**Campylobacter spp. prevalence and mitigation strategies in the broiler production chain**

# *Campylobacter* spp. prevalence and mitigation strategies in the broiler production chain

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**ABSTRACT**

This study aims to discuss the microbial ecology of the broiler gut environment, *Campylobacter* prevalence across the broiler production chain with a follow-up focus on a possible mitigation strategy based on the use of bacteriophages. Scientific literature published from the last two decades was reviewed and data were collected to establish the ranges of *Campylobacter* loads from different samples. Results showed that the pathogen load in the sample is likely to increase from the different stages of the production chain. Contamination of water and feed represents the most notable source of contamination during the primary production, while cross-contamination of broiler carcasses, skin, and meat occurs during the slaughter, dressing, and processing via machinery, work surfaces, water, and air partially due to the leaking of contaminated feces from visceral rupture. Knowledge gaps were identified and included: a lack of studies detecting *Campylobacter* in broilers in most of the European countries over the last decade and a low number of studies determining the bacterial load in crates used to transport broilers to the slaughterhouse. Determining the prevalence of *Campylobacter* in the broiler industry will enable us to set critical control points to produce broiler flocks and meat products with a low risk of *Campylobacter* contamination.

**Keywords**: Microbiota, chicken, bacteriophage, biocontrol, poultry, public health risk

## 1. INTRODUCTION

Zoonoses are defined as those infectious diseases naturally transmitted from vertebrate animals to humans through direct or indirect contact (food or water contamination). Zoonotic agents include a wide variety of bacteria, viruses, protozoa, insects, and helminths. According to the World Health Organization (WHO), almost 600 million cases of food-borne zoonoses were reported worldwide in 2015, of which 52% were caused by pathogenic bacteria (WHO, 2015). *Campylobacter* is the most common pathogen responsible for food-borne zoonotic diseases in humans and it is considered a serious public health issue in both developing and developed countries. The most recent summary report of the European Food Safety Authority (EFSA) stated that campylobacteriosis is the most frequently reported food-borne zoonoses in the European Union (EU), with 220 682 confirmed cases in 2019, reported from 18 different members states (EFSA and European Centre of Disease Prevention and Control (ECDC), 2019). The most common source of infection in humans due to *Campylobacter* are broiler meat and milk (EFSA and European Centre of Disease Prevention and Control (ECDC), 2019).

*Campylobacter* is a genus of microaerophilic and Gram-negative bacteria belonging to the *Proteobacteria* phylum. Bacterial cells generally appear as slender, spirally curved, or comma-shaped rods characterized by inability to form endospores and ability to change to spherical or coccoid form under stress conditions (Lastovica et al., 2014). *Campylobacter* species have an optimal growth temperature, $O_2$, and $CO_2$ concentration of 30- 42°C, 5-10%, and 3-5%, respectively (Lastovica et al., 2014). In terms of biochemical characteristics of *Campylobacter* species, they are oxidase-positive, with the capacity to reduce fumarate to succinate but are not able to metabolize lipids, starch, gelatin, and casein (Lastovica et al., 2014). Currently, 33 species have been taxonomically described from this genus, but this number is increasing due to

61 the identification of new species (bacterio.net). The main natural reservoir of
62 thermophilic/thermotolerant *Campylobacter* species has been extensively reported in warm-
63 blood animals, including most mammals, birds, and food-producing animals (Silva et al., 2011).
64 The mechanism of colonization, adherence, and invasion of host by pathogenic *Campylobacter*
65 species such as *C. jejuni*, has been previously reviewed elsewhere (Elmi et al., 2021). In
66 summary, successful colonization of the bacteria depends on the ability to attach to the gut
67 mucosa, motility, chemotaxis, spiral shape of the cell, the functionality of the flagella, production
68 of toxins and secreted proteins (cytolethal distending toxin -CDT) and other virulence factors
69 (Elmi et al., 2021).

70 Interestingly, *Campylobacter* does not multiply outside a warm-blooded host due to the
71 absence of microaerobic conditions. However, they can survive when protected from dryness. In
72 this regard, it has been shown that *Campylobacter* can survive up to 3 months in slurries and
73 dirty water (Nicholson et al., 2005). The mechanism of survival of the *Campylobacter* species
74 when exposed to stress environments has been explained by its ability to form a biofilm on
75 abiotic surfaces, this biofilm ensures a supply of nutrients and mechanical protection to survive
76 (Johnson et al., 2017). Although the bacteria cannot multiply outside the animal hosts or in food
77 during storage, it has been shown that they are able to develop complex mechanisms of virulence
78 which remain poorly understood.

79 Recent studies have facilitated a greater appreciation of the complex mechanism of
80 virulence of the members of the *Campylobacter* genus. The full genome of *Campylobacter*
81 *jejuni,* has elucidated strain-specific genetic diversity with high genome plasticity (Bacon et al.,
82 2000). The ability to survive and adapt to stress environments indicates that *C. jejuni* harbors
83 complex virulence and fitness factors (Tegtmeyer et al., 2021). Virulence factors and

pathogenicity islands in *C. jejuni* have been identified and are reported elsewhere (Ali et al., 2012; Bacon et al., 2000; Sierra-Arguello et al., 2021; Tang et al., 2020; Zhang et al., 2017). Virulence-associated genes such as *flaA, cadF, cdtA, cdtB, cdtC, cheY, iamA,* and *virB11* were recently identified in *C. jejuni* and *Campylobacter coli* isolates originating from broiler of 31-day or 37-day age at the rearing period to slaughtering process (Tang et al., 2020). Besides the identification of virulence-associated genes, invasion-associated genes, plasmid genes and CDT-associated genes were also recognized in *C. jejuni* strains isolated from cloacal, broiler carcasses, and broiler slaughterhouses (Sierra-Arguello et al., 2021). The aforementioned genes are involved in the adhesion, invasion, chemotaxis, motility, toxin-activity, and host immune system evasion. Interestingly, there is a difference of virulence factors between *C. jejuni* and *C. coli*, where a higher number of virulence genes were retrieved in *C. jejuni* if compared with *C. coli*, this difference might contribute to the higher colonization of *C. jejuni* in the broilers' intestines (Tang et al., 2020; Zhang et al., 2017).

Antibiotic resistance in *Campylobacter* is also considered a global trend. In this regard, *C. jejuni* and *C. coli* had shown a multi-drug resistance to several antibiotics such as tetracyclines, macrolides, aminoglycosides, and β-lactams. However, a higher number of antibiotic resistance genes were retrieved for *C. jejuni* if compared with *C. coli* (Tang et al., 2020). On the increase of antibiotic resistance to more than one class of antibiotics, further research is needed to understand the mechanism of antimicrobial resistance to improve not only human but also animal health.

Besides the genetic makeup, the main factors that influence the occurrence of *Campylobacter* in broilers are related to the host gut environment, production chain, or farm practices (Barker et al., 2020; Djennad et al., 2017; McKenna et al., 2020; Perez-Arnedo and Gonzalez-Fandos,

2019; Sibanda et al., 2018; Tang et al., 2020). A conceptual framework of the factors increasing the occurrence of *Campylobacter* and a prevention guideline to stipulate the best conditions and food processing management to reduce the risk of *Campylobacter* contamination in the broiler production chain has been developed (EFSA, Panel on Biological Hazards, 2011; Lyngstad, Jonsson, Hofshagen, & Heier, 2008).

Several intervention methods have been developed in recent years, such as the combination of strict biosecurity measures, good manufacturing practice (GMP), hazard analysis and critical control points (HACCP), *Campylobacter* vaccines, antibiotic alternatives to control *Campylobacter*, probiotics, and phytochemicals (Deng et al., 2020; European Food Safety Authority Panel on Biological Hazards, 2011; Umar et al., 2016; Ushanov et al., 2020). However, the problem has not been completely eradicated and the prevalence of this pathogen is still high. Ante- and post-mortem veterinary inspections of broilers are routinely used at the slaughterhouse level as a strategy to ensure that meat does not bear fecal or other contaminants. However, the presence of *Campylobacter* in broiler carcasses cannot be detected visually. As an attempt to mitigate this issue, the application of *Campylobacter*-specific bacteriophages has emerged as one of the most promising approaches to be applied within the farm-to-fork poultry process (Atterbury et al., 2003; Fischer et al., 2013; Hammerl et al., 2014; Kittler et al., 2013; Richards et al., 2019). In this context, this review focuses on discussing the most updated scientific achievements made on the microbial ecology of the gastrointestinal (GI) tract of broilers and the interaction between chickens' gut microbiota and *Campylobacter,* *Campylobacter* prevalence across the broiler production chain with a follow up of the application of bacteriophage along the farm-to-fork process.

## 2. MICROBIAL ECOLOGY IN CHICKEN' ECOSYSTEMS

Chicken ecosystems harbor complex, diverse, and dynamic microbial communities composed of bacteria, protozoa, fungi, yeasts, bacteriophages, and other viruses. The integrity of the GI tract and the gut microbiota composition has a direct influence on chicken' health, affecting the development of their digestive and immune systems (Clavijo and Flórez, 2018; Khan et al., 2020). The avian gut microorganisms are mainly responsible for the continuous generation of energy and nutrients, such as vitamins (K and B groups), amino acids, short-chain fatty acids (SCFA like, butyric, propionic, and lactic acids), ammonia, antimicrobial compounds (bacteriocins) and the decrease of triglyceride concentrations in the avian gut (Vispo and Karasov, 1997). The positive effect of the production of SCFA on the host includes the inhibition of pathogens, reduction of the pH levels in the colon, and stimulation of the cell proliferation in the gut epithelium (Christl et al., 1997; Dibner and Richards, 2005; Ricke, 2003). In contrast, chickens' growth can be depressed when competition for energy and protein occurs between the commensal microbiota. As a result of this competition, toxic metabolites (amino acid catabolites) can be produced and impact the growth of the animal (Shang et al., 2018).

As reported in recent years, the composition and function of chickens' microbiota vary depending on biological changes within and between hosts (age, sex, maternal factors, and breed), and environmental factors (biosecurity levels, housing, litter, feed access and antibiotic administration, hygiene, location, and climate) (Kers et al., 2018). Regarding the biological variation within and between hosts, it has been demonstrated that the chickens' microbiota richness increases during the first week of life (Ballou et al., 2016; Crhanova et al., 2011), while the number of different microbial taxa decreases with chicken age (Lu et al., 2003). The

152 microbial composition of chickens does not only change with chicken age but it is also
153 influenced by the location in the digestive tract and diet (Shang et al., 2018).

154 Significant progress has been made in understanding the microbial taxonomic
155 composition of the different sections of the chickens' GI tract. Overall, *Firmicutes* is the phylum
156 most abundant in both ilea and ceca of chickens (Kumar et al., 2018; Lu et al., 2003; Oakley et
157 al., 2014). Interestingly, in the ceca, the relative abundance of members of the *Clostridium* genus
158 increased by 10 fold between weeks 1 and 6 post-hatch (Oakley and Kogut, 2016). Concerning
159 the bacterial community within the small intestine, in this section lactobacilli mainly dominated
160 the microbial ecosystem (Gong et al., 2007; Lu et al., 2003). According to Lu *et al.,* the
161 microbial composition of the ceca and ileum of three days of age broilers (chickens for meat
162 production) fed with a vegetarian corn-soy diet was mainly composed of *Lactobacillus*
163 *delbrueckii* (13 and 42%, respectively), *Clostridium* spp. (31 and 1%, respectively) and
164 *Clostridium perfringens* (13 and 16%, respectively), however differences in the microbial
165 composition between these sections (ceca and ileum) were reported (Lu et al., 2003). Regarding
166 seven to 49 days old chickens, *Clostridium* spp. remained the most abundant bacteria in the ceca,
167 followed by *Ruminococcus,* while differences in the relative abundance of bacterial species
168 overtime were reported in the ileum (Lu et al., 2003). In detail, *Lactobacillus acidophilus* (50-
169 59%) was the most abundant bacteria in chickens of seven to 21 days of age, while a unique
170 community was reported in the ileum of three, 28, and 49 days of age broilers. Interestingly,
171 regardless of the absence of *Clostridium* spp. and *Ruminococcus* spp. in the ileum section at an
172 early age (between three to 14 days), significant differences of the microbiota between the
173 different sections of the GI tract (ileum and ceca) were only found after 14 days of age (Lu et al.,
174 2003).

175 Overall, it is still not clear whether *Proteobacteria* and *Bacteroidetes* are also abundant
176 phyla during the first six weeks in the chicken's ceca (Shang et al., 2018). These contradictory
177 results may be related to the different chicken breeds studied. According to Kers and colleagues
178 (2018) a variation at the phylum level from the ceca samples of broiler breeds (meat production)
179 and laying-type chickens (egg production) were observed at different time points using 16S
180 rRNA gene amplicon sequencing. Specifically, at zero hr, *Firmicutes* was the most abundant
181 phylum reported in meat-type chicks (Pedroso et al., 2016; Danzeisen et al., 2011), while
182 *Proteobacteria* was identified in laying-type chicks (Ballou et al., 2016). This variability may be
183 due to sample types (fecal *vs* cecal), feed intervention, and/or the technical aspects of the
184 microbial identification as reported elsewhere (Shang et al., 2018). However, from one to 42
185 days of age, *Firmicutes* was the most abundant phylum regardless of the type of broiler breeds.
186 Furthermore, *Candidatus arthromitus,* a desirable bacterium commonly associated with healthy
187 GI tracts in animals, has been also identified in the jejunum and ileum of chickens (Gong et al.,
188 2007, 2002).

