Journal of Applied Microbiology 2001, **90**, 96S–114S

Poultry as a source of *Campylobacter* and related organisms

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1. SUMMARY

Approximately 80% of raw chickens sold in the UK are contaminated with thermophilic campylobacters and they can be found on carcasses at levels as high as several thousand per cm^2 of skin. Thus, although they do not multiply on meat, they have a low infectious dose, so that human infection from undercooked meat, or as a result of handling raw poultry, is common. The precise contribution of poultry to human infection is not clear, but comparisons of types infecting humans and animals indicate that there are other important sources of human infection, particularly cattle. Rates of contamination of raw poultry meat with *Arcobacter* species and *Helicobacter pullorum* are also very high. Thermophilic campylobacters and *H. pullorum* colonize the chicken gut and rarely cause disease in poultry. *Arcobacter* species often appear to be environmental contaminants rather than part of the natural gut flora of poultry, although *A. butzleri*, in particular, can cause intestinal infections and abortion in humans.

Thermophilic campylobacters are not generally thought to be transmitted vertically via eggs, nor via feed or litter, provided rearing houses are cleaned and disinfected between flocks, and litter renewed. Flocks usually become infected at about 3 weeks of age. Every bird is usually rapidly colonized, with high levels (10^6–10^7 cfu/g) in the caecal contents. The source of infection can be via unchlorinated water, but in situations where the water supply is not to blame, the precise source of infection is seldom identified. Infection could be via wild birds, rodents, or from farm operatives via boots or clothing. Infection has sometimes been associated with 'thinning' of flocks about a week prior to slaughter. Avoidance of infection during rearing therefore relies mostly on careful attention to hygiene, exclusion of vermin and a clean water supply.

During transport, slaughter and dressing, *Campylobacter*-negative flocks can readily be contaminated from positive flocks. Contamination can be reduced by improved disinfection of transport crates, slaughter of uninfected flocks prior to infected flocks, and by careful attention to major points of cross-contamination on the line. A more effective measure would be to use a terminal decontamination step, such as trisodium phosphate, lactic acid, reduced pressure steam or gamma irradiation.

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2. INTRODUCTION

It is well known that chickens are frequently colonized by *Campylobacter jejuni* subsp. *jejuni* (henceforth referred to as *C. jejuni*) and *C. coli*, and that *C. jejuni* from chicken meat is considered to be the source of most human infection with this species (Hopkins and Scott 1983; Deming *et al.* 1987; Stern and Kazmi 1989; Doyle 1990; Altekruse *et al.* 1994). However, recent studies have indicated that at least one species of *Helicobacter* (*H. pullorum*) is a common inhabitant of the caeca and large intestine of broiler chickens (Atabay *et al.* 1998b) and that *Arcobacter* spp., especially *A. butzleri*, but also *A. cryaerophilus* and *A. skirrowii*, are common on broiler chicken carcasses and in the slaughter house environment, although not in the intestines of the birds (Atabay *et al.* 1998a; Harrass *et al.* 1998; Wesley and Baetz 1999; Gonzalez *et al.* 2000). Other species of intensively raised poultry, particularly turkeys and ducks, are colonized by campylobacters, sometimes to a greater extent than chickens (Luechtefeld and Wang 1981; Prescott and Bruin-Mosch 1981; Acuff *et al.* 1982, 1986; Kasrazadeh and Genigeorgis 1987; Lammerding *et al.* 1988; Ridsdale *et al.* 1998). In addition to *C. coli* and C. jejuni, *C. upsaliensis* has been found in ducks (Ridsdale *et al.* 1998). Arcobacters have also been isolated from commercially reared ducks (Ridsdale *et al.* 1998) and turkeys (Manke *et al.* 1998; Wesley and Baetz 1999) but there are as yet no reports of helicobacters.

3. THE ROLE OF CAMPYLOBACTERACEAE IN DISEASES OF POULTRY AND MAN

It is often stated that campylobacters are harmless commensals in poultry and many wild bird species. However, a severe disease, 'vibrionic hepatitis' was prevalent in the 1950s and 1960s in chickens in North America and Europe, and was apparently caused by *C. jejuni*. The disease was most common in laying flocks, seldom appeared in birds less than 8 weeks of age and caused 10–15% mortality, with up to 35% reduction in egg yield (Peckham 1958; Sevoian *et al.* 1958; Winterfield *et al.* 1958). The reason for the subsequent decline in the incidence of this disease is not clear, but it has been attributed to the change to use of cages for layers (Peckham 1984; Shane 1991). Studies with campylobacters have shown that under normal commercial conditions chicks are seldom colonized before 2 weeks of age (Annan-Prah and Janc 1988; Kazwala *et al.* 1990; Berndtson *et al.* 1996a; Gregory *et al.* 1997; Newell and Wagenaar 2000). However, chicks can be infected by oral dosing with *C. jejuni* from day of hatching onwards and often show symptoms of infection (Ruiz-Palacios *et al.* 1981; Sanyal *et al.* 1984; Welkos 1984; Al-Obaidi 1988; Kaino *et al.* 1988; Young *et al.* 1999). Ruiz-Palacios *et al.* (1981) reported that 88% of 3-day-old chicks given 9×10^7 cfu of a human isolate of *C. jejuni* developed diarrhoea, with a 32% death rate. Nine out of 10 chicks given 90 cells developed watery diarrhoea. Similar results were obtained by Sanyal *et al.* (1984), who also found *C. jejuni* present in significant proportions of spleens, livers and hearts 4–6 d after inoculation. Internal contamination of chicken liver may also account for the identification of chicken liver consumption as a risk factor for human *Campylobacter* enteritis by Schorr *et al.* (1994). Although liver is commonly contaminated externally during processing, Baumgartner *et al.* (1995) found that contamination was internal as well as external, indicating either liver infection in life, or postmortem spread. Neill *et al.* (1984) reported that broiler chickens infected naturally before the age of 2 weeks also showed excess mortality. Enteritis and liver disease in poultry due to *Campylobacter* still seems to be a problem in Eastern Europe (Glunder 1993; Wieliczko 1994; Varga 1997).

3.1. Location and numbers of campylobacters in poultry

Campylobacters in the living bird are found in highest numbers in the large intestine, caecum and cloaca (Welkos 1984; Beery *et al.* 1988; Achen *et al.* 1998). Numbers in the region of 10^5–10^9 cfu per g intestinal contents have commonly been observed (Berndtson *et al.* 1992; Stern *et al.* 1999; Berrang *et al.* 2000), although Wallace *et al.* (1997) reported levels higher than 10^{12} per g in caecal contents. Since total viable microflora counts in caecal or intestinal contents seldom exceed 10^{12} per g, this result seems surprising, and might be an artefact due to the 'most probable number' method of counting used.

