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Comprehensive Review of *Campylobacter* and Poultry Processing

K.M. Keener, M.P. Bashor, P.A. Curtis, B.W. Sheldon, and S. Kathariou

ABSTRACT: *Campylobacter* has been recognized as a leading bacterial cause of human gastroenteritis in the United States, with 40000 documented cases annually. Epidemiological data suggest that contaminated products of animal origin, especially poultry, contribute significantly to campylobacteriosis. Thus, reduction of contamination of raw poultry would have a large impact in reducing incidence of illness. Contamination occurs both on the farm and in poultry slaughter plants. Routine procedures on the farm such as feed withdrawal, poultry handling, and transportation practices have a documented effect on *Campylobacter* levels at the processing plant. At the plant, defeathering, evisceration, and carcass chillers have been documented to cross-contaminate poultry carcasses. Carcass washings and the application of processing aids have been shown to reduce populations of *Campylobacter* in the carcasses by $\log_{10}$ 0.5 to $\log_{10}$ 1.5; however, populations of *Campylobacter* have been shown to enter a poultry processing plant at levels between $\log_{10}$ 5 colony-forming units (CFU)/mL and $\log_{10}$ 8 CFU/mL of carcass rinse. The purpose of this article is to review *Campylobacter*, the infection that it causes, its association with poultry, contamination sources during processing, and intervention methods.

Keywords: *Campylobacter*, carcass washers, antimicrobial, poultry processing, food safety

Bacteriology and ecology

Campylobacter was first described in 1880 by Theodore Escherich (Friedman and others 2000). The name *Campylobacter* is derived from the Greek word "kampylos," which means curved. *Campylobacter* are Gram negative, slender, spiral curved rods having dimensions of 0.2 μm to 0.8 μm wide and 0.5 μm to 5 μm long. Extremely rapid, darting, reciprocating motility can be seen with a phase contrast microscope, with comma-shaped, S, or gull wing–shaped cells.

As *Campylobacter* cells begin to age, they become coccoid in shape (Moran and Upton 1987). Several investigations have shown an association between the transition from the spiral to coccoid morphology with a nonculturable state (Moran and Upton 1986; Rollins and Colwell 1986; Jones and others 1991; Stern and others 1994). Recent studies, however, suggest no correlation between culturability and cell morphology (Medema and others 1992; Hazeleger and others 1995; Lazaro and others 1999). There are mixed reports on the existence and characteristics of the viable but not culturable (VBNC) state of *Campylobacter jejuni* (Rollins and Colwell 1986; Jones and others 1991; Tholozan and others 1999). The mechanism of survival in the putative VBNC state remains unclear.

MS 20030509 Submitted 9/5/03, Accepted 1/20/04, Received 1/20/04. Authors Keener, Bashor, and Kathariou are with Dept. of Food Science, Campus Box 7624, North Carolina State Univ., Raleigh, NC 27695-7624. Author Curtis is with Poultry Product Safety & Quality Program, Auburn Univ., Auburn, Ala. Author Sheldon is with Dept. of Poultry Science, North Carolina State Univ., Raleigh, N.C. Direct inquiries to author Keener (E-mail: kevin_keener@ncsu.edu).

Campylobacter is a fastidious organism that is capable of surviving in a wide range of environments. It has been isolated from rivers, estuarine, and coastal waters, at populations ranging from 10 to 230 colony-forming units (CFU)/100 mL (Bolton and others 1982, 1987). *Campylobacter* is a commensal organism routinely found in cattle, sheep, swine, and avian species. The avian species are the most common host for *Campylobacter*, probably because of their higher body temperature (Skirrow 1977). A study by Bolton and others (1982, 1987) investigated the effect of environmental temperatures over different seasons and nutrients on the survival of *C. jejuni*. They found peak isolation during the late fall and winter months. Willis and Murray (1997) found *Campylobacter* to be at their highest populations on poultry during the warmer months (May through October). During these months, 87% to 97% of the samples tested were positive for *C. jejuni*. They also reported substantial variability in the intestinal colonization of *C. jejuni* across different broiler flocks at different ages in the production cycle.

Campylobacter jejuni and *Campylobacter coli* account for the majority of human infections (Friedman and others 2000) and are commonly referred to as "thermophilic" campylobacters, being able to grow at 37 °C to 42 °C with an optimum growing temperature of 42 °C but incapable of growth below 30 °C. However, a study by De Cesare and others (2002) found that *C. jejuni* survived in excess of 4 h at 27 °C and 60% to 62% relative humidity on some common clean or soiled food contact surfaces.

Campylobacter has a D-value of less than 1 min at 60 °C and is easily inactivated by heat. Freeze-thawing also reduces the population of *Campylobacter* (Stern and Kazmi 1989). *Campylobacter*

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is inactivated by frozen storage at –15 °C in as few as 3 d (Stern and Kotula 1982); however, freezing does not eliminate the pathogen from contaminated foods (Lee and others 1998). Hazeleger and others (1995) discovered that aging *C. jejuni* cells survived the longest at 4 °C. *Campylobacter* will not survive below a pH of 4.9. It is capable of growing in the pH range of 4.9 to 9.0, and grows optimally at pH 6.5 to 7.5.

Campylobacter jejuni is unusually sensitive to oxygen and dehydration. Enzymes present in *C. jejuni* such as superoxide dismutase (SOD), catalase, peroxidase, glutathione synthetase, and glutathione reductase are believed to play a vital role in providing protection against oxygen toxicity (Pesci and others 1994; Purdy and Park 1994). *Campylobacter* requires a special atmosphere, which usually consists of 5% oxygen, 10% carbon dioxide, and 85% nitrogen for growth in or on laboratory media (Stern and Kazmi 1989).

Doyle and Roman (1982b) examined the sensitivity of *C. jejuni* to drying. They demonstrated that several factors influenced the rate of inactivation of *Campylobacter* dried on a glass surface, including bacterial strain, temperature, humidity, and the suspension medium. In all instances, greater survival occurred when organisms were dried in Brucella broth rather than in skim milk. The results of Doyle and Roman (1982a) suggest that *C. jejuni* is quite sensitive to drying and storage at room temperature, but at refrigeration temperatures and appropriate humidity, large numbers may survive drying and remain viable for several weeks. Other studies have also found *C. jejuni* to be quite sensitive to drying at room temperature (Luechtefeld and others 1981).

Campylobacter is oxidase- and catalase-positive and contains a single polar unsheathed flagellum at one or both ends. *C. jejuni* hydrolyzes hippurate, indoxyl, and acetate and reduces nitrate, but is unable to oxidize or ferment carbohydrates. Most strains are resistant to cephalothin. Many are also resistant to fluoroquinolones, a category of antibiotics used to treat animal and human illness (Koenraad and others 1995).

Sources and infection

Campylobacter has long been recognized as a cause of diarrhea in cattle and of septic abortion in both cattle and sheep. Only in the last 25 y has *Campylobacter* been recognized as an important cause of human illness (Friedman and others 2000). The Centers for Disease Control and Prevention reported that *Campylobacter* is a major cause of bacterial diarrheal illness in the United States, with 40000 cases documented annually (CDC 2003). Disease control studies have demonstrated that 50% to 70% of human *Campylobacter* illness is attributed to consuming poultry and poultry products, thus the value of reducing levels associated with raw poultry has drawn considerable attention (Tauxe 1992; Allos 2001). Children less than 1 y of age and young adults aged 15 to 25 y are more susceptible to developing this disease, and individuals with immunosuppression can develop prolonged or unusually severe cases of illness (Friedman and others 2000). Deaths attributed to *Campylobacter* infection in the United States are estimated at 680 to 730/y (Saleha and others 1998).

