimizes the number of birds that have empty and reconcilable tracts (Corry & Atabay, 2001). However, feed withdrawal, as well as the transport from farm to abattoir, is stressful for the birds and can increase fecal shedding of pathogens. Off-feed time that exceeded 14 hours promoted the decrease of tensile strength of intestinal walls, which were more likely to rupture during the evisceration process. According to Keener, et al (Keener et al., 2004) the number of contaminated live birds on the exterior, mainly feathers, can increase 10-fold during transport.

G. Catching and Transport

Traditionally, the bulk of broilers are caught and caged at dusk or at night with dimmed lights to minimize movement of birds. However, with the growing consumer demand for chicken and chicken products, most major processing plants operate under multiple shifts, and it has become necessary to catch a portion of the birds during daylight. Grow-out houses make the use of curtain sidewalls and transportable dark curtains that are hung in the open sidewalls to lower light intensity during catching (2002). Ideally, the birds are picked up gently and placed in transport cages or crates to minimize bruising and injury.

Virtually every aspect of broiler production has been automated over the past few decades except the catching and unloading process related to transport. The process of catching, loading, and unloading is still performed almost entirely by hand. Typically, crews of 7 – 10 persons catch and crate at a rate of 7,000 – 10,000 birds per hour, where a typical flatbed tractor trailer holds ~6,000 – 7,000 birds. The loading time ranges from one-half to one hour per truck (2002).

Birds must be protected from weather conditions that may cause stress after they are loaded into transport cages and placed on the live-haul truck. Birds in transport cages are susceptible to heat-stress during hot weather, and this is especially true if they are caught, loaded, and transported during morning or daytime. Chickens that have been raised in evaporative cooled houses that have not been acclimated to warm temperatures are also at risk (Chauvin et al., 2011). Spraying water on the live haul trucks has proven effective at keeping the birds cool during loading and holding (2002; Chauvin et al., 2011). However, this process is likely to spread *Campylobacter* among chickens. The live haul process should ideally be scheduled to minimize the latent time of the broiler in the crates and prior to processing. This effort can maximize yields by mitigating

weight loss of birds, reduce dead on arrivals (DOAs), reduce contamination and condemnations, as well as maximize use of transport equipment.

Cross-contamination in Transport

Berrang, et al. (2003) investigated the role of previously used dump cages on the contamination of previously negative broilers. After separating out other cross-contamination factors, it was shown that *Campylobacter* soiled onto the dump cages may withstand washing procedures, and the pathogen can contaminate the consequent flock loaded onto the soiled transport cages and survive on the surface of processed carcasses. Newell et al. (Newell et al., 2001) and Slader et al. (Slader et al., 2002) found in their respective studies that the *Campylobacter* subtype isolated from crates prior to loading could be found on previously negative flocks post-holding.

The gut contents and feces of *Campylobacter*-positive broilers have been shown to contain high numbers of *Campylobacter*, in excess of 10^7 CFU/g (Berrang, Dickens, & Musgrove, 2000; Berrang, Northcutt, Fletcher, & Cox, 2003; Epps et al., 2013a). Transportation and stress can increase the rate of *Campylobacter* shedding from positive birds (Epps et al., 2013a). This increased shedding then leads to heavy cage contamination and potential exterior contamination of broilers. The numbers of *Campylobacter* cells recovered from the caeca and the surface of *Campylobacter*-positive broilers are higher following transport than at the farm prior to transport (Epps et al., 2013a).

The role of transport cages in the contamination of previously negative birds was studied by Hansson, et al. (Hansson, Ederoth, Andersson, Vågsholm, & Olsson Engvall, 2005).

Campylobacter was detected on the processed carcasses of previously negative broilers following

exposure to contaminated feces (on the used transport cages) for 2, 4, or 6 hours. In their study, 26 slaughter groups found negative at farm level that were transported in batches of crates from which *Campylobacter* had been isolated. After sampling was performed at the slaughterhouse, *Campylobacter* was detected in 11 of 26 groups (42%) that were previously negative. The concentration of these newly contaminated birds may not be as high as those found in positive flocks, however. Berrang et al. (Berrang et al., 2003) noted in their study that the broiler carcasses contaminated by exposure to the feces of a positive flock had significantly lower numbers of *Campylobacter* per rinse sample than did carcasses from the positive flock itself.

Receiving

Once at the abattoir, shaded areas with fans and evaporative cooling equipment are necessary to maintain the bird's temperature while awaiting processing. During extremely cold weather, birds must be protected from excessive wind chills, rain, and snow during transport by using tarps and wind breaks at the front part of the trailer. Processing begins when trailers loaded with birds are moved from the holding shed into the plant receiving area for slaughter. A forklift is used to remove the full hauling units (compartmentalized cage or drawer systems) from the trucks in the unloading area. Once the compartmentalized cages/drawers are emptied, they are re-stacked onto another trailer. Slaughtering of birds occur approximately 8 to 12 hours after feed withdrawal and crating to mitigate yield loss and fecal contamination (Bell & Weaver, 2002). The unloading of the birds with the container system was identified as a risk factor for *Campylobacter* contamination levels on broiler carcasses (Seliwiorstow et al., 2016).

Transport Cage Cleaning

In a typical commercial operation prior to processing, broiler chickens are caught and

loaded into cages or crates – mechanically or by hand, then are transported on over-the-road trailers to the processing plant where they are held until slaughter (Berrang et al., 2003; Northcutt & Berrang, 2006). The most used method in the United States employs compartmentalized cages. These transport cages are constructed of plastic or wire drawer with a hinged door surrounding a rigid steel base. The cages are usually a five-tiered galvanized frame container with fiberglass floors (Ramesh et al., 2003). Each compartmentalized cage unit holds roughly 300 birds and has 15 separate cages (Bell & Weaver, 2002; Rasschaert et al., 2007).

Soiled poultry transport containers may not be cleaned between uses and thus may act as a vehicle in spreading pathogenic microorganisms such as *Campylobacter* and *Salmonella* from one batch of birds to the next during transportation and flock thinning (Corry & Atabay, 2001; Slader et al., 2002). Northcutt and Jones (Northcutt & Jones, 2004) surveyed chicken processing plants across the United States in 2004 and found that only 28.4% of the respondents wash and sanitize transportation cages and trucks between each use. Even when the cages are washed, no standard commercial procedure exists for washing poultry transportation cages, and research has shown that improperly cleaned transportation cages or crates can harbor pathogenic bacteria, and transfer these microorganisms to subsequent flocks (Berrang, Meinersmann, & Hofacre, 2011; Berrang et al., 2003; Hansson et al., 2005; Harnett, Kelly, Newell, & Woolridge, 2001; McCrea, Tonooka, VanWorth, Boggs, & et al., 2006; Slader et al., 2002). When transport cages were washed, some respondents reported washing cages by hand, whereas others indicated the use of an automated system. Rasschaert et al. (Rasschaert et al., 2007) notes the process of cleaning the cages consists of first soaking in cold water (~15°C) for approximately 2 minutes, spray with cold water, then spray with disinfectant. The cages are then rinsed with high pressure cold water.

Jacobs-Reitsma and Boulder (Jacobs-Reitsma & Boulder, 1998) observed that sometimes 'clean' transport crates were more often contaminated with *Campylobacter* than dirty crates. Moreover, in the study it was noted that birds held for 4 hours or longer became colonized in naturally contaminated crates. Complete removal of all fecal matter during washing requires a great amount of water and time, as is the case of the immersion/sanitation method. The phenomenon of finding *Campylobacter* on cleaned transport crates has been seen in other studies (Hansson et al., 2005; Slader et al., 2002).

Slader et al. (Slader et al., 2002) observed that *Campylobacter* was isolated from crates cleaned under normal conditions on four of five visits to the processing plant but was isolated from wash water only on two visits. When crates were washed using the recommended concentration of detergent of quaternary ammonium compounds (QACs) (0.5 – 1% v/v at 40 – 80°C, *Campylobacter* was isolated from a lower proportion of crates, but only after a second addition of detergent. *Campylobacter* was isolated from all six unwashed crates, and during this visit the temperature of the crate wash water was 30°C.

Biofilms are problematic in the proper cleaning, and consequently, the reduction of pathogens such as *Campylobacter* on transport cages. Slader et al. (Slader et al., 2002) suggested that organisms present on the crates embedded on organic matter (biofilms) were more protected than planktonic cells, thereby neutralizing the effectiveness of the conventional cleaning methods audited by the authors on transport cages. They conclude the number of contaminated crates at the farms and the results of the washing experiments performed under normal conditions demonstrates that the cleaning process had little, if any, effect on the *Campylobacter* and *Salmonella* status of the transport crates. They stress that thorough cleaning, including the

removal of fecal material, needs to occur prior to adequate disinfection can take hold.

Northcutt and Berrang (Northcutt & Berrang, 2006) detected a low initial prevalence (1/27) of *Salmonella* on the surface of cages; yet they found *Campylobacter* on 26% (7/27) of the initial samples. Given that many risk managers focus on the association of the success or shortcomings of the microbiological safety of poultry processing and poultry products to control of *Salmonella*, the impetus for transport cage cleaning may be discounted by some parties involved. An added concern is that there exist few studies in the area of transport cage surveillance and cross-contamination control presently. However, the evidence for the economic and societal impact of *Campylobacter*-associated infections and illnesses is great, and transport-cage washing may be an overlooked avenue to reduce this burden.

The cleaning of transport cages can reduce the number of surface bacteria. Northcutt and Berrang (Northcutt & Berrang, 2006) observed a reduction of approximately 1.6 log CFU/surface area tested in *coliform* counts between (pressure spray) washed and unwashed crates for *coliforms*, and a 2.2 log CFU/surface area tested reduction when a sanitizing chemical solution was used in combination with the washing step. A novel method of transport cage decontamination, which involves immersion of the transport cages (Berrang et al., 2007), (Ramesh et al., 2003) led to a significant reduction in the number of recoverable bacterial numbers from contaminated transport cages. Ramesh et al (Ramesh et al., 2003) demonstrated a 2.4 to 4.2-log reduction in *coliforms* using a combination of high pressure washing and immersion in a tank containing heated water or an aqueous sodium hypochlorite solution of 1000 ppm NaOCl, respectively. Moreover, a synergistic effect of combining heat treatment and chemical treatment was seen.

H. Slaughter and Processing of Chicken

Most chickens that are consumed for their meat are processed in large-scale commercial processing plants using in-line operating speeds of up to 8,000 birds/ hour. The process is virtually fully mechanized and is carried out in defined, factory-like setting. This stage of the chapter provides an overview of the processing of chicken, considering each of the stages to the hazard characterization in this risk assessment.

The initial processing step starts off with the transfer of chickens from transport crates to individual shackles on the processing line, where they are then stunned using electrically charged water or held in a chamber with carbon dioxide gas, prior to bleed out by an incision at the neck. Stunning and slaughter have little effect on the levels or prevalence of *Campylobacter* or *Salmonella* (FAO-WHO, 2009b, 2009c; Giombelli & Gloria, 2014; Harnett et al., 2001; Y. Lehner, Reich, & Klein, 2014).

There are many avenues for changes in the prevalence and concentration of *Campylobacter* during processing. Along with the transport phase, the steps involved in processing broilers for shipment to retail facilities harbor numerous avenues for cross-contamination of *Campylobacter* between slaughtered flocks and birds from the same flock (Allen & Griffith, 2001; Allen et al., 2007; Allen et al., 2000; FAO-WHO, 2009c; Herman et al., 2003; Hermans, Van Deun, et al., 2011). For example, Elvers, et al. (Elvers et al., 2011) reported that during random processing of positive and negative flocks, some carcasses from a *Campylobacter*-negative flock processed immediately after a *Campylobacter*-positive flock can be contaminated with levels of the pathogen comparable to those seen in a positive flock. The pathogen level measured appeared to decline after about 5,000 birds from the negative flock had been processed

and contamination on the end product had waned to a level that was very-low or undetectable.

Elvers et al. (2011) also stated that there was no clear evidence of differences in strains over time.

Scalding

The purpose of scalding is to facilitate removal of feathers from the follicles of the carcass, and to help loosen or remove feces, litter, and bacteria from the skin (Humphrey 1987; Lehner 2014; Stern, 2003). To loosen these feathers, the carcasses are submerged in one or more baths of hot water, in a counter current flow, which serves to denature the protein structures holding the feathers in place (Sams, 2001a). Scalding of chicken carcasses has been implicated as a major potential site for cross-contamination for pathogenic bacteria.

There are two particular combinations of latent time and temperature for scalding that have become industry standards (Conner, Davis, & Zhang, 2001). Scalding at approx. 50°C for two to four minutes is called “soft scalding” and loosens the feather with minimal change to the quality of the outer skin of the carcass. This process leaves the waxy, yellow-pigment cuticle layer of the skin intact. This is the preferred method if producing fresh poultry with a yellow skin exposed, referred to as New York dressed. The resulting color is highly desired in some parts of the world as indicating a healthy bird (Conner et al., 2001). The alternative to the “soft scalding” method is to submerge the carcass in water baths at higher temperatures (58-64°C) for a shorter time (45 s) – a process called “hard scalding.” This method is the predominant method used in processing method in the United States and yields a significant microbial reduction on the surface of the carcasses. Hard scalding allows for easier feather removal than milder scalding conditions (Northcutt, 2001).

Oyarzabal (Oyarzabal, 2005) reviewed that soft scalding is less effective in reducing the

number of *Campylobacter* on carcass skins. Moreover, the reduced temperature scalding water in the scald tank has been implicated as a potential site for cross-contamination (Humphrey & Lanning, 1987; Humphrey, Lanning, & Leeper, 1984). The extent to which this occurs is dependent on the viability of the organisms in the scald tank, the organic load of the water in the tank, and the treatment parameters (Humphrey & Lanning, 1987; Yang, Li, & Johnson, 2002). Variables such as temperature and pH in the scalding process are important determinates of the efficacy of scalding in reducing bacterial numbers (Humphrey, 1981; Humphrey & Lanning, 1987; Humphrey et al., 1984; Kelana & Griffith, 2003; Zweifel, Althaus, & Stephan, 2015). Maintenance of the scald water at a pH of 9.0 ± 0.2 significantly increased the death rates of the skin flora as well as pathogenic bacteria on the carcass and in the scald water, even during soft scalding (Humphrey & Lanning, 1987; Humphrey et al., 1984).

There are conflicting reports on the effect of scalding on levels of *Campylobacter* on the exterior of the carcass. Some studies have shown significant reduction levels of *Campylobacter* contamination (~ 3 -logs) during scalding while others have shown minimal changes in the microbial load (Izat et al., 1988; Keener et al., 2004; Osiriphun et al., 2012; Zweifel et al., 2015). Oosterom, et al (Oosterom et al., 1983) observed on average a 2-log reduction in the level of *Campylobacter* contamination following a high temperature scald (58°C), compared with variable reductions following a low temperature scald (mean reduction 1.3-logs at 51.8°C). Yang et al. (Yang, Li, & Johnson, 2001) is in agreement with the level of reduction as shown by Oosterom et al (Oosterom et al., 1983), where scalding temperatures of ~ 50 and $\sim 60^{\circ}\text{C}$ produced reductions of 1.5 and 6.2 log CFU/ml in the water tank of planktonic cells and <1 and >2 log CFU/cm² on chicken skin, respectively.

Campylobacter counts are generally reduced after scalding; however, the pathogen can remain viable in the intestines and recontaminate the carcass during defeathering and later in evisceration. The microbial populations of the birds immediately post-scald changed during the working day corresponding to changes reported for scald-tank water (Humphrey, 1981). This effect was immediately seen on carcasses entering the plucking machines for defeathering (Alvarado, 2006; Humphrey, 1981; Humphrey et al., 1984) Post-scald treatments of hot water applied, as to not decrease the quality of the carcass, were not effective in lowering *Campylobacter* counts immediately or 30-minutes post scald (Berrang & Dickens, 2000; Humphrey & Lanning, 1987).

Overall, the effects of scalding should produce some reduction of *Campylobacter* numbers on the exterior of the carcasses. While scald water in the bath provides the first potential major source of cross-contamination in the processing plant, its continuous overflow allows for the addition of fresh water and the reduction of microbial levels. Also, when condensate is allowed to accumulate on the ceiling, this can drip onto the product and may become a source for contamination (Bell & Weaver, 2002). The evaluation process of the overall effect that scalding the imparts as a reduction step in the microbial load and the cross-contamination potential as an environmental factor may be confounded in that this step is often conjoined with the defeathering process (Bell & Weaver, 2002). Moreover, the limitation to further increase reduction is limited as the modification of this process stage (scalding system, temperature, and time exposition) may not be cost-effective and the increase in temperature or duration of exposure may lead to a decrease in product quality (Frederick & Huda, 2011; Osiriphun et al., 2012; Sams, 2001a).

Defeathering

Following scalding, the carcasses move to the defeathering process. Defeathering has frequently been implicated as a source of carcass contamination during poultry processing. Using a series of machines referred to as roughers or pickers, the feathers and loose soiling from the skin are removed. The time for the entire operation requires approximately 30 seconds (Bell & Weaver, 2002). These machines incorporate continuous water sprays that aid in flushing away the feathers and other debris as they are removed. A final process of removal of attached feathers may be performed by hand (Bell & Weaver, 2002; FAO-WHO, 2009c; Sams, 2001a).

A significant elevation in the concentration and occurrence of contamination of poultry carcasses with *Campylobacter* is seen after defeathering. Studies have observed an increase in bacterial load of generally 0.5 to 2.5-log, but as much as a 3.7-log increase on carcasses after defeathering has been reported (Berrang et al., 2007; Berrang, Buhr, Cason, & Dickens, 2001; Berrang & Dickens, 2000; Berrang & Dickens, 2004; Berrang, Northcutt, & Dickens, 2004; Guerin et al., 2010). The prevalence of *Campylobacter* infected birds was noted as having a distribution of 25% - 100% per flock with a mean of 75% sampled flocks (Berrang et al., 2007). Increases in prevalence and concentration from carcasses sampled before and after the process can be largely attributed to the escape of fecal material through the cloaca

The principle microbiological problem with defeathering is cross-contamination of carcasses associated with the mechanical action of the machines, the tendency to disperse microbiological contaminants in all directions via aerosols (Berrang, Northcutt, et al., 2004), and contact from other carcasses. It is probable that the pressure applied to the carcass during the process is likely to force out fecal matter through the cloaca by the action of the picker fingers

pressing on the abdomen. (Berrang et al., 2006; Musgrove et al., 1997). Some carcasses may become cross-contaminated by new *Campylobacter* strains between slaughtered flocks (Zweifel et al., 2015).

In the overall scope of broiler processing from defeathering to chilling of carcasses, Zweifel et al. (Zweifel et al., 2015) noted that the concentration of *Campylobacter*, once elevated after the defeathering process tended to remain constant from plucked to chilled carcasses. This is in agreement with previous studies (Berrang & Dickens, 2000; Berrang & Dickens, 2004; Berrang, Northcutt, et al., 2004; Guerin et al., 2010; Hermans, Van Deun, et al., 2011). However, one major note is the contrast with the mechanism modeled in the FAO/WHO model (FAO-WHO, 2009c), in which defeathering results in a significant decrease in concentration and prevalence of *Campylobacter* simulated.

Evisceration

During evisceration, the chicken viscera pack is removed from the carcass, and the edible and inedible giblets are separated. It is a coordinated series of highly automated operations that may vary substantially in sequence and design from plant to plant and from one equipment manufacturer to another (Sams, 2001a). This process is performed to ensure that the bird's digestive tract is not ruptured, which would lead to gross cross contamination with fecal matter (Brizio et al., 2015) (Brizio et al., 2015; Smith et al., 2007). Three basic procedures occur during the evisceration step: 1) the body cavity is opened by making a cut from the posterior tip of the breastbone to the cloaca; 2) the viscera (primarily the gastrointestinal tract and associated organs, reproductive tract, heart, and lungs) is removed; and 3) the edible viscera or "giblets" (heart, liver, and gizzard) are harvested from the extracted viscera, trimmed of adipose tissue, and washed with

water. The neck, usually considered a part of the giblets, is collected later, after inspection of the carcass for wholesomeness (Berrang et al., 2002; Berrang, Smith, et al., 2004; Sams, 2001a).

Evisceration of the birds being processed in the abattoir is a step that yields an increase of *Campylobacter* concentration in the bird; it also serves as a site for cross contamination of other birds being processed (Berrang, Smith, & Meinersmann, 2011; Zweifel et al., 2015). Environmental factors such as knives, scissors, and worker gloves may be a source for cross-contamination since they generally have very high microbial counts prior to startup (Bell & Weaver, 2002). Other environmental sources for cross-contamination include belts, shackles, rail dust, hang back racks, and contaminated air.

Campylobacter colonization of the chicken intestinal tract is a significant avenue to contributing to the overall level of contamination seen in the bird during slaughter and processing (Brizio et al., 2015; Rosenquist et al., 2003). In contaminated birds, the cecum harbors an abundant amount of *Campylobacter* (Berrang, Smith, et al., 2011; Brizio et al., 2015; Hermans, Martel, et al., 2011). *Campylobacter* populations have reported to be (mean log CFU/g) 4.7, 7.3, and 7.2 mean log CFU/g in the crop, ceca, and colon, respectively (Berrang, Buhr, et al., 2000). Van Gerwe, et al. (van Gerwe et al., 2010) detected a caecal content average of 8.5 log CFU/g, and a median crop colonization level of 4.8 log CFU/g.

Campylobacter in the crop of birds can spread as a result of rupture more easily than those spread resultant of an intestinal rupture, which is less commonly seen in processing of broiler carcasses (van Gerwe et al., 2010). Improvements in defeathering and evisceration methods could reduce the amount of fecal contamination on the carcasses and cross-contamination between carcasses in the abattoir, that should result in a reduced *Campylobacter* load on carcasses.

Although care is taken in this process, fecal matter may be spilled or leaked onto the surface of the bird. This contamination must be removed prior to the carcass entering the chill tank, as stipulated by U.S. Department of Agriculture Food Safety Inspection Service (FSIS) regulations (Berrang et al., 2007). The presence of visible fecal/biliary material on the surface of the carcass must be removed by washing or cutting the portion contaminated, since the affected area may indicate probable point of microbiological contamination (Smith et al., 2007). Ideally, when properly performed, this wash/removal step should allow the birds to go back online with the rest of the broilers for further processing.

I. Carcass Washing

After evisceration, the final wash step consists of a cabinet with a series of stationary nozzles spraying water that may contain antimicrobial agents, inside and outside the body cavity (Loretz, Stephan, & Zweifel, 2010; Northcutt & Jones, 2004). Another machine called the inside-outside bird washer consists of a series of probes that penetrate the inside of the body cavity in a spiral motion in which a high-pressure spray is used to remove debris from inside the carcass (Sams, 2001a). At the same time, the outside is being rinsed with a high-pressure spray. This method has been demonstrated to potentially reduce bacterial counts by over 50% and may also be used for in-line processing of fecal contaminated carcasses. Plants may use two final bird washers to meet the regulatory requirement for removing fecal contamination requirement (Bell & Weaver, 2002).

The final washing prior to chilling is designed to remove materials such as blood, membranes, and viscera fragments that may remain on the outside and inside of the carcass.

According to USDA Food Safety Inspection Services (FSIS) regulations, birds that have been tagged to have fecal spots removed that may have resulted from evisceration, must be washed thoroughly before the carcass can enter the post-process chill tank. The washing and chilling of broilers serves as the last major intervention step in mitigating the microbial load of the carcass. When carcass washing is performed properly, this process step is successful for cleaning carcasses and should result in a wholesome product.

The effectiveness of carcass washers for reducing *Campylobacter* spp. is dependent on water volume, water pressure, water temperature, and the available concentration of active ingredients, such as chlorine compounds, in the process water. The integration of chemical decontamination against human pathogens such as *Salmonella* and *Campylobacter* in the processing of chicken carcasses requires considerations regarding the chemistry, consumer acceptance and/or tolerance of the chemical(s) used, human health aspects, development of antimicrobial resistance, environmental impact, cost, microflora changes, sensory conformance, and domestic and import regulations on the use of specific chemicals (Axtell, Russell, & Berman, 2006; Bauermeister et al., 2008; Berrang et al., 2007; Kameyama et al., 2012; Riedel et al., 2009).

In processing of raw poultry and poultry products, monitoring the effects of decontamination steps are usually geared towards total aerobic bacteria, *E. coli*, and/or *Salmonella* (Berrang et al., 2007). However, only a few refer directly to the elimination of *Campylobacter* spp. (Berrang et al., 2007; Corry & Atabay, 2001; Riedel et al., 2009; Zhao & Doyle, 2006). Reidel et al. evaluated the effectiveness of 11 chemical compounds in their effectiveness to reduce *C. jejuni* on chicken skin and meat samples dipped in chemical solutions (Riedel et al., 2009). The compounds were prepared in sterile water immediately before the experiment and

the study was a benchtop model, using artificially contaminated chicken that was inoculated onto frozen *Campylobacter*-negative chickens without giblets. The reductions seen with organic acids (formic acid, 1.57 log; lactic acid, 1.69 log), and the fatty acid salt (capric acid sodium salt, 1.78 log) were statistically similar. The plant extract (grapefruit seed extract, 3.05 log) and bis-bis-guanide compound (chlorhexidine diacetate salt hydrate, 3.81 log) showed better reduction potential. Quaternary compounds (Cetylpyridinium chloride, >4.2 log; benzalkonium chloride, >4.2 log) demonstrated the best reduction potential for *Campylobacter* in this study. As a comparison, sterile water achieved a 0.95 log reduction.

A comprehensive study that monitored industry-used methods across the contiguous U.S. by Berrang et al. (Berrang et al., 2007), which spanned across 13 states and the samplings spaced to represent all four seasons, provides insight into the use of the most common on-line processing chemicals used to reduce *Campylobacter* on broiler carcasses at rehang and post-chill. Hypochlorous acid (HAS) resulted in a 1.47 ± 0.26 log reduction, peroxyacetic acid (PA) resulted in 1.79 ± 0.17 log reduction, acidified sodium chloride (ASC) resulted in a 2.33 ± 0.14 log reduction, food-grade acids (FGA) resulted in 1.32 ± 0.15 log reduction, trisodium phosphate (TSP) resulted in a 2.97 ± 0.07 log reduction, chlorine dioxide (CD) resulted in a 2.41 ± 0.24 log reduction, and treatment processing without chemicals resulted in a 1.78 ± 0.15 log reduction. In all, a mean log reduction of 2.23 ± 0.06 was seen in this stage of broiler processing. In a study of antimicrobial chemicals to compare their effectiveness of eliminating biofilms and pathogens within biofilms, Trachoo and Frank (Trachoo & Frank, 2002) reported that *Campylobacter jejuni* in biofilms was susceptible to chlorine, quaternary ammonium compounds (QAC), peracetic acid (PAA) and a PAA/peroctanoic acid mixture (POA) mixture at 50 and 200 ppm for 45 seconds (Trachoo & Frank,

2002). However, there are mixed reviews on their overall effectiveness against biofilms.

Chlorine Usage

The most commonly used disinfectant in the poultry industry in the United States is chlorine in the form of sodium hypochlorite (NaOH), calcium hypochlorite tablets, and chlorine gas (Brizio et al., 2015). The U.S. Dept. of Agriculture (USDA) Food Safety and Inspection Service (FSIS) allows for the addition of chlorine in processing waters at concentrations up to and including 50 mg/l (ppm) in carcass wash applications and chiller make-up water. In addition, FSIS requires the application of chlorinated water containing a minimum of 20 ppm available chlorine on all surfaces of carcasses when the inner surfaces have been reprocessed due to carcass contamination other than solely by trimming.

Chlorine has been demonstrated to be effective in the control and reduction of *Campylobacter*. The use of chlorine is the predominant disinfectant used for eliminating *Campylobacter* biofilms, along with quaternary ammonium compounds (QAC), peracetic acids and its mixtures (PAA+) (Bauermeister et al., 2008; Trachoo & Frank, 2002). The effectiveness of chlorine, however, is reported to be diminished when bacteria are attached to organic matter, such as chicken carcasses, fats on processing equipment, and debris and fecal matter on transport cages (Bashor et al., 2004; Bucher et al., 2015).

When chlorine is used in poultry processing facilities to disinfect the surfaces of equipment, carcasses, or chiller systems, it encounters a very high organic load and generally loses its microbiocidal properties and does not serve as a disinfectant (Brizio et al., 2015; Eifert, Arritt III, & Kang, 2006). Poultry process waters can have extremely high levels of total organic carbon and a correspondingly high chemical oxygen demand. Any free chlorine added to these high-demand

waters is consumed rapidly, thereby becoming consumed prior to disinfection. If the chemical oxygen demand in these waters is not satisfied, then a true free-chlorine residual cannot be established. The common poultry chill tank can have a chlorine demand of 1,000 to 2,000 ppm that cannot be overcome by 50 ppm chlorine in the makeup of the water.

Bashor et al. (Bashor et al., 2004) states that although a reduction in *Campylobacter* contamination on carcasses may drop due to washing; the reduction is neither consistent nor significant. In one study, the efficacy of inside-outside carcass washers and homemade cabinet washers was evaluated. These systems used 9.73 liters of water per carcass and between 25 – 35 ppm, total chlorine. Although *Campylobacter* concentration was reduced by 0.5 log CFU/ml by the washers, the average concentration after the washer was still 4.3 CFU/ml of carcass rinse (Bashor et al., 2004). Oyarzabal et al. (Oyarzabal et al., 2004) showed that a carcass washer produced modest reductions of *Campylobacter* contamination (between 0.3 and 0.7 log CFU/ml) and left the carcasses with a high incidence (above 90%) of *Campylobacter*.

Prohibition of Specific Antimicrobial Agents in EU Poultry Production

Current EU regulations prohibit the use of chlorine and chlorinated water for chilling, and in Nordic countries, only potable water without additives is used for the chilling process; and products that use the chlorination process is barred for import due to the theoretical link to carcinogenesis (Axtell et al., 2006; Lindblad et al., 2006; Northcutt, 2001). Since 1997, the European Union (EU) has prohibited the import of U.S. poultry products processed with certain pathogen reduction treatments (PRTs) (Johnson, 2015). PRTs are antimicrobial agents used in immersion and rinsing of poultry processing, which includes chlorine dioxide, acidified sodium chlorite, trisodium phosphate, and peroxyacids – that have been approved by the U.S. Department

of Agriculture (USDA 1999a, 1999b, 1999c, 1999d, 2000, 2003, 2004). PRTs are also judged to be safe by European Food Safety Authority (EFSA) (Johnson, 2015). The EU, however, prohibits the use of PRTs and the importation of poultry treated with PRTs, and this has been a source of contention between the two entities. The United States argues the EU ban is a trade barrier that is not based on scientific evidence showing that such treatments are harmful. The EU interests view the use of PRTs as an overreliance on the U.S. standards to meet microbiological wholesomeness and that stronger sanitary practices during production and processing are more appropriate for pathogen control (Johnson, 2015).

J. Post-Wash Chilling

The two most commonly used methods of post-wash chilling poultry throughout the U.S., Europe, and New Zealand/Australia are water and air chilling, (Lindblad et al., 2006; Sams, 2001a). The three major systems currently used for chilling in the European Union (EU) are cold water counterflow immersion chilling, such as that used in the United States sans chlorine usage, cold dry air chilling, and evaporative chilling, which is a combination of air chilling and water spray chilling. The prechiller has some washing and chilling effects on the carcass. Prior to entering the pre-chiller, the carcass temperature is about 38°C, and the skin lipids are still quite fluid (Conner et al., 2001). After the pre-chilling process, the temperature of the carcasses drops to approximately 30°C when they enter the series of chilling process described in sections below (Northcutt et al., 2003).

The primary objective of rapid chilling of poultry is reduction of microbial concentration and retard microbial growth to a level that will optimize both the quality and shelf life for

marketing, where most poultry carcasses are bacterially contaminated on the surfaces of the carcass, both inside and out (Oyarzabal, 2005; Oyarzabal et al., 2004; Sams, 2001a). Post-wash chilling has an effect on the number of common foodborne pathogens found on poultry. A carcass temperature of ~ 4°C is ideally achieved after evisceration, generally 1 – 2 hours postmortem. Current U.S. regulations mandate that carcasses must be chilled to acceptable (4°C or less) temperature within four hours after slaughter for chickens and eight hours for turkeys (Bell & Weaver, 2002; Sams, 2001a). Although post-wash chilling is likely to reduce the level of *Campylobacter* on carcasses (either air chilling or immersion chilling), the impact of decrease is variable on the odds of contamination depending on the chilling process (Bucher et al., 2015). Moreover, in the systemic review meta-analysis conducted by Bucher et al. (Bucher et al., 2015) the results were insignificant with uninformative confidence intervals for the effect chilling interventions have on the prevalence of broilers contaminated with *Campylobacter*.

Immersion Chilling

Most poultry processing plants operating in the United States and Australia use spin chillers (i.e. water immersion chillers). Long flow-through tanks containing water baths of air-agitated ice slush that are usually chlorinated, ensure rapid cooling of poultry carcasses (Berrang et al., 2007; Berrang & Dickens, 2000; Conner et al., 2001; Richardson et al., 2009). Carcasses are removed from the shackle and continually pushed through the water channels by revolving paddles or an Archimedes screw-type system. A pre-chiller, the first stage of the system, has water at about 7 – 12°C and lasts for ~10 to 15 minutes. In the water immersion chiller, the water/ice slush temperature is approximately 4°C at the entrance and ~1°C at the exit, where the total process takes about 45 – 60 minutes (Northcutt et al., 2003). Many immersion chillers are countercurrent,

where clean water is pumped into the chiller near the exit end to maximize heat exchange from the carcasses to water, and maximize cleanliness of the carcasses (Conner et al., 2001; Northcutt et al., 2003). Additionally, to maximize chilling rates of the carcass, air is injected into the bottom of the individual chill tank. Bubbles are created as the air moves toward the water surface and this agitates the water and prevents the formation of thermal layering at the product surface (Northcutt et al., 2003). This prevents the water surface from reaching equilibrium with the carcass temperature and insulating the carcass, which would limit further heat exchange from the carcass to the water. The USDA requires the chiller overflow rate be maintained at one-half gallon of water per carcass chilled to mitigate microbial buildup in the chiller water (Allen et al., 2000). During the time in the chill tanks, as the carcass temperature rapidly drops, the tissue lipids solidify and seal in the water absorbed in the pre-chiller (adding to the carcass weight, and possibly attributing to the internalization of *Campylobacter* on internal tissues).

Free chlorine is maintained in chilling water to reduce cross contamination, yet this practice is not always effective in immersion chillers (Center for Veterinary Medicine, 2000; FAO-WHO, 2009c). Chlorine levels up to 50 ppm at pH 6 may provide adequate control of cross-contamination during chilling (Bashor et al., 2004). However, a potential exists for product flavor and texture changes, as well as increases in biological oxygen demand (BODs) in plant effluent when acids are used to reduce the pH level of chilling water (Bell & Weaver, 2002).

Air-chilling

Evaporative air chilling is commonly used in the European Union (Conner et al., 2001; Lindblad et al., 2006). Air chilling involves passing the shackle lines of carcasses through large rooms of circulating cold air (-7 to 2°C) and maintaining an ambient temperature of 2 – 4°C for 1 to

3 hours until the optimum deep muscle temperature ($<4.4^{\circ}\text{C}$) is achieved (Conner et al., 2001; Lindblad et al., 2006). This can be also done with the birds on racks, but the more common and efficient method is to air-chill carcasses on shackles. To enhance the cooling effects of water, the product can be sprayed with water, which absorbs heat as it evaporates. The relative humidity in the room may also be adjusted to maximize the air's ability to absorb heat from the carcass, where the air's ability to evaporate the surface water for their skin correlates to the drying effect of this chilling method (Lindblad et al., 2006). The dried skin rehydrates, and the appearance usually exhibits a slight weight loss during chilling. This contrasts with the weight gains resulting from water chilling. Such yield differences contribute to restrictions in international trade between countries using the two different chilling methods (Bucher et al., 2015; Johnson, 2015).

However, data on the impact of air chilling on the dissemination and level of bacteria found on the surface of the carcass is still lacking (Bucher et al., 2015; Conner et al., 2001; Guerin et al., 2010; Sanchez, Brashears, & McKee, 1999). Air-chilled carcasses have been reported to contain higher aerobic plate counts and higher *coliform* counts than carcasses that were chilled by water immersion, although in one study, the incidence of pathogenic bacteria was lower in comparison to water immersion (33.3% vs. 56.6%) (Sanchez et al., 1999).

Air-chilling and immersion chilling have been reported to impart different effects on the microbial quality of a poultry carcass (Lindblad et al., 2006). Water chilling washes bacteria from the exterior surface and results in carcasses with a generally lower total bacterial load. However, the extensive bird-to-bird contact in the chill-tank(s) result, in theory, a greater potential for spreading bacteria between carcasses in the water chiller than when air-chilling is used (which is more prevalent in EU countries), where the carcasses are more isolated from each other (Sams,

2001a). This argument for the higher potential for pathogenic cross-contamination during water chilling is another major factor in trade restrictions between countries using the two different chilling methods.

Reduction of Bacterial Counts due to Process Chilling

Most studies indicate that bacterial numbers diminish after the chilling process of carcasses (Berrang et al., 2007; Berrang, Bailey et al., 2008; Berrang, Buhr, et al., 2000; Berrang et al., 2003; Bucher et al., 2015). However, there is also a risk of cross-contamination during the chilling process (Baumgartner & Felleisen, 2011; Chaisowwong et al., 2012; Guerin et al., 2010; Hazards, 2010). Immersion chill tanks may represent an avenue for cross-contamination (Conner et al., 2001; Whyte, Collins, McGill, & Monahan, 2002). Carcasses that do not harbor bacteria may enter the chill tank and become contaminated, conversely, heavily contaminated bacteria may see a marked decrease in concentration after this process as well. Detrimentially, large tanks that contain many carcasses can attribute to the cross-contamination. Bacteria may be transferred off the surface of the contaminated carcass to other carcasses in the water tank.

Allen et al. (Allen et al., 2000) reported that chilling in chlorinated water resulted in decreased levels of aerobic bacteria, *coliforms*, and *Pseudomonas* spp. And, that cold dry air chilling did not affect surface contamination, but it did slightly reduce bacterial numbers in the body cavity. Guerin et al. (Guerin et al., 2010) reviewed two studies that studied the effects of *Campylobacter* concentration and prevalence exposed to air chilling. The reported change in concentration had a minimum decrease of log 0.2 CFU/g and a maximum decrease of log 0.4 CFU/g. The change in concentration pre- and post- water immersion chilling concentrations of *Campylobacter* had a minimum decrease of log 0.8 CFU/mL and a maximum decrease of 1.7

CFU/mL, however, water spray chilling was associated with increased bacterial counts in the body cavity post-chill. In a systemic review meta-analysis conducted by Bucher et al. (Bucher et al., 2015), the team summarized a mean predicted decrease of 0.74 log / carcass (95% CI 0.32 – 1.17), which was a log lower than that predicted for immersion chilling (1.74 log / carcass, (95% CI 1.32 – 2.16).

Prevalence of Campylobacter Post-chill

Continuous immersion chilling has shown to reduce the microbial load on the carcasses. Although there can be an overall decrease in the concentration of *Campylobacter* on chilled carcasses, the overall number of carcasses that are *Campylobacter*-positive can increase after the chill-tank process (Berrang, Meinersmann, et al., 2008; Bucher et al., 2015; Kameyama et al., 2012). In the study by Bahrndorff et al. (Bahrndorff et al., 2015), data from two independent studies were combined and examined for the relationship between *Campylobacter* slaughter group prevalence pre-scalding or pre-chill and the percentage of *Campylobacter* positive carcasses post-chill. The prevalence of contaminated carcasses post-chill increased as intestinal colonization of the birds increased. Only 2% of the carcasses that originated from slaughter groups testing negative pre-scald were positive for *Campylobacter* spp. post-chill. About half of the positive carcasses originated with negative cloacal samples pre-scald, but had positive neck skin samples pre-chill, which indicated that they had been contaminated during processing. A comprehensive review was presented by Guerin et al. (Guerin et al., 2010) for the influence of carcass chilling on the prevalence of *Campylobacter* by comparing results from post-chill samples to samples collected earlier in the processing chain. They reported that of the 18 applicable studies, 10 showed an overall decrease in prevalence, 7 showed an overall increase, and 1 study showed no

change. Of the 7 applicable studies reviewed that used water chill immersion, the change in prevalence ranged from a decrease of 100% to an increase of 26.6%. The increase in prevalence after washing may be attributed to the possibility that water from the final carcass wash could trap bacteria within skin pockets or the abdominal cavity (Wempe, Genigeorgis, Farver, & Yusufu, 1983).

The prevalence of *Campylobacter* on broilers that undergo air chilling is an uncertainty. *Campylobacter* cell death during air chilling may be the result of desiccation of the carcass surface (Berrang, Meinersmann, et al., 2008; Sams, 2001a). A systemic review meta-analysis by Bucher et al. (Bucher et al., 2015) reported that the process did not reduce the prevalence of *Campylobacter* on broiler carcasses, noting that the studies that contributed to the data gathered had conflicting results. The odds of contamination following air chilling could not be predicted in the meta-analysis.

K. *Campylobacter* Survival During In-Plant Storage and Transport to Retail

Post processing, broilers are packed and held in storage until they are ready for transport. Temperature must be maintained below 40°F (5°C) for product in the secondary processing and holding areas and while products is being transported (Bell & Weaver, 2002; Sams, 2001a). Fresh (refrigerated) chickens are stored under chilling conditions and products intended to be marketed frozen are maintained under the specified condition.

Level of Contamination Seen in Retail

Even with the numerous processing steps in preparation of the broilers for retail, the prevalence and, oftentimes, concentration seen in retail products remain high for *Campylobacter*

spp., especially for those products that are stored in refrigeration conditions. Most retail contamination studies concerning *Campylobacter* on broilers focus on fresh products (those kept in refrigeration conditions) more so than frozen products. Moreover, there are few recent reports on the prevalence of *Campylobacter* in the retail setting in the U.S. (Feifei et al., 2009; Williams & Oyarzabal, 2012), and among those studies, even fewer discuss boneless, skinless portioned products (Williams & Oyarzabal, 2012).

The prevalence of *Campylobacter*-positive broilers at the retail stage among industrialized countries around the world is reported to be about 40% - 80% of all poultry meat, regardless of storage condition (Harrison, Griffith, Tennant, & Peters, 2001; Meldrum, Smith, & Wilson, 2006; Suzuki & Yamamoto, 2009). Luber et al. (Luber & Bartelt, 2007) showed prevalence of *Campylobacter* on 87% of retail chicken breast, with a mean count of 1.9×10^3 CFU/fillet for Germany. Harrison, et al. (Harrison 2001) observed an overall contamination rate of 72% of chicken breast pieces (with skin), with a rise to 82% when accounting for only supermarkets at the time of retail purchase in the United Kingdom.

In a survey on the contamination rates of fresh (refrigerated) retail broilers contaminated with *Campylobacter* across the United Kingdom (February 2014 – February 2015) (U.K. FSA, 2016), it was found that 73.2% of skin samples were positive for *Campylobacter*. Overall, 19.7% of the chickens tested were found to contain *Campylobacter* at a level above 1000 CFU/g. In an updated survey of this project, which spanned from August 2016 to July 2017, with a sample size of 3,980 chickens, it was reported that 54.0% of chickens sampled were positive for *Campylobacter*, with 6.5% of all samples contaminated with high levels of *Campylobacter* (>1000 CFU/g) (Agency, 2017). Both figures showed significant decreases from year one of the survey.

The prevalence of *Campylobacter* in raw broiler products in the U.S. is in line as that reported in other developed countries. Regionally, in a one-year study conducted in Baton Rouge, Louisiana from October 2006 to September 2007, it was reported that the overall prevalence of *Campylobacter* spp. on retail whole chickens was 43.3% (Feifei et al., 2009). There was no statistical significance between conventional and organically produced broilers. In the seven supermarket chains surveyed, *Campylobacter* prevalence rates ranged from 19.6% to 87.5%.

Other studies throughout industrialized countries report contamination rates similar to those found in the U.S. Jorgensen et al. (Jorgensen et al., 2002) survey showed that 83% of the chickens sampled in England from 1999 to 2000 were positive for *Campylobacter*. This pathogen was isolated through enrichment from 56% of the samples representing both the inside and outside of the packaging. In that survey, 20% of the retail chickens carried *Campylobacter* at numbers exceeding 10^5 CFU per carcass. *Campylobacter* isolates were comprised of *C. jejuni* (98%) and *C. coli* (2%) and 98 different sero/phagetypes of the isolates were identified. They also noted that the levels of *Campylobacter* spp. in frozen chickens were lower than in chilled chicken. Four out of 26 frozen samples were positive for *Campylobacter* by direct plating, where the level of contamination obtained ranged from 2.8 – 4.4 log₁₀ CFU per chicken.

For most applicable studies reviewed, there seems to be a distinct disparity between fresh and frozen products at retail. In an early study conducted by Stern et al. (Stern, Green, Thacker, Krout, & Chiu, 1984), it was reported that the prevalence of *Campylobacter* on fresh (refrigerated) meats were about 5-fold higher than those of frozen meat. Baumgartner et al. (Baumgartner & Felleisen, 2011) reported that in a survey conducted on retail broiler chickens seen in Switzerland, 53.9% of refrigerated chicken meats (54.9% domestic and 52.2% imported) and 20% of frozen

(28.4% domestic and 14.0% of imported) were positive for *Campylobacter*. For frozen meats, the contamination rates for meats produced domestically were significantly higher than the rates in imported products ($P < 0.001$). This may be attributed to the extended time in deep frozen storage during transport, but this is not discussed in the paper. Considering the total number of carcasses sampled per storage condition, the distribution surveyed in the level of *Campylobacter* contamination were as follows: Refrigeration – 36.0% had concentrations < 10 CFU/g and 1.6% had levels greater than 1000 CFU/g; Frozen – 18.0% had concentrations < 10 CFU/g and no samples had levels greater than 1000 CFU/g. In the study, it was also observed that a combination of skinning and deep-freezing seemed to be highly effective to reduce the *Campylobacter* load.

However, not all studies agree that frozen chickens at retail show a significant decrease in concentration and prevalence of *Campylobacter* to its refrigerated counterpart. In the study performed by Meldrum, et al in the U.K. during 2002 (Meldrum, Tucker, & Edwards, 2004), the percentage of positive samples for *Campylobacter* between fresh ($n=553$, 75% of samples) and frozen ($n=186$, 25% of samples) whole chickens was not significantly different (fresh chickens- 70.2% positive and frozen chickens 72.6% positive).

A study conducted by Williams and Oyarzabal (Williams & Oyarzabal, 2012) sampled a total of 755 skinless boneless retail broiler meat packages (in 1 -2 lb. tray packs) from various sections in Alabama during 2005 through 2011. The average prevalence of *Campylobacter* was 41%, with no statistical difference in prevalence during the seven years of the survey. No distinction was made on storage conditions (refrigerated vs. frozen). It had a contamination rate 39.4% in this study (119 of 302 samples throughout the seven-year period).

Although thermophilic *Campylobacter* is generally regarded as incapable of growth under

normal refrigerated storage conditions, it has been shown from the some studies that they can survive and remain viable for extended periods of time at low temperatures (Meldrum et al., 2004; Sampers et al., 2010). If the effects of chilling and freezing are diminished as suggested (Meldrum et al., 2004), the need for emphasis in proper handling of chicken and raw poultry products, both in home and outside is amplified even greater. Moreover, the importance of implementing effective hazard analysis critical control point (HACCP) programs within commercial processing should also be emphasized even more. The ID₅₀ for humans is estimated to be approximately 500-900 cells (Black et al., 1988; Medema et al., 1996a, 1996b). The potential for human exposure to the pathogen through cross-contamination, poor hygiene, or undercooking in the kitchen is high (Cogan, Bloomfield, & Humphrey, 2002).

Effects of Refrigeration on Survival

Despite the fastidious requirements for growth and apparent survival of *Campylobacter*, the species is maintained remarkably well under refrigeration conditions (Alter & Scherer, 2006; Percival & Williams, 2014). *Campylobacter* spp. survive better at refrigeration temperatures (~4°C) than in ambient temperature (~20 – 25°C) as a function of temperature, pH, and osmotic stress (Alter & Scherer, 2006; Corry & Atabay, 2001; D'Souza et al., 2004; Kelana & Griffith, 2003; Murphy et al., 2006; Percival & Williams, 2014). In surface water as well as on the surface of poultry carcasses and parts, survival is limited to a few days under ambient temperature but under refrigeration conditions, survival is enhanced by several weeks (Buswell et al., 1998; Chan et al., 2001; Percival & Williams, 2014). Kelana and Griffith (Kelana & Griffith, 2003) observed reductions of <1, ~7, and >8 log cycles for bioluminescent *C. jejuni* in broth held at 4, 22, and 30°C for 7 days, respectively, with survival at 22°C being significantly better than at 30°C. And, on finished poultry

products, *Campylobacter* spp. exhibited about a 2.5 – 5 log CFU reduction from (artificially) inoculated initial load when held at ambient room temperature (D'Souza et al., 2004; Murphy et al., 2006).

