The Paternal Effect of *Campylobacter jejuni* Colonization in Ceca in Broilers

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ABSTRACT *Campylobacter jejuni* is one of the most common causes of acute enteritis worldwide. Chickens are believed to be the main reservoir of *C. jejuni*. The role that host genetics play in resistance/susceptibility to *C. jejuni* colonization in broilers is still not clear. Day-old broilers from 2 parental lines (A and B) and their F_1 reciprocal crosses (C and D) were challenged orally with 10^5 cfu of *C. jejuni* to address the role of genetics in determining resistance/susceptibility to *C. jejuni* colonization in broilers. Cloacal swabs were collected on 6, 10, and 13 d postinoculation (dpi), and cecal contents cultured for *C. jejuni* on 7 and 14 dpi. The number of *C. jejuni* colonies in the cloacal swabs and cecal contents of each bird were recorded at each time point. Significantly fewer bacteria were found in the cecal contents from line A than B ($P < 0.05$) and cross D (A♂ × B♀) when compared with cross C (A♀ × B♂) at both 7 and 14 dpi. There was a significant correlation between *C. jejuni* counts in cloacal swabs and those in cecal contents. The results indicated that a paternal effect might be one of the important genetic factors influencing resistance to *C. jejuni* colonization in broilers.

Key words: *Campylobacter jejuni*, broiler, paternal effect, cecal colonization

2008 Poultry Science 87:1742–1747
doi:10.3382/ps.2008-00136

INTRODUCTION

Campylobacter jejuni is a serious human pathogen and is widely known as one of the major causes of diarrhea in humans worldwide (Blaser, 1997; Altekruse et al., 1999; Zilbauer et al., 2008). One to 7 million cases are reported per year in the United States (Solomon and Hoover, 1999). It is believed that chickens are the major reservoir of *C. jejuni* (Solomon and Hoover, 1999; Ahmed et al., 2002) with the consumption and mishandling of poultry and raw poultry products associated with human campylobacteriosis (Kinde et al., 1983; Oosterom et al., 1983; Anonymous, 2006).

Campylobacter jejuni contamination has been found in 88% of fresh broiler carcasses (Hopkins and Scott, 1983; USDA, 1996) as well as on chicken livers (Barot et al., 1983) and wings (Kinde et al., 1983). Chicken farms (Gao et al., 1985; Studer et al., 1999; Denis et al., 2001; Trachoo et al., 2002) and processing plants (Wempe et al., 1983; Prescott and Gellner, 1984) are the main sources of *C. jejuni* contamination. Horizontal transmission (Cawthraw et al., 1996; Pearson et al., 1996; Altekruse et al., 1999; Newell and Fearnley, 2003) has been reported, and vertical transmission of *C. jejuni* (Bang et al., 2003; Anonymous, 2005) has been postulated.

In general, there are no obvious clinical signs observed in chickens infected with *C. jejuni* (Stern et al., 1988; Dhillon et al., 2006) nor are production traits affected during infection (Dhillon et al., 2006). However, the host response to *C. jejuni* infection varied in different chicken lines (Stern et al., 1990; Boyd et al., 2005). Boyd et al. (2005) has shown that bacterial burden of *C. jejuni* in cecal contents could be influenced by a single autosomal dominant locus in an experiment using White Leghorn F_1 crosses and backcrosses. However, the genetic mechanisms controlling the resistance to *C. jejuni* colonization in broilers remain unknown. Previously, we have reported a differential innate immune response and a corresponding differential resistance to bacterial infections between 2 parental broiler lines and between their F_1 reciprocal crosses (Ferro et al., 2004; Swaggerty et al., 2005a,b). The objective of this study was to use the same parental lines and their F_1 reciprocal crosses to examine the (host) genetic effect on *C. jejuni* colonization in broilers.

Received April 1, 2008.
Accepted May 2, 2008.
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MATERIALS AND METHODS

Animals and Bacterial Strain

The chickens used in this study were described previously (Swaggerty et al., 2003b). Two parental lines A and B, and their F_1 reciprocal cross C (A♀ × B♂) and D (A♂ × B♀) were obtained from a commercial breeder company. For the 2 trials, 120 birds in each line (80 infected and 40 noninfected) were used. All chickens were raised in an isolation room (Biosafety Level 2) at the Comparative Medicine Program, Texas A&M University. The floor, feed, water, and shavings were sampled before the chickens were placed and determined to be *C. jejuni*-free. On the day of hatch, 25% of the chickens were screened and confirmed as *C. jejuni*-negative. Only water was provided to the chickens before oral inoculation. Chickens were fed ad libitum with Harkan Teklad certified laboratory chicken diet (Harlan Teklad, Madison, WI) after oral *C. jejuni* inoculation.

