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The colonization of broilers with *Campylobacter*

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Poultry is a major reservoir of Campylobacter jejuni, which is currently the leading cause of acute bacterial diarrhoea in western countries. This results in substantial accumulated economic loss due clinical costs and lost working hours. In developed countries reinforcement of hygienic practices and consumer education have so far been inadequate to significantly decrease numbers of human cases of campylobacteriosis. The control of poultry associated infection in humans may also depend upon the control of Campylobacter colonization in broiler flocks. However, sources of C. jejuni and the transmission routes through which broilers become colonized are not clearly defined. A better understanding of C. jejuni epidemiology is urgently needed. This review summarizes current theories on the epidemiology of C. jejuni broiler colonization.

Keywords: Campylobacter; colonization; epidemiology; broilers.

Introduction

Each species of Campylobacter has a favoured reservoir, Campylobacter enteritis is regarded as a food-borne disease because the natural habitat of Campylobacter species is the intestines of both domestic animals, e.g. dogs (Workman et al., 2005), livestock, e.g. pigs and poultry and wild animals, e.g. migratory birds (Griffiths and Park, 1990; Thomas et al., 1999a). C. jejuni is currently the leading cause of human foodborne gastroenteritis in developed countries and infection and can be followed by severe clinical complications, such as Guillain-Barré syndrome (GBS) (Boyd et al., 2005). Most campylobacteriosis cases are sporadic and a number of foods have been implicated as risk factors for sporadic Campylobacter infection, including barbecued meat, raw milk, bird pecked milk, bottled mineral water and poultry (ACMSF, 2004). Evidence for sporadic sources of human Campylobacter infections is mostly indirect (Corry and Atabay, 2001). Several studies have demonstrated the consumption of undercooked poultry, to be associated with human illness (Studahl and Andersson 2000), and it has been suggested that between 20% and

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40% of sporadic disease might be due to the consumption of chicken (ACMSF, 2004). While not all strains isolated from poultry are the same as those from humans, several studies, where discriminatory typing methods were used, show a close correspondence between the strains of campylobacters found in human infections and those found in chickens (Pearson *et al.*, 1993). However, consumption or occupational contact with livestock or their faeces can also actually be protective and is associated with a significant decrease in the risk of becoming ill with *Campylobacter* (Adak *et al.*, 1995). Controlling *Campylobacter* carriage in the poultry reservoir might have a measurably beneficial effect on human disease incidence (ACMSF, 2004). Despite steadily improving molecular biology identification techniques, *e.g. flu* typing; by PCR-restriction fragment length polymorphism analysis of the *flaA* and *flaB* genes, (Newell *et al., 2001),* the epidemiology of colonization remains unclear. There is an urgent need for further research in this area to help to minimize incidences of consumer *Campylobacter* infection.

Campylobacter and poultry

The ubiquitous nature of *Campylobacter* and its optimal growth in the gastrointestinal tract of poultry ensures that poultry frequently become heavily infected (Evans, 1992). Isolation of campylobacters from poultry carcasses range from 50-60% in the UK, Canada, Switzerland, the USA and up to 94% in Australia (Altekruse *et al.*, 1998; Shapton and Shapton, 1998; Wittwer *et al.*, 2005). In 2001, an extensive Food Safety Authority (FSA) *(Table* 1) survey found the UK national average of *Campylobacter* contamination to be 50% in finished raw chicken meat (FSA, 2001), and C. *jejuni* can also survive routine poultry processing techniques (Evans, 1992).

Table 1 UK regional *Campylobacter* contamination of chicken meat (FSA, 2001).

