Intensification of cattle production increases the risk of zoonotic campylobacteriosis

Vanselow, B.A.[1], Hornitzky, M.A.[2], and Bailey, G.D.[3]
[1]NSW Department of Primary Industries, Beef Industry Centre, UNE, Armidale NSW 2351, Australia
[2]NSW Department of Primary Industries, Regional Veterinary Laboratory, Orange, NSW 2800, Australia
[3]NSW Department of Primary Industries, Elizabeth Macarthur Agricultural Institute, Camden NSW 2570, Australia

Abstract

Campylobacter (including *C. jejuni and C. coli*) is the leading bacterial cause of acute diarrhoea in humans in many industrialised countries. It is also the most common pathogen associated with Guillain-Barré syndrome. Poultry have long been recognised as the main source of *Campylobacter*, but cattle-derived organisms are being increasingly detected in human isolates through the use of phenotypic and genotypic methodology.

An Australian study of 475 slaughter-age cattle and sheep from 19 herds and flocks showed the highest prevalence of *Campylobacter* in feedlot cattle, compared with dairy cattle, pastured cattle and sheep. Wet conditions and animal density were considered to be risk factors. Two of the four feedlots tested had over 70% of animals shedding *Campylobacter*, and studies in other countries have also found similarly high levels. In Australia the number of cattle being finished in feedlots prior to slaughter is increasing, nearly trebling in the decade since 1992 and accounting for 30% of total Australian adult cattle slaughtered. As this number increases so will the zoonotic potential of *Campylobacter* being spread to humans.

Fresh feedlot cattle manure must be considered contaminated and should not enter water supplies or contaminate vegetable and fruit crops. Farm workers also need to be aware of the risk of direct contact with manure or contaminated boots and clothing. Manure is viewed as a valuable fertiliser, but needs to be depleted of pathogens by proper composting or other means.

Introduction

Bacterial pathogens associated with human gastro-intestinal infection may be present in the production animals and therefore be potential sources of contamination. *Campylobacter* is the leading bacterial cause of acute diarrhoea in man in many industrialised countries including Australia (Nachamkin et al. 1992; Stafford et al. 1996; Altekruse et al. 1999) In England the annual incidence of human disease from *Campylobacter jejuni/ coli* has been reported as 1100 per 100,000 (Wallace RB, 1997). It is typically a sporadic disease with no person-to-person transmission. There is acute diarrhoea lasting up to 5 days (with/without blood) and fever. A reactive arthritis develops in 1% of patients at 7 to 10 days and lasts for weeks to months. *Campylobacter* is the most common pathogen associated with the occurrence of Guillain-Barré syndrome (Smith, 1995; Hahn, 1998; Hadden and Gregson, 2001), a serious debilitating condition of an acute polyradiculoneuropathy, causing ascending paralysis. *C. jejuni* and *C. coli*, are the causative agents of *Campylobacter* food-poisoning, with *C. jejuni* most commonly isolated. *C. jejuni* and *C. coli* are almost identical in behaviour and epidemiology (Butzler and Oosterom, 1991) and therefore our discussions relating to *C. jejuni* apply to both organisms. *C. jejuni* is part of the natural intestinal flora of a wide range of birds and animals (Altekruse et al. 1999) and can be pathogenic in these species. Avian species are believed to be the predominant natural hosts of *C. jejuni* because their core temperature is higher than mammals at 42ºC, which is also the optimum growth temperature for *C. jejuni* (Stanley and Jones, 2003). *Campylobacter* appear to have evolved as a commensal organism in the avian gut (Manning et al. 2003) and poultry have long been thought to be the predominant source of human

Campylobacter infection. Transmission of *Campylobacter* to humans is usually via faecal contamination of food and water (Wallace RB, 1997), and common sources are poultry, unpasteurised milk, untreated water and contact with domestic pets (Frost, 2001). Campylobacteriosis is more frequently associated with the consumption of poultry than red meat (Gill and Harris, 1984; Wallace RB, 1997). This is apparently due to the higher prevalence in birds compared to mammals, and because the processing of chicken carcasses results in a wet surface where the bacteria are able to survive, compared with the dry environment on chilled mammalian carcasses. The Public Health Laboratory Network in the United Kingdom conducted an extensive survey of red meat sold at retail outlets and demonstrated that contamination by *Campylobacter* was low (overall 1.6% positive of 6169 samples tested) and when red meat was contaminated, the number of contaminating organisms present was generally very low. This is in sharp contrast to a study of poultry meat where more than 50% of carcasses were contaminated at the point of sale (Nachamkin et al. 1992).