The *C. jejuni* strain 5088 used in the study was isolated from chicken ceca in Iceland. All animal experiments were performed according to the guidelines approved by the Institutional Animal Care and Use Committee, Texas A&M University.

Inoculation and Sampling

For the present studies, to minimize the dose influence and uncover a more accurate response to *C. jejuni* infection in all lines, a preliminary study was conducted to evaluate host response to different doses of *C. jejuni* (data not shown). Based on the study, a dose of 10^5 *C. jejuni* was used. *Campylobacter jejuni* was cultured in Bolton broth (CM0983; Oxoid, Basingstoke, UK) at 42°C for 40 h under a microaerobic environment (5% O_2, 10% CO_2, 85% N_2) and then centrifuged and diluted with PBS (pH = 7.2) to the desired optical density based on the correlation between cfu and absorbance. The actual inoculation dose was determined by direct bacteria plating. Eighty 1-d-old chickens from each line were orally inoculated with 0.5 mL of 3.6×10^5 and 3.7×10^5 cfu *C. jejuni* solution in the first and second trials, respectively, and 40 birds in each line were mock inoculated with 0.5 mL of PBS solution. Cloacal swabs were collected at 6, 10, and 13 d postinoculation (**dpi**) and dipped into 1 mL of PBS (pH = 7.2). At 7 and 14 dpi, 40 challenged chickens and 20 noninfected chickens from each line were killed by CO_2 asphyxiation and cecal contents collected, respectively.

Bacteria Culture and Counting

The cloacal swab samples were directly plated on *Campylobacter*-selective blood free agar (CM739; Oxoid) with CCDA selective supplement (SR155; Oxoid) and incubated in a microaerobic environment (5% O_2, 10% CO_2, 85% N_2) at 42°C for 44 to 48 h. The environment (floor, feed, water, and shavings) and screening samples were enriched in Bolton broth (CM0983; Oxoid) overnight and plated as described above. Cecal contents were filtered using 330 micron sterile filtra bag (Fisher Scientific, Houston, TX) and plated on CCDA agar plates using Whitley Automatic Spiral Plater (Don Whitley Scientific, Frederick, MD). The plates were counted using Protos Colony Counter (Synoptics Ltd., Frederick, MD) after 44-h incubation.

Data Analysis

Results from 2 trials were combined for statistical analysis and data presentation. The number of bacteria in cloacal swab samples were converted to a simple scoring system as follows: 1 = <20 colonies, 2 = 20 to 200 colonies, and 3 = >200 colonies. The number of bacteria in cecal contents were log-transformed $x' = \log(x + 1)$ and analyzed by SAS General Linear Model Analysis of Variance (SAS, Cary, NC). The correlation of bacterial numbers between cloacal swabs and cecal contents was analyzed by univariant analysis using SAS program (SAS). The value $P < 0.05$ was considered as significant.

RESULTS

The Number of Bacteria in Cloacal Swabs

The number of bacteria colonies in the cloacal swabs was obtained for each bird, and the average was calculated for each line on 6, 10, and 13 dpi. The percent *C. jejuni*-positive chickens and converted number of colony scoring in each line are shown in Figures 1 and 2, respectively. The number of bacterial colonies of *C. jejuni* in each line did not increase significantly until 10 dpi. Not all of the birds were determined to be *C. jejuni*-positive by 13 dpi (Figure 1). For the 2 parent lines, the percentages of positive chickens were 36.16 and 59.36% on 6 dpi, 82.13 and 98.65% on 10 dpi, and 97.56 and 100% on 13 dpi for the lines A and B, respectively (Figure 1A). For the F_1 crosses, 39.13 and 11.04% chickens were *C. jejuni*-positive on 6 dpi, 85.73 and 91.25% on 10 dpi, 100 and 100% on 13 dpi for cross C and D, respectively (Figure 1B). The percentages of positive chickens were significantly different between 2 parental lines A and B on 6 and 10 dpi (Figure 1A) and between 2 F_1 crosses on 6 dpi (Figure 1B). Less than 20 cfu (mean of converted number of bacterial colonies <1) were detected in each line on 6 dpi (Figure 2). More than 200 cfu were detected in parental line B (mean of converted number of bacterial colonies >2.06) on both 10 and 13 dpi, whereas fewer than 200 cfu were found in the line A on 10 and 13 dpi (Figure 2A). More than 200 cfu were detected in the cross D on 13 dpi and less than 200 cfu were detected in the cross C on 10 and 13 dpi (Figure 2B).