Refrigeration storage at low temperatures (~4°C) for 3 – 7 days resulted in a reduction of viable *Campylobacter* cell count of 0.34 – 0.81 log CFU in ground chicken and slightly less reduction was seen (0.34 – 0.63 log CFU) for chicken skin artificially inoculated with an initial dose of 8 log CFU (Bhaduri & Cottrell, 2004). After 17 days in refrigeration, the initial load of *Campylobacter* spp. was generally reduced by about 1 – 2 log unit/g. The skin of poultry provides an appropriate micro-environment and serves as a protective factor in the survival of the bacterium. Dykes and Moorhead (Dykes & Moorehead, 2001) attributed the subcutaneous fat layer in protecting the pathogen on the skin, where the fat may act as an insulator. The liquid surrounding the poultry matrix also has been linked to protecting *Campylobacter* spp. from refrigeration and freezing temperatures. Other factors such as oxygen scavengers or different inorganic salts may be responsible for protective effects on the survival of *Campylobacter* in chicken juice and on chicken carcasses (Bhaduri & Cottrell, 2004; Dykes & Moorehead, 2001).

Effects of Freezing on Pathogen Survival

The freezing of food is a traditional method to prevent the growth of or to reduce the number of spoilage and pathogenic bacteria. In comparison to other countries, the United States underutilizes the freezing of poultry for storage. In Nordic countries such as Iceland, Norway, and Denmark, the carcasses of *Campylobacter*-positive flocks are introduced to freezing in processing to further reduce the risk of human campylobacteriosis (Al-Sakkaf & Jones, 2012; Baumgartner & Felleisen, 2011; Sandberg et al., 2005). This method has proven to be effective when processing of

known contaminated flocks are diverted to be finished as frozen carcasses (Sandberg et al., 2005; Stern & Robach, 2003). Deep freezing of broilers to reduce of campylobacteriosis has proven to be a valid risk management measure. Baumgartner et al. (Baumgartner & Felleisen, 2011) reported that prior to 1996, all chicken meat sold in Iceland was regulated to be sold frozen; after abrogation of this requirement, the incidence of campylobacteriosis increased significantly within three years.

During freezing, depending on the cooling velocity, ice nucleation in the cytoplasm of the living cells and cell desiccation result in cell injury and death, although *Campylobacter* spp. can still be isolated from frozen poultry meat and poultry meat products (Alter & Scherer, 2006; Baumgartner & Felleisen, 2011; Boysen & Rosenquist, 2009; Georgsson et al., 2006; Kudra et al., 2012). Freeze-thawing cycles significantly reduce the survival of the pathogen but *C. jejuni* remains viable for multiple freeze-thaw cycles, regardless if the meat product is frozen at -70°C or -20°C. Oxidative damage of the bacterium has been implicated as a mechanism that contributes to freeze-thaw injury due to the probable oxidative burst during thawing from extreme temperatures (Murphy et al., 2006).

With the implementation of freezing as an intervention step in controlling the number of *Campylobacter* on and in food products, the ability of different strains of *Campylobacter* to survive freezing has become extremely relevant for food safety. Therefore, information on how and where *Campylobacter* cells survive prior to raw chicken preparation – adhering to the skin and meat or suspended in the chicken juice – is of significant importance (Birk et al., 2006). Although freezing may be a viable step in mitigating the level of contamination of *Campylobacter* spp. in chicken products, a fraction of the *C. jejuni* population does survive the process or are sub-lethally

injured (Georgsson et al., 2006; I. Sampers et al., 2010).

Studies by Zhao et al. (Zhao et al., 2003) and Moorehead et al. (Moorhead & Dykes, 2002) reported that short-term storage of poultry at freezing temperatures (-18 to -20°C) reduced the number of *Campylobacter* cells markedly. The survival of *C. jejuni* on chicken wings and beef trimmings during freezing have reported reductions of approximately 1.2 and 1.5- log CFU/g, respectively, after 24 hours at freezing temperatures. Other experiments with various meat matrices have demonstrated that detectible reductions of about 1 to 3 logs CFU is manifest within the first few days of frozen storage and maximum levels of reduction are generally obtained within the first two weeks of freezing, followed by a relatively constant viability thereafter (Birk et al., 2006; Moorhead & Dykes, 2002; I. Sampers et al., 2010; Sandberg et al., 2005).

Sampers et al. (Sampers et al., 2010) demonstrated that although freezing affected the prevalence of *Campylobacter* spp. in chicken meat preparations with naturally contaminated *Campylobacter*, the reduction of concentration levels in samples containing frozen skin was not significant in their study. The pathogen could still be detected qualitatively (per 0.1 g) after 84 days. Other studies have addressed the effects of prolonged storage of broilers and broiler parts on the survival of *Campylobacter* on the product, with varying reduction levels being reported, although most report a threshold reducing level seen in extended freezing. During a period of 32 days at freezing temperatures (-18°C), Birk et al. (Birk et al., 2006) tested two model systems that simulated either the chicken skin surface or the “drip fluid” in an around the broiler carcass, which could be a surrogate for environmental contamination due to liquid from the contaminated carcass. The survival rate from the drip fluid model for the strains tested, was ~65% of that of the chicken skin surface model, both demonstrating a rapid decrease in recovery from day 0 to 1, a

moderate decrease within the first week and then a gradual decline over the remaining 32 days with a maximum reduction of approximately 3.5 log (N/N₀). There were some variations in the rate of reductions with the serotypes tested, with a significant difference observed between the survival of *C. jejuni* NCTC 11168 in chicken juice and Brain-Heart Infusion Broth (BHIB) with the liquid model showing about a 1.5 log decrease over the 32 days tested and the skin model exhibiting approximately a 3.5 log decrease during the same time period.

Ritz et al. (Ritz et al., 2007) noted that the reduction of *Campylobacter* in -20°C on the skin, below the skin, and on the muscles (all artificially inoculated with 10⁸ CFU/cm stationary phase cells of *C. jejuni*) was approximately 0.9 – 3.2 log₁₀ when exposed for 5 weeks. The greatest reduction occurred in the first two weeks, and further freezing yielded little additional effect after two weeks. The dynamics of *Campylobacter* reduction due to freezing was similar in other studies. Sandberg et al. (Sandberg et al., 2005) noted that there was an overall 2-log reduction from storage in freezing temperatures after three weeks and only a marginal extra effect was observed when freezing was extended to five weeks using naturally infected carcasses. Additionally, in this study, 80% of samples remained positive for *Campylobacter* using qualitative culturing, even after 120 days of storage at -20°C.

L. Packaging Implications for Microbial Food Safety

The package seal is a significant control point for the prevention of contamination of packaged poultry meat. Although some flexible materials used in packaging of poultry are designed to allow some gas exchange, these packages do not allow microorganisms to penetrate their structure. The seal is a frequent failure point when contamination of the product does occur,

and the packaging material and integrity of the sealing process plays an important role in preventing contamination and purge from leaking into retail cases and surrounding packs. Almost 90% of retail chicken parts are packaged directly into consumer portions using highly oxygen-permeable polystyrene foam trays with a high-oxygen permeable PVC or polymer-based, stretch film wrap (Sams, 2001b).

Contamination of Exterior Retail Packaging

Quality packaging is vital for efficient storage, distribution, and retail display of meat and poultry products with the intention of self-service to the consumer at retail. Foodborne pathogens such as *Campylobacter* spp. as well as *Salmonella* have demonstrated survivability and viability on the interior as well as the exterior of raw chicken packaging purchased from retail stores (U.K. FSA, 2016; Anonymous, 2011; Burgess et al., 2005; Harrison et al., 2001; Kenawi, Abdel-Aal, & Abbas, 2007). The presence of human pathogens such as *Salmonella* and *Campylobacter* spp. on the outer packaging raises concern as consumers would not expect products to be contaminated on the outer packaging – thus, they would likely not do anything to avoid cross-contamination from this route (U.K. FSA, 2016; Jorgensen et al., 2002).

In a study conducted by Burgess et al. (Burgess et al., 2005) in the UK, 895 samplings of raw chicken carcasses and portions were examined for the presence of *Campylobacter* on the exterior surface of packages. Twenty-seven (3%) of the swabbed samples were positive for *Campylobacter* (6.5% for whole chickens and 2.3% for further processed chicken products, respectively.)

Enumerations were not performed for *Campylobacter* on the tested samples.

The results of this study were similar to a study conducted by Harrison, et al. (Harrison et al., 2001), also conducted in the UK, where they noted that among the 300 raw samples (whole

chicken, chicken breast with skin or chicken pieces) purchased and investigated throughout seven months, *Campylobacter* and *Salmonella* were isolated 68% and 29% of retail chicken, respectively. *Campylobacter* was isolated from 3% of external and 34% of whole packaging (internal and external). No breakdown was given regarding the various chicken products regarding package contamination. A similar study was conducted by Wong et al. (Wong et al., 2004) in New Zealand. In this study they found that of the 300 retail supermarket chicken packages sampled, the surfaces of 72 packs (24%) were externally contaminated with *C. jejuni*. Specifically, 10% of the packages that contained breast pieces were contaminated with *C. jejuni*, with concentrations as high as 46 MPN/pack. *Campylobacter*-positive surfaces on raw chicken packaging could be found on visibly leaky packs but also on those that exhibited very little leakage (Wong et al., 2004). The use of absorbent pads placed under the products on the retail package did not prevent leakage – providing plastic bags for holding packaged poultry and raw meat may reduce the risk of cross-contamination.

In an updated survey conducted by the UK Food Standards Agency (UK FSA) during February 2014 to March 2015, 6.7% of the outside of fresh retail chicken packaging was positive for *Campylobacter* (U.K. FSA, 2016). The percentage of chickens with levels greater than 1000 CFU/g of *Campylobacter*-positive samples peaked during June to August. Around 0.1% of the total samples (5 out of the 4,005 samples) harbored greater than 1000 CFU/swab on the exterior of the packaging. The data derived from these studies suggest that exterior contamination on the retail packs could be a source of cross-contamination, but the contribution of this potential source needs to be properly determined by the development of a risk assessment model that takes this into consideration (Anonymous, 2011 Burgess et al., 2005; Harrison et al., 2001; Wong et al.,

2004).

Campylobacter Survival in Modified Atmosphere Packaging (MAP)

Modified atmosphere packaging (MAP), including use of vacuum packaging and oxygen-permeable wraps, is used with greater frequency to package many fresh food products. Combined with low temperature storage, MAP affords the producer and retailer longer periods of shelf-life (Kenawi et al., 2007; Phillips, 1998). However, a potential problem arises in that with MAP packaging, lowered oxygen concentration that is inhibitory for spoilage microorganisms, creates a more favorable growth condition for microaerophilic bacteria such as *Campylobacter* spp. if these have contaminated the food product prior to packaging. *Campylobacter* spp. are thermophilic and do not multiply below 30°C but the survival rate of the organism varies in different atmospheres with survival rate increasing with CO₂ concentration (up to a point) in the environment (Dykes & Moorehead, 2001; Kenawi et al., 2007; Phillips, 1998). If *Campylobacter* spp. can survive MAP techniques for raw poultry, then the risk to public health can include the potential cross-contamination of the product in handling in the domestic kitchen environment to other products and the prepared meal. This is especially problematic due to the low dose of *Campylobacter* spp. required to cause infection and illness (Black et al., 1988; Jones et al., 2004; Medema et al., 1996b; Nauta et al., 2009; Ponce et al., 2000; Teunis & Havelaar, 2000).

Combining chilled storage (4°C) with MAP or vacuum packaging may have the potential to increase the safety of meats. In practice, MAP seems to have only a marginal effect on pathogen survival. Alter et al. (Alter & Scherer, 2006) reported that the magnitude of decrease in cells counts ranged from 0.5 – 4.2 log CFU/g within 25 days. However, Dykes and Moorehead (Dykes & Moorehead, 2001) noted no significant changes in the bacterial cell counts for either MAP or

vacuum packaging regimes over a 41-day storage period at -1°C.

For sampled products packaged using MAP, *Campylobacter* spp. was isolated from 71% of chicken samples, which is similar to that found with non-MAP packaged poultry products at retail (Atterbury, 2003; Frost, 2001; Gormley, 2007; Harrison 2001). In the study conducted by Phillips (Phillips, 1998), 80.0% (32/40) of chickens in MAP packages sampled by the 'use by date' were positive for *Campylobacter* spp. Moreover, *Campylobacter* spp. have been isolated from packaged mixed salads using the MAP process (Phillips, 1998). Combined with mishandling practices seen domestically with fresh poultry and poultry products, this may become especially problematic due to the low dosage level of the pathogen that can cause infection and illness. The prevalence and the consequent viable concentration of *Campylobacter* spp. in the Phillips study (Phillips, 1998), raises discussions in the potential for ready-made products to further contribute to incidences of campylobacteriosis. The majority of the viable concentration was below 80 CFU/g and the highest was reported at 170 CFU/g, which could result in an individual potentially consuming 8,000 to 17,000 organisms from a 100 g serving.

Part 3 – Exposure to *Campylobacter* Due to Poultry Preparation and Consumption

A. Foodborne Campylobacteriosis

The Centers for Disease Control and Prevention (CDC) (CDC, 2017a) estimates 1.4 million total cases of campylobacteriosis annually, with estimated attributable deaths of 76. In the study by Scallan, et al. (Scallan et al., 2011), which is heavily referenced when discussing the burdens of foodborne pathogens in the United States, 80% of all campylobacteriosis is attributed to a food source. Domestically acquired foodborne cases are estimated to be 845,024 with a 90% confidence interval (CI) of (337,031 – 1,611,083). Hospitalizations were estimated at 8,463 (4,300 – 15,227, 90% CI) and deaths at 76 (0 – 332, 90% CI), representing 0.1% (unadjusted) of the cases. The Scallan study updated the foodborne-disease attribution estimates by Mead et al, (Mead et al., 1999) which attributed 1.96 million cases of campylobacteriosis as foodborne (estimated 80% of total cases to foodborne), with a 0.102 hospitalization rate (10,539) and 0.0010 case fatality rate (99). Although there are many cases of campylobacteriosis on an annual basis, there are very few associated mortalities. The estimate for U.S.-based deaths due to campylobacteriosis was reported to be 0.05% (FAO-WHO, 2013), and Mead et al. (Mead et al., 1999) estimated the U.S annual deaths attributed to *Campylobacter* infection to be 0.005%. Denmark estimated mortality due to campylobacteriosis to be 0.004%.

Kirk et al. (Kirk et al., 2015) estimated the burden of global campylobacteriosis in 2010 as 166,175,078 (92,227,873 – 300,877,905 (median with 95% UI – uncertainty intervals)) illnesses, 37,604 (27,738 – 55,101) deaths, and 3,733,822 (2,857,037 – 5,273,652) disability-adjusted life years (DALY). Additionally, a median (with 95% UI) of 58% (44 - 69) was attributed to foodborne

illnesses. Accounting for this proportion, global foodborne campylobacteriosis (median with 95% UI) was attributed as 95,613,970 (51,731,379 – 177,239,714) illnesses, 21,374 (14,604 – 32,584) deaths, and 2,141,926 (1,535,985 – 3,137,980) disability-adjusted life years. These estimates included Guillain-Barré Syndrome cases and deaths. Within this reporting, the figures were further broken down by region (Africa, Americas, Eastern Mediterranean, Europe, South-East Asia, and Western Pacific) as well as by age group (<5 years and ≥5 years). In terms of DALY, *Campylobacter* ranked 5th in the estimates for pathogens acquired from contaminated food, behind *Salmonella*, non-typhoidal *Salmonella* Typhi, enteropathogenic *E. coli*, and Norovirus. In the United States, the annual cost of campylobacteriosis, as estimated by Batz, et al. (Batz, Hoffmann, & Morris, 2014) is at \$1.3 billion.

B. Detection and Reporting of *Campylobacter*-associated Illnesses

Health-outcome attribution typically relies on outbreak data and case-control studies. Source attribution data derived from outbreak surveillance provides the most definitive link to sources by default since an outbreak is, by definition, characterized by cases that share a causal pathway. U.S. foodborne illness outbreak data include food attribution information on cases but are known to underestimate illness (Hoffmann et al., 2007). Sporadic illnesses can be tracked by case-control studies – where they are used to identify variances between the exposure profiles of infection and a control population to determine the attributable fractions associated with exposures found to be statistically significant in a multivariable model (Cole et al., 2014).

Due to their requirements for growth in an environment, *Campylobacter* do not multiply in air or at room temperature. Food handling practices, although important to localized dose-

response between the pathogen and hosts, are less likely to result in the overwhelming spread of the organism from a contamination source (Frost, 2001). Just as important, the relatively long incubation period from infection to illness to signal the potential or probable outbreak from the pathogen is less likely to be recognized by the patient and correlated to the true source of infection.

Friedman, et al (Friedman et al., 2004) provide evidence that the pattern of association with food consumption is likely to vary for outbreak and sporadic cases. Much like in typical cases of sampling, associative connection between foods consumed and foodborne illness observed in case-control studies is often difficult because evidence is typically eaten or destroyed, and food consumption recall is notoriously unreliable (Batz et al., 2005; Hoffmann et al., 2007).

Outbreaks vs. Sporadic Illnesses

The majority of campylobacteriosis are sporadic individual infections, and a wide variety of animal species can harbor the pathogen in high numbers, which complicates tracing and attribution to the original source of infection. Percival and Williams (Percival & Williams, 2014) noted that it is likely that less than 1% of reported infections in the UK are part of known outbreaks, with most infections manifesting as single sporadic illnesses, in which the majority are not source attributed. This holds true for the United States – where only 0.5% of cases during 2004 – 2011 were reported to be outbreak cases using FoodNet (Ebel et al., 2016). The true incidence of foodborne disease is much higher than reported due to sporadic cases. The source of most infections is uncertain, but the authors point to foods as the most likely vehicle – apart from raw milk, the main source of *Campylobacter* exposure is through raw meat – especially poultry products. Risk factors for *Campylobacter* infections differ for outbreak cases and sporadic cases.

The majority of outbreak cases thus far have been attributed to raw milk consumption (Heuvelink et al., 2009; A. Lehner et al., 2000; Percival & Williams, 2014) or contaminated water (Epps et al., 2013a; Frederick & Huda, 2011; Kapperud et al., 2003), while consumption of poultry meat, especially chicken, or exposure to food cross-contaminated by contact with raw poultry makes up the primary risk factor for sporadic infections (Gras et al., 2012; Humphrey, 2001; Wagenaar, French, & Havelaar, 2013; Wagenaar, Mevius, & Havelaar, 2006).

While outbreak investigations provide a valuable insight into the epidemiology of community-acquired infections such as in instances of *Salmonella* enteritis and *E. coli* infection, the sources of sporadic infections are more difficult to trace (Domingues et al., 2012). The nature of *Campylobacter* spp. ensures that reported outbreaks of campylobacteriosis are less likely to be common than those caused by other bacterial pathogens such as *Salmonella* spp. and enteric *E. coli* (Wagenaar et al., 2013). Underreporting is compounded by the practice of investigators being possibly less diligent in the investigation of *Campylobacter* infection than other bacterial infections (Frost, 2001). Rooney et al. (Rooney et al., 2000) reported that while over 90% of local public health authorities investigated reports of *Salmonella* or *E. coli* O157:H7 infection, only 63% always followed up reports of *Campylobacter* infections. *Campylobacter* also scored lowest for follow-up of the 20 likely causes of sporadic food poisoning infections. Most cases of campylobacteriosis are designated as sporadic (Ebel et al., 2016; Hoffmann et al., 2017; Kaakoush, Castano-Rodriguez, et al., 2015). Therefore, source attribution based on outbreak investigation on human campylobacteriosis has limited value (Newell et al., 2017).

Attribution of Campylobacteriosis to Poultry Consumption

The tracking of transmission and attribution of sources of zoonotic pathogens is an integral part of public health. In recent years, methods of sourcing attributable factors of human pathogens have become more sophisticated, incorporating various data sources and laboratory techniques. In practice, the term “source” is used to reference any point across the transmission chain where exposure to the food or environment, as product or livestock, for instance, is identified (Newell et al., 2017).

Frost (Frost, 2001) describes a case-study in England and Wales during 1990 – 1991 which identifies the four main risk factors for sporadic *Campylobacter* infection as handling of raw chicken meat, association with pets with diarrhea, ingestion of untreated water, and consumption of raw or bird-pecked meat. Epidemiological evidence from case-control studies support that undercooked poultry consumption and cross-contamination from handling raw poultry products were among the top risk factors for human campylobacteriosis (Domingues et al., 2012; Neiman et al., 2003; Studahl & Andersson, 2000; Uyttendaele et al., 2006). The sudden decrease in campylobacteriosis seen in Belgium, when affected poultry products were pulled off the market due to the dioxin crisis, further strengthens the validity of this assumption (Newell et al., 2017; Vellinga & Van Loock, 2002).

Case-control studies alone cannot sufficiently attribute human cases to reservoirs (i.e. amplifying hosts) because they can only trace back the sources of human infections up to the point of exposure (e.g. food items consumed, contact with animals, etc.) that may not correspond to the original reservoirs because of, for instance, cross-contamination, or alternative transmission routes (Hazards, 2010, 2011). Up to the mid-2000s, Batz et al. (Batz et al., 2005) argued that case-

control studies of foodborne illness alone in the United States could not yet provide a nationally representative picture of food attribution for sporadic cases.

Along with case-control studies, another modern approach to source attribution is to track genotypically. The multi-locus sequence typing (MLST) approach depends on the genetic sequence of, usually, seven house-keeping genes. This technique was initially developed to investigate the evolutionary structure of biological populations, but is now regularly employed to track source attribution of zoonotic pathogens (Llarena, Taboada, & Rossi, 2017; Nielsen et al., 2010), and is considered the most common approach for tracking source attribution of human campylobacteriosis (Newell et al., 2017). Using mathematical models employing MLST, it was reported that chickens are the most common reservoir/source of *Campylobacter* infection, with attributions varying between 38 – 77% throughout Europe and Canada (Skarp et al., 2016). It is noteworthy that chickens attributed 64.5% of *Campylobacter* infection in Canada (Lévesque et al., 2013), and that chickens (consumption, cross-contamination, and to a lesser extent, insufficient cooking) represented the greatest risk/exposure for human campylobacteriosis, much like in the U.S. and other parts of the industrial world (Pintar et al., 2017). These agree with the MLST-based source attribution model developed by the European Food Safety Authority (EFSA), which estimated 50 – 80% of *Campylobacter* strains infecting humans originate from chickens (EFSA, 2010). In general, source attribution studies have vastly improved the understanding of the relative contributions by various sources to human infection.

The importance of chicken as a major reservoir of *Campylobacter*-associated infection is well established and undeniably linked. Human risk of campylobacteriosis from broiler chickens are reported to be more attributable from meat products with high concentrations of

Campylobacter spp. (Nauta, Jacobs-Reitsma, & Havelaar, 2007; Stern & Robach, 2003; Zweifel et al., 2015). With this known attribution, it has been suggested that human campylobacteriosis can be reduced significantly by targeting control strategies at products with relatively high concentrations of *Campylobacter* spp.

Attribution of Campylobacteriosis to Poultry Handling and Processing

Occupational exposure to chickens and chicken products potentially contaminated with *Campylobacter* can occur. A study conducted by de Perio et al. (de Perio et al., 2013) evaluated 29 cases of laboratory-diagnosed *Campylobacter* infection among workers at a poultry-processing plant during January 2008 – May 2011. Most (62%) cases occurred among workers in the live-hang area even though only 5% of the employees work in the live hang area, suggesting that these employees are disproportionately infected with *Campylobacter* spp. The majority (83%) of symptomatic workers had been working at the abattoir for less than a month.

For poultry workers, the highest risk for work-related *Campylobacter* infections occurs during the first month to a year of work, after which the workers develop immunity that may be protective against future symptomatic infection. However, as Ellström et al. (Ellström et al., 2014) noted, while immunity may develop after prolonged employment at a chicken processing facility, subsequent *Campylobacter* infections to these workers may manifest itself as asymptomatic occurrences. Moreover, a proportion of the asymptomatic infected individuals in the Ellström study only had very limited early exposure to *Campylobacter* – indicating that the true incidence of *Campylobacter*-associated infections may be underrepresented in the chicken processing plants, and in the overall population. The authors conclude that the findings of this study add to the evidence that chicken plays a significant role as the source of human *Campylobacter* infection, but

it also suggests that asymptomatic infections of the pathogen may occur even in individuals with only limited earlier exposure to *Campylobacter*.

Effects of Seasonality on Campylobacteriosis and Campylobacter Prevalence

An apparent correlation exists between the growth and dispersion of foodborne bacterial pathogens to seasonality, climate and the weather (D'Souza et al., 2004; Frost, 2001; Höök, Ekegren, Ericsson, Vasholm, & Danielsson-Tham, 2004; Jagai et al., 2006; Kafetzis et al., 2001; Phillips, 1995; Strachan et al., 2013). Of the two most prevalent bacterial-associated gastrointestinal agents, salmonellosis is most commonly reported in summer, whereas *Campylobacter* infections in humans peak during the spring to summer months in the United States (D'Souza et al., 2004; Frost, 2001; Kafetzis et al., 2001; Meldrum et al., 2004). The same seasonal pattern can be seen in the U.K., with a distinct peak in May and June (Phillips, 1995; Strachan et al., 2013). Most temperate countries exhibit a seasonal peak of infection in the spring while those with milder winters have peaks earlier in the year; countries in the tropical regions have little variation throughout the year (Strachan et al., 2013).

Industrialized countries share commonalities between the peaks of campylobacteriosis and peaks of prevalence and concentration seen on broilers, on the farm as well as in retail. This increase in colonization in broiler chicken flocks is corollary to the peak levels of campylobacteriosis seen in the U.S. (Höök et al., 2004; Stern & Pretanik, 2006). In studies performed by Meldrum et al. (Meldrum et al., 2004) and Baumgartner and Felleisen (Baumgartner & Felleisen, 2011), seasonality played an important role in the incidence of chicken contamination with *Campylobacter*. Peak contamination occurred during the months of June-August (~80-90%) and the lowest rates occurred in January, March, and December (~50%). The observation of

distinct seasonal variation in *Campylobacter* contamination indicates that it is imperative to carry out long-term surveys that cover multiple seasons or years.

A more recent study supports the previous studies conducted. In a UK survey conducted during 2014 to 2015 (U.K. FSA, 2016), the percentage of *Campylobacter*-positive samples in retail peaked from June to September, with these months reporting greater than 80% of the samples being positive for *Campylobacter* (LOD of 10 CFU/g). Although the data was collected over a single 12-month period, and thus does not provide insight into the assessment of this variation is attributable to usual seasonal variation, interventions by chicken suppliers, random fluctuations in weather, or other potential factors, the peak corresponds with other datasets available for seasonality for similar climate regions, including the United States.

In Swedish broiler production, similar seasonality in the prevalence of *Campylobacter* is observable, although the peak in slaughtered broilers appeared somewhat later than human illnesses (Höök et al., 2004). In Sweden, greater than 60% of the reported campylobacteriosis cases occurred from June to September, and peak in the months of July and August (Carrique-Mas et al., 2005; Höök et al., 2004). Average temperature differences between the U.S. and Nordic countries during the summer months may explain the staggering of peaks seen in the seasonality of campylobacteriosis in the respective countries. Nordic countries have shorter summers that occur later in the season.

Correlation between seasonal/temperature variance of campylobacteriosis and peaks of *Campylobacter* prevalence found in broiler chickens has been reported in other industrialized nations. Ishihara et al. (Ishihara et al., 2012) noted seasonal variation of *Campylobacter*-contaminated retail chicken products in Japan collected between April 2009 and March 2010, and

that a higher percentage of sampled chicken products at retail were found to be contaminated from June (39.3%) to November (83.3%). The highest number of human campylobacteriosis cases were reported in June during the same period with the number of cases trending down throughout the year thereafter. Skarp et al. (Skarp et al., 2016) reported that in New Zealand, seasonal peaks in human campylobacteriosis cases usually peaked during their warmer months. Higher isolation rates of *Campylobacter* from chickens were observed in the summertime vs. colder weather months.

The associative impact of seasonal peaks between poultry and human *Campylobacter* infection can be further established by the advent of sequence-based typing methods, especially with the more frequent use of multi-locus sequence typing (MLST) in source attribution (McCarthy et al., 2012; Strachan et al., 2013). MLST has enabled investigation into basic patterns of epidemiology such as temporal and spatial distribution. Even among different serotypes of the same bacterial pathogen, there are occurrences where variations of infection rates may be due to seasonality (Frost, 2001). This could be tied in with the strain/subtype variability as seen in campylobacteriosis infections attributed to poultry consumption.

The practicality in the use of this information to translate into improved food safety, however, has many hurdles. As Guerin et al. (Guerin et al., 2010) speculates, even with the distinction of peaks for *Campylobacter*-positive flocks and seasonal peaks seen in human infection, it is unclear how prevalence fluctuation could affect the logistics of a processing-level HACCP program, since the process is unlikely to be modified for different seasons. Considerations could be made for scheduled slaughter based on *Campylobacter* on-farm testing and more frequent testing and replacement of contaminated water in the plant, and possibly the farm, to help offset

potential seasonal variability of *Campylobacter* in the broiler population entering slaughter. The relationship between climatic temperature and bacterial pathogenesis is shown not just by *Campylobacter*, but other important foodborne pathogens such as *Shigella*, *Vibrio*, and *Salmonella* – this has implications for both the present and future food safety measurements and policy (D'Souza et al., 2004; Ethelberg et al., 2006; Friedman, Neimann, Wegener, & Tauxe, 2000; Höök et al., 2004; Kafetzis et al., 2001; Kassa, Harrington, & Bisesi, 2006; Willis & Murray, 1997). By association of seasonality and levels of illnesses, periods of hot temperatures can indicate to public health authorities that an increase in certain foodborne diseases could be imminent. Preventative public health action could be (and have been) instituted by warning and educating producers of food, managers of commercial food outlets, and the public about the added risks with hot weather, and the importance of due diligence to hygiene, correct storage, and temperature control during food production and preparation (D'Souza et al., 2004).

C. Consumer Food Handling and *Campylobacter* Cross-Contamination

Consumer preparation of food and personal and environmental hygiene of food handlers represent the last link in the food chain. Cross-contamination in the home is a major factor in the transmission of *Campylobacter* to foods that are ultimately ingested by the individual (Borrusso & Quinlan, 2017; Lopez et al., 2015; MacDonald et al., 2015; Todd et al., 2010). The domestic kitchen especially serves as a multifunctional setting in which there can be a constant transfer of pathogens into and out of the environment. Raw and undercooked foods from various sources may be contaminated with pathogenic microorganisms that can be transmitted by the food preparer's hands, cutting utensils, other foods, and food contact surfaces. The wide dissemination

of human pathogens throughout the kitchen via improper handling has been thoroughly discussed (Bolton, Meredith, Walsh, & McDowell, 2014; Boursillon & Riethmuller, 2007; Byrd-Bredbenner et al., 2013; Chardon & Swart, 2016; Fravallo et al., 2009; Gorman, Bloomfield, & Adley, 2002; Haysom & Sharp, 2004; Humphrey, 2001; Kennedy et al., 2011; Kosa et al., 2015; Nauta, 2010; van Asselt, de Jong, & Nauta, 2008; Verhoeff-Bakkenes et al., 2008).

There are many pitfalls to resolving cross-contamination in food preparation – although the “how” may be very self-evident. The “why” presents numerous confounding arguments. This section presents the impact of improper food handling and food hygiene in the domestic setting, the elements of the mechanisms of cross-contamination, the basics of consumer perception and risk communication in the form of education, and the role it plays in food risk assessment.

Contrary to consumer/public perception (Hoelzl et al., 2013; Redmond & Griffith, 2004b), foods prepared in the home are the primary source of disease cases stemming from foods, including outbreaks. Fifty to 87% of all reported foodborne disease outbreaks have been associated with the home (Fischer, Frewer, & Nauta, 2006; Gorman et al., 2002; Redmond & Griffith, 2004b; van Asselt et al., 2008), with a conservative attribution estimate of approximately 60% of all foodborne illness cases (Cogan et al., 2002; Humphrey, 2001). Sporadic illnesses, which make up a majority of illnesses in any given year, are primarily food-linked or environment-linked, such as water, that mostly go unreported but may constitute significant public health burdens and expenditures. Most of these sporadic foodborne illnesses can be traced to domestic preparation. Meredith et al. (Meredith, Lewis, & Haslum, 2001) reviewed that in the UK during the late 1990s, 99.8% of cases were sporadic or restricted to the immediate family. Among common food products, raw poultry possesses one of the greatest potential risk for cross-contamination (Lopez

et al., 2015; MacDonald et al., 2015; Meredith et al., 2001). In instances of sporadic illnesses, it is unlikely that a full investigation would be undertaken by public health officials. Over the years, this aggregates into uncertainty of the true number of incidences in each timeframe and area or the causes and prevention of foodborne illnesses originating in the domestic environment.

Chicken handling, meal preparation, and consumption are major risk factors for *Campylobacter*-associated illnesses in developed countries such as the U.K., Belgium, Denmark, Australia, and the United States. Previous risk attributions have sourced these interactions to account for ~40% of all *Campylobacter* enteritis in adults (Friedman et al., 2004; Neimann et al., 2003; Stafford et al., 2007; Tam et al., 2009; Vellinga & Van Loock, 2002). Recent estimates attribute chicken as the vector for approximately 50 to 80% of all human cases of campylobacteriosis (Hazards, 2011).

Consumer Behaviors and Safe Food Handling in Meal Preparation

Verhoeff-Bakkenes et al. (Verhoeff-Bakkenes et al., 2008) reported that after handling raw meat or poultry, 34 – 58% of consumers do not wash their hands properly, 25 – 39% of consumers do not clean their cutting board adequately, and 29 – 45% do not clean their kitchen utensils sufficiently. Maughan, et al. (Maughan et al., 2016) observed only 39.6% of food handlers wash their hands after handling chicken breast in their study. They noted that many subjects simply rinsed their hands after contact with raw piece of chicken, or did not wash them at all, choosing instead to wipe them on a paper towel.

In the study by Redmond and Griffith (Redmond & Griffith, 2004a), they observed unsafe behaviors that facilitate cross-contamination in greater than 50% of meals either directly from raw chicken and/or associated packaging or via cutting boards and/or knives. Compounding this

problem of inadequate hygiene were judgements of “optimistic bias” and the “illusion of control”, as well as notions of perceived invulnerability to food poisoning from home-prepared foods – “it only happens to others. These attitudes are common among consumers who feel that their hygiene routines in preparing a meal are sufficient because no adverse consequences have been registered over many years of performing the repeated procedures.

In various consumer food handling surveys that have been conducted, respondents often fail to realize the importance of contact transmission in the spread of pathogens throughout their environment as well as from person-to-person (Winter, 2015). And, their reporting of handwashing frequency is often higher than what is observed (Beumer & Kusumaningrum, 2003; Mullan & Wong, 2009; Todd et al., 2010). Moreover, there is a disconnect by the layperson of the importance of fecal-oral route in the transmission of pathogens implicated in illnesses (Todd et al., 2010).

According to Kennedy et al. (Kennedy et al., 2011) a higher level of perceived importance of correct food handling behavior is an associative behavior of higher level of educational attainment in the matter, whereas, the perceived risks of contracting foodborne illness from various food handling behaviors/prevention of food poisoning illness were not linked to better food safety practices. Those who care about food safety issues are more accepting of the information given, whereas those who do not perceive it to be a problem, one way or the other, are more prone to discount or disassociate the issue of safe food handling practices in relation to the illnesses that are present. Consumers must know and be aware of the need for good hygiene at home to prevent the occurrence of infectious diseases (Azevedo et al., 2014; Gorman et al., 2002), and then

need to be motivated to act on that knowledge as a precondition for behavioral change (Abbot et al., 2009; Bánáti, 2011; Cope et al., 2010).

Maughan, et al. (Maughan et al., 2016) observed in their study that the participants relied on multiple methods to determine when their items were perceived to be “fully cooked.” Consumers still rely mainly on visual and textural cues (color and appearance) in ascertaining the doneness of a chicken meal. Only 36.6% of those observed used a thermometer when cooking chicken breasts, and only 33% used the food thermometer as the method to determine doneness. Among those using a thermometer, only 70.3% used it correctly. Additionally, in this study, 24% of the baked chicken breasts and 32% of the turkey patties were not cooked to at least 74°C (165°C) (Maughan et al., 2016). Moreover, there was no statistical difference between those who did and did not use a thermometer in achieving an endpoint temperature of at least 74°C. The authors emphasize that a focal message of food safety is to get food preparers to simply use a food thermometer. If using a food thermometer does not improve the public’s ability to cook items to the correct temperature, due to improper use or to knowledge gaps as to the corrected endpoint temperature, then perhaps more focus needs to be placed on teaching consumers *how* to use a food thermometer in addition to simply possessing the device.

Kosa et al. (Kosa et al., 2015) concluded that based on their survey findings, education is needed to improve consumer handling practices for raw poultry to decrease illness attributed to *Salmonella* and *Campylobacter* in raw poultry products. In their study, poor food safety practices included: improperly storing raw poultry in the refrigerator, not using a food thermometer to determine doneness during cooking and washing raw poultry before cooking. Moreover, as stated in other consumer behavior studies (Millman et al., 2014; Phang & Bruhn, 2011; van Asselt et al.,

2009) there was a disparity (low adherence) between self-reported behavior vs. actual practice in safe food handling, storage, cross-contamination, and cooking procedures.

One of the most basic and integral aspect related to food handling hygiene to prevent cross-contamination is proper handwashing (Humphrey, 2001; Kennedy et al., 2011). Hand washing efficiency is determined by two major mechanisms of the procedure: how well (soaps, friction, and duration) and how often (frequency) it is performed. The duration of the hand washing process is a critical for removing microorganisms (Todd et al., 2010). The duration recommended by the Centers for Disease and Prevention (CDC) is 20 seconds using soap and clean running water (hot or cold) (CDC, 2017b). The same document recommends that handwashing be performed before, during, and after food preparation, as well as before eating (CDC, 2017b). Hoelzl, et al. (Hoelzl et al., 2013) reported that *Campylobacter* can survive 20 minutes on the finger tips, which is sufficient time during meal preparation or food handling for the bacteria to cross-contaminate other food items and surfaces. In the study conducted by Redmond and Griffith (Redmond & Griffith, 2004b), they observed inadequate hand decontamination procedures on at least one occasion after handling raw chicken during all meal preparation. Meredith et al. (Meredith et al., 2001) stressed the importance of proper hand hygiene (washing and drying) in the transfer of pathogens to ready-to-eat (RTE) foods as well as the surrounding environment. Most of the food hygiene interventions (five of eight) involved handwashing following contact either with raw chicken or the waste bin when they tracked the participants in food preparation around the kitchen.

Transfer of Campylobacter to and from Food Contact Surfaces

Foods prepared in the kitchen have the possibility of becoming cross-contaminated with *Campylobacter* by contacting raw products, particularly skinned poultry (Fravalo et al., 2009). In a study performed by Verhoeff-Bakkenes, et al. (Verhoeff-Bakkenes et al., 2008), the authors discussed that irrespective of the transfer route (hands: 0.16%, cutlery: 0.10%, cutting board: 0.08%, or a combination of these routes: 0.19%), a mean of 0.12% of *Campylobacter* initially present on artificially inoculated chicken breast fillets was transferred to prepared salads. Additionally, these transfer rates for *C. jejuni* were also observed when cutting chicken with a knife on a cutting board- chicken to knife (0.02 to 0.26%) and chicken to cutting board (0.02 to 0.10% with plastic; 0.01 to 0.13% with wooden). Also, from this study, cutting chicken with scissors versus a knife transferred more cells onto the cutting utensil (0.08 to 0.31%).

Luber, et al. (Luber et al., 2006) studied chicken parts naturally contaminated with *Campylobacter* on the surface. The study simulated typical scenarios in kitchens and quantified the surface to surface transfer of the pathogen. The mean transfer rate from the chicken parts to hands were 2.9% – 3.8% of viable *Campylobacter*. The transfer from chicken parts to plate was 0.3%, and the transfer from chicken parts to cutting board and knife was 1.1%. The average transfer rates from hands to kitchen utensils to ready-to-eat foods ranged from 2.9% - 27.5%. Redmond and Griffith (Redmond & Griffith, 2004b), also using naturally contaminated chicken parts, isolated from nearly a third of all meal preparations where positive raw chicken breasts were handled. They also confirmed *C. jejuni* strains found on raw chicken pieces were found on the prepared chicken salads. Thus, confirming microbial transfer from raw chicken breasts to finished products.

Fravelo et al, (Fravalo et al., 2009) studied the quantitative transfer between naturally contaminated chicken thighs to cutting boards. A mean of 60.5% were transferred for an exposure time of one minute and 80.6% for samples that were in contact with the cutting board for 10 minutes. The percent transfer rate varied from 0.05 – 35.7% and was significantly different and inversely related to the initial counts on poultry skin. A linear relationship between the initial load of *Campylobacter* on the skin and in the value of the percent transfer rate.

Sarjit and Dykes (Sarjit & Dykes, 2017) noted that rinsing with water significantly reduced the attachment on most of the preparation surfaces, in agreement with the van Asselt study (van Asselt et al., 2008) but in contradiction with the study conducted by de Jong et al. (de Jong et al., 2008), who reported no significant differences between rinsing with water alone and non-washing. Humphry (Humphrey, 2001) noted that regarding the temperature of the (hot) rinse water the highest temperature recorded was 57°C at the start, coming down to 47°C by the end, which was adequately hot. At the other end of the scale, another participant had washing-up water that started out at 40°C and had gone down to 31°C by the end of the rinse. The average temperature in the study was 47.5°C, which feels hot on the skin but does not affect pathogenic microorganisms in question, especially if protected with food residues. He advocates that hot water rinses should be as hot as they can, with the donning of rubber gloves and the use of a brush rather than a cloth for food contact surfaces, because that combination will mean that the washing-up water will start off hotter than it would if one did not have gloves on and if one washed with a cloth.

The relative cross-contamination transfer between hands and cutting board may be explained by the difference in the sub-environment. Boursillon and Riethmuller (Boursillon &

Riethmuller, 2007) stated that it is generally recognized that microorganisms penetrate the pores of the wooden cutting board where they become trapped. In their experiment, it was shown that pine boards perform slightly better than beech or plastic cutting boards when used under optimal conditions, i.e., boards that were clean and dry (Boursillon & Riethmuller, 2007). Certain wooden cutting boards, especially pine, expresses antimicrobial activity, albeit slow acting. An explanation of the difference in survival of *Campylobacter* on various cutting board surfaces was noted by Corry, et al. (Corry & Atabay, 2001). Survival of *Campylobacter* stressed by aeration was enhanced by wood, due to the surface's characteristic of having pores of 16- μm in diameter or less, which have interesting parallels with the protective effect of chicken skin.

Boursillon and Riethmuller (Boursillon & Riethmuller, 2007) pointed out that plastic cutting boards, which are the standard in the processing environment, are not as easy to clean as wooden boards. Moreover, the type of wood constituting the cutting board determines the recoverability of microorganisms contaminated on the cutting board. Pine cutting boards exhibited much lower recovery of the contaminants on RODAC discs when compared to boards made of polyethylene and beech, probably due to its marked retention potential, in addition to its antimicrobial properties. Also discussed by the authors was that due to polyethylene's non-absorbing properties, without a cleaning step, bacteria can remain viable in high numbers for at least 24 hours post-contamination. Moreover, even after cleaning with detergent, it was demonstrated that polyethylene boards could not be reduced to a clean level – partly because plastic boards are often structured to allow for utility or aesthetic purposes.

Tang, et al. (Tang et al., 2011) demonstrated that although there was no significant difference in the mean transfer rate of *C. jejuni* from raw chicken onto the type of cutting board

(rubberwood or polyethylene), they did notice that scoring that occurs with the repeated use of the cutting board produced crevices on the surface of soft rubberwood whereas scoring on hard polyethylene boards caused numerous cuts that created niches to shelter bacteria. Significantly more of the pathogen was transferred from raw fillets and skin to the scored cutting boards, versus non-scored cutting boards. This was in agreement with the study conducted by Sarjit and Dykes (Sarjit & Dykes, 2017), although the study observed a higher transfer of *C. coli* from plastic cutting boards vs. wooden ones. The authors (Sarjit & Dykes, 2017; Tang et al., 2011) noted that the ability of scored rubberwood/ non-pine boards to pick up significantly more bacteria than polyethylene boards suggested that crevices were more efficient than surface peeling in trapping the bacteria. More importantly, cooked chicken picked up *C. jejuni* more frequently from scored contaminated boards, compared to unscored cutting boards. Scored rubberwood transferred significantly higher numbers of the pathogen to the prepared meal. They also demonstrated that although the transfer rate was greatly reduced, *C. jejuni* harboring on contaminated cutting boards from raw chickens could contaminate cooked chicken even if the cooked meal were initially as hot as $100\pm0.5^{\circ}\text{C}$.

The constitution of cutting boards in processing facilities and in the retail and domestic kitchens may attribute to the level of cross-contamination. A change in material – whether in potential antimicrobial properties or durability – to mitigate transfer (with considerations to safety, ease-of-cleanability) of the cutting board would likely result in the reduction of foodborne illnesses. Cutting boards represent an important factor in the cross-contamination of foodborne pathogens to hands and ingested meals.

Moore et al. (Moore, Sheldon, & Jaykus, 2003) found that, although natural desiccation reduces the concentration of viable *Campylobacter* on stainless steel surfaces, relatively high numbers could still be recovered and transferred to ready-to-eat foods one to two hours after brief contact with the material. The mean percent transfer from this study ranged from 16 to 38% for dry lettuce and from 15 to 27% for wet lettuce during the 80-minute sampling period from artificially inoculated stainless-steel coupons (25 cm²). The percent transfer increased with sampling time up to 40 minutes and then decreased at the 60 and 80-minute sampling periods for both wet and dry lettuce.

Presence and Persistence of Campylobacter in the Kitchen Environment

Campylobacter spp. is a fastidious, microaerophilic pathogen that does not survive well in the environment, and it is also difficult to isolate and identify successfully. In the kitchen environment, Azevedo et al. (Azevedo et al., 2014) noted that *Campylobacter* was not found in any of 15 kitchens sampled in their study. However, Borruso and Quinlan (Borruso & Quinlan, 2017) isolated the organism in 3% of homes sampled in their study. Although the positive isolation of the pathogen was low, the fact that viable *C. jejuni* was isolated at all from kitchens in which food-preparation was not being actively performed indicates that the environmental reservoir must be considered as a factor in the cross-contamination process.

Transfer of *Campylobacter* to kitchen surfaces during meal preparation can occur frequently (Cogan, Bloomfield, & Humphrey, 1999; Gorman et al., 2002; Lopez et al., 2015; Nauta, 2010). The pathogen could be isolated from non-food surfaces such as doorknobs, and it could also be found three hours after kitchen cleaning (Cogan et al., 1999). In their study, fewer preparation surfaces were contaminated after cleaning but more positive samples were recovered

from the taps, utensils, and condiments than were found prior to cleaning, indicating that the bacteria may have been transferred to these sites during the 'cleaning' process. Although not explicitly modelled within the body of work in this risk assessment, Kennedy et al. (Kennedy et al., 2011) also found a very strong relationship between contaminated hands on taps followed by the failure of over half of the participants to thoroughly wash their hands after handling raw meats.

Maintaining control in the spread and persistence of foodborne pathogens in the domestic environment by practicing proper hygienic practices and disinfection of food-contact surfaces is of the utmost importance to mitigate foodborne diseases. The aggregation of the studies reviewed link contaminated raw foods with important cross-contamination paths to other raw and RTE foods. Cross contamination is a significant risk for campylobacteriosis given the transfer rates of viable organisms from raw chicken products to surface, and back from the contaminated surface to RTE meals and hands observed in these studies. The low infective dose of *Campylobacter* in humans may help to explain the sporadic nature of infections caused by this pathogen that originate from the home. In the review of pertinent literature, it is evident that there needs to be an increase in awareness for the potential and impact of cross-contamination and hygienic practices by the consumer, as well as to give more focus to the survival characteristics of this "fragile" pathogen.

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CHAPTER II - Characterizing of the spread and survival of *Campylobacter* from Pre-Processing through Consumption on Boneless Skinless Chicken Breast Fillets Domestically Prepared in the United States

ABSTRACT

A descriptive farm-to-fork quantitative microbial risk assessment (QMRA) was developed that initiates with estimates of the prevalence and concentration of *Campylobacter* on individual chickens based on the average infection frequency observed, and ends with human exposure to *Campylobacter* and the estimated cases of illness within boneless skinless chicken breast prepared and consumed domestically in the United States. It considered the impact of reductions of control measures and avenues for cross-contamination within different phases of processing, retail, home storage, and product handling.

This study follows the modular process risk model (MPRM) methodology for food chain QMRA. The model consists of interlinking modules, with each module representing a distinctive step in the farm-to-fork process. The data generated is incorporated into the model to estimate the risk to populations and the disease burden. The annual simulated cost-of-illness incurred in the U.S. was used as a metric to gauge public health burden.

The model estimates 108,174 annual illnesses, 834 hospitalizations, and 37 deaths in the contiguous United States associated with the hazard characteristic. Each year, 96 patients are expected to develop Guillain-Barré Syndrome (GBS), and 9,613 cases will develop Irritable Bowel Syndrome (IBS). The total annual economic burden is expected to be approximately \$366 million.