When a broiler flock first becomes infected with campylobacters, the organisms usually spread so rapidly that close to 100% of birds are reported to become colonized in a very short time (Lindblom *et al.* 1986; Jacobs-Reitsma *et al.* 1995; Newell and Wagenaar 2000), although Glunder (1993), Achen *et al.* (1998) and Payne *et al.* (1999) found strains of *C. jejuni* which colonized significantly lower proportions of flocks. There is some evidence that carriage rates fall as the birds get older (Pokamunski *et al.* 1986; Achen *et al.* 1998), that individual birds excrete campylobacters intermittently (Achen *et al.* 1998), and that birds infected when older excrete campylobacters for a shorter time (Lindblom *et al.* 1986; Kaino *et al.* 1988). In commercial flocks infection can be with multiple types, with a succession of strains appearing (Jacobs-Reitsma *et al.* 1995; Stern *et al.* 1997; Studer *et al.* 1999; Newell and Wagenaar 2000).

3.2. *Helicobacter pullorum* and *Arcobacter* species

H. pullorum can cause hepatitis in broiler chickens, and it has also been isolated from the liver, duodenum and caeca of

asymptomatic birds (Stanley *et al.* 1994; Atabay *et al.* 1998b; Fox *et al.* 1999). In addition, it has occasionally been reported as a cause of enteritis in humans (Burnens *et al.* 1994; Steinbreuckner *et al.* 1997, 1998; Kusters and Kuipers 1998; Melito *et al.* 1999). A. *butzleri*, and more rarely A. *cryaerophilus*, have been implicated as causes of diarrhoea and other infections in humans (Lerner *et al.* 1994; Lauwers *et al.* 1996; On 1996; Wesley 1996; Engberg *et al.* 2000). A. *butzleri*, *A. cryaerophilus* and *A. skirrowii* have all been found on poultry carcasses (Atabay *et al.* 1998a), but it appears that they may not colonize the poultry intestinal tract. Atabay and Corry (1997) examined four intestinal sites (gizzard, small intestine, caecum and colon) from 15 carcasses which were positive for arcobacters. From these 60 samples, *Arcobacter* was isolated only once, from colon contents. Similar results were obtained by Harrass *et al.* (1998), who examined 170 broiler carcasses and caecal contents. Fifty-seven percent of skin samples were positive for *Arcobacter* spp., but none of the caecal contents. On the other hand, Ridsdale *et al.* (1998) found *Arcobacter* spp. both on and in ducks, and Wesley and Baetz (1999) found 15% of 407 chicken cloacae positive for arcobacters. However, when these workers tried to infect, by oral dosing, 20 chicks with four strains of *A. butzleri* isolated from chicken carcasses, they were unsuccessful. A similar experiment with four *A. butzleri* strains from turkey meat succeeded in infecting only 4/67 turkey poults.

3.3. Contribution of poultry to human *Campylobacter* infections

Evidence for poultry meat being the prime source of human *Campylobacter* infections is mostly indirect, because most cases are sporadic, and not traced to a specific source. Thus most evidence has been gained by use of case control studies or comparison of strains isolated from various sources with those causing infections in humans. A few outbreaks have been attributed to poultry meat, usually undercooked, or cross-contaminated from raw poultry (Rosenfield *et al.* 1985; Tauxe 1992; Pebody *et al.* 1997). Outbreaks have also occurred from raw or improperly pasteurized cow's milk and from sewage-polluted water (Finch and Blake 1985; Tauxe 1992). Besides the sporadic nature of most cases, another handicap has been the lack of a readily available method of typing of strains. Earlier work tended to use serotyping schemes; these were not very discriminating, but confirmed the link between strains found in chickens and those in humans, and indicated that some infections probably originated from other sources (Jones *et al.* 1984; Banffer 1985; Pokamunski *et al.* 1986; Fricker and Park 1989; Elhamakijelinek and Awadmasalmeh 1992; Koenrad *et al.* 1995). More recently, genotypic typing methods, such as pulsed field gel electrophoresis (PFGE) and flagellin gene restriction fragment length polymorphism (Fla-typing) have been used (Newell *et al.* 2000). Fla-typing examines the variations in base sequence in the variable regions of the flagellin genes. Recent studies using these newer typing methods confirm that a significant proportion of human strains appear not to be of poultry origin (Koenrad *et al.* 1995; Hudson *et al.* 1999) and that some originate from cattle (Nielsen *et al.* 1997; On *et al.* 1998; Nielsen and Nielsen 1999). This is surprising, given the very low level of contamination reported for beef. However, cattle frequently carry large numbers of campylobacters in their intestinal contents, and sometimes suffer from mastitis due to *Campylobacter*, so that the source of infection for humans could be contaminated milk, or surface water contaminated from farm manure. Red meat offal also tends to be more highly contaminated than meat (Fricker and Park 1989; Bolton *et al.* 1999). Another explanation might be that cattle and humans are infected from another common source.

Case control studies have identified various risk factors besides poultry, including contact with cats and dogs, drinking raw milk or untreated water, eating sausages from a barbecue, and drinking milk from bottles pecked by wild birds (Hopkins *et al.* 1984; Deming *et al.* 1987; Kapperud *et al.* 1992; Ikram *et al.* 1994; Bloomfield 1997). One study even suggested that handling raw chicken was protective (Adak *et al.* 1995), although the high prevalence of *Campylobacter* infection among young male college students has been attributed to their preparing raw chicken in the kitchen (Hopkins and Scott 1983; Pearson *et al.* 1987). This apparent contradiction can be explained if people who habitually handle raw poultry become resistant to infection, while new college students, who had previously not cooked for themselves, would be susceptible. Anecdotal and published reports indicate that staff new to poultry abattoirs frequently contract *Campylobacter* diarrhoea during their first few weeks of employment (Christensen *et al.* 1983; Grados *et al.* 1983; Hopkins and Scott 1983; Berndtson *et al.* 1996a).

It is clear that many strains of *Campylobacter* colonizing poultry are not pathogenic to man, and that some human strains do not readily colonize poultry (Clark and Bueschkens 1988; Korolik *et al.* 1995, 1998). The possibility that man is occasionally the source of infection for poultry and other animals should not be ignored, especially as human intervention is frequently implicated as the source of infection for broiler chickens on growing farms (Cherkassy *et al.* 1991; see section 6).