Doses as low as 500 organisms have been reported to cause illness (Robinson 1981; Black and others 1988; Friedman and others 2000). There are several species of *Campylobacter* (*C. jejuni*, *C. coli*, *C. lari*, and *C. uppsaliensis*) capable of causing human illness. However, *C. jejuni* is implicated in about 85% of the cases of human campylobacteriosis, with the remaining cases being primarily caused by *C. coli* (Fricker and Park 1989; Friedman and others 2000).

The most common clinical symptoms of campylobacteriosis are fever, abdominal pain, and diarrhea that occur within 2 to 5 d of ingestion of food or water contaminated with *C. jejuni* (Robinson 1981; Black and others 1988). Symptoms are usually self-limiting and are resolved within a period of 3 to 10 d, and most cases do not require the use of antibiotics. When antibiotics are necessary, erythromycin and fluoroquinolones are usually prescribed.

In about 1 of 1000 cases, the infection is followed 2 to 3 wk later with Guillain-Barre Syndrome (GBS), a debilitating inflammatory polyneuritis characterized by fever, pain, and weakness that progresses to paralysis. Other possible autoimmune diseases from *Campylobacter* infections include Miller Fisher syndrome (MFS) and Reiter's syndrome or reactive arthritis (Kuroki and others 1993; Nachamkin and others 1998).

Several studies have suggested that the consumption of undercooked poultry and/or the handling of raw poultry are risk factors for human *Campylobacter* infection and illness (Skirrow 1982; Hopkins and others 1984; Oosterom and others 1984; Tauxe and others 1985; Harris and others 1986; Kapperud and others 1992; Blaser 1997; Altekruse and others 1999). A large number of serotypes of *C. jejuni* isolated from chicken carcasses are frequently linked to human cases of campylobacterosis, thus confirming that poultry is an important contributor in the epidemiology of human campylobacterosis (Stern and Kazmi 1989).

Most *Campylobacter* infections are sporadic, that is, they involve individual cases. Outbreaks of *Campylobacter* infections have been traced to raw milk, contaminated water, and contact with pets and farm animals (Kapperud and others 1992; Altekruse and others 1999). *Campylobacter jejuni* may be present in milk from fecal contamination during milking or an udder infection (Doyle and Roman 1982a; Hutchinson and others 1985; Orr and others 1995). Raw milk has been identified as a vehicle in *Campylobacter* human gastroenteritis (Blaser and others 1979; Robinson and others 1979; Porter and Reid 1980; Potter and others 1983; Korlath and others 1985). Vogt and others (1982) and Sacks and others (1986) reported contaminated water as a source of *Campylobacter* outbreaks. Of 8 reported waterborne outbreaks, 4 involved surface water, 2 unchlorinated deep-well water, and 2 chlorinated deep-well systems (McNeil and others 1981; Mentzing 1981; Vogt and others 1982; Palmer and others 1983; Taylor and others 1983). Investigation of these waterborne outbreaks either failed to recover *Campylobacter* or recovered strains that were not the same serovar as those isolated from clinical cases in these outbreaks (Sacks and others 1986). This failure to recover the same serovars from environmental and clinical samples could be due to the lack of advanced technology for identifying strains at that time.

On July 25, 1996, the U.S. Dept. of Agriculture Food and Safety Inspection Service (USDA FSIS) attempted to improve food safety by implementing the Pathogen Reduction; Hazard Analysis and Critical Control Point (HACCP) Systems final rule (USDA 1996a). In this rule, FSIS stated its goal to reduce the risk of foodborne illness associated with the consumption of meat and poultry products to the maximum extent possible and ensure that appropriate and feasible measures are taken at each step in the food production process where hazards can enter and where procedures and technologies exist or can be developed to prevent the hazard or reduce the likelihood it will occur.

Pathogen-specific performance standards for raw products are an essential component of the FSIS food safety strategy because they provide a direct measure of progress in controlling and reducing pathogens. There is some indication that FSIS may implement a *Campylobacter* performance standard in the near future; however, there are less data available on *Campylobacter* levels on raw poultry carcasses during processing than *Salmonella*.

Poultry and poultry products often have been implicated in the transmission of *Campylobacter*. Case-control studies of foodborne infection rates have estimated that 50% to 70% of *Campylobacter* illness is due to poultry and poultry products, thus re-

ducing levels of *Campylobacter* contamination associated with raw poultry may be warranted (Tauxe 1992; Allos 2001). Many factors contribute to *Campylobacter* contamination of broiler carcasses, and some of these factors may be difficult to control to meet performance standards. Because of the strict growth environment requirements for *Campylobacter,* many would believe that it would be easy to remove the organism from the processing plant. This does not appear to be the case. Most likely the high levels of contamination and their populations (log_{10} 5 to log_{10} 8 CFU/mL of carcass rinse) constantly reintroduce the organism (Bashor and others 2004).

Potential for cross-contamination of *Campylobacter* is very high inside the poultry processing plant. Since *Campylobacter* enters the plant at such high levels, it can easily be spread. Poultry that enters the processing plant have *Campylobacter* populations ranging from log_{10} 5 to log_{10} 8 CFU/g of feces, and the bacteria are found in the crop as well as fecal material (Byrd and others 1998a).

The problem of *Campylobacter* contamination of food must be addressed. To date, no practical or effective control measures have been available (Newell and Wagenaar 2000). At the consumer level, accidental ingestion of 1 drop of raw chicken juice can easily constitute an infectious dose, which is as little as 500 organisms (Newell and Wagenaar 2000; Friedman and others 2000). Infections can occur during the improper handling of raw chicken carcasses, by eating insufficiently cooked chicken, and via cross-contamination of other foods by contact with knives or cutting boards used to prepare raw chicken. Although some reduction in cross-contamination of poultry carcasses can be achieved by improved sanitation during processing, the elimination of *Campylobacter* populations from birds before processing is desirable but may not be practical or feasible.

Contamination on the farm

Campylobacter contamination of live birds can be traced back to the farm (Byrd and others 1998b). The available research on bacterial populations in poultry production is mainly from studies on chickens. In a typical broiler processing operation, freshly laid fertile eggs are collected and incubated at a hatchery. After they hatch, the chicks are delivered to farms where they are reared until they are ready for slaughter, and then transported to a processing plant.

Various on-farm strategies have been advanced to reduce the incidence rates of poultry contamination: introduction of competing microbial populations into newly hatched chicks, chlorination of poultry drinking water, vaccination, or selective breeding of poultry for resistance to pathogens. Sound management practices incorporating good husbandry and hygiene practices also play a part in limiting the occurrence of *C. jejuni* in poultry flocks (Saleha and others 1998). Farms that use these practices tend to have lower rates of intestinal colonization with *Campylobacter* spp. (Sjogren and Kaijser 1989; Humphrey and others 1993; Kazwala and others 1993).

Flock colonization

Campylobacter is considered to be a commensal organism in many avian species, including those grown commercially. In most flocks, colonization is not detectable until at least 10 d and continues for many weeks (Newell and Wagenaar 2000). Spread of C*ampylobacters* among hatch mates is rapid if infected birds are introduced into the population. Under laboratory conditions, 3 d of contact with artificially inoculated seeder birds is sufficient for the majority of the brood to be colonized (Shanker and others 1990). Young (1- to 2-wk-old) chicks are highly susceptible to *Campylobacter* colonization (Stern 1992). Chickens are coprophagic, which facilitates the fecal–oral spread of C*ampylobacters;* however, the rapidity of the shift from uncolonized to 100% colonized suggests that *Campylobacters* are also spread from chick to chick via their communal source of drinking water (Montrose and others 1985).