1. INTRODUCTION

Human campylobacteriosis is one of the most significant food-borne diseases worldwide. The disease ranks in the top three bacteria-acquired food attributed illnesses in the U.S. (Scallan et al., 2011; Scallan et al., 2015). Scallan et al. (Scallan et al., 2011) estimates campylobacteriosis attributes for ~845,000 illnesses, ~8,500 hospitalizations, and ~80 deaths annually in the United States, of which 80% are attributed to a foodborne origin. The previous estimate for campylobacteriosis in the U.S., the first comprehensive estimates of foodborne diseases in the U.S., as reported by Mead et al. (Mead et al., 1999) was ~2.4 million illnesses (~1.9 million foodborne), ~13,000 hospitalizations (~10,500 foodborne), and 124 deaths (99 foodborne). Consumption of chicken products is a major source of human campylobacteriosis (Indikova, Humphrey, & Hilbert, 2015; Smadi & Sargeant, 2013). In some industrialized countries, *Campylobacter*-associated gastroenteritis has been estimated to be two to seven times more common than *Salmonella*, *Shigella*, or *Escherichia coli* O157:H7 (Allos, 2001). *Campylobacter* infection is of concern and a socio-economic burden due to its large number of cases incurred annually.

The primary aim of this quantitative microbial risk assessment (QMRA) was to estimate the public health burden of a commonly consumed food commodity on a yearly basis in the United States as well as to assess the impact of reductions of control measures within different phases of processing, retail, home storage, and product handling. This risk assessment considers the impact of major control measures applied throughout the farm to consumption model chain in terms of human campylobacteriosis cases in humans. A descriptive farm-to-fork QMRA was developed that starts with estimates of the prevalence and concentration of *Campylobacter* on individual chickens

based on the average infection frequency observed and ends with human exposure to *Campylobacter* and the *estimated* cases of illness within a selected food product (boneless skinless chicken breast).

This study follows the modular process risk model (MPRM) methodology for food chain quantitative microbial risk assessment (QMRA) (Schaffner, 2008). The model consists of interlinking modules, with each module representing a distinctive step in the farm-to-fork process. The processing of chicken carcasses is a sequential process that has vulnerabilities for contamination with foodborne pathogens, in this case, *Campylobacter*. Figure II. A represents a general overview of the pathway.

Campylobacter spp. are common bacteria of poultry and poultry products. At the beginning of processing, greater than 80% and up to 88.2% of processed poultry are contaminated with *C. jejuni* or *C. coli* (Jacobs-Reitsma, 2000; Solow, Cloak, & Fratamico, 2003; Willis & Murray, 1997; U.S. Food and Drug Administration, CVM, 2000). The farm/baseline module estimates the extent of external contamination that occurs while the birds are on the farm, and the external contamination that occurs during transportation of the flock to the slaughter facility. In the model, if a bird was positive for *Campylobacter*, the predominant species isolated was assumed to be *C. jejuni*.

There are various pathways in the processing of the chicken carcass to reduce bacterial load or gain bacteria from environmental contamination. Among the processes, scalding is one of the most effective processes to reduce the level of *Campylobacter* on processed broilers. This is due to the effects of washing with heated water to remove dirt and fecal matter off the carcass exterior (FAO-WHO, 2009a; Guerin et al., 2010; Osiriphun et al., 2012). However, the process of

scalding has limitations in reducing the level of contaminants in that it cannot eliminate excessively high numbers of pathogens on the exterior, and it is very dependent on the method of scalding used (FAO-WHO, 2009a). Therefore, a static level of reduction was used for this module. A reduction of 2 – 3 logs/carcass is seen within this process (Byrd & McKee, 2005).

Defeathering is a mechanical process that follows scalding, which facilitates the removal of feathers and loose soil from the skin. A series of in-line machines, containing banks of counter-rotating assemblies holding multiple rubber “fingers,” scour the surface of each carcass. These machines incorporate continuous water sprays that aid in flushing away the feathers and other debris as they are removed. A final process of hand-finishing of attached feathers may be performed by hand (2002; FAO-WHO, 2009c; Sams, 2001a). The principal microbiological problem with defeathering is cross-contamination of carcasses associated with the mechanical action of the machines, and the tendency to disperse microbiological contaminants in all directions via aerosols. In most cases, the levels of *Campylobacter* and numbers of carcasses contaminated significantly increase (Berrang, et al, 2001; Berrang & Dickens, 2000; Berrang & Dickens, 2004; Berrang, Northcutt, & Dickens, 2004). For modeling reduction or cross-contamination in this module, the explicit effects of seeder carcasses were ignored, preferring the probabilistic effects of reduction or cross-contamination that could occur due to defeathering by combining the datasets of two relevant studies (Berrang & Dickens, 2004; Seliwiorstow et al., 2015).

The evisceration phase involves removal of viscera of the carcass and harvesting of edible offal. The need to minimize rupture of the exposed intestines and prevent the spread of fecal matter are of the utmost importance in controlling bacterial pathogens associated with chickens.

The evisceration process is another phase in chicken processing that poses an avenue for cross-contamination.

The process of washing reduces the level of contaminants such as *Campylobacter* and other bacteria from the carcass. In processing raw poultry and poultry products, monitoring of the effects of decontamination steps are usually geared towards total aerobic bacteria, *E. coli*, and/or *Salmonella*. However, few refer directly to the elimination of *Campylobacter* spp. (Berrang et al., 2007; Corry & Atabay, 2001). This process step presents another challenge in that there may be cross-contamination from the surviving bacteria in the wash-off in the chill tanks. Rosenquist et al. (Rosenquist et al., 2006) reported that the incidence of *Campylobacter* spp. increased from 86.4% to 90.8% for pre-chill to post-chill carcasses. This may be due to the possibility that large tanks that hold many carcasses can be responsible for cross-contamination between carcasses.

Washing and chilling of chicken carcasses has demonstrated that it reduces the number of *Campylobacter* on the carcass (Kameyama et al., 2012; Northcutt & Jones, 2004; Seliwiorstow et al., 2015; Shin, Martin, & Sánchez-Plata, 2011; Yang, Li, & Johnson, 2001; Zweifel, Althaus, & Stephan, 2015). However, cross contamination has been shown to occur in the chill tanks (FAO-WHO, 2009a; Guerin et al., 2010; Rosenquist et al., 2006).

Following processing of the chicken meat, USDA regulations require that poultry meat must be maintained at temperatures above -3°C (26°F) at the meat core to be labeled “fresh” (Sams, 2001b). Based on NASS data, 86% of the products are stored refrigerated (2 – 8°C) and 14% were stored frozen ($\leq 0^{\circ}\text{C}$) prior to transport to retail. During retail storage, the average overall shelf-life for commercially eviscerated fresh cut-up carcasses stored at 4.4°C is 6 – 8 days (Sams, 2001b). The USDA (U.S. Dept. of Agriculture, FSIS, 2014) states that whole chicken carcasses may be frozen

indefinitely (9 months for chicken parts, for quality). In the modeling process, frozen chickens were assumed to be consumed within 31 days' post-purchase. The USDA also recommends that chicken products stored in refrigeration conditions to be consumed within 1 – 2 days' post-purchase.

The model is designed to be comprehensive. The improper handling of raw chicken products is a major factor in the dissemination and ultimate ingestion of pathogens such as *Campylobacter* and *Salmonella* spp. Two features that are unique in the home storage portion and preparation of the meal are cross-contamination from the outer package and internalization of *Campylobacter* in the muscles of the chicken breast fillet. For cross contamination from the outside package (Outside package--->Hand), estimations were developed and modelled using data published in the UK for fresh retail chickens and their packaging (U.K. FSA, 2016). The survey represented over 4,000 samples collected over a 13-month period from February 2014 to March 2015. Internalization of *Campylobacter* in poultry meat has so far been discounted as a significant source of the pathogen in accounting for human campylobacteriosis in quantitative microbial risk modeling. In the current model, data was adapted from Lubert and Bartelt (Lubert & Bartelt, 2007) to factor in the significance of modeling such an event during cooking.

Various factors are measured in this QMRA. In addition to the farm-to-fork approach, the model provides more than relative risks for the illness. The probabilities of infection and illness are modelled, and using these derived rates, an annual burden in the contiguous United States is simulated for the food pathogen/food interaction, which includes morbidity, hospitalization, and mortality. Moreover, the social and economic burdens are estimated, as well as the distribution/stratification of the illness by gender, age group, and seasonality. The model also provides insight into the burden estimates for secondary illnesses following acute

campylobacteriosis in modeling the rates of Guillain-Barré Syndrome (GBS) and Irritable Bowel Syndrome (IBS). Direct and indirect costs are measured for IBS and for GBS, further analysis of the impact of ventilated vs. non-ventilated cases are performed.

2. MATERIALS AND METHODS

2.1. Development of Modules to Model the Risk of Campylobacteriosis

The microbial exposure assessment factored in the occurrence of contamination as well as concentration of *Campylobacter* that may be present on processed chicken breast product. These processed food products were ultimately destined for home preparation and consumption within the contiguous United States. As illustrated in Figure II. A, the risk assessment model presented is based on an interlinked modular structure, where the output generated from a previous module may be the driver for input in the sequential module. The baseline model possesses five main compartments, 1) Farm to Processing, 2) Retail to Home Preparation, 3) Consumption and Dose-Response, 4) Disease Distribution and 5) Public Health/Economic Burden. Following exposure assessment (Processing to Home Preparation), a hazard characterization (consumption → dose-response, probabilities of infection and illness) is used to develop the risk characterization of the model. The risk characteristic is the integration of the information collected during the hazard identification, the probability and magnitude of exposure as individual risk, or the risk per serving of chicken (exposure assessment), and hazard characterization steps to generate the estimates of the probability of adverse events that can be predicted to follow the preparation and consumption of chicken. The data generated is incorporated into the model to estimate the risk to populations

and the disease burden. The annual simulated cost-of-illness incurred in the U.S. was used as a metric to gauge public health burden.

2.1.1. Probability distribution functions and Monte Carlo (Latin Hypercube) simulation

Experimental data collected from literature were transformed into appropriate probability distribution functions. When appropriate, aggregated data sets were fitted to theoretical distributions using BestFit® (@Risk software version 8.0, Palisade Corporation, Ithaca, N.Y.). Akaike's Information Criterion (AIC) was used to calculate the log-likelihood function of the BestFit parameters. AIC was developed for model selection – the process of ranking among fitted distributions (Palisade, 2015). Information criteria is better suited for model selection due to the accounting of, among other things, the number of free parameters of the fitted distribution. The theoretical underpinnings rely on Bayesian analysis. The AIC is recommended for use by the @Risk software.

For some distributions, the accuracy of fit of a distribution was ranked using the Anderson-Darling (A-D) test, which emphasizes fitting a distribution at the tails as well as at the main body (Vose, 2000). A Monte Carlo simulation with Latin-Hypercube sampling was utilized to simulate the distribution of contamination probabilities and concentration of *Campylobacter* spp. in a prepared meal as a result of cross contamination (Luber et al., 2006). When applicable, an initial seed value of one was used for simulations. The simulation model considers a grouping of 1,225 birds from a random flock at the point of pre-transport, assigns them a baseline contamination number, and follows them in a hypothetical processing plant in the contiguous United States. There are several processing steps (stages) that are described within modules from farm to consumption. At each of

these stages, the number of *Campylobacter* on the bird, carcass, and processed foodstuff is calculated along with the proportion that are contaminated. This simulates the effects of that step on the changes on the number of organisms on or in the food. For each model simulation, 10,000 iterations were used for the set of 1,225 birds.

Variability is incorporated by means of probability distributions. Throughout the modules there are variabilities over the treatment processes and the chickens. The iteration number of the Monte Carlo simulation determines the number of times a quantity is resampled which varies over conditional or treatment processes. A quantity varying over individual chickens/food products is resampled for every chicken/product and every iteration.

A simulation using 1,225 individual observations with 10,000 iterations is sufficient to show convergence, where the output distributions become steady for extrapolation of useful statistics such as means, standard deviations, and percentiles. Convergence may be measured and used to assess confidence of reliability of the resulting distributions. In contrast, the realization of random variables used for variability across chicken carcasses represent uniquely different numbers of the chickens – these numbers are not meant to converge. The modules used to model the risk of campylobacteriosis, from the farm through processing, retail, home storage and preparation and consumption are summarized in Figure II. A, and described in the proceeding sections.

2.2. Farm module

2.2.1. *Baseline Prevalence*

A Uniform distribution of 0.65 – 0.80 was set as the initial proportion of chickens that are *Campylobacter*-positive. This range was chosen to represent upper and lower bounds of the

occurrence of contamination with the mitigation of under- and over-representation of the true incidence. The concentration of the *Campylobacter*-positive (+) of the representative chicken was set using a Weibull distribution. The concentration estimate was derived from datasets derived from various studies (Cason et al., 2007). These and other model input distributions are listed in Appendix A. The initial prevalence and concentration of *Campylobacter* (+) birds at the farm level are easily modifiable as data becomes updated and available.

The sample size of each iteration within this model is 1,225 sample sets. This number set provides reasonable levels of precision for a national prevalence, and is extrapolated from a previous risk assessment performed by the U.S. FDA Center for Veterinary Medicine (CVM) (U.S. Food and Drug Administration, CVM, 2000).

2.2.2. Transport

In a typical commercial processing operation, chickens are caught, mechanically or by hand, loaded into cages or crates, and transported on over-the-road trailers to the processing plant where they are held until slaughter (Berrang, Northcutt, Fletcher, & Cox, 2003; Northcutt & Berrang, 2006). A transport cage washing compliance of 28.4% was used based on a survey that indicated this proportion of broiler processors wash and sanitize transportation cages and trucks between each use (J. K. Northcutt & Jones, 2004). A binomial distribution was used in conjunction with this point estimate of compliance to designate if the bird being processed was held in a crate that had previously been washed prior to loading.

Crate Washing: The washing and sanitizing of transport crates may be an avenue to potentially mitigate the cross-contamination of *Campylobacter*, especially in negative flocks.

Although the process reduces the occurrence of cross-contamination, it does not eliminate it.

Northcutt and Berrang (2006) reported that even if the transport cage is washed and sanitized, 2 of 27 samples were still positive for *Campylobacter*. This phenomenon was modeled using a Beta-binomial distribution. Further explanation is provided in Appendix A.

Off-Feed/Transport Times: The total of time off-feed encompasses the interval from the time birds are loaded onto transport vehicles from the grow-out houses to the time the birds are shackled for slaughter and processing in the abattoir. This includes transport and resting (lairage) in the holding cages (Epps et al., 2013). Time off feed and time spent in a clean or unclean cage can affect the level of *Campylobacter* contamination of each bird. The total time off-feed used in modeling was adapted from data by Epps, et al. (Epps et al., 2013) and Wesley, et al. (Epps et al., 2013; (Wesley et al., 2009), where the total time off-feed for each of the six premises was based on drivers' logs and the records of the slaughter plants used in the study, as well as (Epps et al., 2013) which was based on the transport of turkeys. The off-times for broilers are comparable to that of the descriptions used for turkeys (~8 – 12 h.) (Corry & Atabay, 2001; Hansson, Ederoth, Andersson, Vågsholm, & Olsson Engvall, 2005), and it is in line with practices seen in the EU as well for off-feed time for chickens (Seliwiorstow, Baré, Van Damme, Uyttendaele, & De Zutter, 2015). Each phase of the transport time was bootstrapped for 10,000 iterations and fitted for the distribution using the BestFit feature of @Risk using AIC as the ranking criterion for the use of an appropriate distribution.

The main difference between the two poultry products (chickens vs turkeys) is the number of birds loaded onto the cages, however, the mechanistic principles are similar (Alvarado, 2006; Conner, Davis, & Zhang, 2001; Rasschaert, Houf, & De Zutter, 2007). Off-feed time used in

modeling (in hours) were separated to the following: 1) Feeders lifted pre-load out; 2) Loading time; 3) Transit time; and 4) Holding time. The total off-feed time estimate used was a mean of 10.7 hours. The details of off-times used in estimations are described in (Wesley, Muraoka, Trampel, & Hurd, 2005). At the beginning of processing, the USDA estimates that 70 – 80% of carcasses at rehang to be contaminated with *Campylobacter* (U.S. Food and Drug Administration, CVM, 2000; U.S. Dept. of Agriculture, FSIS, 2008). The level of colonization at the point of slaughter is based on the output generated after the transport module.

2.3. Processing Module

The Processing stage consists of a series of modules that simulates the possible level of contamination on a carcass within a given module. The model considers the level of contamination and occurrence of contamination on a carcass after scalding, defeathering, evisceration, washing and chilling, portioning, and in-plant storage. Each module within the processing stage uses datasets from studies that capture key elements of processing and rationalized assumptions when needed. The output from the previous module, feeds into the input of the present module, e.g. the outcome of the concentration and occurrence of contamination of *Campylobacter* seen on the carcasses from scalding is the input for evisceration. The overall outcome from the Processing stage is the estimate of the probability of contamination of *Campylobacter* seen from sampling and a distribution for the number of bacteria present on each carcass.

2.3.1. Scalding

A reduction of 2 – 3 logs/carcass was combined with a normal distribution to model the effect of scalding on the level of *Campylobacter* contamination. Variabilities in the scalding process, such as the direct impact of pH, water temperature, flow rate of the scald water, and exposure time to log reduction, were not directly accounted for in the module. These uncertainties in the process can be incorporated as robust data are recognized.

2.3.2. Defeathering

A beta-binomial distribution was used to indicate the use of two separate distribution sets. Of the 17 sets/batches combined, 4 resulted in potential reducing effects. With the standard deviation given, however, it is possible for a slight increase if this “decrease” derived distribution is used. Within the change in contamination, the effect was initially fitted using a dataset generated with the “Distribution Fitting” (“Distribution, Define” @Risk 8.0) option of @Risk.

2.3.3. Evisceration

The change in contamination level is dependent on the concentration on the birds as estimated in the “Defeathering” phase, provided they are *Campylobacter*-positive. The level of cross-contamination is split between *Campylobacter*-positive carcasses up to 3.4 log/carcass and those that are greater than 3.4 log/carcass. In each case, for the *Campylobacter*-positive carcass, a Lognormal distribution is used to estimate the level of cross-contamination. Change in the level of *Campylobacter* can either increase or decrease in this module, depending on the actions in the pathway. Further explanation is provided in Appendix A.

2.3.4. *Washing and Chilling*

Within this module, there are seven possible antimicrobial chemicals used during washing and/or chilling in this simulation. The proportions are based on 800 processing activities in the 20 plants sampled by Berrang et al. (Berrang et al., 2007). This risk assessment assumes that the proportion of chemicals used in these plants is representative of that used by all processing plants in the contiguous U.S. The seven chemicals used are: 1) Hypochlorous acid (HAS), 2) Peroxyacetic acid (PA), 3) Acidified sodium chlorite (ASC), 4) Food grade acids (FGA), 5) Trisodium phosphate (TSP), 6) Chlorine dioxide (CD), and 7) no on-line reprocessing chemical used. In the baseline model, the proportion links to a dialogue box that can be modified with updated data, as necessary.

The reductions in *Campylobacter* on carcasses are correlated to a Normal distribution when these chemicals, if used, are combined with water chilling. Moreover, within this module the usage of chlorine in the chill tank or water spray is modeled in a Beta-binomial distribution, where 16 out of 20 plants used chlorine in their chill tank. All carcasses processed in the contiguous U.S. are assumed to undergo water chilling (as opposed to air chilling) in this risk assessment. The occurrence of cross-contamination due to water chilling is also considered. A beta-binomial distribution is used to capture this phenomenon. We assume if cross-contaminated, the increase due to cross-contamination is normally distributed, with a mean of 0.45 log/carcass increase.

2.3.5. *Portioning*

Due to the lack of experimental data on the effects of portioning of a contaminated carcass to a final product such as skinless, boneless filets, assumptions were made to fill in the data gap

experienced in this module. Following washing and chilling, the carcass is processed to the final product – skinless, boneless breast pieces. The mechanism of portioning plays a role in the number of *Campylobacter* on the product. Each carcass was assigned a weight using a Uniform distribution of 1,130 to 1,700 g based on USDA, Agricultural Marketing Service guidelines (AMA) (U.S. Dept. of Agriculture, AMA, 2013). An assumption is made that the filet produced for modeling is in proportion to the carcass weight. From the total weight, a filet weight of a Uniform distribution of 6 – 8 % is considered.

The redistribution of the contaminants, if present, is assumed to be proportional to the contamination from carcass to filet. The number of *Campylobacter* reduced by the removal of skin in the breast filet preparation is estimated by a Normal distribution, where the magnitude of 0.22 logs is multiplied to the total contaminant level (log) to derive a log reduction effect seen by this process. The chance of cross-contamination from the environment, e.g. carcass splitter, exterior belt line or manual contact through personnel is modeled by a Beta-binomial (15,5) distribution. If cross-contamination occurred from exterior sources, the level of *Campylobacter* increase on the surface of the filet by Normal (SD) (log 0.27,0.027). The product is considered contaminated if one or more CFU of *Campylobacter* is present.

Cold Storage of Portioned Breast Fillets: There are three separate stages within the risk assessment model that estimate the correlation of pathogen prevalence and concentration on the product due to typical temperature conditions seen in storage – post processing, retail, and home. Post storage, a proportion of the processed food item is stored under refrigeration conditions or frozen based on USDA National Agricultural Statistics Service (NASS) data. This proportion carries over to transport and retail storage. Using available data, the effect of time-dependent reduction

of *Campylobacter* in thermal conditions was estimated to predict contamination levels on a product post-storage.

The modules involving the storage of the finished, raw product (end-of-processing, retail, and home) considers the effects of refrigeration and freezing independently. Both refrigeration and freezing reduce the number of *Campylobacter* on the surface of chicken carcasses, given time (number of days), albeit at different rates. Time-dependent thermal death models (Chilled/Refrigerated/"Fresh" and Frozen) for *Campylobacter* are utilized on products post-storage. A log-linear inactivation model employed – with a caveat that a weakness of this model is that this model cannot incorporate shoulders or tails of the survival curve. Tailing may be particularly in the context of risk assessment, where the omission of the tails may lead to an underestimation of risks. However, due to the lengths of time used for freezing and chilling and the adequacy of fit derived in the developed survival/inactivation curve, a log-linear inactivation model should be sufficient to capture the dynamics of the survival of the pathogen in the given conditions.

2.3.6. In-Plant Storage

The finished, portioned, and packaged product is held under two distinct storage conditions, chilled (refrigerated) and frozen. The initial setup distributes 86% of the products as being stored refrigerated ($2 - 8^{\circ}\text{C}$), and 14% stored frozen ($\leq 0^{\circ}\text{C}$). These proportions are easily modifiable as data becomes available to ascertain the effects of storage conditions. For those products stored under refrigeration, the duration of processing plant storage and transport which maintained the designated temperatures followed a Uniform distribution of 0.3 – 1.5 days. A PERT

distribution of (1,16,31) days was used for those products stored frozen until reaching market. The reductions of pathogen concentrations on the food product used the log-linear activation models for refrigerated and frozen chicken products listed in Appendix A (In-Plant Storage).

2.4. Retail, Home Storage and Handling Modules

2.4.1. *Transport to Retail and Retail Storage*

The storage condition from which the filets are delivered from processing site are assumed to carry over to the retail space. Uniform distributions of 0.5 – 8 days were used for storage under refrigeration, and 0.5 – 45 days was used for products stored frozen. The tail ends of the distributions used assumed that approximately a half-day was necessary to unload, inspect, and shelve the product. The end-of-holding time for frozen products was truncated to 45 days. The reductions of pathogen concentrations on the chicken breast used the log-linear activation models for refrigerated and frozen chicken products, as described previously. The effects of storage on ice in retail (in refrigeration) was not modelled, as the common retail practice used presently holds the packaged meat on temperature-controlled shelves or bins (for frozen packs). In cases where one or more cells of *Campylobacter* were detected, the cell was tallied as “positive.”

2.4.2. *Home Storage*

Product storage conditions in the home were modelled with the following three scenarios: 1) prepared and consumed day-of purchase, 2) further stored in refrigeration until being prepared and consumed, or 3) frozen until being prepared and consumed.

In the baseline model, Uniform distributions of 1 – 3 days is used for storage in refrigeration, and 1 – 31 days is used for products stored frozen. If the product was purchased refrigerated, 50% is prepared and consumed on the day of purchase, 15% is stored frozen, and 35% is stored in the refrigerator. In cases where the product is purchased frozen, it is assumed that 25% is prepared and consumed on the day of purchase, 45% stored frozen, and 30% stored in the refrigerator. The cell was tallied as “positive” if one or more cells of *Campylobacter* was detected on the filet.

Although the model is comprehensive, it does not take into consideration the scenario where the refrigerated chicken filets were further stored frozen after the expiration of storage limits in chilled conditions (“use by or freeze” date). The impact of this exclusion of this scenario can be further developed – however, assumption on the frequency of this phenomenon must be assumed, due to a lack of data for this process. Moreover, the thermal dynamics imparted on the survival of *Campylobacter* on frozen chicken breasts (room temperature, refrigeration, or microwave defrosting) was not considered.

2.4.3. Cross-Contamination from Package (Outside package--->Hand)

External package contamination was estimated for this model. Only fresh (refrigerated) packs at retail were considered for external package contamination. It was assumed that frozen packages were devoid of external contamination due to the nature of storage in freezing conditions. For chicken packs that were stored in refrigeration, reduction on the outer package due to storage was estimated using the reduction rate for chilling used in other storage modules. The transfer of *Campylobacter* cells to the hands from contaminated exterior of the chicken packs

were calculated using the transfer rate to the hands, and hands to product was calculated using the transfer rate from meat to hands and hands to meat as used in the “Cross-Contamination from Handling of Filets” of the Cross-Contamination module in this model.

2.4.4. Cross-Contamination from Handling of Filets

This module in the risk assessment model focuses on three distinct sites for potential cross-contamination when preparing raw chicken: hands, cutting boards, and utensils, such as knives and forks. Each of these avenues are presented in sub-modules to lay out the impact each has on the overall transfer to the respective environment, and the secondary transfer from these potentially contaminated surface areas to the finished, cooked product, which is then ingested by the consumer.

Hands: Four pathways are given to the effects that hands play in cross-contamination. Default probabilities were designated as Normal distributions for “No wash”, “Rinse”, “Wash with soap”, and “No contact” (meaning that the handler used disposable gloves or generally avoided contact with the uncooked chicken filet).

Cutting Boards: The potential effects of cross-contamination via cutting board was modelled in the risk assessment. Like cross-contamination on hands, there were four pathways available (No wash, rinse, wash with soap and water, and no contact – meaning the initial cutting board was replaced with a clean one in the subsequent preparation of the meal).

Utensils: The last potential cross-contamination activity modelled in the preparation of the meal was the interaction between the raw chicken filet and the utensil used in preparation. The

utensil designated was a knife used to manipulate the food product. This sub-module was formatted much like that developed in the cutting board sub-module.

The number of *Campylobacter* cells (N CFU/filet) ingested was the aggregate number of transferred and surviving *Campylobacter* in the food preparation environment (hands, board, utensil) onto the finished (cooked) meal. The number of cells remaining is assumed to be transferred to the cooked, prepared meal using the same transfer rate used previously in this module, and each of those cells are ingested along with the cells that were on or in the meal that survived after cooking. The calculated transfer from the environment to finished food was developed using datasets derived from studies relevant to transfer of *Campylobacter* on to and off of relevant surface materials, bootstrapped for 10,000 iterations, and estimated with the BestFit feature of the @Risk software to develop the redistribution of the pathogen back to the food (Cools et al., 2005; Jensen, 2012; Lindqvist & Lindblad, 2008; Lubber, Byrnestad, et al., 2006; Mylius, Nauta, & Havelaar, 2007; Sampers et al., 2008; van Asselt, de Jong, & Nauta, 2008).

2.4.5. Cooking

Proper cooking is one of the most critical steps in the preparation of a meal from a food safety standpoint. This module addresses the exposure pathway of ingestion of *Campylobacter* that survives the process of cooking the prepared chicken filet in a domestic kitchen setting. The frequency of undercooking where the cooked meal may remain contaminated and the number of surviving cells on and within the food product are considered. The initial concentration of *Campylobacter* on the chickens that will undergo cooking was set up from the preceding modules in the simulation.

Thermal Reduction of *Campylobacter*: In this module, the thermal effect of cooking on reduction of *Campylobacter* was estimated using D-values (time (minutes) to achieve a one log reduction) with a Uniform distribution of 1.93 – 1.976. The z-value was not considered, and it was assumed the same temperature was used in cooking the meal(s). This D-value and estimated cooking time was based on the work of Van Asselt et al. (van Asselt et al., 2009) and Lubert et al. (Lubert, Byrnestad, et al., 2006). Each dataset was bootstrapped for 10,000 iterations, where then data was fitted using the BestFit feature of @Risk using the AIC. Uncertainty of cooking time can be separated using the simulation feature of @Risk. Each distribution set was assigned a number in which the datapoint selected corresponded with the spread derived from a specific study. The first set, as stated by, “Distribution chooser Cooking=15” refers to the cooking time as observed in Lubert et al. (Lubert, Byrnestad, et al., 2006). The second set, assigned as, “Distribution_chooser Cooking=25” refers to the cooking time as observed in Van Asselt et al. (Van Asselt, Fischer et al. 2009). The third set, assigned as, “Distribution chooser Cooking=35” combines data from the study equally. These “Distribution chooser Cooking” sets can be run as separate simulations to see the effects of using these parameters in the model with minimal model manipulation. The Log CFU/filet reduction (time/D-value) was calculated using these two generated parameters.

Internalization of *Campylobacter* into Meat: An assumption was made that the phenomenon of *Campylobacter* internalization occurs only if the broiler was positive for *Campylobacter* on the surface as well to carry over the proportion from surface to internal contamination, as shown in the study. In the module, the proportion of internally contaminated meat from the surface was derived from the study, where a binomial distribution with a probability of 0.23 of those qualified being positive internally. Among those products where

Campylobacter was found internally, the concentration was derived from an assigned Pert distribution (0.442,0.491,0.788) as a proportion to surface contamination level. The reduction in concentration of internalized *Campylobacter* was factored into the final value when reducing the pathogen levels by cooking.

2.5. Meal Consumption, Ingestion of *Campylobacter*, Dose Response Modules

The ingestion of *Campylobacter* from consuming chicken breasts is simulated by the aggregation of the cells on the individually prepared meals in the model and is determined in the preceding modules. The prepared meal is considered a single serving. It is assumed all servings are consumed in their entirety in one sitting.

2.5.1. Dose-Response

The probability of illness depends on the manifestation of three conditional probabilities—the probability that the organism is ingested; the probability that the organism can survive and infect the host once it is ingested; and the probability of the host becoming ill once infected. The simulation data obtained from the number of cells ingested by the individual was combined with dose-response information to generate probabilistic estimates of risk. Two core methods of estimations were performed using the data generated through the risk simulation. The first method employed a dose-dependent dose-response estimate, and the second utilized a dose-response model based on the ‘single-hit principle’, which are common with microbiological risk assessments, i.e. a single CFU of *Campylobacter* can colonize a host and potentially cause illness, albeit with a relatively low probability (Haas, 2002). In both methods the level of ingested dose is

dependent on the likelihood that the pathogen establishes infection and consequent illness. The method of estimation implies that there is no existence of a 'safe' level of *Campylobacter* ingestion since any level above zero may potentially cause disease in the human population, unlike the threshold method. However, it is also implied that the risk of illness at low exposure levels is considerably less than the risk at higher levels and ingestion of a certain 'threshold' concentration would not necessarily imply automatic illness. The level of contamination of *Campylobacter* on chicken meat and the fraction of these bacteria that is ultimately ingested vary over a wide range.

A Beta-Poisson model for the probability of infectivity was used in this model, where it is assumed to follow a Beta distribution. The following approximation was used:

$$P_{inf} = 1 - [1 - P_{inf}(1)]^D$$

Where

$P_{inf}(D)$: The probability of infection from the dose

$P_{inf}(1)$: The probability of infection from one organism, sampled from the beta-distribution Beta (0.21,59.95)

D : the number of organisms estimated to be in the meal (Dose)

The conditional probability of illness, given infection, P_{ill} , may be positively or negatively dependent on the dose or dose-independent (Teunis & Havelaar, 2000). For this QMRA, the FAO/WHO approach to the probability of illness was used (FAO-WHO, 2002, 2009a, 2009b). P_{ill} was assumed to be dose-independent and was described by the following beta-distribution. Further explanation of the conditional probability, given infection can be found in the 2009 report (FAO-WHO, 2009c). To account for the uncertainty in the conditional probability, a Beta distribution, with parameters that are based on the referenced study ($\alpha = 30$ {number ill+1} and $\beta = 61$ {number infected – number ill+1}) was used: $P_{ill} \text{ given infection} = \text{Beta}(30,61)$.

This risk assessment model estimates the annual number of infections as well as the annual number of illnesses (morbidity).

2.5.2. Consumption of Boneless Skinless Domestically Reared Chickens in the U.S.

Chicken consumption per capita was based on the 2019 U.S. population (contiguous states) post-census estimates from the U.S. Census Bureau (U.S. Census Bureau, 2019). For 2019, this U.S. population was 326,092,106, excluding population estimates for Alaska, Hawaii, and Puerto Rico from the total U.S. estimate (331,433,217 persons). An annual per-capita value representing measurable human exposure to chicken in the United States was extrapolated from USDA data (U.S. Dept. of Agriculture, ERS, 2016). Per-capita consumption of broilers in the U.S., ranged from 79.8 lbs. in 2009 to 88.9 lbs. in 2015 (National Chicken Council, 2017). A Uniform distribution of 55.90 – 62.30 lbs. was used for the model as the consumption weight which excludes bones, feathers, neck, gizzard, and loss of weight due to processing. The fraction of chicken consumed at home (Uniform distribution of 30-50%) and the proportion that is boneless, skinless chicken breast (Uniform distribution of 50-65%) breast consumption proportion was based on surveys done by the National Chicken Council (National Chicken Council, 2017). The "Average serving size" assumes that one chicken filet constitutes one "meal", which in this case is representative of a "serving".

2.5.3. Modeling of Campylobacteriosis due to Consumption of Chicken Product

Morbidity and Hospitalization: The infection and illness rates as presented were calculated by multiplying the probability of infection (P_{inf}) or probability of illness (P_{ill}) to the total annual servings of boneless, skinless chicken breast filets prepared and consumed in the home. The

dataset was compiled for calculated and observed values as mentioned in section 2.5.1. Each of the four distribution sets were assigned a value in which the datapoint selected corresponded with the value derived from a specific calculation, as follows:

- The first set, as stated by, “Distribution Chooser Model Output=15” refers to P_{inf} (or P_{ill}) per dose-dependent dose-response estimate from calculated probability of consumption of a contaminated meal (P_{cons}).
- The second set, assigned as “Distribution Chooser Model Output=25”, refers to P_{inf} (or P_{ill}) per dose-dependent dose-response estimate from compiled (observed) probability of consumption of a contaminated meal (P_{cons}).
- The third set, assigned as “Distribution Chooser Model Output=35”, refers to P_{inf} (or P_{ill}) per dose-response model based on the ‘single-hit principle’ estimate from calculated probability of consumption of a contaminated meal (P_{cons}).
- The fourth set, assigned as “Distribution Chooser Model Output=45”, refers to P_{inf} (or P_{ill}) per dose-response model based on the ‘single-hit principle’ estimate from compiled (observed) probability of consumption of a contaminated meal (P_{cons}).

As discussed in the Cooking section, these “Distribution Chooser Model Output” sets can run independent simulations to see the effects of using these parameters in the model with minimal model manipulation.

Estimated campylobacteriosis illnesses are based on 1) the estimations as presented by Mead, et al. (Mead et al., 1999); 2) the estimations compiled from appropriate U.S. state data; and 3) estimations as presented by Scallan, et al. (Scallan et al., 2011). Mead et al., (Mead et al., 1999) estimated ~2.4 million annual illnesses of foodborne campylobacteriosis in the U.S where the minimum and maximum estimates are based on a factor of 30 and 50 times the number of the confirmed cases of 1997. The state estimate for the annual campylobacteriosis cases in the U.S. is estimated by extracting a rate of 12.54 cases/100,000 persons by compiling state public health

data. The number of cases (38,129) derived was multiplied by a factor of 30, 38, or 50 for minimum, most likely, or maximum, respectively. The most updated and referenced foodborne illness estimates acquired in the United States is authored by Scallan, et al. (Scallan et al., 2011). The mean number of domestically acquired foodborne campylobacteriosis cases is 845,024 (90% credible interval of 337,031 – 1,611,083), and the mean hospitalization rate associated with campylobacteriosis was 17.1%. The attribution of the role of poultry consumption is estimated to be 40 – 70% of all campylobacteriosis cases, as per peer-reviewed journals and reports. The state data generated was derived from calculating the accrued available state data from State Agency Public Health Offices from a three to four-year range of confirmed cases of campylobacteriosis and weighing each state dataset by calculating its rate per 100,000 individuals, if not already available. This calculated data was compared with the reported rate. Louisiana, Massachusetts, New Hampshire, Nevada, and Texas were excluded due to the unavailable data from these states. Gender variations were also calculated from the aggregate available data reported to generate campylobacteriosis differences between men and women. Lastly, age stratifications were also generated from state datasets, and each age range was adjusted to be weighed equally for states reporting these values.

Additionally, acute campylobacteriosis is modelled in terms of the number of hospitalizations, mean number of days hospitalized, general medical costs and fees, general productivity losses due to non-work – direct, and productivity losses due to non-work – indirect (e.g. productivity losses incurred by individuals due to caretaking), the aggregate general economic burden, and disutility, as per FDA, USDA ERS, and works from Buzby, et al. Roberts, and Hoffmann, et al. (Buzby, 2011; Buzby & Roberts, 2009; Economic Research Service (ERS), 2014; Hoffmann, et

al., 2012; Roberts, 2007). The available cost of campylobacteriosis estimates were adjusted using the inflation values from U.S. Department of Labor, Bureau of Labor Statistics (Bureau of Labor Statistics, 2016a, 2016b, 2016c).

As model validation, a secondary file was constructed using previously calculated estimates of boneless, skinless chicken consumption in the U.S. and applying it to the Mead, State, and Scallan estimates for campylobacteriosis expected from chicken consumption from the U.S. This was run for three simulations at 10,000 iterations with 1,225 sample sets each to generate a comparative model. Moreover, considering only the generated state estimates and the Scallan estimates, a “target” estimation for campylobacteriosis was established, where a simulation was performed for 10,000 iterations considering datapoints associated with these two estimates. The Mead estimate was excluded from this measurement due to more current data being available.

Mortality: A Pert distribution of (0.00005, 0.00038, 0.005) was attributed to the number of illnesses per year (N_{ill}/yr) generated in the model output to estimate the attributable mortality related to human campylobacteriosis.

Sequelae – Guillain-Barré Syndrome (GBS): The details of campylobacteriosis-associated Guillain-Barré Syndrome (GBS) is described in detail in Chapter I. In this risk assessment, GBS is modelled using a proportion of those that have a campylobacteriosis illness (Uniform distribution of 0.0009 – 0.0011). A secondary distribution is described as a proportion of the subpopulation of those that are ill by using a Weibull distribution (as 0.03% at 5%; 0.07% at 50%; and 0.15% at 95% of the distribution curve).

Only those cases associated with sequelae from campylobacteriosis are considered: the total estimated GBS cases are split into two types of patients, those who need no aid of ventilation

(non-ventilated, Uniform distribution of 0.7 – 0.8) and those that require ventilation (ventilated, residual value from non-ventilated cases). Public health burden is measured by medical costs, estimated productivity losses – resumed work, estimated productivity losses – cannot work, associate death due to GBS complications, and estimated annual costs due to GBS.

Additionally, GBS is modelled in terms of the number of hospitalizations, mean number of days hospitalized, general medical costs and fees, general productivity losses due to non-work – direct, and productivity losses due to non-work – indirect (e.g. productivity losses incurred by individuals due to caretaking), the aggregate general economic burden, and disutility, as per FDA. The modeling is stratified into age groups (all, less than 5 years, 5 – 9 years, 10 – 14 years, 15 – 17 years, 18 – 44 years, 45 – 64 years, 65 – 84 years, and 85+ years).

Sequelae – Irritable Bowel Syndrome (IBS): Much like modeling *Campylobacter*-associated GBS, the details of campylobacteriosis-associated Irritable Bowel Syndrome (IBS) are described in detail in Chapter I. In this QMRA model, a Pert distribution of (0.085, 0.088, 0.09) was multiplied to the annual number of campylobacteriosis cases expected from the consumption of boneless, skinless chicken breast pieces prepared at the home to establish the estimated number of yearly IBS-sequelae stemming from *Campylobacter*-related illnesses.

To obtain the distribution of direct costs incurred in relation to IBS, a Pert distribution (\$2,269, \$6,182, \$12,007) was used, reflecting inflation adjusted 2012 – 2013 USD from 2001 values, using the datasets based on Nellesen et al. (Nellesen, et al, 2013) and Buono et al. ((Buono et al., 2017). The direct cost was multiplied by the estimated number of IBS patients due to the consumption of skinless, boneless chicken breast meals prepared at the home. Direct costs are defined as those incurred through medication, hospitalizations, outpatient services, emergency

room visits (ER), and laboratory tests. Indirect costs attributable to IBS were also constructed in this manner, using a Pert distribution (\$1,356, \$2,722, \$9,993). Indirect costs are measured as lost productivity due to IBS symptoms or healthcare-seeking behaviors spread out per capita. The total attributable costs were the summation of the direct and indirect costs incurred for IBS sequelae.

Further analysis of direct costs on IBS were performed for the time spent by patients on physician office visits, emergency room visits, and hospitalizations. Normal distributions (days, standard deviation) were used for all three phases with truncations set for 0 days. The distributions used were 1) physician office visits (10.3, 7.7); 2) emergency room visits (2, 2.3); and 3) hospitalizations (1.2, 0.6). The number of days were derived from Buono et al. (Buono et al., 2017) and the days aggregated to number of IBS patients are generated from the model.

3. RESULTS AND DISCUSSION

The results in the following sections were generated from one simulation using 10,000 iterations of the baseline QMRA model for a set of 1,225 chickens. Appendix B includes an example of the inputs and outputs of the model as organized into four tabbed sheets within a Microsoft Excel file. This example is displayed as follows:

Appendix B1 - Model Inputs: Transport and Processing

Appendix B2 - Model Inputs: Storage, Handling, Cooking and Consumption

Appendix B3 - Model Outputs: Pre-Transport, Processing and Preparation

Appendix B4 - Model Outputs: Ingestion, Dose response, Economic burden

3.1. *Campylobacter* Prevalence after Transport from Farm

The initial prevalence of contaminated broiler chickens on the exterior surface with *Campylobacter* spp. at the farm level was 72.51% (median = 72.49%, min. = 61.71%, max. = 82.94%). The bacterial log CFU/broiler load estimate is 4.60 (median = 4.60, min. = 4.52, max. = 4.69). The progression of changes in the occurrence of *Campylobacter* contamination and concentration through the major modules simulated in processing, retail, and home steps are shown in Figure II. B and Figure II. C.

As birds left the farm, in preparation for and transport to the abattoir, the average total time in transport cages was 7.97 hours (min. = 7.76, max. = 8.18). The mean occurrence of cages contaminated with *Campylobacter* is determined to be 22.69% (min. = 18.29%, max. = 27.35%). In cases where cross-contamination occurred in transport with previously negative (-) birds, the increase in log CFU for a previously (-) bird was 0.82 (min. = 0.39, max= 1.37). Among those birds that were already contaminated prior to transport, there was an average log CFU/previously (+) bird increase of 2.12 (min. = 2.01, max= 2.26). Contrarily, of those positive birds, 22.9% (~135/590) saw a decrease in their initial load (0.32 log CFU/previously (+) bird decrease). Overall, following transport of broilers from the farm to processing, it is expected that 78.7% ($\pm 0.0840\%$ 90% CI) of all broilers will be contaminated with *Campylobacter*, with an average log CFU/bird of 7.23 (min. = 7.06, max. = 7.41).

3.2. *Campylobacter* Levels Through Processing

3.2.1. *Scalding*

The scalding process represents one of the most effective methods for the reduction of *Campylobacter* and other external bacteria on the surface of a broiler carcass. The reduction in *Campylobacter* load estimated in this stage is 3.10 log CFU/carcass. Following the scalding phase, the baseline model predicts that a mean of 72.99% (median = 72.98%, min. = 62.86%, max. = 82.45%) of all broiler carcasses will harbor *Campylobacter*, with a bacterial CFU/carcass of 4.51 (median = 4.50, min. = 4.21, max. = 5.63).

3.2.2. *Defeathering*

During this process, 79.63% (median = 79.66%, min. = 61.27%, max. = 99.27%) of all carcasses sampled are predicted to undergo some change in *Campylobacter* load. In total, 64.65% of carcasses experience an increase in concentration, with a mean log CFU/carcass addition of 1.78 (min. = 1.68, max. = 1.89 increase). The remaining sample set experiences a decrease or no change in *Campylobacter* load. The mean decrease expected is 0.99 log CFU/carcass (median = 0.99, min. = 0.90, max. = 1.11 log CFU/carcass decrease). Furthermore, the baseline model estimates 87.15% of all broiler carcasses have some level of *Campylobacter* contamination after the defeathering stage. The mean log CFU/carcass on the individual bird expected is 6.93 (median = 6.90 min. = 6.22, max. = 8.64).

3.2.3. Evisceration

During evisceration, the bacterial load per carcass may increase or decrease. The model simulation estimates an overall mean increase of 0.34 log CFU/bird (median = 0.34, min. = 0.31, max. = 0.36 log CFU/bird). Among birds sampled, 69.8% (min. = 65.8%, max. = 75.7%) are predicted to have an increase in *Campylobacter* load in this stage, with the remainder having a decrease or remaining the same. Among those that gain *Campylobacter*, the average gain is 0.49 log CFU/bird. Among birds that lose *Campylobacter*, the average decrease is only 0.01 log CFU/bird. Following the evisceration phase, the baseline model predicts that 93.56% (min. = 89.88%, max. = 96.33%) of all broiler carcasses will harbor *Campylobacter*, and the mean concentration is estimated to be 6.93 log CFU/bird (median = 6.89, min. = 6.22, max. = 8.63).

3.2.4. Washing and Chilling

As discussed in Chapter I, chilling of poultry carcasses in the United States involves the use of immersion chilling, where carcasses are immersed in large tanks of cold water that are typically chlorinated. The use of water immersion chilling may wash off pathogens – both inside and out – which can transfer to other carcasses in the same chill tank. After the chilling process, the model predicts the mean occurrence of contamination of *Campylobacter* will be 69.00% (median = 68.98%, min. = 61.22%, max. = 75.43%), with an overall concentration of 5.15 log CFU/carcass (median = 5.11, min. = 4.34, max. = 6.27) of *Campylobacter*. A detailed comparison of antimicrobial chemical use and effectiveness during this step is presented in Table II. A.

3.2.5. Portioning

The average file size is 99.05 g (min. = 97.33 g, max. = 100.45 g) after breasts are split and the skin removed. Immediately after the splitting stage, 48.60% of all filets are expected to harbor *Campylobacter* (median = 48.57%, min. = 40.49%, max. = 56.41%). In removing the skin from the broiler carcass, a reduction of *Campylobacter* is realized on the exterior surface. This process reduces *Campylobacter* load by 0.54 log CFU/filet. However, *Campylobacter* may be picked up in the environment through cross contamination at this stage. This even occurs at a frequency of 75.00% (median = 75.02%, min. = 70.86%, max. = 80.00%), with an average increase of 0.27 log CFU/filet. Accounting for the total loss in skin removal and cross-contamination due to the environment, 85.08% (median = 85.06%, min. = 80.98%, max. = 89.22%) are positive for *Campylobacter*, with a load of 2.70 log CFU/filet (median = 2.67, min. = 2.20, max. = 3.66 log CFU/filet) after this stage.

3.2.6. In-Plant Storage / Transport to Retail

During in-plant storage, it is predicted that 63.57% (min. = 57.71%, max. = 70.12%) of these chicken breasts will harbor *Campylobacter*, with an average log CFU/filet of 2.44 (median = 2.42, min. = 1.87, max. = 3.50). There is a significant difference in bacterial load for those that are stored under refrigeration conditions (2.50 log CFU/breast portion) vs. those that are frozen (0.90 log CFU/breast portion). During storage, could expect a log CFU/filet decrease of 0.19 if refrigerated and a 1.68 log CFU/filet decrease if frozen prior to storage in retail.

3.3. *Campylobacter* Levels Through Retail and Home Storage

3.3.1. *Transport to Retail and Retail Storage*

Overall, 31.72% (median = 31.67%, min. = 25.55%, max. = 38.53%) of all chicken breast pieces are expected to harbor *Campylobacter* during retail storage. The *Campylobacter* load (CFU/filet) is estimated to be 95.80 (median = 78.26, min. = 20.67, max. = 879.74 CFU/filet). The level and occurrence of contamination differed greatly dependent on storage at refrigeration vs. frozen temperatures. Among those that are stored chilled, 36.64% (median = 36.60%, min. = 29.72%, max. = 44.34%) are expected to be contaminated with a detectable level (≥ 1 CFU/filet) of *Campylobacter* on the exterior surface. The *Campylobacter* load for positive chicken breast pieces is 111.34 CFU per breast portion (median = 90.88, min. = 24.28, max. = 1,009.99). However, if the breast pieces are stored frozen, only a mean of 1.42% (median = 1.23%, min. = 0%, max. = 7.36%) are expected to be contaminated, with a mean load of fewer than 1 CFU/breast portion (mean = 0.12, min. = 0, max. = 15.30 CFU/breast portion).