189 ***2.1 Chicken diet and intestinal microbiota interaction***

190 The characteristics of the chickens, feed management, the use of medications or vaccines,
191 the environmental conditions of the poultry house, and the housing systems are known factors
192 that have a short- or long-term effect on the intestinal microbiota composition and immune
193 system development of chickens (Kers et al., 2018). The effect of feed management on the
194 intestinal microbiota has been extensively studied (Apajalahti, 2005; McKenna et al., 2020;
195 Singh et al., 2014; Stanley et al., 2012; Takeshita et al., 2021). Differences in the relative
196 abundance of bacterial species in fecal and cecal samples of broilers following high and low
197 growth diets were reported elsewhere (Singh et al., 2014; Stanley et al., 2012). Knarreborg and

198 colleagues (2002) demonstrated that the divergence in feed can also increase or decrease the
199 relative abundance of a specific bacterial group: the aforementioned study shows how pellet feed
200 increases the number of *Enterococcus* spp. and coliforms and decreases lactobacilli species and
201 *C. perfringens* in the ileum of broilers when compared with mash feed (Knarreborg et al., 2002).
202 In contrast, when chickens consumed corn, this diet favors a decrease in the number of clostridia,
203 enterococci, and lactobacilli, while when chickens consumed wheat, it favors the increase of
204 bifidobacteria (Apajalahti, 2005). Interestingly, the amount of protein in the chicken feed also
205 changed microbial composition, where high amount of protein showed a lower relative
206 abundance of lactobacilli species compared with chicken feed with a low amount of proteins diet
207 (Takeshita et al., 2021). The difference in microbial community structure between production
208 systems together with different management paraments such as stocking density has also shown
209 to alter the microbiota of broilers (McKenna et al., 2020). To date, broilers' microbiota studies
210 have focused on identifying bacterial composition while the identification of other components,
211 such as fungi, phages, or viruses, remain unclear. A better understanding of the role and
212 interactions between mycobiota, phagobiota, and virobiota with the broiler microbial ecosystem
213 may help to improve chicken productivity, health, and welfare and develop novel strategies for
214 controlling the prevalence of *Campylobacter* spp. in broilers (Silva et al., 2011).

215 ***2.2 Campylobacter and its interactions with gut microbiota in chickens***

216 *Campylobacter* typically occurs within two weeks onwards and increases during broilers
217 life cycle (Ijaz et al., 2018; Kalupahana et al., 2013; McKenna et al., 2020; Neill et al., 1984;
218 Thibodeau et al., 2015). It usually grows in the ceca and cloaca and can also colonize the spleen,
219 blood, and liver (Lin, 2009). *C. jejuni* and *C. coli* are the most commonly isolated
220 *Campylobacter* species in broiler samples at different production stages. Interestingly, recent

studies show contradictory results on how microbial ecology influences *Campylobacter* colonization. According to Sofka and colleagues the presence of *Campylobacter* decreased the diversity of intestinal microbiota when compared with non-colonized broilers (Sofka et al., 2015). In alignment with the aforementioned study, a significant difference in the relative abundance of the microbial operational taxonomic units detected in the ceca of chickens fed with different diets, at different ages from three different commercial broiler farms were reported between *Campylobacter*-positive and -negative chickens (Takeshita et al., 2021). Interestingly, the decrease in lactobacilli abundance in chicken ceca was associated with high levels of *Campylobacter,* while the growth of *Campylobacter* increased the levels of *Enterobacteriaceae* (Sakaridis et al., 2018). However, according to McKenna and colleagues the presence of *Campylobacter* in broilers reared under standard industrial growing systems increased the cecal microbial community structure (McKenna et al., 2020). Whereas the transplantation of cecal microbial in chickens shows no significant difference in the ceca microbial communities of different inbred chickens (Chintoan-Uta et al., 2020). The factors affecting host-pathogen ecology in terms of the microbiome and the microbial dynamics and *Campylobacter* presence remain poorly studied at an industrial or small-scale farm level.

## *3. CAMPYLOBACTER* PREVALENCE IN THE BROILER PRODUCTION CHAIN

In terms of *Campylobacter* diversity in the broiler chain production, a recent study has demonstrated that *C. jejuni* predominates during the rearing broiler period while more isolates of *C. coli* were detected during the slaughtering process (Chen et al., 2010; Tang et al., 2020; Zhang et al., 2018).

243 *Campylobacter* from chicken reservoirs may reach humans through the environment or by
244 direct contact and mainly through the consumption of raw and undercooked contaminated broiler
245 meat and meat products. The Scientific Opinion of the Panel on Biological Hazards (BIOHAZ),
246 EFSA in 2010 estimated that the majority of human campylobacteriosis is attributed to the
247 chicken reservoir as a whole (50 – 80%), while the handling, preparation, and consumption of
248 broiler meat may account only for 20 to 30% (EFSA, 2010). Other food products such as
249 unpasteurized dairy products and contaminated water are also vehicles of transmission of this
250 pathogen, but *Campylobacter* infections are less common from these sources compared with
251 meat products (Josefsen et al., 2015). To provide an indication of the possible control points for
252 *Campylobacter* at the broiler meat production chain, this review describes the prevalence of
253 *Campylobacter* along the process and discusses the risk factors that influence the level of
254 contamination (Figure 1).

255 ***3.1 Primary production***

256 The increases of *Campylobacter* loads during the first weeks of age of commercial flocks
257 (Umar et al., 2016) indicate that vertical transmission of the pathogen does not commonly occur;
258 however, it still represents an important risk factor for the *Campylobacter* colonization in
259 broilers (Bull et al., 2008; European Food Safety Authority Panel on Biological Hazards, 2011;
260 Humphrey, 2006; Tang et al., 2020). The protection of young broilers' GI tract against
261 colonization of *Campylobacter* has been associated with *Campylobacter*-specific maternal
262 antibodies (MAB) (Sahin et al., 2001; Vandeplas et al., 2010). MAB, predominantly
263 immunoglobulin G (IgG) class, are transported from the egg yolk across the yolk sac membrane
264 into the embryonic circulation to protect young broiler chickens from *C. jejuni* infections
265 (Linden and Roth, 1978). However, MAB levels change with the increase in the broiler age. In

detail, the highest level of MAB is reported during the first two weeks after hatching, followed by a decrease, reaching minimal values at the third and fourth weeks of age (Sahin et al., 2001; Vandeplas et al., 2010). The increased risk of *Campylobacter* colonization in broilers from week three to week six was demonstrated elsewhere, as shown in Table 1 (Ingresa-Capaccioni et al., 2016; Perez-Arnedo and Gonzalez-Fandos, 2019; Tang et al., 2020; Tangkham et al., 2016). Interestingly, the prevalence of *Campylobacter* spp. and *C. jejuni* at the end of the rearing period is higher in open housing compared with environmentally controlled housing (Tangkham et al., 2016). Noteworthy, a higher prevalence of *C. jejuni* isolates compared with *C. coli* was observed from the broiler rearing period at the farm level in China (Tang et al., 2020), while in Spain, *C. coli* has not been detected throughout chickens progeny (Ingresa-Capaccioni et al., 2016; Perez-Arnedo and Gonzalez-Fandos, 2019).

Natural colonization of broilers by single or multiple *Campylobacter* species rapidly occurs through horizontal transmission from vectors such as domestic pets, insects, rodents and migratory birds, farm equipment, transport vehicles, farmworkers, drinking water, feed, litter, air, or thinning (Bang et al., 2002; Carvalho et al., 2010; Gharib Naseri et al., 2012; Koolman et al., 2014; Robyn et al., 2013; Schroeder et al., 2014; Stern et al., 2001). Based on the most recent studies considered in this review, the highest prevalence of *Campylobacter* presence has been detected on drinking water, feed, workers boots, and broilers' fecal and cecal samples after thinning, while the lowest prevalence of this bacteria was found on samples from the litter, air and the infrastructure of the farm (Table 1).

The colonization of the flocks with *Campylobacter* can also be introduced from external visitors to the farm, maintenance personnel, bird catching crew, close livestock, when operators visit more than one farm or are negligent regarding hygienic practices (Berndtson et al., 1996;

Cardinale et al., 2004; Hald et al., 2000; Johnsen et al., 2007; Kapperud et al., 1993; A. M. Ridley et al., 2011). Besides human traffic, the proximity of the fresh litter, larger thinning crews, increasing the number of broiler houses on-site, and the presence of dead broilers in the house enhances the survival of *Campylobacter* and thus the risk of positive flocks is increased (Cardinale et al., 2004; Koolman et al., 2014; Lyngstad et al., 2008). Inadequate biosecurity such as broiler houses older than 15 years, absence of anterooms and barriers in each house, the use of shared tools between houses, long downtime, and drinking systems with bells or cups represent a common risk factor for *Campylobacter* colonization of broiler flocks (Sommer et al., 2016). Partial thinning of broiler flocks has also been considered as a potential risk factor for *Campylobacter* colonization of the remaining animals, due to the difficulty of keeping biosecurity measures during the process (Hermans et al., 2011).

The dispersion of *Campylobacter* originating from broilers into the environment represents an important factor leading to increased contamination. In broiler, *C. jejuni* is the most predominant species colonizing the flocks, followed by *C. coli* and occasionally by other species (Rossler et al., 2019; Umar et al., 2016). Once the first bird is infected, *Campylobacter* is horizontally transmitted to most of the birds in a flock within only a few days, reaching between $10^6$ and $10^8$ CFU/g in their intestinal tract, and they remain colonized until slaughter (Marotta et al., 2015).

The increased water consumption by chickens during summer months increases the risk of drinking water contaminated with *Campylobacter* (Herman et al., 2002; Lyngstad et al., 2008). This association has been observed in northern European countries, such as Sweden, Denmark, Norway, and the Netherlands (Jore et al., 2010). The changes in the temperature throughout the year can explain the increase in water consumption during summer. However, other factors such

as the increment in the abundance of transmission vectors of *Campylobacter* (flies) and the need for ventilation in the poultry house of broilers can also increase the probabilities of the introduction of *Campylobacter* into the environment (Hald et al., 2008; Jore et al., 2010). Also, it must be underlined that, as expected, climate change is an important factor that might increase the prevalence of *Campylobacter* in the future, placing public health at risk. It is worth noting that colonized birds are predominantly asymptomatic, with no negative effect on their health (Pielsticker et al., 2012).

### *3.2 Transportation before slaughter*

The transportation step consists of moving the broilers from farm facilities to the slaughterhouse. During this process, the animals are confined in crowded crates or cages, deprived of water and feed, and undergo continuous stress that affect not only the meat quality but also the *Campylobacter* prevalence, which could increase due to cross-contamination. If a single broiler is colonized, *Campylobacter* will be spread to the environment and will be transmitted to the rest through fecal matter or feathers contact (Stern et al., 2001; Whyte et al., 2001).

In comparison with the primary production, the pre- and post-transportation steps have been less studied over the last two decades (Table 2). Overall, from the literature available we observed that during transportation, the floor and transportation cage/crates account for the highest risk of cross-contamination in broilers (Stern et al., 2001; Willis et al., 2002), while no clear contamination level in fecal samples between pre-and post-transportation steps is observed (Whyte et al., 2001). One likely explanation for this phenomenon is that catching and placing the chickens in cages/crates to transport them to the processing plant increases the risk of contamination, mostly due to cross-contamination during transportation (Slader et al., 2002).

However, recent literature has demonstrated the ineffectiveness of cleaning naturally contaminated crates before using them to transport broilers and reduce *Campylobacter* infections (A. Ridley et al., 2011). Further, transportation stress alters excretion rates of *Campylobacter* in the fecal material of broilers (Whyte et al., 2001).

### *3.3 Slaughter, dressing, and processing*

Colonization of *Campylobacter* in broilers during rearing and transportation steps contributes to the contamination of this bacteria in the slaughterhouse causing cross-contamination. During the slaughter process, the transmission of *Campylobacter*-infected flocks to non-infected (Shange et al., 2019; Umar et al., 2016) is mainly due to the leaking of contaminated feces from visceral rupture to the chickens (García-Sánchez et al., 2019; Hermans et al., 2011). In the European Union (EU), it was observed that batches of broilers whose intestines were colonized with *Campylobacter* yielded carcasses with high numbers of *Campylobacter* (EFSA, Panel on Biological Hazards, 2011). However, *Campylobacter* counts on carcasses varied among slaughterhouses. These differences found on the levels of *Campylobacter* loads among slaughterhouses might be related to the different hygiene practices used between countries. Interestingly, *Campylobacter* strains in chickens are not necessarily the same as those isolated from processed carcasses, which suggests that cross-contamination occurs during processing (Slader et al., 2002).