	England	Wales	Scotland	N. Ireland
Fresh	52%	47%	87%	89%
Frozen	31%	29%	35%	41%
Number of samples	2,481	800	800	800

Campylobacters in living broilers are found in highest numbers in the large intestine, caecum and cloaca (Shane, 2000; Corry and Atabay, 2001). When colonizing intestines, C. *jejuni* expresses several virulence factors, *e.g.* chernotaxis (Takata *et al.*, 1992; van Vliet and Ketley, 2001). The organism is located in the mucosal film (Shane, 2000; Corry and Atabay, 2001) as it is attracted to L-fucose, a terminal sugar of the glycoprotein constituent of mucin (Shane, 2000). Heat shock proteins (HSPs) are associated with the thermal stress response of bacteria and are important virulence factors *e.g. DnaJ* mutants are unable to colonize chickens (Konkel *et al.*, 1998; van Vliet and Ketley 2001). *Campylobacter* penetrate the mucous layer covering the intestinal cells using their polar flagella and 'cork-screw' motion (Szymanski *et al.*, 1995). Adhesion and invasion are dependent on both motility and flagellar expression, as *C. jejuni* mutants with decreased motility due to paralysed flagella show reduced adhesion and no invasion, indicating that while flagella are involved in adherence, other adhesions are involved in subsequent internalization (Yao *et al.*, 1994; van Vliet and Ketley, 2001). The outer-membrane adhesion proteins *CadF* and *PEBI* are involved in adherence and invasion. The *PEBI* protein is encoded by the *peb1A* locus, with significantly decreased adherence, invasion of HeLa cells and reduced colonization of mouse models associated with *peb1A* mutants

(Konkel *et al.,* 1997; Pei *et al.,* 1998; Ziprin *et al..* 1999; van Vliet and Ketley, 2001). *CadF* mutants are unable to bind fibronectin and colonize newly hatched leghorn chickens (Konkel *et al.,* 1997; Pei *et al.,* 1998; Ziprin *et al.,* 1999; van Vliet and Kelley, 2001). The major outer membrane constituents. lipo-oligosaccharide (LOS) and lipopolysaccharides (LPS) are involved in serum resistance, endotoxicity and adhesion (Jin *et a/. 2001;* van Vliet and Ketley, 2001). The superoxide dimutase (SOD) protein *SoaB is* the main component of the C. *jejuni* superoxide stress defence and *sodB* mutants show significantly decreased intracellular survival in human embryonic intestinal (INT-407) cells (Pesci *et al.,* 1994; Purdy and Park, 1994).

Numbers in the region of 10^5-10^9 colony forming units (CFUs) per g of intestinal contents have been commonly observed (Corry and Atabay, 2001; Newell, 2001). Poultry faecal samples may contain high concentrations of C. *jejuni* cells, *e.g.* 10^7 CFUs per g (Pearson *et al.,* 1993). Organisms may also be recovered from throughout the gastrointestinal tract, as well as the spleen and liver, indicating some systemic infection has occurred (Newell, 2001). Rapid and cross-contamination of poultry carcasses occurs during processing (Pearson *et al.,* 1996; Corry and Atabay, 2001). Experimental challenge of chicks of all ages, appears to induce an identical outcome to that observed during natural infection, where C. *jejuni* act as a commensal in this host because colonization is both extensive and asymptomatic (Newell, 2001). C. *jejuni* can be pathogenic for young birds, but is generally not so for adult birds (Pearson *et al.* 1996). Newly hatched chicks can become easily colonized, without morbidity, by single oral dosage or cloacal inoculation, or by repeated low level inoculation through drinking water (Evans, 1992; Altekruse *et al.,* 1998). For example, 1-d-old chicks orally challenged with as few as 30 CFUs of fresh isolates of C. *jejuni* can achieve caecal colonization with levels of up to 1×10^{10} CFUs per g of caecal contents within 3 d (Newell, 2001). The level and extensiveness of colonization in chick models varies according to the genetic lineage of the birds, the challenge strain and the degree of laboratory adaptation of the strain (Newell, 2001). Specific flocks that become infected, show a rapid rate of intra-house transmission and a high isolation rate from caecal swabs, water and litter (Hafez *et al.,* 2001). Within colonized flocks, the organism usually spreads so rapidly that the proportion of birds colonized is often close to 100% (Pearson *et al.,* 1993; Corry and Atabay, 2001). Coprophagy may partly explain the rapid transmission of the organism between birds, because it has been demonstrated that the virulence of invasive C. *jejuni* isolates is enhanced by passage through chicks (Evans, 1992). Once excreted into the environment, the organism does not multiply, due to its relatively high minimal growth temperature of >30°C (van de Giessen *et* al., 1996).