When a flock of broiler chickens becomes positive for *Campylobacter*, the prevalence of infection among birds is high, often reaching 100% of birds tested (Pokamunski and others 1986; Gregory and others 1997). As a commensal organism in poultry, *Campylobacter* colonizes the intestinal mucus layer in the crypts of the intestinal epithelium (Beery and others 1988). There are 2 modes of transmission of *Campylobacter* in poultry: horizontal and vertical. Both have been shown to occur.

Horizontal transmission is believed to be mainly through contaminated water, litter, insects, wild birds, rodents, fecal contact, and by farm personnel via their boots (Aarts and others 1995; Evans and Sayers 2000; Line 2001). Feed has not been implicated in the spread of *Campylobacter* because it is too dry to favor survival.

Chickens can harbor very high levels of *Campylobacter* in the gut, up to 9.0 log_{10} CFU/g of cecal content, without symptoms, and the microorganism can be transmitted among birds within a flock (Berndtson and others 1992; Evans 1997; Altekruse and others 1999). Once 1 bird in a flock is colonized, the infection spreads very quickly (Beery and others 1988). Gregory and others (1997) found almost complete colonization within a flock by the end of the grow-out period (49 d), and 50% to 100% of birds in a flock were colonized in other studies. In 1995, a survey of feces from 19 broiler flocks found 96.4% were positive with *Campylobacter* with an average population of log_{10} 4.6 CFU/g. A follow-up study reported in 2003 (Stern and Robach 2003) found that 94% of feces tested positive for *Campylobacter* with an average population of log_{10} 5.17 CFU/g.

A Dutch study revealed that 67% (29/43) of breeder flocks were colonized with *Campylobacter* (Jacobs-Reitsma 1995). The level of colonization is lower in Norway and Sweden where 6% (10/176) and 14% (522/3727) of the broiler flocks were shown to be colonized with *Campylobacter* (Kapperud and others 1993).

Wallace and others (1998) conducted a comprehensive study on the colonization of turkeys by *Campylobacter*. They found a 100% carriage rate by day 21 in all samples. The results indicated that peaks in *Campylobacter* populations isolated from fecal samples correlated with an increase in the number of birds with diarrheal symptoms. The maximum number of *Campylobacter* excreted was 7.78 log_{10}. *Campylobacter jejuni* was the only species isolated, but comprised several different biotypes. From turkeys that were slaughtered and stored at 4 °C for 7 d, these authors recovered 6.15 log_{10} and 6.98 log_{10} CFU/mL *Campylobacter* from the cecum and feces, respectively. This is comparable to what was found in chickens; however, in an earlier study, Rosef and others (1984) showed that the carriage rate of *Campylobacter* on turkey carcasses was greater than that of either hens or broiler chickens.

Other studies have shown vertical transmission as a means of contamination of a breeder flock (Van de Giessen and others 1992). *Campylobacter jejuni* isolates from a parent flock were found to be the same clonal origin as those from the offspring in a broiler flock. Egg transmission of *Campylobacter* from the breeder flock has not been recognized as a source of entry because of the inability to culture *Campylobacter* from the hatchery samples or from newly hatched chicks (Acuff and others 1982; Doyle 1984; Neill and others 1984; Jones and others 1991). Clark and Bueschkens (1985) inoculated fertile chicken eggs with *C. jejuni* and found that 11% of the resulting chicks at hatch had the inoculated *Campylobacter* in their intestinal tract. Other studies have demonstrated that chickens raised in a laboratory environment without exposure to any farm environment continued to become colo-

nized by *C. jejuni* (Lindblom and others 1986). Chuma and others (1994) determined the carrier rate of *C. jejuni* in the cecal content of newly hatched chicks to be as high as 35%. These data suggest that the chicks were colonized before delivery to the farm. Pearson and others (1996) found no difference between the types of *Campylobacter* isolated in the hatcheries and the types of *Campylobacter* isolated in the subsequent broiler chickens, suggesting that the *Campylobacter* contamination may have occurred by way of vertical transmission.

Feed withdrawal

Byrd and others (1998b) studied the effect of feed withdrawal on *Campylobacter* in the crops of market age broiler chickens. The purpose of feed withdrawal is to allow the clearance of the gastrointestinal tract and thus reduce the potential fecal contamination of poultry carcasses during slaughter. Feed withdrawal caused a significant increase in *Campylobacter* positive crop samples taken from 7 of 9 houses sampled. They found 90 of 360 birds tested before feed withdrawal were positive for *Campylobacter,* whereas 254 of 359 birds tested after feed withdrawal were positive for *Campylobacter*. Feed withdrawal did not significantly alter the *Campylobacter* isolation frequency from ceca. During feed withdrawal, the pH of the crop decreases, affecting the microflora present (Hinton and others 2000). This has been suggested as an important factor in the increased prevalence of *Campylobacter*-contaminated crops. Byrd and others (1998a) reported that the incidence of *Campylobacter* contamination in crop contents may exceed that of cecal contents by as much as 37-fold in some broiler flocks, and may represent a critical preprocessing control point in reducing *Campylobacter* entry into the processing plant. A recent study by Northcutt and others (2002) found that broiler age also plays a role in *Campylobacter* contamination, with 42-d-old birds being 100% positive with an average population of $\log_{10}$ 2.7 CFU/bird compared with 56-d-old birds being 90% positive with an average population of $\log_{10}$ 2.0 CFU/bird.

Transportation

Commercially grown poultry flocks are collected on the farm, placed into crates, transported to the processing plant, and processed on the same day. *Campylobacter* populations have been shown to increase during transport and holding before slaughter (Stern and others 1995a). It is known that stress can cause a disturbance of intestinal functions and may lower the resistance of the live animal and increase spreading of intestinal bacteria. Ceca, blind pouches between the ileum and the colon of the broiler intestinal tract, can harbor large numbers of *Campylobacter* (Duke 1986). Moran and Bilgili (1990) found that pathogens, including *Campylobacter*, consumed orally by the bird before or during crating and transportation may colonize the ceca where they may be retained throughout processing. Line and others (1997) studied ways to reduce *Salmonella* and *Campylobacter* populations associated with broiler chickens subjected to transport stress using a yeast culture. They found that feeding *Saccharomyces boulardii*, a nonpathogenic yeast, could reduce the frequency of *Salmonella* colonization to lower than pre-stress levels. The frequency of *Campylobacter* isolation from the ceca was not affected by treatment.

A survey by Jacobs-Reitsma and others (1994) found *Salmonella* in 27% of 181 broiler flocks surveyed and *Campylobacter* in 82% of 187 flocks. Potential sources of *Campylobacter* contamination on poultry carcasses included fecal contamination of feathers and skin during transport to the slaughter facility, leakage of fecal content from the cloaca, intestinal breakage, and contact with contaminated equipment, water, or other carcasses (Jacobs-Reitsma 2000). Feathers may carry 8 $\log_{10}$ total bacteria/g (Barnes 1975) and skin may carry 6 $\log_{10}$bacteria/cm^2 (Wilkerson and others 1961). It was shown that poultry carcasses can become contaminated with *Campylobacter* from their intestinal contents during the slaughter process (Wempe and others 1983; Genigeorgis and others 1986). *Campylobacter* populations on the feathers of cooped and transported birds are 10-fold greater than those remaining on the farm (Stern and others 1995b). A recent study (Stern and others 2001) also found that many coops are not properly cleaned between flocks, which may contribute to increased contamination levels observed at the plant.