There is increasing evidence worldwide that non-poultry sources of human clinical infection have been previously underestimated, and that *Campylobacter* can be a significant environmental contaminant. Increasingly, cattle production is being implicated in human outbreaks (Clark et al. 2003).

As part of a larger project investigating Shiga toxin-producing *Escherichia coli* and *Salmonella* in cattle and sheep, a "snapshot" study of 19 properties was undertaken to ascertain the prevalence of *Campylobacter* (*C. jejuni* and *C. coli*), *Listeria* (*L. monocytogenes* and *L. ivanovii*) and *Yersinia* (*Y. enterocolitica*) in faeces from slaughter-age animals in NSW and Queensland (Bailey et al. 2003). Our aim was to test animals that were about to be slaughtered but had not yet left the farm. This paper describes our findings in relation to *Campylobacter*, the changing cattle industries in Australia and discusses the worldwide findings implicating cattle manure as a source of environmental *Campylobacter*.

Materials and Methods

Property Selection

Nineteen commercial cattle and sheep properties in NSW and Queensland were selected to cover all production systems producing red meat: six dairy cattle properties, four feedlot-beef cattle properties, four pasture-beef cattle properties, two prime-lamb properties and three mutton-sheep properties (Table 1). These properties were a subset of 215 properties from a larger research project, in which properties were selected with and without a history of *Salmonella* in the preceding two years. Of the 19 properties in this study, nine had a history of *Salmonella*. There was no selection in relation to a history of *Campylobacter spp.* in this study.

Animal Selection and Sampling

From each property, 25 animals were selected at random from those meeting the following criteria: all animals were within one month of expected slaughter date or of equivalent age; grazing animals were fresh off pasture and sampled within four hours of yarding and not yarded overnight; feedlot cattle were on feed for a minimum of 60 days; dairy cattle were greater than four years old and in the last 100 days of a lactation cycle. Groups of 25 animals available for slaughter from the selected properties were sampled and this sample size was considered large enough to demonstrate potential significant differences. At least 2 g of faeces was collected per rectum (using a new sterile glove for each animal), and placed in individually numbered sterile specimen containers. Specimens were transported chilled to arrive at the laboratory within 24 hours of collection.

Data Collection

Statistical analysis was done using Genstat. Questionnaires for each production system were prepared in Epi Info 6.04 and analysed by Chi-square analysis (Centers for Disease Control and

Prevention, Atlanta, Georgia, USA). They were designed to identify possible risk factors associated with the excretion of the bacterial pathogens and included approximately 100 questions under the following headings: *Property management, Environment, Management of sampled animals, Access to manures, Nutrition and feed, Water, Health status* – animal and human. The questionnaire was completed by the producer and veterinarian.

Laboratory Testing

Campylobacter culture

Faeces (1.0 g) was inoculated into 10 mL Preston Selective Enrichment Broth (Oxoid), incubated at 42^{o}C for 48 hours in a microaerophilic atmosphere (Campy*Gen*, Oxoid or a gas mixture consisting of 5% O_2, 10%CO_2, 85%N_2), then subcultured (10 µL) onto Preston *Campylobacter* Selective Agar (Oxoid) plates which were incubated as described above.

Identification of Campylobacter spp.

Up to three suspect colonies (based on characteristic morphological appearance) were subcultured. Isolates were considered to be *Campylobacter spp*. if they were oxidase positive, motile and Gram stained smears of suspect colonies revealed small tightly coiled spiral organisms. Isolates were identified as *C. jejuni* or *C. coli* as described by Barrow *et al*. (1993)

Results

The results and prevalence rates of *Campylobacter*, in each of the 19 herds and flocks sampled are summarised in Table 1 and Figure 1.