3.3.2. *Home Storage*

Home storage of the breast pieces may occur at different temperatures and for different lengths of time. For examples, the chicken may be consumed on the day of purchase or stored in a refrigerator or in a freezer. These storage conditions affect the level of *Campylobacter* prior to preparation for cooking. The model estimates that after home storage stage, 25.34% (median = 25.31%, min. = 19.84%, max. = 31.92%) of all purchased chicken breast filets harbor *Campylobacter*, with a bacterial load of 61.40 CFU/filet (median = 48.02, min. = 12.28, max. =

867.91). Table II. B describes the *Campylobacter* contamination frequency and relevant concentrations after the various scenarios for chicken breast filets during home storage.

3.3.3. Cross-Contamination from Package (Outside package--->Hand)

As simulated, the mean occurrence of contamination on the outside package at time of purchase is 5.86% (median = 5.87%, min. = 3.67%, max. = 8.73%). The model predicts that packages contaminated with *Campylobacter* have a mean of 86.98 cells/package (median = 86.97, min. = 46.02, max. = 173.18 cells/package). Among packages that are contaminated, at time of meal preparation, there is an average transfer of 13.62 cells. Overall, this pathway provides minimal effect on the overall change of *Campylobacter* contamination on the raw chicken.

3.3.4. Cross-contamination from Handling of Filets

This part of the risk assessment process contains many “moving pieces” in terms of how the food is handled, the presence/absence of *Campylobacter* prior to handling, the bacterial transfer from raw pieces, and the bacterial transfer to cooked and prepared pieces. Overall, the percentage of meals contaminated with *Campylobacter* after preparation and handling and just prior to ingestion is 2.61% (median = 2.61%, min. = 0.98%, max. = 4.65%). The mean CFU ingested in a meal from all mishandlings is 1.19 (median = 0, min. = 0, max. = 1,514). If accounting for just contaminated incidences, the mean CFU ingested increases to 50.48 (median = 12.00, min. = 1.00, max. = 59,808). A detailed comparison of cross-contamination scenarios for *Campylobacter* occurrence and concentration are described in Table II. C.

3.3.5. Cooking

The process of cooking a meal is a major source of bacterial reduction in a raw ingredient. The model simulates an average reduction of 6.37 log CFU/chicken breast (median = 6.27, min. = 5.68, max. = 7.39). The frequency of cooking chickens less than 8 minutes, which was found to be a critical threshold in the near-elimination of pathogens on the chicken, was 32.22% (median = 32.08%, min. = 26.61%, max. = 39.84%) in cooking processes simulated. The percentage of cooked chickens still harboring *Campylobacter* after cooking was predicted to be 0.65% (median = 0.65%, min. = 0%, max. = 1.96%).

This module also simulated internalization of *Campylobacter* in the muscle of the chicken breast. This phenomenon was estimated to occur in 58.4% (min. = 34.0%, max. = 83.0%) of chicken breast pieces that were also contaminated externally. If *Campylobacter* was internalized in the muscle of the chicken breast, the mean bacterial load was 0.63 log CFU/chicken breast (median = 0.61, min. = 0.0064, max. = 2.33). The significance of accounting for internalized *Campylobacter* in the muscle of the chicken filet should be nominal in the ingestion of bacteria after the cooking process.

3.4. Ingestion of *Campylobacter* and Dose Response

The final stage of the “Handling → Consumption” stage of the model is the ingestion of *Campylobacter* in a prepared meal. The model predicts the average *Campylobacter* CFU ingested from all meals is 1.34 (median = 0, min. = 0, max. = 1,514). Accounting for just contaminated meals, the mean CFU ingested is 47.57 (median = 13, min. = 2, max. = 48,792). The probability of consuming a contaminated meal, is given by (calculation) as $P_{cons} (calc)$ of 3.24% (median = 3.24%

min. = 1.31%, max. = 5.58%). The probability of consuming a contaminated meal, by tallying up the number of positive meals in the model (observed) is $P_{cons} (obs)$ of 3.02% (median = 3.02%, min. = 1.22%, max. = 5.22%). The model also predicts the average CFU ingested from all meals considered. The probability of infection and illness was estimated using several methods to determine the uncertainty of the output values, within the restraints of the methodologies used. The details of the outputs are presented in Table II. D.

3.4.1. Consumption of Boneless Skinless Domestically Reared Chicken in the U.S.

The annual per-capita consumption of boneless chickens in retail weight was estimated to be 59.1 lbs. (median = 59.1, min. = 55.9, max. = 62.3). The fraction of home chicken meal preparation and consumption was estimated to be 40.0%, which translates to a domestic annual per capita consumption of 23.6 lbs. (median = 23.6, min. = 16.9, max. = 31.0). The fraction of skinless boneless chicken breasts of a chicken meal prepared and consumed domestically was estimated to be 57.50% of the above or 13.6 lbs. (min. = 9.0 lbs., max. = 19.6 lbs.) annually. Since the average serving size was estimated to be 6.00 oz. (170.10 g), the annual servings per capita was estimated to be 36.33 (median = 36.01, min. = 21.35, max. = 56.92). The U.S. Census data estimates the contiguous U.S. population (Bureau, 2019) to be 326,092,106, therefore the total domestic consumption of skinless boneless chicken breasts is 1.19×10^{10} servings (min. = 7.72×10^9 servings, max. = 1.79×10^{10} servings).

3.4.2. Foodborne *Campylobacteriosis* Estimates due to Chicken Consumption

The model simulation estimates that 328,257 incidences of *Campylobacter* infections/year (median = 278,211, min. = 7,268, max. = 2,163,872) would occur in the contiguous United States from the domestic preparation and consumption of boneless, skinless chicken breasts. Among those infections, 108,174 annual illnesses are predicted (median = 91,244, min. = 2,481, max. = 653,318), and among those who get sick, 834 annual hospitalizations are expected (median = 690, min. = 15, max. = 5,587). Moreover, the baseline model estimates that it would lead to an annual death toll of 37 (median = 31, min. = 1, max. = 240) from the domestic preparation and consumption of boneless, skinless chicken breasts due to campylobacteriosis. Table II. E. includes further detail for a model output summary of annual infections, illnesses, cases where physicians were and were not consulted, hospitalizations, and deaths due to acute campylobacteriosis.

In Figure II. D., model estimates for domestically acquired foodborne campylobacteriosis from boneless, skinless chicken breast fillets are compared with illness estimates based on 1) Mead, et al. (Mead et al., 1999); 2) U.S. state data compilations; and 3) Scallan, et al. (Scallan et al., 2011). The generated data from the secondary simulations are as follows: 1) Mead estimates (mean = 282,342, min. = 118,689, max. = 580,128); 2. U.S. state data compilations (mean = 166,815, min. = 66,215, max. = 327,926); and Scallan estimates (mean = 93,934, min. = 13,865, max. = 1,038,476). The “target” estimate, considering datapoints generated from only the Scallan and state data, had a mean of 129,964 campylobacteriosis cases (as compared to the model estimate of 108,174 annual illnesses) from the consumption of boneless, skinless, chicken breasts prepared at the home.

Detailed summaries of illnesses and burdens pertaining to the sequelae Guillain-Barré Syndrome (GBS) and Irritable Bowel Syndrome (IBS) following an onset of campylobacteriosis is provided in Tables II. F. and G. (GBS) and Tables II. F. and H. (IBS). GBS is stratified into non-ventilated patients and ventilated patients, whether the severity of the illness caused the patients to eventually resume working (if applicable), be incapacitated, or in the most severe cases, die. The medical losses, as well as productivity losses are estimated. Overall, it is estimated among the ~108,000 campylobacteriosis cases that roughly 100 patients (additive mean = 96) will experience GBS, with approximately 25% of those being put under ventilation due to GBS-associated paralysis, with a mortality rate of 3.13%.

More cases of *Campylobacter*-associated IBS are expected relative to GBS. The number of IBS cases due after the onset of campylobacteriosis from consumption of boneless, skinless chicken breast meals prepared in the home on an annual basis is estimated to be 9,613 (median = 8,248, min. = 277, max. = 63,206). In terms of days spent per annum in relation to medical procedures related to IBS, specifically IBS with diarrhea (IBS-D), is broken down to the following: 1. Physician office visits, 2. Emergency room visits, 3. Hospitalizations. It is expected that on an annual basis, approximately 111,600 days are lost due to physician office visits, ~26,000 days are lost by visits to the emergency room, and ~11,700 days are spent in hospitalized care (Table II. H.).

The model further estimates the yearly incidence of campylobacteriosis by gender, by month or season, and by age group. Table II. I. presents estimates of illness, hospitalizations, deaths for males and females. In each case, the mean and median yearly estimates are generally >25% higher for males and females, while the maximum yearly estimates for hospitalizations, deaths are similar for males and females. Table II. J provides analysis for campylobacteriosis cases

by month. For examples, illnesses, hospitalizations, and deaths are expected to be higher in June, July or August and lowest in February and November. Table II. K provides analysis by stratifying age groups. When the population is grouped into five-year intervals, the highest number of illnesses are expected in the age group of 0-4 years and age 60-65 years. While the mean number of illnesses ranged from approximately 8,000 to 30,000 for each five-year age group, the minimum and maximum estimated illnesses for each group varied widely, especially for age 0-4 years.

3.5. Economic Burden of Campylobacteriosis Including Guillain-Barré Syndrome (GBS) and Irritable Bowel Syndrome (IBS)

3.5.1. *Costs Associated with GBS and IBS Caused by Campylobacteriosis*

Further analysis of Guillain-Barré Syndrome following the onset of campylobacteriosis was presented in Table II. G. Here, the sequela is separated further to assess the burdens stemming from ventilated and non-ventilated GBS cases. Medical costs and productivity losses are analyzed, as well as death-related costs. The overall annual cost associated with non-ventilated GBS cases is estimated to be approximately \$44.6 million, and the overall annual cost associated with ventilated GBS cases is roughly \$32.7 million. Combined, the costs associated with this secondary illness, as constructed, has an annual economic burden of \$77,346,035 (median = \$60,974,952, min. = \$726,026, max. = \$1,156,000,000).

The output summary of *Campylobacter*-associated IBS was presented in Table II. H. Although cases of IBS are much more prevalent (9,501 IBS cases estimated vs. 96 GBS cases estimated), the total burden estimated is only 1.25X seen in GBS. The measured total annual cost associated with IBS is estimated to be \$96,890,826 (median = \$80,033,709, min. = \$2,845,985,

max. = \$139,897,041). The attributable annual costs seen in IBS are divided into direct (~\$61.8 million) and indirect costs (~\$35.1 million), with direct costs further separated into outpatient costs (~\$29.4 million), inpatient costs (~10.4 million), and costs associated with drug treatment (~\$13.6 million).

3.5.2. Total Costs Associated with Campylobacteriosis Due to Chicken Consumption

The total yearly economic burden for acute campylobacteriosis derived from the consumption of boneless, skinless, chicken breast filets prepared domestically in the contiguous United States is estimated to be approximately \$192 million. Table II. L. provides a detailed look into the analysis. The bulk of the costs (65.6%) are attributed with the premature deaths associated with this illness. It is estimated that \$125,855,412 (median = \$63,617,175, min. = \$676,829, max. = $\$1.69 \times 10^9$) of the total cost can be linked to premature death. The economic burden for individuals that did not visit a physician is \$23,190,223 (79,345 cases), which attributes to 12% of the cost for 73.3% of the cases. Economic burden for physician visits and hospitalizations are also captured in this analysis. In combination with the costs associated with the sequelae captured in this risk assessment, the total annual economic burden due to this system is estimated to be approximately \$366 million.

4. CONCLUSION

This QMRA provides an in-depth, comprehensive window into how a pathogen human importance such as *Campylobacter* persists and spreads throughout the manipulation steps in the

processing, packaging, storage, handling, and preparation of a common food commodity. It presents the characterization of the pathogen through the “farm to fork” model, the accompanying number of illnesses caused from a specific food commodity, as well as the overall disease burden. It quantifies the impact this pathogen has on a specific food source in great detail.

Although pathogen-reduction steps exist within this process to mitigate pathogen persistence and bacterial load, there are still ways to further improve the mitigation strategies. In the next chapter, we will delve into locales within the various modules to explore possible mitigation steps to optimize the reduction of *Campylobacter* concentration and persistence on a finished meal. This will present the opportunity to simulate how these alternative scenarios translate into reductions in campylobacteriosis and accompanying sequelae.

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Figure II. A Modules used to model the risk of campylobacteriosis: farm, processing, retail to home, consumption

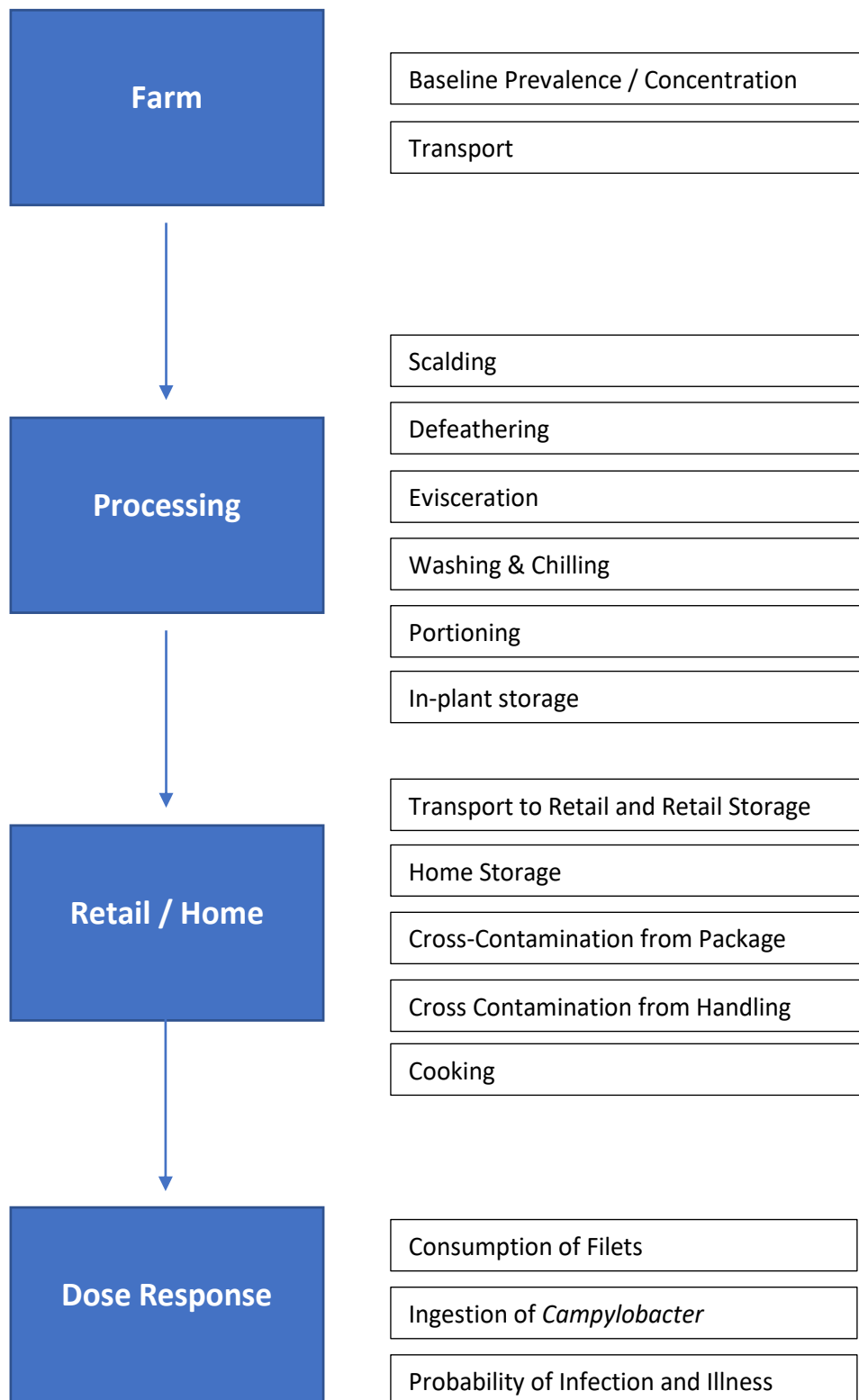


Figure II. B. Progression of *Campylobacter* prevalence throughout Processing/Handling chain

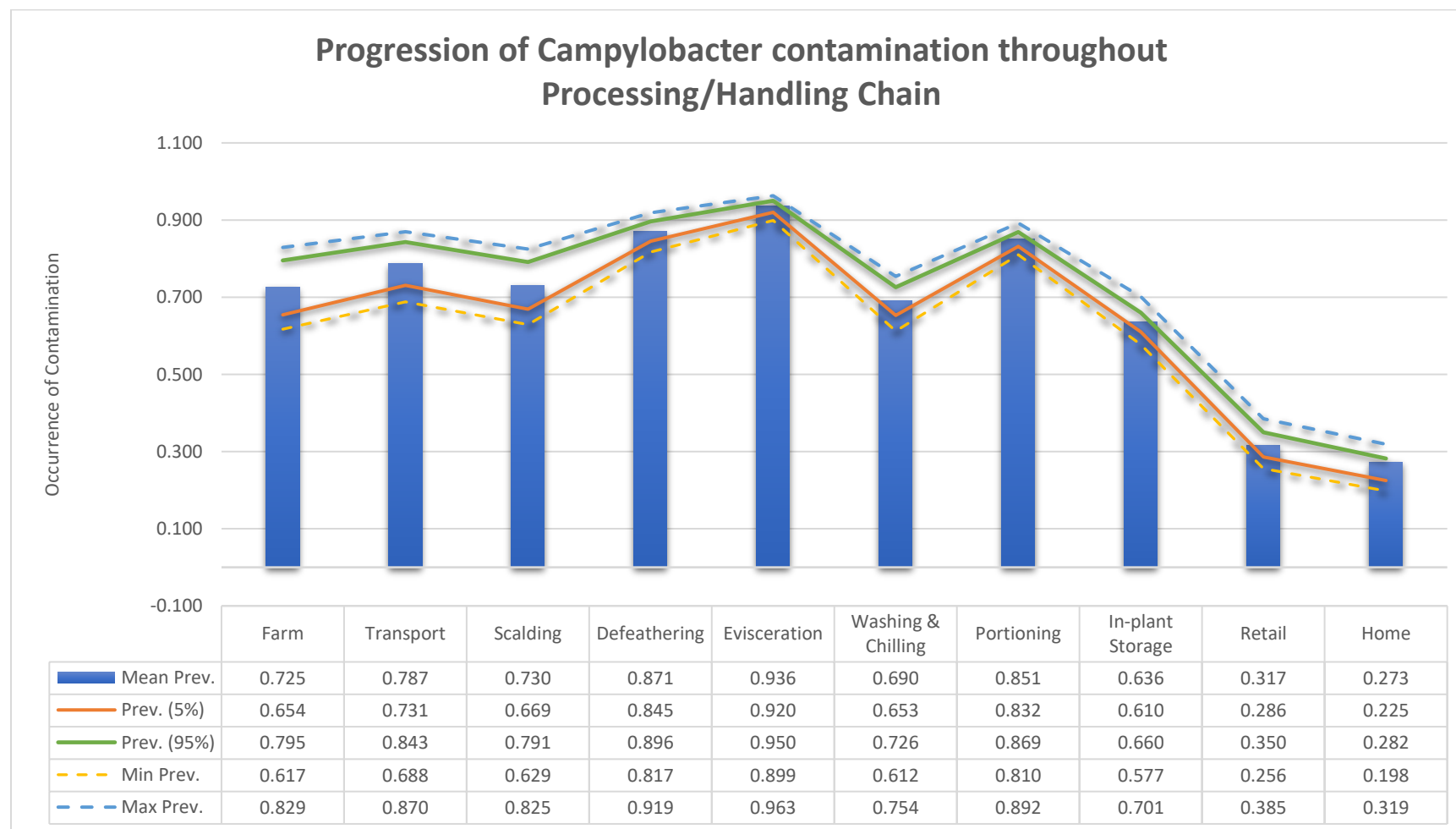


Figure II. C. Concentration of *Campylobacter* contamination throughout Processing/Handling chain

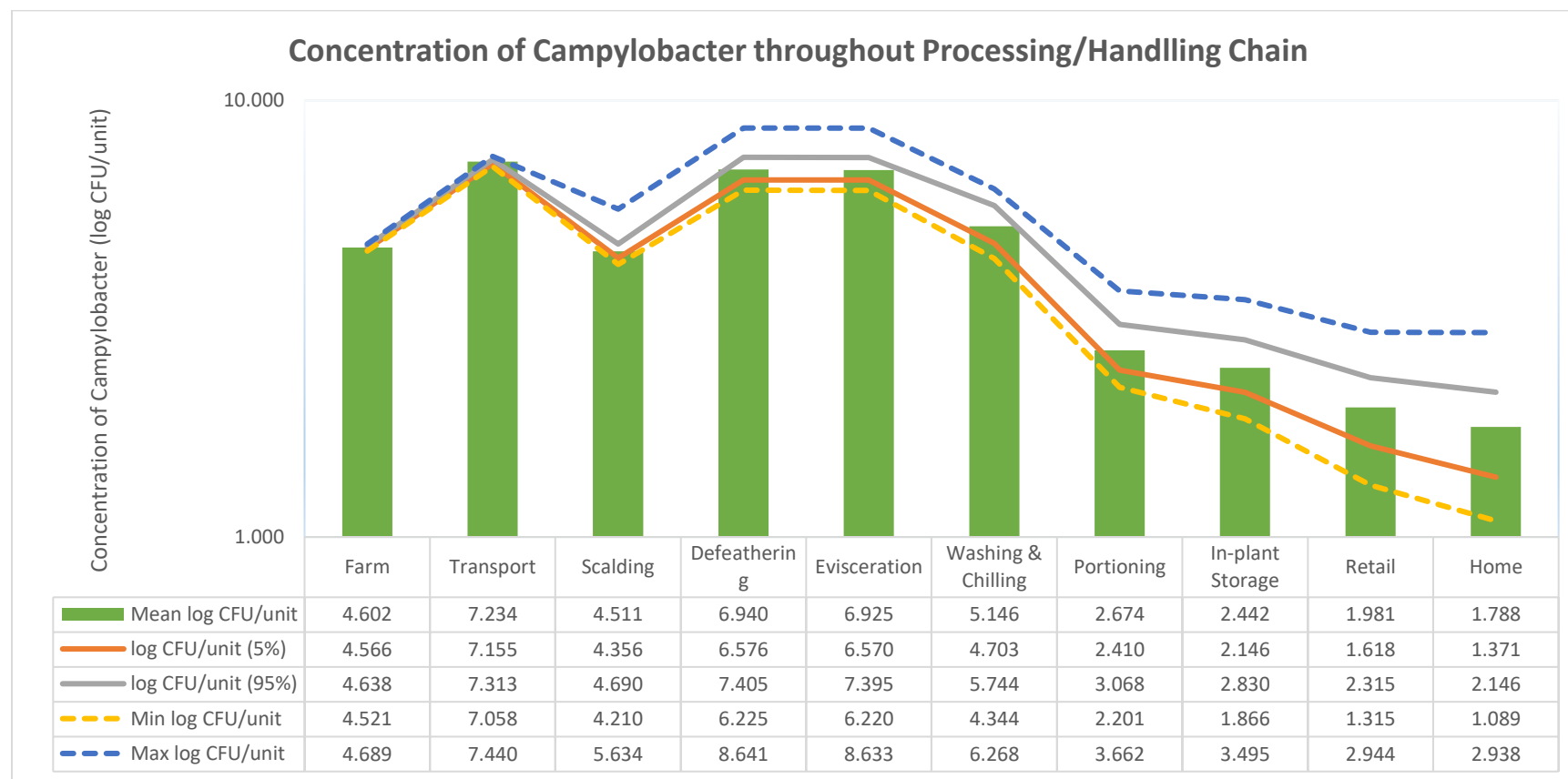


Figure II. D. Comparison of model output with other estimates of domestically acquired foodborne campylobacteriosis from boneless, skinless chicken breast fillets

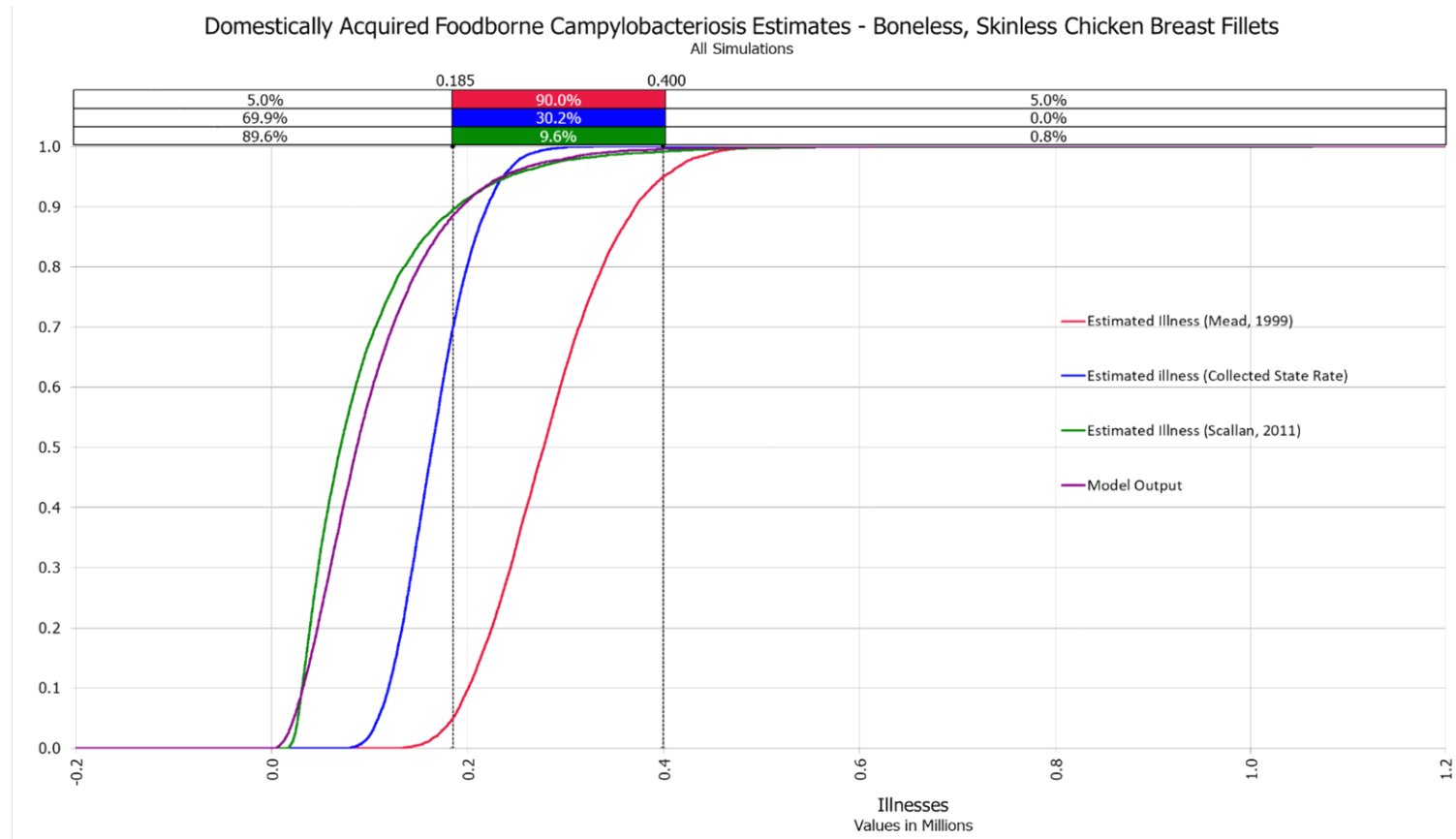


Table II. A. Model outputs for chemical usage and *Campylobacter* contamination frequency and concentration during washing and chilling of carcasses

Chemical + Washing and Chilling Descriptor	Proportion of Chemical Usage in Plant			Mean log CFU/carcass Reduction		
	Mean	Min	Max	Mean	Min	Max
Hypochlorous Acid (HAS)	0.050	0.025	0.076	1.47	1.33	1.59
Peroxyacetic acid (PA)	0.125	0.093	0.164	1.79	1.73	1.84
Acidified sodium chlorite (ASC)	0.175	0.133	0.223	2.33	2.29	2.37
Food grade acids (FGA)	0.150	0.113	0.189	1.32	1.28	1.37
Trisodium phosphate (TSP)	0.313	0.265	0.362	2.97	2.96	2.98
Chlorine Dioxide (CD)	0.088	0.060	0.118	2.41	2.31	2.49
No on-line reprocessing chemicals used (None)	0.100	0.064	0.136	1.78	1.73	1.84

Descriptor	Mean	Min	Max
Percentage of <i>Campylobacter</i> (+) Carcasses after Washing and Chilling (%)	69.00%	61.22%	75.43%
Average log CFU/carcass after Washing and Chilling Stage	5.15	4.34	6.27
Percentage of Chlorinated Water Usage (%)	77.27%	72.73%	81.55%
Occurrence of Cross-contamination during water chilling process	0.15	0.12	0.20
Additional bacterial load (log CFU/carcass) due to Cross Contamination	0.45	0.44	0.46

Table II. B. Model Outputs for *Campylobacter* contamination frequency and concentration during home storage of filets

	Number Samples			Occurrence of Contamination			Average N (s, home) CFU/filet		
	Mean	Min	Max	Mean	Min	Max	Mean	Min	Max
Bought Chilled, Consumed Day-of	580	516	646	0.367	0.268	0.461	113.23	18.77	1,720.25
Bought Chilled, Chilled	316	261	387	0.253	0.147	0.370	28.00	1.68	690.97
Bought Chilled, Frozen	158	115	205	0.107	0.006	0.217	4..47	0.019	393.42
Bought Frozen, Consumed Day-of	43	21	73	0.014	0	0.139	0.118	0	72.12
Bought Frozen, Chilled	51	29	81	0.0012	0	0.013	7.3E-03	0	2.07
Bought Frozen, Frozen	77	49	109	5.5E-05	0	7.9E-03	1.4E-04	0	0.10

Occurrence of contaminated filets at home: Mean: 0.253; Median = 0.2534, Min: 0.198; Max: 0.319

Overall average N CFU/filet at home storage: Mean: 61.40; Median = 48.02, Min: 12.28; Max: 867.91

Table II. C. Home handling (cross-contamination) output

Cross-contamination Descriptor	Occurrence of Meat → Hands cross-contamination			Average CFU transferred if contaminated		
	Mean	Min	Max	Mean	Min	Max
Meat → Hands (No Wash) Sampled	102	51	172	3.23	0	1,508
Meat → Hands (Rinse) Sampled	714	527	863	3.27	0	4,670
Meat → Hands (Wash) Sampled	307	191	451	0.0169	0	21
Meat → Hands (No Contact) Sampled	102	48	169	0	0	0
All Meat → Hands Sampled	1,225			2.14	0	2,714

Cross-contamination Descriptor	Occurrence of Meat → Board cross-contamination			Average CFU transferred if contaminated		
	Mean	Min	Max	Mean	Min	Max
Meat → Board (No Wash) Sampled	154	77	230	3.09	0	811
Meat → Board (Rinse, Cold) Sampled	103	55	159	25.96	0	4,574
Meat → Board (Rinse, Hot) Sampled	102	51	160	0.00160	0	3
Meat → Board (Wash) Sampled	52	21	91	0	0	0
Meat → Board (No Contact) Sampled	815	667	948	0	0	0
All Meat → Board Sampled	1,225			2.56	0	373

Cross-contamination Descriptor	Occurrence of Meat → Utensils cross-contamination			Average CFU transferred if contaminated		
	Mean	Min	Max	Mean	Min	Max
Meat → Utensils (No Wash) Sampled	254	202	314	2.94	0	290
Meat → Utensils (Rinse, Cold) Sampled	77	48	107	0.0598	0	74
Meat → Utensils (Rinse, Hot) Sampled	77	47	110	0.00210	0	3
Meat → Utensils (Wash) Sampled	2	0	9	0	0	0
Meat → Utensils (No Contact) Sampled	817	743	878	0	0	0
All Meat → Utensils Sampled	1,225			0.494	0	59

Descriptor	Mean	Min	Max
Average CFU Transferred to Meal from All Handlings	5.41	0.238	2,715
Average CFU Transferred to Meal if Contaminated via Handling	84.14	4	43,769
Average CFU Ingested in Meal from All Handlings	1.19	0	1,514
Average CFU Ingested in Meal if Contaminated via Handling	50.48	1	59,808

P(cross-contamination) of Handling Filets at Home: Mean: 0.0261; Min: 0.00980; Max: 0.0.0465

Table II. D. Probability of infection and illness per dose or per CFU ingested

Probability of infection and illness per dose ingested

		Mean	Median	Min	Max
<i>Probability of Infection</i>	<i>from calculated $P_{consumption}$</i>	2.882×10^{-5}	2.602×10^{-5}	1.721×10^{-6}	0.00012
	<i>from observed $P_{consumption}$</i>	2.681×10^{-5}	2.429×10^{-5}	1.726×10^{-6}	0.00011
<i>Probability of Illness</i>	<i>from calculated $P_{consumption}$</i>	9.496×10^{-6}	8.547×10^{-6}	5.004×10^{-7}	4.188×10^{-5}
	<i>from observed $P_{consumption}$</i>	8.833×10^{-6}	7.983×10^{-6}	5.019×10^{-7}	3.875×10^{-5}

Probability of infection and illness per CFU ingested

		Mean	Median	Min	Max
<i>Probability of Infection</i>	<i>from calculated $P_{consumption}$</i>	2.873×10^{-5}	2.344×10^{-5}	4.480×10^{-7}	0.00023
	<i>from observed $P_{consumption}$</i>	2.673×10^{-5}	2.190×10^{-5}	4.247×10^{-6}	0.00020
<i>Probability of Illness</i>	<i>from calculated $P_{consumption}$</i>	9.468×10^{-6}	7.671×10^{-6}	1.554×10^{-7}	8.031×10^{-5}
	<i>from observed $P_{consumption}$</i>	8.808×10^{-6}	7.178×10^{-6}	1.474×10^{-7}	6.904×10^{-5}

Table II. E. Model output summary of annual illnesses, hospitalizations, deaths due to acute campylobacteriosis in the contiguous United States

Yearly Illnesses

Model Output Descriptor	Mean	Median	Min	Max
$P_{ill}(calc) - \text{per dose ingested } N_{ill} / \text{yr}$	112,558	99,407	6,348	580,165
$P_{ill}(observed) - \text{per dose ingested } N_{ill} / \text{yr}$	104,702	92,864	6,104	536,749
$P_{ill}(calc) - \text{per CFU ingested } N_{ill} / \text{yr}$	112,248	88,749	1,468	1,111,933
$P_{ill}(observed) - \text{per CFU ingested } N_{ill} / \text{yr}$	104,424	83,062	1,392	955,872

Yearly Hospitalizations

Model Output Descriptor	Mean	Median	Min	Max
$P_{ill}(calc) - \text{per dose ingested } N_{ill} / \text{yr}$	868	754	54	5,069
$P_{ill}(observed) - \text{per dose ingested } N_{ill} / \text{yr}$	807	705	42	4,007
$P_{ill}(calc) - \text{per CFU ingested } N_{ill} / \text{yr}$	866	676	12	7,955
$P_{ill}(observed) - \text{per CFU ingested } N_{ill} / \text{yr}$	806	632	10	8,435

Yearly Deaths

Model Output Descriptor	Mean	Median	Min	Max
$P_{ill}(calc) - \text{per dose ingested } N_{ill} / \text{yr}$	39	33	1	239
$P_{ill}(observed) - \text{per dose ingested } N_{ill} / \text{yr}$	36	31	2	200
$P_{ill}(calc) - \text{per CFU ingested } N_{ill} / \text{yr}$	39	30	0	452
$P_{ill}(observed) - \text{per CFU ingested } N_{ill} / \text{yr}$	36	28	0	408

Combined Output Distribution

Model Output Descriptor	Mean	Median	Min	Max
Yearly Infections	328,257	278,211	7,268	2,163,872
Yearly Illnesses	108,174	91,244	2,481	653,318
Did not Visit Physician, Survived	79,345	66,927	1,820	479,202
Visited Physician, Survived	27,754	23,410	637	167,620
Yearly Hospitalizations	834	690	15	5,587
Yearly Deaths	37	31	1	240

Table II. F. Model output summary of annual sequelae (GBS and IBS) following acute campylobacteriosis in the contiguous United States

Yearly Campylobacter-associated GBS

Model Output Descriptor	Mean	Median	Min	Max
$P_{iii}(\text{calc})$ – per dose ingested N_{iii} / yr	99	84	4	841
$P_{iii}(\text{observed})$ – per dose ingested N_{iii} / yr	92	78	3	674
$P_{iii}(\text{calc})$ – per CFU ingested N_{iii} / yr	100	74	1	1,392
$P_{iii}(\text{observed})$ – per CFU ingested N_{iii} / yr	92	42	1	1,049

Yearly Campylobacter-associated IBS

Model Output Descriptor	Mean	Median	Min	Max
$P_{iii}(\text{calc})$ – per dose ingested N_{iii} / yr	9,887	8,728	557	51,609
$P_{iii}(\text{observed})$ – per dose ingested N_{iii} / yr	9,196	8,146	533	46,863
$P_{iii}(\text{calc})$ – per CFU ingested N_{iii} / yr	9,859	7,796	128	98,610
$P_{iii}(\text{observed})$ – per CFU ingested N_{iii} / yr	9,173	7,301	122	85,153

Combined Output Distribution

Yearly Campylobacter-associated GBS	96	77	1	1,373
Yearly Campylobacter-associated IBS	9,501	8,026	217	56,424

Table II. G. Output summary of yearly incidence and costs of Guillain-Barré Syndrome (GBS) caused by campylobacteriosis

Descriptor	Mean	Median	Minimum	Maximum
Non-Ventilated Patients	72	57	1	1,003
Non-Ventilated Patients, Resumed Work	58	46	1	812
Non-Ventilated Patients, Cannot Work	14	11	0	191
Medical Costs, Non-Ventilated	\$8,587,729	\$6,804,402	\$119,375	\$119,733,606
Productivity Losses, Non-Ventilated, Resumed Work	\$342,620	\$273,617	\$4,451	\$5,245,774
Productivity Losses, Non-Ventilated, Cannot Work	\$35,755,833	\$27,528,413	\$393,507	\$562,578,944
Annual Loss, Non-Ventilated	\$44,686,182	\$34,865,108	\$517,333	\$687,558,324
Ventilated Patients	24	20	0	370
Ventilated Patients, Resumed Work	15	12	0	233
Ventilated Patients, Cannot Work	6	5	0	98
Ventilated Patients, Died	3	2	0	39
Medical Costs, Ventilated	\$14,912,661	\$11,790,529	\$0	\$229,605,039
Productivity Losses, Ventilated, Resumed Work	\$342,942	\$272,906	\$4,174	\$4,941,200
Productivity Losses, Ventilated, Cannot Work	\$12,450,187	\$9,668,007	\$149,016	\$151,448,535
Productivity Losses, Ventilated, Died	\$4,954,062	\$4,954,062	\$55,503	\$81,995,362
Annual Loss, Ventilated	\$32,659,852	\$25,961,793	\$208,693	\$467,990,136
Total Costs	\$77,346,035	\$60,974,952	\$726,026	\$1,156,000,000

Table II. H. Output summary of yearly incidence and costs of Irritable Bowel Syndrome (IBS) caused by campylobacteriosis

Descriptor	Mean	Median	Minimum	Maximum
Estimated Annual Number of IBS Patients due to Campylobacteriosis from Boneless Skinless Domestic Chicken Consumption	9,501	8,026	217	56,424
Direct Costs	\$61,755,289	\$50,174,337	\$1,496,101	\$506,698,717
Indirect Costs	\$35,135,527	\$27,280,465	\$731,515	\$297,226,936
Total	\$96,890,816	\$80,033,709	\$2,845,985	\$656,201,691
Distribution of Health Care Costs, Direct - Outpatient Costs, IBS	\$29,426,686	\$23,276,118	\$386,634	\$281,583,988
Distribution of Health Care Costs, Direct - Inpatient Costs, IBS	\$10,412,415	\$7,768,671	\$186,779	\$126,455,208
Distribution of Health Care Costs, Direct - Pharmacy or Drug Costs, IBS	\$13,587,766	\$10,292,120	\$256,786	\$139,897,041
Physician Office Visits, Number of Days/Year, IBS-D	111,638.69	79,654.77	8.01	1,350,364.26
Emergency Room Visits, Number of Days/Year, IBS-D	26,284.33	18,293.59	2.53	260,2146.98
Hospitalizations, Number of Days/Year, IBS-D	11,722.50	8,933.31	6.59	129,763.30

Table II. I. Output summary of yearly incidence of campylobacteriosis by gender

Descriptor	Mean (Male / Female)	Median (Male / Female)	Minimum (Male / Female)	Maximum (Male / Female)
Yearly Illnesses	59,722 / 48,453	50,226 / 40,857	1,285 / 1,196	343,504 / 317,216
Yearly Hospitalizations	460 / 373	382 / 307	8 / 7	3,096 / 2,652
Yearly Deaths	34 / 27	28 / 22	2 / 2	247 / 259

Table II. J. Output summary of yearly incidence of acute campylobacteriosis by month

Months	Mean Seasonal Distribution (90% CI, $\pm$)	Mean Illnesses (90% CI, $\pm$)	Mean Hospitalizations (90% CI, $\pm$)	Mean Deaths (90% CI, $\pm$)
January	6.48% (0.016%)	7,011 (80.27)	54 (0.65)	2.42 (0.030)
February	5.18% (0.018%)	5,624 (67.14)	43 (0.54)	1.94 (0.025)
March	5.75% (0.013%)	6,217 (71.17)	48 (0.58)	2.14 (0.027)
April	6.43% (0.016%)	6,955 (80.20)	54 (0.65)	2.40 (0.030)
May	8.15% (0.025%)	8,810 (103.64)	68 (0.84)	3.04 (0.039)
June	10.17% (0.028%)	11,015 (127.96)	85 (1.03)	3.81 (0.048)
July	12.48% (0.031%)	13,489 (154.78)	104 (1.25)	4.66 (0.057)
August	11.83% (8.74 x 10 ⁻⁵ %)	12,801 (142.92)	99 (1.16)	4.41 (0.053)
September	9.22% (0.024%)	9,954 (113.83)	77 (0.92)	3.43 (0.043)
October	9.16% (0.015%)	9,904 (111.56)	76 (0.90)	3.41 (0.042)
November	6.54% (0.016%)	7,076 (81.51)	55 (0.66)	2.45 (0.031)
December	8.17% (0.035%)	8,850 (108.71)	68 (0.88)	3.06 (0.040)

Table II. K. Output summary of yearly incidence of campylobacteriosis by age distribution

Months	Mean Seasonal Distribution (90% CI, $\pm$)	Mean Illnesses (90% CI, $\pm$)	Mean Deaths (90% CI, $\pm$)
0 – 4 years	12.46% (0.24%)	15,942 (244.91)	5.50 (0.090)
5 – 9 years	5.59% (0.066%)	6,438 (83.56)	2.22 (0.031)
5 – 14 years	9.87% (0.058%)	11,242 (130.50)	3.89 (0.049)
10 – 14 years	4.45% (0.021%)	4,923 (56.24)	1.69 (0.021)
15 – 19 years	6.65% (0.044%)	6,722 (80.87)	2.32 (0.030)
15 – 24 years	11.82% (0.087%)	12,732 (152.66)	4.40 (0.056)
20 – 29 years	11.92% (0.035%)	12,929 (145.11)	4.46 (0.054)
30 – 39 years	12.09% (0.050%)	12,729 (145.61)	4.39 (0.054)
40 – 49 years	13.41% (0.067%)	14,522 (167.35)	5.01 (0.062)
50 – 59 years	13.03% (0.087%)	13,759 (164.41)	4.74 (0.061)
60+ years	16.87% (0.091%)	18,193 (210.61)	6.28 (0.078)
65+ years	11.06% (0.037%)	11,872 (135.01)	4.10 (0.050)

Table II. L. Output summary of yearly economic burden of acute campylobacteriosis

Descriptor	Mean	Median	Minimum	Maximum
Did not Visit Physician, Survived	\$23,190,223	\$12,599,803	\$204,741	\$253,190,223
Visited Physician, Survived	\$32,142,257	\$23,458,095	\$469,229	\$275,417,510
Hospitalized, non-sequelae, Survived	\$10,639,626	\$8,426,920	\$148,751	\$80,720,120
Premature Deaths	\$125,855,412	\$63,617,175	\$676,829	\$1.69 x 10 ⁹
Total	\$191,834,441	\$147,421,293	\$4,151,505	\$1.86 x 10 ⁹

CHAPTER III – Analysis of Simulations of Alternative Scenarios with the Baseline Quantitative Microbial Risk Assessment (QMRA) Model to Reduce Campylobacteriosis from Home Chicken Preparation and Consumption

ABSTRACT

Chapter II established the “baseline model” in describing in the annual burdens associated with the production, preparation, and consumption of a boneless chicken breast portion in the domestic setting contaminated with *Campylobacter* in the United States. This chapter describes the effects of modifying specific modules along the QMRA chain.

Analyses were performed on modifying the baseline model within the farm-to-fork process. Two scenarios represented the effects of changing either the level of *Campylobacter* prevalence or *Campylobacter* concentrations on the bird prior to processing, one analyzed the impact transport crate washing, one examined the impact of storage conditions post processing/ pre-retail, and an alternative scenario model estimated the impact of handwashing in the preparation and handling of a meal. A combination *Campylobacter* mitigation model was also developed using the inputs from reducing concentration of *Campylobacter* on the bird prior to transport to the abattoir and the effects of increasing the proportion of frozen filets held post processing/ pre-retail section of the modeling process.

The effects of these interventions varied depending on the location and intensity of the mitigation steps. The mean yearly illnesses are estimated to decrease from 103,989 to 57,631 if on-farm *Campylobacter* prevalence was lowered to 35-50% from the baseline. An additional ~50,000 illnesses is expected if the non-washing in the preparation of the meal increases from 8.3% (baseline) to 20%, and it was established the combined effects of reducing on-farm *Campylobacter* concentrations and increasing in-plant freezing proportion have greater impacts on reducing yearly campylobacteriosis and associated burdens than either intervention alone.

1. INTRODUCTION

In Chapter II, numerous stages of chicken processing and preparation were evaluated for their effect on changing the prevalence and concentration of *Campylobacter* from the live bird to the final prepared and consumed chicken portion. Many of the process and preparation steps can cause and increase or decrease in the *Campylobacter* prevalence or concentration (or both) for the chicken carcass and ultimately a boneless chicken breast portion. Any of these process or preparation steps could be adjusted or modified in a way that could result in reduced *Campylobacter* prevalence, reduced *Campylobacter* concentrations, and possibly reduced cases of campylobacteriosis. Sensitivity analysis, which can determine the importance of model parameters with significant impact on the output was performed on the baseline model. Within this analysis, 16 sensitive inputs/data gaps representing potential areas in input that significantly affect the uncertainty around the annual illness mean output were identified. The tornado graph is presented in Figure III. A. Sensitivity analysis may be considered for food safety interventions.

This chapter describes the effect that selected interventions or scenarios could have on reducing *Campylobacter* prevalence, concentrations, cases of campylobacteriosis and associated public health costs. The scenarios presented in this chapter utilize the Quantitative Risk Assessment (QMRA) model as described in Chapter II – also referred in this chapter as the “baseline model.” Modifications were implemented to the baseline model to assay various stepwise alterations within the farm-to-fork process model. The alternative scenarios were developed to represent a broad range within the QMRA system. The effects of modifying he

baseline model's inputs within the farm-to-fork process were studied as follows: Post grow-out (1) prevalence and (2) concentration of *Campylobacter* on chickens at the farm prior to transport, 3) transport crate cleaning frequency prior to loading, 4) the storage conditions post processing/ pre-retail, and 5) the frequency of handwashing in the preparation and handling of a chicken meal. Lastly, a combination *Campylobacter* mitigation model was developed using the inputs from a post grow-out intervention model (reducing concentration of *Campylobacter* on the bird prior to transport to the abattoir) and the post processing/ pre-retail model (increase proportion frozen).

Wassenaar, et al (Wassenaar 2011) described three levels of interventions that have been considered by the poultry industry: (1) reducing the numbers of flocks colonized by *Campylobacter*; (2) reducing the bacterial load on and in the birds prior to slaughter from positive to negative birds; and (3) to reserve contaminated meat for frozen products, which can reduce the number of viable organisms. The alternative scenarios represented in this chapter were developed independent of this journal article but practicality in application and a baseline sensitivity analysis were considered.

2. MATERIALS AND METHODS

Five scenarios were selected where the prevalence and/or concentration of *Campylobacter* could be varied and evaluated. Each alternative scenario model was developed using five simulations with stepwise modifications to the baseline level distributions. All

simulations were run for 10,000 iterations using Latin Hypercube Monte Carlo analysis. The alternative scenarios included the following:

- 1) Reduce *Campylobacter* Prevalence at Farm Grow-out, Pre-transport
- 2) Reduce *Campylobacter* Concentrations at Farm Grow-out, Pre-Transport
- 3) Transport Compliance – Cleaning and Sanitizing Transport Crates
- 4) Increase Proportion Frozen during In-Plant Storage
- 5) Cross-Contamination from Changes in Handwashing during Preparation

A sixth scenario evaluated a combination of scenario 2) and 4) above (Reduce On-farm *Campylobacter* Concentrations and Increase In-Plant Freezing).