4. METHODS OF DETECTION

Probably the first published evidence of poultry meat as a source of campylobacters was by Smith and Muldoon (1974), who found only 3/165 samples positive. This low

prevalence was probably because the method of isolation was poor until the media of Skirrow (1977) and Blaser *et al.* (1978) were devised. These and other media, until recently, concentrated on the common thermophilic species, particularly *C. jejuni* subsp. *jejuni*, *C. coli* and *C. lari* (Corry *et al.* 1995a), incubating at 42° or 43°C. These methods are often not effective for isolating other species. For instance, *H. pullorum* does not always grow on mCCDA (modified cefoperazone charcoal deoxycholate agar) or many other popular *Campylobacter* isolation media, particularly those containing polymyxin, and requires a microaerobic atmosphere including hydrogen (Burnens *et al.* 1994; Atabay *et al.* 1996, 1998a). It could also be mistaken for *C. coli* (Burnens *et al.* 1994; Melito *et al.* 1999). Use of the membrane filter method of Steele and McDermott (1984), which relies on the ability of the target organisms to penetrate through a 0·45 or 0·65 μm pore membrane filter, allows many of the less well-known species to be isolated without the use of selective media. For example, blood agar can be used. The limiting factor is that numbers of organisms in the region of 10^4–10^5 per ml of initial suspension must be present (Moreno *et al.* 1993). Also incubation for up to 8 d, and an incubation temperature of 37°C enables higher recoveries of species such as *C. fetus* subsp. *fetus*, *C. jejuni* subsp. *doylei* or *Arcobacter* spp. This method has been used successfully to isolate *H. pullorum* from chicken caecal contents (Atabay and Corry 1997; Atabay *et al.* 1998b). Several selective media have also recently been devised for the isolation of *Arcobacter* spp. (Collins *et al.* 1996a; De Boer *et al.* 1996; Lammerding *et al.* 1996; Atabay and Corry 1997, 1998; Corry 1997; Corry and Atabay 1997; Johnson and Murano 1999a, b; Corry *et al.* 2001).

When examining carcass rinses or intestinal contents of poultry for thermophilic campylobacters, which often contain large numbers, it is frequently not necessary to enrich (Furanetto *et al.* 1991; Koenrad *et al.* 1996); in fact, there are indications that some strains may predominate over others after enrichment, giving a false impression of the types or even species originally present (Koenrad *et al.* 1996; Dr R. Madden, pers. comm.). For instance, when examining carcass rinses using an enrichment medium with CAT (cefoperazone, teicoplanin, amphotericin) supplement, we isolated thermophilic campylobacters only by direct plating, and arcobacters only after enrichment (Atabay and Corry 1997).

As with many cultural techniques, use of more than one enrichment and/or plating method enables detection of more positive samples and/or a greater variety of species (e.g. Van Etterijck *et al.* 1996). For samples from the poultry processing or farm environment, such as water, litter or surfaces, a resuscitation step can be beneficial (Corry *et al.* 1995a). This often involves delayed addition of some or all selective agents to the enrichment broth (Stern and Line 1992; Humphrey *et al.* 1995; Mason *et al.* 1999). However, pre-enrichment was not found beneficial by Mason *et al.* (1999) for examining chicken skin, even after freezing and thawing. Wet surfaces are more likely to yield campylobacters (Humphrey *et al.* 1995) since these organisms are sensitive to drying. Chlorine, which is frequently used in drinking water and in water used in abattoirs, must immediately be neutralized with thiosulphate, in order to avoid unnecessary damage to campylobacters.

The question of whether VNC (viable but non-culturable) campylobacters are able to infect poultry and/or humans has not been resolved (D. Jones *et al.* 1991; Stern *et al.* 1994; van de Giessen *et al.* 1996); neither can live, dead or VNC campylobacters be distinguished using non-culture (e.g. PCR-based) techniques on naturally contaminated samples.

5. PROPORTION OF CONTAMINATED CARCASSES AND NUMBERS PRESENT

The proportion of carcasses or raw chicken portions contaminated with thermophilic campylobacters at retail outlets or immediately postslaughter and dressing has been studied by a large number of workers and the results summarized by Bryan and Doyle (1995), Waldroup (1996) and Jacobs-Reitsma (2000). Waldroup comments that the proportion of *Campylobacter*-contaminated products seems to have increased over the past 20 years, probably because of improved methods of examination. Jacobs-Reitsma suggests that the wide variation in percent positive samples could be the result of the different methods of sampling (e.g. whole carcass rinse, 10 g or 25 g samples of meat (minus skin), surface swabs, excised skin or exudate) and examination used. Recent reports appear to show levels of 80–90% contamination in England and Wales, The Netherlands and the USA (Stern and Line 1992; Jacobs-Reitsma *et al.* 1994; Cason *et al.* 1997; Bolton *et al.* 1999), and a lower level in Sweden, Finland and Norway (Hanninen *et al.* 2000; Berndtson, pers. comm.). Stern *et al.* (1999), however, reported that the level in carcasses from USA plants using chlorinated water chilling was about 30%.

Numbers of campylobacters per bird were estimated by Hood *et al.* (1988) to be up to 1.5×10^6 for conventional carcasses and 10-fold higher for New York Dressed (uneviscerated) carcasses in England. Somewhat lower levels (10^4–10^5 per carcass) were reported from North America by Gill and Harris (1984) and Cason *et al.* (1997), and a lower level still ($\approx 10^2$ per carcass) by Stern *et al.* (1999). This could be due to the use of chilling with chlorinated water in North America rather than air chilling, which is more commonly used in Europe. Numbers on frozen

carcasses are usually lower since freezing reduces numbers by 1–2 log cycles, although the remainder seem to survive freezing for months (Oosterom *et al.* 1983b; Yogasundram and Shane 1986).

Species of thermophilic campylobacters detected are frequently not reported, but from the data in Table 1 it is evident that *C. coli* can comprise from 6 to 50% of the strains isolated. Other species occasionally reported in poultry include *C. lari*, *C. intestinalis* and *C. upsaliensis*.