This cross-contamination can occur during the entire slaughter, dressing, and processing including the chilling room, plucking, evisceration and portioning areas, or via machinery, work surfaces, process water and air (Allen et al., 2003; Arnold and Silvers, 2000; Corry and Atabay, 2003; Haas et al., 2005; Hue et al., 2010; Johnsen et al., 2007). Overall, based on the most recent studies considered in this review, the highest prevalence of *Campylobacter* spp. was detected on

the defeathering, evisceration, operation tables, worker's gloves, shackles, and conveyor belt equipment, while the lowest prevalence of *Campylobacter* spp. was detected on the sink, floor and chopping boards and knife swabs during slaughter, dressing, and processing as shown in Table 3 (García-Sánchez et al., 2017; Khan et al., 2018; Tang et al., 2020; Zhang et al., 2018). In agreement with data from the primary production, where the surfaces and equipment in the facility are the main sources for *Campylobacter*, a high prevalence of this bacteria in broiler samples and carcasses has been also reported during the slaughter, dressing and processing (Table 3) (Carrillo et al., 2014; Casagrande Proietti et al., 2018; García-Sánchez, Melero, Diez, Jaime, & Rovira, 2018; Ingresa-Capaccioni et al., 2016; Khan et al., 2018; Korsak, Maćkiw, Rożynek, & Żyłowska, 2015; Perez-Arnedo & Gonzalez-Fandos, 2019; Williams & Oyarzabal, 2012; Zhang et al., 2018).

The variation of the prevalence of *Campylobacter* spp. during the different processes' steps can also be explained due to technical aspects of *Campylobacter* detection (sampling procedures, storage, DNA extraction, selection of targeting region, and PCR primers and the sequencing platforms used). Culture-based isolation approaches is considered as a standard method for the detection and enumeration of the different *Campylobacter* spp. of products intended for human consumption, animal feeding, environmental samples in the area of food and feed production, and samples from the primary production stage (ISO, 2017). However, the limitations of this technique rely on the difficulties to isolate *Campylobacter* from samples with heavy contamination and the rapid loss in cultivability of isolates. To overcome the challenges in traditional phenotype-based methods for the identification of *Campylobacter*, different DNA-based approaches have become widely used due to the speed, and reproducibility to confirm *Campylobacter* identification (Johannessen et al., 2020). Nevertheless, besides the great

advances made in establishing a less time-consuming sampling protocol and more amenable to couple with DNA-base methods, currently, there is no standard procedure for a fast screening of *Campylobacter* at the retail level. One also notes that pathogenesis or virulence factors that certain *Campylobacter* sequence types may have is an important feature to consider for predicting future *Campylobacter* outbreaks and accurate identification in the context of risk assessment.

## 4. CONTROL STRATEGIES

A direct relationship between the reduction of *Campylobacter*'s load at the different broiler production stages and the reduction of public health risk has been linked to an effective control strategy. Reducing the numbers of *Campylobacter* on the carcasses by one $\log_{10}$-unit, would reduce the public health risk by between 50 and 90%, and, reducing counts by more than two $\log_{10}$-units would reduce the public health risk by more than 90% (European Food Safety Authority Panel on Biological Hazards, 2011). To reduce *Campylobacter* loads, the EU has recently developed the Commission Regulation (EU) 2017/1495, which sets microbiological limits regarding *Campylobacter* spp. in carcasses of broilers (European Commission, 2017). In recent years several *Campylobacter* control strategies have been developed; most of them focused on the reduction of *Campylobacter* colonization at the farm level which consequently decreased *Campylobacter* loads into the slaughterhouse, resulting in a low concentration or absence of the pathogen on the final product (Wagenaar et al., 2006).

The main strategies to control *Campylobacter* spp. colonization at the farm level is based on the reduction of environmental exposure (biosecurity and hygienic measures), the increase of

broiler resistance to colonization (competitive exclusion, vaccination, application of pre-and pro-biotics, organic acids, or phytocompounds etc.), the use of alternative antimicrobials (bacteriophage therapy and bacteriocin treatment), and/or selection of specific breeding to increase the resistance of broiler chickens to colonization (European Food Safety Authority Panel on Biological Hazards, 2011; Umar et al., 2016). Besides, the BIOHAZ sets sanitation practices during thinning to prevent *Campylobacter* from entering broiler houses at primary production, and the application and monitor system of the decontamination of carcasses (using chemical or physical treatments) are recommended (EFSA, Panel on Biological Hazards, 2011).

At the transportation stage the improvement of hygienic measures by removing feed and litter, cleaning and disinfecting transport crates, are the main strategies studied (Meunier et al., 2016). However, besides the importance of the transportation step, at the moment the BIOHAZ has not published any recommendation to prevent and/or reduce the contamination of *Campylobacter* during this processing step. At slaughter, dressing, and processing the most common and effective strategies used to reduce *Campylobacter* loads is the application of specific food safety protocols and strict hygienic practices (HACCP), separating *Campylobacter*-infected flocks from non-infected, physical treatments (scalding, chilling) and chemical decontamination of carcasses using chlorine compounds or chlorine-based antimicrobials (Osimani et al., 2017; Silva et al., 2011). One of the disadvantages of using physical treatments is that it contributes to the change of organoleptic properties of the food products, which would make them less desirable to the consumers. In addition, physical decontamination is allowed in the United States but not in the EU.

In the EU, bacteriophages or bacteriocins in the feed are used to reduce the load of *Campylobacter* in the GI tract of broilers before slaughtering, a reduction of the slaughter age of

broilers, implementation and improvement of the sanitation practices during slaughter (including the design of adequate equipment with the prevention of fecal leakage), and training food handlers with better hygienic practices to prevent or reduce the *Campylobacter* colonization in the slaughter, dressing and processing steps are recommendations made by the EFSA to promote good processing practices (EFSA, Panel on Biological Hazards, 2011).

## 5. THE APPLICATION OF BACTERIOPHAGES TO COMBAT *CAMPYLOBACTER* IN BROILER PRODUCTION

Despite the extensive efforts from the broiler industry, food safety authorities, and academia, there is no effective, reliable, and practical intervention control strategy able to reduce the prevalence of *Campylobacter* from the farm-to-fork process. As the incidence of antibiotic-resistant *Campylobacter* strains is increasing, the development of novel non-antibiotic anti-*Campylobacter* treatments is becoming critical (Johnson et al., 2017). Treatment strategies that have shown highly promising results for *Campylobacter* control in broiler chickens are currently under development. Among them, the use of specific bacteriophages (phages) as biocontrol agents is considered one of the most promising strategies to reduce the prevalence of *Campylobacter* in the broiler production chain (Atterbury et al., 2003; Carvalho et al., 2010; El-Shibiny et al., 2009; Fischer et al., 2013; Hammerl et al., 2014). Bacteriophages are viruses that specifically infect and kill bacteria, widely distributed in the environment from the human GI tract to the deep ocean, and often naturally present in animals such as, broilers (Dion et al., 2020; Nafarrate et al., 2021). The interest in using phages as a safety strategy in food production relies on its selectivity towards the pathogen of concern, it is harmless to humans, animals, and plants, and does not affect the existing commensal microbiota or alter food properties.

449 *Campylobacter*-specific phage cocktail (phiCcolBB12, phiCcolBB35, and phiCcolBB37) has
450 been applied at pre-slaughter and post-slaughter stages to reduce bacterial loads (Carvalho et al.,
451 2012). In detail, the application of *Campylobacter*-specific bacteriophages in the broiler
452 production chain has been tested in several studies, focusing on the administration of
453 bacteriophages into the drinking water during the rearing cycle of broilers (Loc Carrillo et al.,
454 2005; El-Shibiny et al., 2009; Fischer et al., 2013; Hammerl et al., 2014; Kittler et al., 2013;
455 Richards, Connerton, & Connerton, 2019) or using phages on raw and processed meat or raw
456 liver (Atterbury et al., 2003; Firlieyanti et al., 2016; Goode et al., 2003).

457 During the rearing cycle of broilers, the reduction rates in the cecal content achieved by the
458 addition of single bacteriophages or bacteriophage cocktails (phage NCTC 12673, 12674, and
459 12678) showed promising potential reducing bacterial loads between 2.5 to 3.2 log CFU/g
460 (Fischer et al., 2013; Kittler et al., 2013). In addition, it was demonstrated that no adverse effects
461 on the broiler microbiota occur after administering a phage cocktail (CP20 and CP30A) in
462 contrast to administering broad-spectrum antibiotic treatments, which can yield dysbiosis in the
463 gut microbiota (Richards et al., 2019). Overall, the results from most of the studies in broilers
464 conclude that bacteriophages (NCTC 12672, 12673, 12674, 12678, 12669, 12671, 12684, CP8,
465 CP34, CP81, Cj6, phiCcolIBB35, phiCcolIBB37, and phiCcolIBB12) can effectively reduce
466 *Campylobacter* levels if they are administered 24-48 h prior to slaughter (Ushanov et al., 2020).

467 Besides the use of bacteriophages in the primary production, a mean reduction of
468 approximately one log CFU/g of *Campylobacter* on broiler products was obtained using single
469 bacteriophages (NCTC 12674 and 12673) during meat processing (Atterbury et al., 2003; Goode
470 et al., 2003). In this case, different authors suggest the application of bacteriophages
471 (phiCcolIBB35, phiCcolIBB37, and phiCcolIBB12) at high titers to achieve successful

472 reductions in *Campylobacter* counts (Carvalho et al., 2012). In addition, bacteriophages (CP8
473 and CP30) have also shown successful dispersal of *Campylobacter* biofilms and a reduction of
474 viable cells ranging from one to three log CFU/cm$^2$ (Siringan et al., 2011), indicating an
475 additional potential field for phage application to target biofilms in meat processing facilities.

476 From an epidemiological point of view, the bacteriophage treatment in the production chain
477 can contribute to a drastic reduction of the infection risk for the consumer. According to the
478 model prediction, phage therapy could lead to a reduction of *Campylobacter* in the fecal matter
479 and on the surface of broiler chicken meat (one log each), resulting on a 90% risk reduction for
480 consumer infection (Havelaar et al., 2007). Clinical data suggest the tolerability and/or
481 effectiveness of phage therapy to reduce antibiotic-resistant infections in humans, but also phage
482 resistance (El Haddad et al., 2019; Zhvania et al., 2017). Bacteria can promote phage attack
483 mainly through spontaneous chromosomal mutations governed by Darwinian dynamics, leading
484 to the emergence of phage resistance and consequently treatment failure (Luong et al., 2020).
485 Identifying new phages with different binding sites to improve efficacy may aid in the prevention
486 of problems related to phage resistance (Wright et al., 2019).

487 More research is needed to find routes of administration, phage selection, the order of phage
488 exposure frequency of administration, dosage, phage resistance, pharmacokinetic and
489 pharmacodynamic properties of the phages, and improve bacteriophage efficacy against
490 *Campylobacter* and broiler meat safety. It is worth noting that bacteriophages should not be
491 considered as a substitute for the control strategies developed so far, but rather seen as a
492 complementary strategy. Successful control of *Campylobacter* could probably be achieved by
493 implementing strict biosecurity and hygiene measures in combination with bacteriophage
494 treatments.

## 6. CONCLUSION

The high occurrence of *Campylobacter* along the broiler production chain is a serious threat to public health. This review revealed that abiotic factors have the potential to contribute to cross-contamination of *Campylobacter*. Furthermore, the transfer of contaminated content of the bird intestine, or persisting biofilm on equipment/surfaces represents likely the source of cross-contamination during the broiler production. Effective *Campylobacter* control measures along the whole broiler production chain are, therefore, needed to improve broiler meat safety, resulting in a reduction of the incidence of human campylobacteriosis. The use of *Campylobacter*-specific bacteriophages at different points from farm-to-fork (in livestock, slaughter, and/or processing facilities) has been proposed as an additional strategy of a multistage bio-security measure to assure safer chicken products for the consumer. The use of multi-omics approaches can help us to increase our understanding of the ability of this foodborne pathogen to persist through the water and the food chain, its environmental niche, and how it interacts with bacteriophages. Progress in this field will help us to better understand how to assess the environmental conditions and nutritional requirements to reduce the risk of *Campylobacter* contamination in the broiler production chain.

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L.L; Validation, Review and Editing, K.R, L.L, I.F, L.C, A.L, I.N, J.MG; Writing-original Draft Preparation, J.MG; Supervision, K.R.

