Epidemiological analysis of *Campylobacter* spp. Isolated from Poultry and Humans in Senegal using pulsed-field gel electrophoresis and antibiotic susceptibility

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ABSTRACT:
Aims: The main objectives of this study were to investigate the diversity of *Campylobacter* genotypes circulating in Senegal and to determine the frequency of antibiotic resistance. **Methods and Results**: Strains of *C. jejuni* isolated from poultry (n=99) and from patients (n=10) and *C. coli* isolated from poultry (n=72) were subtyped by pulsed-field gel electrophoresis (PFGE). The pulsotypes obtained after digestion by *Sma*I and *Kpn*I revealed a significant genetic diversity in both species, but without any predominant pulsotypes. However, farm-specific clones were identified in the majority of poultry houses (76.5 %). Human and poultry isolates of *C. jejuni* had common PFGE patterns. High quinolone-resistance rates were observed for *C. jejuni* (43.4 %) and *C. coli* (48.6 %) isolates obtained from poultry.
Conclusions: The results showed a genetic diversity of *Campylobacter* between farms indicating multiple sources of infection; but specific clones had the ability to colonize the broiler farms. The antimicrobial resistance patterns were not related to any specific PFGE pattern suggesting that resistance was due to the selective pressure of antibiotic usage. *Campylobacter* with similar genotypes were circulating in both human and poultry.

Keywords: *Campylobacter*, poultry, human, PFGE, antimicrobial resistance, Senegal

INTRODUCTION

Thermophilic *Campylobacter* spp. (especially *C. jejuni* and *C. coli*) are among the most important agents of human gastrointestinal infections in the developed world (Altekruse *et al.* 1999; Van de Giessen *et al.* 1998). *Campylobacter* enteritis is an also emerging public-health problem in developing countries (Gedlu and Aseffa 1996; Simango and Nyahanana 1997; Prasad *et al.* 2001) and is particularly serious for immuno-compromised individuals (Obi and Bessong 2002).

Contaminated food is a common source of human illness and consumption or handling of poultry is considered to be a major route of infection, at least in developed countries (Stern *et al.* 2001). In Senegal, *C. jejuni* and *C. coli* have been recovered from commercial poultry produced under the conditions described below (Cardinale *et al.* 2004). However, their involvement in human infections remains unknown, as does the significance of chicken meat as a vehicle of infection, since no epidemiological analysis has been made previously in that country. The Senegalese chicken industry was established only recently. The birds are reared in open-sided houses with no artificial ventilation and the material used for litter is usually wood shavings (sometimes chopped straw or shredded paper). The basic foodstuffs for the birds are imported maize and locally produced peanut cake and fish meal. Typically, each flock is vaccinated against Newcastle disease and Gumboro disease and medicated to protect it from coccidiosis. Most day-old chicks come from hatching eggs that are imported from Europe or Brazil. The birds are normally slaughtered at 40 days of age and eviscerated manually without chilling. The resultant carcasses are sold in retail shops, of which there different kinds in Dakar. Some have no shelter and are exposed to direct sunlight; the food display area and sales counter are table-like structures made from wooden boards; the meat itself is not refrigerated. Other shops are well-established commercial outlets, with an electrical supply, water available and refrigeration equipment. Such shops are purpose-built and have employees who handle and sell the food.

In attempting to trace sources of infection and routes of transmission, use has been made of phenotypic methods, such as biotyping and serotyping (Garcia *et al.* 1985; Nielsen *et al.* 1997). In practice, these methods have only limited discriminatory power and various genetic methods have been developed for sub-typing purposes (Wassenaar and Newell 2000). Pulsed-field gel electrophoresis (PFGE) has been widely recognized as a sensitive method for molecular fingerprinting of *Campylobacter* isolates (On *et al.* 1998; Nielsen *et al.* 2000).

Most cases of human enteritis from *Campylobacter* do not require antimicrobial treatment, being brief, clinically mild and self-limiting. However, complications, such as septicaemia, may occur and require treatment. Macrolides and fluoroquinolones are the most useful antimicrobial drugs for treating *Campylobacter* infections, but increasing resistance has been reported (Piddock 1995; Saenz *et al.* 2000; Engberg *et al.* 2001;). In Senegal, it was found that 34 % of *Campylobacter* isolates from poultry were resistant to ciprofloxacin (Cardinale *et al.* 2003).