Most commercial chickens are colonized with C. *jejuni,* without clinical signs, around 3 weeks of age (Evans, 1992; Fields and Swerdlow, 1999; Boyd *et* al., 2005). The reasons for this delay are unclear, but may include maternal antibodies in young chicks, as most broiler-breeder flocks are *Campylobacter-positive* and anti -*Campylobacter* antibodies may be present in egg yolks, and the presence in young birds of bacterial floras antagonistic to *Campylobacter* spp. (ACMSF, 2004). In the UK, individual broiler flocks are colonized by a limited number of subtypes of C. *jejuni* (Newell *et al.,* 2001). In some, but not all cases, the same subtypes, isolated from the ceca, contaminate the end product as observed in carcass washes (Newell *et al.,* 2001).

Following prolonged environmental stresses, *e.g.* low nutrient conditions in water, certain strains of C. *jejuni* can enter a dormant coccal form, where it is no longer possible to culture the organism using conventional media (Jones *et al.,* 1991). After campylobacters are no longer culturable, electron microscopy has demonstrated that portions of cocci do not show signs of degeneration, which is also consistent with continued viability, i.e. a viable, but not culturable state (VBNC) (Jones *et al.,* 1991). The

animal gut remains the only natural site of multiplication for thermophilic campylobacters (Jones *et al.*. 1991). The epidemiological significance of VBNC *C. jejuni is* based upon the potential of the form to resuscitate and give rise to infection (Thomas *et al.,* 1999b). However, only a limited proportion of isolates produce the VBNC form (Tholozan *et al.,* 1999), suggesting that this form is not significant in transmission to poultry flocks and it is not a universal survival mechanism.

Vertical transmission

The theory of vertical transmission is a controversial issue. *Campvlobacter is* present in the reproductive tracts and semen of poultry which could lead to vertical transmission of *Campylobacter* from the hen to the chick (Cole *et al.,* 2004a; Cole *et al.,* 2004b). However, most evidence suggests that the control of infection in breeder flocks appears to have little importance, since most researchers have found no evidence that campylobacters are transmitted vertically, or in the egg (Shane, 1992; Corry and Atabay, 2001). Low C. *jejuni* isolation rates from naturally or experimentally infected eggs (Shane *et al.,* 1986; Pearson *et al.,* 1996; Sahin *et al.,* 2003), lack of egg penetration (Doyle, 1984; Shanker *et al.,* 1986; Evans, 1992), no multiplication inside eggs, except in yolks and poor survival on eggs shells (Corry and Atabay, 2001) collectively suggest that vertical transmission of *C. jejuni* through eggs is a rare event and does not play a major role in the introduction of *Campvlobacter* to chicken flocks. Shane (1992) found that faecal shedders of C. *jejuni* did not produce infected eggs. The theory of vertical transmission may be supported by the fact that the same *C. jejuni* strains may be present in both breeder flocks and their progeny *(Cox et al.,* 2002). However, this could equally be due to low-level horizontal transmission of a persistent source or reservoir external to broiler house environments, *e.g.* cattle (On *et al.,* 1998; Shreeve *et al.,* 2002). Therefore, control measures should be directed at limiting the sources of infection for broilers and reducing the rate of transmission within flocks (van de Giessen *et al.,* 1996; Evans, 1992).

Horizontal transmission

Confinement of poultry flocks is critical because many warm-blooded animals and insects may serve as vectors (Altekruse *et al.,* 1998) and broilers readily pick up *C. je juni* from the environment, so there may be many potential sources of infection (Evans, 1992; Pearson *et al.,* 1993). Flock positivity is generally higher (up to 100%) in organic and free-range flocks compared to intensively reared flocks, presumably reflecting the level of environmental exposure of such birds, as well as the increased age of the birds at slaughter (Newell and Fearnly, 2003). An elevated risk of broiler *Campvlobacter* infection is associated with the presence of other farm animals, *e.g.* cattle and sheep (Annanm-Prah and Janc 1988; Cardinale *et al.,* 2004). Cross-contamination of *Campylobacter* between sheds by contaminated air, dust, insects or litter, are possibilities (Pearson *et al.,* 1993; Whyte *et al.,* 2001). Lack of hygiene barriers, uncemented poultry-house floors (Cardinale *et al.,* 2004) dividing flocks into batches for staggered slaughter and down periods of less than 14 d all significantly increase *Campvlobacter* prevalence in broilers (Hald *et al.,* 2000; Hald *et al.,* 2001). Transportation of broilers prior to processing significantly increases *Campylobacter* colonization of the ceca and carcass contamination (Stern *et al.,* 1995; Hald *et al.,* 2001). This is probably because trucks, forklifts, pallets, crates, drivers' and catchers' boots are all potential sources of *C. jejuni* for broilers (Ramabu *et al.,* 2004).