Contamination in the processing plant

At the processing plant, birds are unloaded, shackled, killed, scalded, defeathered, eviscerated, washed, cooled, and packaged (Figure 1).

Scalding

The scalding procedure is used to open the feather follicles to facilitate the removal of feathers. The potential for bacterial cross-contamination during scalding and picking is well recognized (Bailey and others 1987), and *Campylobacter* has been periodically recovered from scald water (Stern and others 2001). It is hypothesized that the follicles might remain open throughout the processing until the carcass is chilled. When the follicles close during chilling, the microorganisms may be retained. A study by Cason and others (1999) evaluated the microbiological effect of removing feathers from the carcasses while they are out of the scald water and moving between the tanks of a multiple-tank scalder. The data showed no reduction in populations of aerobic bacteria, *Escherichia coli*, or *Campylobacter* on carcasses during scalding and defeathering. Chicken skin has been shown to harbor and support the survival of *C. jejuni* (Lee and others 1998). Berrang and Dickens (2000) found 3.80 $\log_{10}$ CFU/g of *Campylobacter* in breast skin before entering the scald tank.

Berrang and Dickens (2000) studied the presence and level of *Campylobacter* on broiler carcasses throughout the processing plant. In this study, samples were collected immediately before scald, post-scald, post-pick before transfer to the evisceration line, immediately after the removal of the viscera, after the final washer, and post-chill. *Campylobacter* carcass rinse counts were found to be the highest when carcasses were sampled pre-scald (4.73 $\log_{10}$), then the counts dropped significantly after the carcasses were scalded (1.80 $\log_{10}$). They also found 3.80 $\log_{10}$ CFU/g of *Campylobacter* in breast skin sampled before entering the scald tank.

Defeathering

Wempe and others (1983) isolated *C. jejuni* from 94.4% of the feather picker drip water samples, and the population of organisms present was high. They believe that this is an area where cross-contamination may occur, since the rubber fingerlike projections that beat the feathers from the bird become contaminated and may pass the organism from bird to bird. They observed that the water used in rinsing the birds in the feather picker physically removed the *Campylobacter* organism and thus reduced the number of organisms on the edible parts. They recovered *C. jejuni* from all recycled water samples tested. The use of recycled water to clean the gutters may further contaminate the receiving room with *C. jejuni*. Further distribution of *C. jejuni* may also occur through movement of plant personnel from the receiving area to other areas of the plant.

Berrang and Dickens (2000) found that after defeathering, the counts increased significantly (3.70 $\log_{10}$). An increase in *Campylobacter* counts after defeathering has been previously reported (Acuff and others 1986; Izat and others 1988). It has been suggested that the rubber fingers in the mechanical picker act to cross-con-

taminate birds that previously had low or undetectable levels of *Campylobacter* (Acuff and others 1986; Stern and others 1995b).

Evisceration

Chicken skin has been shown to harbor and support the survival of *C. jejuni* (Lee and others 1998). Berrang and others (2001b) studied the presence and level of *Campylobacter*, coliforms, *E. coli*, and total aerobic bacteria recovered from broiler parts with and without skin. Samples were taken from defeathered carcasses before evisceration. No *Campylobacter* were recovered from meat collected from the breasts or thighs, and only 2 of 10 drumstick meat samples had detectable levels of *Campylobacter*. However, 9 of 10 breast skin, 10 of 10 thigh skin, and 8 of 10 drumstick skin samples were positive for *Campylobacter*, with levels between 2 $\log_{10}$ and 3 $\log_{10}$ CFU/g of *Campylobacter* after evisceration. In a related study, Altmeyer and others (1985) collected 50 muscle samples from broilers and found no *Campylobacter*. Kotula and Pandya (1995) found higher counts on breast tissue of broiler meats than on the thigh or drumstick.

The high incidence of contaminated neck flaps and breast tissue suggest that the crop contents may be an important source of *Campylobacter* contamination during processing. The crop has been found to be a significant source of *Campylobacter*, thus potentially contributing to carcass contamination (Byrd and others 1998a). Berrang and others (2000) reported that 100% of the crops of 18 broilers were positive for *Campylobacter*. The study also showed that *Campylobacter* could be found on the skin of carcasses in the early stages of processing even with no contamination from internal organs. A recent study (Berrang and others 2002) found that removal of skin before processing reduces *Campylobacter* levels by 0.7 $\log_{10}$ CFU/carcass.

Jeffery and others (2001) studied the prevalence of *Campylobacter* from skin, crop, and intestine of commercial broiler chicken carcasses at processing. They sampled 6 to 12 carcasses from 22 flocks just before evisceration and found skin samples 78% positive, crops 48% positive, and the intestines 94% positive.

Berndtson and others (1992) isolated *Campylobacter* in 89% of neck skin samples, 93% of peritoneal cavity swab samples, and 75% of subcutaneous samples. They also found that muscle samples were only very sparsely contaminated, and believed it was likely that the feather follicles were the orifices where *Campylobacter* is introduced into the subcutaneous layer. Overall, *Campylobacter* counts dropped as the flocks moved through the plant (Berrang and Dickens 2000).

Carcass washers

Carcasses are commonly washed with systems of washers using chlorinated water to remove contamination, such as blood, tissue fragments, and fecal contamination as part of the regular processing procedures. Carcass washing has been allowed for poultry since 1978 as an alternative to knife trimming because studies have shown it to be equally effective in removing fecal contamination