The objectives of the present study were to (i) investigate the diversity of genotypes occurring on poultry farms and in broiler chicken meat in Dakar, and to compare these to clinical isolates collected from the same area; (ii) evaluate the frequency of antibiotic resistance in poultry isolates and determine whether resistance is associated with specific genotypes.

MATERIALS AND METHODS

Bacterial isolates: The poultry isolates of *C. jejuni* and *C. coli* originated from farms and retail shops and were collected between January 2000 and December 2003 in the Dakar (capital city) region. Forty broiler farms were chosen at random. These farms belonged to the modern poultry sector and all showed similar characteristics. At each farm, five broilers were slaughtered and breast-skin samples (each weighing *ca* 25 g) were taken. Farm managers were asked to complete a questionnaire about any drugs used during the life-span of the flock. In addition, 10 randomly selected retail shops were investigated and skin samples taken from five chicken carcasses in each case. The samples were analysed as described by Cardinale *et al.* (2004). Briefly, each sample was added to 225 ml of Preston broth containing Preston antibiotic supplement (Oxoid, Basingstoke, UK) and incubated at 42° C for 24 hours under microaerobic conditions (Campygen: Oxoid). The sample was then streaked onto Virion plates (Mueller-Hinton agar: Merck, Germany; Bacto agar: Difco, Detroit, USA; with 5 % defibrinated horse blood: AES Laboratoire, Combourg, France) and onto Karmali plates (Oxoid). Plates were incubated at 42° C under microaerobic conditions for 48 hours. Isolates were identified using a commercial identification method (API Campy®, bioMérieux, France) and multiplex PCR (Denis *et al.* 2001).

Ten human isolates, obtained from epidemiologically-unrelated domestic patients during 2001 and 2002, were provided by the Pasteur Institute of Dakar. These originated from faecal samples, cultured on plates of *Campylobacter* blood-free selective medium and identified as described previously.

PFGE analysis: Prior to the extraction of DNA, each isolate was cultivated at 42° C overnight on Karmali agar (AES Laboratoire) under microaerobic conditions.
DNA preparation: the bacterial lawn thus obtained was then suspended in 2.5 ml of Tris NaCl (0.01 mol.l^{-1} Tris-HCl, 1 mol.l^{-1} NaCl, pH 7.6). The cells were washed twice with 2 ml of the same medium. Each bacterial suspension was adjusted to an OD value of 1.5 at 600 nm. An agarose plug was prepared by adding 250 •l of adjusted cell suspension to 250 •l of 1 % agarose (Agarose Standard, Eurobio, France), mixing thoroughly and using 100 •l for the plug. The solidified agarose plug was then incubated in ESP lysis buffer (0.5 mol.l^{-1} EDTA, pH 9, 1 % N-lauryol Sarcosine, and 1 mg ml^{-1}). Proteinase K was inactivated by incubating the agarose plug in 2 mol.l^{-1} of aminoethyl-benzesulphonyl fluoride (Pefabloc, Boehringer, Mannheim). The plug was then washed five times with TE buffer and cut into four thin slices.

Restriction endonuclease digestion and PFGE conditions: One of the four plug slices was used for restriction endonuclease digestion in a separate reaction with 40 U of *Sma*I and *Kpn*I (Roche Molecular Biochemicals) under conditions recommended by the manufacturer, in a final volume of 100 •l. The process involved incubation for 6 h at the appropriate temperature (25°C for *Sma*I and 37°C for *Kpn*I).
PFGE was carried out using the CHEF-DRIII system (Biorad). Agarose gel (1 %) prepared in 0.5 x TBE (Tris 45 mmol.l^{-1}, boric acid 45 mmol.l^{-1}, EDTA 1 mmol.l^{-1}) was subjected to electrophoresis for 22 h at 6,6 V/cm, 14° C, with ramped pulse-times from 15 to 45 s for the first 20 h and 5 to 8 s for the two last hours for *Sma*I. Fragments generated by *Kpn*I digestion were separated by electrophoresis for 23 h at 6,6 V/cm, 14° C, with ramped pulse-times from 2 to 25 s. Macrorestriction patterns were compared with the use of BioNumerics software (Applied Maths, Sint-Martens Latem, Belgium). Similarity fingerprints were determined using the Dice coefficient and a band-position tolerance of 1 %. A hierarchic unweighted pair group method with averaging algorithm was used to generate dendrograms describing the relationship among *Campylobacter* pulsotypes; these pulsotypes were clustered at an 80%

similarity level, which corresponded to a three-band difference and to the lowest level of epidemiological relatedness, according to the guidelines given by Tenover *et al.* (1995).