Wild birds can acquire *Campylobacter* by feeding on raw sewage and rubbish, and can

spread these agents to humans directly or by contaminating commercial poultry operations (Reed et al., 2003). The lesser mealworm, Alphitobius diaperinus (Panzer), is a carrier of Campylobacter in poultry facilities and could acquire and harbour Campylobacter from an environmental source (Strother et al., 2005). The lesser mealworm is capable of passing viable bacteria to chickens that consume the beetle, and should be included in attempts to reduce Campylobacter prominence in poultry facilities (Strother et al., 2005).

The presence of Campylobacter in the intestinal tract implicates ingestion of a contaminated source, e.g. feeds and water (Montorse et al., 1985). Neither feeds nor fresh litter seem to be likely sources of campylobacters (Pearson et al., 1993). The moisture content of commercial feed is about 8-10% (Montorse et al., 1985). Feed is dried and pelleted, may be pasteurized and is air blown into silos (Pearson et al., 1993). C. *jejuni* is very sensitive to dehydration and dies rapidly in aerosols and the litter used on farms is generally wood shavings, which are dry and resinous, being mainly softwood and normally comes directly from sawmills (Pearson et al., 1993). Spent wood shaving litter is extremely inhibitory to the growth of salmonellas (Pearson et al., 1993). Its effect on campylobacters is less well understood, but experimental work has shown that litter artificially contaminated with campylobacters can infect chickens, under laboratory conditions (Pearson et al., 1993).

Drinking water

Drinking water has sometimes been found to be a significant source of infection, or a very significant risk factor (Kapperud et al., 1993; Pearson et al., 1993). There is evidence that low (10-10^2 per litre) numbers of campylobacters in water supplies can colonize poultry flocks (Shapton and Shapton, 1998). Although the levels of chlorination normally used for potable water would usually be considered lethal to planktonic C. jejuni, water-borne protozoa, such Tetrahymena pyrifonnis and Acanthamoeba castellanii, have strong potential to act as protective reservoirs for C. *jejuni* in the drinking water systems of intensively reared broilers (Snelling et al., 2005). In-vitro, experimental co-cultivation of C. jejuni with such protozoa can significantly reduce the susceptibility of the bacteria to chlorine (King et al., 1988) as well as to the industrial disinfectant Virudene ($p>0.05$) and can significantly ($p>0.05$) prolong the viability of Campylobacter at 25°C, the temperature at which intensively reared broilers are maintained (Snelling et al., 2005). These factors may partially explain observations by Stern et al. (2002) that chlorination of broiler drinking water had no effect on the C. jejuni colonization of broilers (Newell et al., 2003). Although no link between resistance to disinfectants and antibiotics has been observed (Avrain et al., 2003), water-borne protozoa could putatively act as protective delivery vehicles for Campylobacter (Snelling et al., 2005) with antiobiotic resistance genes, which could then be exchanged between strains (Wittwer et al., 2005).

Despite these new findings, overall evidence suggests that C. jejuni-contaminated water constitutes a low risk of colonization for broiler flocks (Newell and Fearnly, 2003). Further research is required to establish the role of water line and reservoir contamination in flock colonization and the value of water sanitizers as control measures (Newell and Feamley, 2003).

