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The Prevalence of Antibiotic and Biocide Resistance Among *Campylobacter coli* and *Campylobacter jejuni* from Different Sources

*Ana Mavri, Marija Kurinčič and Sonja Smole Možina**

Department of Food Science and Technology, Biotechnical Faculty, University of Ljubljana, Jamnikarjeva 101, SI-1000 Ljubljana, Slovenia

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Summary

The increasing levels of antimicrobial resistance among foodborne bacteria are recognised as an important emerging public health problem. Reduced susceptibility to biocides also appears to be increasing. A potential concern is the possibility that the widespread use of biocides is responsible for the selection and maintenance of antibiotic-resistant bacteria. Here, we examine the prevalence of erythromycin, ciprofloxacin, triclosan, benzalkonium chloride, chlorhexidine diacetate, cetylpyridinium chloride, trisodium phosphate and sodium dodecyl sulphate resistance among 27 isolates of *Campylobacter coli* and 15 isolates of *Campylobacter jejuni* from food, animal, human and environmental water sources. These antimicrobial susceptibilities were determined by the broth microdilution method. In the 42 *Campylobacter* strains studied, different antibiotic resistance levels were seen. The resistance to erythromycin and ciprofloxacin was observed in 14.3 % of *Campylobacter* strains. A higher rate of erythromycin resistance and multi-resistance was observed among isolated *C. coli* than among *C. jejuni* strains. Similar situations were seen for triclosan. Conversely, the level of benzalkonium chloride resistance was higher in *C. jejuni* than in *C. coli*. No correlation between biocide and antibiotic resistance was observed. This study does not provide evidence to confirm that tolerance to biocides is connected to antibiotic resistance in *Campylobacter.*

Key words: *Campylobacter*, biocides, antibiotics, disinfectants, multidrug resistance

Introduction

The thermotolerant *Campylobacter* spp., and especially *Campylobacter coli* and *Campylobacter jejuni*, have become the most commonly reported bacterial cause of foodborne gastroenteritis in humans worldwide. Usually they are transmitted to humans by contaminated food and drinking water (*1*). As well as this widespread occurrence, *Campylobacter* spp. have become increasingly resistant to antibiotics, including macrolides, fluoroquinolones and tetracyclines. These are drugs of choice for the treatment of clinical campylobacteriosis, so this resistance greatly compromises the effectiveness of these antibiotic treatments, indicating a growing public health problem (*2*,*3*).

Biocides comprise various chemical agents that can efficiently inactivate microorganisms. They are regularly used in the food industry and in housekeeping to prevent bacterial contamination during food processing, to disinfect, sanitise and/or sterilise objects and surfaces, and to preserve materials or processes from microbiological contamination (*4*). In comparison with antibiotic resistance, the mechanisms of bacterial resistance to biocides have been described more recently and less studied overall (*5*,*6*). However, there are numerous reports of bacterial resistance to biocides, including resistance to

*Corresponding author; Phone: ++386 1 320 3751; Fax: ++386 1 256 6296; E-mail: sonja.smole@bf.uni-lj.si

triclosan, quaternary ammonium compounds, chlorhexidine and trisodium phosphate (*7–9*).

A potential concern is the possibility that mechanisms providing resistance to biocides may also provide cross-protection against the activity of antibiotics. Antibiotic resistance mechanisms, such as reduced cellular uptake, drug efflux and inactivation, and mutation at a target site, can also apply to biocides (*6,10*). A possible link between biocide and antibiotic resistances in bacteria has been reported in several studies (*11–13*). However, contrary evidence has appeared in the literature to suggest that this phenomenon does not cause a real problem in practice (*14–16*).

There is a lot of data relating to biocide resistance among many different bacteria, like *Salmonella*, *Listeria*, and *Pseudomonas* (*13,17,18*). However, little is known about biocide resistance in *Campylobacter* spp. In this study, we examine the prevalence of resistance among 27 *C. coli* and 15 *C. jejuni* isolates from food, animal, human and environmental water sources to a broad range of biocides: triclosan, benzalkonium chloride (BC), cetylpyridinium chloride (CPC), chlorhexidine diacetate (CHA) and trisodium phosphate (TSP). We also include the anionic surfactant sodium dodecyl sulphate (SDS), the microbicide with protein denaturing potency that is added to a large class of cleaning agents to assist in the cleaning (*19,20*). Due to the possibility that biocide resistance is linked to antibiotic resistance in bacteria, the prevalence of resistance to the clinically important antibiotics erythromycin and ciprofloxacin will also be examined.