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## 7. REFERENCES

Ali, A., Soares, S.C., Santos, A.R., Guimarães, L.C., Barbosa, E., Almeida, S.S., Abreu, V.A.C., Carneiro, A.R., Ramos, R.T.J., Bakhtiar, S.M., Hassan, S.S., Ussery, D.W., On, S., Silva, A., Schneider, M.P., Lage, A.P., Miyoshi, A., Azevedo, V., 2012. Campylobacter fetus subspecies: Comparative genomics and prediction of potential virulence targets. Gene 508, 145–156. https://doi.org/10.1016/j.gene.2012.07.070

536 Allen, V.M., Tinker, D.B., Hinton, M.H., Wathes, C.M., 2003. Dispersal of micro-organisms in
537 commercial defeathering systems. Br. Poult. Sci. 44, 53–59.
538 https://doi.org/10.1080/0007166031000085436

539 Apajalahti, J., 2005. Comparative gut microflora, metabolic challenges, and potential
540 opportunities of gastrointestinal. J. Appl. Poult. Res. 14, 444–453.
541 https://doi.org/https://doi.org/10.1093/japr/14.2.444

542 Arnold, J.., Silvers, S., 2000. Comparison of poultry processing equipment surfaces for
543 susceptibility to bacterial attachment and biofilm formation. Poult. Sci. 79, 1215–1221.
544 https://doi.org/https://doi.org/10.1093/ps/79.8.1215

545 Atterbury, R.J., Connerton, P.L., Dodd, C.E.R., Rees, C.E.D., Connerton, I.F., 2003. Application
546 of host-specific bacteriophages to the surface of chicken skin leads to a reduction in
547 recovery of Campylobacter jejuni. Appl. Environ. Microbiol. 69, 6302–6306.
548 https://doi.org/10.1128/AEM.69.10.6302-6306.2003

549 Bacon, D.J., Alm, R., Burr, D.H., Hu, L., Kopecko, D.J., Ewing, C.P., Trust, T.J., Guerry, P.,
550 2000. Involvement of a plasmid in virulence of Campylobacter jejuni 81-176. Infect.
551 Immun. 68, 4384–4390. https://doi.org/10.1128/IAI.68.8.4384-4390.2000

552 bacterio.net, n.d. List of prokaryotic names with standing in nomenclature (LPSN): Genus,
553 Campylobacter [WWW Document]. URL https://www.bacterio.net/genus/campylobacter
554 (accessed 3.19.21).

555 Ballou, A.L., Ali, R.A., Mendoza, M.A., Ellis, J.C., Hassan, H.M., Croom, W.J., Koci, M.D.,
556 2016. Development of the chick microbiome: How early exposure influences future

557 microbial diversity. Front. Vet. Sci. 3, 1–12. https://doi.org/10.3389/fvets.2016.00002

558 Bang, D.D., Wedderkopp, A., Pedersen, K., Madsen, M., 2002. Rapid PCR using nested primers
559 of the 16s rRNA and the hippuricase (hipO) genes to detect Campylobacter jejuni and
560 Campylobacter coli in environmental samples. Mol. Cell. Probes 16, 359–369.
561 https://doi.org/10.1006/mcpr.2002.0434

562 Barker, C.R., Painset, A., Swift, C., Jenkins, C., Godbole, G., Maiden, M.C.J., Dallman, T.J.,
563 2020. Microevolution of Campylobacter jejuni during long-term infection in an
564 immunocompromised host. Sci. Rep. 10. https://doi.org/10.1038/s41598-020-66771-7

565 Berndtson, E., Danielsson-Tham, M.L., Engvall, A., 1996. Campylobacter incidence on a
566 chicken farm and the spread of Campylobacter during the slaughter process. Int. J. Food
567 Microbiol. 32, 35–47. https://doi.org/10.1016/0168-1605(96)01102-6

568 Bull, S.A., Thomas, A., Humphrey, T., Ellis-Iversen, J., Cook, A.J., Lovell, R., Jorgensen, F.,
569 2008. Flock health indicators and Campylobacter spp. in commercial housed broilers reared
570 in Great Britain. Appl. Environ. Microbiol. 74, 5408–5413.
571 https://doi.org/10.1128/AEM.00462-08

572 Cardinale, E., Tall, F., Guèye, E.F., Cisse, M., Salvat, G., 2004. Risk factors for Campylobacter
573 spp. infection in Senegalese broiler-chicken flocks. Prev. Vet. Med. 64, 15–25.
574 https://doi.org/10.1016/j.prevetmed.2004.03.006

575 Carrillo, C.D., Plante, D., Iugovaz, I., Kenwell, R., Belanger, G., Boucher, F., Poulin, N.,
576 Trottier, Y.L., 2014. Method-dependent variability in determination of prevalence of
577 Campylobacter jejuni and Campylobacter coli in Canadian retail poultry. J. Food Prot. 77,

578 1682–1688. https://doi.org/10.4315/0362-028x.jfp-14-133

579 Carvalho, C.M., Gannon, B.W., Halfhide, D.E., Santos, S.B., Hayes, C.M., Roe, J.M., Azeredo,
580 J., 2010. The in vivo efficacy of two administration routes of a phage cocktail to reduce
581 numbers of Campylobacter coli and Campylobacter jejuni in chickens. BMC Microbiol. 10,
582 232. https://doi.org/https://doi.org/10.1186/1471-2180-10-232

583 Casagrande Proietti, P., Pergola, S., Bellucci, S., Menchetti, L., Miraglia, D., Franciosini, M.P.,
584 2018. Occurrence and antimicrobial susceptibility of Campylobacter spp. on fresh and
585 refrigerated chicken meat products in Central Italy. Poult. Sci. 97, 2895–2901.
586 https://doi.org/10.3382/ps/pey147

587 Chen, X., Naren, G.-W., Wu, C.-M., Wang, Y., Dai, L., Xia, L.-N., Luo, P.-J., Zhang, Q., Shen,
588 J.-Z., 2010. Prevalence and antimicrobial resistance of Campylobacter isolates in broilers
589 from China. Vet. Microbiol. 144, 133–139. https://doi.org/10.1016/j.vetmic.2009.12.035

590 Chintoan-Uta, C., Wisedchanwet, T., Glendinning, L., Bremner, A., Psifidi, A., Vervelde, L.,
591 Watson, K., Watson, M., Stevens, M.P., 2020. Role of cecal microbiota in the differential
592 resistance of inbred chicken lines to colonization by campylobacter jejuni. Appl. Environ.
593 Microbiol. 86. https://doi.org/10.1128/AEM.02607-19

594 Christl, S.U., Bartram, H.P., Paul, A., Kelber, E., Scheppach, W., Kasper, H., 1997. Bile acid
595 metabolism by colonic bacteria in continuous culture: Effects of starch and pH. Ann. Nutr.
596 Metab. 41, 45–51. https://doi.org/10.1159/000177977

597 Clavijo, V., Flórez, M.J. V, 2018. The gastrointestinal microbiome and its association with the
598 control of pathogens in broiler chicken production: A review. Poult. Sci. 97, 1006–1021.

https://doi.org/10.3382/ps/pex359

Corry, J.E.L., Atabay, H.I., 2003. Poultry as a source of Campylobacter and related organisms. J. Appl. Microbiol. 90, 96S-114S. https://doi.org/10.1046/j.1365-2672.2001.01358.x

Crhanova, M., Hradecka, H., Faldynova, M., Matulova, M., Havlickova, H., Sisak, F., Rychlik, I., 2011. Immune response of chicken gut to natural colonization by gut microflora and to Salmonella enterica serovar enteritidis infection. Infect. Immun. 79, 2755–2763. https://doi.org/10.1128/iai.01375-10

Danzeisen, J.L., Kim, H.B., Isaacson, R.E., Tu, Z.J., Johnson, T.J., 2011. Modulations of the chicken cecal microbiome and metagenome in response to anticoccidial and growth promoter treatment. PLoS One 6. https://doi.org/10.1371/journal.pone.0027949

Deng, W., Dittoe, D.K., Pavilidis, H.O., Chaney, W.E., Yang, Y., Ricke, S.C., 2020. Current perspectives and potential of probiotics to limit foodborne Campylobacter in poultry. Front. Microbiol. 11. https://doi.org/10.3389/fmicb.2020.583429

Dibner, J.J., Richards, J.D., 2005. Antibiotic growth promoters in agriculture: History and mode of action actual usage of antimicrobials in Denmark voluntary and legislated bans. Poult. Sci. 84, 634–643. https://doi.org/https://doi.org/10.1093/ps/84.4.634

Dion, M.B., Oechslin, F., Moineau, S., 2020. Phage diversity, genomics and phylogeny. Nat. Rev. Microbiol. 18, 125–138. https://doi.org/10.1038/s41579-019-0311-5

Djennad, A., Nichols, G., Loiacono, G., Fleming, L., Kessel, A., Kovats, S., Lake, I., Sarran, C., Elson, R., Lane, C., Hoeser, C., Bailey, T., 2017. The seasonality and effects of temperature

619 and rainfall on Campylobacter infections. Int. J. Popul. Data Sci. 1, 1–10.
620 https://doi.org/10.23889/ijpds.v1i1.51

621 El-Shibiny, A., Scott, A., Timms, A., Metawea, Y., Connerton, P., Connerton, I., 2009.
622 Application of a group II campylobacter bacteriophage to reduce strains of campylobacter
623 jejuni and campylobacter coli colonizing broiler chickens. J. Food Prot. 72, 733–740.
624 https://doi.org/10.4315/0362-028X-72.4.733

625 El Haddad, L., Harb, C.P., Gebara, M.A., Stibich, M.A., Chemaly, R.F., 2019. A systematic and
626 critical review of bacteriophage therapy against multidrug-resistant ESKAPE organisms in
627 humans. Clin. Infect. Dis. 69, 167–178. https://doi.org/10.1093/cid/ciy947

628 Elmi, A., Nasher, F., Dorrell, N., Wren, B., Gundogdu, O., 2021. Revisiting Campylobacter
629 jejuni virulence and fitness factors: Role in sensing, adapting, and competing. Front. Cell.
630 Infect. Microbiol. 10. https://doi.org/10.3389/fcimb.2020.607704

631 European Commission, 2017. Commission Regulation (EU) 2017/1495 of 23 August 2017
632 amending Regulation (EC) No 2073/2005 as regards Campylobacter in broiler carcases.
633 Off. J. Eur. Union L 218, 1–6.

634 European Food Safety Authority (EFSA), 2010. Analysis of the baseline survey on the
635 prevalence of Campylobacter in broiler batches and of Campylobacter and Salmonella on
636 broiler carcasses, in the EU, 2008 - Part B. EFSA J. 8, 1522.
637 https://doi.org/10.2903/j.efsa.2010.1522

638 European Food Safety Authority and European Centre of Disease Prevention and Control (EFSA
639 and ECDC), 2019. The European union one health 2018 zoonoses report. EFSA J. 17.

https://doi.org/10.2903/j.efsa.2019.5926

European Food Safety Authority Panel on Biological Hazards, (BIOHAZ), 2011. Scientific opinion on Campylobacter in broiler meat production: control options and performance objectives and/or targets at different stages of the food chain. EFSA J. 9, 2105. https://doi.org/10.2903/j.efsa.2011.2105

Firlieyanti, A.S., Connerton, P.L., Connerton, I.F., 2016. Campylobacters and their bacteriophages from chicken liver: The prospect for phage biocontrol. Int. J. Food Microbiol. 237, 121–127. https://doi.org/10.1016/j.ijfoodmicro.2016.08.026

Fischer, S., Kittler, S., Klein, G., Glünder, G., 2013. Impact of a single phage and a phage cocktail application in broilers on reduction of Campylobacter jejuni and development of resistance. PLoS One 8, 1–13. https://doi.org/10.1371/journal.pone.0078543

García-Sánchez, L., Melero, B., Diez, A.M., Jaime, I., Rovira, J., 2018. Characterization of Campylobacter species in Spanish retail from different fresh chicken products and their antimicrobial resistance. Food Microbiol. 76, 457–465. https://doi.org/10.1016/j.fm.2018.07.004

García-Sánchez, L., Melero, B., Jaime, I., Hänninen, M.L., Rossi, M., Rovira, J., 2017. Campylobacter jejuni survival in a poultry processing plant environment. Food Microbiol. 65, 185–192. https://doi.org/10.1016/j.fm.2017.02.009

García-Sánchez, L., Melero, B., Jaime, I., Rossi, M., Ortega, I., Rovira, J., 2019. Biofilm formation, virulence and antimicrobial resistance of different Campylobacter jejuni isolates from a poultry slaughterhouse. Food Microbiol. 83, 193–199.

https://doi.org/10.1016/j.fm.2019.05.016

Gharib Naseri, K., Rahimi, S., Khaki, P., 2012. Comparison of the effects of probiotic, organic acid and medicinal plant on campylobacter jejuni challenged broiler chickens. J. Agric. Sci. Technol. 14, 1485–1496.