Conclusion

In several on-farm studies where horizontal transmission was suspected, and where epidemiological typing of *Campylobacter* isolates were performed, the exact source of

Campylobacter infection in broilers were not established (Jacobs-Reitsma et al., 1995; van de Giessen et al., 1996). In theory, problems like these should be addressed with rapidly evolving molecular biology methods to provide more sensitive Campylobacter detection methods and typing methods with more accuracy. However, because Campylobacter spp. are ubiquitous in the environment, cases are mostly sporadic, outbreaks are rare and due to the lack of global protocol standardization, source tracing can be difficult (Wassenaar and Newell, 2000). It is extremely difficult to identify the most suitable Campylobacter genotyping method, e.g. pulsed-field gel electrophoresis (PFGE), that fulfils all of the requirements for molecular epidemiological investigations (Wassenaar and Newell, 2000). However, these issues are now starting to be addressed by international consortia, e.g. CAMPYNET (Wassenaar and Newell, 2000) and PulseNet, to try to attain standard methods for strain comparison. This should help to give a greater insight into both the population structure and distribution of Campylobacter over the next few years.

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References

ADAK, G.K., COWDEN, J.M., NICHOLAS, S. and EVANS, H.S. (1995) The Public Health Laboratory Service national case-control study of primary indigenous sporadic cases of *Campvlobacter* infection. *Epidemiology and Infection* **115***:* 15-22.

ADVISORY COMMITTEE ON THE MICROBIOLOGICAL SAFETY OF FOOD (A.C.M.S.F.) (2004) Second Report on *Campylobacter. http://www.food.gov.uk/multimedia/pdfs/acmsfcampyloreport.pdf*

ALTEKRUSE, S.F., SWERDLOW, D.L. and STERN, N.J. (1998) *Campylobacter jejuni. Veterinary Clinics of North America-Food Animal Practice* **14**: 31-40.

ANNANM-PRAH, A. and JANC, M. (1988) The mode *of* spread of *Campylobacter jejuni* and *Campylobacter* coli to broiler flocks. *Journal of Veterinary Medicine* **35***:* 11-18.

AVRAIN L., ALLAIN L., VERNOZY-ROZAND C. and KEMPF I. (2003) Disinfectant susceptibility testing of avian and swine *Campylobacter* isolates by a filtration method. *Veterinary Microbiology* **96**: 35-40.

BOYD, Y., HERBERT, E.G., MARSTON, K.L., JONES, M.A. and BARROW, P.A. (2005) Host genes affect intestinal colonisation *of* newly hatched chickens by *Campylobacter jejuni. Immunogenetics* **57***:* 248-253.

CARDINALE, E., TALL, F., GUEYE, E.F., CISSE, M. and SALVAT, G. (2004) Risk factors for *Campvlobacter spp.* infection in Senegalese broiler-chicken flocks. *Preventive Veterinary Medicine* **64**: 15-25.

COLE, K., DONOGHUE, A.M., BLORE, P.J. and DONOGHUE, D.J. (2004a) Isolation and prevalence *of Campylobacter* in the reproductive tracts and semen of commercial turkeys. *Avian Diseases* **48**: 625-630.

COLE, K., DONOGHUE, A.M., BLORE, P.J., HOLLIMAN, J.S., COX, N.A., MUSGROVE, M.T. and DONOGHUE, D.J. (2004b) Effects of aeration and storage temperature on *Campylobacter* concentrations in poultry semen. *Poultry Science* **83**: 1734-1738.

CORRY, J.E.L. and ATABAY, H.I. (2001) Poultry as a source of *Campylobacter* and related organisms. *Journal of Applied Microbiology* **90***:* 96S-114S.

COX, N.A., STERN, N.J., HIETT, K.L. and BERRANG, M.E. (2002) Identification of a new source of *Campylobacter* contamination in poultry: from breeder hens to broiler chickens. *Avian Diseases* **46**: 535-541.

DOYLE, M. (1984) Association of *Campylobacter jejuni* with laying hens and eggs. *Applied and Environmental Microbiology* **47***:* 533-536.

EVANS, S.J. (1992) Introduction and spread of thermophilic campylobacters in broiler flocks. *The Veterinary Record* **131**: 574-576.

FIELDS, P.I. and SWERDLOW, M.D. (1999) *Campylobacter jejuni. Clinics in Laboratory Medicine* **19***:* 489504.