Antibiotic Resistance: Several colonies of each strain were suspended in 5ml of Mueller-Hinton broth to achieve a turbidity equal to a 0.5 MacFarland standard. The suspensions were inoculated onto Mueller-Hinton agar containing 5 % sheep blood, using sterile swabs. After the application of E-test® strips (AB Biodisk, Sweden), the plates were incubated at 37° C for 48 h under microaerobic conditions. The MICs were determined by two different readers, according to the recommendations of the manufacturer.

The breakpoints were those recognised by the Antimicrobial Committee of the French Society for Microbiology (Soussy *et al.* 2000). Breakpoints for resistance susceptibility were respectively >16 $mg.l^{-1}$ for amoxicillin (Am), >16/2 $mg.l^{-1}$ for amoxicillin-clavulanic acid (Ac), >4 $mg.l^{-1}$ for erythromycin (E), >16 $mg.l^{-1}$ for nalidixic acid (Na) and >2 $mg.l^{-1}$ for ciprofloxacin (Ci).

Determination of mutations in the quinolone resistance determining region (QRDR) of the *gyrA* gene: 26 nalidixic acid- and ciprofloxacin-resistant, and two nalidixic acid-resistant and ciprofloxacin-susceptible strains of *C. jejuni* from poultry were analysed. Chromosomal DNA was extracted from each strain by boiling, as described previously (Bachoual *et al.* 2001). The oligonucleotide primers used to amplify a 220-bp QRDR-containing fragment of *gyrA* from *C jejuni* (codon 54-126) were also described previously (Kazwala *et al.* 1990). The PCR was carried out in a 50 • l mixture containing 8 • l of chromosomal DNA, 1X buffer (Promega), 2 mM of $MgCl_2$, 0.6 mM of dNTP, 50 pmol of each primer and 2.5 U of Taq DNA polymerase (Promega). PCR cycling conditions were as described previously (Kazwala *et al.* 1990), except that the annealing temperature was 50° C. PCR products were purified with a QIAquick Gel Extraction Kit (Qiagen, Hilden, Germany), for use in sequencing reactions. These were carried out with a sequencer Biosystem 3100. The sequences of both strands were determined with the BigDye Terminator Cycle Sequencing Ready Reaction Kit and an ABI PRISM 377 DNA sequencer (Applied Biosystems).

RESULTS

Campylobacter spp. were isolated from 68.4 % of the poultry samples. The contamination rate was similar at both types of test location (69 % at the farms *vs.* 66 % at the retail shops). Four farms and four retail shops harboured both *C. jejuni* and *C. coli*. The species distribution was *C. jejuni* 57.9 % and *C. coli* 42.1 %. Only ten human isolates were examined; all belonged to *C. jejuni*.

PFGE Analyses

Campylobacter jejuni: Macrorestriction with *Sma*I and *Kpn*I yielded 24 different patterns consisting of seven to 10 fragments and 25 patterns of nine to 19 fragments respectively (Fig. 1). Twenty-five pulsed-field profiles were obtained with both enzymes (Table 1). None of these patterns indicated a predominant genotype. Most of the human isolates (40 %) were grouped in pattern SJ6KJ6. The dendrogram (Fig. 1) showed several clusters but only one was significant (18 isolates).

Campylobacter coli: Digestion with restriction enzymes *Sma*I and *Kpn*I demonstrated 18 patterns consisting of 10 to 16 fragments and 17 patterns of 12 to 19 fragments respectively (Fig. 2). Nineteen pulsed-field profiles were obtained with both enzymes (Table 2). There was

no predominant genotype. The dendrogram (Fig. 2) showed several clusters that gathered together only a few isolates.

Most of the farms (76.5 %) harboured a unique genotype. Within the same species, 17.6 % of the farms exhibited different subtypes, but one genotype was predominant. Strains from the retail shops isolates were also characterized according to species and within-species subtypes. Only two retails shops exhibited a unique pulsotype.