6. MODE OF INFECTION AND METHODS OF CONTROL IN LIVE POULTRY

Many studies have been published on this topic, recently reviewed by Newell and Wagenaar (2000); see also Pattison (2001), in this issue. While measures against salmonella infection in poultry, including treatment of feed, biosecurity in the hatchery, in the feedmill and on the farm (e.g. careful cleaning and disinfection of houses between flocks, control of vermin, restricted access, provision of clean clothing, disinfection of footwear), salmonella-free parent and grand-parent flocks, vaccination of breeders and competitive exclusion (Mead 2000), finally appear to be succeeding in reducing levels of salmonellas in broilers (Davies *et al.* 2001), similar measures seem to be ineffective against campylobacters.

Control of infection in breeder flocks appears to have little importance, since most researchers have found no evidence that campylobacters are transmitted vertically in or on the egg (Neill *et al.* 1985; Shanker *et al.* 1986; Kasrazadeh and Genigeorgis 1987; van de Giessen *et al.* 1992; Jacobs-Reitsma 1995, 1997; Chuma *et al.* 1997). The reason for the apparent lack of vertical transmission is thought to be due to: (a) poor survival of campylobacters on egg shells and inability to multiply inside the egg except in yolk (Clark and Bueschkins 1986; Shane *et al.* 1986); (b) the low proportion of eggs either laid by infected hens, or after challenge with high numbers of campylobacters in faecal suspension, found to contain campylobacters (Doyle 1984; Shane *et al.* 1986). However, Pearson *et al.* (1996) and Cox *et al.* (1999) did find evidence, by typing strains, of low-level vertical transmission of *Campylobacter* by the breeding flocks to their progeny. If infection does take place by vertical transmission, the apparent delay of about 2 weeks before the birds become infected needs to be explained. Chuma *et al.* (1994) suggest this is due to the infection being at a very low level. They were able to detect *C. jejuni* by DNA–DNA hybridization in chicks from the day of arrival to 3 weeks of age, when it could not be detected in the same samples by conventional means. However, a later study examining Fla-types concluded that vertical transmission was unlikely (Chuma *et al.* 1997).

Table 1 Species of *Campylobacter* isolated from live broiler chickens and carcasses, and from human patients with diarrhoea

Source	*Campylobacter* spp.				Reference (country)
	C. jejuni subsp. *jejuni*	*C. coli*	*C. lari*	Others	
Live birds	90	7·6	2·6		Kazwala *et al.* 1990 (Ireland)
	66	34	–		Jacobs-Reitsma *et al.* 1994 (Netherlands)
	94	6	–		Wallace *et al.* 1997 (England)
	92	7		1	Miflin *et al.* 1999 (Australia)
	85	11		4 (*lari 'intestinalis' upsaliensis*)	Wedderkopp *et al.* 1999 (Denmark)
Carcasses	94	6	0		Lammerding *et al.* 1988 (Canada)
	97*	0·7	2·.2		
	70	30	0		Manzano *et al.* 1995 (France)
	85	7	5		Uyttendaele *et al.* (1996) (Belgium)
	50	50	–		Madden *et al.* (1998) (N. Ireland)
	59	39	2		Osano and Arimi (1999) (Kenya)
	87	8	5		Hald *et al.* 2000 (Denmark)
	45	25		29†	Flynn *et al.* (1994) (Northern Ireland)
Humans	89.5	10·3		0·2	ACMSF 1993 (England and Wales)
	94	6			Nielsen *et al.* 1997 (Denmark)
	98	2			Steinhauserova and Fojikova (1999) (Czech Republic)
	93	6·5	0·5	*jejuni* subsp. *doylei*, *fetus* subsp. *fetus*, *upsaliensis*	PHLS Campylobacter Reference Laboratory 1999, personal communication (England and Wales)

*Turkey.
†21% identified to '*Campylobacter* sp.', 8% identified as *jejuni* subsp. *doylei*, *fetus* subsp. *fetus* or '*Campylobacter cryaerophila*'.

Drinking water has sometimes been found to be the source of infection (Pearson *et al.* 1993) or a very significant risk factor (Kapperud *et al.* 1993) but water supplies are normally chlorinated and often contamination of drinking water seems to follow rather than precede infection of the flock. Similar observations have been made concerning detection of campylobacters in the air and in flies and other sources, such as feed (Rosef and Kapperud 1983; Kazwala *et al.* 1990; Berndtson *et al.* 1996a; Gregory *et al.* 1997). However, Buswell *et al.* (1998) suggested that *C. jejuni* can survive, although not multiply, in the biofilm which forms inside the water supply system; this would justify chlorination of all water, and regular thorough dismantling and cleaning of the water supply system.

In North America, litter is frequently not changed between flocks, and this does not appear to be a source of infection unless it is damp (Smitherman *et al.* 1984; Montrose *et al.* 1985; Genigeorgis *et al.* 1986; Berndtson *et al.* 1996b; Payne *et al.* 1999). Damp areas frequently occur near drinkers.

Most studies have concluded that the most important measures in preventing infection are concerned with biosecurity: particularly hygiene precautions requiring workers and visitors to disinfect or change their footwear (and other protective clothing) and wash their hands when entering the growing house (Humphrey *et al.* 1993; Kapperud *et al.* 1993; Hald *et al.* 2000). The practice of harvesting part of the flock early ('thinning') has also been identified as a risk factor (Hald *et al.* 2000), while F. Jones *et al.* (1991) thought that delayed removal of dead birds was a risk factor. The lack of convenient and rapid typing methods for distinguishing between strains has hampered investigations into modes of infection. In all published studies (e.g. Kazwala *et al.* 1990; Berndtson *et al.* 1996a) campylobacters have apparently either been detected in suspect sources *after* the broilers have become infected, implying that the broilers were the source, or, when campylobacters were isolated from the environment *before* the flock became infected, the types differed (Stern *et al.* 1997). The most important methods of control appear to be: (a) treatment (e.g. chlorination) of drinking water (if it is from a borehole or other non-public supply, or if there is a holding tank), as well as thorough dismantling and cleaning of the drinking systems between flocks; (b) hygiene measures for human workers/visitors: obligatory disinfection or, better, change of boots (and preferably other clothes) and minimization of visits; (c) control of wild birds, rodents, and flies.

Improved methods of detection would help to elucidate modes of infection. Polymerase chain reaction (PCR)-based methods undoubtedly detect more campylobacters (Studer *et al.* 1999), and it is possible to identify and type strains, but such methods do not distinguish between live and dead (or VNC) organisms.