FOOD STANDARDS AGENCY (FSA) (2001) FSA News: UK-wide survey of Salmonella and Campylobacter contamination of fresh and frozen chicken on retail sale. http://www.foodstandards.gov.uk/news/ chikensum.htm.

GRIFFITHS, P.L. and PARK, R.W.A. (1990) Cain pylobacters associated with human diarrhoeal disease. Journal *of Applied* Bacteriology **69**:281-301.

HAFEZ, H.M., SCHROTH, S., STADLER, A. and SCHULZE, D. (2001) Detection of Salmonella. Campylobacter, and verotoxin producing E. coli in turkey flocks during rearing and processing. *Archiv fur Geflügelkunde* **65:** 130-136

HALD, B, WEDDERKOPP, A. and MADSEN, M. (2000) Thermophilic Campylobacter spp. in Danish production: a cross-sectional survey and a retrospective analysis of risk factors for occurrence in broiler flocks. Avian Pathology **29**: 123-131.

HALD, B., RATTENBORG, E. and MADSEN, M. (2001) Role of batch depletion of broiler houses on the occurrence of Campylobacter spp. in chicken flocks. Letters im Applied Microbiology **32**: 253-256.

JACOBS-REITSMA, W.F., VAN DE GIESSEN, A.W., BOLDER, N.M. and MULDER, R.W.A.W. (1995) Epidemiology of Campylobacter spp. at two Dutch broiler farms. *Epidetniologv and Infection* **114**: 413-421.

JIN, S., JOE, A., LYNETT, J., HANI, E.K., SHERMAN, P. and CHAN, V .L. (2001) JlpA. a novel surfaceexposed lipoprotein specific to Campylobacter *jejuni*, mediates adherence to host epithelial cells. Molecular Microbiology **39**: 1225-1236.

JONES, D.M., SUTCLIFFE, E.M. and CURRY, A. (1991) Recovery of viable, but non-culturable *Campylobacter jejuni.* Journal of General Microbiology **137**: 2477-2482.

KAPPERUD, G., SKJERVE, E., VIK., L., HAUGE, K., LYSAKER, A., AALMEN, 1., OSTROFF, S.M. and POTTER, M. (1993) Epidemiological investigation of risk factors for Campylobacter colonisation in Norwegian Broiler flocks. *Epidemiology and Infection* **111**: 245-255.

KING, C.H., SHOTTS, E.B., WOOLEY, R.E. and PORTER, K.G. (1988) Survival of coliforms and bacterial pathogens within protozoa during chlorination. *Applied and Environmental Microbiology* ***54***: 3023-3033.

KONKEL, M.E., GARVIS, S.G., TIPTON, S.L., ANDERSON, D.E. and CIEPLAK, W. (1997) Identification and molecular cloning of a gene encoding a fibronectin-binding protein *(CadF)* from Campylobacter jejuni. Molecular Microbiology **24**: 953-963.

KONKEL, M.E., KIM, B.J., KLENA, J.D., YOUNG, C.R. and ZIPRIN, R. (1998) Characterization of the thermal stress respopse of Campylobacter jejuni. *Infection and Immunity* **66**: 3666-3672.

MONTROSE, M.S., SHANE, S.M., and HARRINGTON, K.S. (1985) Role of litter in the transmission of Campylobacter jejuni. Avian Diseases **29**: 392-399.

NEWELL, D.G. (2001). Animal model of Campylobacter jejuni colonization and disease and the lessons to be learned from similar Helicobacter pylori models. Journal of Applied Microbiology **90**: 57S-67S.

NEWELL, D.G. and FEARNLEY, C. (2003) Sources of Campylobacter colonization in broiler chickens. Applied *and Environmental* Microbiology **69**: 4343-4351.

ON, S.L.W., NIELSEN, E.M., ENGBERG, J. and MADSEN, M. (1998) Validity of Smal-defined genotypes of Campylobacter jejuni examined by Sall, *KpnI,* and BatnHI polymorphisms: evidence of identical clones infecting humans, poultry, and cattle. *Epidemiology and Injection* **120**: 231-237.