Materials and Methods

Bacterial strains and growth conditions

The 27 *C. coli* and 15 *C. jejuni* strains from food, animal, human and environmental water sources used in this study were isolated and identified phenotypically and by multiplex PCR based on amplification of the aspartokinase gene in *C. coli* and the hippuricase gene in *C. jejuni*, as described previously (*3,21*). The cultures were stored at –80 °C in brain-heart infusion broth (Biolife, Milan, Italy) with 20 % horse blood (Oxoid, Hampshire, UK) and 20 % glycerol (Kemika, Zagreb, Croatia) (*22*). The isolates were cultivated at 42 °C under micro-aerophilic conditions (3 % O_2, 10 % CO_2 and 87 % N_2) in gas-tight containers on Karmali or Columbia agar, supplemented with 5 % horse blood (Oxoid). *C. coli* ATCC 33559, *C. jejuni* ATCC 33560 and NCTC 11168 were used as reference.

Antimicrobial susceptibility testing

The minimal inhibitory concentrations (MICs) of triclosan (Merck, Darmstadt, Germany), BC, CHA, CPC and TSP (Sigma-Aldrich, St. Louis, MO, USA), SDS (Invitrogen, Carlsbad, CA, USA), erythromycin (Sigma-Aldrich) and ciprofloxacin (Fluka, Buchs, Switzerland) were determined using the broth microdilution method. This was carried out in Müller Hinton broth (Oxoid) with inocula of 10^6 bacteria/mL using 96-well microtitre plates, as described previously (*23*). Ethanol (Merck) was used as the erythromycin and triclosan co-solvent, at concentrations previously shown to be non-inhibitory for bacterial cells. Twofold serial dilutions of erythromycin, ciprofloxacin, triclosan and BC were used at concentrations of 0.016 to 512 μg/mL, of CHA and CPC at 0.016 to 2 μg/mL, of SDS at 0.125 to 1024 μg/mL, and of TSP at 0.5 to 64 mg/mL. The microtitre plates were incubated for 24 h at 42 °C under microaerophilic conditions. The MICs were defined as the lowest concentration of the antimicrobial drug where no viable cells were present, and they were determined on the basis of the fluorescent signal measured by the microplate reader (Tecan, Männedorf, Switzerland) after adding 20 μL CellTiter-Blue® Reagent (Promega, Madison, WI, USA) to the culture media, following the manufacturer's instructions. The assays were repeated twice in duplicate, to confirm the reproducibility of these MIC data.

The antibiotic resistance breakpoints were defined according to the recommendations of the Clinical and Laboratory Standards Institute (*24*). The breakpoints are shown in Table 1. Multi-resistance was defined as the resistance to erythromycin and ciprofloxacin. Sensitive strains were those susceptible to erythromycin and ciprofloxacin. *C. coli* ATCC 33559, *C. jejuni* ATCC 33560 and NCTC 11168 reference strains were used as susceptibility test control.

Table 1. The range of concentrations of biocides and antibiotics tested and breakpoints for antibiotics (according to CLSI (*24*))

Biocide/antibiotic	γ	Resistance breakpoint
	μg/mL	μg/mL
triclosan	8–64	
benzalkonium chloride (BC)	0.016–4	
cetylpyridinium chloride (CPC)	0.25–4	
chlorhexidine diacetate (CHA)	0.063–2	
trisodium phosphate (TSP)	2000–16000	
sodium dodecyl sulphate (SDS)	128–1024	
erythromycin	0.125–1024	>32
ciprofloxacin	0.125–128	>4

Statistical analysis of MIC data

Statistical analyses were performed with IBM® SPSS® software v. 17.0 (IBM Corporation, Armonk, NY, USA). The MICs of antimicrobial assays were compared with the independent samples *t*-test to prove or disprove the significance of the differences in resistance between *C. coli* and *C. jejuni*, and between strains sensitive and resistant to antibiotics, as well as between sensitive and multi-resistant strains. The correlation of antibiotic and biocide MIC distributions was compared by Pearson's χ^2 test. Results were considered significant when $p \geq 0.05$. A Pearson's correlation coefficient was calculated for the correlation matrix between biocide and antibiotic MIC distributions.

Results and Discussion

In total, 42 *C. coli* and *C. jejuni* isolates from food, animal, human and environmental water sources were tested for their susceptibilities to erythromycin, ciproflox-

acin, five biocides (triclosan, BC, CHA, CPC and TSP) and an anionic surfactant SDS. The range of concentrations for antibiotics and biocides (in twofold increases) and the breakpoints for antibiotics are shown in Table 1.