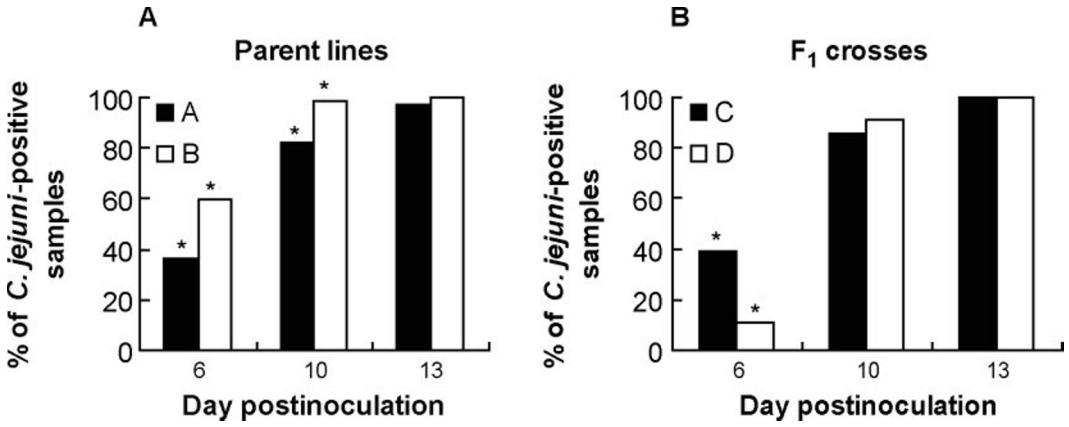

Figure 1. The percentage of *Campylobacter jejuni*-positive samples for cloacal swabs in each line on 6, 10, and 13 d postinoculation. *$P <$ 0.05.

The Number of Bacteria in Cecal Contents

The average $\log_{10}$ bacterial cfu in the cecal contents in each line was calculated and shown in Figure 3. On 7 dpi, significantly higher bacterial cfu was found in line B (3.50) than in parental line A (1.39; Figure 3A), and in F_1 cross C (1.91) than in cross D (0.31; Figure 3B; $P < 0.05$). On 14 dpi, the bacterial cfu was significantly higher in parental line B (6.19) than in line A (5.22; Figure 3A), and F_1 cross C (5.95) higher than cross D (5.43; Figure 3B; $P < 0.05$).

The Correlation of Number of Bacterial Colonies Between Cloacal Swabs and Cecal Contents

The correlation of the number of bacterial colonies between cecal contents and cloacal swabs, and within swabs and within cecal contents between different time intervals were analyzed (Table 1). Bacterial colonies of cecal contents on 7 dpi were significantly correlated with cloacal swabs on 6 and 13 dpi with corresponding correlation coefficients of 0.16 and 0.15, respectively ($P < 0.05$). Bacterial colonies of cecal contents on 14 dpi correlated with cloacal swabs on 10 and 13 dpi ($P < 0.05$) with corresponding correlation coefficients of 0.28 and 0.37, respectively. The significant correlation was found between cloacal swabs on 13 dpi and cloacal swabs on 6 dpi (0.22) and between 13 and 10 dpi (0.13; $P < 0.05$).

DISCUSSION

The number of bacteria colonizing the chicken is associated with bacterial strains, chicken lines, inoculation dose, and housing methods (floor or cage). The cecum is more susceptible to *C. jejuni* colonization than other tissues including spleen, lung, heart, and liver in the chicken (Knudsen et al., 2006). *Campylobacter jejuni* isolated from both humans and chickens are capable of colonizing chickens (Stern et al., 1990; Knudsen et al., 2006; Ringoir et al., 2007).

Day-old chickens can be colonized by as few as 2 to 100 cfu *C. jejuni* (Stern et al., 1988; Wassenaar et al., 1993; Young et al., 1999; Dhillon et al., 2006; Knudsen et al., 2006). With a higher challenge dose and a higher bacterial colonization in the cecum, a shorter latent period is observed (Stern et al., 1990; Stas et al., 1999).