7. CONTROL DURING TRANSPORT, SLAUGHTER AND PROCESSING

Poultry abattoirs are often called 'processing plants', possibly because slaughter, dressing and packing are carried out more like a food factory than a red meat abattoir. Birds are delivered to the plants in crates slotted into pallets on large lorries. The crates are usually made of plastic and hold up to 24 birds. Journey time plus holding time before slaughter is usually short in the UK and many other European countries (a few hours), but in North America may total 18 h without food or water. Many plants deal with 12 000 birds or more per hour, and run for 19 or 20 h per day, stopping only for cleaning and disinfection. In contrast to other food animal species, poultry are eviscerated without opening the carcass, and the skin is not normally removed. The process involves suspending the live birds by their legs on shackles on a moving line. They are stunned by electric shock and killed by bleeding. The feathers are loosened by submerging the carcasses in a bath of warm water (temperatures of 50–53°C for 'soft scald' and 58–60°C for 'hard scald'). Hard-scalding is sometimes used for carcasses that will subsequently be sold frozen as the appearance of the skin is less important, but is now less commonly used than soft scalding in both Europe and North America. The feathers are removed on a plucking machine by means of a series of rotating discs, each with several rubber fingers, and aided by copious water sprays. Together with various procedures to remove head, feet, neck, and lungs, the carcasses are eviscerated mechanically and finally chilled, packed, and then either frozen or distributed chilled. Many carcasses are further processed into portions, with or without bones and skin. Water, sometimes with high concentrations of chlorine, is used liberally to wash both carcasses and equipment at frequent intervals along the line. The most important washing point is immediately prior to chilling, when the carcasses should be washed thoroughly inside and out.

It should be noted that microbial contamination of poultry meat is largely a surface phenomenon, and as the skin is normally not removed, many of the contaminants are found on and in the skin. The pathogens, especially the campylobacters, contaminate either directly from the intestinal contents and faeces, or indirectly via equipment. The nature of the poultry processing system makes cross-contamination from *Campylobacter*-infected to *Campylobacter*-free carcasses unavoidable (Mead 1989; ICMSF 1998). Lillard (1989) summarized the means by which poultry skin was contaminated with microbes during processing. The skin absorbs water during scalding and water chilling, and from water sprays during processing. Microbes (already present or

introduced from scald or chill water, intestinal contents, faeces or contaminated machinery) adhere to the skin surface first by various physico-chemical mechanisms and later by more permanent bonds, forming a biofilm which is difficult to remove unless rinsed by clean water immediately after contamination. In particular, microbes will be present in the layer of water that is retained by the carcass after immersion scalding or chilling. Immediate rinsing after the carcass has been immersed in contaminated water will remove most contamination, but the remainder will persist and, as the skin takes up water and swells, microbes become trapped in folds and crevices of the skin, particularly in the feather follicles.

Most sampling techniques for campylobacters during processing involve carcass rinses, skin swabbing, or excising and homogenizing neck skin flaps, or other parts of the skin. Few workers have examined deep muscle or other parts of the carcass. Oosterom *et al.* (1983a) aseptically dissected carcasses from *Campylobacter*-positive flocks immediately postscalding and isolated campylobacters from 9/20 lung samples and 3/25 liver samples. Examining carcasses from *Campylobacter*-positive flocks at the end of processing, Berndtson *et al.* (1992) tested two breast and two thigh muscles from each of 85 carcasses (340 samples) and found nine samples positive, while 75% of feather follicles contained campylobacters.

7.1. Transport

Feed is usually withdrawn for about 8–12 h prior to slaughter. This reduces the volume of intestinal contents, and is thought to reduce the contamination of the carcass with intestinal contents, and hence campylobacters. However, feed withdrawal, as well as the journey from farm to abattoir, is stressful, and can increase faecal shedding of pathogens such as *Campylobacter* and *Salmonella* (Mulder 1995). Byrd *et al.* (1998) observed that feed withdrawal increased the proportion of *Campylobacter*-positive crop contents in 7/9 flocks prior to transportation, relative to the proportion of positive caecal contents. Numbers per g of caecal contents and per carcass were found by Stern *et al.* (1995) to have increased after transportation and holding for a total of 16–18 h before slaughter, compared with birds slaughtered at the farm (mean $\log_{10}$ cfu/g caecal contents 5·44 at farm vs. 6·15 at abattoir; mean 3·66 per carcass at farm vs. 7·11 at abattoir). However, increased numbers on *Campylobacter*-infected chickens may not result in increased numbers at the end of processing. Buhr *et al.* (2000) found that transport in crates with solid floors resulted in more faecal contamination on the outside of the carcasses before plucking, but that after plucking there was no significant difference in numbers of *Escherichia coli* or *Campylobacter* between carcasses transported on solid or wire flooring. The proportion of *Salmonella*-positive carcasses was also unaffected.

Transport crates are frequently not adequately washed and disinfected after use (Mead *et al.* 1994, 1995; Berndtson *et al.* 1996a; Corry *et al.* unpublished observations). Jacobs-Reitsma and Bolder (1998) observed that sometimes 'clean' crates were more often contaminated with *Campylobacter* than were dirty crates, and that birds became colonized after 4 h in naturally contaminated crates (8/10 positive oesophagus; 1/10 positive ileum and 1/20 positive caecum). Numbers on the outside of the carcass increased from $< 5 \times 10^2$ cfu to $1{\cdot}5 \times 10^4$.

7.2. Slaughter and processing

Tables 2 and 3 illustrate the effect of processing on numbers of campylobacters on carcasses at various points. In a study carried out by Oosterom *et al.* (1983a; Table 2) two processing plants were compared. Plant A used a high scald temperature and water chilling; plant B used a lower scald temperature and air chilling. The hard scald resulted in lower numbers of campylobacters after scalding and plucking, but after evisceration, numbers were similar. Water chilling reduced *Campylobacter* load significantly, while air chilling had a variable effect. Numbers on two occasions were lower after than before air chilling, and on one occasion, higher. Izat *et al.* (1988; Table 3) monitored numbers of campylobacters on carcasses in three processing plants, visiting each plant twice. All used water chilling. No information was provided on scald temperatures, nor on how much chlorine was added to the wash or chill water. Their results were similar to those obtained by Oosterom *et al.* in the plant with water chill. These results are typical of those obtained by other researchers, showing that when *Campylobacter*-infected flocks are processed, high numbers of campylobacters can be found on carcasses at all stages, as well as on the processing machinery, in chill water and in the scald water with temperature ≤ 53°C (Oosterom *et al.* 1983a; Izat *et al.* 1988; Berndtson *et al.* 1992, 1996a; Mead *et al.* 1995). Campylobacters can also often be recovered from the