With regard to antibiotic resistances of these *Campylobacter* strains, 33 out of the 42 strains (78.6 %) were susceptible and 9 out of 42 (21.4 %) were resistant to erythromycin, and half of these strains tested (51.4 %) were resistant to ciprofloxacin. The resistance to both antibiotics tested was observed in 6 out of 42 (14.3 %) tested strains. The erythromycin resistance data determined in our previous study (*23*) were in some cases different from those obtained in the present study. Instability of the erythromycin resistance phenotype was observed in three *C. coli* strains: VC7114, VC110722 and VC110725. Erythromycin resistance (MIC>512 μg/mL) was determined in strain VC7114 in our previous antimicrobial testing, while no resistance (MIC=1 μg/mL) was seen in further investigations with the same method. Additionally, previous erythromycin medium-level resistance of strains VC110722 and VC110725 dropped from 8 μg/mL to a sensitive phenotype (4 and 2 μg/mL, respectively). A similar situation was reported previously (*23*), where we reported the presence of the A2075G mutation in the 23S rRNA gene as responsible for this erythromycin resistance. No mutation has been identified in any sensitive strain, as well as in the resistant strain VC7114, which appears to have an unstable resistance phenotype. This reduction of the erythromycin resistance to a sensitive phenotype occurred only in *C. coli*. These data suggest that there is erythromycin resistance that is not mediated by the A2075G mutation in the 23S rRNA gene but by other mechanism(s), and that this might be conditionally (temporarily) induced to provide the bacterial pathogens with rapid adaptation to environmental changes. As with our findings, other studies have reported unstable phenotypes: *Campylobacter* mutants that show low-to-intermediate levels of erythromycin resistance and lack 23S rRNA mutations were not stable in culture media or on the animal host, and easily lost their resistance phenotype in the absence of macrolide antibiotics (*25–27*).

There is a lot of data available that relate to biocide resistance among different foodborne pathogenic bacteria; however, the data about biocide resistance in *Campylobacter* spp. are scarce. This study of 42 *Campylobacter* strains from different sources indicates that there is biocide resistance in *C. coli* and *C. jejuni*. Since there is neither literature data about breakpoints of resistance levels to biocides, the MICs in the *Campylobacter* spp. in the present study were compared to MICs determined in other bacteria.

In the present study, the MICs of triclosan among *C. coli* and *C. jejuni* strains ranged from 8 to 64 μg/mL. In this way, these data resemble the recently reported observations for strains of *Salmonella enterica*, where three distinct triclosan resistance phenotypes were observed: triclosan low-level resistance (MIC≥8 μg/mL), triclosan medium-level resistance (MIC=16–32 μg/mL), and triclosan high-level resistance (MIC>32 μg/mL) (*8,11,28*).

In 41 of the 42 strains tested (97.6 %), BC MICs were between 0.016 and 2.0 μg/mL. Only one human clinical multi-resistant isolate had a MIC of 4 μg/mL for BC. These data for BC susceptibility are similar to those reported for *Listeria monocytogenes* food and industrial isolates, where two BC resistance phenotypes were observed: BC sensitive with MICs of 2 μg/mL or below and BC resistant with MICs of 4 μg/mL or above (*17,29*). The same situation was observed for CPC, another biocide from the group of quaternary ammonium compounds that was tested in this study. However, no statistically significant correlation between the resistances of these tested strains to these two biocides was seen. The MIC values for triclosan, BC, CPC and CHA in our isolates were lower than those reported for *Acinetobacter*, *Citrobacter* and *Pseudomonas* industrial isolates (*15*), *Pseudomonas stutzeri* (*18*), and *Campylobacter* isolates from slaughterhouses (*30*). In these previous studies the MICs for triclosan ranged from 4 to above 100 μg/mL, the MICs for BC from 0.5 to 340 μg/mL, for CPC from 2.5 to 250 μg/mL and for CHA from 1 to 100 μg/mL. However, these other MICs were determined using the agar dilution method, which is known to give higher values than the MICs determined in broth (*29*).