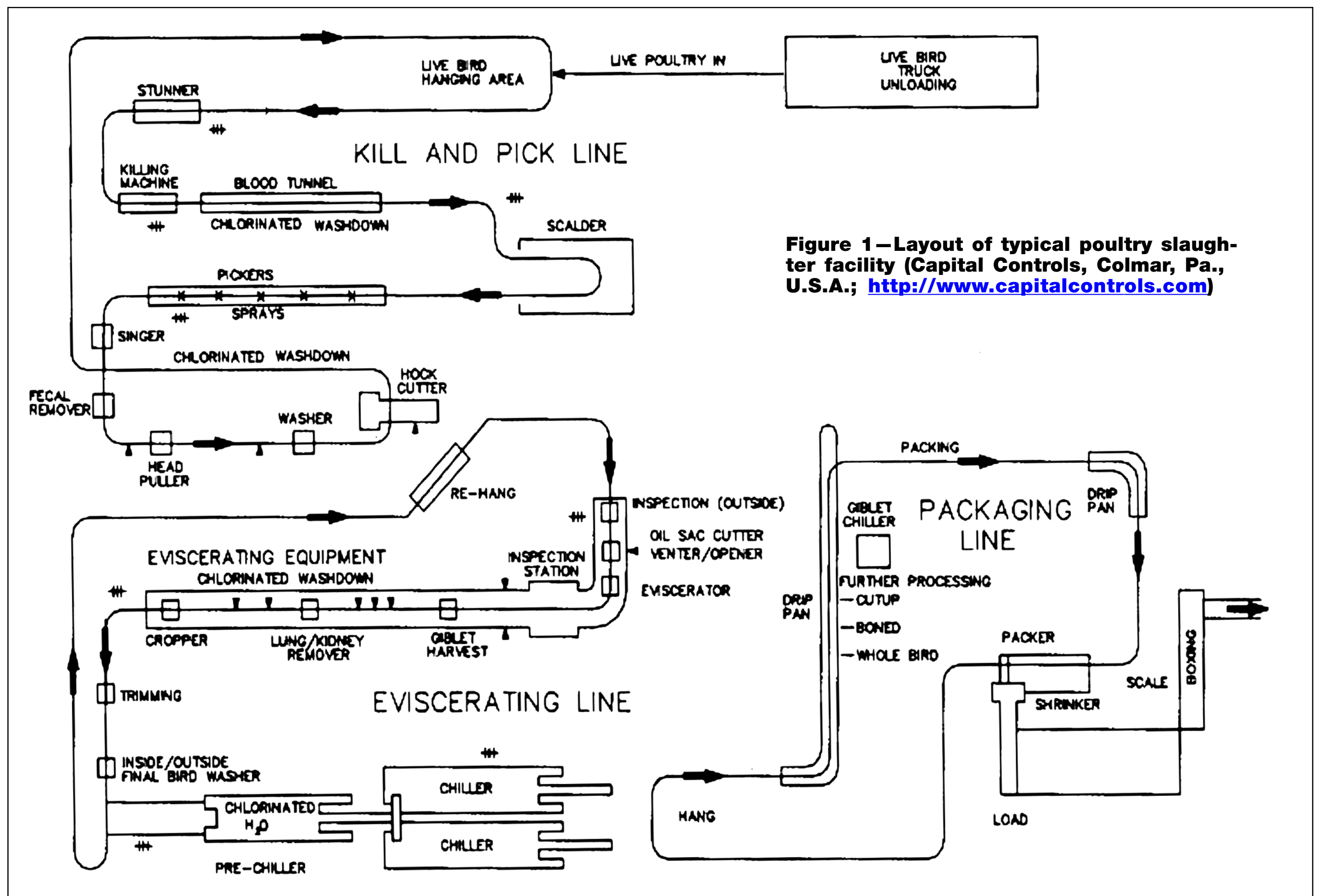

Figure 1—Layout of typical poultry slaughter facility (Capital Controls, Colmar, Pa., U.S.A.; http://www.capitalcontrols.com)

(SCVMRPH 1998). The development of new washer systems makes it very difficult for a processing plant to know which type or system would be best for them. Limited studies have been conducted on evaluating the performance and effectiveness of poultry washers and sanitizing treatments within the processing plant (Anand and others 1989; Dickens and Cox 1992; Bautista and others 1997). Sales representatives from carcass washer manufactures have provided design and performance data for their respective wash systems, but no comparison data are available. In addition, many plants have made individual modifications to these systems, and experience indicates that wash systems installed in 1 plant may not perform equally well in another plant. There are numerous parameters that affect the overall effectiveness/efficiency of the carcass washing system, including number of washers and types. For specific washers, operating parameters include wash water temperature, water pressure, nozzle type, nozzle arrangement, flow rate, line speed, and surfactant/sanitizing agents used.

There are several types of carcass washers currently used in poultry processing plants. These include brush washers, cabinet washers, and inside/outside bird washers. A brush washer acts similarly to that of a car wash. It has many rubber fingers that gently remove debris with the aid of water from the outside of the carcass. A cabinet washer has a series of nozzles enclosed by a cabinet that spray water on the outside of the carcass. In an inside/outside carcass washer, the carcasses enter with the breast facing toward the inside of the machine. As the machine rotates, a probe with a single spray nozzle enters the bird's intestinal cavity and washes the inside thoroughly. Then a series of spray nozzles wash the entire outside of the bird. Many inside/outside bird washers are equipped with pressure nozzles operating between 40 and 180 psi for removing visible fecal contamination on carcasses. These wash systems use 20 to 50 ppm of chlorine as an antimicrobial agent and generally consume 25 to 50 gallons/min (GPM) of water. The 3 primary suppliers of poultry carcass washers are Stork Gamco Inc. (Stork Gamco Inc., Airport Parkway, Gainesville, Ga., U.S.A.), Linco USA (LindHolst & Co., Trige, Denmark), and Baader Johnson (Johnson Food Equipment, Inc., Kansas City, Kans., U.S.A.).

The washer systems currently used for inside and outside surface cleaning of chicken carcasses have shown a limited effectiveness for *Campylobacter* removal (Bashor and others 2004). The primary reason is that washing with cold water, regardless of pressure and flow volume, does not lower water surface tension, an important factor in bacterial/fecal removal. Some plants use more than 9 L of water per bird for carcass washing with a minimal (0.5 $\log_{10}$) reduction in *Campylobacter levels* (Bashor and others 2004). An average poultry processing plant spends $500000 to $1 million dollars per year on water for carcass washers (Jackson and others 1999). Further design modifications of carcass washers are needed to reduce water consumption, provide scrubbing action, and introduce surfactants during the washing process that can lower water surface tension and aid in bacteria and fecal removal.

Carcass chillers

Poultry carcasses are required to be cooled rapidly to prevent bacterial growth (USDA 2003a). Many poultry processors use water chillers for rapid cooling of carcasses. Recent studies on *Campylobacter* document its potential for cross-contamination in water chillers (Sanchez and others 2002; Whyte and others 2002). In the chill tank, chlorination with up to 50 mg/L at pH 6.0 is required to control cross-contamination of poultry carcasses due to the increased organic load (Goresline and others 1951; Ziegler and Stadelman 1955; Dawson and others 1956; McVicker and others 1958; Mallman and others 1959). To reduce cross-contamination, USDA (1995) has required the addition of 20 to 50 ppm chlorine to water chillers to prevent cross-contamination.

USDA has also allowed, in addition to chlorine, the use of ozone (USDA 2003b) and chlorine dioxide (1996c) in chillers to prevent cross-contamination. In addition, researchers and industry are exploring air and cryogenic nitrogen chilling. A recent study by Sanchez and others (2002) found that *Campylobacter* levels on chilled carcasses were significantly higher in immersion chilling than air chilling. A commercial cryogenic nitrogen chiller is commercially available and being used in poultry plants (Air Products 2003).

Processing aids

When assessing the right treatment to use, there are many factors that should be considered such as overall efficacy, levels of microbial contamination, potential for introducing other food safety hazards, impact on the environment, effects on sensory properties and quality of the product, feasibility, and consumer perception (SCVMRPH 1998). Demonstration of efficacy should include not only laboratory tests but also in-plant investigations. Possible treatments for reducing microbial contamination of poultry carcasses include physical methods, chemical methods, and irradiation. The most common physical methods include cold water, hot water, and high pressure. Less common physical methods include steam and steam vacuum. Popular chemical methods include organic and inorganic acids and bases, and/or chlorine and related compounds. A less popular, although effective method, for bacterial reductions is irradiation.

Water. Washing with potable water is used extensively in poultry processing and may result in an overall reduction of surface contamination by 90% to 99% (SCVMRPH 1998). Warm water (40 °C to 70 °C) washing has received limited study in poultry processing. A study by Li and others (2002) found that a 60 °C hot water spray reduced *Campylobacter* levels 0.78 $\log_{10}$ CFU/carcass compared with a 20 °C water. A recent study by Purnell and others (2004) found that a 70 °C, 40-s rinse showed no detrimental effect on chicken skin and produced a 1.6 $\log_{10}$ reduction in *Campylobacter*/mL. There are currently poultry processing plants in the United States using a warm water washer to precondition carcasses before further washing and processing aid treatment (Bashor 2002). It is suspected that warm water rinsing kills bacteria directly and also reduces the surface tension of the water, which may enhance bacteria and fecal removal.