Table 2 Mean numbers of campylobacters ($\log_{10}$ cfu per g pericloacal skin) from three flocks passing through each of two different abattoirs (Plant A: hard scald, water chill; Plant B: soft scald, air chill) (from Oosterom *et al.* 1983a)

After	Plant A	Plant B
Bleeding	3·08	3·16
Scalding	1·04 (58°C)	1·82 (52°C)
Plucking	1·97	2·24
Evisceration	2·54	2·45 (+ wash)
Chilling	1·35 (water)	3·73 (air)

Table 3 Mean numbers (duplicate samples from two trials) of campylobacters (log_{10} cfu per 1000 cm^2) on carcasses during processing in three different abattoirs (from Izat *et al.* 1988)

	Plant A	Plant B	Plant C
Prescald	$3{\cdot}74^{a,z}$	$3{\cdot}56^{a,z}$	$3{\cdot}03^{a,z}$
Postscald	$< 1{\cdot}26^{f,z}$	$1{\cdot}26^{e,z}$	$1{\cdot}19^{b,z}$
Postpick	$2{\cdot}37^{cd,y}$	$3{\cdot}68^{a,z}$	$2{\cdot}82^{a,y}$
Postviscera pull	$2{\cdot}84^{bc,z}$	$3{\cdot}04^{b,z}$	$3{\cdot}11^{a,z}$
Postviscera removal	$3{\cdot}12^{b,z}$	$3{\cdot}49^{a,z}$	$3{\cdot}49^{a,z}$
Prewash	$2{\cdot}83^{bc,y}$	$2{\cdot}94^{b,y}$	$3{\cdot}50^{a,z}$
Postwash	$1{\cdot}71^{ef,y}$	$2{\cdot}39^{c,yz}$	$3{\cdot}04^{a,z}$
Postchill	$1{\cdot}43^{ef,z}$	$1{\cdot}85^{d,z}$	$1{\cdot}18^{b,z}$
Prepackage (whole carcass)	$1{\cdot}92^{de,z}$	$1{\cdot}92^{d,z}$	$1{\cdot}21^{b,y}$
Prepackage (cut up)	$1{\cdot}68^{ef,z}$	$1{\cdot}69^{de,z}$	$< 1{\cdot}15^{b,z}$

[a–f]Means in the same column with no common superscripts differ significantly ($P < 0{\cdot}05$).
[y,z]Means in the same row with no common superscripts differ significantly ($P < 0{\cdot}05$).

air in the 'hanging on' area, near the plucking machine and sometimes in the evisceration and chilling areas (Oosterom *et al.* 1983a; Berndtson *et al.* 1996a). *Campylobacter*-free flocks become extensively contaminated if processed after infected flocks (Genigeorgis *et al.* 1986; Rivoal *et al.* 1999), and the proportion of *Campylobacter*-positive carcasses increases during processing (F. Jones *et al.* 1991). Cross-contamination to flocks processed the following day, after the plant has been cleaned and disinfected, has even been reported (Genigeorgis *et al.* 1986).

With such widespread contamination, it is difficult to do much to prevent cross-contamination or to minimize numbers of campylobacters on the final product from infected flocks. The best option lies either in obtaining *Campylobacter*-free flocks, or in decontaminating the final product by physical or chemical means. At first sight, use of a high scald temperature should be helpful. It does indeed result in minimal numbers of campylobacters on carcasses immediately postscald, but subsequent steps release more organisms from the intestine and recontaminate the outside of the carcass (Wempe *et al.* 1983). In addition, there is evidence that hard scalding damages the epidermis more than soft scalding, exposing tissues that allow firmer adherence of pathogens subsequently coming into contact (Slavik *et al.* 1995). Even techniques such as simultaneous scalding and plucking have not been found beneficial (Cason *et al.* 1999).

Use of chlorine in water sprays and in chill water has been found useful in reducing numbers but is not generally thought useful for eliminating campylobacters from the final product (Acuff *et al.* 1986; Bolder and van der Hulst 1987; James *et al.* 1992; Waldroup *et al.* 1992; Mead *et al.* 1995; Cason *et al.* 1997). However, Stern *et al.* (1999) found that while 52% of birds were colonized at slaughter, only 30% of their carcasses were contaminated (with $\approx 10^2$ cfu per carcass). This reduction in the rate of contamination was attributed to the chlorination of chill tank water. Addition of chlorine to chill tank water has been identified as a critical control point (CCP) in a generic Hazard Analysis Critical Control Points (HACCP) study of poultry contamination by all pathogens (McNamara 1997). A risk-analysis study by Fazil *et al.* (1999) for *Campylobacter* in fresh chicken concluded that chlorination of chill tank water can reduce the risk of foodborne *Campylobacter* infection by 25%, and that the risk of infection from the drip fluid is 2000 times greater than that from consuming the chicken.

7.3. Further processing

Dividing into portions tends to cause more cross-contamination, although, as might be expected, removing the skin reduces numbers of campylobacters (Uyttendaele *et al.* 1999). Campylobacters are more heat sensitive than most other vegetative bacteria, and inactivated relatively easily by cooking; infection is therefore most likely from handling or eating undercooked products or via cross-contamination onto food not subsequently heated (De Boer and Hahné 1990).

8. METHODS OF DECONTAMINATING RAW POULTRY

Many investigations of methods for decontaminating raw red and poultry meat have been carried out, monitoring the effect on total viable counts or coliforms or sometimes *Salmonella* or *Escherichia coli* (Corry *et al.* 1995b; James and James 1997; Genigeorgis 1999; Hinton and Corry 1999), but few refer to campylobacters. This seems to be because they tend to be more sensitive than most other vegetative bacteria to decontaminating agents, and also because they are more difficult to work with on account of their special growth requirements. Decontamination of poultry is also made more difficult because some of the organisms are trapped in the skin and/or protected by their attachment to the skin. Suitable treatments for poultry are those that can easily be inserted on existing production lines, i.e. they need to be rapid and preferably economical in terms of space and cost.

8.1. Chlorine

Although chlorination of washing and chill water (25–50 ppm) has been reported to reduce total numbers of campylobacters on carcasses, it seems that this is due to inactivation of the organisms washed off into solution, not those still attached. Chlorine should, however, reduce cross-contamination of campylobacters, as it does with salmonellas

(Yogasundram *et al.* 1987; Mead 1989; Genigeorgis 1999). As mentioned previously (section 7.2), Stern *et al.* (1999) were of the opinion that chlorination of chiller water reduced numbers of *Campylobacter*-positive carcasses, as well as numbers of campylobacters per carcass.