Gong, J., Forster, R.J., Yu, H., Chambers, J.R., Wheatcroft, R., Sabour, P.M., Chen, S., 2002. Molecular analysis of bacterial populations in the ileum of broiler chickens and comparison with bacteria in the cecum. FEMS Microbiol. Ecol. 41, 171–179. https://doi.org/https://doi.org/10.1111/j.1574-6941.2002.tb00978.x

Gong, J., Si, W., Forster, R.J., Huang, R., Yu, H., Yin, Y., Yang, C., Han, Y., 2007. 16S rRNA gene-based analysis of mucosa-associated bacterial community and phylogeny in the chicken gastrointestinal tracts: From crops to ceca. FEMS Microbiol. Ecol. 59, 147–157. https://doi.org/10.1111/j.1574-6941.2006.00193.x

Goode, D., Allen, V.M., Barrow, P.A., 2003. Reduction of experimental Salmonella and Campylobacter contamination of chicken skin by application of lytic bacteriophages. Appl. Environ. Microbiol. 69, 5032–5036. https://doi.org/10.1128/AEM.69.8.5032-5036.2003

Haas, D., Posch, J., Schmidt, S., Wüst, G., Sixl, W., Feierl, G., Marth, E., Reinthaler, F.F., 2005. A case study of airborne culturable microorganisms in a poultry slaughterhouse in Styria, Austria. Aerobiologia (Bologna). 21, 193–201. https://doi.org/10.1007/s10453-005-9003-x

Hald, B., Skovgård, H., Pedersen, K., Bunkenborg, H., 2008. Influxed insects as vectors for Campylobacter jejuni and Campylobacter coli in Danish broiler houses. Poult. Sci. 87, 1428–1434. https://doi.org/10.3382/ps.2007-00301

682 Hald, B., Wedderkopp, A., Madsen, M., 2000. Thermophilic Campylobacter spp. in Danish
683 broiler production: A cross-sectional survey and a retrospective analysis of risk factors for
684 occurrence in broiler flocks. Avian Pathol. 29, 123–131.
685 https://doi.org/10.1080/03079450094153

686 Hammerl, J.A., Jäckel, C., Alter, T., Janzcyk, P., Stingl, K., Knüver, M.T., Hertwig, S., 2014.
687 Reduction of campylobacter jejuni in broiler chicken by successive application of group II
688 and group III phages. PLoS One 9, 1–17. https://doi.org/10.1371/journal.pone.0114785

689 Havelaar, A.H., Mangen, M.J.J., De Koeijer, A.A., Bogaardt, M.J., Evers, E.G., Jacobs-Reitsma,
690 W.F., Van Pelt, W., Wagenaar, J.A., De Wit, G.A., Van Der Zee, H., Nauta, M.J., 2007.
691 Effectiveness and efficiency of controlling Campylobacter on broiler chicken meat. Risk
692 Anal. 27, 831–844. https://doi.org/10.1111/j.1539-6924.2007.00926.x

693 Herman, L., Heyndrickx, M., Grijspeerdt, K., Vandekerchove, D., Rollier, I., De Zutter, L., 2002.
694 Routes for Campylobacter contamination of poultry meat: Epidemiological study study
695 from hatchery to slaughterhouse. Epidemiol. Infect. 131, 1169–1180.
696 https://doi.org/https://doi.org/10.1017/S0950268803001183

697 Hermans, D., Van Deun, K., Messens, W., Martel, A., Van Immerseel, F., Haesebrouck, F.,
698 Rasschaert, G., Heyndrickx, M., Pasmans, F., 2011. Campylobacter control in poultry by
699 current intervention measures ineffective: Urgent need for intensified fundamental research.
700 Vet. Microbiol. 152, 219–228. https://doi.org/10.1016/j.vetmic.2011.03.010

701 Hue, O., Le Bouquin, S., Laisney, M.J., Allain, V., Lalande, F., Petetin, I., Rouxel, S., Quesne,
702 S., Gloaguen, P.Y., Picherot, M., Santolini, J., Salvat, G., Bougeard, S., Chemaly, M., 2010.

703 Prevalence of and risk factors for Campylobacter spp. contamination of broiler chicken
704 carcasses at the slaughterhouse. Food Microbiol. 27, 992–999.
705 https://doi.org/10.1016/j.fm.2010.06.004

706 Humphrey, T., 2006. Are happy chickens safer chickens? Poultry welfare and disease
707 susceptibility. Br. Poult. Sci. 47, 379–391. https://doi.org/10.1080/00071660600829084

708 Ijaz, U.Z., Sivaloganathan, L., McKenna, A., Richmond, A., Kelly, C., Linton, M., Stratakos,
709 A.C., Lavery, U., Elmi, A., Wren, B.W., Dorrell, N., Corcionivoschi, N., Gundogdu, O.,
710 2018. Comprehensive longitudinal microbiome analysis of the chicken cecum reveals a shift
711 from competitive to environmental drivers and a window of opportunity for Campylobacter.
712 Front. Microbiol. 9. https://doi.org/10.3389/fmicb.2018.02452

713 Ingresa-Capaccioni, S., Jiménez-Trigos, E., Marco-Jiménez, F., Catalá, P., Vega, S., Marin, C.,
714 2016. Campylobacter epidemiology from breeders to their progeny in Eastern Spain. Poult.
715 Sci. 95, 676–683. https://doi.org/10.3382/ps/pev338

716 ISO 10272-2:2017, 2017. Microbiology of the food chain — Horizontal method for the detection
717 , and enumeration of Campylobactcer spp.

718 Johannessen, G.S., Garofolo, G., Di Serafino, G., Koláčková, I., Karpíšková, R., Wieczorek, K.,
719 Osek, J., Christensen, J., Torp, M., Hoorfar, J., 2020. Campylobacter in chicken – Critical
720 parameters for international, multicentre evaluation of air sampling and detection methods.
721 Food Microbiol. 90, 103455. https://doi.org/10.1016/j.fm.2020.103455

722 Johnsen, G., Kruse, H., Hofshagen, M., 2007. Genotyping of thermotolerant Campylobacter
723 from poultry slaughterhouse by amplified fragment length polymorphism. J. Appl.

724 Microbiol. 103, 271–279. https://doi.org/10.1111/j.1365-2672.2006.03238.x

725 Johnson, T.J., Shank, J.M., Johnson, J.G., 2017. Current and potential treatments for reducing
726 Campylobacter colonization in animal hosts and disease in humans. Front. Microbiol. 8,
727 2019. https://doi.org/10.3389/fmicb.2017.00487

728 Jore, S., Viljugrein, H., Brun, E., Heier, B.T., Borck, B., Ethelberg, S., Hakkinen, M., Kuusi, M.,
729 Reiersen, J., Hansson, I., Olsson Engvall, E., Løfdahl, M., Wagenaar, J.A., van Pelt, W.,
730 Hofshagen, M., 2010. Trends in Campylobacter incidence in broilers and humans in six
731 European countries, 1997-2007. Prev. Vet. Med. 93, 33–41.
732 https://doi.org/10.1016/j.prevetmed.2009.09.015

733 Josefsen, M.H., Bhunia, A.K., Engvall, E.O., Fachmann, M.S.R., Hoorfar, J., 2015. Monitoring
734 Campylobacter in the poultry production chain - From culture to genes and beyond. J.
735 Microbiol. Methods 112, 118–125. https://doi.org/10.1016/j.mimet.2015.03.007

736 Kalupahana, R.S., Kottawatta, K.S.A., Kanankege, K.S.T., Van Bergen, M.A.P., Abeynayake,
737 P., Wagenaar, J.A., 2013. Colonization of Campylobacter spp. in broiler chickens and
738 laying hens reared in tropical climates with low-biosecurity housing. Appl. Environ.
739 Microbiol. 79, 393–395. https://doi.org/10.1128/AEM.02269-12

740 Kapperud, G., Skjerve, E., Vik, L., Hauge, K., Lysaker, A., Aalmen, I., Ostroff, S.M., Potter, M.,
741 1993. Epidemiological investigation of risk factors for Campylobacter colonization in
742 Norwegian broiler flocks. Epidemiol. Infect. 111, 245–256.
743 https://doi.org/10.1017/S0950268800056958

744 Kers, J.G., Velkers, F.C., Fischer, E.A.J., Hermes, G.D.A., Stegeman, J.A., Smidt, H., 2018.

745 Host and environmental factors affecting the intestinal microbiota in chickens. Front.
746 Microbiol. 9, 1–14. https://doi.org/10.3389/fmicb.2018.00235

747 Khan, J.A., Rathore, R.S., Abulreesh, H.H., Qais, F.A., Ahmad, I., 2018. Prevalence and
748 antibiotic resistance profiles of Campylobacter jejuni isolated from poultry meat and related
749 samples at retail shops in Northern India. Foodborne Pathog. Dis. 15, 218–225.
750 https://doi.org/10.1089/fpd.2017.2344

751 Khan, S., Moore, R.J., Stanley, D., Chousalkar, K.K., 2020. The gut microbiota of laying hens
752 and its manipulation with prebiotics and probiotics to enhance gut health and food safety.
753 Appl. Environ. Microbiol. 86. https://doi.org/10.1128/AEM.00600-20

754 Kittler, S., Fischer, S., Abdulmawjood, A., Glünder, G., Kleina, G., 2013. Effect of
755 bacteriophage application on Campylobacter jejuni loads in commercial broiler flocks.
756 Appl. Environ. Microbiol. 79, 7525–7533. https://doi.org/10.1128/AEM.02703-13

757 Knarreborg, A., Simon, M. A., Engberg, R. M., Jensen, B. B., & Tannock, G.W., 2002. Effects
758 of dietary fat source and subtherapeutic levels of antibiotic on the bacterial community in
759 the ileum of broiler chickens at various ages. Appl. Environ. Microbiol. 68, 1189–1195.
760 https://doi.org/10.1128/AEM.68.12.5918

761 Koolman, L., Whyte, P., Bolton, D.J., 2014. An investigation of broiler caecal Campylobacter
762 counts at first and second thinning. J. Appl. Microbiol. 117, 876–881.
763 https://doi.org/10.1111/jam.12580

764 Korsak, D., Maćkiw, E., Rożynek, E., Żyłowska, M., 2015. Prevalence of Campylobacter spp. in
765 retail chicken, turkey, pork, and beef meat in Poland between 2009 and 2013. J. Food Prot.

78, 1024–1028. https://doi.org/10.4315/0362-028x.jfp-14-353

Kumar, S., Chen, C., Indugu, N., Werlang, G.O., Singh, M., Kim, W.K., Thippareddi, H., 2018. Effect of antibiotic withdrawal in feed on chicken gut microbial dynamics, immunity, growth performance and prevalence of foodborne pathogens. PLoS One 13, 1–23. https://doi.org/10.1371/journal.pone.0192450

Lastovica, A.J., On, S.L.W., Zhang, L., 2014. The family Campylobacteraceae, in: Rosenberg, E., DeLong, E.F., Lory, S., Stackebrandt, E., Thompson, F. (Eds.), The Prokariotes. Springer Berlin Heidelberg, Berlin, Heidelberg, pp. 307–335. https://doi.org/10.1007/978-3-642-39044-9_274

Lin, J., 2009. Novel approaches for Campylobacter control in poultry. Foodborne Pathog. Dis. 6, 755–765. https://doi.org/10.1089/fpd.2008.0247

Linden, C.D., Roth, T.F., 1978. IgG receptors on foetal chick yolk sac. J. Cell Sci. 33, 317–328.

Loc Carrillo, C., Atterbury, R.J., Dillon, E., Scott, A., Connerton, I.F., Connerton, P.L., 2005. Bacteriophage therapy to reduce Campylobacter jejuni colonization of broiler chickens. Appl. Environ. Microbiol. 71, 6554–6563. https://doi.org/10.1128/AEM.71.11.6554

Lu, J., Idris, U., Harmon, B., Hofacre, C., Maurer, J.., Lee, M., 2003. Diversity and succession of the intestinal bacterial community of the maturing. Appl. Environ. Microbiol. 69, 6816–6824. https://doi.org/10.1128/AEM.69.11.6816

Luong, T., Salabarria, A.-C., Roach, D.R., 2020. Phage therapy in the resistance era: Where do we stand and where are we going? Clin. Ther. 42, 1659–1680.

https://doi.org/10.1016/j.clinthera.2020.07.014

Lyngstad, T.M., Jonsson, M.E., Hofshagen, M., Heier, B.T., 2008. Risk factors associated with the presence of Campylobacter species in Norwegian broiler flocks. Poult. Sci. 87, 1987–1994. https://doi.org/10.3382/ps.2008-00132

Marotta, F., Garofolo, G., Di Donato, G., Aprea, G., Platone, I., Cianciavicchia, S., Alessiani, A., Di Giannatale, E., 2015. Population diversity of Campylobacter jejuni in poultry and its dynamic of contamination in chicken meat. Biomed Res. Int. 2015. https://doi.org/10.1155/2015/859845

McKenna, A., Ijaz, U.Z., Kelly, C., Linton, M., Sloan, W.T., Green, B.D., Lavery, U., Dorrell, N., Wren, B.W., Richmond, A., Corcionivoschi, N., Gundogdu, O., 2020. Impact of industrial production system parameters on chicken microbiomes: mechanisms to improve performance and reduce Campylobacter. Microbiome 8, 128. https://doi.org/10.1186/s40168-020-00908-8