2.1. Reduce *Campylobacter* Prevalence at Farm Grow-out, Pre-transport

For this phase of the scenario, we investigated the impact of the initial prevalence/occurrence of *Campylobacter*-positive birds on the overall system. Concurrently, the initial concentration is integrated with the level of contamination seen at the post grow-out stage, where the concentration level is directly tied in the overall decrease (or increase) seen for the birds. The default setting for the occurrence of *Campylobacter*-positive birds at the time of post grow-out is a Uniform distribution of 65% - 80%. The dialog box on probability on Prevalence (position C4) is modified at each simulation to reflect the distribution range set for the simulations tested. For this phase, the estimated *Campylobacter* prevalence drops in each scenario interval by approximately 15%. Even though the lower prevalence levels may be very difficult to achieve, it is important to study if reduced prevalence could lead to significant improvements in public health impacts. The alternative scenario process distributions are designed in the following manner:

On-farm prevalence of *Campylobacter* on bird exterior (cell C4), pre-transport:

- Scenario 1, Baseline – Uniform (0.65,0.80)
- Scenario 2 – Uniform (0.50,0.65)
- Scenario 3 – Uniform (0.35,0.50)
- Scenario 4 – Uniform (0.20,0.35)
- Scenario 5 – Uniform (0.05,0.20)

2.2. Reduce *Campylobacter* Concentrations at Farm Grow-out, Pre-Transport

We looked at the impact of modifying the concentration of *Campylobacter* on the pathogen-specific birds at the farm, prior to transport to the processing plant. The occurrence of contamination in this model was kept constant at the baseline for this alternative scenario QMRA model. We used five simulations as the structure of the model, with scenario 1 representing the baseline value, and each subsequent scenario uniformly distributed stepwise around a one log interval as follows:

Concentration of *Campylobacter*, individual birds on farm (pre-transport)

- Five simulations (linked to *SIMTABLE_Processing*, designation 10,20,30,40,50)
- Position row 16 (C(ext), Carcass (process) log CFU/carcass)
- Concentration of *Campylobacter* at the farm level
 - Scenario 1, Baseline – $0.435 * \text{LN}(\text{Default_Prevalence}) + \text{RiskWeibull}(12.543, 7.0643, \text{RiskShift}(-2.5494), \text{RiskTruncate}(1, 8))$
 - Scenario 2 – Uniform(3.5,4.5)
 - Scenario 3 – Uniform(2.5,3.5)
 - Scenario 4 – Uniform(1.5,2.5)
 - Scenario 5 – Uniform(0.5,1.5)

2.3. Transport Compliance – Cleaning and Sanitizing Transport Crates

Washing and sanitizing of crates reduces the cross-contamination levels seen in the birds transported, especially in those birds not harboring any *Campylobacter* at the farm level (Slader 2002, Rasschaert, Houf et al. 2007). Nationally, 28.4% of transport crates used in moving live broilers from the grow-out house of the farm to the processing plant are washed and sanitized between each use (Northcutt and Jones 2004). In this scenario, we analyze the effect

on the level of contamination, the concentration of *Campylobacter* found, as well the effects in the overall illness incidence as compliance for crate washing is increased in a stepwise progression. Transport crate washing and sanitizing compliance were set using five simulations with 10,000 iterations per simulation. They were stratified as described, using the values set in the dialog box on Probability on Compliance (E,F38: Compliance): 1) Baseline compliance level of 28.4%, 2) an increase in washing and sanitizing using a Uniform distribution of (30 – 40%), 3) an increase in washing and sanitizing using a Uniform distribution of (40 – 60%), 4) an increase in washing and sanitizing using a Uniform distribution of (70 – 80%), and the final simulation set at (90 – 100%) of the transport crates washed and sanitized prior to loading of the broilers at the farm.

- Five simulations (linked to *SIMTABLE_Processing*, designation 10,20,30,40,50)
- Dialog Box on Probability (E38, F38; Compliance)
- Compliance to Cleaning and Sanitizing of Crates at the Transport Level
 - Scenario 1, Baseline – (0.284)
 - Scenario 2 – Uniform(0.35,0.45)
 - Scenario 3 – Uniform(0.55,0.65)
 - Scenario 4 – Uniform(0.75,0.85)
 - Scenario 5 – Uniform(0.90,1.0)

2.4. Increase Proportion Frozen during In-Plant Storage

As noted in Chapter I and Chapter II, freezing is a proven, effective measure to reduce and control the number of *Campylobacter* cells on the surface of the food commodity (Moorhead 2002, Zhao 2003). In Nordic countries such as Iceland, Norway, and Denmark, the carcasses of *Campylobacter*-positive flocks are introduced to freezing in processing to further reduce the risk of human campylobacteriosis (Sandberg, Hofshagen et al. 2005, Baumgartner

and Felleisen 2011, Al-Sakkaf and Jones 2012). This method has proven to be effective when processing of known contaminated flocks are diverted to be finished as frozen carcasses (Stern and Robach 2003, Sandberg, Hofshagen et al. 2005). Deep freezing of broilers to reduce the number of cases of campylobacteriosis has proven to be a valid risk management measure. Baumgartner et al. (Baumgartner and Felleisen 2011) reported that prior to 1996, all chicken meat sold in Iceland was regulated to be sold frozen; after abrogation of this requirement, the incidence of campylobacteriosis increased significantly within three years.

Although freezing may be a viable step in mitigating the level of contamination of *Campylobacter* spp. in chicken products, a fraction of the *C. jejuni* population does survive the process or are sub-lethally injured (Georgsson 2006, Sampers, Habib et al. 2010). Studies by Zhao et al. (Zhao 2003) and Moorhead et al. (Moorhead 2002) reported that short-term storage of poultry at freezing temperatures (-18 to -20°C) reduced the number of *Campylobacter* cells markedly. The survival of *C. jejuni* on chicken wings and beef trimmings during freezing have reported reductions of approximately 1.2 and 1.5- log CFU/g, respectively, after 24 hours at freezing temperatures. Other experiments with various meat matrices have demonstrated that detectable reductions of about 1 to 3 logs CFU is manifest within the first few days of frozen storage and maximum levels of reduction are generally obtained within the first two weeks of freezing, followed by a relatively constant viability thereafter (Moorhead 2002, Sandberg, Hofshagen et al. 2005, Birk, Rosenquist et al. 2006, Sampers, Habib et al. 2010). For this scenario, five simulations were carried out where scenario 1 represented a baseline value of 13.8% of chickens are frozen, and each subsequent scenario level has increasingly larger proportions of frozen product as follows:

- Five simulations (linked to *SIMTABLE_Processing*, designation 10,20,30,40,50)
- Position F180, Processing, =@RiskSimtable({9,19,29,39,49})
- Dialog Box on Probability (Frozen/Chilled)
- Chilled = reciprocal of freezing probability
- Freezing
 - Scenario 1, Baseline – 0.138
 - Scenario 2 – Uniform(0.15,0.35)
 - Scenario 3 – Uniform(0.35,0.55)
 - Scenario 4 – Uniform(0.55,0.75)
 - Scenario 5 – Uniform(0.75,1.0)

2.5. Cross-Contamination from Changes in Handwashing during Preparation

Departing from examining alternative scenarios as possible mitigation steps in controlling *Campylobacter*, this section examined the ramifications in campylobacteriosis when there is a breakdown in the control process. The following simulations are designed to estimate the effects of a stepwise increase in the non-washing of hands in the preparation and handling of boneless, skinless chicken breast filets that are prepared in the home. Although non-washing of hands is the main factor for analysis, correlating actions tied to the preparation and handling are also affected. In the baseline level, no washing events occurred 8.3% of the time (Rinsing 58.3%, washing with soap 25%, and no contact 8.4%). The scenarios simulated are as outlined below:

- Alternative Scenario on (Meat + Hands)
- Five simulations (linked to *Simtable_Retail_Consumer*, designation 9, 19, 29, 39, 49)
- Position C4, Handling-→Consumption, =@RiskSimtable({9,19,29,39,49})
- Increase in unhygienic practice (no-wash)
 - No Wash
 - Scenario 1, Baseline – Normal (0.083,0.0083)
 - Scenario 2 – 0.2
 - Scenario 3 – 0.4
 - Scenario 4 – 0.6

- Scenario 5 – 0.8
- Rinse
 - Scenario 1, Baseline – Normal (0.583,0.0583)
 - Scenario 2 – 0.5
 - Scenario 3 – 0.4
 - Scenario 4 – 0.2
 - Scenario 5 – 0.1
- Wash w. Soap
 - Scenario 1, Baseline – Normal(0.25,0.025)
 - Scenario 2 – 0.217
 - Scenario 3 – 0.117
 - Scenario 4 – 0.117
 - Scenario 5 – 0.017
- No contact
 - Scenario 1, Baseline – Normal (0.084,0.0084)
 - Scenario 2 – 0.083
 - Scenario 3 – 0.083
 - Scenario 4 – 0.083
 - Scenario 5 – 0.083

2.6. Combination of Interventions – Reduce On-farm *Campylobacter* Concentration and Increase In-Plant Freezing

This section describes some of the combination of individual interventions previously discussed in the chapter. Wagenaar et al. (Wagenaar, French et al. 2013) posits that multi-level approaches should be pursued, aiming at reducing the level of *Campylobacter* contamination of food products. The choices in selecting the possible synergistic effects in the combination of two analyzed interventions were based on applicability into the risk assessment model as well as the feasibility of implementation in real-life settings. The first combination process modelled was the reduction of post grow-out *Campylobacter* concentrations at the farm (see section 2.2) and increasing the proportion of frozen vs. chilled finished and packaged boneless skinless chicken breasts at the in-process storage (prior to retail) (see section 2.4).

The alternative scenario input structures are the same as the individual alternative scenarios as described previously in the chapter. The model was modified as little as possible to mitigate the sources of possible confoundment in the effects seen. Both modifiers are linked to the simulation controller *SIMTABLE_Processing*, designation 10,20,30,40,50 in the “Processing” worksheet. Again, simulation 5 represents the most extreme intervention steps (reduce *Campylobacter* concentrations to ~0.5 – 1.5 log CFU/carcass and 75 – 100% of chicken will be frozen in-plant) and it is expected that any modification would perform between this simulation and the baseline (~ 4.60 log CFU *Campylobacter*/carcass and 13.8% of chicken frozen in-plant).

3. RESULTS AND DISCUSSION

3.1. Reduce *Campylobacter* Prevalence at Farm Grow-out, Pre-transport

Table III. 1A. represents the prevalence output of the five simulations. The changes in magnitude from the simulations range from 1.261 (scenario 2) to 5.8 (scenario 5) decrease from the baseline mean. The average external concentration of *Campylobacter* (4.60 log CFU/bird) does not change for any scenario (Table III. 1B.).

There is a gradual stepwise downward shift shown in each simulation through the preparation in the home, for the occurrence of contamination present as well as the mean *Campylobacter* concentration (Tables III. 1C. – III. 1J.). For example, from Tables III. 1E. and 1F., the proportion of positive fillets during in-plant storage decreases from a mean of 63.6% (baseline) to a mean of 52.0% (scenario level 5). At this stage, the mean *Campylobacter* concentration decreases from 2.44 log CFU/filet (baseline) to 2.12 log CFU/filet (scenario level

5). The decrease in change of magnitude for the probability of ingestion (Table III. 1K.) mirrors that found in the occurrence of contaminated filets at home (Table III. 1G.).

Tables III. 1L. – 1O. exhibit the reduction in illnesses, hospitalizations, deaths and total yearly costs of acute cases of campylobacteriosis due to the reduction of *Campylobacter* (+) birds at the post grow-out / pre-processing stage in the QMRA model. The intervention steps focusing on reducing the occurrence of *Campylobacter*-positive birds at the farm/pre-transport phase of the process presented in this model demonstrated a steady, substantial decrease throughout the processing, retail, and home pathways. Overall, as seen in Table III. 1A., a decrease in prevalence from a mean of 0.725 (baseline) to 0.575 (scenario 2) results in approximately a 20,000 annual case reduction of campylobacteriosis in the contiguous United States from ingesting boneless, skinless chicken breast meals prepared in the home (Table III. 1L.). This reduction in illnesses translates into a reduction in annual costs attributed to acute campylobacteriosis of ~ \$50 million (Table III. 1O.). Additionally, the changes in magnitude from baseline for simulations 2 to 5 were consistent for estimates of annual illnesses, as well as disease burden due to sequelae. There is nearly a two-fold change seen for simulation 2 and nearly a three-fold reduction for simulation 4. Simulation 5, which represents the extreme end of the mitigation process, exhibits nearly a seven-fold reduction from the baseline mean. For example, mean yearly illnesses may decrease from 103,989 to 57,631 if on-farm *Campylobacter* prevalence was 35-50%, and further decrease to 15,017 if on-farm prevalence was 5-20% (Table III. 1L.).

3.2. Reduce *Campylobacter* Concentrations at Farm Grow-out, Pre-Transport

The change in magnitude for the contamination of carcasses at the farm follows a logarithmic scale for the scenarios analyzed, as shown in Table III. 2A. Comparing scenario 2 to 5, there is a ~4.5-fold to ~4500-fold decrease in average concentration seen vs. the baseline (~4.6 log CFU/bird). As processed carcasses move through processing both the prevalence and concentrations of *Campylobacter* decrease in each scenario. For example, after washing and chilling, mean prevalence of *Campylobacter* decreases from 69.0 to 40.0% and mean concentrations decrease from 5.15 log CFU/carcass to 1.97 log CFU/carcass when the baseline scenario is compared with the scenario 5 outputs (Tables III. 2C. and 2D.). During retail storage (Tables III. 2E. and 2F.), the occurrence of *Campylobacter* drops from 31.4 to 27.3% and the mean number of CFU/filet decreases from 93.6 to 32.7 CFU/filet when mean on-farm concentrations are reduced from 4.60 to 3.95 log CFU/carcass (scenario level 2).

The probability of ingesting a *Campylobacter*-positive chicken breast meal falls from ~3% (Table III. 2G.) to ~2%, ~0.8%, ~0.3%, and ~0.1% in the respective simulations. This reduction in probability directly correlates to the burdens seen in the estimations by the model. The values obtained in the yearly illnesses, hospitalizations, deaths, and sequelae from campylobacteriosis as well as the annual (acute) values of the cost of burden from the five simulations within this alternative QMRA provides some of the most effective mitigation processes seen in this chapter (Table III. 2H.). Even scenarios 2 and 3, which represent the mid-tier intervention steps, proved to be effective in significantly reducing the illnesses and burdens associated with campylobacteriosis. Table III. 2H. illustrates the reductions seen in illnesses by implementing the intervention steps. It is expected at by reducing the concentration of

Campylobacter at the farm level as seen in scenario 2, the estimated illness is reduced from ~100,000 to ~35,000 annually, and by reducing the concentration level of positive birds to that seen in scenario 3, the estimated annual campylobacteriosis cases seen in the contiguous United States by the consumption of boneless, skinless chicken breast meals prepared at the home is approximately 4,000. Proportional decreases in public health costs can be seen in Table III. 2I. The effect of reducing the average concentration level seen in the infected bird, can have a greater effect in mitigating the level of annual human campylobacteriosis than reducing the overall occurrence of infection in chickens in the pre-processing population.

3.3. Transport Compliance – Cleaning and Sanitizing Transport Crates

This scenario provides a unique look at an implementation of an intervention that provides no significant impact of pathogen reduction in the overall processing line. Although the initial impact of the occurrence of transport cages contaminated with *Campylobacter* aligns with each level of washing and sanitizing compliance, as well as impacting the level of positive birds seen after the transport stage, convergence is seen for the occurrence of *Campylobacter*-positive carcasses at the end of the Defeathering stage as it progresses through the processing stage due to environmental cross-contamination. From Table III. 3A., the mean occurrence of *Campylobacter* contaminated cages drops from 22.7 to 11.2% as the proportion of cages that are washed and sanitized increases from the baseline level of 28.4% to 90-100% (scenario level 5). Increased transport cage sanitation appears to have little effect on *Campylobacter* prevalence for subsequent processing steps. The mean percentage of positive birds after transport decreases from 78.7 to 75.6% as the cleaning and sanitizing frequency increases from

the baseline level through scenarios 2 – 5 (Table II. 3B). Furthermore, the percent positive carcasses after scalding only decreases from 72.3 to 71.6% (Table III. 3C.) and after defeathering, the mean percent positive (87.1%) does not differ for each of the transport crate washing scenarios (Table III. 3D.).

3.4. Increase Proportion Frozen during In-Plant Storage

Freezing at the pre-retail stage provided significant and immediate reductions in occurrence of contamination of *Campylobacter*. For example, increasing the proportion of chicken frozen in-plant from 13.8% (baseline level) to 35-55% (scenario 3) would result in a mean reduction in the proportion of contaminated fillets from 63.8% to 47.6% (Table III. 4A.). Additionally, the *Campylobacter* load following the freezing intervention is estimated to drop from 2.4 log CFU/filet to 2.2 log CFU/fillet where the freezing proportion increased to 35-55%. For the 5th scenario (75-100 % freezing schedule), the mean log reduction is ~0.9 log CFU/filet from the baseline freezing concentration (Table III. 4B.). Tracking the bacterial load and the occurrence of contamination from retail, home storage, preparation, and ultimately consumption of the prepared meal, each stepwise scenario showed progressively larger impacts from the baseline (Table III. 4C.). There was an approximately 37 to 38-fold decrease, from the baseline to the 5th scenario, in the number of annual expected illnesses (Table III. 4D.) and the total annual costs attributed to acute campylobacteriosis in the contiguous United States (Table III. 4E.).

3.5. Cross-Contamination from Changes in Handwashing during Preparation

Table III. 5A exhibits the changes in the percentage of samples contaminated as handwashing frequency decreases through the scenarios simulated. For the baseline situation where 8.3% preparers do not wash their hands (and 58.3% rinse their hands) the model calculates that 2.5% of the cooked filets and prepared meals can become contaminated while 7.2% of the meals may become contaminated in the scenario where 80% of preparers do not wash their hands. For each handwashing scenario ~ 6 CFU would be transferred to the prepared meal, but a noticeable increase occurs in the proportion of contaminated filets with an increase of non-washing of hands. This increase carries over to the probability of ingestion of *Campylobacter* in a prepared meal where more than a two-fold increase (~3.1 to 7.7%) is observed with an increase from baseline 8.3% to 80% for not handwashing after handling raw chicken filets (Table III. 5B.).

The results of this section exhibit the ramifications with a breakdown of proper hygiene in the preparation of a meal. Since it occurs so far down the pathway modelled, the impact in just one part of the preparation stage extrapolated into an increase of ~50,000 additional illnesses when there is an increase in the non-washing of the hands in preparation of a chicken meal from a mean of 8.3% to 20% (Table III. 5C.). In scenario 5 (80% non-handwashing) the model estimates a ~5.5-fold increase in acute and secondary illnesses and public health costs of over \$1 billion when just considering total costs attributed to acute campylobacteriosis (Table III. 5D.). Cross-contamination of a cooked chicken meal with *Campylobacter* can result in a large increase in additional cases of campylobacteriosis.

3.6. Combination of Interventions – Reduce On-farm *Campylobacter* Concentrations and Increase In-Plant Freezing

The outputs presented in this combination intervention start off at the occurrence of contaminated filets during in-plant storage due to this secondary intervention occurring further downstream in the QMRA pathway. Tables III. 6A. through 6D. present data generated from the scenario analysis model for the synergistic effects of the two combined interventions. A more comprehensive dataset is contained within the QMRA alternative scenario model.

The effects of combination intervention steps are explored in this chapter as well. Although not every combination was analyzed with every intervention step, there were promising synergistic effects estimated in the models developed. The effects noted in combining the control of *Campylobacter* concentration at the post grow-out level at the farm prior to processing, and increasing the frequency of freezing boneless, skinless chicken filets prior to retail shipment yielded favorable results utilizing low to mid-tier interventions (simulations). For example, where in-plant freezing could reduce *Campylobacter* occurrence to 47.6% (scenario level 3, freezing of 35-55% of fillets), if on-farm bird concentrations were reduced ~2 logs (scenario level 3) prior to this level of in-plant freezing, then *Campylobacter* occurrence could drop to a mean 38.4% (from 47.6%) (Table III. 6A.). Furthermore, for the previous example, the combined interventions would result in a mean *Campylobacter* level after in-plant freezing of ~1.07 log CFU/filet whereas the level was ~2.23 log CFU/filet based on the increased freezing proportion alone (Table III. 6B.). The combined effects of reduced on-farm *Campylobacter* concentrations and increased in-plant freezing proportion will have greater impacts on reducing yearly campylobacteriosis and associated costs than either intervention alone (Table III. 6C. and 6D.).

4. CONCLUSION:

Other alternative scenario analyses can be performed in various locations within the QMRA system. The alternative scenarios represented in this chapter were chosen for their applicability in terms feasibility in implantation. The sensitive analysis provides locations where changes are most impactful. It can determine intervention steps which would have the most meaning if applicability is also realized. A cost-benefit or benefit-benefit analyses are imperative to whether these mitigation factors are realized. Moreover, the practicality of scalability must also be considered (Wegener 2010, Sears, Baker et al. 2011, Raut, Shashidhar et al. 2012). As can be seen in comparing the four applicable scenarios in terms of impact between the number of illnesses and consequently, the health costs ascertained per scenario and level of implementation, there is a varying range of mitigation (or breakdown, in the case of handwashing hygiene) in the outputs. Notably, these alternative scenarios as presented may not be comparative to “apples to apples”. The process in the alternative scenario used in the “Transport Cage Wash and Clean” compliance step provides an example where the attempt at the mitigation of occurrence in contamination, concentration in samples, and ultimately – illnesses provide nominal effectiveness due to the location in the intervention step in the processing pathway.

Tables III. 7A. and 7B. provide side-by-side comparison of the more pertinent issues within the QMRA outputs, such as annual illnesses/year, health costs/year, and impact on sequelae (GBS and IBS). It is notable to state that simulation 5 represents the most extreme scenario for each respective analysis. They represent the bookend to the baseline in terms of

change. It is expected for any practical implementation that any potential improvements seen in the mitigation of the occurrence of contamination and concentration of the food commodity/meal – translating to the number of illnesses and the accompanying societal burdens and costs would fall between the baseline and the extreme effects.

Wassenaar, et al (Wassenaar 2011) reviewed that most of the interventions presented in this chapter have been targeted in multiple levels internationally – including, but not limited to (1) reducing the number of *Campylobacter*-positive flocks at the farm; (2) reducing the bacterial load on and in the birds prior to slaughter from positive to negative birds; and (3) to reserve contaminated meat for frozen products, which reduces the number of viable organisms. There is difficulty in implementing these measures for success in large-scale, low cost production and these interventions may not be cost effective in highly competitive markets.

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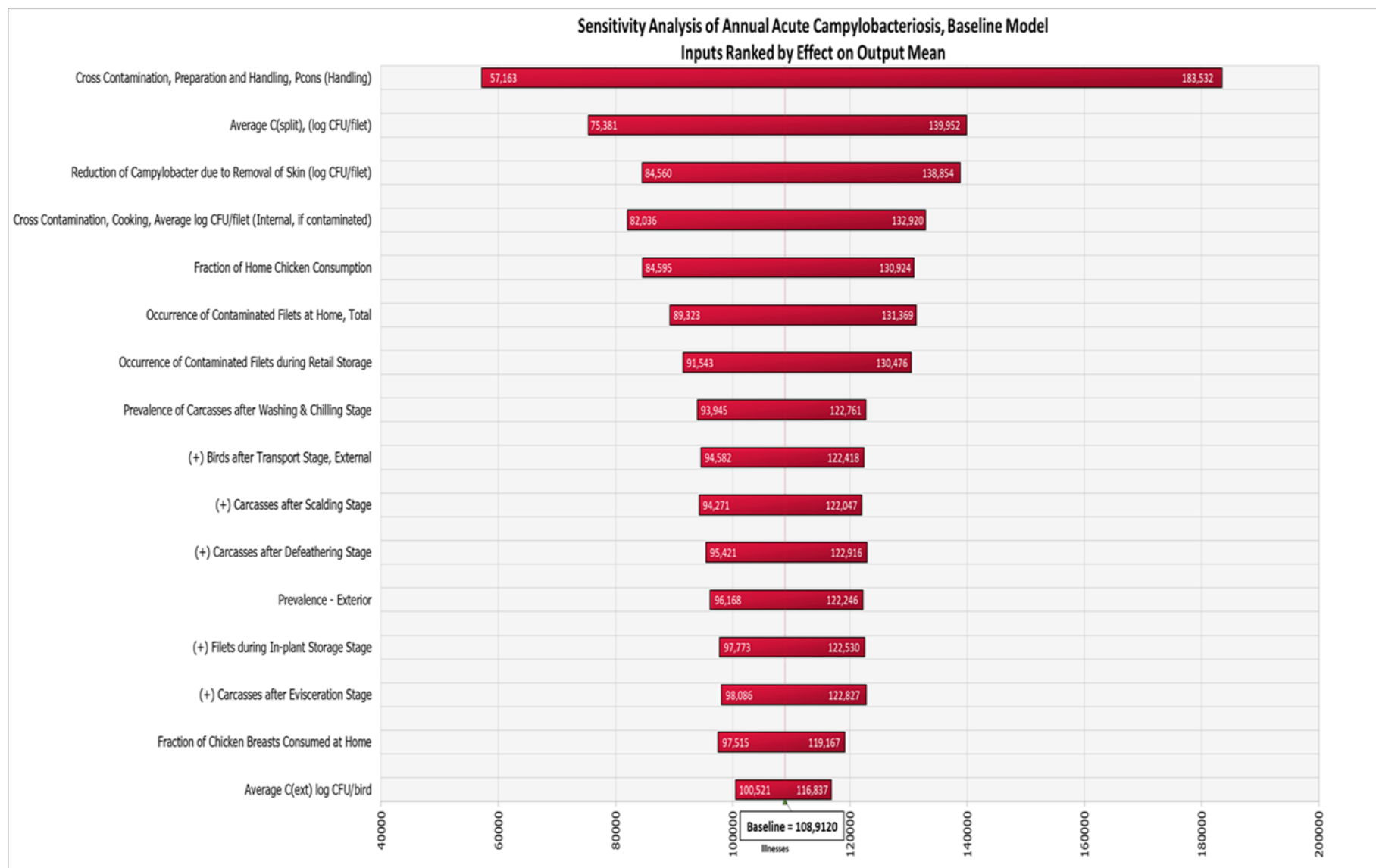
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FIGURE III. A. – Sensitivity Analysis of Acute Campylobacteriosis, Inputs Ranked by Output Mean



TABLES: III. 1A. – 1O.: Reduce *Campylobacter* Prevalence at Farm Grow-out, Pre-transport

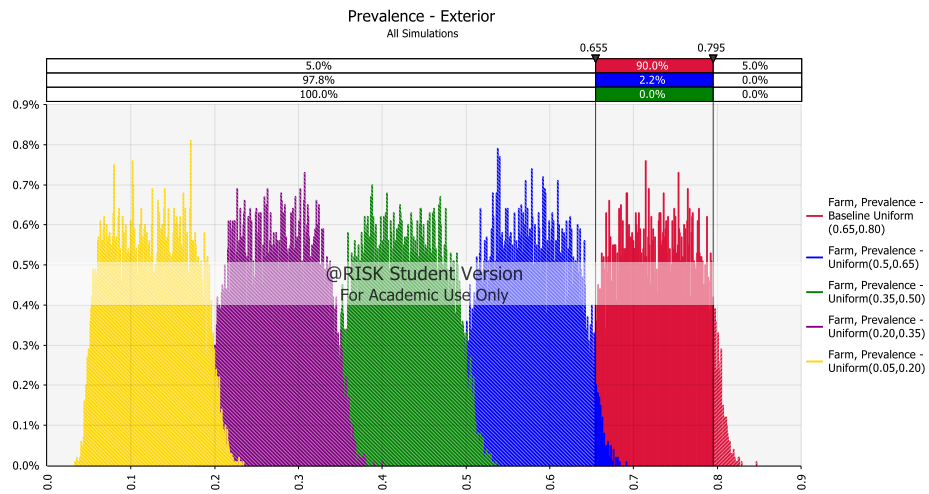


Table III. 1A. - Farm (Pre-Transport, Post grow-out), Prevalence—Exterior (C4)

Scenario	Mean	Median	5% CI	95% CI	Min	Max	Δ magnitude, Baseline
1, Baseline	0.725	0.726	0.655	0.795	0.611	0.847	-----
2	0.575	0.575	0.504	0.647	0.468	0.691	-1.261
3	0.425	0.425	0.353	0.496	0.318	0.536	-1.706
4	0.275	0.275	0.204	0.345	0.168	0.401	-2.636
5	0.125	0.125	0.056	0.194	0.033	0.234	-5.800

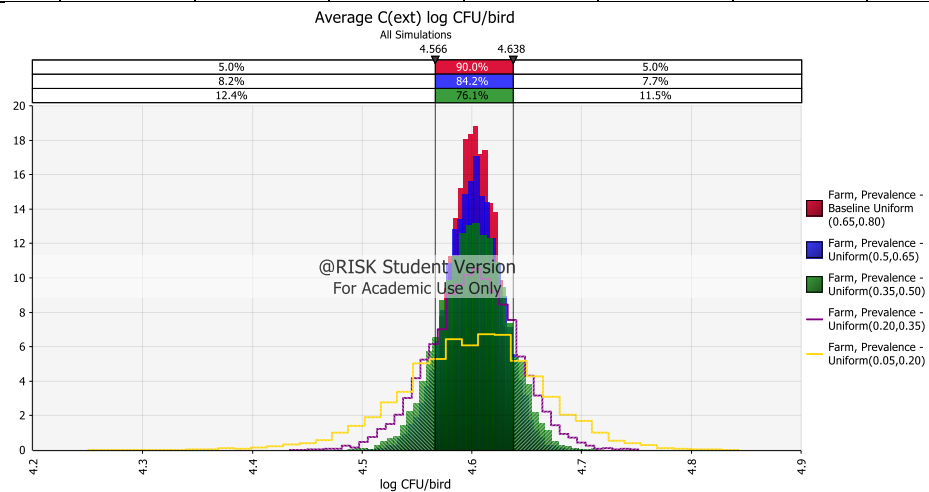


**Table III. 1B. – Contamination of Carcass - Farm (Pre-Transport, Post grow-out)
Average C(ext) log CFU/bird – (C5)**

Scenario	Mean	Median	5% CI	95% CI	Min	Max	Δ magnitude, Baseline
1, Baseline	4.602	4.602	4.566	4.638	4.509	4.684	-----
2	4.602	4.602	4.550	4.643	4.502	4.701	1.000
3	4.602	4.602	4.552	4.651	4.475	4.715	1.000
4	4.601	4.602	4.536	4.664	4.434	4.752	1.000
5	4.599	4.601	4.496	4.701	4.250	4.843	-1.001

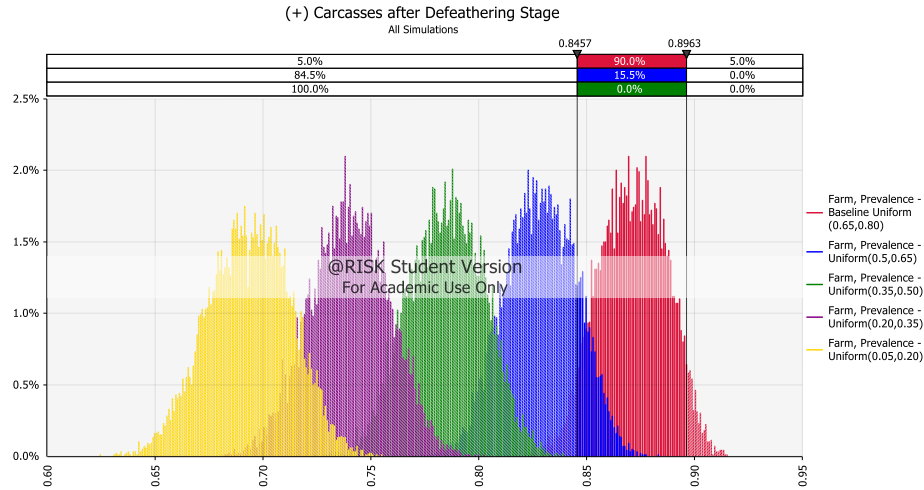


Table III. 1C. – Positive Carcasses after Defeathering Stage (C88)

Scenario	Mean	Median	5% CI	95% CI	Min	Max	Δ magnitude, Baseline
1, Baseline	0.871	0.872	0.846	0.896	0.814	0.915	-----
2	0.829	0.829	0.802	0.856	0.770	0.883	-1.051
3	0.785	0.785	0.757	0.813	0.724	0.839	-1.110
4	0.740	0.740	0.710	0.770	0.681	0.802	-1.177
5	0.694	0.694	0.661	0.725	0.625	0.755	-1.255

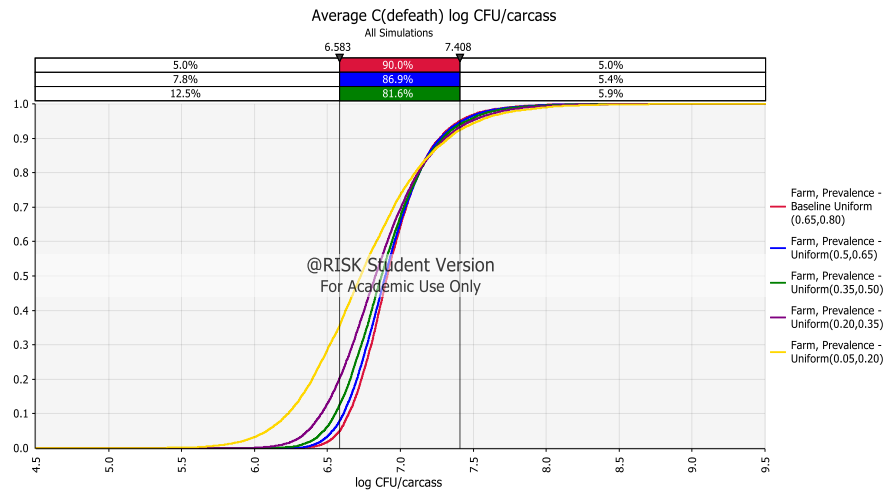


Table III. 1D. – Average C(defeat) log CFU/carcass after Defeathering Stage (C41)

Scenario	Mean	Median	5% CI	95% CI	Min	Max	Δ magnitude, Baseline
1, Baseline	6.942	6.910	6.583	7.408	6.210	8.658	-----
2	6.926	6.896	6.534	7.420	6.211	8.708	-1.038
3	6.903	6.870	6.473	7.453	6.022	8.698	-1.094
4	6.864	6.828	6.364	7.488	5.793	8.882	-1.197
5	6.758	6.728	6.084	7.537	4.906	9.066	-1.528

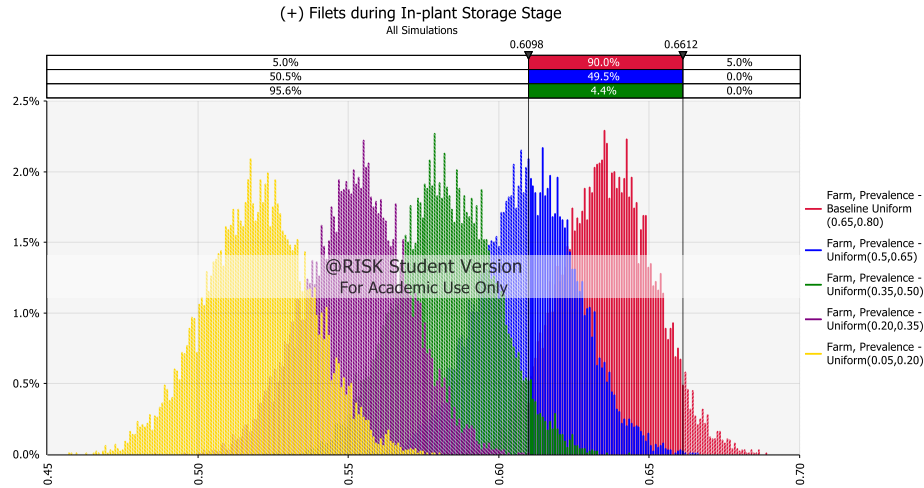


Table III. 1E. – Positive Filets during In-plant Storage (C88)

Scenario	Mean	Median	5% CI	95% CI	Min	Max	Δ magnitude, Baseline
1, Baseline	0.636	0.636	0.610	0.661	0.575	0.689	-----
2	0.610	0.610	0.583	0.636	0.549	0.666	-1.043
3	0.582	0.582	0.555	0.609	0.522	0.643	-1.093
4	0.553	0.553	0.531	0.580	0.486	0.610	-1.150
5	0.520	0.520	0.491	0.549	0.457	0.580	-1.223

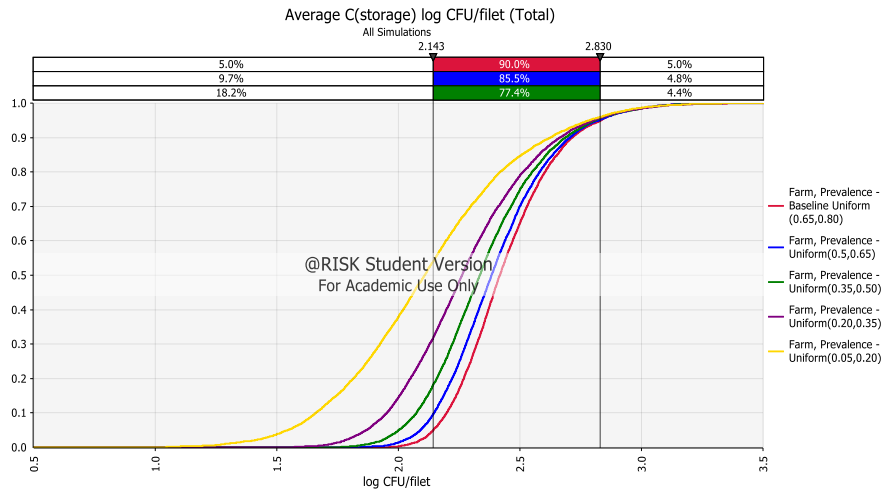


Table III. 1F. – Average C(storage) log CFU/filet during In-plant Storage (C89)

Scenario	Mean	Median	5% CI	95% CI	Min	Max	Δ magnitude, Baseline
1, Baseline	2.442	2.417	2.144	2.830	1.818	3.397	-----
2	2.408	2.383	2.090	2.825	1.779	3.432	-1.081
3	2.360	2.335	2.000	2.808	1.700	3.400	-1.208
4	2.288	2.262	1.868	2.800	1.417	3.400	-1.426
5	2.124	2.108	1.545	2.783	0.642	3.401	-2.080

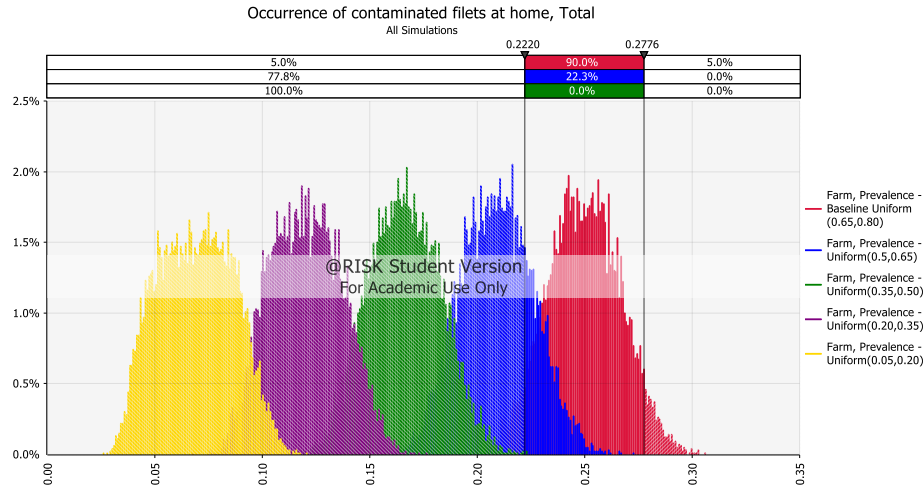


Table III. 1G. – Occurrence of Contaminated Filets at Home (C112)

Scenario	Mean	Median	5% CI	95% CI	Min	Max	Δ magnitude, Baseline
1, Baseline	0.250	0.250	0.222	0.278	0.189	0.306	-----
2	0.209	0.209	0.182	0.237	0.160	0.273	-1.196
3	0.166	0.166	0.140	0.193	0.118	0.223	-1.506
4	0.120	0.120	0.094	0.148	0.068	0.176	-2.083
5	0.069	0.069	0.042	0.097	0.026	0.121	-3.623

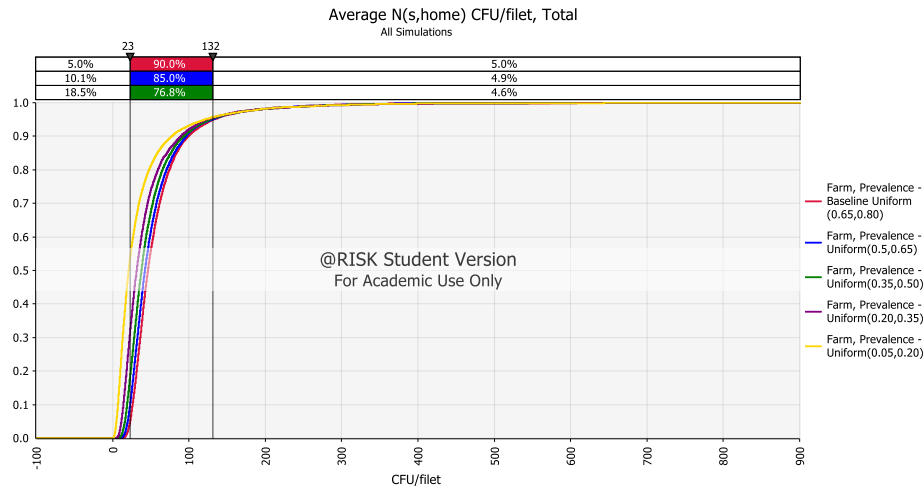


Table III. 1H. – Average N(home) CFU/Filet at Home (C113)

Scenario	Mean	Median	5% CI	95% CI	Min	Max	Δ magnitude, Baseline
1, Baseline	58.55	46.33	23.11	131.74	10.81	860.59	-----
2	55.11	42.35	19.78	130.35	8.83	810.44	-1.062
3	51.04	37.76	16.16	127.68	6.76	714.66	-1.147
4	45.73	31.52	11.39	123.25	2.79	735.53	-1.280
5	36.53	20.98	4.95	120.23	0.471	898.33	-1.603

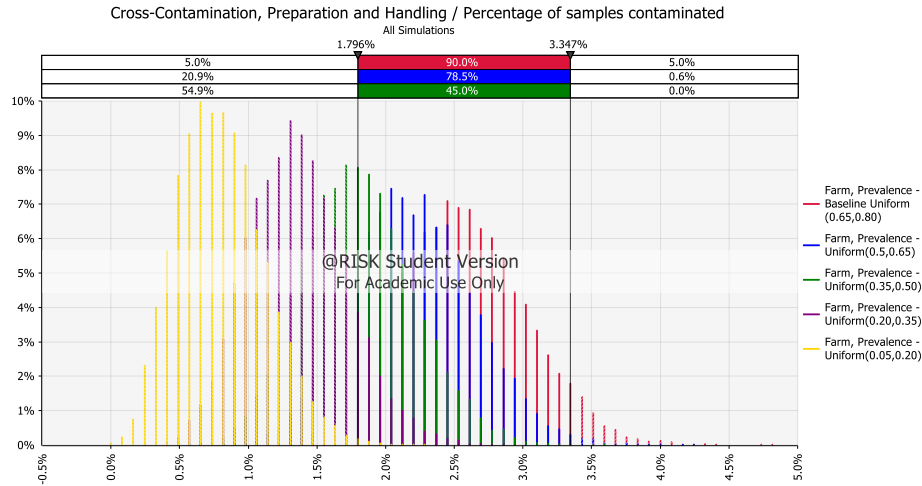


Table III. 1I. – Percentage of Samples Contaminated, Preparation and Handling (C183)

Scenario	Mean	Median	5% CI	95% CI	Min	Max	Δ magnitude, Baseline
1, Baseline	2.552%	2.531%	1.796%	3.347%	0.898%	4.816%	-----
2	2.198%	2.204%	1.469%	2.939%	0.816%	4.245%	-1.161
3	1.803%	1.796%	1.143%	2.531%	0.490%	3.592%	-1.415
4	1.357%	1.306%	0.816%	1.959%	0.245%	2.857%	-1.881
5	0.802%	0.816%	0.327%	1.388%	0%	2.286%	-3.182

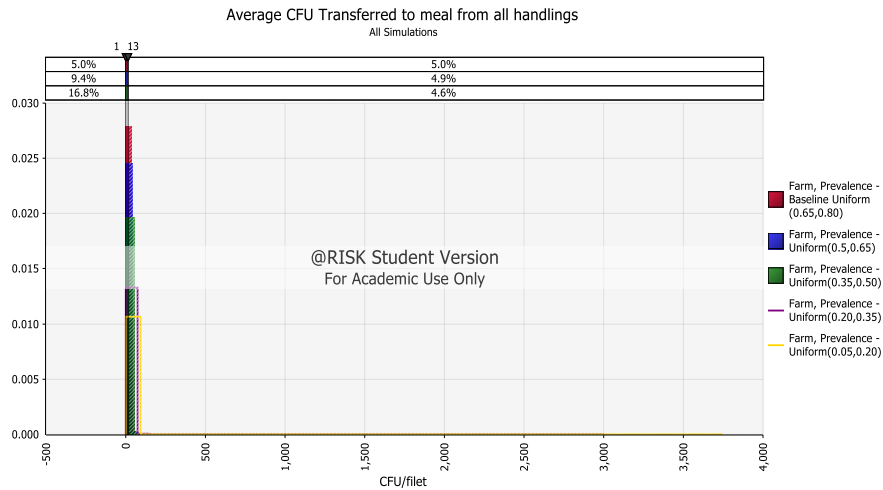


Table III. 1J. – Average CFU Transferred to Meal from Preparation and Handling (C179)

Scenario	Mean	Median	5% CI	95% CI	Min	Max	Δ magnitude, Baseline
1, Baseline	4.84	2.32	0.772	12.97	0.298	1,420.20	-----
2	4.60	2.04	0.626	12.72	0.173	1,613.77	-1.052
3	4.32	1.72	0.471	12.12	0.124	2,018.80	-1.120
4	4.05	1.33	0.302	11.30	0.0645	2,994.58	-1.200
5	3.32	0.753	0.0971	9.73	0.00898	3,741.31	-1.458

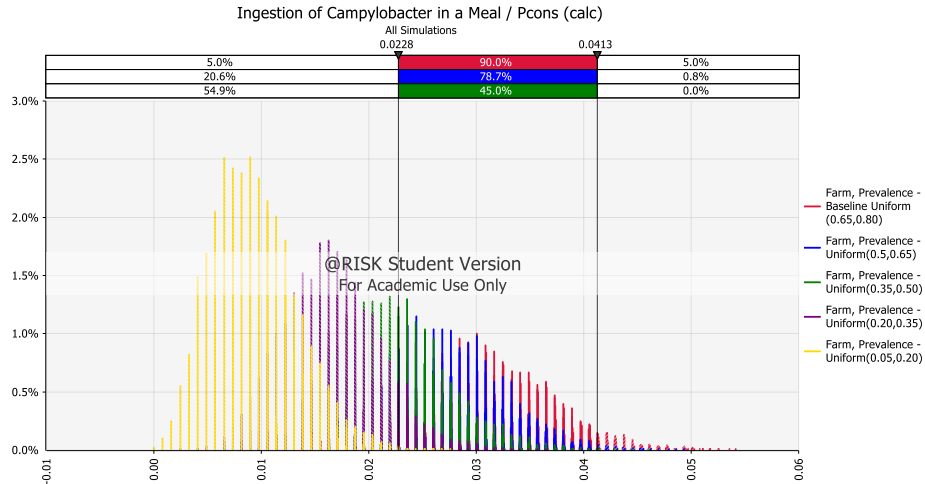


Table III. 1K. – Ingestion of *Campylobacter* in a Meal – Pcons(calc) (C6)

Scenario	Mean	Median	5% CI	95% CI	Min	Max	Δ magnitude, Baseline
1, Baseline	0.0317	0.0316	0.0228	0.0413	0.0122	0.0542	-----
2	0.0274	0.0269	0.0187	0.0365	0.0098	0.0493	-1.170
3	0.0226	0.0221	0.0147	0.0309	0.0057	0.0460	-1.403
4	0.0171	0.0171	0.0106	0.0244	0.0024	0.0364	-1.853
5	0.0102	0.0098	0.0041	0.0171	0	0.0276	-3.108