PEARSON, A.D, GREENWOOD, M.H., HEALING, T.D., ROLLILAS, D., SHAHAMAT, M., DONALDSON, J. and COLWELL, R.R. (1993) Colonization of broiler chickens by waterborne Campylobacter *jejuni.* Applied *and Environmental* Microbiology **59**: 987-996.

PEARSON , A.D., MELODY, H., GREENWOOD, M.H., FELTHAM, R.K.A., HEALING, T.D., DONALDSON, J., JONES, D.M. and COLWELL, R.R. (1996) Microbial ecology of Campylobacter *jejuni* in a United Kingdom chicken supply chain: intermittent common source, vertical transmission, and amplification by flock propagation. Applied and *Environmental* Microbiology **62**: 4614-4620.

PEI, Z., BURUCOA, C., GRIGNON, B., BAQAR, S., HUANG, X.Z., KOPECKO, D.J., BOURGEOIS, A.L., FAUCHERE, J.L. and BLASER, M.J. (1998) Mutation in the peblA locus of Campylobacter *jejuni* reduces interactions with epithelial cells and intestinal colonization of mice. *Infection and* Immunity **66**: 93843.

PESCI, E.C., COTTLE, D.L. and PICKETT, C.L. (1994) Genetic, enzymic, and pathogenic studies of the iron superoxide dismutase of Campylobacter jejuni. *Infection and Immunity* **62**: 2687-2694.

PURDY, D. and PARK, S.F. (1994) Cloning, nucleotide sequence and characterization of a gene encoding superoxide dimutase from Campylobacter jejuni and Campylobacter coli. *Microbiology* **140**: 1203-1208.

RAMABU, S.S., BOXALL, N.S., MADIE, P. and FENWICK, S.G. (2004) Some potential sources for transmission of Campylobacter jejuni to broiler chickens. Letters im Applied *Microbiology* **39**: 252-256.

REED, K.D., MESCE, J.K., HENKEL, J.S. and SHUKLA, S.K. (2003) Birds, Migration and Emerging Zoonoses: West Nile Virus, Lyme Disease. Influenza A and Enteropathogens. Clinical *Medicine and Research* **1**: 5-12.

SAHIN, O., KOBALKA, P. and ZHANG, Q. (2003) Detection and survival of Campylobacter in chicken eggs. Journal *of Applied* Microbiology **95**:1070-1079.

SHANE, S.M., GIFFORD, D.H. and YOGASUNDRAM, K. (1986) Campylobacter *jejuni* contamination of eggs. *Veterinary Research Communication* **10**: 487-492.

SHANE, S.M. (1992) The significance of *Campylobacter jejuni* infection in poultry: A Review. *Avian pathology* **21**: 189-213.

SHANE, S.M. (2000) *Campylobacter* infection of commercial poultry. *Revue Scientifique et Technique de L'Of International des Epizooties* **19**: 376-395.

SHANKER, S. LEE, A. and SORRELL, T.C. (1986) *Campylobacterjejuni* in broilers: the role of vertical transmission. *Journal of Hygiene* **96**: 153-159.

SHAPTON, D.A. and SHAPTON N.F. (1998) Pathogenicity and pathogen profiles, in: *Principles and Practices for the Safe Processing of Foods, (D.A.* Shapton and N.F. Shapton eds), pp. 287-288, Cambridge: Woodhead Publishing Ltd.

SHREEVE, J.E., TOSZEGHY, M., RIDLEY, A. and NEWELL, D.G. (2002) The carry-over of *Campylobacter* isolates between sequential poultry flocks. *Avian Diseases* **46**: 378-385.

SNELLING, W.J., MCKENNA, J.P., LECKY, D.M. and DOOLEY, J.S.G. (2005) Survival of *Campylobacterjejuni* in water-borne protozoa. *Applied and Environmental Microbiology* **71**: 5560-5571.

STERN, N.J., CLAVERO, M.R.S., BAILEY, J.S., COX, N.A. and ROBACH, M.C. (1995) *Camp) lobacter spp. in* broilers on the farm and after transport. *Poultry Science* **74**: 937-941.