8.2. Chlorine dioxide and other chlorine derivatives

Chlorine dioxide has been reported by various workers (cit. Genigeorgis 1999) to be more effective than chlorine in reducing levels of bacteria on carcasses and in reducing numbers of *Salmonella*-positive carcasses when used in water chilling. It is also less affected by organic matter and raised pH and is less unpleasant for workers or corrosive to processing machinery.

Salmide™ (Bioxy Inc, Raleigh, NC, USA) is an aqueous mixture of sodium chlorite, sodium chlorate, sodium borate, sodium sulphate and hydrogen peroxide, and its active ingredients include superoxide, hypochlorite and chlorine dioxide. It was reported by Mullerat *et al.* (1994) to reduce numbers of salmonellas on poultry skin, with its activity being enhanced by EDTA. EDTA plus Salmide had a similar activity to trisodium phosphate alone (see below).

Sodium chlorite acidified with citric or phosphoric acid, used as a dip for 5 s immediately after the inside/outside wash and prior to chilling, was reported by Kemp *et al.* (2000) to reduce numbers of *E. coli* on poultry carcasses by about 2 log cycles. Citric was preferred to phosphoric acid for environmental reasons, and because campylobacters had been found to be more sensitive to the version containing citric acid (unpublished).

8.3. Organic acids

Comparison of different studies is difficult because of the variations in experimental design. Genigeorgis (1999) reviewed the literature for the effects of organic acids on microbes in general on carcasses. With respect to campylobacters, Cudjoe and Kapperud (1991) found that 1 or 2% lactic acid sprayed on carcasses at 4°C reduced numbers of inoculated campylobacters over the next few days' storage at 4°C, but did not completely eliminate them. Using 0·5% lactic acid with 0·05% sodium benzoate and inoculating chicken legs, Hwang and Beuchat (1995) obtained rather better results. However, Stern *et al.* (1985), who treated chilled naturally contaminated chickens by dipping for 50 s in 0·5% lactic acid at 50°C, found only small decreases in numbers of campylobacters, but did not investigate the effect of chilled storage on survival. These studies indicate that lower lethality may be observed when chicken is naturally contaminated rather than inoculated, that treatment at higher temperature may be more effective, and that a progressive decline in viability may occur following treatment.

8.4. Trisodium phosphate (TSP)

Trisodium phosphate is marketed by Rhone-Poulenc as Avgard™ for use in controlling *Salmonella* contamination of poultry, and few studies have been published concerning its effect on campylobacters. Significant reduction in numbers of campylobacters (≈ 1 log cycle) was reported by Slavik *et al.* (1994) following chilled storage for 1 and 6 d after tre- atment of chilled naturally contaminated chicken carcasses with 10% TSP at 50°C, but not at 10°C. Federighi *et al.* (1995) observed a mean 1·3 log reduction in numbers of campylobacters immediately after immersion of naturally contaminated carcasses (17 tests and 17 controls) in 10% TSP at room temperature. Enumeration (by MPN) was carried out immediately. The temperature of the carcasses was not stated.

Use of more dilute TSP, with other agents, such as lysozyme or nisin, has been suggested (Demelo *et al.* 1998).

8.5. Irradiation

Campylobacters are more sensitive to gamma irradiation than most vegetative Gram-negative bacteria, including salmonellas and *E. coli* O157, with *D*-values of about 0·12–0·32 kGy in chilled meat (Lambert and Maxcy 1984; Radomyski *et al.* 1994; Patterson 1995; Thayer 1995; Collins *et al.* 1996b). Irradiation decontamination of poultry, designed to eliminate salmonellas, uses 2·5–3 kGy (Mulder *et al.* 1977; ICGFI 1991; Murano 1995) and would thus be sufficient to inactivate numbers in the region of 10^9 campylobacters per carcass. Data on the radiation resistance of arcobacters are sparse, but Collins *et al.* (1996b), examining one strain of *A. butzleri* in vacuum-packed ground pork found a *D*-value of 0·27 kGy: more resistant than most campylobacters, but still relatively sensitive. Resistance in frozen foods would be significantly higher; for instance, Lambert and Maxcy (1984) found that *D*-values of *C. jejuni* in turkey meat were a mean of 0·19 kGy at 0–5°C and 0·29 at −30°C ± 10°C, but 2·5–3·0 kGy treatment would still be ample to eliminate campylobacters from all but the most grossly contaminated carcass. Irradiation has the added advantage that, unlike most other decontamination treatments, it would inactivate organisms in skin folds, crevices and feather follicles, as well as those on the skin surface. It can be applied to portions at the end of production, using an electron accelerator, or to chilled or frozen wrapped carcasses or portions at a specialist gamma irradiation plant. Mechanically deboned chicken meat is irradiated in France for use mostly by the food industry. Small quantities of irradiated

poultry are sold through a few retail outlets in the USA (Mulder 1999).

8.6. UV light

C. jejuni is more sensitive to UV (ultra violet) light than is *E. coli* or *Yersinia enterocolitica* (Butler *et al.* 1987) and UV could be used to decontaminate water (e.g. in crate washing). Evenly exposing raw meat was found difficult in early work (Haines and Smith 1933). Since then investigations on the efficiency of UV have met with varying degrees of success (James and James 1997). However, in recent work at Bristol, UV at 3·4–3·7 mW/cm^2 for 10 s reduced total counts on raw chicken by approximately two log cycles (Stephen James, pers. comm.). Ultra violet light might be applied during air chilling, but it is difficult to envisage UV treatment penetrating skin crevices and feather follicles; nor is it clear how the body cavity of carcasses could be treated on a moving line. Promising results were also obtained by Wong *et al.* (1998) with *E. coli* and *Salmonella senftenberg* on pork skin and muscle.

8.7. Heat

Various forms of heat have been suggested for decontaminating poultry and other raw meats, but most studies have targeted salmonellas, *E. coli* or total viable numbers. However, as with other decontamination methods, a number of publications indicate that campylobacters are more sensitive to heat than other Gram-negative pathogens (ICMSF 1996). Blankenship and Craven (1982) found *D*-values for *C. jejuni* ranging from 8·8 min at 51°C to 0·8 min at 57°C in ground chicken meat. Humphrey *et al.* (1984) reported a *D*-value of 62 min at 52°C for *S. typhimurium* on chicken skin. Hilton *et al.* (2000) examined the heat resistance of one strain of *A. butzleri* and found it to be less resistant than *C. jejuni*.