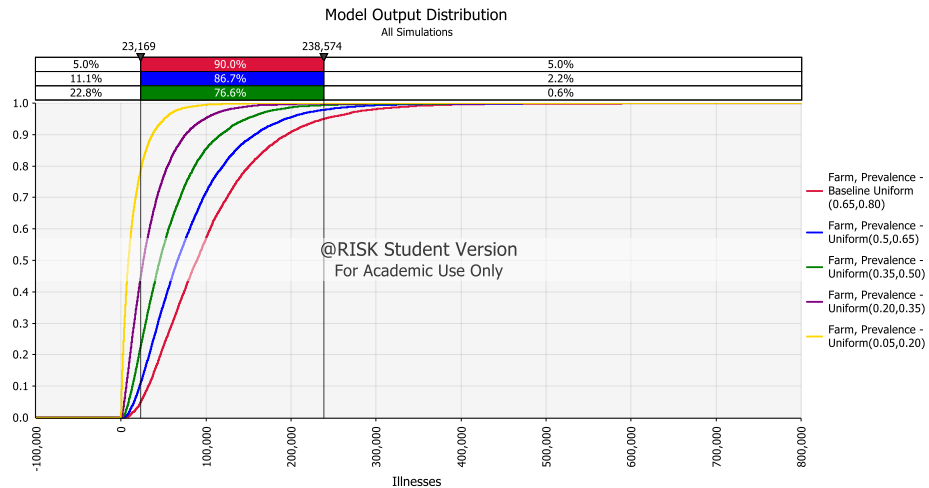


Table III. 1L. – N illnesses / year (D53)

Scenario	Mean	Median	5% CI	95% CI	Min	Max	Δ magnitude, Baseline
1, Baseline	103,989	89,151	23,169	238,574	1,516	749,364	-----
2	80,526	66,335	15,270	193,391	1,180	640,097	-1.294
3	57,631	45,610	8,909	147,210	215	575,660	-1.804
4	35,501	26,266	3,732	98,518	84	499,351	-2.929
5	15,017	8,623	440	50,687	0	237,184	-6.925

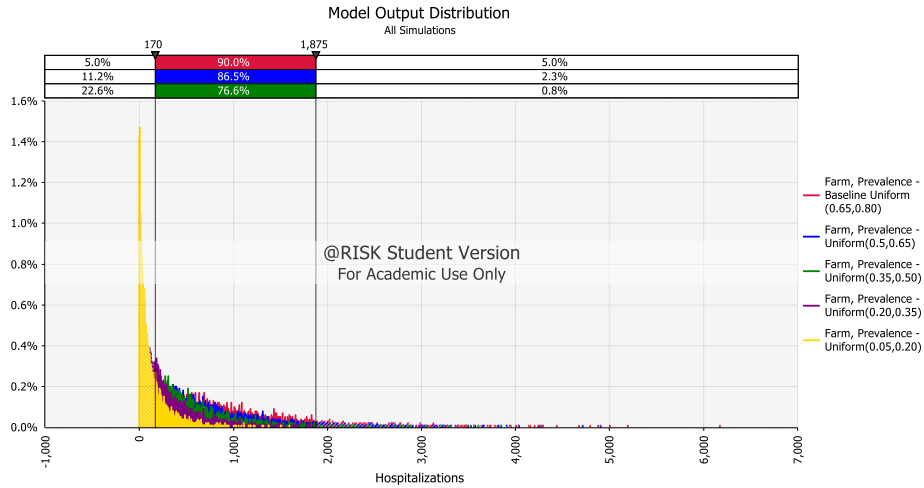


Table III. 1M. – N Hospitalizations / year (E53)

Scenario	Mean	Median	5% CI	95% CI	Min	Max	Δ magnitude, Baseline
1, Baseline	802	668	170	1,875	14	6,171	-----
2	621	502	111	1,510	10	4,908	-1.291
3	444	345	65	1,144	2	3,848	-1.806
4	274	199	27	766	1	2,989	-2.927
5	116	66	3	398	0	1,595	-6.914

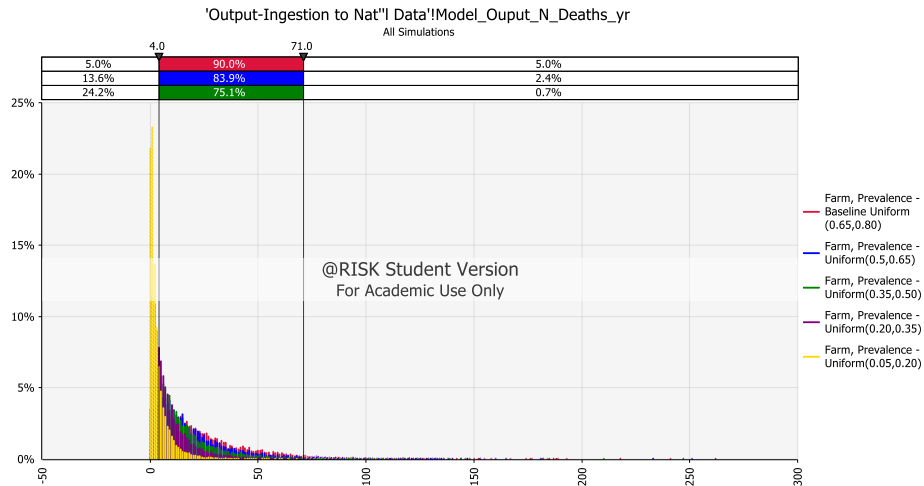


Table III. 1N. – N Deaths / year (F53)

Scenario	Mean	Median	5% CI	95% CI	Min	Max	Δ magnitude, Baseline
1, Baseline	26	19	4	71	0	262	-----
2	20	14	2	57	0	251	-1.300
3	14	10	1	43	0	247	-1.857
4	9	6	1	28	0	182	-2.889
5	4	2	0	14	0	68	-6.500

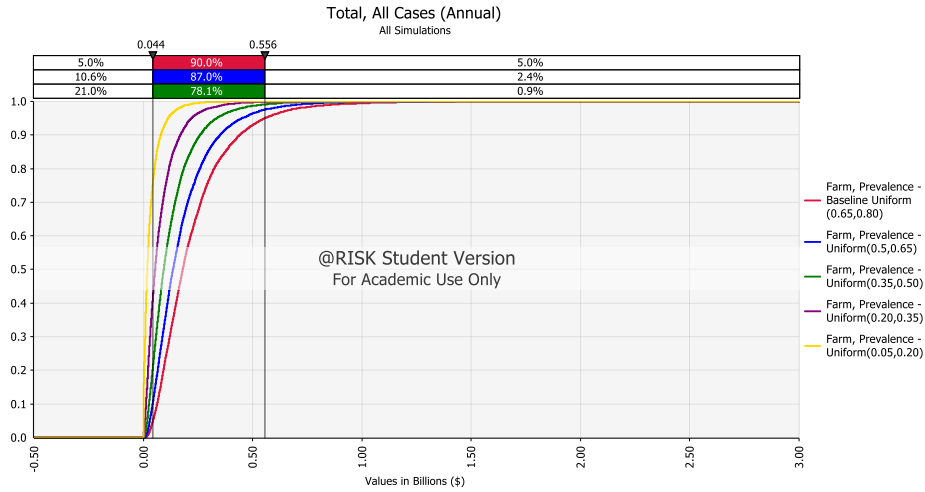


Table III. 10. – Total Costs - Acute / year (C77)

Scenario	Mean	Median	5% CI	95% CI	Min	Max	Δ magnitude, Baseline
1, Baseline	\$224,836,288	\$180,375,079	\$43,817,991	\$555,519,676	\$3,359,504	\$2.547 x 10 ⁹	-----
2	\$174,280,127	\$135,296,494	\$29,463,903	\$451,464,250	\$1,414,007	\$2.443 x 10 ⁹	-1.290
3	\$124,408,229	\$93,253,714	\$18,040,204	\$335,159,989	\$117,235	\$2.404 x 10 ⁹	-1.807
4	\$76,844,532	\$53,668,042	\$7,672,214	\$169,103,509	\$32,876	\$1.243 x 10 ⁹	-2.926
5	\$32,379,155	\$17,900,103	\$374,145	\$112,356,888	\$0	\$597,749,596	-6.944

Tables: III. 2A. – 2I.: Reduce *Campylobacter* Concentrations at Farm Grow-out, Pre-Transport

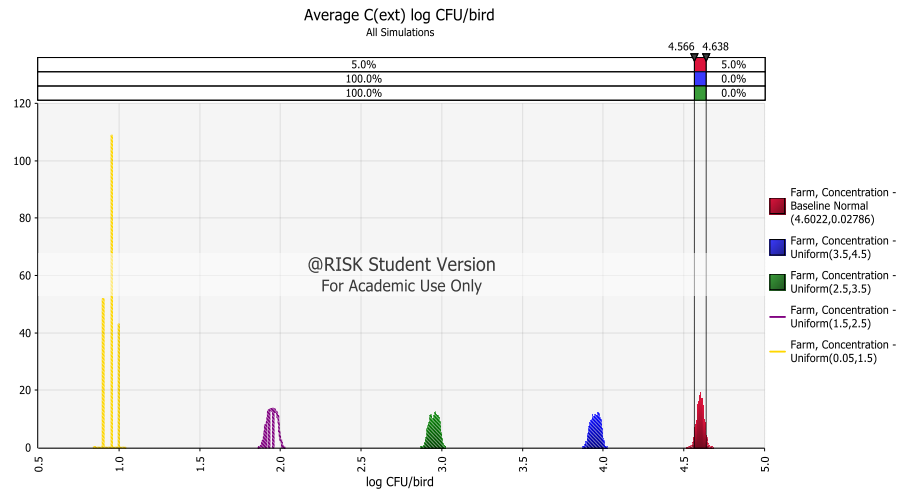


Table III. 2A. – Contamination of Carcass - Farm (Pre-Transport, Post grow-out),
Avg C(ext) log CFU/bird – (C5)

Scenario	Mean	Median	5% CI	95% CI	Min	Max	Δ magnitude, Baseline
1, Baseline	4.602	4.603	4.566	4.638	4.515	4.682	-----
2	3.951	3.952	3.904	3.995	3.871	4.027	-4.477
3	2.951	2.952	2.905	2.996	2.870	3.025	-44.771
4	1.951	1.954	1.903	1.996	1.863	2.029	-447.713
5	0.951	0.954	0.903	1.000	0.845	1.041	-4477.133

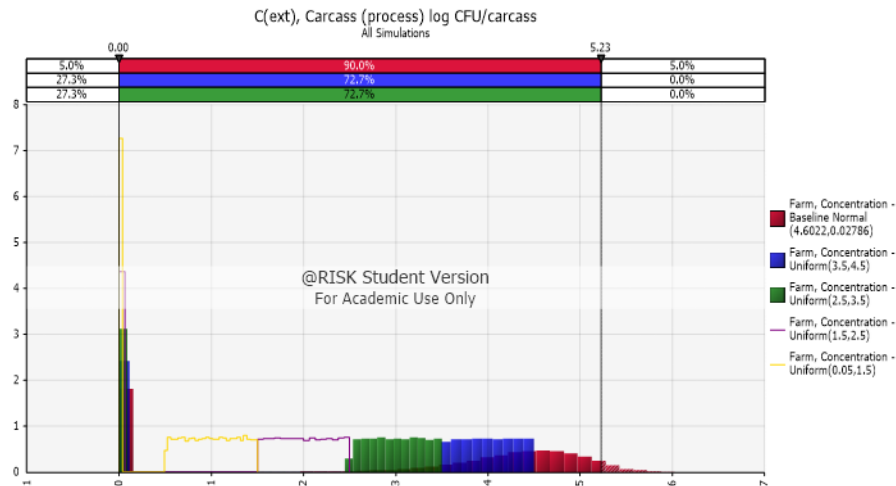


Table III. 2B. – Contamination of Carcass- Farm (Pre-Transport, Post grow-out)
C(ext) Carcass (process) log CFU/carcass – individual bird sampling (C16)

Scenario	Mean	Median	5% CI	95% CI	Min	Max	Δ magnitude, Baseline
1, Baseline	3.177	4.121	0	5.228	0	6.031	-----
2	2.910	3.812	0	4.433	0	4.500	-1.849
3	2.182	2.814	0	3.434	0	3.500	-9.886
4	1.455	1.812	0	2.433	0	2.500	-52.723
5	0.727	0.812	0	1.429	0	1.500	-281.838

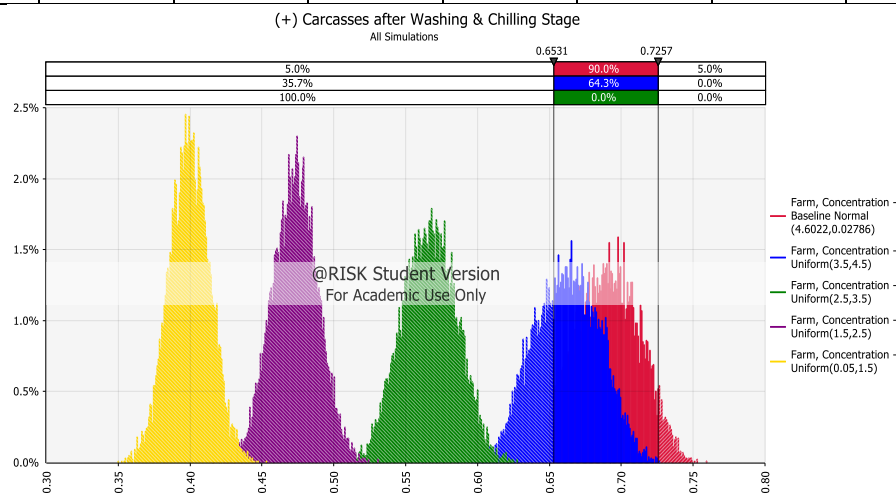


Table III. 2C. – Occurrence of Contaminated Carcasses after Washing and Chilling (C70)

Scenario	Mean	Median	5% CI	95% CI	Min	Max	Δ magnitude, Baseline
1, Baseline	0.690	0.691	0.653	0.723	0.620	0.760	-----
2	0.662	0.663	0.625	0.699	0.587	0.727	-1.042
3	0.566	0.567	0.536	0.598	0.501	0.628	-1.219
4	0.473	0.473	0.447	0.499	0.416	0.531	-1.459
5	0.400	0.399	0.376	0.423	0.350	0.453	-1.725

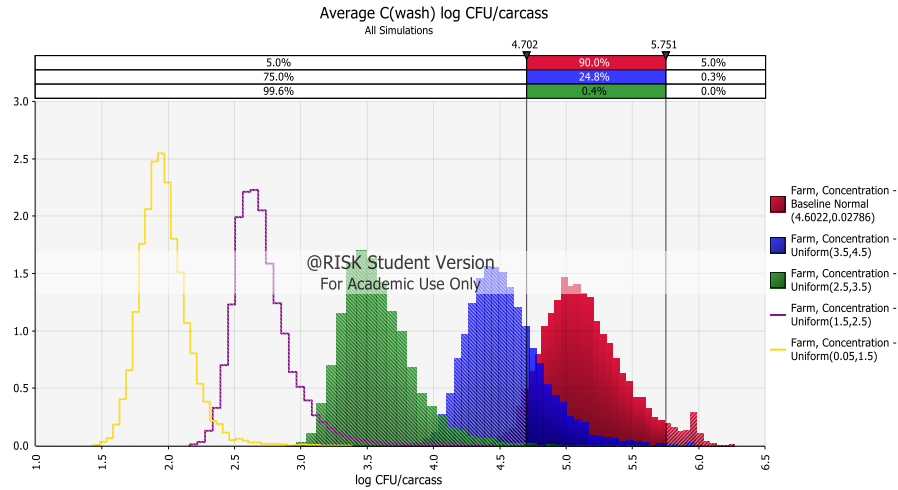


Table III. 2D. – Average C(wash) log CFU/carass after Washing and Chilling (C71)

Scenario	Mean	Median	5% CI	95% CI	Min	Max	Δ magnitude, Baseline
1, Baseline	5.145	5.105	4.702	5.751	4.331	6.275	-----
2	4.545	4.501	4.149	5.070	3.756	6.018	-3.981
3	3.580	3.538	3.215	4.082	2.889	5.914	-36.728
4	2.712	2.673	2.426	3.132	2.161	4.462	-271.020
5	1.968	1.951	1.710	2.283	1.436	3.383	-1503.142

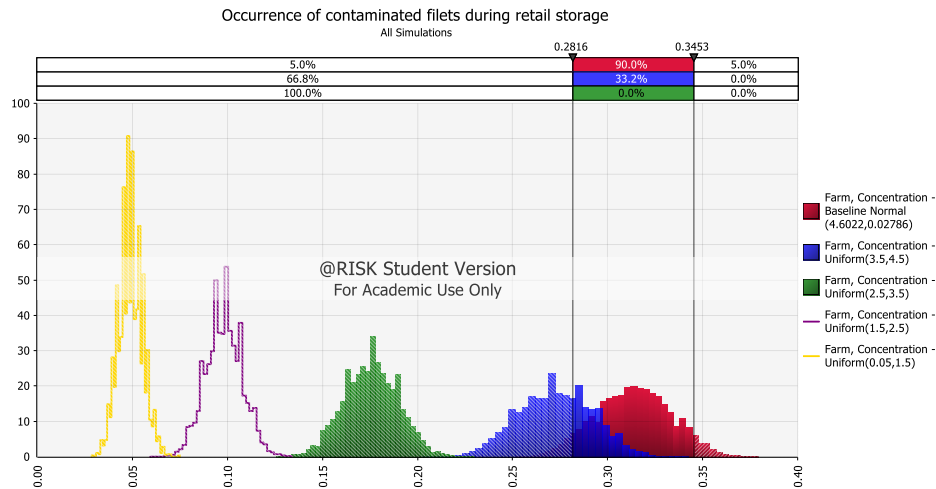


Table III. 2E. – Occurrence of Contaminated Filets during Retail Storage (C99)

Scenario	Mean	Median	5% CI	95% CI	Min	Max	Δ magnitude, Baseline
1, Baseline	0.314	0.314	0.282	0.345	0.251	0.380	-----
2	0.273	0.273	0.242	0.304	0.202	0.343	-1.150
3	0.176	0.176	0.153	0.198	0.126	0.230	-1.784
4	0.098	0.098	0.082	0.114	0.060	0.134	-3.204
5	0.049	0.048	0.039	0.060	0.029	0.075	-6.408

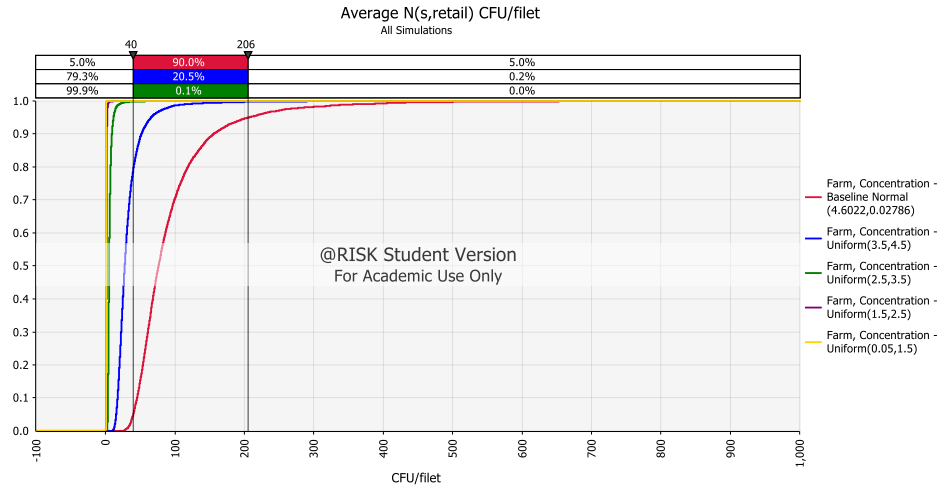


Table III. 2F. – Average N(s,retail) CFU/filet (C100)

Scenario	Mean	Median	5% CI	95% CI	Min	Max	Δ magnitude, Baseline
1, Baseline	93.55	76.16	39.92	205.64	20.22	917.43	-----
2	32.72	27.51	15.21	65.44	7.55	479.69	-2.859
3	5.93	5.04	3.01	11.22	1.53	339.65	-15.776
4	1.22	1.10	0.69	2.14	0.39	20.21	-76.680
5	0.287	0.265	0.161	0.412	0.083	2.27	-325.958

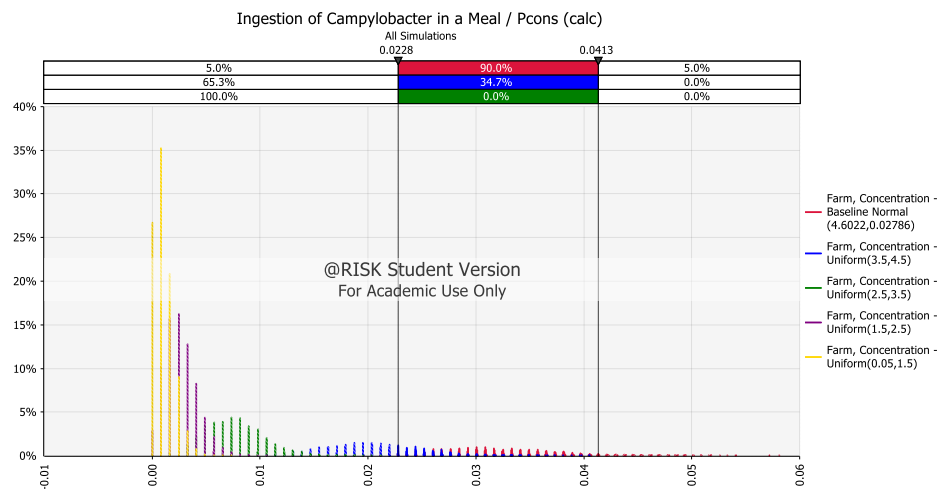


Table III. 2G. – Ingestion of *Campylobacter* in a Meal – Pcons(calc) (C6)

Scenario	Mean	Median	5% CI	95% CI	Min	Max	Δ magnitude, Baseline
1, Baseline	0.0316	0.0316	0.0212	0.0413	0.0106	0.0581	-----
2	0.0210	0.0211	0.0130	0.0292	0.0049	0.0405	-1.505
3	0.00821	0.00815	0.00408	0.0130	0	0.0212	-3.849
4	0.00293	0.00245	0.000816	0.00571	0	0.0106	-10.785
5	0.00108	0.000816	0	0.00245	0	0.00735	-29.259

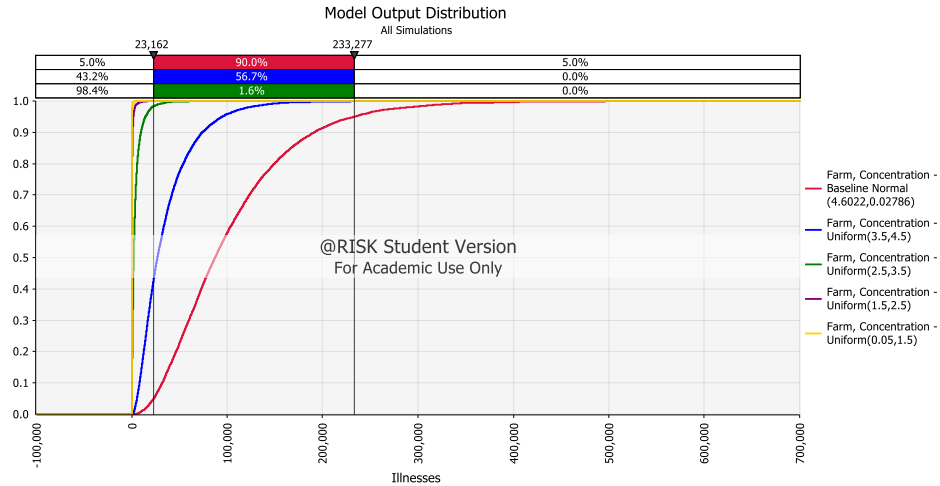


Table III. 2H. – N illnesses / year (D53)

Scenario	Mean	Median	5% CI	95% CI	Min	Max	Δ magnitude, Baseline
1, Baseline	102,356	87,315	23,162	233,277	1,996	681,043	-----
2	35,678	26,963	5,555	94,983	170	365,997	-2.869
3	4,083	2,180	197	14,363	0	83,170	-25.069
4	471	144	0	1,873	0	33,786	-217.316
5	76	9	0	317	0	9,724	-1346.789

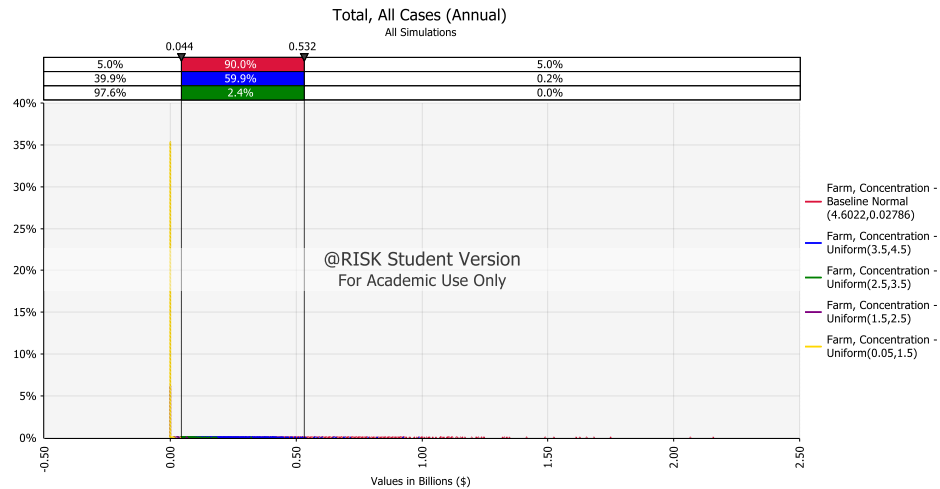


Table III. 2I. – Total Costs - Acute / year (C77)

Scenario	Mean	Median	5% CI	95% CI	Min	Max	Δ magnitude, Baseline
1, Baseline	\$220,204,195	\$178,327,507	\$43,924,693	\$532,355,755	\$3,757,727	\$2.156 x 10 ⁹	-----
2	\$76,822,054	\$55,426,608	\$10,983,055	\$211,842,178	\$49,352	\$990,125,477	-2.866
3	\$8,530,287	\$4,062,910	\$122,609	\$30,917,484	\$0	\$322,261,958	-25.814
4	\$784,979	\$104,738	\$0	\$3,609,931	\$0	\$66,010,520	-280.522
5	\$96,579	\$4,920	\$0	\$288,184	\$0	\$42,354,870	-2280.042

Tables: III. 3A. – 3D.: Transport Compliance – Cleaning and Sanitizing Transport Crates

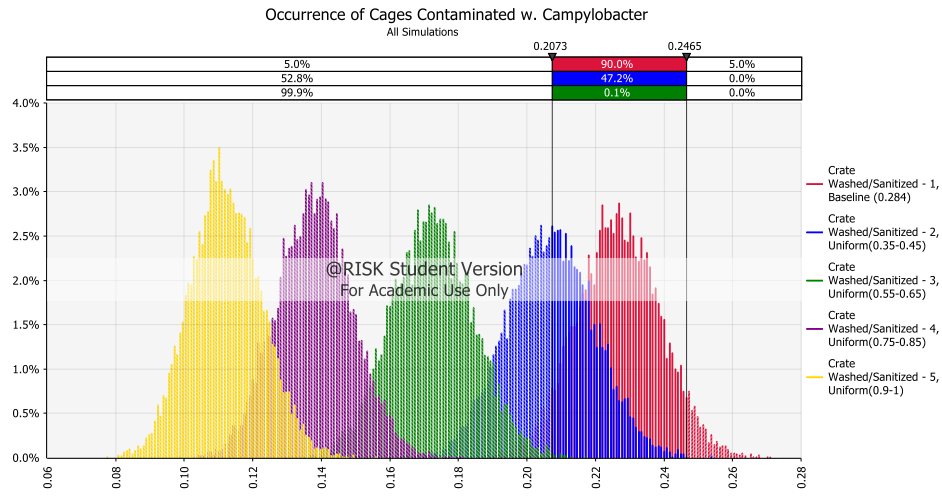


Table III. 3A. – Occurrence of Cages Contaminated with *Campylobacter* (C14)

Scenario	Mean	Median	5% CI	95% CI	Min	Max	Δ magnitude, Baseline
1, Baseline	0.227	0.227	0.204	0.247	0.176	0.271	-----
2	0.207	0.207	0.186	0.228	0.160	0.254	-1.100
3	0.172	0.171	0.153	0.192	0.130	0.217	-1.320
4	0.138	0.138	0.120	0.157	0.099	0.181	-1.645
5	0.112	0.112	0.096	0.130	0.078	0.156	-2.027

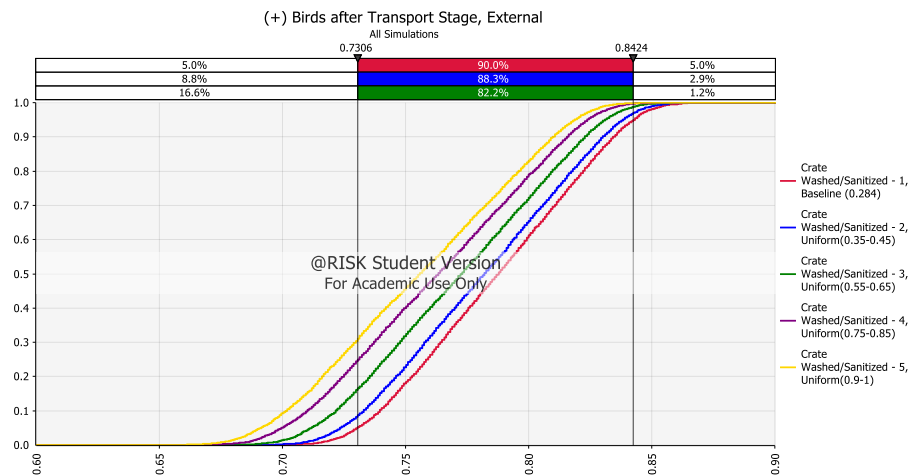


Table III. 3B. – Positive Birds after Transport Stage (C24)

Scenario	Mean	Median	5% CI	95% CI	Min	Max	Δ magnitude, Baseline
1, Baseline	0.787	0.788	0.731	0.822	0.695	0.881	-----
2	0.782	0.782	0.723	0.838	0.684	0.878	-1.006
3	0.772	0.773	0.712	0.831	0.675	0.863	-1.019
4	0.763	0.763	0.700	0.824	0.659	0.856	-1.031
5	0.756	0.756	0.691	0.819	0.659	0.851	-1.041

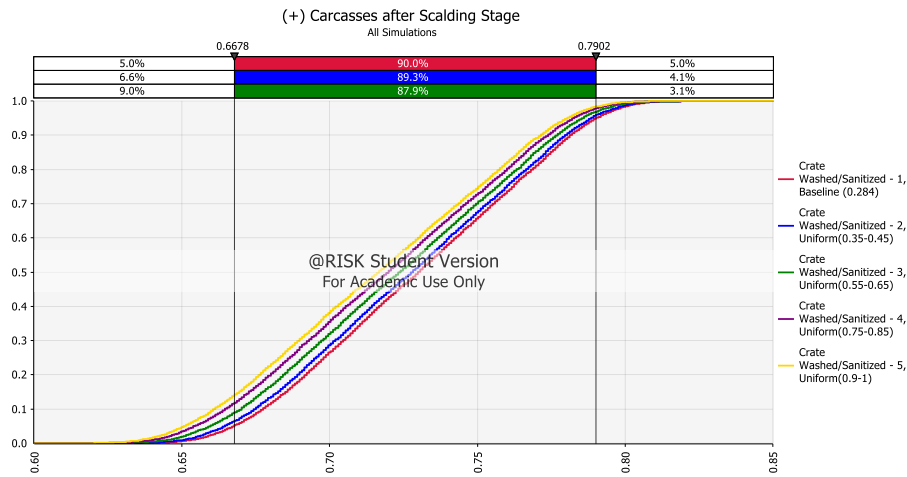


Table III. 3C. – Positive Carcasses after Scalding Stage (C29)

Scenario	Mean	Median	5% CI	95% CI	Min	Max	Δ magnitude, Baseline
1, Baseline	0.723	0.730	0.668	0.790	0.632	0.828	-----
2	0.727	0.728	0.664	0.789	0.630	0.826	+0.994
3	0.723	0.724	0.652	0.786	0.620	0.826	1.000
4	0.719	0.720	0.655	0.783	0.614	0.821	-1.006
5	0.716	0.716	0.651	0.780	0.610	0.816	-1.010

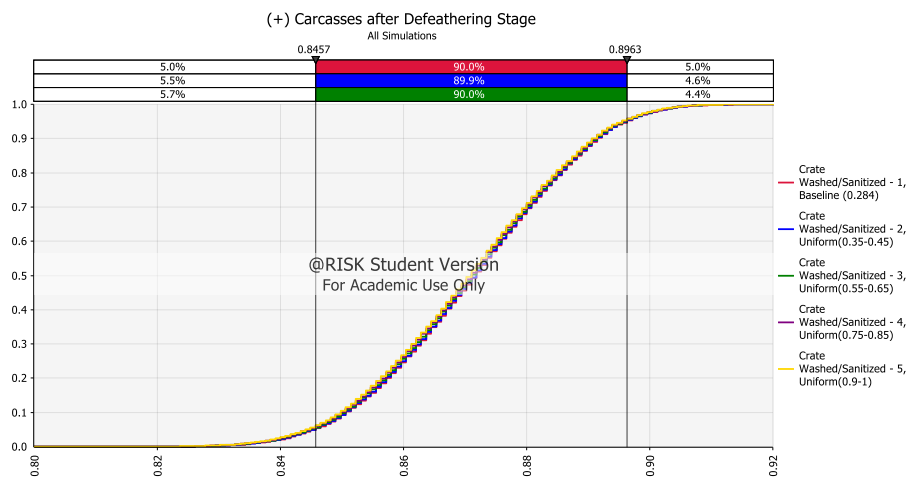


Table III. 3.D. – Positive Carcasses after Defeathering Stage (C35)

Scenario	Mean	Median	5% CI	95% CI	Min	Max	Δ magnitude, Baseline
1, Baseline	0.871	0.872	0.846	0.896	0.815	0.918	-----
2	0.871	0.876	0.846	0.896	0.814	0.918	1.000
3	0.871	0.871	0.845	0.896	0.814	0.918	1.000
4	0.871	0.871	0.844	0.896	0.814	0.918	1.000
5	0.871	0.871	0.844	0.891	0.810	0.918	1.000

Tables: III. 4A. – 4E.: Increase Proportion Frozen during In-Plant Storage

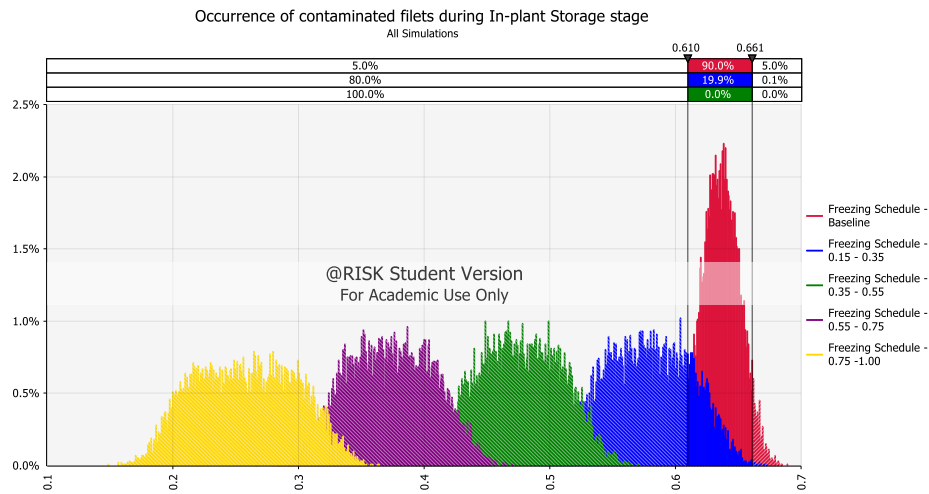


Table III. 4A. – Occurrence of Contaminated Filets during In-Plant Storage (C88)

Scenario	Mean	Median	5% CI	95% CI	Min	Max	Δ magnitude, Baseline
1, Baseline	0.638	0.636	0.610	0.661	0.579	0.689	-----
2	0.578	0.578	0.524	0.633	0.486	0.673	-1.104
3	0.476	0.476	0.422	0.529	0.376	0.571	-1.340
4	0.373	0.373	0.319	0.428	0.267	0.470	-1.710
5	0.258	0.258	0.195	0.322	0.149	0.364	-2.472

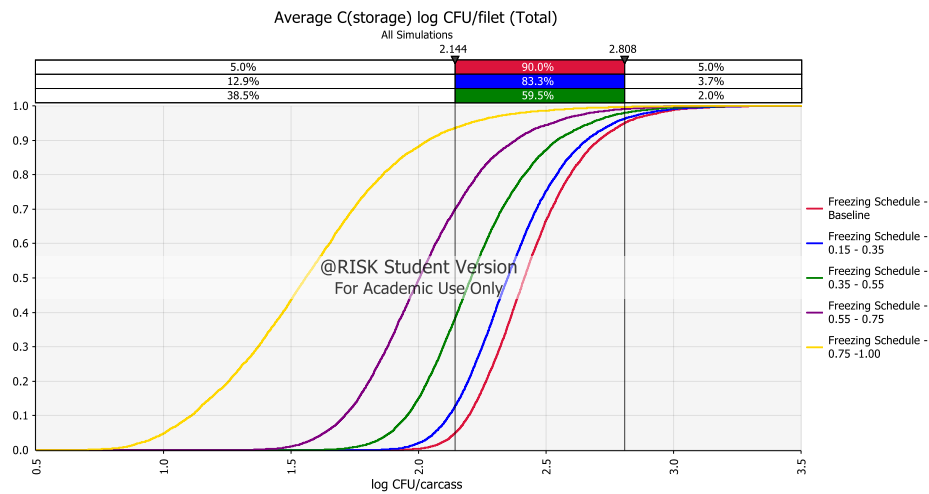


Table III. 4B. – Average C(storage) log CFU/filet during In-Plant Storage (C89)

Scenario	Mean	Median	5% CI	95% CI	Min	Max	Δ magnitude, Baseline
1, Baseline	2.437	2.415	2.144	2.808	1.847	3.393	-----
2	2.373	2.349	2.062	2.765	1.685	3.387	-1.159
3	2.231	2.205	1.887	2.677	1.550	3.372	-1.607
4	2.028	2.004	1.631	2.525	1.229	3.189	-2.564
5	1.571	1.556	1.006	2.201	0.518	3.169	-7.345

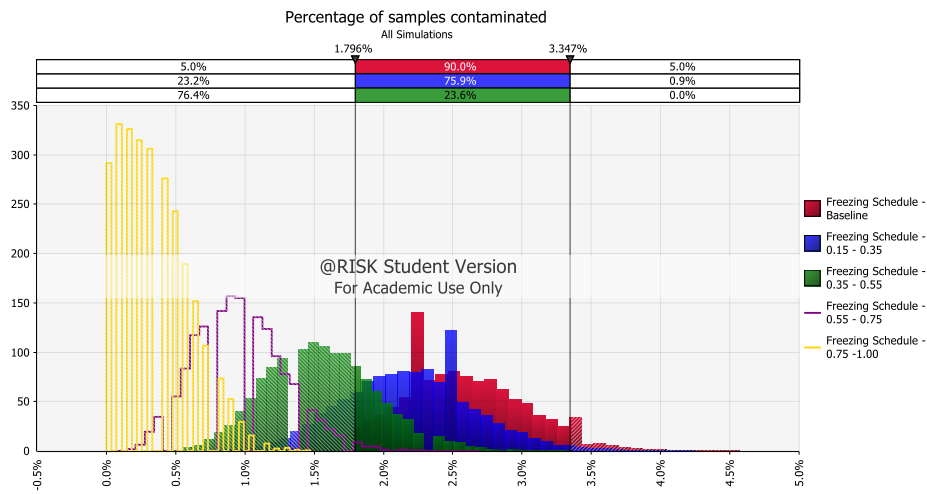


Table III. 4C. – Percentage of Samples Contaminated, Preparation and Handling (C183)

Scenario	Mean	Median	5% CI	95% CI	Min	Max	Δ magnitude, Baseline
1, Baseline	2.551%	2.531%	1.796%	3.347%	1.061%	4.571%	-----
2	2.189%	2.204%	1.469%	3.020%	0.816%	4.245%	-1.165
3	1.561%	1.551%	0.980%	2.204%	0.327%	3.347%	-1.634
4	0.971%	0.980%	0.490%	1.551%	0.082%	2.694%	-2.627
5	0.348%	0.327%	0%	0.816%	0%	1.469%	-7.330

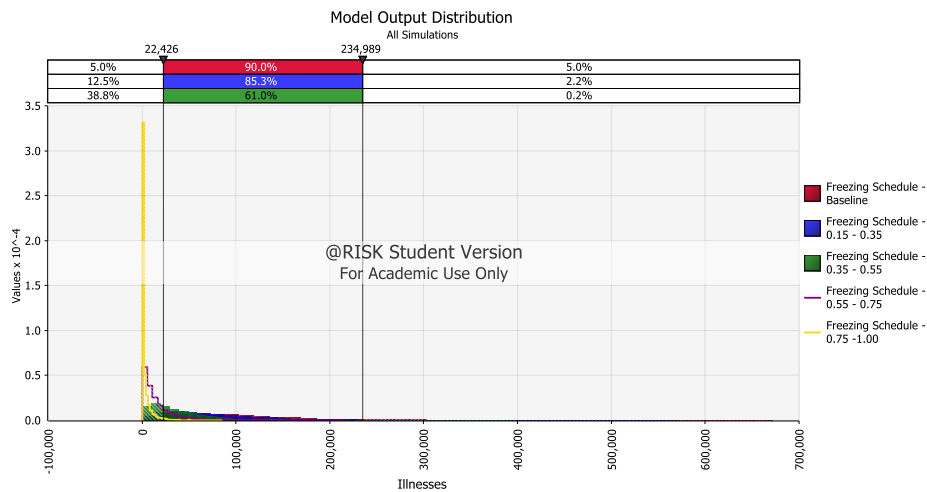


Table III. 4D. – N illnesses / year (D53)

Scenario	Mean	Median	5% CI	95% CI	Min	Max	Δ magnitude, Baseline
1, Baseline	103,616	88,513	22,426	234,989	1,657	671,707	-----
2	76,936	63,413	13,715	187,754	1,449	572,126	-1.347
3	39,461	29,840	4,780	105,606	9	393,762	-2.626
4	15,802	9,814	476	51,108	1	219,491	-6.557
5	2,822	586	0	13,264	0	84,786	-36.717

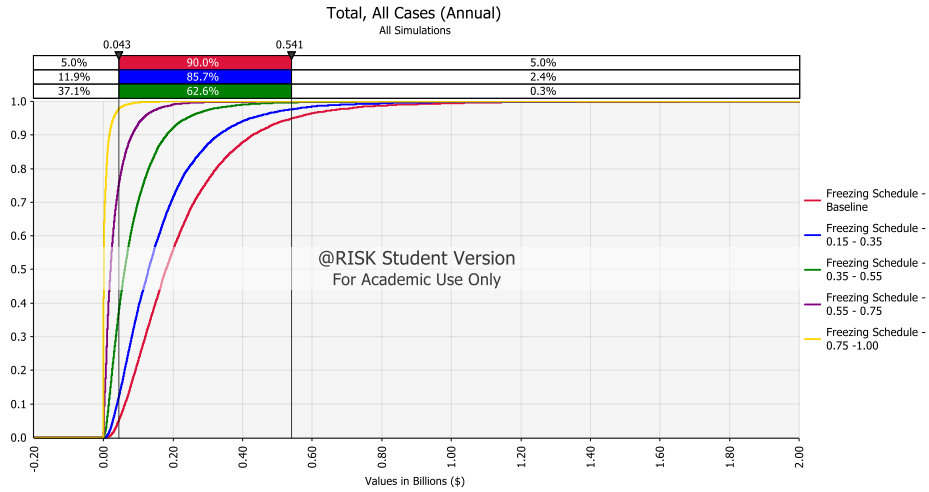


Table III. 4E. – Total Costs - Acute / year (C77)

Scenario	Mean	Median	5% CI	95% CI	Min	Max	Δ magnitude, Baseline
1, Baseline	\$222,515,505	\$179,250,505	\$43,289,299	\$541,090,951	\$3,681,711	$\$1.949 \times 10^9$	-----
2	\$165,064,389	\$129,563,053	\$26,588,092	\$421,895,437	\$1,720,178	$\$1.571 \times 10^9$	-1.348
3	\$84,674,345	\$60,785,289	\$9,435,054	\$238,344,087	\$3,033	$\$1.041 \times 10^9$	-2.628
4	\$33,813,696	\$20,137,950	\$1,039,309	\$113,125,325	\$251	\$518,883,841	-6.581
5	\$5,846,130	\$773,520	\$0	\$28,225,526	\$0	\$217,301,751	-38.062

Tables: III. 5A. – 5D.: Cross-Contamination from Changes in Handwashing during Preparation

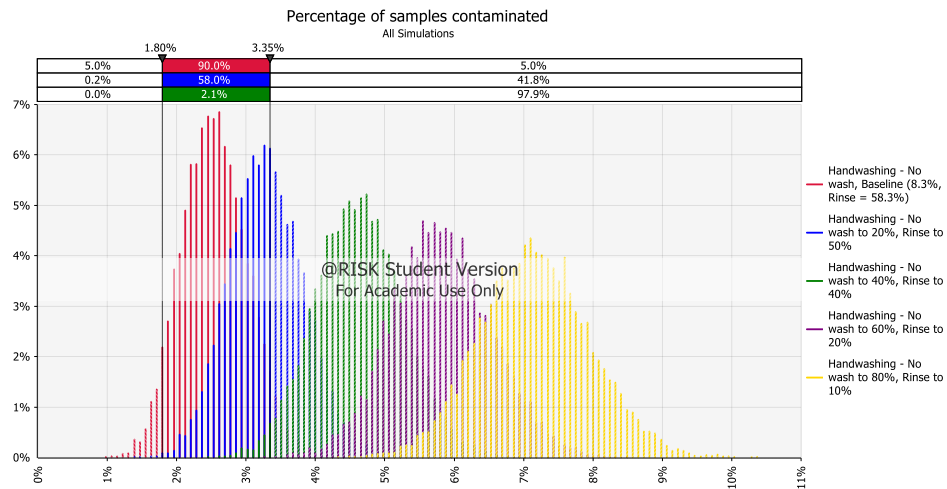


Table III. 5A. – Percentage of Samples Contaminated, Preparation and Handling (C183)

Scenario	Mean	Median	5% CI	95% CI	Min	Max	Δ magnitude, Baseline
1, Baseline	2.549%	2.531%	1.796%	3.34%	0.980%	4.490%	-----
2	3.266%	3.266%	2.449%	4.163%	1.388%	5.388%	+1.275
3	4.639%	4.6531	3.592%	5.714%	2.449%	7.189%	+1.820
4	5.822%	5.796%	4.653%	7.020%	3.265%	8.490%	+2.284
5	7.165%	7.184%	5.878%	8.489%	4.327%	10.37%	+2.811

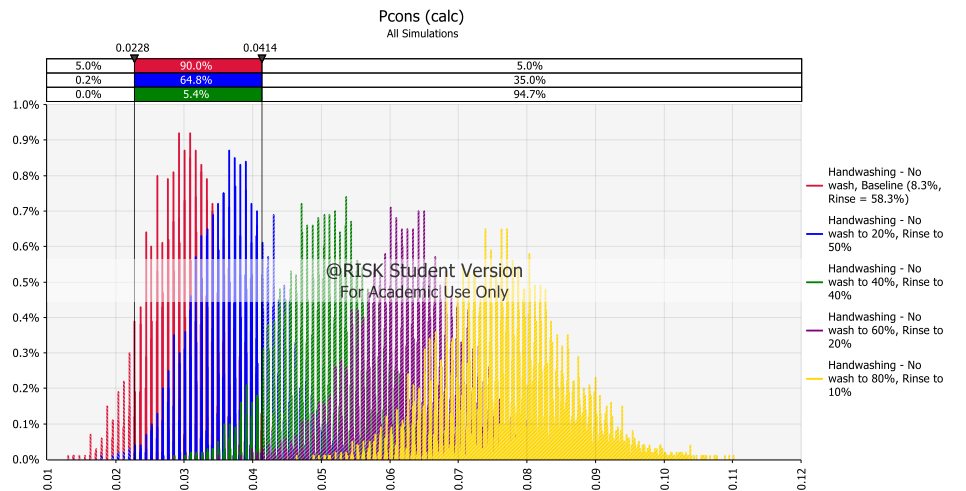


Table III. 5B. – Ingestion of *Campylobacter* in a Meal – Pcons(calc) (C6)

Scenario	Mean	Median	5% CI	95% CI	Min	Max	Δ magnitude, Baseline
1, Baseline	0.0316	0.0316	0.0221	0.0414	0.0130	0.0557	-----
2	0.0390	0.0390	0.0285	0.0495	0.0179	0.0615	+1.234
3	0.0523	0.0520	0.0407	0.0647	0.0285	0.0793	+1.655
4	0.0641	0.0640	0.0512	0.0777	0.0350	0.0929	+2.028
5	0.0774	0.0771	0.0634	0.0923	0.0464	0.1102	+2.449