STERN, N.J., ROBACH, M.C., COX, N.A. and MUSGROVE, M.T. (2002) Effect of drinking water chlorination on *Campylobacter spp.* colonization of broilers. *Avian Diseases* **46**: 401-404.

STROTHER, K.O., STEELMAN, C.D. and GBUR, E,E. (2005) Reservoir competence of lesser mealworm (Coleoptera : Tenebrionidae) for *Campylobacter jejuni* (Campy lobacterales: Campylobacteraceae). *Journal of Medical Entomology* **42**: 42-47.

STUDAHL, A. and ANDERSSON, Y. (2000) Risk factors for indigenous *Campylobacter* infection: a Swedish case-control study. *Epidemiology and Infection* **125**: 269-275.

SZYMANSKI, C.M., KING, M, HAARDT, M. and ARMSTRONG, G.D. (1995) *Campylobacter jejuni* motility and invasion of Caco-2 cells. *Infection and Immunity* **63**: 4295-4300.

TAKATA, T., FUJIMOTO, S. and AMAKO, K. (1992) Isolation of non-chemotatic mutants of *Campylobacter jejuni* and their colonization of the mouse intestinal tract. *Infection and Immunity* **60**: 3596-3600.

THOLOZAN, J.L., CAPPELIER, J.M., TISSIER, J.P., DELATTRE, G. and FEDERIGHI, M. (1999) Physiological characterization of viable-but-nonculturable *Campylobacter jejuni* cells. *Applied and Environmental Microbiology* **65**: 1110-1116.

THOMAS, C., HILL, D.J. and MABEY, M. (1999a). Evaluation of the effect of Temperature and nutrients on the survival of *Campylobacter spp. in* water microcosms. *Journal ofApplied Microbiology* **86**: 1024-1032.

THOMAS, C., GIBSON, H., HILL, D.J. and MABEY, M. (1999b) *Campylobacter* epidemiology: an aquatic perspective. *Journal ofApplied Microbiology* **85**: 168S-177S.

VAN DE GIESSEN, A.W., HEUVELMAN, C.J. and ABEE, T. (1996) Experimental studies on the infectivity of non-culturable forms of *Campylobacter spp. in* chicks and mice. *Epidemiology and Infection* **117***:* 463-470.

VAN VLIET, A.H. and KETLEY, J.M. (2001) Pathogenesis of enteric *Campylobacter* infection. *Symposium Series Society for Applied Microbiology* **30**: 45S-56S.

WASSENAAR T.M. and NEWELL D.G. (2000) Genotyping of *Campylobacter spp. Applied and Environmental Microbiology* **66**: 1-9.

WHYTE, P., COLLINS, J.D., MCGILL, K., MONAHAN, C. and O'MAHONY, H. (2001) Distribution and prevalence of airborne microorganisms in three commercial poultry processing plants. *Journal of Food Protection* **64**: 388-391.

WITTWER, M., KELLER, J., WASSENAAR, T.M., STEPHAN, R., HOWALD, D., REGULA, G. and BISSIG-CHOISAT, B. (2005) Genetic diversity and antibiotic resistance patterns in a *Campylobacter* population isolated from poultry farms in Switzerland. *Applied and Environmental Microbiology* **71***:* 28402847.

WORKMAN, S.N, MATHISON, G.E. and LAVOIE, M.C. (2005) Pet Dogs and Chicken Meat as Reservoirs of *Campylobacter spp. in* Barbados. *Journal of Clinical Microbiology* **43**: 2642-2650.

YAO, R., BURR, D.H., DOIG, P., TRUST, T.J., NIU, H. and GUERRY, P. (1994) Isolation of motile and nonmotile insertional mutants of *Campylobacter jejuni:* the role of motility in adherence and invasion of eukaryotic cells. *Molecular Microbiology* **14**: 883-893.

ZIPRIN, R.L., YOUNG, C.R., STANKER, L.H., HUME, M.E. and KONKEL, M.E. (1999) The absence of cecal colonization of chicks by a mutant of *Campylobacter jejuni* not expressing bacterial fibronectin-binding protein. *Avian Diseases* **43**: 586-589.