Antibiotic resistance

Campylobacter jejuni: Thirty-eight poultry isolates (38.4 %) were susceptible to all the antibiotics tested. Forty-three poultry isolates (43.4 %) were resistant to the quinolones (Table 3). The most commonly observed resistance pattern was Am Na Ci, accounting for 22.2 % of the isolates. This antimicrobial resistance profile was also seen in two of the human isolates (Table 1).

Campylobacter coli: Only 16 isolates (22.2 %) were fully susceptible to the antibiotics tested and 35 (48.6 %) were resistant to the quinolones. Am Na Ci was the most common pattern, accounting for 36.1 % of the isolates. Eight isolates (11.1 %) were each resistant to four of the drugs (Table 2).

The antimicrobial resistance patterns were not related specifically to the PFGE patterns: on the one hand a single subtype could exhibit different antibiotics resistance profiles, as with SJ5KJ8, SJ6KJ6, SJ8KJ12, SJ14KJ17, SJ23KJ7, or SC5KC5, SC7KC7, SC8KC8, SC9KC13; on the other hand, a particular antibiotics resistance profile could be associated with different pulsotypes.

Resistance to quinolones was found in isolates from most of the farms (85 %) that used quinolones to treat poultry during the rearing period.

Sequence analysis of the QRDR of the *gyrA* gene: A mutation in codon 86 of the *gyrA* gene with a Threonin-Isoleucine substitution is reported to be the main cause of high-level resistance to quinolones (Piddock *et al.* 2003). Among the highly quinolone-resistant strains of *C jejuni* (ciprofloxacin MIC 8- >32 $mg.l^{-1}$, nalidixic acid MIC 32- >256 $mg.l^{-1}$), 18 showed the Threonine86-Isoleucine substitution, four the Threonine86-Alanine substitution and four showed no mutation in the QRDR. Two strains with low-level resistance to nalidixic acid (MIC = 16 $mg.l^{-1}$), but susceptible to ciprofloxacin, had the Threonine86-Alanine substitution.

DISCUSSION

To study the epidemiology of *Campylobacter* infections, several genetic typing methods have been developed in order to differentiate isolates below species level (Wassenaar and Newell 2000). However, PFGE has been widely recognized as the most discriminatory method for both *C. jejuni* (Fitzgerald *et al.* 2001a) and *C. coli* (Fitzgerald *et al.* 2001b). It has also been demonstrated that macrorestriction analysis with the two enzymes, *Sma*I and *Kpn*I, was the method of choice for subtyping (On *et al.* 1998; Michaud *et al.* 2001).

In the present study, 68.4 % of chicken carcasses were contaminated with *Campylobacter*. These results are consistent with those of a previous study, where *Campylobacter* was isolated from 63 % of broiler flocks (Cardinale *et al.* 2004). The findings are also in accordance with the results obtained from other developing countries, such as Kenya and China, where thermophilic *Campylobacter* spp. have been isolated from 77 % and 76 % of chicken samples respectively (Osano and Arimi 1999; Shih 2000).

In this study, we identified a significant diversity of PFGE patterns among strains from different farms, but with specific clones occurring in most of the farms. Some flocks may be infected by two or more different strains; however, the method used did not allow multiple strains on any one chicken carcass to be detected. Moreover, strains that were present in lower numbers and grow less well in enrichment media or are more sensitive to oxidative stress and damage are likely to be overlooked (Petersen and Wedderkopp 2001).

The presence of one predominant strain at a particular farm has already been demonstrated in several studies (Berndtson *et al.* 1996; Jacobs Reitsma 1997). In fact, studies on the dynamics of infection in experimentally-infected chicken flocks have concluded that some strains of *Campylobacter* are able to become dominant, while preventing colonisation by other strains (Korolik *et al.* 1998; Barrow and Page 2000). In Senegalese poultry production, numerous sources of infection exist and a previous study has highlighted several possible risk factors (Cardinale *et al.* 2004). Poultry flocks appear to become infected mainly by horizontal pathways via the farm environment. This is consistent with earlier reports showing that the main source of infection is likely to be the immediate surroundings of the house, in spite of the use of hygiene barriers (Kazwala *et al.* 1990; Van de Giessen *et al.* 1998).