Meunier, M., Guyard-Nicodème, M., Dory, D., Chemaly, M., 2016. Control strategies against Campylobacter at the poultry production level: Biosecurity measures, feed additives and vaccination. J. Appl. Microbiol. 120, 1139–1173. https://doi.org/10.1111/jam.12986

Nafarrate, I., Mateo, E., Miranda-Cadena, K., Lasagabaster, A., 2021. Isolation, host specificity and genetic characterization of Campylobacter specific bacteriophages from poultry and swine sources. Food Microbiol. 97. https://doi.org/10.1016/j.fm.2021.103742

Neill, S.D., Campbell, J.N., Greene, J.A., 1984. Campylobacter species in broiler chickens. Avian Pathol. 13, 777–785. https://doi.org/10.1080/03079458408418574

807 Nicholson, F.A., Groves, S.J., Chambers, B.J., 2005. Pathogen survival during livestock manure
808 storage and following land application. Bioresour. Technol. 96, 135–143.
809 https://doi.org/10.1016/j.biortech.2004.02.030

810 Oakley, B.B., Kogut, M.H., 2016. Spatial and temporal changes in the broiler chicken cecal and
811 fecal microbiomes and correlations of bacterial taxa with cytokine gene expression. Front.
812 Vet. Sci. 3, 1–12. https://doi.org/10.3389/fvets.2016.00011

813 Oakley, B.B., Lillehoj, H.S., Kogut, M.H., Kim, W.K., Maurer, J.J., Pedroso, A., Lee, M.D.,
814 Collett, S.R., Johnson, T.J., Cox, N.A., 2014. The chicken gastrointestinal microbiome.
815 FEMS Microbiol. Lett. 360, 100–112. https://doi.org/10.1111/1574-6968.12608

816 Osimani, A., Aquilanti, L., Pasquini, M., Clementi, F., 2017. Prevalence and risk factors for
817 thermotolerant species of Campylobacter in poultry meat at retail in Europe. Poult. Sci. 96,
818 3382–3391. https://doi.org/10.3382/ps/pex143

819 Pedroso, A.A., Batal, A.B., Lee, M.D., 2016. Effect of in ovo administration of an adult-derived
820 microbiota on establishment of the intestinal microbiome in chickens. Am. J. Vet. Res. 77,
821 514–526. https://doi.org/https://doi.org/10.2460/ajvr.77.5.514

822 Perez-Arnedo, I., Gonzalez-Fandos, E., 2019. Prevalence of Campylobacter spp. in poultry in
823 three Spanish farms, a slaughterhouse and a further processing plant. Foods 8, 111.
824 https://doi.org/10.3390/foods8030111

825 Pielsticker, C., Glünder, G., Rautenschlein, S., 2012. Colonization properties of Campylobacter
826 jejuni in chickens. Eur. J. Microbiol. Immunol. 2, 61–65.
827 https://doi.org/10.1556/eujmi.2.2012.1.9

828 Richards, P.J., Connerton, P.L., Connerton, I.F., 2019. Phage biocontrol of campylobacter jejuni
829 in chickens does not produce collateral effects on the gut microbiota. Front. Microbiol. 10,
830 1–10. https://doi.org/10.3389/fmicb.2019.00476

831 Ricke, S.C., 2003. Perspectives in the use of organic acids and short chain fatty acids as
832 microbials. Poult. Sci. 82, 632–639. https://doi.org/https://doi.org/10.1093/ps/82.4.632

833 Ridley, A., Morris, V., Gittins, J., Cawthraw, S., Harris, J., Edge, S., Allen, V., 2011. Potential
834 sources of Campylobacter infection on chicken farms: Contamination and control of broiler-
835 harvesting equipment, vehicles and personnel. J. Appl. Microbiol. 111, 233–244.
836 https://doi.org/10.1111/j.1365-2672.2011.05038.x

837 Ridley, A.M., Morris, V.K., Cawthraw, S.A., Ellis-Iversen, J., Harris, J.A., Kennedy, E.M.,
838 Newell, D.G., Allen, V.M., 2011. Longitudinal molecular epidemiological study of
839 thermophilic campylobacters on one conventional broiler chicken farm. Appl. Environ.
840 Microbiol. 77, 98–107. https://doi.org/10.1128/AEM.01388-10

841 Robyn, J., Rasschaert, G., Hermans, D., Pasmans, F., Heyndrickx, M., 2013. Is allicin able to
842 reduce campylobacter jejuni colonization in broilers when added to drinking water? Poult.
843 Sci. 92, 1408–1418. https://doi.org/10.3382/ps.2012-02863

844 Rossler, E., Signorini, M.L., Romero-Scharpen, A., Soto, L.P., Berisvil, A., Zimmermann, J.A.,
845 Fusari, M.L., Olivero, C., Zbrun, M. V., Frizzo, L.S., 2019. Meta-analysis of the prevalence
846 of thermotolerant Campylobacter in food-producing animals worldwide. Zoonoses Public
847 Health 1–11. https://doi.org/10.1111/zph.12558

848 Sahin, O., Zhang, Q., Meitzler, J.C., Harr, B.S., Morishita, T.Y., Mohan, R., 2001. Prevalence,

849 antigenic specificity, and bactericidal activity of poultry anti-Campylobacter maternal
850 antibodies. Appl. Environ. Microbiol. 67, 3951–3957.
851 https://doi.org/10.1128/AEM.67.9.3951-3957.2001

852 Sakaridis, I., Ellis, R.J., Cawthraw, S.A., van Vliet, A.H., Stekel, D.J., Penell, J., Chambers, M.,
853 La Ragione, R.M., Cook, A.J., 2018. Investigating the association between the caecal
854 microbiomes of broilers and Campylobacter burden. Front. Microbiol. 9, 927.
855 https://doi.org/10.3389/fmicb.2018.00927

856 Schroeder, M.W., Eifert, J.D., Ponder, M.A., Schmale, D.G., 2014. Association of
857 Campylobacter spp. levels between chicken grow-out environmental samples and processed
858 carcasses. Poult. Sci. 93, 734–741. https://doi.org/10.3382/ps.2013-03646

859 Shang, Y., Kumar, S., Oakley, B., Kim, W.K., 2018. Chicken gut microbiota: Importance and
860 detection technology. Front. Vet. Sci. 5, 254. https://doi.org/10.3389/fvets.2018.00254

861 Shange, N., Gouws, P., Hoffman, L.C., 2019. Campylobacter and Arcobacter species in food-
862 producing animals: prevalence at primary production and during slaughter. World J.
863 Microbiol. Biotechnol. 35. https://doi.org/10.1007/s11274-019-2722-x

864 Sibanda, N., McKenna, A., Richmond, A., Ricke, S.C., Callaway, T., Stratakos, A.C.,
865 Gundogdu, O., Corcionivoschi, N., 2018. A review of the effect of management practices
866 on campylobacter prevalence in poultry farms. Front. Microbiol. 9, 1–9.
867 https://doi.org/10.3389/fmicb.2018.02002

868 Sierra-Arguello, Y.M., Perdoncini, G., Rodrigues, L.B., Ruschel dos Santos, L., Apellanis
869 Borges, K., Quedi Furian, T., Pippi Salle, C.T., de Souza Moraes, H.L., Pereira Gomes,

870 M.J., Pinheiro do Nascimento, V., 2021. Identification of pathogenic genes in
871 Campylobacter jejuni isolated from broiler carcasses and broiler slaughterhouses. Sci. Rep.
872 11. https://doi.org/10.1038/s41598-021-84149-1

873 Silva, J., Leite, D., Fernandes, M., Mena, C., Gibbs, P.A., Teixeira, P., 2011. Campylobacter spp.
874 As a foodborne pathogen: A review. Front. Microbiol. 2.
875 https://doi.org/10.3389/fmicb.2011.00200

876 Singh, K.M., Shah, T.M., Reddy, B., Deshpande, S., Rank, D.N., Joshi, C.G., 2014. Taxonomic
877 and gene-centric metagenomics of the fecal microbiome of low and high feed conversion
878 ratio (FCR) broilers. J. Appl. Genet. 55, 145–154. https://doi.org/10.1007/s13353-013-
879 0179-4

880 Siringan, P., Connerton, P.L., Payne, R.J.H., Connerton, I.F., 2011. Bacteriophage-mediated
881 dispersal of Campylobacter jejuni biofilms. Appl. Environ. Microbiol. 77, 3320–3326.
882 https://doi.org/10.1128/AEM.02704-10

883 Slader, J., Domingue, G., Mcalpine, K., Owen, R.J., Bolton, F.J., Humphrey, T.J., 2002. Impact
884 of transport crate reuse and of catching and processing on Campylobacter and Salmonella
885 contamination of brioler chickens. Society 68, 713–719.
886 https://doi.org/10.1128/AEM.68.2.713

887 Sofka, D., Pfeifer, A., Gleiss, B., Paulsen, P., Hilbert, F., 2015. Changes within the intestinal
888 flora of broilers by colonisation with Campylobacter jejuni. Berl. Munch. Tierarztl.
889 Wochenschr. 128, 104–110.

890 Sommer, H. M., Høg, B. B., Larsen, L. S., Sørensen, A. I. V., Williams, N., Merga, J. Y., Osek,

J., 2016. Analysis of farm specific risk factors for Campylobacter colonization of broilers in six European countries. Microb. Risk Anal. 2, 16–26. https://doi.org/https://doi.org/10.1016/j.mran.2016.06.002

Stanley, D., Denman, S.E., Hughes, R.J., Geier, M.S., Crowley, T.M., Chen, H., Haring, V.R., Moore, R.J., 2012. Intestinal microbiota associated with differential feed conversion efficiency in chickens. Appl. Microbiol. Biotechnol. 96, 1361–1369. https://doi.org/10.1007/s00253-011-3847-5

Stern, N.J., Fedorka-Cray, P., Bailey, J.S., Cox, N.A., Craven, S.E., Hiett, K.L., Mead, G.C., 2001. Distribution of Campylobacter spp. in selected U.S. poultry production and processing operations. J. Food Prot. 64, 1705–1710. https://doi.org/10.4315/0362-028x-64.11.1705

Stern, N.J., Svetoch, E.A., Eruslanov, B. V., Perelygin, V. V., Mitsevich, E. V., Mitsevich, I.P., Pokhilenko, V.D., Levchuk, V.P., Svetoch, O.E., Seal, B.S., 2006. Isolation of a Lactobacillus salivarius strain and purification of its bacteriocin, which is inhibitory to Campylobacter jejuni in the chicken gastrointestinal system. Antimicrob. Agents Chemother. 50, 3111–3116. https://doi.org/10.1128/AAC.00259-06

Takeshita, N., Watanabe, T., Ishida-Kuroki, K., Sekizaki, T., 2021. Transition of microbiota in chicken cecal droppings from commercial broiler farms. BMC Vet. Res. 17. https://doi.org/10.1186/s12917-020-02688-7

Tang, Y., Jiang, Q., Tang, H., Wang, Z., Yin, Y., Ren, F., Kong, L., Jiao, X., Huang, J., 2020. Characterization and prevalence of Campylobacter spp. from broiler chicken rearing period

912 to the slaughtering process in eastern China. Front. Vet. Sci. 7.
913 https://doi.org/10.3389/fvets.2020.00227

914 Tangkham, W., Janes, M., Lemieux, F., 2016. Prevalence and distribution of Campylobacter
915 jejuni in small-scale broiler operations. J. Food Prot. 79, 75–81.
916 https://doi.org/10.4315/0362-028x.jfp-15-331

917 Tegtmeyer, N., Sharafutdinov, I., Harrer, A., Soltan Esmaeili, D., Linz, B., Backert, S., 2021.
918 Campylobacter virulence factors and molecular host-pathogen interactions. Curr. Top.
919 Microbiol. Immunol. 431, 169–202. https://doi.org/10.1007/978-3-030-65481-8_7

920 Thibodeau, A., Fravalo, P., Yergeau, E., Arsenault, J., Lahaye, L., Letellier, A., 2015. Chicken
921 caecal microbiome modifications induced by Campylobacter jejuni colonization and by a
922 non-antibiotic feed additive. PLoS One 10. https://doi.org/10.1371/journal.pone.0131978

923 Umar, S., Maiyah, A.T., Mushtaq, A., 2016. Campylobacter infections in poultry: Update on
924 challenges and potential immune interventions. Worlds. Poult. Sci. J. 72, 381–390.
925 https://doi.org/10.1017/S0043933916000167

926 Ushanov, L., Lasareishvili, B., Janashia, I., Zautner, A.E., 2020. Application of Campylobacter
927 jejuni phages: Challenges and perspectives. Animals 10.
928 https://doi.org/10.3390/ani10020279

929 Vandeplas, S., Beckers, Y., Théwis, A., Dubois-Dauphin, R., Thonart, P., Palm, R., 2010.
930 Prevalence and sources of Campylobacter spp. contamination in free-range broiler
931 production in the southern part of Belgium. Biotechnol. Agron. Soc. Environ. 14, 279–288.