The distributions of antibiotic and biocide MICs across *C. coli* and *C. jejuni* are presented in Table 2. A significant difference in erythromycin resistance was observed between *C. coli* (29.6 %) and *C. jejuni* (6.7 %) (*t*-test, p=0.019). Moreover, a higher rate of multi-resistance was found in *C. coli* (20.8 %) than in *C. jejuni* (6.7 %). Higher rates of erythromycin resistance among *C. coli* than among *C. jejuni* had been reported previously (*3,16,23,31*). A similar situation was observed for the triclosan resistance. *C. coli* strains were in general more resistant than *C. jejuni* strains (*t*-test, p=0.02). Conversely, the level of BC resistance was higher in *C. jejuni* than in *C. coli* (*t*-test, p=0.021). However, there were no significant differences among *C. coli* and *C. jejuni* strains for other tested biocides, SDS and ciprofloxacin. Cases of linked biocide and antibiotic resistances (*10,11,32*) and a lack of any such link (*14–16*) have already been reported in the literature. Resistance to the clinically important antibiotics erythromycin and ciprofloxacin in *Campylobacter* spp. might also be attributed to its active efflux (*23*), which is known to mediate multidrug resistance to antibiotics and disinfectants in bacteria (*11,12,33*). To investigate cross-resistance to antibiotics and biocides, *Campylobacter* strains with different resistance levels to erythromycin and ciprofloxacin were included in this study. The distributions of different biocide MICs across the erythromycin- and ciprofloxacin-sensitive and -resistant strains, as well as across sensitive and multi-resistant strains are presented in Table 3. The overall level of biocide resistance was not significantly different in strains with different antibiotic resistance level (*t*-test, p>0.05), with an exception of SDS. Interestingly, a higher level of SDS resistance was found in erythromycin-sensitive than in erythromycin-resistant, as well as in sensitive than in multi-resistant strains (t-test, p=0.035 and p=0.007, respectively). A Pearson's correlation matrix was calculated for two distributions of antibiotics, six distributions of biocides and SDS. A statistically significant correlation between MIC distribution of CPC and CHA (p=0.002, R_{xy}=0.47), and of CPC

Table 2. The distribution of MICs of biocides tested in *C. coli* and *C. jejuni*

Biocide/antibiotic	Distribution of MIC/%										
γ(triclosan)/(μg/mL)	8	16	32	64							
C. coli	0	14.8	59.3	25.9							
C. jejuni	13.3	33.3	46.7	6.7							
total	4.8	21.4	54.8	19							
γ(BC)/(μg/mL)	0.016	0.063	0.25	0.5	1	2	4				
C. coli	0	3.7	28.6	44.4	22.2	0	0				
C. jejuni	6.7	6.7	0	6.7	60	13.3	6.7				
total	2.4	4.8	19	31	35.7	4.8	2.4				
γ(CPC)/(μg/mL)	0.25	0.5	1	2	4						
C. coli	7.4	37	22.2	29.6	3.7						
C. jejuni	13.3	13.3	26.7	46.7	0						
total	9.5	28.6	23.8	35.7	2.4						
γ(CHA)/(μg/mL)	0.063	0.125	0.25	0.5	1	2					
C. coli	3.7	18.5	37	33.3	3.7	3.7					
C. jejuni	0	20	40	33.3	6.7	0					
total	2.4	19	38.1	33.3	4.8	2.4					
γ(TSP)/(mg/mL)	2	4	8	16							
C. coli	0	3.7	55.6	37							
C. jejuni	6.7	20	55.3	26.7							
total	2.4	9.5	54.8	33.3							
γ(SDS)/(μg/mL)	128	256	512	1024							
C. coli	3.1	55.5	25.9	14.8							
C. jejuni	6.7	40	53.3	0							
total	4.8	50	35.7	9.5							
γ(erythromycin)/(μg/mL)	0.125	0.25	0.5	1	2	4	512	1024			
C. coli	11.1	11.1	14.8	14.8	14.8	1	11.1	18.5			
C. jejuni	6.7	13.3	53.3	20	0	0	6.7	0			
total	9.5	11.9	28.6	16.6	4	1	9.5	11.9			
γ(ciprofloxacin)/(μg/mL)	0.063	0.125	0.25	1	2	4	8	16	32	64	128
C. coli	4.5	13.6	13.6	0	0	9.1	4.5	22.7	22.7	4.5	4.5
C. jejuni	0	20	13.3	13.3	6.7	6.7	20	13.3	0	0	6.7
total	2.7	16.2	13.5	5.4	2.7	8.1	10.8	18.9	13.5	2.7	5.4

Table 3. The distribution of MICs of biocides tested in antibiotic-sensitive, antibiotic-resistant and multi-resistant *Campylobacter* strains