By contrast, the samples from retails shops harboured a variety of strains. In Senegal, retail outlets are small shops that sell only a relatively low number of chickens and are supplied by numerous farms. Thus, each shop is a site of possible cross-contamination from chickens of multiple origins.

In this study, the human and poultry isolates of *C. jejuni* shared common PFGE patterns: SJ2KJ2, SJ6KJ6, SJ20KJ23 or SJ23KJ7. Because of the low number of human isolates, it is difficult to draw firm conclusions about the potential for transmission from broiler chickens to humans, as is the case in other countries (Hanninen *et al.* 2000; Petersen *et al.* 2001). In Senegal, many enteritis sufferers do not seek medical attention and, even among those that do, only some will have a stool specimen cultured for enteric pathogens. This is especially true for *Campylobacter*, because there is no systematic investigation of *Campylobacter* cases in medical laboratories. Further investigation of human isolates is badly needed.

In our survey, high rates of resistance to quinolones were observed for both species of *Campylobacter* isolated from chicken samples. This was similar to the results obtained in several European countries (Piddock 1995) and in Japan (Chuma *et al.* 2001). As reported by Saenz *et al.* (2000), cross-resistance between nalidixic acid and ciprofloxacin has also been observed in Spain. Since 1991, when Endtz *et al.* (1990) identified the first quinolone-resistant *C. jejuni* and *C. coli* in the Netherlands, the resistance of *Campylobacter* to quinolones has increased throughout the world (Saenz *et al.* 2000; Engberg *et al.* 2001). It has long been suggested that this resistance was related to the introduction of the antibiotics into veterinary medicine (Endtz *et al.* 1990; Saenz *et al.* 2000). In Senegal, fluoroquinolones (norfloxacin, enrofloxacin) were first used in poultry production in 1996 to treat respiratory and intestinal diseases, such as colibacillosis and salmonellosis. Due to treatment failures with other antibiotics, they became the first-line drugs in 2000. Thus, according to our observations, the high prevalence of quinolone-resistance could be related to the introduction of these drugs in the industry (McDermott *et al.* 2002). Moreover, there was no relationship between antimicrobial resistance and PFGE patterns. This suggests the absence of any dominant clones that might have developed resistance in the past and then flourished under selective pressure. Instead, the observed resistance was probably generated *de novo* by

genetic modification from the selective pressure of antibiotic usage. Most of our highly-quinolone-resistant strains exhibited the Threonine86-Isoleucine substitution, but we also found a Threonine86-Alanine substitution in four isolates and no mutation in four others. The Thr-86-Ala change was described previously by Bachoual *et al.* (2001) for one clinical strain of *C. jejuni* with high-level resistance to nalidixic acid (MIC 64 • $g.ml^{-1}$), but low-level resistance to ciprofloxacin (MIC 2 • $g.ml^{-1}$). Piddock *et al.* (2003) also found a lack of mutation in *gyrA* in two resistant isolates (MICs for ciprofloxacin of 16 and 32 $mg.l^{-1}$ respectively), and they suggested that development of resistance in the absence of a mutation in *gyrA* is extremely rare. However, we believe that other mutational changes might exist in *gyrA* outside the critical region. It has been shown that factors other than *gyrA* QRDR mutations, such as efflux pumps and *parC*, may contribute to the resistance phenotype (Gibreel *et al.* 1998; Lin *et al.* 2002).

At present, Senegal is one of the biggest producers of poultry meat in West Africa (http://faostat.fao.org/). While there still is a traditional sector, a modern system of poultry production has been developed over the last fifteen years. At the same time, *Campylobacter* has emerged as a potential hazard to consumers, not only in relation to food poisoning, but also through the spread of antibiotic resistance. Although further investigations are needed to explore the relationship between human and poultry isolates, this study has highlighted the importance of continuous, routine surveillance to monitor the *Campylobacter* situation in Senegal.

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Fig 1: Dendrogram based on *Sma*I and *Kpn*I-digested DNA from *C. jejuni* isolates from poultry and humans (Dakar-region, Senegal, 2000 – 2003). Similarity fingerprints were determined by using the Dice coefficient and a band-position tolerance of 1 %. Dendrograms were generated by the unweighted pair-group method with arithmetic means. Twenty-five patterns were obtained from 109 isolates.