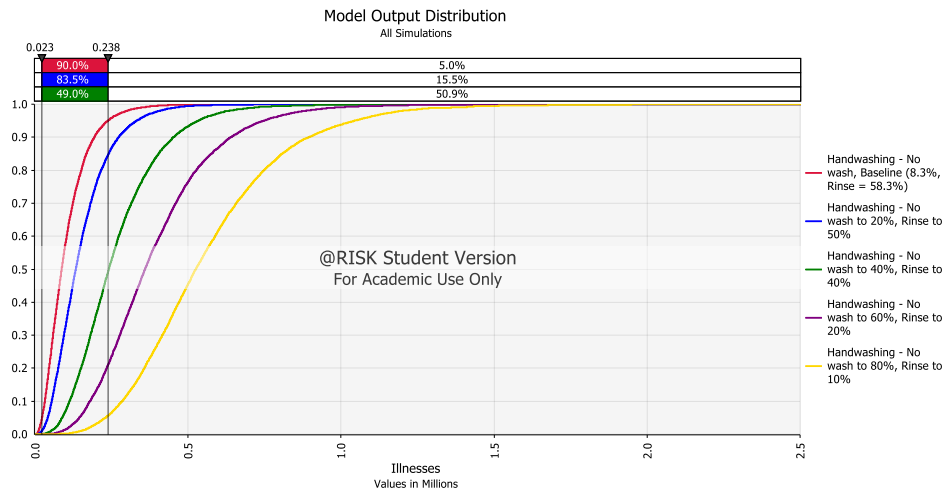


Table III. 5C. – N illnesses / year (D53)

Scenario	Mean	Median	5% CI	95% CI	Min	Max	Δ magnitude, Baseline
1, Baseline	103,046	86,993	22,627	238,247	2,430	607,130	-----
2	152,282	132,850	41,372	331,986	6,506	888,912	+1.478
3	266,956	214,234	88,472	532,353	9,954	1,306,439	+2.591
4	391,558	359,149	144,502	746,722	24,795	1,774,702	+3.800
5	565,805	524,877	229,437	1,043,033	49,152	2,411,500	+5.491

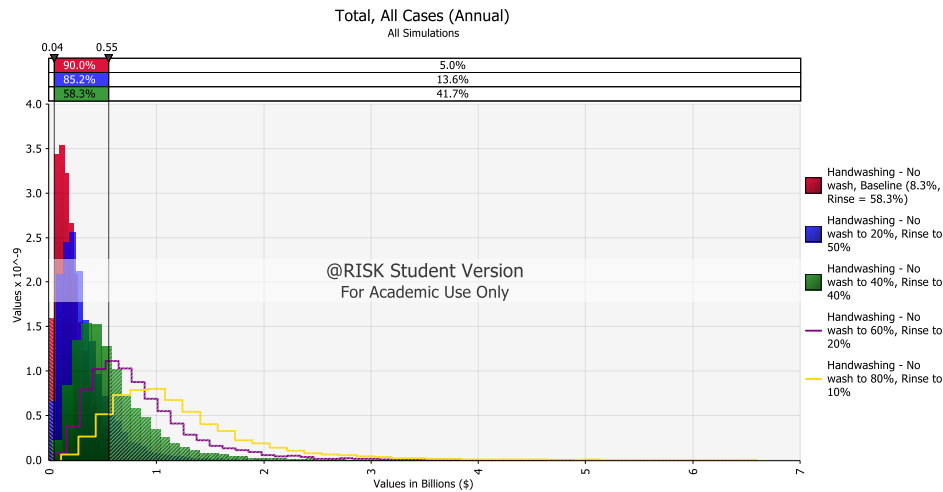


Table III. 5D. – Total Costs - Acute / year (C77)

Scenario	Mean	Median	5% CI	95% CI	Min	Max	Δ magnitude, Baseline
1, Baseline	\$222,021,997	\$176,109,828	\$44,119,718	\$552,103,229	\$6,812,997	\$1.750 x 10 ⁹	-----
2	\$328,240,549	\$266,619,587	\$78,128,274	\$784,535,817	\$12,254,182	\$2.397 x 10 ⁹	+1.478
3	\$575,370,229	\$486,873,675	\$166,209,332	\$1.278 x 10 ⁹	\$28,405,146	\$3.679 x 10 ⁹	+2.592
4	\$843,138,569	\$731,157,643	\$270,040,537	\$1.823 x 10 ⁹	\$37,721,771	\$4.895 x 10 ⁹	+3.798
5	\$1.219 x 10 ⁹	\$1.064 x 10 ⁹	\$423,031,075	\$2.564 x 10 ⁹	\$107,023,330	\$6.598 x 10 ⁹	+5.491

TABLES: III. 6A. – 6D.: Combination of Interventions – Reduce On-farm *Campylobacter* Concentrations and Increase In-Plant Freezing

Table III. 6A. – Change in *Campylobacter* Occurrence during In-Plant Storage (C88) with Reduced On-farm *Campylobacter* Concentrations and Increased In-Plant Freezing

Scenario	Concentration (log CFU/bird); Freeze Schedule, Proportion	Combination Mean	Farm Concentration Mean	In-Plant Freezing Mean	Combination Δ magnitude	Farm Concentration Δ magnitude	In-Plant Freezing Δ magnitude
1, Baseline	~4.6; 0.138	0.636	0.636	0.638	-----	-----	-----
2	3.5–4.5; 0.15-0.35	0.552	0.611	0.578	-1.152	-1.041	-1.104
3	2.5–3.5; 0.35-0.55	0.384	0.558	0.476	-1.656	-1.140	-1.340
4	1.5–2.5; 0.55-0.75	0.228	0.519	0.373	-2.789	-1.225	-1.710
5	0.5–1.5; 0.75-1	0.079	0.495	0.258	-8.051	-1.285	-2.472

Table III. 6B. – Change in *Campylobacter* Concentrations during In-Plant Storage (C89) with Reduced On-farm *Campylobacter* Concentrations and Increased In-Plant Freezing

Scenario	Concentration (log CFU/bird); Freeze Schedule, Proportion	Combination Mean	Farm Concentration Mean	In-Plant Freezing Mean	Combination Δ magnitude	Farm Concentration Δ magnitude	In-Plant Freezing Δ magnitude
1, Baseline	~4.6; 0.138	2.441	2.442	2.437	-----	-----	-----
2	3.5–4.5; 0.15-0.35	1.930	1.995	2.373	-3.243	-2.799	-1.159
3	2.5–3.5; 0.35-0.55	1.069	1.270	2.231	-23.550	-14.859	-1.607
4	1.5–2.5; 0.55-0.75	0.247	0.643	2.028	-156.315	-62.951	-2.564
5	0.5–1.5; 0.75-1	-0.748	0.166	1.571	-1,545	-188.799	-7.345

Table III. 6C. – Change in mean number illnesses/year (D53) with Reduced On-farm *Campylobacter* Concentrations and Increased In-Plant Freezing

Scenario	Concentration (log CFU/bird); Freeze Schedule, Proportion	Combination Mean	Farm Concentration Mean	In-Plant Freezing Mean	Combination Δ magnitude	Farm Concentration Δ magnitude	In-Plant Freezing Δ magnitude
1, Baseline	~4.6; 0.138	104,307	102,356	103,616	-----	-----	-----
2	3.5–4.5; 0.15-0.35	27,055	35,678	76,936	-3.855	-2.869	-1.347
3	2.5–3.5; 0.35-0.55	1,675	4,083	39,461	-62.273	-25.069	-2.626
4	1.5–2.5; 0.55-0.75	98	471	15,802	-1,064	-217.316	-6.557
5	0.5–1.5; 0.75-1	6	76	2,822	-17,385	-1346.789	-36.717

Table III. 6D. – Change in Total Costs (Acute)/year (C77) with Reduced On-farm *Campylobacter* Concentrations and Increased In-Plant Freezing

Scenario	Concentration (log CFU/bird); Freeze Schedule, Proportion	Combination Mean	Farm Concentration Mean	In-Plant Freezing Mean	Combination Δ magnitude	Farm Concentration Δ magnitude	In-Plant Freezing Δ magnitude
1, Baseline	~4.6; 0.138	\$224,759,387	\$220,204,195	\$222,515,505	-----	-----	-----
2	3.5–4.5; 0.15-0.35	\$58,168,643	\$76,822,054	\$165,064,389	-3.864	-2.866	-1.348
3	2.5–3.5; 0.35-0.55	\$3,248,003	\$8,530,287	\$84,674,345	-69.199	-25.814	-2.628
4	1.5–2.5; 0.55-0.75	\$132,740	\$784,979	\$33,813,696	-1,693	-280.522	-6.581
5	0.5–1.5; 0.75-1	\$5,248	\$96,579	\$5,846,130	-42,828	-2280.042	-38.062

Tables: III. 7A. – 7B. Comparison of Alternative Scenarios

Scenario Level	Reduce on Farm Prevalence			Reduce on Farm Concentration			Freezing for in Plant Storage			No Handwashing during Preparation		
	% bird prevalence	Illnesses/ year	Health costs/ year (Million \$)	log CFU/ carcass	Illnesses/ year	Health costs/ year (Million \$)	% filets frozen	Illnesses/ year	Health costs/ year (Million \$)	preparers % no wash	Illnesses/ year	Health costs/ year (Million \$)
1 (baseline)	65 - 80	103,989	224.8	4.6	102,356	220.2	13.8	103,616	222.5	8.3	103,046	222.0
2	50 - 65	80,526	174.3	3.5 - 4.5	35,678	76.8	15 - 35	76,936	165.1	20	152,282	328.2
3	35 - 50	57,631	124.4	2.5 - 3.5	4,083	8.5	35 - 55	39,461	84.7	40	266,956	575.4
4	20 - 35	35,501	76.8	1.5 - 2.5	471	0.785	55 - 75	15,802	33.8	60	391,558	843.1
5	0.05 - 20	15,017	32.4	0.5 - 1.5	76	0.0966	75 - 100	2,822	5.8	80	565,805	1219

Table III. 7A. – Comparison in Illnesses/Year and Health Costs/year for Individual Alternative Scenarios (Excluding Transport Compliance)

Scenario Level	Reduce on Farm Prevalence			Reduce on Farm Concentration			Freezing for in Plant Storage			No Handwashing during Preparation		
	% bird prevalence	GBS / year	IBS / year	log CFU/ carcass	GBS / year	IBS / year	% filets frozen	GBS / year	IBS / year	preparers % no wash	GBS / year	IBS / year
1 (baseline)	65 - 80	93	9132	4.6	91	8,992	13.8	92	9,102	8.3	92	9,050
2	50 - 65	73	7072	3.5 - 4.5	32	3,134	15 - 35	68	6,759	20	135	13,374
3	35 - 50	52	5061	2.5 - 3.5	4	359	35 - 55	35	3,466	40	237	23,444
4	20 - 35	32	3118	1.5 - 2.5	0.381	41	55 - 75	14	1,388	60	347	34,387
5	0.05 - 20	13	1319	0.5 - 1.5	0.049	7	75 - 100	3	248	80	501	49,690

Table III. 7B. – Comparison in Sequelae (GBS and IBS) implicated for campylobacteriosis for Individual Alternative Scenarios (Excluding Transport Compliance)

SUMMARY

Quantitative microbial risk assessment (QMRA) is a powerful analytic tool which can be utilized to model the dynamics between the food pathogen and food commodity, where it provides insight into the impacts of the process in its interaction and its surrounding environment. If designed properly and the inputs used in modelling is robust and accurate, the risk assessment model can be an efficient and effective method to estimate the mitigation steps necessary to control food safety hazards.

The first chapter provided an overall scope of the food safety problem, in the form of the risk of *Campylobacter* ingestion on boneless, skinless chicken breast filets prepared and consumed in the home. An in-depth literature review was conducted – covering a wide range in scope in describing the microbial pathogen, the disease, its impact to society, and the role it plays within the chicken industry. A literature review was also conducted in earnest on the production, processing, storage, and preparation of the broiler chicken and ultimately the meal. These analyses provided the collection of empirical data to compile the necessary inputs that would drive a comprehensive QMRA, which was constructed to identify the mechanisms in the characterization of the hazard. This is described in Chapter II. The model is comprehensive in its analysis and employs a second-order analysis, where variability and uncertainty are separated. The major benefit of this risk assessment model is that it is constructed in a series of interlinked modules and sub-modules, which can be swapped out and the data updated in a logical fashion.

Overall, it was expected that the campylobacteriosis due to the consumption of boneless, skinless chicken breast meal on an annual basis in the contiguous United States is ~ 100,000 – 110,000 cases. Among those cases, approximately 27,000 hospitalizations, and 40

deaths are estimated. Campylobacteriosis-associated secondary illnesses are as follows on an annual basis: ~1,300 cases of GBS and ~9,500 cases of IBS. The total economic burden associated with this risk assessment is estimated to be ~ \$366 million annually.

Chapter III posits what to do with the data generated in the QMRA. This process is ultimately what is needed for policy analysis in government and industry. The effects of modifying the baseline model's inputs within the farm-to-fork process were studied as follows: Post grow-out (1) prevalence and (2) concentration of *Campylobacter* on chickens at the farm prior to transport, 3) transport crate cleaning frequency prior to loading, 4) the storage conditions post processing/ pre-retail, and 5) the frequency of handwashing in the preparation and handling of a chicken meal, and 6) the combination *Campylobacter* mitigation models using the inputs from (2) and (3).

The effects of these interventions varied depending on the location and intensity of the mitigation steps. The mean yearly illnesses are estimated to decrease by half if on-farm *Campylobacter* prevalence was lowered to 35 – 50% from the baseline. Moreover, it was established the combined effects of reducing on-farm *Campylobacter* concentrations and increasing in-plant freezing proportion have greater impacts on reducing yearly campylobacteriosis and associated burdens than either intervention alone. On the flip-side of the equation, the importance of cross-contamination during the preparation of a meal was modelled. An additional ~50,000 cases of campylobacteriosis/year are expected if the non-washing in the preparation of the meal increases from 8.3% (baseline) to 20%.

Appendix A. Input Distributions and Calculations for @RISK based model

<i>Processing Tabbed Sheet</i>			
Farm:	Cell	Model Input	Notes / References
<u>Default Prevalence</u>	C6	=RiskUniform(0.65,0.8)	The probability of a Campylobacter-contaminated bird on the farm
<u>Number sampled</u>	C3	=COUNT(C10:AUE10)	Aggregation of food pieces observed
Contamination of Carcasses:	Cell	Model Input	Notes / References
<u>Prevalence (Exterior/Skin)</u> 0 = No, 1 = Yes	C15	=@RiskBinomial(1,Default_Prevalence,RiskName("Prevalence, farm - Exterior"))	Probability of a contaminated bird, where 0 = negative bird, 1 = positive bird
<u>Campylobacter concentration (log CFU/carcass):</u>	C16	=IF(C15=1,-0.435*LN(Default_Prevalence)+@RiskWeibull(12.543,7.0643,RiskName("Preliminary C(ext) log CFU/carcass"),RiskShift(-2.5494),RiskTruncate(1.8)),0)	
Transport:	Cell	Model Input	Notes / References
<u>Crates washed and sanitized prior to loading? 0 = No, 1 = Yes</u>	C26	=@RiskBinomial(1,Transport_Cage_Wash_Compliance,RiskName("Crate washed and sanitized"))	• U.S. survey found 28.4% of respondents wash and sanitize transportation cages and trucks between each use. • Refers to "Compliance" box (F38) • 0 = Crates not washed/sanitized prior to loading birds • 1 = Crates washed/sanitized prior to loading birds (Northcutt and Jones 2004)
<u>Development of Off-Feed/Transport Times</u>			Truncated to 11 hours, simulation of 10000 iterations gives max holding time of 13 hours – supplemental model developed. (Wesley 2009)

<u>Loading Time (Hours)</u>	C27	=@RiskNormal(0.816667,0.057155,RiskName("Loading time (h)"))	Loading time for chickens for transport from farm
<u>Transit Time (Hours)</u>	C28	=RiskBetaGeneral(2.7338,1.6183,0,4,RiskName ("Transit time (h)"))	Transit time for chickens to processing facility
<u>Holding Time (Hours)</u>	C29	=RiskInvgauss(1.8612,1.1424,RiskName("Holding time (h)"), RiskShift(2.9438),RiskTruncate(,11))	Holding time for chickens to be unloaded at processing facility after transport
<u>Time in Cages (hours)</u>	C30	=SUM(C27:C29)	Total holding time from farm to processing plant
<u>Occurrence of Cages Contaminated Campylobacter</u>	C31	=@IF(C26=0,RiskBinomial(1,RiskBeta(8,21),RiskName("Transport crates unwashed and positive for Campy")),IF(C26=1,RiskBinomial(1,RiskBeta(3,26),RiskName("Transport crates washed and positive for Campy"))))	Depending on outcome of "Crates washed and sanitized prior to loading? 0 = No, 1 = Yes" a Beta binomial distribution is used to estimate if the crates are contaminated with <i>Campylobacter</i> (Northcutt and Berrang 2006)
<u>Increase (External) - if previously negative</u>	C32	=IF(AND(C15=0,C31=1),((0.05*C30/2)^2-(0.05*C30/2)+3.15),IF(AND(C15=0,C31=0),0,"NA"))	*(Berrang, Northcutt et al. 2003) *Assumption that if the bird is already harboring <i>Campylobacter</i> and is put in contaminated cages, there is an increase to the exterior concentration, however, the effect of the concentration is not as great as seen with negative birds. Due to transfer of pathogen from the bird to the cage as well as the transfer from the cage to the bird, a uniform distribution (0.7-0.9) is used to show this effect. *If the bird sampled is negative at the farm but is cross-contaminated in the transfer cage, the full transfer from the cage to the bird is used.
<u>Change due to transfer of Campylobacter on previously positive birds</u>	C33	=@IF(C15=1,RiskBinomial(1,RiskBeta(12,3)),IF(C15=0,"NA"))	If "Change" from previous row is 1, then the interaction between cage and birds to the birds will result in an increase in concentration. If "Change" from previous row is 0, the resulting interaction will reflect a decrease in concentration due to bird(x) giving off <i>Campylobacter</i> to the environment. (Stern, Clavero et al. 1995)
	C34	=IF(C33=1,RiskNormal(2.49,1.343,RiskTruncate(0.35,3.55)),IF(C33=0,-@RiskNormal(0.29,0.198),"NA"))	

Scalding:	Cell	Model Input	Notes / References
<u>Reduction in contamination, log CFU/carcass</u>	C63	=@RiskOutput("Reduction in contamination, log CFU/carcass")+IF(SIMTABLE_Processing=10,-@RiskNormal(3.1,0.58),IF(SIMTABLE_Processing=20,-@RiskNormal(3.1,0.58)))	*~3 log reduction. * (Berrang and Dickens 2000) - Data: Prescald count (avg) of 4.73 log CFU/carcass, post-scald count of 1.80 log CFU/carcass. * Uncertainty – two levels of reduction as presented in SIMTABLE_Processing
Defeathering:	Cell	Model Input	Notes / References
<u>Increase or Decrease by Effect of Defeathering (0 = Increase, 1 = Decrease)</u>	C76	=@RiskBinomial(1,RiskBinomial(1,RiskBeta(4+1,17-4+1)))	• (Berrang and Dickens 2004) and (Seliwiorstow, Baré et al. 2015) • (Seliwiorstow) Of the 12 batches sampled throughout four processing plants, 4 batches resulted in a reduction of Campylobacter "After plucking" when compared to "After bleeding" • (Berrang) All five batches samples in the study showed increase in contamination when compared to pre-picking. • Each sampling or batch considered equivalent (6 chickens vs 10 chickens)
<u>Change in contamination, log CFU/carcass</u>	C77	=@IF(C76=0,RiskWeibull(9.2976,10.38,RiskShift(-8.3961),RiskTruncate(-4.7),RiskName("Defeathering, Increase set")),RiskNormal(-1.02747,0.49678,RiskName("Defeathering, Reduction set")))	Increase due to cross-contamination in defeathering (Berrang and Dickens 2004)
Evisceration:	Cell	Model Input	Notes / References
<u>Change in contamination, log CFU/carcass</u>	C95	=IF(AND(C80>0,C80<3.4),RiskLognorm(1.163,0.015193,RiskName("Avg log reduction / Flock 2 (1.428-2.400)")),RiskShift(-0.66324)),IF(AND(C80>=3.4,C80<5.3),RiskLognorm(0.93601,0.0096039,RiskName("Avg log reduction / Flock 1 (2.438-	• Refers to "C(defeather), log CFU/carcass" (C80) • (Rosenquist, Sommer et al. 2006) • (Berrang and Dickens 2000) can also be supplemented.

		3.054")),RiskShift(-0.45596)),IF(C80>=5.3,RiskNormal(-0.009907,0.0062792,RiskName("Avg log reduction (combined for flocks 3 & 4))),0)))	
Washing and Chilling:	Cell	Model Input	Notes / References
<u>Type of On-line reprocessing chemical used</u>	C112, C113	=@RiskDiscrete({10,20,30,40,50,60,70},Chemical_Used__Washing_Chilling,RiskName("Type of On-line reprocessing chemical used"),RiskCollect()) =IF(C112=10,"HAS",IF(C112=20,"PA",IF(C112=30,"ASC",IF(C112=40,"FGA",IF(C112=50,"TSP",IF(C112=60,"CD","None")))))	Links to "Type of On-line reprocessing chemical used" (C112) Linked to "Chemical Used, Proportion" (E124 – E129, F124 – F129)
<u>Reduction in Contamination Due to Washing (log) - Chemicals & Water Chilling</u>	C114	=@RiskOutput("Reduction due to Chemical Washing and Chilling (log)")+IF(C113="HAS",-@RiskNormal(1.47,0.26),IF(C113="PA",-@RiskNormal(1.79,0.17),IF(C113="ASC",-@RiskNormal(2.33,0.14),IF(C113="FGA",-@RiskNormal(1.32,0.15),IF(C113="TSP",-@RiskNormal(2.97,0.07),IF(C113="CD",-@RiskNormal(2.41,0.24),-@RiskNormal(1.78,0.15)))))))	(Berrang, Bailey et al. 2007)
<u>Use of Chlorinated water in chilling</u>	C115, C116	=@RiskBinomial(1,RiskBeta(16+1,20-16+1)) =IF(C115=1, "Yes", "No")	Of the 20 plants visited, 16 used chilling w/ chlorinated water
<u>Cross Contamination Due to Water Chilling</u>	C117, C118	=@IF(C115=1,RiskBinomial(1,RiskBeta(2,8),RiskName("Cross contamination due to water chilling"),RiskCollect()),0) =IF(C117=1, "Yes", "No")	- Refers to "Use of Chlorinated water in chilling 0 = No, 1 = Yes" (C105), Reference: (Rosenquist, Sommer et al. 2006) - FAO/WHO data combined with two data results for chilling in (Rosenquist, Sommer et al. 2006) - FAO: Decrease 5/6 times, increase 1/6; Rosenquist, decrease 2/2 times
<u>Increase due to cross-contamination log CFU/carcass</u>	C119	=@IF(C117=1,RiskNormal(0.45,0.045),0)	Refers to "Cross Contamination Due to Water Chilling" (C117)

<u>C(wash), log CFU/carcass</u>	C120	=IF(C97+C114+C119>9,9,IF(C97+C114+C119>0,C97+C114+C119,0))	Refers to “C(evisc), log CFU/carcass (C97), “Reduction in Contamination due to Washing (log) – chemicals and water chilling” (C114), “Increase Due to Cross-Contamination log CFU/carcass” (C119);
Portioning to Breast Filets:	Cell	Model Input	Notes / References
<u>Carcass Weight (g)</u>	C148	=@RiskUniform(1130,1700)	* As per Agricultural Marketing Service, USDA - "Consumer Pack Cut-Up(205050)", "Cut-up(205140)", "Breast and Leg Quarters (221140)" -..."Must be produced from ready-to-cook broiler/fryer chickens which weigh 2.50 to 2.75 pounds (1.13-1.70 kg) each, without neck and giblets.
<u>Filet Size (g)</u>	C149	=C148*(@RiskUniform(0.06,0.08))	Refers to “Carcass Weight (g)” (C148) One portion equivalent to 6 – 8% of total carcass weight Assumption to match regular filet size
<u>Redistribution of <i>Campylobacter</i> N(split) CFU/filet</u>	C150	=IF(C121>0,ROUND(C121*(C149/C148),0),0)	The redistribution is set up so that there is a proportionality of contamination from carcass to filet (assumption) Transfer rate of 10% - taken from WTO model, "Level of contamination at slaughter for negative flocks – transport. (World Health Organization 2000) * Handling of chickens through splitter and/or manual contact through personnel
<u>Reduction of <i>Campylobacter</i> due to removal of skin (log)</u>	C153	=IF(C120>0,C120* -@RiskNormal(0.22,0.02),0)	
<u>Cross Contamination Due to Environment? 1 = Yes, 0 = No</u>	C152	=RiskBinomial(1,RiskBeta(15,5))	

<u>Increase in <i>Campylobacter</i> due to cross-contamination from Environment (log)</u>	C155	=IF(C154=1,RiskNormal(0.27,0.027),0)	Refers to “Cross Contamination Due to Environment?” (C152) Assumptions made in redistribution flow through this section - cross-contamination from splitter & worker hands/utensils handling of processed filets
In-Plant Storage:	Cell	Model Input	Notes / References
<u>Refrigerated/Chilled? Frozen?</u> 0 = Chilled, 1 = Frozen	C172, C173	=@RiskOutput("Refrigerated/Chilled? Frozen?")+@RiskBinomial(1,Frozen_Prob_In_Plant_Baseline) =IF(C172=1,"Frozen","Chilled")	Links to “Frozen/Chilled – Probability (E180 – E181, F180 – F181)
<u>Number of Days</u>	C174	=IF(C173="Chilled",RiskUniform(0.3,1.5),RiskPert(1,16,31))	Assumption that refrigerated chickens stay in plant storage from 0.3 – 1.5 days and frozen chickens stay in retail storage from 1 - 31 days, with most chickens being shipped by the 16th day
<u>Reduction (log)</u>	C175	=IF(C173="Frozen",- (0.4991*LN(C174)+0.3358),- (0.0018*C174^3 - 0.0357*C174^2 + 0.2462*C174))	- Reduction derived from "Freezing and chilling Trendline" worksheet - Reference: (Georgsson 2006) (Freezing and chilling); (Davis 2007) (Chilling) - Combination of short-term and long-term data
<u>C log CFU / filet (storage)</u>	C176	=IF(C156+C175>0,C156+C175,0)	Refers to “Adjusted <i>Campylobacter</i> due to removal and cross-contamination log CFU/filet” (C156) and “Reduction (log)” (C175)

Handling → Consumption Tabbed Sheet

RETAIL AND HANDLING	Cell	Model Input	Notes / References
<u>Number sampled</u>	C3	=COUNT(C10:AUE10)	Aggregation of food pieces observed
Transport to Retail & Retail Storage:	Cell	Model Input	Notes / References
<u>Delivered from Plant Frozen or Chilled</u>	C11	=IF(Processing!C173="Chilled","Chilled","Frozen")	- Links for C173 of "Processing" - Assumed that the storage condition from processing plant is carried over to the transport and retail storage
<u>Number of Days in Retail Storage</u>	C12	=IF(C11="Frozen",RiskUniform(0.5,45,RiskName("Retail Storage - Freezing")),RiskCollect()),RiskUniform(0.5,8,RiskName("Retail Storage - Chilling"),RiskCollect()))	Assumption that frozen chickens stay in retail storage from 0.5 – 45 days and chilled chickens stay in retail storage from 0.5 – 8 days post-delivery from processing facility
<u>Aggregate number of days in storage (in-plant, transport, retail)</u>	C13	=Processing!C174+C12	Adds total number of days in processing facility and retail facility
<u>Reduction from finished product in-plant</u>	C14	=IF(C11="Frozen",- (0.499*LN(C13)+0.3358),- (0.0018*C13^3-0.0357*C13^2+0.2462*C13))	- Links to "Deliver from Plans Frozen or Chilled" (C11) - D-value derived from frozen and chilled environment
<u>Total reduction due to plant and retail storage (log CFU/filet)</u>	C15	=(Processing!C175+C14)	Links to "Reduction (log)" (C175, Processing); and Reduction from finished product in-plant (C14)
Home Storage:	Cell	Model Input	Notes / References
<u>Consumed at Day 0? Frozen? Chilled?</u>	C38, C39	=@IF(C11="Frozen",RiskDiscrete({0,10,20},Frozen_Retail_Purchase,RiskName("Storage, home -	(Assumption) Pathways for home storage, where 0 = product consumed day of purchase 10 = product stored frozen 20 = product refrigerated

		frozen")),RiskDiscrete({0,10,20},Chilled_Retail_Purchase,RiskName("Storage, home - chilled")) =IF(C38=0,"Day 0",IF(C38=10,"Frozen","Chilled"))	(Assumption) Products purchased frozen or chilled, if not consumed the day of purchase may be stored in the same condition as purchase or may be stored under different conditions Discrete distribution refers to conditional probability (point distribution) on the dialogue boxes (F49:52 – if bought frozen; F56:58 – if bought chilled)
<u>If not consumed on purchase date, duration of storage at home (Days)</u>	C40	=IF(C38=10,RiskUniform(1,31),IF(C38=20,RiskUniform(1,3),0))	- Duration of home storage if not consumed day of purchase - Assume that most frozen chickens are consumed within 31 days (1 month) - Recommend that chilled chickens be consumed within 1-3 days post purchase - Uniform distribution - frozen (1 - 31 days), chilled (1 - 3 days)
<u>Aggregate number of days in storage (Until preparation and consumption)</u>	C41	=IF(AND(C11="Chilled",C39="Chilled"),C13+C40,IF(AND(C11="Frozen",C39="Frozen"),C13+C40,IF(C39="Day 0",0,"NA")))	- Refers to “Delivered from Plant Frozen or Chilled” (C11); “Consumed at Day 0? Frozen? Chilled? (C39); “Aggregate number of days in storage (in-plant, transport, retail)” (C13); “If not consumed on purchase date, duration of storage (Days)” (C40) - Adds total number of days in storage
<u>Reduction from finished product in-plant</u>	C42	=IF(AND(C11="Frozen",C39="Frozen"),-(0.499*LN(C41)+0.3358),IF(AND(C11="Chilled",C39="Frozen"),-(0.499*LN(C40)+0.3358),IF(AND(C11="Chilled",C39="Chilled"),-(0.0018*C41^3 - 0.0357*C41^2 + 0.2462*C41),IF(AND(C11="Frozen",C39="Chilled"),-(0.0018*C40^3 - 0.0357*C40^2 + 0.2462*C40),0))))	- Refers to “Delivered from Plant Frozen or Chilled” (C11); “Consumed at Day 0? Frozen? Chilled? (C39); “If Not Consumed on Purchase Date, Duration of Storage (Days) (C39); “Aggregate number of days in storage (Until Preparation and Consumption)” (C40) - D-values for freezing and chilled environment
<u>Total Reduction from "Storage (Plant, Retail, Home)" module (log CFU/filet)</u>	C43	=IF(C39="Day 0",0,(C15+C42))	Refers to “Total reduction due to plant and retail storage (log CFU/filet)” (C15); “Reduction from finished product in-plant” (C42)

			Shows possible reduction from just home storage phase
<u>C(s,home) log CFU/filet</u>	C44	=IF(C16+C42>0,C16+C42,0)	- Refers to “Delivered from Plant Frozen or Chilled” (C16); “Consumed at Day 0? Frozen? Chilled? (C42); - Tabulates reduction and changes concentration in log magnitude
<u>N(s,home) CFU/filet</u>	C44	= IF(C44>0,ROUND(10^C44,0),0)	
Cross-Contamination from Outside Package to Hands:	Cell	Model Input	Notes / References
Occurrence of contamination	C80, C81	=@IF(C11="Chilled",RiskBinomial(1,RiskBinomial(1,RiskBeta(272+1,4005-272+1))),0) =IF(C80=1,"Yes","No")	Outside package cross-contamination is considered only on chilled retail products.
<u>Tier of contamination</u>	C82	=RiskDiscrete({1,2,3},{0.768,0.213,0.0183})	- Refers to the level of contamination (point estimates) initially on the surface of package where: 1 = probability of 0.768, with 10 – 99 cells 2 = probability of 0.213, with 100 – 1000 cells 3 = probability of 0.018, with 1,001 – 10,000 cells - Upper bound of 100,000 CFU/package - Uses dialogue boxes (E91:G91, level 1, E92:G92, level 2, E93:G93, level 3) (UK Food Safety Agency, 2016) (Byrd-Bredbenner, Berning et al. 2013)
<u>Contamination on Outside of Package (Log CFU/package) - initial</u>	C83	=IF(AND(C81="Yes",C82=1),LOG(@RiskUniform(10,99),10),IF(AND(C81="Yes",C82=2),LOG(@RiskUniform(100,1000),10),IF(AND(C81="Yes",C82=3),LOG(@RiskUniform(1000,100000),10),0)))	(UK Food Safety Agency, 2016)
<u>Reduction due to Storage (log CFU/package) - Home</u>	C84	=IF(AND(C11="Chilled",C39="Chilled"),-(0.0018*C40^3-0.0357*C40^2+0.2462*C40),0)	Accounts for further storage in refrigeration at home.

<u>Contamination on Outside of Package (log CFU/package) - Final</u>	C85	=IF(C83>0,C83+C84,0)	Refers to Contamination on Outside of Package (Log CFU/package) – initial and Reduction due to Storage (log CFU/package) - Home
<u>Transfer N(hands)</u>	C87	=ROUND((C83*@RiskGamma(22.788,0.023925,RiskName("Transfer from packaging to hands"),RiskShift(0.003933))),0)	Uses transfer rate from Cross Contamination from Handling of Filets (transfer to hands)
<u>Hands(package)--->Carcass</u>	C88	=@RiskOutput("Hands(package)--->Carcass")+ROUND((@RiskPearson6(0.63149,2.7351,0.10528,RiskName("Packaging to hands to meat"),RiskShift(0.01045))*C87),0)	Uses transfer rate from Cross Contamination from Handling of Filets (transfer from hands)
Cross Contamination from Handling of Filets	Cell	Model Input	Notes / References
<u>Handling path, Meat--->Hands</u>	C118, C119	=@RiskDiscrete({1,2,3,4},\$E\$124:\$E\$127) =IF(C118=1,"No Wash",IF(C118=2,"Rinse",IF(C118=3,"Wash","NC")))	Handwashing practices probabilities – Normal distribution: “No wash” (0.083,0.0083), “Rinse” (0.583,0.0583), “Wash with soap” (0.25,0.025), and “No contact” (0.083,0.0083) Wash = Wash w. soap Rinse = Rinse No Wash = No Wash NC = No Contact Uses dialogue boxes (C124:E124, level 1, C125:E125, level 2, C126:E127, level 3, C128:E128, level 4) (Luber 2006)
<u>Transfer rate for cross-contamination from meat to hands</u>	C120	=@IF(C118=1,(RiskPearson6(0.63149,2.7351,0.10528,RiskShift(0.01045))),IF(C118=2,(10^@RiskNormal(-6.46,1.26))/(10^@RiskNormal(-2.88,0.68))),IF(C118=3,(10^@RiskNormal(-7.04,0.66))/(10^@RiskNormal(-2.88,0.68)),0)))	(Luber 2006) (van Asselt, de Jong et al. 2008)

Overall number of cells transferred from raw meat to hands	C121	=ROUND((C103+C105)*C120,0)	(Luber 2006) (van Asselt, Fischer et al. 2009)
<u>Handling path, Meat--->Board</u> • If Rinsed (2), 10=Cold, 20=Hot, with 0.5 p for each; If not rinsed 0 = "Other" • Transfer Rate: No Wash; Rinsing (cold),(hot); Washing • Truncation, if necessary (Limit = transfer rate of 0.7)	C130, C131, C132, C133	=@RiskDiscrete({1,2,3,4},\$E\$138:\$E\$141) =IF(C130=1,"No Wash",IF(C130=2,"Rinse",IF(C130=3,"Wash","NC"))) =@IF(C130=2,RiskDiscrete({10,20},\$H\$139:\$H\$140),"Other") =IF(C132=10,(10^@RiskNormal(-3.51,0.43))/(10^@RiskNormal(-3.15,0.49)),IF(C132=20,(10^@RiskNormal(-7.26,0.05))/(10^@RiskNormal(-3.15,0.49)),IF(C132="Other",0,C133))) =IF(C133>0.7,0.7,C133)	• Cutting board cleaning probabilities were designated as Normal distributions (No wash – 0.125,0.0125; Rinse – 0.167,0.0167; Wash with soap and water – 0.042, 0.0042; and No contact – 0.667, 0.0667). Wash = Wash w. soap Rinse = Rinse No Wash = No Wash NC = No Contact Uses dialogue boxes (C138:E138, level 1, C139:E139, level 2, C140:E140, level 3, C141:E141, level 4) • The “rinse” pathway was further divided into rinsing with cold or hot water (probability of each use at 0.5). • The transfer rate was truncated to 0.7 logs to maintain realistic scenarios. (Luber 2006), (Nauta 2010)
<u>Handling path, Meat--->Utensil</u> If Rinsed (2), 10=Cold, 20=Hot, with 0.5 p for each; If not rinsed 0 = "Other" Transfer Rate: No Wash; Rinsing (cold),(hot); Washing, No Contact (NC)	C144, C145, C146, C147	=@RiskDiscrete({1,2,3,4},\$E\$151:\$E\$154) =IF(C144=1,"No Wash",IF(C144=2,"Rinse",IF(C145=3,"Wash","NC"))) =@IF(C144=2,RiskDiscrete({10,20},\$H\$152:\$H\$153),"Other") =IF(C146=10,(10^@RiskNormal(-6.76,0.67))/(10^@RiskNormal(-3.06,0.56)),IF(C146=20,(10^@RiskNormal(-7.17,0.01))/(10^@RiskNormal(-3.06,0.56)),IF(C144=4,"NC","No Wash"))	• Cutting board cleaning probabilities were designated as Normal distributions (No wash – 0.125,0.0125; Rinse – 0.167,0.0167; Wash with soap and water – 0.042, 0.0042; and No contact – 0.667, 0.0667). Wash = Wash w. soap Rinse = Rinse No Wash = No Wash NC = No Contact

		=IF(C144=1,ROUND((@RiskPearson6(0.63149,2.7351,0.10528,RiskName("CFU transfer to knife"),RiskShift(0.01045))*C105),0),IF(C146=10,ROUND((C147*C105),0),IF(C146=20,ROUND((C147*C105),0),0)))	Uses dialogue boxes (C151:E151, level 1, C152:E152, level 2, C153:E153, level 3, C154:E154, level 4) • The “rinse” pathway was further divided into rinsing with cold or hot water (probability of each use at 0.5). (Luber 2006); (van Asselt, Fischer et al. 2009)
<u>Overall number of cells transferred from the raw meat to utensils</u>	C148	=IF(C144=1,ROUND((@RiskPearson6(0.63149,2.7351,0.10528,RiskName("CFU transfer to knife"),RiskShift(0.01045))*C105),0),IF(C146=10,ROUND((C147*C105),0),IF(C146=20,ROUND((C147*C105),0),0)))	(Luber 2006) (van Asselt, Fischer et al. 2009)
<i>Total Transfer from Cross-Contamination, Handling</i>	Cell	Model Input	Notes / References
<u>Total Transfer N(Hands,board,utensil)</u>	C157	=@RiskOutput("Total Transfer N(Hands,board,utensil)") + SUM(C121,C135,C148)	(Luber 2006), (van Asselt, Fischer et al. 2009)
<u>(Hands → Food)</u>	C159	=ROUND((C121*@RiskGamma(22.788,0.023925,RiskName("Hands to Food"),RiskShift(0.003933))),0)	(Luber 2006), (van Asselt, Fischer et al. 2009)
<u>(Board → Food)</u>	C160	=ROUND((C135*@RiskInvgauss(0.026215,0.006511,RiskName("Board to Food"),RiskShift(0.0025741))),0)	(Luber 2006), (van Asselt, Fischer et al. 2009), (Nauta 2010)
<u>(Utensil → Food)</u>	C161	=ROUND((C148*@RiskInvgauss(0.026215,0.006511,RiskName("Knife to Food"),RiskShift(0.0025741))),0)	(Luber 2006), (van Asselt, Fischer et al. 2009)
<u>Contaminated?</u>	C162	=IF(C163>0,"Yes","No")	
<u>N CFU/filet Ingested (Nch)</u>	C163	=@RiskOutput("N CFU/filet Ingested (Nch)") + SUM(C159:C161)	

Internalization of <i>Campylobacter</i> in the muscles of the broiler	Cell	Model Input	Notes / References
<u>Internal Contamination?</u> 0=No, 1=Yes	C209	=@IF(C105>0,RiskBinomial(1,0.23),0)	Carcasses that are negative (surface) prior to cooking (cell C105) are assumed to be negative internally. If positive, the filet has a 0.23 probability of being positive internally Refers to “Final C(home), N/filet (Luber 2006, Luber and Bartelt 2007)
<u>C(Filet,internal)</u>	C210	=IF(C209=1,C104*@RiskPert(0.442,0.491,0.788),0)	Proportion to the internal counts to surface count (Luber 2006, Luber and Bartelt 2007, Agency 2016)
Cooking	Cell	Model Input	Notes / References
<u>D-Value used in Model</u>	C205	=RiskUniform(1.93,1.976,RiskName("D-value for cooking poultry"))	(Luber 2006) (van Asselt, Fischer et al. 2009) Each dataset was weighed equally in developing the parameter estimations. The dataset was bootstrapped for 10,000 iterations, where then data was fitted using the BestFit feature of @Risk using the AIC. Uncertainty of cooking time can be separated using the simulation feature of @Risk.
<u>Cooking under 8 minutes</u>	C211	=@IF(Distribution_Chooser__Cooking=25,RiskBinomial(1,RiskBeta(9,17,RiskName("Cooking time under 8 minutes, Cooking simulation 02"))),"NA")	(van Asselt, Fischer et al. 2009)
<u>Cooking Time (Minutes)</u>	C212	=@IF(Distribution_Chooser__Cooking=15,RiskGamma(3.0325,3.8708,RiskName("Cooking time, simulation 01 (minutes)")),RiskShift(0.303),RiskTruncate(1.5,45),RiskCollect()),IF(AND(Distribution_Chooser__Cooking=25,C211=1),RiskWeibull(3.9142,4.5479,RiskName("Cooking time, <= 8 minutes, simulation 02")),RiskShift(1.5),RiskCollect()),IF(AND(Distribution_Chooser__Cooking=25,C211=0),RiskPearson5(2.5066,12.27,RiskName("Cooking time, >8 minutes, simulation 03")),RiskShift(1.5),RiskCollect()))	“Distribution chooser__Cooking=15” refers to the cooking time as observed in Luber et al. (Luber 2006). The second set, assigned as, “Distribution_chooser__Cooking=25” (Van Asselt, Fischer et al. 2009). The third set, assigned as, “Distribution chooser__Cooking=35” combines datasets “Distribution chooser__Cooking” sets can be run as separate simulations to see the effects using

		02"),RiskShift(7.45),RiskCollect()),IF(Distribution_Chooser__Cooking=35,RiskBetaGeneral(1.229,3.5284,2,45,RiskName("Cooking time, simulation 03"),RiskCollect()))))	these parameters in the model with minimal model manipulation.
Log CFU/filet Reduction (time/D-value)	C213	=-C212/D_Value__Cooking	
Dose Response	Cell	Model Input	Notes / References
<u>Pcons (calc)</u>	C231	=Pcons__handling+pcons_cooking-(Pcons__handling*pcons_cooking)	(Black, Levine et al. 1988) (FAO-WHO 2009)
<u>Pcons (obs)</u>	C232	=COUNTIF(Dose_Response__CFU_ingested_from_product,"Yes")/Total_Samples	
Ingestion of Campylobacter	Cell	Model Input	Notes / References
<u>Total Ingested N(cons)</u>	C235	=@RiskOutput("Total Ingested N(cons)")+SUM(C163,C215)	Adds number of cells ingested from (C163 - handling - cross contamination onto food from hands, plate, utensils) and (C215 - cooking - survivors from cooking process)
Probability of Infection and Illness per dosage ingested	Cell	Model Input	Notes / References
<u>Pinf(D)</u>	C244	=1-(1+C235/59.95)^-0.21	(Teunis, Van den Brandhof et al. 2005), (Black, Levine et al. 1988)
<u>Pinf (from calculated Pcons)</u>	C245	=\$C\$231*C244	(Black, Levine et al. 1988)
<u>Pinf (from observed Pcons)</u>	C246	=\$C\$232*C244	
<u>Pill inf</u>	C247	=@RiskBeta(30,61)	(FAO-WHO 2009)
<u>Pill (from calculated Pcons)</u>	C248	=C245*C247	
<u>Pill (from observed)</u>	C249	=C246*C247	

<i>Probability of Infection and Illness per CFU ingested</i>	Cell	Model Input	Notes / References
<u>Pinf(1)</u>	C258	=@RiskBeta(0.21,59.95)	(Black, Levine et al. 1988)
<u>Pinf(D)</u>	C259	=1-(1-C258)^C235	(Black, Levine et al. 1988)
<u>Pinf (from calculated Pcons)</u>	C260	=C231*C259	
<u>Pinf (from observed Pcons)</u>	C261	=C232*C259	
<u>Pill inf</u>	C262	=@RiskBeta(30,61)	(FAO-WHO 2009)
<u>Pill (from calculated Pcons)</u>	C263	=C260*C262	
<u>Pill (from observed)</u>	C264	=C261*C262	

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Appendix B1 - Model Inputs: Transport and Processing:

Default Prevalence	Contaminated
Reciprocal	0.76
Number Sampled	0.24
Simulation Table	1225
	10

Major sections
Carcass or filet Sampling(s)
Constants
Summary/outputs
Subsections
Uncertainties
Simulation Tables

TRANSPORT & PROCESSING

Contamination of carcasses	1	2	3	4	5	6	7	1225
Prevalence/Contaminated (Exterior/Skin) 0 = No, 1 = Yes	1	0	0	1	1	1	1	0
C(ext), Carcass (process) log CFU/carcass	3.31	0.00	0.00	4.43	4.07	4.79	4.30	0.00
N(ext), Carcass (process) CFU/carcass, (log 0/carcass not detectible)	2,056	0	0	26,907	11,827	62,370	20,029	0

Prevalence - Exterior	0.77
Average C(ext) log CFU/bird	4.62
Average N CFU/bird - External	41,690

Transport	1	2	3	4	5	6	7	1225
Crates washed and sanitized prior to loading? 0 = No, 1 = Yes	0	1	1	1	1	0	1	0
Loading time (h)	0.78	0.91	0.71	0.88	0.82	0.89	0.82	0.71
Transit time (h)	3.35	1.20	2.07	3.55	3.47	1.19	3.65	0.99
Holding time (h)	3.89	4.49	3.31	3.24	4.88	3.39	4.47	5.48
Time in Cage (hours)	8.02	6.60	6.09	7.66	9.17	5.46	8.94	7.19
Occurrence of Cages Contaminated <i>Campylobacter</i>	0	0	0	0	0	0	0	0
Increase (External) - if previously negative	NA	0.00	0.00	NA	NA	NA	NA	0.00
Change due to transfer of <i>Campylobacter</i> on previously positive birds	0	NA	NA	1	1	1	1	NA
Change due to transfer of <i>Campylobacter</i> on previously positive birds	(0.33)	NA	NA	2.60	1.99	2.99	1.91	NA
C(ext), Carcass (transport) log CFU/carcass	2.99	0.00	0.00	7.03	6.06	7.79	6.21	0.00
N(ext), Carcass (transport) CFU/carcass (log 0/carcass not detectible)	970	0	0	10,670,022	1,145,778	61,643,241	1,609,718	0

Crates Washed and Sanitized prior to Loading	0.278
Loading time (h)	0.820
Transit time (h)	2.532
Holding time (h)	4.667
Average Total Time in Cage (Hours)	8.019
Occurrence of Cages Contaminated <i>Campylobacter</i>	24.08%
Samples of Previously (-) Birds	282
Occurrence of Cross-contamination in Transport (prev (-) bird)	0.21
Average C(transport,external) log CFU/bird Increase for Previously (-) Birds	0.59
Average N(transport,external) CFU/bird Increase for Prev (-) Birds	4
Samples w. Increase for Previously (+) Birds	771
Average C(transport,external) log CFU/bird increase for prev (+) birds	2.15
Samples w. Decrease (or No Change) for Previously (+) Birds	172
Average Change C(transport,external) log CFU/bird (or no change) for Previously (+) Birds	(0.30)
(+) Birds after Transport Stage, External	0.82
Average C(post transport,external) log CFU/bird after Transport Stage	7.28
Average N(post transport,external) CFU/bird after Transport Stage	18,904,447

Compliance	0.284
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Scalding	1	2	3	4	5	6	7	1225
Reduction in contamination, log CFU/carass	(4.08)	(2.95)	(2.64)	(2.96)	(3.94)	(3.61)	(4.16)	(3.84)
C(scald), log CFU/carass	0.00	0.00	0.00	4.07	2.12	4.18	2.05	0.00
N(scald), N CFU/carass	0	0	0	11,663	131	15,140	112	0

(+) Carcasses after Scalding Stage	0.76
Change in contamination due to Defeathering (log CFU/carass)	(3.13)
Average C(scald) log CFU/carass	4.46
Average N(scald) CFU/carass	28,934