Vispo, C., Karasov, W.H., 1997. The interaction of avian gut microbes and their host: An elusive symbiosis. Gastrointest. Microbiol. 116–155. https://doi.org/10.1007/978-1-4615-4111-0_5

Wagenaar, J.A., Mevius, D.J., Havelaar, A.H., 2006. Campylobacter in primary animal production and control strategies to reduce the burden of human campylobacteriosis. Rev. Sci. Tech. 25, 581–94.

Whyte, P., Collins, J.D., McGill, K., Monahan, C., O'Mahony, H., 2001. The effect of transportation stress on excretion rates of campylobacters in market-age broilers. Poult. Sci. 80, 817–820. https://doi.org/10.1093/ps/80.6.817

Williams, A., Oyarzabal, O.A., 2012. Prevalence of Campylobacter spp. in skinless, boneless retail broiler meat from 2005 through 2011 in Alabama, USA. BMC Microbiol. 12, 184. https://doi.org/10.1186/1471-2180-12-184

Willis, W.L., Murray, C., Talbott, C., 2002. Campylobacter isolation trends of cage versus floor broiler chickens: A one-year study. Poult. Sci. 81, 629–631. https://doi.org/10.1093/ps/81.5.629

World Health Organization (WHO), 2015. Who estimates of the global burden of foodborne diseases. Foodborne disase Burd. Epidemiol. refeence Gr. 265.

Wright, R.C.T., Friman, V.-P., Smith, M.C.M., Brockhurst, M.A., 2019. Resistance evolution against phage combinations depends on the timing and order of exposure. MBio 10. https://doi.org/10.1128/mBio.01652-19

Zhang, X., Tang, M., Zhou, Q., Zhang, J., Yang, X., Gao, Y., 2018. Prevalence and

952 characteristics of Campylobacter throughout the slaughter process of different broiler
953 batches. Front. Microbiol. 9, 1–9. https://doi.org/10.3389/fmicb.2018.02092

954 Zhang, X., Yin, T., Du, X., Yang, W., Huang, J., Jiao, X., 2017. Occurrence and genotypes of
955 Campylobacter species in broilers during the rearing period. Avian Pathol. 46, 215–223.
956 https://doi.org/10.1080/03079457.2016.1248374

957 Zhvania, P., Hoyle, N.S., Nadareishvili, L., Nizharadze, D., Kutateladze, M., 2017. Phage
958 therapy in a 16-year-old boy with netherton syndrome. Front. Med. 4.
959 https://doi.org/10.3389/fmed.2017.00094
960

961 **Table legend**

962 **Table 1.** Prevalence of *Campylobacter* spp, *Campylobacter jejuni* and *Campylobacter coli*
963 from different sample types at different sampling points during the broiler primary production
964 using culture-based methods for the detection of *Campylobacter* expresses as percentage or log
965 CFU/g

966 **Table 2.** Prevalence of *Campylobacter* spp, *Campylobacter jejuni* and *Campylobacter coli*
967 from fecal samples and different equipment used to transport broilers during the pre-and post-
968 transportation of broilers to the slaughterhouse using culture-based methods for the detection of
969 *Campylobacter* expresses as percentage

970 **Table 3.** Prevalence of *Campylobacter* spp, *Campylobacter jejuni* and *Campylobacter coli*
971 from different sample types during slaughter, dressing and processing using different types of
972 polymerase chain reaction for the detection of *Campylobacter* expresses as percentage

973

974 **Figure legend**

975 **Figure 1.** Key steps identified and used to assess the prevalence of *Campylobacter* in the
976 broiler production chain. The location of studies investigating the effect of bacteriophage is
977 indicated with a bacteriophage icon. Color in blue is the prevalence of *Campylobacter* spp. and
978 color in yellow is the prevalence of *Campylobacter jejuni*, both expressed as percentage.

979 **Table 1**

| FACTORS | DESCRIPTION | SAMPLE | *CAMPYLOBACTER* | *C. JEJUNI* | *C. COLI* | REFERENCE |
|---|---|---|---|---|---|---|
| **Broiler age** | | | | | | |
| | 14 d | Cloacal | 5.00 % | 100.00 % | | (Ingresa-Capaccioni et al., 2016) |
| | 42 d | Cloacal | 62.00 % | 67.00 % | | (Ingresa-Capaccioni et al., 2016) |
| | 14 | Cloacal | 0.00 % | | | (Perez-Arnedo and Gonzalez-Fandos, 2019) |
| | 42 d | Cloacal | 0.00 – 100.00 % | | | (Perez-Arnedo and Gonzalez-Fandos, 2019) |
| | 31 d | Cloacal | 39.30 % | 46.00 % | 26.00 % | (Tang et al., 2020) |
| | 37 d | Cloacal | 60.00 % | 74.00 % | 38.00 % | (Tang et al., 2020) |
| | 7 d (control housing) | Fecal | 5.30 % | 5.30 % | | (Tangkham et al., 2016) |
| | 42 d (control housing) | Fecal | 26.00 % | 26.00 % | | (Tangkham et al., 2016) |
| | 21 d (control housing) | Fecal | 83.30 % | 18.70 % | | (Tangkham et al., 2016) |
| | 7 d (open housing) | Fecal | 0.00 % | 0.00 % | | (Tangkham et al., 2016) |
| | 21 d (open housing) | Fecal | 93.30 % | 70.70 % | | (Tangkham et al., 2016) |
| | 42 d (open housing) | Fecal | 37.30 % | 00 - 37.30 % | | (Tangkham et al., 2016) |
| **Water** | | | | | | |
| | Environmentally control | | 0.00 - 66.70 % | 0.00 - 16.70 % | | (Tangkham et al., 2016) |
| | Open | | 0.00 - 83.30 % | 0.00 - 83.30 % | | (Tangkham et al., 2016) |
| | Water of 31 d age | | 0.00 % | | | (Tang et al., 2020) |
| | Water of 37 d age | | 0.00 % | | | (Tang et al., 2020) |
| **Feed** | | | | | | (Tangkham et al., 2016) |
| | Environmentally control | | 0.00 - 83.30 % | 0.00 - 33.30 % | | (Tangkham et al., 2016) |
| | Open | | 0.00 - 33.30 % | 0.00 - 33.30 % | | (Tangkham et al., 2016) |

| | | | | | |
|---|---|---|---|---|---|
| | Feed of 31 d age | | 0.00 % | | (Tang et al., 2020) |
| | Feed of 37 d age | | 0.00 % | | (Tang et al., 2020) |
| **Litter** | | | | | |
| | Covering shoe | | 20.00 % | | (Schroeder et al., 2014) |
| **Air** | Gelatin sample | | 15.00 % | | (Schroeder et al., 2014) |
| | Air filter samples | | 0.00 - 10.00 % | | (Johannessen et al., 2020) |
| **Workers** | Workers' boots swabs | | 0.00 - 60.00 % | | (Johannessen et al., 2020) |
| **Infrastructure of the farm** | | | | | |
| | | | 0.00 - 12.50 % | | (Bang et al., 2002) |
| | Floor 31 d age | | 0.00 % | | (Tang et al., 2020) |
| | Floor 37 d age | | 0.00 % | | (Tang et al., 2020) |
| | Bedding 31 d age | | 10.00 % | | (Tang et al., 2020) |
| | Bedding 37 d age | | 10.00 % | 1.00 % | (Tang et al., 2020) |
| | Sole 31 d age | | 4.70 % | 1.00 % | (Tang et al., 2020) |
| | Sole 37 d age | | 10.50 % | 2.00 % | (Tang et al., 2020) |
| | Net 31 d age | | 0.00 % | | (Tang et al., 2020) |
| | Net 37 d age | | 0.00 % | | (Tang et al., 2020) |
| | Stool 31 d age | | 0.00 % | | (Tang et al., 2020) |
| | Stool 37 d age | | 6.70 % | 1.00 % | (Tang et al., 2020) |
| **Management** | | | | | |
| | First thinning | Cecal | 27.00 - 100.00 % | | (Koolman et al., 2014) |
| | Second thinning | Cecal | 90.00 - 100.00 % | | (Koolman et al., 2014) |
| | Drinking water + allicin | Cecal | | 5.38 log CFU/g | (Robyn et al., 2013) |
| | Feed | | | | |

| | | | | |
|---|---|---|---|---|
| | | Cecal | 4.2 - 7.5 log CFU/g | (Gharib Naseri et al., 2012) |
| | | Fecal | 6.3 - 7.2 log CFU/g | (Gharib Naseri et al., 2012) |
| | Drink water + probiotics | | | |
| | | Cecal | 4.1 - 6.6 log CFU/g | (Gharib Naseri et al., 2012) |
| | | Fecal | 5.4 - 6.4 log CFU/g | (Gharib Naseri et al., 2012) |
| | Feed + plant derivate | | | |
| | | Cecal | 4.2 - 6.3 log CFU/g | (Gharib Naseri et al., 2012) |
| | | Fecal | 5.5 - 6.5 log CFU/g | (Gharib Naseri et al., 2012) |
| | Feed + organic acids | | | |
| | | Cecal | 4.0 - 6.2 log CFU/g | (Gharib Naseri et al., 2012) |
| | | Fecal | 4.1 - 5.6 log CFU/g | (Gharib Naseri et al., 2012) |
| | Feed + bacteriocin | Fecal | ND | (Stern et al., 2006) |
| | Feed + bacteriophages | Fecal | 5.00 log CFU/g | (Carvalho et al., 2010) |

980 **Abbreviations:** ***C. jejuni****; Campylobacter jejuni,* ***C. coli;*** *Campylobacter coli,* ***ND;*** *Not determined*

981

982 **Table 2**

| FACTORS | DESCRIPTION | PREVALENCE (%) | | | REFERENCE |
|---|---|---|---|---|---|
| | | *CAMPYLOBACTER* | *C. JEJUNI* | *C. COLI* | |
| **Equipment** | | | | | |
| | Cage | | 36.80 | ND | (Willis et al., 2002) |
| | Floor | | 65.40 | ND | (Willis et al., 2002) |
| | Pre-transportation cage | 6.20 - 30.00 | | ND | (Stern et al., 2001) |
| | Post-transportation cage | 42.50 - 85.00 | | ND | (Stern et al., 2001) |
| **Fecal** | | | | | |
| | Pre-transportation | 57.10 - 80.00 | | ND | (Whyte et al., 2001) |
| | Post-transportation | 60.00 - 80.00 | | ND | (Whyte et al., 2001) |

983 **Abbreviations:** ***C. jejuni;*** *Campylobacter jejuni,* ***C. coli;*** *Campylobacter coli,* ***ND;*** *Not determined*

984

985 **Table 3**

| PRODUCTION STAGE | FACTORS | DESCRIPTION | PREVALENCE (%) | | | | REFERENCE |
|---|---|---|---|---|---|---|---|
| | | | *CAMPYLOBACTER* | *C. JEJUNI* | *C. COLI* | *AMPLIFICATION GENE* | |
| **Slaughter** | Environment | Dirty defeathering swabs | 60.00 | 80.00 | 20.00 | *hipO-F, hipoO-R, hipO-P, ceuE-F, ceuE-R, ceuE-R, ceuE-P*[A] | (García-Sánchez et al., 2017) |
| | | Clean defeathering swabs | 54.50 | 90.00 | 10.00 | *hipO-F, hipoO-R, hipO-P, ceuE-F, ceuE-R, ceuE-R, ceuE-P*[A] | (García-Sánchez et al., 2017) |
| | | Dehairing swabs | 31.80 | 3.00 | 4.00 | 16S rRNA, *mapA, ceuE*[C] | (Tang et al., 2020) |
| | | | | | | | |
| | | Dirty evisceration swabs | 78.00 | 100.00 | 0.00 | *hipO-F, hipoO-R, hipO-P, ceuE-F, ceuE-R, ceuE-R, ceuE-P*[A] | (García-Sánchez et al., 2017) |
| | | Clean evisceration swabs | 56.40 | 100.00 | 0.00 | *hipO-F, hipoO-R, hipO-P, ceuE-F, ceuE-R, ceuE-R, ceuE-P*[A] | (García-Sánchez et al., 2017) |
| | | Evisceration | 31.80 | 1.00 | 7.00 | 16S rRNA, *mapA, ceuE*[C] | (Tang et al., 2020) |
| | | | | | | | |
| | | Dirty sink swabs | 100.00 | 100.00 | 0.00 | *hipO-F, hipoO-R, hipO-P, ceuE-F, ceuE-R, ceuE-R, ceuE-P*[A] | (García-Sánchez et al., 2017) |
| | | Clean sink swabs | 20.00 | 50.00 | 50.00 | *hipO-F, hipoO-R, hipO-P, ceuE-F, ceuE-R, ceuE-R, ceuE-P*[A] | (García-Sánchez et al., 2017) |
| | | | | | | | |
| | | Dirty floor swabs | 22.70 | 100.00 | 0.00 | *hipO-F, hipoO-R, hipO-P, ceuE-F, ceuE-R, ceuE-R, ceuE-P*[A] | (García-Sánchez et al., 2017) |
| | | Clean floor swabs | 30.00 | 83..30 | 16.70 | *hipO-F, hipoO-R, hipO-P, ceuE-F, ceuE-R, ceuE-R, ceuE-P*[A] | (García-Sánchez et al., 2017) |
| | | | | | | | |
| | | Operation table swabs | | 10.00 - 80.00 | 0.00 - 100.00 | 16S rRNA, *mapA, ceuE*[C] | (Zhang et al., 2018) |
| | | Partition | 9.10 | 1.00 | 1.00 | 16S rRNA, *mapA, ceuE*[C] | (Tang et al., 2020) |
| | | Workers' gloves swabs | | 20.00 - 60.00 | 40.00 - 80.00 | 16S rRNA, *mapA, ceuE*[C] | (Zhang et al., 2018) |