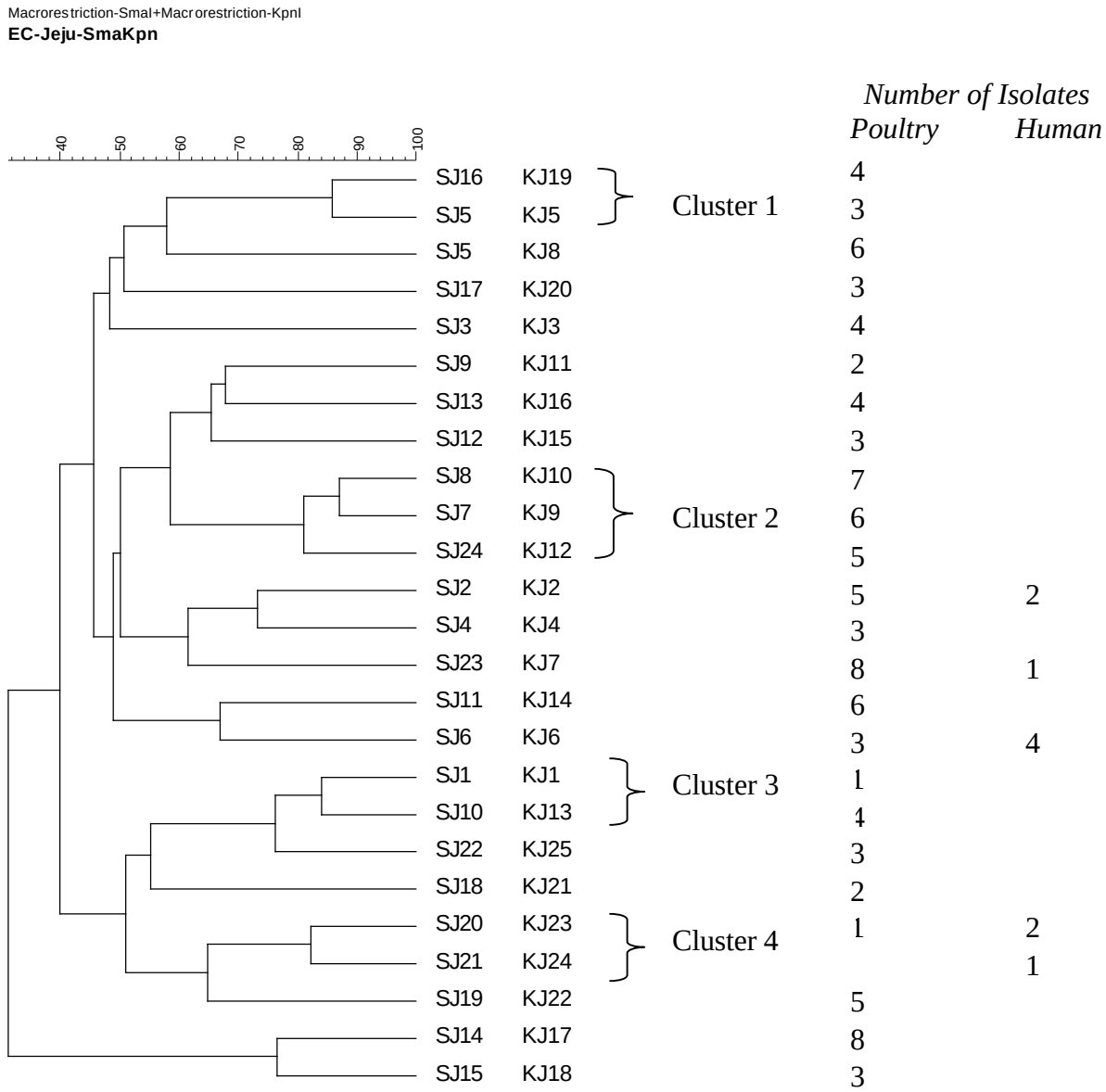

Table 1: Antimicrobial resistance and PFGE patterns for human and poultry isolates of C. *jejuni* (Dakar region, Senegal, 2000-2003, 109 isolates)