Hot water treatment of poultry carcasses (dipping or spraying) has been investigated by a number of workers with respect to numbers of *Salmonella* (Pickett and Miller 1966; Avens and Miller 1972; Teotia and Miller 1972; Cox *et al.* 1974; Notermans and Kampelmacher 1975a; De Ledesma *et al.* 1996). Reduction in numbers was generally considerably lower than anticipated, possibly because many of the organisms were protected by their location with respect to the microtopology of the skin and by being members of a biofilm (Notermans and Kampelmacher 1974, 1975a, b; Brown and Gilbert 1993; Genigeorgis 1999). In order to achieve significant reduction in numbers of bacteria, relatively long contact times and temperatures above 65°C were required, resulting in an unacceptable (cooked) appearance. If the only aim were to eliminate campylobacters, lesser treatments might be adequate, but it should be remembered that carcasses often carry thousands of campylobacters, compared to hundreds, or less, of salmonellas.

Other methods of applying heat include steam at atmospheric, high or reduced pressure, high intensity dry heat or microwave heating. Due to the release of latent heat, steam can transfer a large amount of heat rapidly to a surface as it condenses, and it can also penetrate small cavities, crevices and feather follicles. The main problem in applying steam decontamination to poultry carcasses is ensuring even treatment of the whole carcass, especially the internal cavity (Klose *et al.* 1971; Davidson *et al.* 1985). The process also needs to be applied on a fast-moving production line. Morgan *et al.* (1996a, b) developed a device that used very rapid cycles of high pressure steam (to heat) and vacuum (to cool) poultry carcasses. Treatment at 145°C for 25 ms reduced numbers of *Listeria innocua*, which is more heat resistant than salmonellas or campylobacters, by 4 log cycles. However, the system would be expensive to install and could not operate at the rate of modern poultry processing lines (> 6000 per h). Steam at atmospheric pressure is more promising, as the carcasses could be passed through the steam. There is, however, liable to be some residual effect on appearance (Goksoy *et al.* 2000a). Microwave heating is capable of decontaminating surfaces, but suffers from the seemingly insoluble problem of uneven heating, i.e. hot and cold spots (Goksoy *et al.* 2000b).

In summary, all methods of decontamination have disadvantages. With the exception of irradiation, treatment of air-chilled carcasses is best immediately before chilling. Water-chilled carcasses could be treated after chilling (TSP, organic acid, irradiation, heat), or in some cases during chilling (chlorine dioxide). Of the chemical methods, dipping in TSP or acidified sodium chlorite seems promising. Irradiation would be very effective, and has the added advantage of penetrating beyond the surface, but is relatively expensive and is unpopular with consumers. Heat is liable to cause changes in appearance.

9. FACTORS AFFECTING SURVIVAL OF CAMPYLOBACTERS DURING STORAGE OF FOOD

As mentioned previously, the thermophilic campylobacters require unusual conditions for growth (atmosphere with ≈ 10% carbon dioxide and 6% oxygen, temperature above 30°C and a high relative humidity or a_w). Multiplication in food or the food processing environment thus seems unlikely, at least in temperate climates. The situation for arcobacters may well be different, but has not been investigated. Circumstantial evidence suggests that arcobacters multiply in the warm, wet environment of poultry

processing plants, and possibly also in sewage and effluent. Minimum temperatures for growth are 15–25°C, so they are unlikely to multiply in refrigerated poultry, but they are able to multiply in air.

For the thermophilic campylobacters, then, with their low infective dose, the problem is how long they can survive in food, rather than how to prevent them growing. This topic has been reviewed in depth by Stern and Kazmi (1989), Park *et al.* (1991), ICMSF (1998) and Jacobs-Reitsma (2000). Various workers have found that survival in food is better at lower (e.g. 4°C) than higher (e.g. 20°C) temperatures (Svedhem *et al.* 1981; Blankenship and Craven 1982; Hanninen *et al.* 1984; Reynolds and Draughton 1987; Phebus *et al.* 1991; Curtis *et al.* 1995). Other factors that influence their survival are: pH (better at pH 6·4 than 5·8, (Gill and Harris 1982, 1983)); sodium chloride levels (worse with > 0·5% NaCl (Hanninen 1981; Doyle and Roman 1982b)); oxygen levels (better in atmospheres without oxygen at 4°C, but no difference at 21°C (Phebus *et al.* 1991)). Their sensitivity to drying is well known, although there are few specific reports besides that of Doyle and Roman (1982a) and observations made by Oosterom *et al.* (1983b) that campylobacters survive much less well on the skin of pig carcasses than on poultry carcass skin, linking this with the drier state of pig skin. There is also the possibility that the improvements in methods of isolation of sublethally injured organisms since these two publications might show that campylobacters survive better than previously thought. The study of Humphrey *et al.* (1995) on isolation from contaminated surfaces demonstrated that this was not the case: campylobacters are very unlikely to be recovered from dry surfaces. An interesting aspect of survival on surfaces is the study of Boucher *et al.* (1998), who found that survival of campylobacters stressed by aeration was enhanced by wood, due to its physical structure (pores 16 μm diameter or less). There are interesting parallels with the protective effect of chicken skin.

Apart from on their growth temperature and atmospheric preferences, information on growth/survival conditions for arcobacters is very sparse, but they seem to have similar optimum pH requirements to campylobacters, and to be more heat sensitive (Hilton *et al.* 2000).

10. CONCLUSIONS

In spite of extensive literature on the relationship between thermophilic campylobacters and poultry, it is still far from clear: (a) what proportion of human illness is caused by campylobacters originating from poultry; (b) how to prevent them colonizing broiler chickens. The answers to these questions may become clearer with the application of better and standardized methods of strain typing, as well as better methods of detection.

Most hazards to consumers could be eliminated if all carcasses were decontaminated immediately after slaughter and dressing. Ionizing radiation would be most effective because it could be applied to warm, chilled or frozen carcasses, and would affect appearance and organoleptic properties least. However, many different methods could be used, since campylobacters are generally less robust than most other pathogenic bacteria.

The other varieties of campylobacteria (*Arcobacter* spp., *H. pullorum*, *C. fetus*, *C. intestinalis* and *C. upsaliensis*) associated with poultry appear to be much less important with regard to public health, but more information concerning their ecology and effect on poultry and human health is needed.

11. ACKNOWLEDGEMENT

We are grateful to Professor G.C. Mead for his helpful comments on this manuscript.

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