Organic acids. There are several organic acids that have proven to be effective in poultry processing such as acetic acid, lactic acid, citric acid, and succinic acid. They all work very well in killing bacteria, especially *Salmonella*, because of their ability to penetrate and disrupt the cell membrane, and to acidify the cell contents (SCVMRPH 1998). They are very stable in the presence of organic material often present in poultry processing plants. Because they are acids, they can corrode equipment and can cause off flavors, odors, and colors. Thomson and others (1976) compared the effect of acid, heat treatment, and chlorine on inactivating *Salmonella* on broiler carcasses and observed that succinic acid alone (1% at 55 °C) reduced *Salmonella* prevalence by 50%. Lillard and others (1987) reported that 0.2% to 0.5% acetic acid added to the scalding water reduced the total aerobic plate count and *Enterobacteriaceae* populations. Okrend and others (1986) added 0.1% acetic acid to scald water and observed a reduction in populations of *Salmonella* Typhimurium and *C. jejuni* from 0.5 to 1.5 $\log_{10}$ CFU/mL. Bautista and others (1995) studied the effect of lactic acid, chlorine (50 ppm), and trisodium phosphate (TSP) sprays under various pressures on treating turkey carcasses. They observed that 1.25% and 4.25% lactic acid caused a 2.4 and 4.4 $\log_{10}$ reduction in aerobic plate count. The acid spray had an even greater impact on the reduction of coliforms and also reduced *Salmonella*.

Chlorine. Chlorine has been used in poultry processing for more than 40 y to reduce spoilage bacteria, control the spread of pathogens, and prevent build-up of microorganisms on working surfaces and equipment such as chill tanks (Bailey and others 1986). When sodium hypochlorite is injected into water, it forms hypochlorous acid, the form of chlorine responsible for its antimicrobial properties (Gavin and Weddig 1995). The addition of chlorine gas to processing water is easily controlled. However, most waters contain organic impurities that will react with the initial amount of added chlorine reducing the amount of available chlorine to form hypochlorous acid. Chlorine added to water will continue to react and be reduced by these impurities until the impurities have been completely oxidized. The amount of chlorine required for this purpose is known as the chlorine demand of the water.

Any chlorine present over the chlorine demand of the water exists as combined residual chlorine or free residual chlorine. The concentration of chlorine where free residual chlorine exists is called the break point. Chlorine combines loosely with nitrogenous (organic) matter to form chloramines and other chloro–nitrogen compounds. These are forms of combined residual chlorine and exhibit relatively weak germicidal properties (Gavin and Weddig 1995).

The rate at which bacteria are killed is proportional to the concentration of free residual chlorine. The pH of the water after the addition of chlorine determines how fast the microorganisms will be killed. The lower the pH (below 7.5), the faster the microorganisms are killed, and as the pH increases, the effectiveness of the chlorine decreases (Gavin and Weddig 1995). Many present-day chill tank water treatment programs operate with the cooling water pH in the range of 8.0 to 8.5 or higher. As a result, oxidizing microbiocides such as chlorine are less effective. At a pH of 6.0, chlorine hydrolyzes almost completely to hypochlorous acid (HOCl), which is the most effective form of chlorine for microbiological control; however, at a pH of 8.5, only 8% goes to HOCl, thus requiring a much higher dosage of chlorine to control bacteria. Contamination of processing equipment is progressively reduced by increasing the chlorine concentration to 70 mg/L at pH 6.5 (Bailey and others 1986). Chlorine is active against a wide range of microorganisms, with various degrees of susceptibility. At a pH of 6.0, 0.1 mg/L of free available chlorine killed 99% of *C. jejuni* (Blaser and others 1986). The necessary contact time varied between 5 min and 15 min at 25 °C.

Under conditions of commercial processing, not all studies involving chlorine have shown a reduction in carcass contamination. Mead and others (1975) showed that neither the levels of contamination of bacteria nor the occurrence of cross-contamination were reduced by spray-washing in chlorinated water after evisceration. Sanders and Blackshear (1971) showed little effect of chlorine in the final carcass wash unless at least 40 mg/L were used. Washing carcasses post-chilled with water containing 50 mg/L of chlorine did not reduce the proportion of *Salmonella*-positive samples (Kotula and others 1967). These studies emphasized the importance of adequate contact time, which is not usually achieved in a washing operation.

A study by Waldroup and others (1993) examined the modification of broiler processing procedures to include 20 ppm of chlorine through the processing line and include 1 to 5 ppm of free chlorine in the chill tank overflow. These concentrations resulted in a 0.2 $\log_{10}$ to 0.6 $\log_{10}$ reduction in aerobes, 0.0 $\log_{10}$ to 0.3 $\log_{10}$ reduction in coliforms, and 0.0 $\log_{10}$ to 0.4 $\log_{10}$ reduction in *E. coli*.

Chlorine dioxide. Chlorine dioxide (ClO_2) is an antimicrobial compound recognized for its disinfectant properties since the early 1900s. In 1967, the Environmental Protection Agency (EPA) first registered the liquid form of chlorine dioxide for use as a disinfectant and sanitizer. In 1988, the EPA registered chlorine dioxide gas as a sterilizing agent. Chlorine dioxide is a synthetic yellowish-green gas with chlorine-like odor. ClO_2 is unstable as a gas and will undergo decomposition into chlorine gas (Cl_2), oxygen gas (O_2), and heat. However, ClO_2 is stable and soluble in an aqueous solution and does not form hypochlorous acid or react with ammonia. It functions independent of pH and can provide excellent control at a fraction of the chlorine dosage because it can be used at much lower doses (Lillard 1979). The smaller dosage also makes chlorine dioxide more cost-effective. Chlorine dioxide kills microorganisms by disrupting transport of nutrients across the cell wall. It can be generated in a gas or liquid form and smells like chlorine bleach. The additive may be used to control the microbial population in poultry processing chill water in an amount not to exceed 3 ppm residual chlorine dioxide (Liem 2002).

Chlorine dioxide reduces microbial contamination of carcasses much the same way as chlorine but is up to 7 times more active, can be used at lower concentrations such at 3 to 5 mg/L in the chill tank, and is less corrosive (Lillard 1979). Chlorine dioxide does not appear to have an effect on meat flavor but tends to result in a slightly lighter skin color (Thiessen and others 1984).

Trisodium phosphate. The TSP system was created to eliminate the need for off-line reprocessing. It is a white, free-flow crystalline material that complies with the specifications of the Food Chemicals Codex. It has been certified by the National Sanitation Foundation (NSF International) for use in the treatment of drinking water at a maximum dosage of 41.5 mg/L. Trisodium phosphate has a pH of 11.8 at a concentration of 12% (SCVMRPH 1998). The use of TSP can be costly because of the level needed to clean the carcass. Residual TSP on the carcasses entering the chiller causes the pH to increase dramatically from 7.0 to over 11.0. In plants where TSP is used, chiller water pH can be in the 10 to 11 range. This high pH level greatly negates the antimicrobial properties of chlorine (SCVMRPH 1998).

The bactericidal effect of TSP is well documented in the scientific literature and confirmed in several industrial studies. Trisodium phosphate is more active on Gram-negative pathogens such as *Salmonella*, *Campylobacter*, and *E. coli* than against Gram-positive ones such as *Listeria monocytogenes*. There are several mechanisms for the TSP mode of action: surfactant properties; destructive effect on bacteria at the high pH (pH 11); removal of bacteria that are not yet firmly attached to the skin surface; removal of some surface fat, which facilitates the removal of bacteria by the washing process; and an effect on the bacterial cell wall.

The overall activity of TSP against *Salmonella* has been well documented (Bender 1992; Gudmunsdottir and others 1993; Li and others 1994; Hwang and Beuchat 1995; Lillard 1995). In a study using broiler carcass wings, Rodrigues de Ledesma and others (1996) compared the effect of water at 95 °C for 10 s to the effect of dipping in 10% TSP or 10% sodium carbonate for 10 s. Trisodium phosphate alone caused reductions of 84.3%, 65.3%, and 60.2% of *S.* Typhimurium, *Staphylococcus aureus*, and *L. monocytogenes*, respectively. A combination of TSP and hot water resulted in reductions of 98.6%, 99.5%, and 99.7%, respectively.