From the 19 herds or flocks, *Campylobacter spp.* were found in all production systems and 73.7% (14/19) of all herds and flocks tested. *C. jejuni* was isolated from all production systems and one isolation of *C. coli* was made from one dairy cow. Within individual properties there was an apparent higher prevalence in cattle than in sheep, with *Campylobacter* being most commonly isolated from feedlot cattle. The median prevalences and ranges were: for dairy cattle, 6% (0 – 24%), feedlot-beef cattle, 58% (12 - 92%) and pasture-beef cattle, 2% (0 – 52%), mutton sheep, 0% (0 – 4%) and prime lambs 8%. Log Linear modelling identified significantly ($P < 0.05$) higher numbers of *Campylobacter* positive isolates in feedlot-beef cattle faecal samples than all other production systems.

For the four feedlots sampled, two factors, stocking density and weather conditions, were identified from the questionnaires as possible contributors to the number of animals shedding *Campylobacter*. Feedlot-beef property 4, which had the lowest prevalence of *Campylobacter*, also had the lowest cattle density and was the only feedlot where dry weather conditions prevailed. Of the pastured animals, dairy cattle, pasture-beef cattle, mutton sheep and prime lambs, dairy cattle had the highest stocking rates and also the highest prevalence of *Campylobacter*. Of the four pasture-beef properties sampled, property 1 had higher levels of *Campylobacter* (56% of samples positive) than the other pasture-beef properties (0-4% of samples positive). From the questionnaire, a further possible risk factor was identified: because of drought conditions, the beef cattle on property 1 had been grazed near the house septic tank absorption trench. No other property reported that sampled animals had grazed near an absorption trench. Because of the limited nature of this survey, results from the questionnaires were not proven to be statistically significant associations, but have been reported as possible associations.

Three of the six dairies and one of the four pasture beef properties reported diarrhoea in the family or workers at the property in the two months prior to collecting the cattle faecal samples. No causative agent was identified for any of these human cases. The three dairies, but not the pasture

beef property were detected to have animals shedding *Campylobacter*, but there was no statistically significant association with the human illness reported.

Table 1 Prevalence of *Campylobacter* in cattle and sheep from 19 herds or flocks, based on testing 25 faecal samples per property

Prod type & property no.	Date	District	Approximate stocking rate (DSE/ hectare)	Approximate stocking rate No. head/m^2	*Campylobacter* Isolations (%)	Total number of positive samples/total samples	P^+
Feedlot beef 1	5/08/98	Southern Qld		0.09	92	23/25	
" " **2**	16/07/98	Central NSW		0.09	76	19/25	
" " **3**	21/07/98	South-east Qld		0.10	40	10/25	
" " **4**	22/04/98	Southern NSW		0.06	12	3/25	
					Median 58 **Mean 55** **95% CI 57.3**	**55/100**	
Dairy cattle 1	1/06/98	Northern NSW	24		24	6/25	
" " **2**	26/08/98	Southern NSW	20		24	6/25	
" " **3**	2/06/98	Northern NSW	23		8	2/25	
" " **4**	12/05/98	Central NSW	7		4	1/25	
" " **5**	26/05/98	Southern NSW	10		4*	1/25	
" " **6**	13/05/98	Southern NSW	20		0	0/25	
					Median 6 **Mean 10.7** **95% CI 11.2**	**16/150**	**<0.001**
Pasture beef 1	28/10/98	Southern NSW	2		56	14/25	
" " **2**	4/08/98	Western NSW	1		4	1/25	
" " **3**	5/08/98	Western NSW	1		0	0/25	
" " **4**	28/10/98	Southern NSW	3		0	0/25	
					Median 2 **Mean 15** **95% CI 43.6**	**15/100**	**<0.001**
Mutton sheep 1	7/05/98	South-west NSW	1		4	1/25	
" " **2**	13/05/98	South-west NSW	1		0	0/25	
" " **3**	10/06/98	Central NSW	3		0	0/25	
					Median 0 **Mean 1.3** **95% CI 5.7**	**1/75**	**<0.001**
Prime lambs 1	11/08/98	Southern NSW	3		8	2/25	
" " **2**	30/06/98	Central NSW	4		8	2/25	
					Median 8 **Mean 8** **95% CI 0**	**4/50**	**<0.001**

DSE = Dry Sheep Equivalent
Y = Yes, N = No, CI = Confidence Interval
* = *Campylobacter coli*, all other *Campylobacter* isolates were *Campylobacter jejuni*
P^+ = P value determined by log linear modelling comparing total number of *Campylobacter* positive and negative samples for each production system compared with Feedlot Beef.