Biocide	Distribution of MIC/%						
γ(triclosan)/(μg/mL)	8	16	32	64			
erythromycin-sensitive	3	27.3	51.5	18.2			
erythromycin-resistant	11.1	0	66.7	22.2			
ciprofloxacin-sensitive	5.6	33.3	38.9	16.7			
ciprofloxacin-resistant	5.3	10.5	57.9	26.3			
sensitive	6.7	40	40	13.3			
multi-resistant	16.7	0	66.7	16.7			
γ(BC)/(μg/mL)	0.016	0.063	0.25	0.5	1	2	4
erythromycin-sensitive	3	3	18.2	33.3	36.4	6.1	0
erythromycin-resistant	0	11.1	22.2	22.2	33.3	0	11.1
ciprofloxacin-sensitive	5.6	5.6	11.1	27.8	38.9	11.1	0
ciprofloxacin-resistant	0	5.3	21.1	26.3	42.1	0	5.3
sensitive	6.7	6.7	6.7	26.7	40	13.3	0
multi-resistant	0	16.7	16.7	16.7	33.3	0	16.7

Table 3. – continued

Biocide	Distribution of MIC/%					
γ(CPC)/(μg/mL)	0.25	0.5	1	2	4	
erythromycin-sensitive	9.1	30.3	18.2	39.4	3	
erythromycin-resistant	11.1	22.2	44.4	22.2	0	
ciprofloxacin-sensitive	16.7	33.3	5.6	44.4	0	
ciprofloxacin-resistant	5.3	15.8	42.1	36.8	0	
sensitive	13.3	33.3	6.7	46.7	0	
multi-resistant	0	16.7	66.7	16.7	0	
γ(CHA)/(μg/mL)	0.063	0.125	0.25	0.5	1	2
erythromycin-sensitive	3	18.2	33.3	39.4	3	3
erythromycin-resistant	0	22.2	55.6	22.2	0	0
ciprofloxacin-sensitive	0	16.7	33.3	44.4	5.6	0
ciprofloxacin-resistant	5.3	21.1	47.3	21.1	5.3	0
sensitive	0	13.3	46.7	33.3	6.7	0
multi-resistant	0	16.7	66.7	16.7	0	0
γ(TSP)/(mg/mL)	2	4	8	16		
erythromycin-sensitive	3	9.1	51.5	36.4		
erythromycin-resistant	0	11.1	66.7	22.2		
ciprofloxacin-sensitive	5.6	11.1	66.7	16.7		
ciprofloxacin-resistant	0	5.3	47.4	47.4		
sensitive	6.7	6.7	66.7	13.3		
multi-resistant	0	0	66.7	33.3		
γ(SDS)/(μg/mL)	128	256	512	1024		
erythromycin-sensitive	6.1	42.4	39.4	12.1		
erythromycin-resistant	0	77.8	22.2	0		
ciprofloxacin-sensitive	5.6	33.3	50	11.1		
ciprofloxacin-resistant	5.3	68.4	21.1	5.3		
sensitive	6.7	33.3	46.7	13.3		
multi-resistant	0	100	0	0		

and TSP (p=0.014, R_{xy}=0.376) was observed. Another correlation was seen for triclosan and BC (p=0.048, R_{xy}= –0.307) and for ciprofloxacin and SDS (p=0.05, R_{xy}=–0.307). No statistically significant correlation was observed between antibiotic and biocide MICs.

Conclusion

The results of our study demonstrate that there is antimicrobial resistance among thermotolerant *Campylobacter* spp. Higher rates of erythromycin resistance were found among *C. coli* than *C. jejuni* isolates, which is in agreement with previously published data (*3*,*16*,*23*). A similar situation was observed for triclosan resistance. A comparison of our data on erythromycin resistance in *Campylobacter* spp. in this and our previous study (*23*) indicates the appearance of unstable resistance to erythromycin among *C. coli* that is not mediated by mutations in 23S rRNA. Therefore, the erythromycin resistance is also mediated by other mechanism(s). As erythromycin is an antibiotic of choice for the treatment of clinical campylobacteriosis, this temporarily induced mechanism might greatly compromise the effectiveness of such antibiotic treatment, which could cause a serious public health problem. The present study does not provide evidence to confirm that tolerance to biocides is connected to antibiotic resistance in *Campylobacter* spp. Findings obtained in the present study and the reported occurrence of cross--resistance to disinfectants and antibiotics among pathogens, and to common resistance mechanisms, like non--specific active efflux (*4*,*11*,*31*,*33*), indicate the need to further monitor the prevalence of antibiotic resistance and biocide resistance of zoonotic bacteria in food, animals and humans. The data obtained in such comprehensive epidemiological studies should contribute to better traceability and understanding of the phenomenon of biocide and antibiotic resistance, and the links between them, and they would highlight the need for prudent use of antimicrobials in different areas of work, including the food production chain.

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