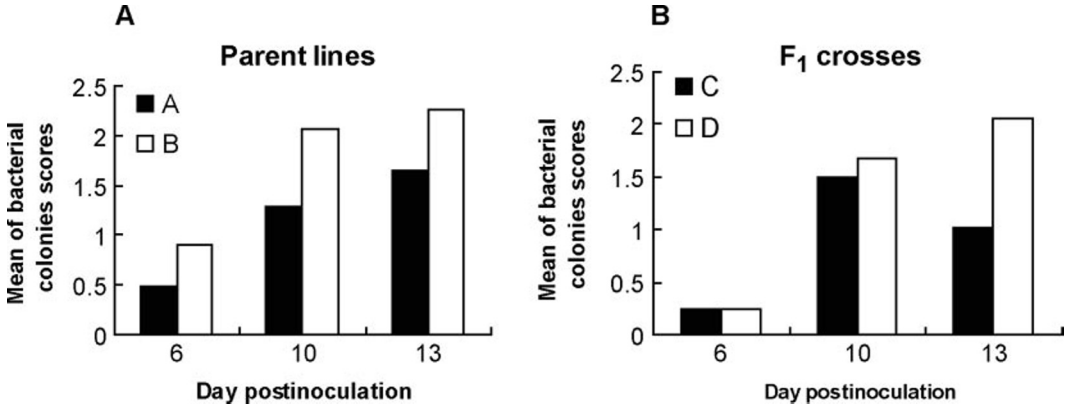

Figure 2. Bacterial colony scores of cloacal swabs in each line on 6, 10, and 13 d postinoculation. Scores: 1 = <20 colonies, 2 = 20 to 200 colonies, 3 = >200 colonies.

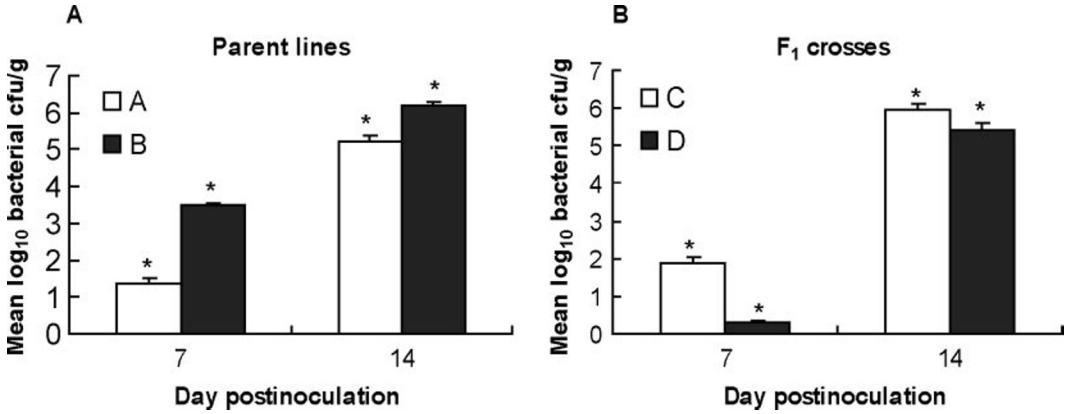

Figure 3. The mean $\log_{10}$ cfu of cecal contents ± SE in each line on d 7 and 14 postinoculation. *$P < 0.05$.

In the present study, fewer than 20 colonies were detected in cloacal swabs in all lines and $10^{3.5}$ cfu/g in cecal contents in line B at 7 dpi compared with 10^6 cfu/g 5 dpi as reported by Dhillon et al. (2006). The variations between these 2 studies may be due to the different bacterial strains and chicken lines used. Not all chickens were *C. jejuni*-positive before 7 dpi in the current study, which is contrary to previous findings (Ringoir et al., 2007). Both the frequency of *C. jejuni*-positive chickens and colonization quotient significantly increased in both parental lines and F_1 crosses after 7 dpi. Two possible reasons might explain these findings: 1) the chickens likely picked up fecal droppings from the floor leading to a secondary infection; 2) the potential horizontal transmission of *C. jejuni* between chickens. A similar experiment was conducted in cages, and the number of bacteria colonies recovered in the ceca decreased significantly after 7 dpi (X. Li and H. Zhou, Texas A&M University, unpublished data). This result suggested that the secondary infection through picking up fecal droppings from the floor was likely the major cause of *C. jejuni* colonization in chicken production, thus further demonstrating that horizontal transmission is likely a major cause in the prevalence of *C. jejuni* in broilers on poultry farms.