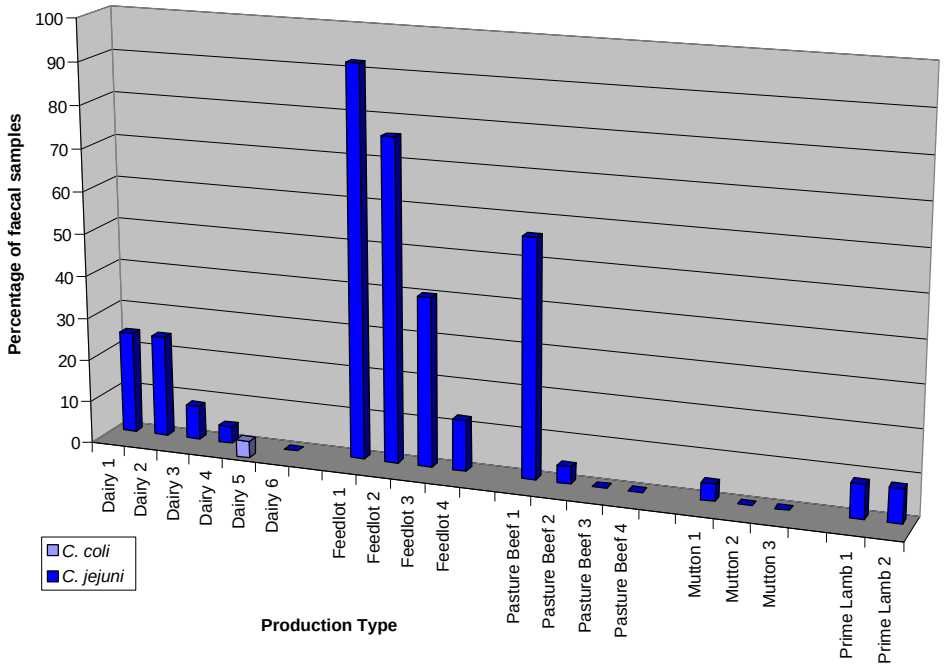

Figure 1 Percentage of faecal samples positive for *Campylobacter*

Feedlot Cattle Numbers in Australia

The feedlot industry in Australia has grown rapidly over 2 decades with numbers nearly tripling since 1992 (Figure 2) (MLA, 1999; MLA, 2002; MLA, 2004)and currently 30% of Australian beef cattle are finished in feedlots (MLA, 2004).

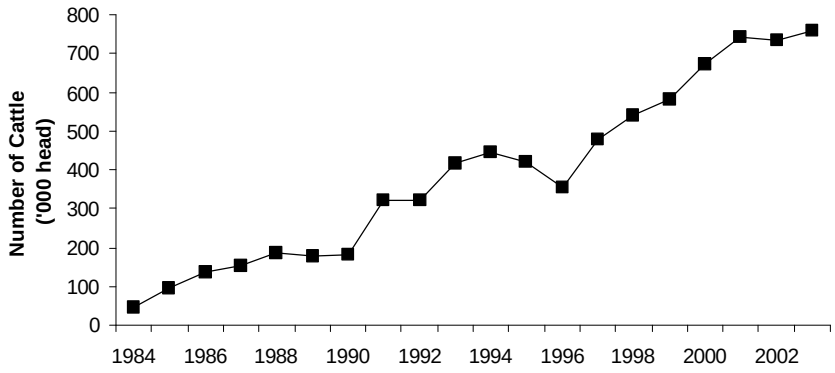

Figure 2 The growth of numbers of cattle in Australian feedlots

Discussion

C. jejuni and C. coli are bacterial pathogens that cause enteritis in humans. In our study, *C. jejuni* was commonly isolated and there was a higher prevalence in cattle than in sheep. This study

demonstrated a difference between cattle from different production systems, with feedlot cattle having a significantly higher prevalence than either dairy cattle or pasture-beef cattle.

From the results of the questionnaires, high stocking density and wet weather were identified as possible contributors to the number of feedlot animals shedding *Campylobacter*. Both these factors would increase the level of moisture in the pen and encourage the survival of the organisms. In addition to these possible contributing factors, feedlot rations are high in carbohydrate and therefore may provide a suitable environment in the gastrointestinal tract for *Campylobacter* to survive and proliferate. The higher stocking rate in dairies, compared with other grazing cattle and sheep, was identified as a possible risk factor for *Campylobacter* prevalence. Three pasture-beef properties had none or low levels of *Campylobacter*, but one property had a prevalence of 56%. For this property, a possible risk factor of cattle grazing near the house septic tank-absorption trench, was identified. Fluid from the trench may have been a source, although, as with many of the properties in this study, cats and dogs were also present, so the proximity to dog and cat faeces may have been a risk factor.