Salvat and others (1997) and Coppen and others (1998) investigated TSP treatment of poultry carcasses and found a 2 $\log_{10}$ reduction of *Salmonella*. Pre-chill spraying of chicken with TSP solution (10%) resulted in 2.1 $\log_{10}$ to 2.2 $\log_{10}$ reduction in *Salmonella*. The effect of TSP on *C. jejuni* has been demonstrated, with differences noted between in vitro studies and industrial tests. Using artificial biofilms or cell suspensions, Somers and others (1994) reported a reduction of 5 $\log_{10}$. Slavik and others (1994) and Federighi and others (1995) demonstrated in an industrial trial a reduction of 1.2 $\log_{10}$ to 1.5 $\log_{10}$. It has been reported to remove significant numbers of *E. coli*, *Enterobacteriaceae*, *Campylobacter*, *Salmonella*, and total aerobes from poultry carcasses by

more than 2 $\log_{10}$ when concentrations are between 10% and 12% (pH 11.5 to 13) (Ellerbroek and others 1992; Somers and others 1994; Federighi and others 1995; Salvat and others 1996).

The Rhodia corporation (Rhodia Inc., Cranbury, N.J., U.S.A.) has conducted several studies using the AvGard® TSP spray and its affect on *E. coli* 0157:H7. Using a 12% solution at 21 °C, the results indicate an average reduction of 2 $\log_{10}$, and showed an even greater reduction at 38 °C. Bashor (2002) found a 1.2 $\log_{10}$ reduction from broiler carcass rinse samples collected before and after TSP spray in a commercial poultry processing plant.

It is hypothesized that the increased wetting ability of hot water and TSP physically remove bacteria in addition to killing them. To conserve water and further improve food safety of poultry, further research into the development of soaps and surfactants specifically designed for removing microbial contamination from poultry carcasses should be initiated.

Acidified sodium chlorite. Acidified sodium chlorite (ASC) had been used as a disinfectant in hospitals, dental operations, and pharmaceutical clean rooms (Kemp and others 2001). It has also been approved by the Food and Drug Administration (FDA), EPA, and the USDA as an antimicrobial for use on poultry, red meats, fruits, vegetables, and seafood (Kemp and others 2001b). The Alcide Corporation (Redmond, Wash., U.S.A.) markets the Sanovaä spray system that is a commonly used ASC system in poultry processing plants. The chemistry of ASC is related to that of chlorine dioxide. When a solution of sodium chlorite is acidified with a weak organic acid, chlorous acid is formed. The formation of chlorous acid is instantaneous when chlorite and acid are combined. As the pH of the mixed solution decreases from pH 4, the proportion of chlorite that dissociates to chlorous acid increases. Acidified sodium chlorite typically operates in a pH range of 2.3 to 3.2 and acts as a broad-spectrum disinfectant by oxidizing the microbial cell wall, attacking the sulfide and disulfide linkage of proteins. It provides a nonspecific attack on the amino acid component of the cell membrane. Acidified sodium chlorite is applied at ambient temperature, either by a spray or immersion dip, with a concentration averaging 1000 ppm and a dosage rate of 1.3 to 1.5 oz of ASC solution/bird. It is considered a broad-spectrum oxidative antimicrobial, effective on pathogenic bacteria as well as viruses, fungi, yeast, molds, and some protozoa.

Kemp and others (2001b) investigated the effectiveness of using ASC in poultry processing plants under conditions similar to those in commercial poultry facilities. This study showed that the application of ASC was effective in reducing *E. coli* populations on broiler carcasses from an initial level of 3.1 $\log_{10}$ by 2.2 $\log_{10}$ and total coliforms with initial levels of 2.2 $\log_{10}$ by 1.5 $\log_{10}$.

A study conducted by the Alcide Corporation with the Sanova system in a broiler processing plant detected populations of *Campylobacter* spp. of 3.7 $\log_{10}$ after evisceration, 2.6 $\log_{10}$ postwash, and 1.1 $\log_{10}$ after the Sanova spray (Kemp and others 2001a). It was found that 73.2% of the carcasses were positive for *Campylobacter* after evisceration, whereas 49.1% were positive after the Sanova spray. The incidence of *Salmonella* spp. on broiler carcasses decreased from 37% to 10% after the application of the Sanova system.

Irradiation. The biological effects of ionizing radiation on cells can be attributed to direct interactions with critical cell components and to indirect actions by molecular entities such as free radicals formed in the water (SCVMRPH 1998). The DNA of the cell is the most critical target of ionizing radiation, and the inactivation of microorganisms is primarily due to damage to the DNA. Gram-positive bacteria seem to show the most resistance. Patterson (1995) investigated the sensitivity of *C. jejuni*, *C. coli*, and *Campylobacter fetus* to irradiation in poultry meat. The D_{10} values ranged from 0.12 to 0.25 kGy. There was a significant difference in the radiation sensitivity between *Campylobacter* species and within strains of the same species. The values indicated that *Campylobacter* was more sensitive to irradiation than *Salmonella* and *L. monocytogenes*. The FDA and USDA have approved irradiation of chicken at a maximum dose of 3 kGy to control foodborne pathogens such as *Salmonella* and *Campylobacter* (USDA 1996b).

Others. New and improved processing aids are being developed all the time. Recent studies examining alternative methods for reducing *Campylobacter* have included electrolyzed water (Park and others 2002), organic acid mixtures (Chaveerach and others 2002), and mixtures of phosphates and fatty acids (Hinton and Ingram 2003). Results have documented reductions similar to existing processing aids. Currently there is no treatment available to completely remove *Campylobacter* from poultry carcasses except irradiation.

On-line reprocessing

A study conducted by Fletcher and others (1997) evaluated on-line reprocessing on visible contamination and microbiological quality of broilers. Before this study, off-line reprocessing procedures for visually contaminated carcasses included the removal of the bird from the processing line, reprocessing in an approved off-line area, then chlorination and finally reinspection. Approved procedures included washing, vacuuming, and trimming (Fletcher and others 1997). On-line reprocessing would allow the carcass to proceed through evisceration and a chlorinated inside/outside carcass washer before examination for visual contamination at the pre-chill checkpoint. Carcasses that still had visible contamination would then be removed for manual off-line reprocessing. Results based on visual scores indicated that the on-line reprocessing reduced the need for off-line reprocessing by 73% to 84%. The incidence of *Salmonella* or *Campylobacter* was not affected by the treatment. This study showed that on-line processing of visually contaminated carcasses could greatly reduce the number of carcasses currently being subjected to off-line reprocessing without negative effects on total bacterial counts or presence of pathogenic organisms.

Post-processing contamination

The Minnesota Dept. of Health data showed that levels of contamination of retail poultry remain high despite interventions made at the processing plant (Smith and others 1999). Stern and Line (1992) detected *Campylobacter* spp. in 98% of retail-packaged broilers sampled from grocery stores. Another study by Willis and Murray (1997) found 69% (229/330) of raw commercial broilers were positive for *C. jejuni*. A study from New Zealand showed that *Campylobacter* could be isolated from 63% of chicken carcasses at retail outlets (Bongkot 1997).

Kinde and others (1983) indicated that the presence of *Campylobacter* in market broilers diminishes over time during refrigerated storage. Blankenship and Craven (1982) detected viable strains of *C. jejuni* stored in sterile ground chicken meat. The growth and extended survival observed at 37 °C with ambient atmosphere incubation suggest that the test strains were readily able to locate favorable microaerophilic conditions for growth within the ground meat after surface inoculation.