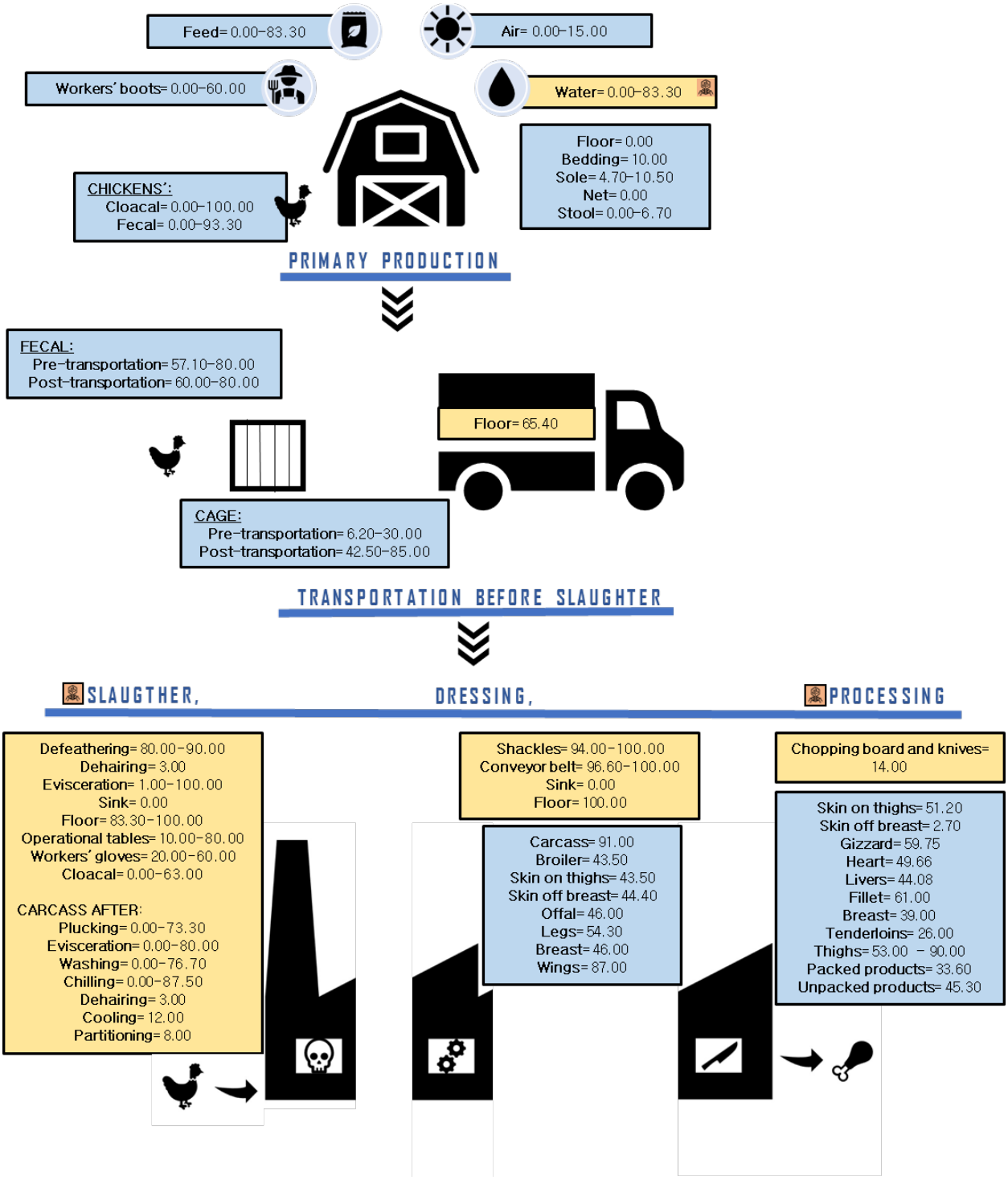
| | | | | | | | |
|---|---|---|---|---|---|---|---|
| | | Cooling | 36.40 | 5.00 | 5.00 | 16S rRNA, *mapA*, *ceuE*[C] | (Tang et al., 2020) |
| | Broiler slaughterhouse | Cloacal swabs | | 0.00 - 63.00 | 0.00 - 13.60 | 16S rRNA, *mapA*, *ceuE*[C] | (Zhang et al., 2018) |
| | | Carcass after plucking | | 0.00 - 73.30 | 0.00 - 85.70 | 16S rRNA, *mapA*, *ceuE*[C] | (Zhang et al., 2018) |
| | | Carcass after evisceration | | 0.00 - 80.00 | 0.00 - 80.00 | 16S rRNA, *mapA*, *ceuE*[C] | (Zhang et al., 2018) |
| | | Carcass after washing | | 0.00 - 76.70 | 0.00 - 95.20 | 16S rRNA, *mapA*, *ceuE*[C] | (Zhang et al., 2018) |
| | | Carcass after chilling | | 0.00 - 87.50 | 0.00 - 100.00 | 16S rRNA, *mapA*, *ceuE*[C] | (Zhang et al., 2018) |
| | | Carcass entrance (41 - 44 days age) | 41.40 | 38.00 | 22.00 | 16S rRNA, *mapA*, *ceuE*[C] | (Tang et al., 2020) |
| | | Carcass after dehairing | 12.90 | 3.00 | 23.00 | 16S rRNA, *mapA*, *ceuE*[C] | (Tang et al., 2020) |
| | | Carcass after evisceration | 53.40 | 37.00 | 69.00 | 16S rRNA, *mapA*, *ceuE*[C] | (Tang et al., 2020) |
| | | Carcass after cooling | 14.80 | 12.00 | 16.00 | 16S rRNA, *mapA*, *ceuE*[C] | (Tang et al., 2020) |
| | | Carcass after partition | 13.60 | 8.00 | 18.00 | 16S rRNA, *mapA*, *ceuE*[C] | (Tang et al., 2020) |
| **Dressing** | Environment | Dirty shackles swabs | 41.80 | 100.00 | 0.00 | *hipO-F, hipoO-R, hipO-P, ceuE-F, ceuE-R, ceuE-R, ceuE-P*[A] | (García-Sánchez et al., 2017) |
| | | Clean shackles swabs | 38.00 | 94.70 | 5.30 | *hipO-F, hipoO-R, hipO-P, ceuE-F, ceuE-R, ceuE-R, ceuE-P*[A] | (García-Sánchez et al., 2017) |
| | | Dirty conveyor belt swabs | 87.90 | 96.60 | 3.40 | *hipO-F, hipoO-R, hipO-P, ceuE-F, ceuE-R, ceuE-R, ceuE-P*[A] | (García-Sánchez et al., 2017) |
| | | Clean conveyor belt swabs | 3.30 | 100.00 | 0.00 | *hipO-F, hipoO-R, hipO-P, ceuE-F, ceuE-R, ceuE-R, ceuE-P*[A] | (García-Sánchez et al., 2017) |
| | | Dirty sink swabs | 0.00 | 0.00 | 0.00 | *hipO-F, hipoO-R, hipO-P, ceuE-F, ceuE-R, ceuE-R, ceuE-P*[A] | (García-Sánchez et al., 2017) |
| | | Clean sink swabs | 0.00 | 0.00 | 0.00 | *hipO-F, hipoO-R, hipO-P, ceuE-F, ceuE-R, ceuE-R, ceuE-P*[A] | (García-Sánchez et al., 2017) |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| | | Dirty floor swabs | 9.10 | 100.00 | 0.00 | *hipO-F, hipoO-R, hipO-P, ceuE-F, ceuE-R, ceuE-R, ceuE-P*[A] | (García-Sánchez et al., 2017) |
| | | Clean floor swabs | 10.00 | 100.00 | 0.00 | *hipO-F, hipoO-R, hipO-P, ceuE-F, ceuE-R, ceuE-R, ceuE-P*[A] | (García-Sánchez et al., 2017) |
| | Broiler | Carcass processing plant | 91.00 | | | Not mentioned[B] | (Perez-Arnedo and Gonzalez-Fandos, 2019) |
| | | Skin on thighs | 43.50 | | | *glyA, hipO*[C] | (Casagrande Proietti et al., 2018) |
| | | Skin off breast | 44.40 | | | *glyA, hipO*[C] | (Casagrande Proietti et al., 2018) |
| | | Legs | 54.30 | | | Not mentioned[B] | (Perez-Arnedo and Gonzalez-Fandos, 2019) |
| | | Breast | 46.00 | | | Not mentioned[B] | (Perez-Arnedo and Gonzalez-Fandos, 2019) |
| | | Wings | 87.00 | | | Not mentioned[B] | (Perez-Arnedo and Gonzalez-Fandos, 2019) |
| **Processing** | Environment | Chopping board and knives swabs | | 14.00 | | *hipO*[B] | (Khan et al., 2018) |
| | Partition | | 13.60 | 8.00 | 18.00 | 16S rRNA, *mapA, ceuE*[C] | (Tang et al., 2020) |
| | Broiler parts | Skin on thighs | 51.20 | | | *glyA, hipO*[C] | (Casagrande Proietti et al., 2018) |
| | | Skin off breast | 2.70 | | | *glyA, hipO*[C] | (Casagrande Proietti et al., 2018) |
| | | Unpacked thighs | 51.60 | | | 23S rRNA, *glyA, hipO, sapB2*[C] | (García-Sánchez et al., 2018) |
| | | Unpacked breast | 51.60 | | | 23S rRNA, *glyA, hipO, sapB2*[C] | (García-Sánchez et al., 2018) |
| | | Unpacked minced | 21.90 | | | 23S rRNA, *glyA, hipO, sapB2*[C] | (García-Sánchez et al., 2018) |
| | | Unpacked marinated | 56.30 | | | 23S rRNA, *glyA, hipO, sapB2*[C] | (García-Sánchez et al., 2018) |
| | | Mean unpacked | 45.30 | | | 23S rRNA, *glyA, hipO, sapB2*[C] | (García-Sánchez et al., 2018) |
| | | Packed thighs | 56.30 | | | 23S rRNA, *glyA, hipO, sapB2*[C] | (García-Sánchez et al., 2018) |
| | | Packed breast | 45.30 | | | 23S rRNA, *glyA, hipO, sapB2*[C] | (García-Sánchez et al., 2018) |

| | | | | | | |
|---|---|---|---|---|---|---|
| | Packed minced | 14.00 | | | 23S rRNA, *glyA, hipO, sapB2*[C] | (García-Sánchez et al., 2018) |
| | Packed marinated | 18.70 | | | 23S rRNA, *glyA, hipO, sapB2*[C] | (García-Sánchez et al., 2018) |
| | Mean packed | 33.60 | | | 23S rRNA, *glyA, hipO, sapB2*[C] | (García-Sánchez et al., 2018) |
| | Raw broiler meat | | 36.00 | | *hipO*[B] | (Khan et al., 2018) |
| | Broiler intestine | | 24.00 | | *hipO*[B] | (Khan et al., 2018) |
| | Feathers | | 8.00 | | *hipO*[B] | (Khan et al., 2018) |
| | Gizzard | 59.75 | | | 23S rRNA, *mapA, ceuE, hipO*[B] | (Korsak et al., 2015) |
| | Heart | 49.66 | | | 23S rRNA, *mapA, ceuE, hipO*[B] | (Korsak et al., 2015) |
| | Livers | 44.08 | | | 23S rRNA, *mapA, ceuE, hipO*[B] | (Korsak et al., 2015) |
| | Fillet | 61.00 | | | 23S rRNA, *mapA, ceuE, hipO*[B] | (Korsak et al., 2015) |
| | Breast | 39.00 | | | *glyA, hipO, ask*[C] | (Williams and Oyarzabal, 2012) |
| | Tenderloins | 26.00 | | | *glyA, hipO, ask*[C] | (Williams and Oyarzabal, 2012) |
| | Thighs | 53.00 - 90.00 | 94.50 | 5.50 | *glyA, hipO, ask*[AC] | (García-Sánchez et al., 2017; Williams and Oyarzabal, 2012) |

986 **Abbreviations: *C. jejuni;*** *Campylobacter jejuni,* ***C. coli;*** *Campylobacter coli,* [A] Real-Time Polymerase Chain Reaction (PCR), [B]PCR, [C]Multiplex PCR

987 **Figure 1**

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