It is interesting to note that, from the results of the questionnaire, diarrhoea in humans was recorded at three of the six dairy properties, one of three pasture-beef properties, but not from the other production systems. The causative agents for the diarrhoea in humans were unknown and there was no statistically significant association between diarrhoea in humans and any of the pathogens isolated from animals on the same property. Nonetheless, it was found that the three dairy properties with cases of human diarrhoea also had animals shedding *Campylobacter*. This organism is the most common bacterial cause of diarrhoea in humans, and in Canadian studies, Thompson (Thompson et al. 1986) found a strong association between human campylobacterioisis and living on a farm. Our report of diarrhoea in dairy workers warrants further investigation. The management of dairy cattle exposes the workers to cattle faeces much more commonly than in any other production system.

Studies from other countries report a wide variation of *Campylobacter* carriage rate in domestic food producing animals. This may reflect the different geographic/climatic conditions, and management practices (Nachamkin et al. 1992). New Zealand abattoir studies in both dairy cattle and sheep demonstrated higher prevalence rates than our study: New Zealand dairy cattle had isolation rates for *C. jejuni* or *C. coli* from rectal swabs of 24%, 31% and 12% during summer, autumn and winter respectively (Meanger and Marshall, 1989), while New Zealand sheep had prevalence rates of 2.4% for lambs and 14% for adult sheep (Gill and Harris, 1982). In the study of New Zealand dairy cattle, approximately half of the isolates were *C. jejuni* and the other half, *C. coli*. Interestingly, we only isolated *C. coli* from one animal, a dairy cow, in our study. Dairy calves have been shown to be high shedders of *Campylobacter* (Stanley et al. 1998). An abattoir study in Australia by Grau (1988) found *C. jejuni* in 54% calf faecal samples and 12.5% of cow faecal samples and also observed that lot-fed cattle were more likely to have *C. jejuni* in their intestinal tracts and on their carcasses than pasture-fed cattle. Garcia et al. (1985) also found a high incidence of *C. jejuni* in cattle raised in feedlots compared with cattle on pasture. In a Canadian study of 60 beef steers in a simulated feedlot setting it was found that 100% of the steers monitored over 4 months shed *Campylobacter*, and a high percentage were chronic shedders of large quantities of *Campylobacter* (Inglis et al. 2004) In a small study of a *Campylobacter* positive UK dairy herd it was concluded that a small proportion of the herd may be shedding high numbers of *Campylobacter* at any one time (Stanley and Jones, 2003). Our "snapshot" study demonstrated that feedlot cattle had a statistically higher ($P < 0.05$) number of animals shedding *Campylobacter* than pastured beef cattle, dairy cattle, mutton sheep or prime lambs.

Campylobacter prevalence has been reported to be seasonal, with both humans and animals (Altekruse et al. 1999; Meanger and Marshall, 1989) having higher levels during the warmer

months. Our study was conducted in the cooler months of the year (between May and October), so we could anticipate higher levels during warmer months. The animals in this study were healthy animals still on-farm, but about to leave the farm for slaughter. We did not study the confounding effects of transport to the abattoir, and the time in lairage before slaughter, nor did we study the levels of carcass contamination. Other studies indicate that the chilling and dessication of carcasses at the abattoir may limit the carriage of *Campylobacter* on meat (Wallace RB, 1997). The prevalence of *Campylobacter* on retail red meat in Australia is not available although in the UK low levels have been detected. It could be expected that bovine livers carry *Campylobacter* as observed by Saito et al. (2005).. In a UK study, lambs liver, ox liver and pigs liver had 72.9, 54.2 and 71.7 % positive for *Campylobacter* (Kramer et al. 2000).