The number of *C. jejuni* in the cecal contents could represent real bacterial colonization in chickens. However, the birds must be sacrificed to measure bacterial colonization in cecal content (the colonization quotient), whereas cloacal swabs can be easily and repetitively collected without invasive harm to the birds. The relationship between the number of bacteria in cloacal swabs and cecal contents was calculated to evaluate if it is feasible to examine *C. jejuni* infection using cloacal swabs. In general, there was no correlation between the number of bacteria in the cecal contents and in the cloacal swabs. However, there was a strong correlation between the sampling times of cloacal swabs (6 dpi) and culture of cecal contents (7 dpi). Therefore, based on the results in the current study, cloacal swabs could provide a rough estimate of the number of bacteria in the cecum of chickens.

The chicken lines used in this study have been used for numerous *Salmonella enteritidis* and *Enterococcus gallinarum* in vivo challenge studies (Swaggerty et al., 2003b, 2004, 2005a,b, 2006a,b). To our knowledge, the 2 parental lines, A and B, are not selected for resistance to any specific pathogen. Parental line A and cross D are more responsive and resistant to *Salmonella enteritidis* and *Enterococcus* infections than line B and cross C (Ferro et al., 2004; Swaggerty et al., 2003a,b, 2005b). These trends were also observed in the present *C. jejuni* infection study. Both *Salmonella enteritidis* and *C. jejuni* are gram-negative bacteria and *Enterococcus* is a gram-positive bacterium. This common phenomenon among these 3 bacteria suggests that chickens might have similar defense systems to protect against bacterial colonization, although further studies should be considered.

There was a significant genetic effect on the resistance or susceptibility to *C. jejuni* colonization in ceca found in the current study. The resistance or susceptibility to *C. jejuni* colonization in the F_1 crosses (D and C) was associated with the sires of parental lines (A

Table 1. Correlation coefficients of the number of bacteria between cloacal swabs and cecal contents at days post oral inoculation (dpi)

Item	Swab-6 dpi	Swab-10 dpi	Swab-13 dpi	Cecal content-7 dpi	Cecal content-14 dpi
Swab-6 dpi	1.00	0.10	0.22*	0.16*	0.17
Swab-10 dpi		1.00	0.13*	0.08	0.28*
Swab-13 dpi			1.00	0.15*	0.37*
Cecal content-7 dpi				1.00	0.10
Cecal content-14 dpi					1.00

*$P < 0.05$.

and B) in the present study. In other words, the D cross (line A sire) was more resistant than the C cross (line B sire). This indicated there might be a paternal effect involved in resistance, susceptibility, or both to *C. jejuni* colonization in broilers.

Boyd et al. (2005) reported that the difference of resistance to *C. jejuni* colonization is not linked with the W chromosome and controlled by one major quantitative trait locus or gene in the autosomal chromosome. The results in the current study also provide evidence that resistance/susceptibility to *C. jejuni* colonization was not associated with genotype of K loci on Z chromosome (X. Li and H. Zhou, Texas A&M University, unpublished data). Results of F_1 reciprocal crosses indicated the sire had more influence on resistance to *C. jejuni* colonization in chickens. No maternal effect was found based on the current results. Gene imprinting could be one of the mechanisms to explain these findings. The expression of an imprinted gene depends on the parent from which that allele was inherited (Reik and Walter, 2001). Many orthologs of mammalian imprinted genes are found in chickens (Dunzinger et al., 2005, 2007), which makes it feasible to uncover this phenomenon through imprinted genes. Imprinted gene(s) from sires may be the main gene(s) that regulate resistance to *C. jejuni* infection in broilers. Further investigation on the bacterial colonization in a pedigreed sire family from both lines A and B would help understand the sire effect on *C. jejuni* colonization in broilers.

In conclusion, genetics played a significant role in resistance to *C. jejuni* colonization in chickens. The lines A and D (A♂ × B♀) were more resistant than the lines B and C (A♀ × B♂). The variation of genetic resistance should be controlled by many genes and gene networks; therefore, it is imperative to utilize high throughput microarray technology to reveal the molecular mechanism of genetic control of *C. jejuni* persistency in chickens. Determining the host response to *C. jejuni* infection among high bacterial burden (more susceptible), low bacterial burden (more resistant) and noninfected birds within lines A and B using the chicken 44K Agilent microarray (Li et al., 2008) is under way in our laboratory.

ACKNOWLEDGMENTS

This project was supported by National Research Initiative Grant no. 2007–35604–17903 from the USDA Cooperative State Research, Education, and Extension Service Animal Genome program. The authors thank Laura H. Ripley (Agricultural Research Service, USDA, College Station, TX) for help with animal care and sample collection and Cabrera Elisa (Texas A&M University, College Station) for help with autoplating. Mention of commercial products is for the sole purpose of providing specific information; not recommendation/endorsement by the USDA.

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