PFGE pattern	Antimicrobial resistance pattern	Number of poultry isolates	Number of human isolates	Origin of poultry isolates	Use of anti-biotics
SJ1KJ1		1		Farm	No
SJ2KJ2	Am	5		Farm	Yes
SJ2KJ2	Am		2		
SJ3KJ3	Na Ci	4		Farm	Yes
SJ4KJ4	Na Ci	3		Farm	Yes
SJ5KJ5	Na Ci	3		Farm	Yes
SJ5KJ8	Am Na Ci	1		Farm	Yes
SJ5KJ8		5		Farm	Yes
SJ6KJ6	Na Ci	1		Farm	Yes
SJ6KJ6			1		
SJ6KJ6	Am Na Ci		2		
SJ6KJ6	Am Na Ci	2		Retail shop	
SJ6KJ6	E Na Ci		1		
SJ7KJ9	A Na Ci	6		Farm	Yes
SJ8KJ10		7		Farm	No
SJ9KJ11	Am Na Ci	2		Farm	Yes
SJ10KJ13	Na Ci	2		Farm	Yes
SJ10KJ13	Na Ci	2		Retail shop	
SJ11KJ14	Am Na Ci	6		Farm	Yes
SJ12KJ15		1		Farm	Yes
SJ12KJ15		2		Retail shop	
SJ13KJ16		4		Farm	No
SJ14KJ17		5		Farm	No
SJ14KJ17	Am	3		Retail shop	
SJ15KJ18		3		Farm	No
SJ16KJ19		4		Farm	No
SJ17KJ20		3		Retail shop	
SJ18KJ21	Na Ci	2		Retail shop	
SJ19KJ22	Am E Na	5		Farm	Yes
SJ20KJ23	Am		2		
SJ20KJ23	Am	1		Retail shop	
SJ21KJ24	Am		1		
SJ22KJ25	Am Na Ci	3		Retail shop	
SJ23KJ7		2		Retail shop	
SJ23KJ7	Am	4		Farm	Yes
SJ23KJ7	Am		1		
SJ23KJ7	Am Na Ci	2		Retail shop	
SJ24KJ12	E Na Ci	4		Farm	Yes
SJ24KJ12		1		Retail shop	

Ac: amoxicillin-clavulanic acid ; Am: amoxicillin; Ci : ciprofloxacin ; E: erytrhromycin; Na: nalidixic acid

Fig 2: Dendrogram based on *Sma*I and *Kpn*I-digested DNA from *C. coli* isolates from poultry (Dakar-region, Senegal, 2000 – 2003). Similarity fingerprints were determined by using the Dice coefficient and a band-position tolerance of 1 %. Dendrograms were generated by the unweighted pair-group method with arithmetic means. Nineteen patterns were obtained from 72 isolates.