Worldwide there is growing evidence of the significance of cattle as a source of human *Campylobacter* infection. The greatest risk to humans from *Campylobacter* in cattle is through manure contaminating water, contaminated unpasteurised milk and direct contact with cattle or manure (Stanley and Jones, 2003). *Campylobacter* has been commonly associated with untreated drinking water (Merritt et al. 1999) with some infections being combined with *E. coli* O157:H7, an organism commonly associated with cattle (Stanley and Jones, 2003; Vanselow et al. 2005). *Campylobacter* infection occurred in 2 Canadian communities following contamination of well-water. Through both phenotypic and genotypic methodology the more recent outbreak in Walkerton, 2000 which was both *E. coli* O157:H7 and *Campylobacter* infection, the *Campylobacter* types identified in human patients were indistinguishable from those isolated from one nearby farm. This combined with the epidemiological and hydrogeological data suggested that bacteria from cattle manure entered groundwater and contaminated a well for the town (Clark et al. 2003). Consumption of unpasteurised milk was implicated as the source of 30 out of 80 outbreaks of human campylobacteriosis reported to the Centers for Disease Control and Prevention/US (CDC) between 1973 and 1992 (Altekruse et al. 1999) and four of 21 outbreaks reported to the Communicable Disease Surveillance Centre (CDSC) from England and Wales between 1992 and 1994 (Stanley and Jones, 2003). Many studies have found that the presence of farm animals, such as cattle and sheep, on broiler farms is associated with increased risk of *Campylobacter* infection in broiler flocks (Stanley and Jones, 2003). A study of the frequency and spatial distribution of environmental *Campylobacter* spp in a 10 by 10 km square of rural Cheshire showed that humans were at risk through occupational and recreational exposure to cattle manure. *C. jejuni* was also readily isolated from waterways (Brown et al. 2004). Brown et al. (2004) postulate that direct and indirect exposures to cattle faeces may be responsible for many sporadic cases of human *Campylobacter* infection. *Campylobacter* in humans is predominantly a sporadic disease.
There is increasing evidence that non-poultry sources of human clinical infection have been previously underestimated. Nielsen et al. (1997) demonstrated an overlap between serotypes of *C. jejuni* found in humans, poultry, and cattle, indicating that poultry and cattle should be considered in the transmission to humans. Certain strains from poultry, cattle, sheep and humans are indistinguishable by various molecular subtyping methods such as Pulsed-field gel electrophoresis (PFGE), *fla* polymerase chain reaction-restriction fragment length polymorphism (PCR-RLFB) and ribotyping. As such, ruminants carry and excrete *Campylobacter* genotypes that are capable of causing disease in the local community (Stanley and Jones, 2003). Nielsen et al. (2000) identified 2 isolates from cattle and human patients that were identical using six methods of subtyping including: PFGE, *fla* –RLFB, ribotyping, random amplified polymorphic DNA typing (RAPD), denaturing gradient gel electrophoresis of *fla*A (*fla*-DGGE) and Penner heat-stable serotyping. In a Japanese study serotypic and genotypic data indicated a possible link between sporadic human campylobacteriosis and *C. jejuni* from bovine bile and faeces (Saito et al. 2005).

Intensive cattle industries such as feedlots and dairies have stored manure or slurry which can be a potential source of *Campylobacter* to pathogen vectors such as birds, rodents and flies. These

vectors can reinfect cattle. Contaminated drinking water has been implicated in cattle shedding *Campylobacter* (Humphrey and Beckett, 1987).

Campylobacter are susceptible to dessication and do not survive well on chilled carcasses in an abattoir. This is good news re consumption of beef, but we must not overlook the role of cattle in zoonotic transmission by direct contact with cattle or manure or through contaminated water and milk. Feedlots and dairies in particular are likely to have a high level of *Campylobacter* in manure. Fresh feedlot cattle manure must be considered contaminated and should not enter water supplies or contaminate vegetable and fruit crops. Farm workers also need to be aware of the risk of direct contact with manure or contaminated boots and clothing. Manure is viewed as a valuable fertiliser, but needs to be depleted of pathogens by proper composting or other means.

As the number of feedlots increases along with increasing human populations and urban encroachment into rural areas in Australia, so will the risk of zoonotic transmission of *Campylobacter* to humans through direct contact with cattle or manure or contaminated run-off entering human water supplies. The geographical siting of feedlots, dairies and abattoirs must be such that any water run-off does not enter human water supplies. Any water supplies at any risk of contamination must be carefully monitored and chlorinated.