Defeathering	1	2	3	4	5	6	7	1225
<i>Increase or Decrease by Effects of Defeathering (0 = Increase, 1 = Reduction)</i>	1	0	1	0	1	0	0	0
Change in contamination, log CFU/carass	(0.64)	(1.06)	(0.71)	2.01	(0.91)	2.71	2.06	0.81
Contaminated after Defeathering Stage? 0 = No, 1 = Yes	0	0	0	1	1	1	1	1
Contaminated after Defeathering Stage?	No	No	No	Yes	Yes	Yes	Yes	Yes
C(defeather), log CFU/carass	0.00	0.00	0.00	6.07	1.21	6.89	4.11	0.81
N(defeather) N CFU/carass	0	0	0	1,187,312	16	7,710,804	13,004	6

(+) Carcasses after Defeathering Stage	0.89
Change in contamination due to Defeathering (log CFU/carass)	0.76
Samples w. Increase in Contamination for Defeathering	778
Average Increase (log CFU/carass)	1.76
Samples w. Decrease (or no change) in Contamination for Defeathering	447
Average Change (log CFU/carass)	(0.98)
Average C(defeath) log CFU/carass	6.82
Average N(defeath) CFU/carass	6,568,384

Evisceration	1	2	3	4	5	6	7	1225
Change in contamination, log CFU/carass	0.00	0.00	0.00	(0.01)	0.50	(0.01)	0.48	0.50
Cross contaminated? 0 = No, 1 = Yes	1	1	0	1	1	1	1	1
C(evisc), log CFU/carass	0.50	0.50	0.00	6.07	1.71	6.88	4.59	1.31
N(evisc) CFU/carass	3	3	0	1,166,244	51	7,545,809	39,137	20

(+) Carcasses after Evisceration Stage	0.95
Change in contamination due to Evisceration	0.34
Samples w. Increase in Contamination for Evisceration	862
Average Increase (log CFU/carass)	0.49
Samples w. Decrease (or no change) in Contamination for Evisceration	363
Average Change (log CFU/carass)	(0.01)
Average C(evisc) log CFU/carass	6.80
Average N(evisc) CFU/carass	6,382,110

Washing & Chilling	1	2	3	4	5	6	7	1225
Type of On-line reprocessing chemical used	20	10	60	50	70	70	40	50
Type of On-line reprocessing chemical used	PA	HAS	CD	TSP	None	None	FGA	TSP
Reduction in contamination due to washing (log) - chemicals & water chilling	2.00	1.48	2.65	2.95	1.84	1.86	1.42	2.93
Use of Chlorinated water in chilling 0 = No, 1 = Yes	1	1	1	0	1	1	0	1
Use of Chlorinated water in chilling	Yes	Yes	Yes	No	Yes	Yes	No	Yes
Cross contamination due to water chilling 0 = No, 1 = Yes	0	0	1	0	0	0	0	0
Cross contamination due to water chilling	No	No	Yes	No	No	No	No	No
Increase due to cross-contamination, log CFU/carcass	0.00	0.00	0.41	0.00	0.00	0.00	0.00	0.00
C(wash), log CFU/carcass	0.00	0.00	0.00	3.12	0.00	5.02	3.17	0.00
N(wash) CFU/carcass	0	0	0	1,316	0	104,840	1,482	0

(+) Carcasses after Washing & Chilling Stage	0.72
<i>Average C(wash) log CFU/carcass</i>	5.57
<i>Average N(wash) CFU / carcass</i>	371,888
<i>Occurrence of cross-contamination in chilling</i>	0.16
<i>Increase due to cross-contamination (log CFU/carcass)</i>	0.44
Percentage of chlorinated water usage	78.29%
Percentage of Hypochlorous acid (HAS)	4.49%
<i>Average Reduction using HAS + water chilling</i>	(1.48)
Percentage of Peroxyacetic acid (PA)	12.82%
<i>Average Reduction using PA + water chilling</i>	(1.78)
Percentage of Acidified sodium chlorite (ASC)	18.29%
<i>Average Reduction using ASC + water chilling</i>	(2.33)
Percentage of Trisodium phosphate (TSP)	30.69%
<i>Average Reduction using TSP + water chilling</i>	(2.97)
Percentage of no on-line reprocessing chemical	8.24%
<i>Average Reduction using no on-line reprocessing chemical + water chilling</i>	(1.75)
Percentage of Blend of food-grade acids (FGA)	16.33%
<i>Average Reduction using FGA + water chilling</i>	(1.32)
Percentage of Chlorine dioxide (CD)	9.14%
<i>Average Reduction using CD + water chilling</i>	(2.43)

Chemical Used	Proportion
HAS	0.050
PA	0.125
ASC	0.175
FGA	0.150
TSP	0.313
CD	0.088
None	0.100

Portioning to Breast Filets	1	2	3	4	5	6	7	1225
Carcass Weight (g)	1,312.83	1,184.26	1,615.55	1,643.35	1,189.69	1,220.68	1,214.71	1,271.63
Filet size (g)	98.59	81.98	117.15	129.92	88.03	79.77	90.71	79.26
Redistribution of Campylobacter N(split) CFU/filet	0	0	0	104	0	6,851	111	0
C(filet) log CFU/filet (detection limit of 1 CFU/filet)	0.00	0.00	0.00	2.02	0.00	3.84	2.05	0.00
Occurrence of Contamination 0 = No, 1 = Yes	0	0	0	1	0	1	1	0
Reduction of Campylobacter due to removal of skin (log)	0.00	0.00	0.00	(0.63)	0.00	(1.04)	(0.74)	0.00
Cross contamination due to environment? 0 = No, 1 = Yes	1	1	1	1	0	1	1	1
Increase in Campylobacter due to cross-contamination from env (log)	0.31	0.27	0.28	0.31	0.00	0.24	0.25	0.28
Adjusted Campylobacter due to removal and cross-contamination log CFU/filet	0.31	0.27	0.28	1.70	0.00	3.04	1.55	0.28
Adjusted Campylobacter due to removal and cross-contamination N CFU/filet	2	2	2	51	0	1,092	36	2
Contaminated?	Yes	Yes	Yes	Yes	No	Yes	Yes	Yes

Average filet size (g)	98.54
Prevalence After Splitting from Carcass	0.48
Reduction of Campylobacter Due to Removal of Skin (log CFU/filet)	(0.52)
Frequency of Cross-contamination Due to Environment	0.77
Avg. Increase in Campylobacter Due to Cross-contamination from Environment (log CFU/filet)	0.27
(+) Filet after Splitting Stage	0.85
Average C(split) log CFU/filet	2.97
Average N(split) CFU/filet	936

In-Plant Storage	1	2	3	4	5	6	7	1225
Refrigerated/Chilled? Frozen? 0 = Chilled, 1 = Frozen	0	0	0	0	0	0	0	1
Refrigerated/Chilled? Frozen?	Chilled	Chilled	Chilled	Chilled	Chilled	Chilled	Chilled	Frozen
Number of Days	0.77	0.89	0.83	1.02	1.22	1.27	0.58	13.94
Reduction (log)	(0.17)	(0.19)	(0.18)	(0.22)	(0.25)	(0.26)	(0.13)	(1.65)
C log CFU / filet (storage)	0.14	0.08	0.10	1.49	0.00	2.78	1.42	0.00
N CFU / filet (storage)	1	1	1	31	0	602	26	0

(+) Filets during In-plant Storage stage	0.63
Average C(storage) log CFU/filet (Total)	2.67
Average N(storage) CFU/filet (Total)	471
Chilled samples	1,055
Reduction from Chilling Process	(0.19)
Average C(chill) log CFU/filet	2.73
Average N(chill) CFU/filet	543
Frozen samples	170
Reduction from Freezing Process	(1.69)
Average C(freeze) log CFU/filet	1.35
Average N(freeze) CFU/filet	22

	Probability
Frozen	0.14
Chilled	0.86

Appendix B2 - Model Inputs: Storage, Handling, Cooking and Consumption:

Number Sampled	1225
Simtable - Retail/Consumer	25

RETAIL AND HANDLING

Transport to Retail & Retail Storage	1	2	3	4	5	6	7	8	1225
Delivered from Plant Frozen or Chilled	Chilled	Chilled	Chilled	Chilled	Chilled	Chilled	Chilled	Chilled	Frozen
Number of days in retail storage	2.36	6.88	1.15	3.03	7.51	7.28	3.38	7.84	28.06
Aggregate number of days in storage (in-plant, transport, retail)	3.13	7.78	1.98	4.05	8.73	8.55	3.96	8.59	28.06
Reduction from finished product in-plant (Retail storage - reduction)	(0.48)	(0.60)	(0.36)	(0.53)	(0.63)	(0.62)	(0.53)	(0.62)	(2.00)
Total reduction due to plant and retail storage (log CFU/filet)	(0.65)	(0.80)	(0.54)	(0.75)	(0.88)	(0.88)	(0.66)	(0.79)	(2.00)
C(s,retail) log CFU/filet (detection limit of 1 CFU/filet)	0.00	0.00	0.00	0.96	0.00	2.16	0.89	1.77	0.00
N(s,retail) CFU/filet	0	0	0	9	0	144	8	59	0
Contaminated? 0 = No, 1 = Yes	0	0	0	1	0	1	1	1	0
Contaminated?	No	No	No	Yes	No	Yes	Yes	Yes	No

(+) Filets during retail storage	0.33
Average C(s,retail) log CFU/filet	2.10
Average N(s,retail) CFU/filet	127
Number sampled (Chilled)	1,054
Reduction from chilling process	(0.53)
<i>Occurrence of contaminated filets, (Chilled)</i>	0.37
Average C(s,home) log CFU/filet, (Chilled)	2.17
Average N(s,home) CFU/filet, (Chilled)	148
Number sampled (Frozen)	171
Reduction from freezing process	(2.12)
<i>Occurrence of contaminated filets, (Frozen)</i>	0.02
Average C(s,home) log CFU/filet, (Frozen)	(0.82)
Average N(s,home) CFU/filet, (Frozen)	0.15

Home Storage	1	2	3	4	5	6	7	8	1225
Consumed at Day 0? Frozen? Chilled? (0 = day of, 10 = Frozen, 20 = Chilled)	0	20	10	0	0	10	0	0	0
Consumed at Day 0? Frozen? Chilled?	Day 0	Chilled	Frozen	Day 0	Day 0	Frozen	Day 0	Day 0	Day 0
If not consumed on purchase date, duration of storage at home (Days)	0.00	1.74	12.74	0.00	0.00	27.71	0.00	0.00	0.00
Aggregate number of days in storage (Until preparation and consumption)	0.00	9.51	NA	0.00	0.00	NA	0.00	0.00	0.00
Reduction from finished product in-plant	0.00	(0.66)	(1.61)	0.00	0.00	(1.99)	0.00	0.00	0.00
Total Reduction from "Storage (Plant, Retail, Home)" module (log CFU/filet)	0.00	(1.46)	(2.15)	0.00	0.00	(2.87)	0.00	0.00	0.00
C(s,home) log CFU/filet (detection limit of 1 CFU/filet)	0.00	0.00	0.00	0.96	0.00	0.17	0.89	1.77	0.00
N(s,home) CFU/filet	0	0	0	9	0	1	8	59	0
Contaminated? 0 = No, 1 = Yes	0	0	0	1	0	1	1	1	0
Contaminated?	No	No	No	Yes	No	Yes	Yes	Yes	No

Occurrence of contaminated filets at home, Total	0.27
Average C(s,home) log CFU/filet, Total	1.82
Average N(s,home) CFU/filet, Total	66
Number sampled (Bought Chilled, consumed day of)	597
Occurrence of contaminated filets, (Bought Chilled, consumed day of)	0.41
Average C(s,home) log CFU/filet, (Bought Chilled, consumed day of)	2.02
Average N(s,home) CFU/filet, (Bought Chilled, consumed day of)	105
Number sampled (Bought Chilled, chilled)	291
Occurrence of contaminated filets, (Bought Chilled, chilled)	0.22
Average C(s,home) log CFU/filet, (Bought Chilled, chilled)	1.80
Average N(s,home) CFU/filet, (Bought Chilled, chilled)	63
Number sampled (Bought Chilled, frozen)	166
Occurrence of contaminated filets, (Bought Chilled, frozen)	0.11
Average C(s,home) log CFU/filet, (Bought Chilled, frozen)	0.20
Average N(s,home) CFU/filet, (Bought Chilled, frozen)	1.58
Number sampled (Bought Frozen, consumed day of)	40
Occurrence of contaminated filets, (Bought Frozen, consumed day of)	0.05
Average C(s,home) log CFU/filet, (Bought Frozen, consumed day of)	(0.49)
Average N(s,home) CFU/filet, (Bought Frozen, consumed day of)	0
Number sampled (Bought Frozen, chilled)	61
Occurrence of contaminated filets, (Bought Frozen, chilled)	0.00
Average C(s,home) log CFU/filet, (Bought Frozen, chilled)	0.00
Average N(s,home) CFU/filet, (Bought Frozen, chilled)	0
Number sampled (Bought Frozen, frozen)	70
Occurrence of contaminated filets, (Bought Frozen, frozen)	0.00
Average C(s,home) log CFU/filet, (Bought Frozen, frozen)	0.00
Average N(s,home) CFU/filet, (Bought Frozen, frozen)	0

If Bought Frozen (from Retail)	
	Probability
Day 0	0.250
Frozen	0.450
Chilled	0.300

If Bought Chilled (from Retail)	
	Probability
Day 0	0.55
Frozen	0.15
Chilled	0.30

Home Storage	1	2	3	4	5	6	7	8	1225
Consumed at Day 0? Frozen? Chilled? (0 = day of, 10 = Frozen, 20 = Chilled)	0	20	10	0	0	10	0	0	0
Consumed at Day 0? Frozen? Chilled?	Day 0	Chilled	Frozen	Day 0	Day 0	Frozen	Day 0	Day 0	Day 0
If not consumed on purchase date, duration of storage at home (Days)	0.00	1.74	12.74	0.00	0.00	27.71	0.00	0.00	0.00
Aggregate number of days in storage (Until preparation and consumption)	0.00	9.51	NA	0.00	0.00	NA	0.00	0.00	0.00
Reduction from finished product in-plant	0.00	(0.66)	(1.61)	0.00	0.00	(1.99)	0.00	0.00	0.00
Total Reduction from "Storage (Plant, Retail, Home)" module (log CFU/filet)	0.00	(1.46)	(2.15)	0.00	0.00	(2.87)	0.00	0.00	0.00
C(s,home) log CFU/filet (detection limit of 1 CFU/filet)	0.00	0.00	0.00	0.96	0.00	0.17	0.89	1.77	0.00
N(s,home) CFU/filet	0	0	0	9	0	1	8	59	0
Contaminated? 0 = No, 1 = Yes	0	0	0	1	0	1	1	1	0
Contaminated?	No	No	No	Yes	No	Yes	Yes	Yes	No

Occurrence of contaminated filets at home, Total	0.27
Average C(s,home) log CFU/filet, Total	1.82
Average N(s,home) CFU/filet, Total	66
Number sampled (Bought Chilled, consumed day of)	597
Occurrence of contaminated filets, (Bought Chilled, consumed day of)	0.41
Average C(s,home) log CFU/filet, (Bought Chilled, consumed day of)	2.02
Average N(s,home) CFU/filet, (Bought Chilled, consumed day of)	105
Number sampled (Bought Chilled, chilled)	291
Occurrence of contaminated filets, (Bought Chilled, chilled)	0.22
Average C(s,home) log CFU/filet, (Bought Chilled, chilled)	1.80
Average N(s,home) CFU/filet, (Bought Chilled, chilled)	63
Number sampled (Bought Chilled, frozen)	166
Occurrence of contaminated filets, (Bought Chilled, frozen)	0.11
Average C(s,home) log CFU/filet, (Bought Chilled, frozen)	0.20
Average N(s,home) CFU/filet, (Bought Chilled, frozen)	1.58
Number sampled (Bought Frozen, consumed day of)	40
Occurrence of contaminated filets, (Bought Frozen, consumed day of)	0.05
Average C(s,home) log CFU/filet, (Bought Frozen, consumed day of)	(0.49)
Average N(s,home) CFU/filet, (Bought Frozen, consumed day of)	0
Number sampled (Bought Frozen, chilled)	61
Occurrence of contaminated filets, (Bought Frozen, chilled)	0.00
Average C(s,home) log CFU/filet, (Bought Frozen, chilled)	0.00
Average N(s,home) CFU/filet, (Bought Frozen, chilled)	0
Number sampled (Bought Frozen, frozen)	70
Occurrence of contaminated filets, (Bought Frozen, frozen)	0.00
Average C(s,home) log CFU/filet, (Bought Frozen, frozen)	0.00
Average N(s,home) CFU/filet, (Bought Frozen, frozen)	0

If Bought Frozen (from Retail)	
	Probability
Day 0	0.250
Frozen	0.450
Chilled	0.300

If Bought Chilled (from Retail)	
	Probability
Day 0	0.55
Frozen	0.15
Chilled	0.30

Cross-Contamination from Package (Outside pack--->Hand)	1	2	3	4	5	6	7	8	1225
Occurrences of contamination 0 = No, 1 = Yes	0	0	0	0	0	0	0	0	0
Contaminated?	No	No	No	No	No	No	No	No	No
Tier of Contamination	1	1	2	1	2	2	1	1	1
Contamination on Outside of Package (Log CFU/package) - initial	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Reduction due to Storage (log CFU/package) - Home	0.00	(0.33)	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Contamination on Outside of Package (log CFU/package) - Final	0	0	0	0	0	0	0	0	0
Contamination on Outside of Package (N CFU/package) - Final	0	0	0	0	0	0	0	0	0
Transfer N(hands)	0	0	0	0	0	0	0	0	0
Hands(package)--->Carcass	0	0	0	0	0	0	0	0	0

Occurrence of Package Contamination - At Time of Purchase	0.06
Avg. Contamination on Outside of Packge (log CFU/package), Total - Initial (Retail)	0.12
Avg. Contamination on Outside of Packge (log CFU/package), If Contaminated - Initial (Retail)	1.91
Occurrence of Package Contamination - At Time of Meal Preparation	0.06
Avg. Contamination on Outside of Packge (log CFU/package), Total - Final (Retail)	0.11
Avg. Contamination on Outside of Packge (log CFU/package), If Contaminated - Final (Retail)	1.84
Avg. Transfer N(hands) - Total	0.07
Avg. Transfer N(hands) - If Contaminated	1.11
Avg. Hands(package)--->Carcass - If Contaminated	0.00

Level	Probability	
10 - 99	1	0.768
100 - 1,000	2	0.213
1,000-100,000	3	0.018

Campylobacter on Product before Food Preparation	1	2	3	4	5	6	7	8	1225
CFU Campylobacter remaining on hands	0	0	0	0	0	0	0	0	0
Final C(home), log CFU / filet	0.00	0.00	0.00	0.95	0.00	0.00	0.90	1.77	0.00
Final N(home), CFU / filet	0	0	0	9	0	1	8	59	0

Occurrence of Contamination	0.27
Average log CFU/filet (home)	1.82
Average N CFU/filet (home)	66

CROSS-CONTAMINATION (HANDLING & COOKING)

Cross Contamination from Handling of Filets	1	2	3	4	5	6	7	8	1225
Handling path, Meat-->Hands	4	3	2	2	2	3	2	2	3
Handling path, Meat-->Hands	NC	Wash	Rinse	Rinse	Rinse	Wash	Rinse	Rinse	Wash
Transfer Rate	0.00E+00	2.04E-04	7.99E-02	4.67E-02	1.68E-03	1.64E-03	7.50E-03	1.96E-05	5.25E-04
Transfer N(hands)	0.00E+00	0	0	0	0	0	0	0	0

Handling		Probability
No Wash	1	0.09
Rinse	2	0.52
Wash w. soap	3	0.22
No contact	4	0.07

Handling path, Meat-->Board	4	4	4	4	2	4	1	1	4
Handling path, Meat-->Board	NC	NC	NC	NC	Rinse	NC	No Wash	No Wash	NC
If Rinsed (2), 10=Cold, 20=Hot, with 0.5 p for each; If not rinsed 0 = "Other"	Other	Other	Other	Other	10	Other	Other	Other	Other
Transfer Rate: No Wash; Rinsing (cold),(hot); Washing	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.21E-01	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Truncation, if necessary (Limit = transfer rate of 0.7)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.21E-01	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Transfer N(board)	0	0	0	0	0	0	0	7	0

Handling		Probability
No Wash	1	0.127
Rinse	2	0.152
Wash w. soap	3	0.038
No contact	4	0.654

If Rinse, usage of:	
Cold water	0.5
Hot water	0.5

Handling path, Meat-->Utensil	1	4	2	4	1	4	1	1	1
Handling path, Meat-->Utensil	No Wash	NC	Rinse	NC	No Wash	NC	No Wash	No Wash	No Wash
If Rinsed (2), 10=Cold, 20=Hot, with 0.5 p for each; If not rinsed 0 = "Other"	Other	Other	10	Other	Other	Other	Other	Other	Other
Transfer Rate: No Wash; Rinsing (cold),(hot); Washing, No Contact (NC)	No Wash	NC	5.56E-06	NC	No Wash	NC	No Wash	No Wash	No Wash
Transfer N(Utensil)	0	0	0	0	0	0	0	1	0

Handling		Probability
No Wash	1	0.207
Rinse	2	0.125
Wash w. soap	3	0.002
No contact	4	0.666

If Rinse, usage of:	
Cold water	0.5
Hot water	0.5

Total Transfer from Cross-Contamination, Handling									
Total Transfer N(Hands,board,utensil)	0	0	0	0	0	0	0	8	0
C(posthandling) log CFU/Fillet	0.00	0.00	0.00	0.95	0.00	0.00	0.90	1.71	0.00
Hands-->Food	0	0	0	0	0	0	0	0	0
Board-->Food	0	0	0	0	0	0	0	0	0
Utensil-->Food	0	0	0	0	0	0	0	0	0
Contaminated?	No	No	No	No	No	No	No	No	No
N CFU/filet Ingested (Nch)	0	0	0	0	0	0	0	0	0

Pcons (handling)	2.78E-02
Meat--->Hands (No Wash) Sampled	119
Meat--->Hands (Rinse) Sampled	722
Meat--->Hands (Wash) Sampled	296
Meat--->Hands (No Contact) Sampled	88
Avg N CFU from all Meat--->Hands sampled	0
Avg N CFU from Meat--->hands sampled if contaminated (No Wash)	1
Avg N CFU from Meat--->hands sampled if contaminated (Rinse)	0
Avg N CFU from Meat--->hands sampled if contaminated (Wash, soap)	0
Avg N CFU from Meat--->hands sampled if contaminated (No Contact)	0
Meat--->Board (No Wash) Sampled	155
Meat--->Board (Rinse, cold) Sampled	90
Meat--->Board (Rinse, hot) Sampled	97
Meat--->Board (Wash, soap) Sampled	45
Meat--->Board (No Contact) Sampled	838
Avg N CFU from all Meat--->Boards sampled	2
Avg N CFU from Meat--->Board sampled if contaminated (No Wash)	3
Avg N CFU from Meat--->Board sampled if contaminated (Rinse, cold)	17
Avg N CFU from Meat--->Board sampled if contaminated (Rinse, hot)	0
Avg N CFU from Meat--->Board sampled is contaminated (Wash, soap)	0
Avg N CFU from Meat--->Board sampled if contaminated (No contact)	0
Meat--->Utensil (No Wash) Sampled	263
Meat--->Utensil (Rinse, cold) Sampled	87
Meat--->Utensil (Rinse, hot) Sampled	57
Meat--->Utensil (Wash, soap) Sampled	2
Meat--->Utensil (No Contact) Sampled	816
Avg N CFU from all Meat--->Utensil sampled	0
Avg N CFU from Meat--->Utensil sampled if contaminated (No Wash)	1
Avg N CFU from Meat--->Utensil sampled if contaminated (Rinse, cold)	0
Avg N CFU from Meat--->Utensil sampled if contaminated (Rinse, hot)	0
Avg N CFU from Meat--->Utensil sampled is contaminated (Wash, soap)	0
Avg N CFU from Meat--->Utensil sampled if contaminated (No contact)	0
Average CFU Transferred off filet from all handlings	2
Average CFU Transferred to meal if contaminated via handling	29
Average CFU Ingested in meal from all handlings	0
Average CFU Ingested in meal - from contaminated meals	5
Percentage of Samples having <i>Campylobacter</i>- Contaminated filets from Handling	2.78%

D-Value	1.97									
Distribution Chooser - Cooking	35	22								
COOKING	1	2	3	4	5	6	7	8	1225	
C(posthandling) log CFU/Fillet	0.00	0.00	0.00	0.95	0.00	0.00	0.90	1.71	0.00	0.00
Internal Contamination? 0=No, 1=Yes	0	0	0	0	0	0	0	0	0	0
C(Fillet, internal)	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Cooking under 8 minutes	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Cooking Time (Minutes)	9.71	3.30	8.08	10.14	3.50	10.51	6.88	4.40	17.99	
Log CFU/filet Reduction (time/D-value)	(4.94)	(1.68)	(4.11)	(5.15)	(1.78)	(5.34)	(3.50)	(2.24)	(9.14)	
Contamination Log CFU/filet (not detectible at <=log 0)	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	
N CFU/filet Ingested (Ncu)	0	0	0	0	0	0	0	0	0	
Contaminated?	No	No	No	No	No	No	No	No	No	No

Pcons (cook)	9.80E-03
% of Internal Contamination	6.204%
Average log reduction/filet	6.63
Average log CFU/filet (Internal, if contaminated)	0.65
Average CFU ingested for all meals	0.00E+00
Average CFU ingested if meal is contaminated	2.000
Percentage of contaminated cooked filets	0.98%
Occurrence of Cooking under 8 minutes	32.2%

DOSE-RESPONSE

Pcons (calc)	3.73E-02								
Pcons (obs)	3.51E-02								
Ingestion of Campylobacter	1	2	3	4	5	6	7	8	1225
Campy CFU ingested from filet?	No	No	No	No	No	No	No	No	No
Total Ingested N(cons)	0	0	0	0	0	0	0	0	0

Average Ingested from all meals	0.00
Average CFU ingested if from contaminated meals	8
Pcons (calc)	3.73E-02
Pcons (obs)	3.51E-02

Probability of Infection and Illness per dosage ingested	1	2	3	4	5	6	7	8	1225
Pinf(D)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Pinf (from calculated Pcons)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Pinf (from observed Pcons)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Pill inf	3.10E-01	2.95E-01	2.74E-01	3.14E-01	2.81E-01	3.23E-01	4.72E-01	4.22E-01	2.75E-01
Pill (from calculated Pcons)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Pill (from observed)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00

Pinf (from calculated Pcons)	2.81E-05
Pinf (from observed Pcons)	2.65E-05
Pill (calc)	9.15E-06
Pill (obs)	8.61E-06

Probability of Infection and Illness per CFU ingested	1	2	3	4	5	6	7	8	1225
Pinf(1)	2.27E-03	1.39E-02	3.63E-04	1.09E-02	6.48E-04	4.57E-03	1.32E-04	2.98E-05	4.31E-05
Pinf(D)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Pinf (from calculated Pcons)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Pinf (from observed Pcons)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Pill inf	2.79E-01	3.54E-01	4.07E-01	4.03E-01	2.99E-01	3.37E-01	3.49E-01	3.39E-01	3.35E-01
Pill (from calculated Pcons)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Pill (from observed)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00

Pinf (from calculated Pcons)	1.96E-05
Pinf (from observed Pcons)	1.85E-05
Pill(calc)	7.03E-06
Pill(obs)	6.62E-06

Appendix B3 - Model Outputs: Pre-Transport, Processing and Preparation:

Farm (Pre-transport, Post grow-out)	
<i>Prevalence - Exterior</i>	0.73
Average C(ext) log CFU/bird	4.60
Transport	
Loading time (h)	0.82
Transit time (h)	2.51
Holding time (h)	4.64
Average Total Time in Cage (Hours)	7.97
Occurrence of Cages Contaminated w. Campylobacter	0.23
Samples of Previously (-) Birds	337
Occurrence of Cross-contamination in Transport (prev (-) bird)	0.23
Average C(transport,external) log CFU/bird Increase for Previously (-) Birds	0.82
Samples w. Increase for Previously (+) Birds	723
Average C(transport,external) log CFU/bird increase for prev (+) birds	2.12
Samples w. Decrease (or No Change) for Previously (+) Birds	165
Average Change (C(transport,external) log CFU/bird) (or no change) for Previously (+) Birds	(0.32)
(+) Birds after Transport Stage, External	0.79
Average C(post transport,external) log CFU/bird after Transport Stage	7.23
Scalding	
(+) Carcasses after Scalding Stage	0.73
Change in contamination due to Scalding (log CFU/carcass)	(3.10)
Average C(scald) log CFU/carcass	4.51

Defeathering	
(+) Carcasses after Defeathering Stage	0.87
Change in contamination due to Defeathering	0.80
Samples w. Increase in Contamination for Defeathering	792
Average Increase (log CFU/carcass)	1.78
Samples w. Decrease in Contamination for Defeathering	433
Average Decrease (log CFU/carcass)	(0.99)
Average C(defeath) log CFU/carcass	6.93

Evisceration	
(+) Carcasses after Evisceration Stage	0.94
Change in contamination due to Evisceration	0.34
Samples w. Increase in Contamination for Evisceration	862
Average Increase (log CFU/carcass)	0.49
Samples w. Decrease in Contamination for Evisceration	363
Average Decrease (log CFU/carcass)	(0.01)
Average C(evisc) log CFU/carcass	6.93

Washing and Chilling	
Proportion of HAS Usage	0.05
<i>Average Reduction (log CFU/carcass) using HAS + water chilling</i>	(1.47)
Proportion of PA Usage	0.13
<i>Average Reduction (log CFU/carcass) using PA + water chilling</i>	(1.79)
Proportion of ASC Usage	0.18
<i>Average Reduction (log CFU/carcass) using ASC + water chilling</i>	(2.33)
Proportion of TSP Usage	0.31
<i>Average Reduction (log CFU/carcass) using TSP + water chilling</i>	(2.97)
Proportion of No on-line Reprocessing Chemical Usage	0.10
<i>Average Reduction (log CFU/carcass) using no on-line reprocessing chemical + water chilling</i>	(1.78)
Proportion of FGA Usage	1.50
<i>Average Reduction (log CFU/carcass) using FGA + water chilling</i>	(1.32)
Proportion of CD Usage	0.09
<i>Average Reduction (log CFU/carcass) using CD + water chilling</i>	(2.41)
 (+) Carcasses after Washing & Chilling Stage	0.69
<i>Average C(wash) log CFU/carcass</i>	5.16
<i>Occurrence of cross-contamination in chilling</i>	0.15
<i>Increase due to cross-contamination (log CFU/carcass)</i>	0.45
Percentage of chlorinated water usage	77.24%

Portioning	
Average filet size (g)	98.56
Prevalence After Splitting from Carcass	0.49
Reduction of Campylobacter Due to Removal of Skin (log CFU/filet)	(0.54)
Frequency of Cross-contamination Due to Environment	0.75
Avg. Increase in Campylobacter Due to Cross-contamination from Environment (log CFU/filet)	0.27
(+) Filet after Splitting Stage	0.85
Average C(split) log CFU/filet	2.70

<i>In-Plant Storage</i>	
<i>(+) Filets during In-plant Storage Stage</i>	<i>0.64</i>
Average C(storage) log CFU/ilet (Total)	2.44
Chilled samples	1,056
Reduction (log CFU/ilet) from chilling process	(0.19)
Average C(chill) log CFU/ilet	2.50
Frozen samples	169
Reduction (log CFU/ilet) from freezing process	(1.68)
Average C(freeze) log CFU/ilet	0.90

<i>Retail</i>	
<i>Occurrence of contaminated filets during retail storage</i>	<i>0.32</i>
Average N(s,retail) CFU/ilet	96
Number sampled (Chilled)	1,054
Reduction (log CFU/ilet) from chilling process	(0.53)
<i>Occurrence of contaminated filets, (Chilled)</i>	<i>0.37</i>
Average N(s,home) CFU/ilet, (Chilled)	111.34
Number sampled (Frozen)	171
Reduction from freezing process	(2.12)
<i>Occurrence of contaminated filets, (Frozen)</i>	<i>0.014</i>
Average N(s,home) CFU/ilet, (Frozen)	0.12

Home Storage	
Occurrence of contaminated filets at home, Total	0.23
Average N(s,home) CFU/ilet, Total	37
Number sampled (Bought Chilled, consumed day of)	564
<i>Occurrence of contaminated filets, (Bought Chilled, consumed day of)</i>	<i>0.34</i>
<i>Average N(s,home) CFU/ilet, (Bought Chilled, consumed day of)</i>	<i>72</i>
Number sampled (Bought Chilled, chilled)	347
<i>Occurrence of contaminated filets, (Bought Chilled, chilled)</i>	<i>0.23</i>
<i>Average N(s,home) CFU/ilet, (Bought Chilled, chilled)</i>	<i>13</i>
Number sampled (Bought Chilled, frozen)	143
<i>Occurrence of contaminated filets, (Bought Chilled, frozen)</i>	<i>0.10</i>
<i>Average N(s,home) CFU/ilet, (Bought Chilled, frozen)</i>	<i>1</i>
Number sampled (Bought Frozen, consumed day of)	43
<i>Occurrence of contaminated filets, (Bought Frozen, consumed day of)</i>	<i>0.02</i>
<i>Average N(s,home) CFU/ilet, (Bought Frozen, consumed day of)</i>	<i>3.95E-01</i>
Number sampled (Bought Frozen, chilled)	46
<i>Occurrence of contaminated filets, (Bought Frozen, chilled)</i>	<i>0.00</i>
<i>Average N(s,home) CFU/ilet, (Bought Frozen, chilled)</i>	<i>0.00E+00</i>
Number sampled (Bought Frozen, frozen)	82
<i>Occurrence of contaminated filets, (Bought Frozen, frozen)</i>	<i>0.00</i>
<i>Average N(s,home) CFU/ilet, (Bought Frozen, frozen)</i>	<i>0.00E+00</i>

Packaging (Outside) Contamination	
Occurrence of Package Contamination - At Time of Purchase	0.06
<i>Avg. Contamination on Outside of Packge (log CFU/package), Total - Initial (Retail)</i>	<i>0.11</i>
<i>Avg. Contamination on Outside of Packge (log CFU/package), If Contaminated - Initial (Retail)</i>	<i>1.92</i>
Occurrence of Package Contamination - At Time of Meal Preparation	0.06
<i>Avg. Contamination on Outside of Packge (log CFU/package), Total - Final (Retail)</i>	<i>0.10</i>
<i>Avg. Contamination on Outside of Packge (log CFU/package), If Contaminated - Final (Retail)</i>	<i>1.81</i>
<i>Avg. Transfer N(hands) - Total</i>	<i>0.06</i>
<i>Avg. Transfer N(hands) - If Contaminated</i>	<i>1.12</i>
<i>Avg. Hands(package)--->Carcass - If Contaminated</i>	<i>0.00</i>

Cross-Contamination, Preparation and Handling	
Pcons (handling)	2.61E-02
Meat--->Hands (No Wash) Sampled	104
Meat--->Hands (Rinse) Sampled	770
Meat--->Hands (Wash) Sampled	278
Meat--->Hands (No Contact) Sampled	73
Avg N CFU from all Meat--->Hands sampled	1
Avg N CFU from Meat--->hands sampled if contaminated (No Wash)	1
Avg N CFU from Meat--->hands sampled if contaminated (Rinse)	1
Avg N CFU from Meat--->hands sampled if contaminated (Wash, soap)	0
Avg N CFU from Meat--->hands sampled if contaminated (No Contact)	0
Meat--->Board (No Wash) Sampled	168
Meat--->Board (Rinse, cold) Sampled	101
Meat--->Board (Rinse, hot) Sampled	87
Meat--->Board (Wash, soap) Sampled	30
Meat--->Board (No Contact) Sampled	839
Avg N CFU from all Meat--->Boards sampled	0
Avg N CFU from Meat--->Board sampled if contaminated (No Wash)	2
Avg N CFU from Meat--->Board sampled if contaminated (Rinse, cold)	2
Avg N CFU from Meat--->Board sampled if contaminated (Rinse, hot)	0
Avg N CFU from Meat--->Board sampled is contaminated (Wash, soap)	0
Avg N CFU from Meat--->Board sampled if contaminated (No contact)	0
Meat--->Utensil (No Wash) Sampled	245
Meat--->Utensil (Rinse, cold) Sampled	74
Meat--->Utensil (Rinse, hot) Sampled	82
Meat--->Utensil (Wash, soap) Sampled	0
Meat--->Utensil (No Contact) Sampled	824
Avg N CFU from all Meat--->Utensil sampled	0
Avg N CFU from Meat--->Utensil sampled if contaminated (No Wash)	1
Avg N CFU from Meat--->Utensil sampled if contaminated (Rinse, cold)	0
Avg N CFU from Meat--->Utensil sampled if contaminated (Rinse, hot)	0
Avg N CFU from Meat--->Utensil sampled is contaminated (Wash, soap)	0
Avg N CFU from Meat--->Utensil sampled if contaminated (No contact)	0
Average CFU Transferred to meal from all handlings	1
Average CFU Transferred to meal if contaminated via handling	23
Percentage of samples contaminated	2.61%

<i>Cross-Contamination, Cooking</i>	
Average log reduction	(6.26)
Average log CFU/filet (Internal, if contaminated)	0.77
Percentage of contaminated cooked filets	0.41%
Occurrence of Cooking under 8 minutes	0.33

Appendix B4 – Model Outputs: Ingestion, Dose response, Economic burden:

Ingestion of Campylobacter in a Meal	
Average CFU Ingested from all meals	1
Average CFU ingested if from contaminated meals	48
<i>Pcons (calc)</i>	3.24E-02
<i>Pcons (obs)</i>	3.02E-02
Probability of Infection and Illness per dosage ingested	
<i>Pinf (from calculated Pcons)</i>	2.882E-05
<i>Pinf (from observed Pcons)</i>	2.681E-05
<i>Pill (calc)</i>	9.496E-06
<i>Pill (obs)</i>	8.833E-06
Probability of Infection and Illness per "single hit" method	
<i>Pinf (from calculated Pcons)</i>	2.873E-05
<i>Pinf (from observed Pcons)</i>	2.673E-05
<i>Pill (calc)</i>	9.468E-06
<i>Pill (obs)</i>	8.808E-06
Exposure to Product	
Per capita consumption/yr, boneless retail weight (lbs) (2012 - 2015, yearly disappearance)	59.10
Fraction of home chicken consumption	40%
lbs consumed at home	23.64
grams consumed at home	10723.10
Fraction of breast consumption (home)	58%
lbs breast consumed at home	13.59
Per capita consumption (home)/yr, breast (g)	6165.34
"Average" serving size (g)	170.10
Servings of breast per capita/yr (home)	36.33
Current contiguous U.S. Population (2019)	326,092,106
Total Servings of breasts consumed/yr (home)	11,846,926,211

Simtable	3
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Domestically Acquired Foodborne Campylobacteriosis Estimates	3	Target N ill for chicken consumption/yr	For filet/yr (home consumption)
Estimated Illness (Mead, 1999) Campylobacteriosis	2,407,452	1,221,611	280,971
Estimated Illness, Collected State Rate, (2006-2008)	1,570,398	796,866	183,279
Estimated Illness, (Scallan, 2011), Campylobacteriosis	289,429	146,865	33,779

Attribution of Campylobacteriosis due to Chicken Consumption	50.74%
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"target" N ill for chicken consumption/yr - State and Scallan data	146,865
For filet/yr (home consumption)	33,779

Distribution Chooser - Model Output	1					
	N inf / yr	N ill / yr	N Hosp. / yr	N Deaths / yr	N Guillain-Barre / yr	N IBS / yr
Model output Pill(calc) - per dose ingested	341,428	112,498	1,123	45	120	9,953
Model output Pill(obs) - per dose ingested	317,616	104,644	1,047	35	114	9,291
Model output Pill (calc) - per CFU ingested	340,362	112,167	1,011	41	118	9,890
Model output Pill (obs) - per CFU ingested	316,668	104,348	900	48	97	9,134

Distribution Chooser - Model Output	35	Model Output Distribution	340,362	112,167	1,011	41	118	9,890
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Economic Burden

Distribution Chooser - Economic Burden					
	Didn't Visit Physician; Survived	Visited Physician; Survived	Hospitalized, non-GBS; Survived	Premature Deaths	Total
Model Output Distribution - ERS Calculator	\$ 7,021,505	\$ 20,199,603	\$ 9,602,492	\$ 154,599,735	\$ 231,108,389
Model Output Distribution - FDA Disutility	\$ 79,706,444	\$ 81,415,710	\$ 16,958,140	\$ 183,817,824	\$ 231,108,389
Model Output Distribution - ERS and FDA Disutility Combined	\$ 79,706,444	\$ 81,415,710	\$ 16,958,140	\$ 183,817,824	\$ 231,108,389
Model Output Distribution - Roberts et al.	\$ 34,546,589	\$ 37,181,774	\$ 7,041,780	\$ 59,273,836	\$ 138,043,979
Model Output Distribution - Buzby et al.	\$ 46,812,726	\$ 48,365,085	\$ 15,522,111	\$ 46,194,363	\$ 156,894,285
Model Output Distribution - Roberts et al. & Buzby et al. Combined	\$ 46,812,726	\$ 48,365,085	\$ 15,522,111	\$ 46,194,363	\$ 156,894,285
Number of Cases	82,273	28,778			

Combined - All	\$ 46,812,726	\$ 48,365,085	\$ 15,522,111	\$ 46,194,363	\$ 156,894,285
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Distribution Chooser					
	Didn't Visit Physician; Survived	Visited Physician; Survived	Hospitalized, non-sequelae; Survived	Premature Deaths	Total
Gender					
Men	\$ 23,464,967	\$ 24,243,089	\$ 7,780,487	\$ 23,155,011	\$ 78,643,554
Women	\$ 23,347,760	\$ 24,121,995	\$ 7,741,624	\$ 23,039,352	\$ 78,250,731

Total, All Cases (Annual)	\$ 231,108,389
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Distribution Chooser		15			
Age Distribution	Didn't Visit Physician; Survived	Visited Physician; Survived	Premature Deaths	Total	
Less than 5 years	\$ 7,964,658	\$ 8,228,774	\$ 7,886,843	\$ 24,080,274	
5 - 9 years	\$ 2,974,983	\$ 3,073,636	\$ 3,380,075	\$ 9,428,695	
5 - 14 years	\$ 5,126,923	\$ 5,296,937	\$ 4,506,767	\$ 14,930,628	
10 - 14 years	\$ 2,176,698	\$ 2,248,879	\$ 2,253,384	\$ 6,678,960	
15 - 19 years	\$ 2,700,343	\$ 2,789,889	\$ 2,253,384	\$ 7,743,616	
15 - 24 years	\$ 5,490,985	\$ 5,673,072	\$ 5,633,459	\$ 16,797,516	
20 - 29 years	\$ 5,617,750	\$ 5,804,040	\$ 5,633,459	\$ 17,055,249	
30 - 39 years	\$ 5,357,763	\$ 5,535,432	\$ 5,633,459	\$ 16,526,653	
40 - 49 years	\$ 6,290,245	\$ 6,498,836	\$ 6,760,151	\$ 19,549,231	
50 - 59 years	\$ 5,788,663	\$ 5,980,621	\$ 5,633,459	\$ 17,402,743	
60+ years	\$ 7,880,936	\$ 8,142,275	\$ 7,886,843	\$ 23,910,054	
65+ years	\$ 5,095,416	\$ 5,264,385	\$ 4,506,767	\$ 14,866,568	

Age Distribution: Hospitalization, survived; non-Sequelae related	N. est Hospitalizations, survived, non-sequelae	No. days hospitalized	Medical Costs & Fees	Productivity Losses due to Non-work, Direct	Productivity Losses due to Non-work, Indirect
Less than 5 years	46	2.48	\$ 405,529	* \$	48,420
5 - 9 years	36	2.16	\$ 315,513	* \$	27,201
10 - 14 years	34	3.35	\$ 484,760	* \$	28,652
15 - 17 years	23	2.58	\$ 293,556	\$ 7,583	\$ 18,979
18 - 44 years	252	3.51	\$ 4,133,482	\$ 352,625	\$ 177,779
45 - 64 years	217	3.62	\$ 4,053,573	\$ 238,082	\$ 170,725
65 - 84 years	180	4.02	\$ 3,180,517	\$ 45,059	\$ 185,504
85+ years	35	4.70	\$ 728,759	\$ 2,021	\$ 29,646
Aggregate	822	21,726	\$ 13,595,689	\$ 645,369	\$ 686,906

Age Distribution: Guillain-Barre Syndrome	N. est Hospitalizations	No. days hospitalized (Average)	Medical Costs & Fees	Productivity Losses due to Non-work, Direct	Productivity Losses due to Non-work, Indirect	Total
Less than 5 years	3	7.78	\$ 147,956	* \$	8,973	\$ 156,929
5 - 9 years	3	8.10	\$ 164,515	* \$	10,929	\$ 175,444
10 - 14 years	3	8.93	\$ 181,857	* \$	12,791	\$ 194,649
15 - 17 years	3	8.26	\$ 223,880	\$ 2,869	\$ 4,275	\$ 231,024
18 - 44 years	35	12.91	\$ 4,032,765	\$ 174,491	\$ 101,542	\$ 4,308,798
45 - 64 years	38	11.81	\$ 3,954,595	\$ 103,702	\$ 100,725	\$ 4,159,023
65 - 84 years	30	12.88	\$ 3,219,600	\$ 43,687	\$ 127,797	\$ 3,391,084
85+ years	3	13.18	\$ 234,617	\$ -	\$ 6,772	\$ 241,389
Total	118	1,022	\$ 12,159,785	\$ 324,750	\$ 373,805	\$ 12,858,340

Guillain-Barre Syndrome	
Est. No. of Non-Ventilated Patients (Annual)	92
Est. No. of Ventilated Patients (Annual)	26
Medical Costs - Non-ventilated	\$ 10,982,544
Medical Costs - Ventilated	\$ 16,134,408
Est. Prod. Losses from Campy-associated GBS, Non-ventilated, Resumed Work	\$ 393,746
Est. Prod. Losses from Campy-associated GBS, Non-ventilated, Cannot Work	\$ 32,276,777
Est. No. of Non-Ventilated Patients, Resumed Work	75
Est. No. of Non-Ventilated Patients, Cannot Work	17
Est. Prod. Losses from Campy-associated GBS, ventilated, Resumed Work	\$ 434,232
Est. Prod. Losses from Campy-associated GBS, ventilated, Cannot Work	\$ 9,522,835
Est. Prod. Losses from Campy-associated GBS, ventilated, Died	\$ 4,469,562
Est. No. of Ventilated Patients, Resumed Work	16
Est. No. of Ventilated Patients, Cannot Work	7
Est. No. of Ventilated Patients, Died	3
Est. Annual Costs, non-ventilated GBS	\$ 43,653,067
Est. Annual Costs, ventilated GBS	\$ 30,561,037
Est. Total Costs, GBS	\$ 74,214,104

Sequela - Irritable Bowel Syndrome (IBS)	
Est. No. of IBS Patients due to Campylobacteriosis from Boneless Skinless Domestic Chicken Consumption (Annual)	9,890
Direct Costs, IBS	\$ 84,361,445
Indirect Costs, IBS	\$ 21,131,440
Total Costs, IBS	\$ 105,492,885
Distribution of Health Care Costs, Direct - Outpatient Costs, IBS	\$ 33,765,772
Distribution of Health Care Costs, Direct - Inpatient Costs, IBS	\$ 15,243,980
Distribution of Health Care Costs, Direct - Pharmacy or Drug Costs, IBS	\$ 14,030,590
Physician Office Visits, Number of Days/Year, IBS-D	18,136.47
Emergency Room Visits, Number of Days/Year, IBS-D	26,214.19
Hospitalizations, Number of Days/Year, IBS-D	6,215.47

Distribution Chooser - Seasonal		15			
Seasonal	Seasonal Distribution (Weighted)	N est. Ill/yr	N est. hosp./yr	N est. Deaths/yr	
January	4.38%	4,914	44	2	
February	5.70%	6,395	58	2	
March	6.18%	6,934	63	3	
April	6.78%	7,607	69	3	
May	7.43%	8,335	75	3	
June	9.40%	10,545	95	4	
July	11.62%	13,032	117	5	
August	11.85%	13,297	120	5	
September	8.79%	9,854	89	4	
October	9.36%	10,494	95	4	
November	6.68%	7,487	67	3	
December	9.41%	10,551	95	4	

Distribution Chooser - Gender		10	22		
Gender	Gender Distribution	N est. Ill/yr	N est. hosp./yr	N est. Deaths/yr	
Men	50.13%	56,224	507	21	
Women	49.87%	55,943	504	20	

Distribution Chooser - Age Distribution		25		
Age Distribution: Estimated Illness and Deaths, Non-Sequelae	Freq. of Occurrence (Weighted)	N est. Ill/yr	N est. Deaths/yr	
0 - 4 years	17.01%	19,084	7	
5 - 9 years	6.36%	7,128	3	
5 - 14 years	10.95%	12,285	4	
10 - 14 years	4.65%	5,216	2	
15 - 19 years	5.77%	6,470	2	
15 - 24 years	11.73%	13,157	5	
20 - 29 years	12.00%	13,461	5	
30 - 39 years	11.45%	12,838	5	
40 - 49 years	13.44%	15,072	6	
50 - 59 years	12.37%	13,870	5	
60+ years	16.84%	18,883	7	
65+ years	10.88%	12,209	4	