A study in the United Kingdom estimated a population range of *Campylobacter* organisms on the surface of fresh chicken carcasses from 3 to 6 $\log_{10}$ CFU per chicken (Friedman and others 2000). Kanenaka (2000) conducted a survey from 2 large retail markets in Hawaii to characterize strains of *C. jejuni* isolated from clinical and poultry samples. She found that samples collected at Oahu were 83.3% positive for *C. jejuni* on whole chicken samples and 91.7% positive on chicken parts, whereas mainland samples were 93.8% positive for whole chickens, 35.7% for chicken parts, and 56.8% for the total number of *C. jejuni* posi-

Table 1—*Campylobacter* incidence and populations measured in poultry production and processing

Source of contamination	Incidence	Populations (colony-forming units/g)
Farm	87.5%[a]	3 $\log_{10}$ to 9 $\log_{10}$[b]
	62%[b]	1995 feces, 4.93 $\log_{10}$[c]
	1995, 96.4%[c]	2001 feces, 5.16 $\log_{10}$[c]
	2001, 94%[c]	
Transport/initial level entering plant	0 h: Feed withdrawal, 0 %[b]	Fecal, 6 $\log_{10}$[e]
	0 h: Feed withdrawal, 90%[d]	
	8 h: Feed withdrawal, 85%[b]	Cecal, 6.5 $\log_{10}$[f]
	8 h: Feed withdrawal, 92%[d]	Cecal, 7.3 $\log_{10}$[g] Crop, 4.7 $\log_{10}$[h]
Scalding	Pre-scald: feathers, 77.5%[h]	Pre-scald: feather, 5.4 $\log_{10}$[g] Pre-scald: feathers, 7.4 $\log_{10}$[h]
	Pre-scald: skin, 52.5%[h]	Pre-scald: skin, 3.8 $\log_{10}$[g] Pre-scald: skin, 6.6 $\log_{10}$[h]
Feathers	90%[i]	8 $\log_{10}$[i]
Evisceration	42 d old, 100%[d]	Skin, 6 $\log_{10}$[j]
	56 d old, 96%[d]	Crop, 3.5 $\log_{10}$[f] 42 d old, 2.7 $\log_{10}$[d] 56 d old, 2.0 $\log_{10}$[d]
Carcass washers	Pre-wash, 87%[k] Post-wash, 80%[k]	Pre-wash, 4.78 $\log_{10}$[k] Post-wash, 4.3 $\log_{10}$[k]
Carcass chilling	Pre-chill, 99%[d]	Pre-chill, 4.75 $\log_{10}$[c] Pre-chill, 2.9 $\log_{10}$[d] Post-chill, 21% to 41%[a]
	Post-chill, 83%[d]	Post-chill, 3.03 $\log_{10}$[c] Post-chill, 1.6 $\log_{10}$[d]
Packaging/retail	69%[l]	4.64 $\log_{10}$[m]

[a]Stern and others (2001).
[b]Byrd and others (1998b).
[c]Stern and Robach (2001).
[d]Northcutt and others (2003).
[e]Oosterom and others (1983).
[f]Achen and others (1998).
[g]Berrang and others (2000).
[h]Kotula and Pandya (1995).
[i]Barnes (1975).
[j]Wilkerson (1961).
[k]Bashors and others (2004).
[l]Willis and Murray (1997).
[m]Kanenaka (2002).

tive samples. Overall, *Campylobacter* was shown to be present in 70% of samples. It was also observed that certain fryers were found to have substantial contamination with an estimated mean population of $\log_{10}$ 4.6 CFU/carcass.

Uyttendaele and others (1999) studied the incidence of *Salmonella, C. jejuni*, and *L. monocytogenes* on poultry carcasses and different types of poultry products on sale at a Belgium retail market. *Salmonella* was found in 36.5% of the samples, *C. jejuni* in 28.5% of the samples, and *L. monocytogenes* in 38.2% of the samples. They also found that contamination of poultry meat increases with further processing, but skinless parts were less likely to be contaminated with *Salmonella*, *Campylobacter*, and *L. monocytogenes* than those parts with skin. The prevalence of *Campylobacter* spp. in poultry and poultry meat products in Germany was studied by Atanassova and Ring (1999). Of 509 samples taken from poultry flocks, 41.1% were *Campylobacter*-positive, whereas broiler carcasses were 45.9% positive.

Most fresh poultry products are marketed in an air atmosphere, but some products are stored in a carbon dioxide atmosphere to extend shelf-life. The spoilage flora that develops on poultry during low-temperature air storage is predominantly *Pseudomonas* species, whereas in a carbon dioxide atmosphere, *Lactobacillus* species are most prevalent (Balley and others 1979). Blankenship and Craven (1982) found that *C. jejuni* survived quite well with spoilage flora that developed during both air and carbon dioxide atmosphere storage.

Thawing of poultry products at ambient room temperature for extensive periods is not recommended. Lee and others (1998) have reported that *C. jejuni* cells could replicate at room temperatures and under refrigeration at 4 °C. Many health authorities recommend thawing poultry rapidly and cooking it thoroughly to an internal end point temperature of 75 °C. It is also important to avoid cross-contamination of cooked and raw foods.

Dawkins and others (1984) examined work surfaces, sinks, and floors of areas where fresh and frozen chicken had been processed. Cleaning with detergent and hot water (or steam) and drying was sufficient to remove *C. jejuni* from the environment. They reported that drying surfaces after washing was an important factor in controlling persistence in the environment.

A recent study by Kramer and others (2000) compared the prevalence of *C. jejuni* and *C. coli* in fresh bovine, porcine liver, and chicken portions from retail outlets and compared the strain subtype distribution with those associated with cases of human campylobacteriosis from the same time period and study area.

Campylobacter were isolated from 73.2% of 489 samples, with chicken exhibiting the highest contamination rate (83.3%). Of the human isolates, 89.3% were *C. jejuni* and 10.7% *C. coli*. A significant proportion of the chicken and lamb isolates shared identical subtypes with the human strains, indicative of their role as potential sources of infection.

Summary

The illness caused by *Campylobacter* contamination is clearly a major issue in our food system. Because such a large majority of contamination is associated with poultry (50% to 70%), it is important to focus on this vehicle. Control strategies have been improved in recent years including on-farm techniques and methods during poultry processing. However, these strategies still require significant improvements to completely remove or significantly reduce the threat of *Campylobacter* contamination.

Current trends find that some poultry processors are starting to monitor bacterial levels in birds when they are received at the plant and compensate growers based on these data. Processing plants that have implemented monitoring programs indicate that incidences of fecal and *Salmonella* failures have been greatly reduced. A majority of poultry processing plants do not measure *Campylobacter* levels regularly.

Table 1 summarizes *Campylobacter* incidence and populations measured in poultry production and processing. Significant *Campylobacter* populations can be found on a majority of chickens entering the processing plant. This contamination is easily spread from carcass to carcass during processing. Further effort is needed to design more efficient and effective washing systems. An average poultry processing plant spends $500000 to $1 million dollars per year on water for washing chicken carcasses with minimal reductions in bacteria. A large portion of the bacterial reduction occurs from the application of processing aids such as TSP and ASC. Although *Campylobacter* contamination is reduced during processing, they are still present on the carcass after processing at levels of 2 to 4 $\log_{10}$ organisms/gm. Although these reductions are significant, they still may not reduce the levels of contamination below the threat to public health since as few as 500 organisms can make a person ill.

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