Conclusions

- Large numbers of intensively reared cattle in feedlots shed *Campylobacter*.
- High stocking density and moisture suspected as risk factors.
- Three-fold increase in the number of cattle in feedlots in Australia since 1992.
- 30% of Australian beef cattle finished in feedlots.
- Increasing evidence worldwide of the significance of cattle as a source of human campylobacterioisis.
- The risk to humans is not through meat but through environmental contamination and direct contact with animals and manure.

Acknowledgments

This work was funded by Meat and Livestock Australia and NSW Department of Primary Industries (previously NSW Agriculture). Valuable assistance was given by John Cronin, Queensland Department of Primary Industry, Keith Walker, Steven Hum, Graeme Eamens, Paul Gill of NSW Department of Primary Industries, Rural Lands Protection Boards of NSW veterinarians and staff, and property owners.

References

Altekruse, S.F., Stern, N.J., Fields, P.I. and Swerdlow, D.L. (1999) *Campylobacter jejuni* - An emerging foodborne pathogen. *Emerging Infectious Diseases* 5 (1):28-35.

Bailey, G.D., Vanselow, B.A., Hornitzky, M.A., Hum, S.I., Eamens, G.J., Gill, P.A., Walker, K.H. and Cronin, J.P. (2003) A study of the foodborne pathogens: *Campylobacter*, *Listeria* and *Yersinia*, in faeces from slaughter-age cattle and sheep in Australia. *Communicable Diseases Intelligence* 27 (2):249-257.

Barrow, G., Cowan, S., Steel, K. and Feltham, R. (1993) *Cowan and Steel's Manual for the Identification of Medical Bacteria.*, Cambridge; New York: Cambridge University Press.

Brown, P.E., Christensen, O.F., Clough, H.E., Diggle, P.J., Hart, C.A., Hazel, S., Kemp, R., Leatherbarrow, A.J.H., Moore, A., Sutherst, J., Turner, J., Williams, N.J., Wright, E.J. and French, N.P. (2004) Frequency and spatial distribution of environmental *Campylobacter*

spp. *Applied and Environmental Microbiology* 70 (11):6501-6511.
Butzler, J.P. and Oosterom, J. (1991) *Campylobacter*: pathogenicity and significance in foods. *International Journal of Food Microbiology* 12 (1):1-8.
Clark, C.G., Price, L., Rafiq Ahmed, Woodward, D.L., Melito, P.L., Rodgers, F.G., Jamieson, F., Ciebin, B., Li AiMin and Ellis, A. (2003) Characterization of waterborne outbreak-associated *Campylobacter jejuni* , Walkerton, Ontario. *Emerging Infectious Diseases* 9 (10):1232-1241.
Frost, J.A. (2001) Current epidemiological issues in human campylobacteriosis. *Journal of Applied Microbiology* 90 (s6):85S-95S.
Garcia, M.M., Lior H, Stewart, R., Ruckerbauer, G., Trudel, J. and Skljarevski, A. (1985) Isolation, characterization, and serotyping of *Campylobacter jejuni* and *Campylobacter coli* from slaughter cattle. *Applied and Environmental Microbiology* 49 (3):667-672.
Gill, C.O. and Harris, L.M. (1982) Contamination of red-meat carcasses by *Campylobacter fetus* subsp. *jejuni*. *Applied and Environmental Microbiology* 43 (5):977-980.
Gill, C. and Harris, L. (1984) Hamburgers and broiler chickens as sources of human *Campylobacter* enteritis. *Journal of Food Protection* 47 (2):96-99.
Grau, F.H. (1988) *Campylobacter jejuni* and *Campylobacter hyointestinalis* in the intestinal tract and on the carcasses of calves and cattle. *Journal of Food Protection* 51 (11):857-861.
Hadden, R.D.M. and Gregson, N.A. (2001) Guillain-Barré syndrome and *Campylobacter jejuni* infection. *Journal of Applied Microbiology* 90 (s6):145S-154S.
Hahn, A. (1998) Guillain-Barré syndrome. *The Lancet* 352**:** 635-641.
Humphrey, T.J. and Beckett, P. (1987) *Campylobacter jejuni* in dairy cows and raw milk. *Epidemiology and Infection* 98 (3):263-269.
Inglis, G.D., Kalischuk, L.D. and Busz, H.W. (2004) Chronic shedding of *Campylobacter* species in beef cattle. *Journal of Applied Microbiology* 97 (2):410-420.
Kramer, J.M., Frost, J.A., Bolton, F.J. and Wareing, D.R.A. (2000) *Campylobacter* contamination of raw meat and poultry at retail sale: identification of multiple types and comparison with isolates from human infection. *Journal of Food Protection* 63 (12):1654-1659.
Manning, G., Dowson, C.G., Bagnall, M.C., Ahmed, I.H., West, M. and Newell, D.G. (2003) Multilocus sequence typing for comparison of veterinary and human isolates of *Campylobacter jejuni*. *Applied and Environmental Microbiology* 69 (11):6370-6379.
Meanger, J.D. and Marshall, R.B. (1989) Seasonal prevalence of thermophilic *Campylobacter* infections in dairy cattle and a study of infection of sheep. *New Zealand Veterinary Journal* 37 (1):18-20.
Merritt, A., Miles, R. and Bates, J. (1999) An outbreak of *Campylobacter* enteritis on an island resort, north Queensland. *Communicable Disease Intelligence* 23 (8):215-219.
MLA (1999) Statistical Review July 1998-June 1999. *Meat & Livestock Australia, Marketing Information Services, Sydney*
MLA (2002) Statistical Review July 2001-June 2002. *Meat & Livestock Australia, Marketing Information Services, Sydney*
MLA (2004) Australian Beef Industry. Production and Sale of Beef Cattle. *Meat & Livestock Australia. Australian Beef 04.3*
Nachamkin, I., Baser, M. and Tomkins, LS.E. (1992) *Campylobacter jejuni*; Current Status and Future Trends. American Society for Microbiology, Washington DC.
Nielsen, E.M., Engberg, J., Fussing, V., Petersen, L., Brogren, C.H. and On, S.L.W. (2000) Evaluation of phenotypic and genotypic methods for subtyping *Campylobacter jejuni* isolates from humans, poultry, and cattle. *Journal of Clinical Microbiology* 38 (10):3800-3810.
Nielsen, E.M., Engberg, J. and Madsen, M. (1997) Distribution of serotypes of *Campylobacter jejuni* and *C. coli* from Danish patients, poultry, cattle and swine. *FEMS Immunology and Medical Microbiology* 19 (1):47-56.
Saito, S., Yatsuyanagi, J., Harata, S., Ito, Y., Shinagawa, K. , Suzuki, N., Amano, K. and Enomoto,

K. (2005) *Campylobacter jejuni* isolated from retail poultry meat, bovine feces and bile, and human diarrheal samples in Japan: comparison of serotypes and genotypes. *FEMS Immunology and Medical Microbiology* 45 (2):311-319.

Smith, J.L. (1995) Arthritis, Guillain-Barré syndrome, and other sequelae of *Campylobacter jejuni* enteritis. *Journal of Food Protection* 58 (10):1153-1170.

Stafford, R., Tenkate, T. and McCall, B. (1996) A five year review of *Campylobacter* infection in Queensland. *Communicable Diseases Intelligence* 20: 478-482.

Stanley, K. and Jones, K. (2003) Cattle and sheep farms as reservoirs of *Campylobacter*. *Journal of Applied Microbiology* 94 (s1):104-113.

Stanley, K.N., Wallace, J.S., Currie, J.E., Diggle, P.J. and Jones, K. (1998) The seasonal variation of thermophilic campylobacters in beef cattle, dairy cattle and calves. *Journal of Applied Microbiology* 3: 472-480.

Thompson, J.S., Cahoon, F.E. and Hodge, D.S. (1986) Rate of *Campylobacter* spp. isolation in three regions of Ontario, Canada, from 1978 to 1985. *Journal of Clinical Microbiology* 24 (5):876-878.

Vanselow, B.A., Krause, D.O. and McSweeney, C.S. (2005) The Shiga toxin-producing *Escherichia coli*, their ruminant hosts, and potential on-farm interventions: a review. *Australian Journal of Agricultural Research* 56 (3):219-244.

Wallace RB (1997) *Campylobacter*. In: Hocking, A., Arnold, G., Jenson, I., Newton, K. and Sutherland, P., (Eds.) *Foodborne Microorganisms of Public Health Significance*, 5th edn. pp. 265-284. North Sydney NSW 2059: Australian Institute of Food Science and Technology Inc. NSW Branch, Food Microbiology Group]