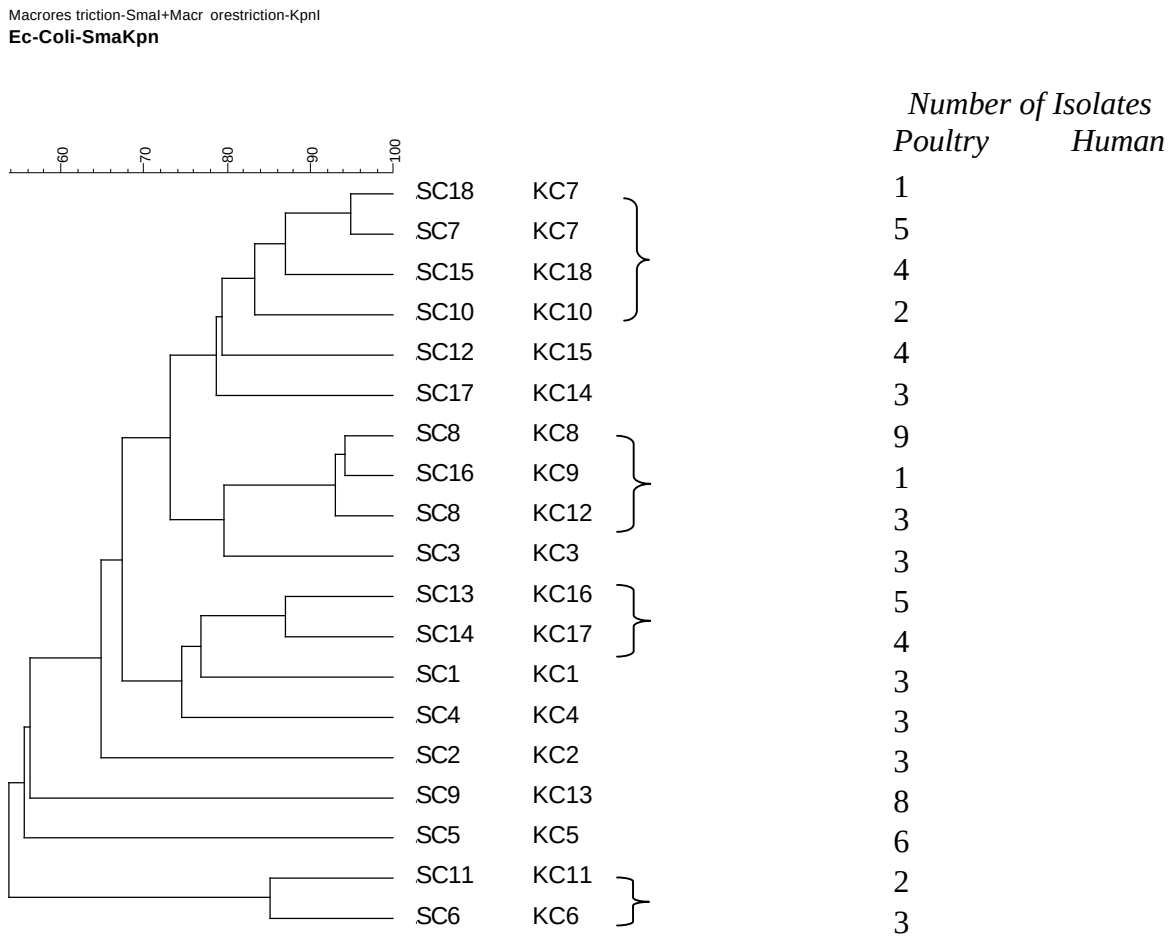

Table 2: Antimicrobial resistance and PFGE patterns for human and poultry isolates of C. *coli* (Dakar region, Senegal, 2000-2003, 72 isolates)

PFGE pattern	Antimicrobial resistance pattern	Number of poultry isolates	Number of human isolates	Origin of poultry isolates	Use of anti-biotics
SC1KC1	Am Na Ci	3		Farm	Yes
SC2KC2	Am Na Ci	3		Farm	Yes
SC3KC3	Am Na Ci	3		Farm	Yes
SC4KC4		3		Farm	Yes
SC5KC5	Am Ac Na Ci	5		Farm	Yes
SC5KC5	Am Na Ci	1		Farm	Yes
SC6KC6	Am Ac Na Ci	3		Farm	Yes
SC7KC7		4		Farm	No
SC7KC7	Am Na Ci	1		Farm	No
SC8KC8	Am	4		Farm	Yes
SC8KC8	Am Na Ci	4		Farm + Retail shop	Yes
SC8KC8	Am E Na	1		Farm	No
SC8KC12	Am	3		Farm	Yes
SC9KC13		5		Farm	No
SC9KC13	Am Na	3		Farm	Yes
SC10KC10	Am	2		Retail shop	
SC11KC11	Am Na Ci	2		Retail shop	
SC12KC15		4		Farm	No
SC13KC16	Am Na Ci	4		Farm	Yes
SC13KC16	Am Na Ci	1		Retail shop	
SC14KC17	Am Na Ci	4		Farm	Yes
SC15KC18	E	4		Retail shop	
SC16KC9	Am Na Ci	1		Retail shop	
SC17KC14	Am E Na	3		Farm	Yes
SC18KC7	Na Ci	1		Farm	No

Ac: amoxicillin-clavulanic acid ; Am: amoxicillin; Ci : ciprofloxacin ; E: erytrhromycin; Na: nalidixic acid

Table 3: Percentage of strains with antimicrobial resistance for *Campylobacter jejuni* and *Campylobacter coli* isolated from humans and poultry (Dakar region, Senegal, 2000 – 2003, 181 isolates).

	Campylobacter jejuni		*Campylobacter coli*	
	Poultry isolates	Human isolates	Poultry isolates	Human isolates
Amoxicillin	40.4 (40)*	80 (8)	70.8 (51)	_____
Amoxicillin – clavulanic aic	0 (0)	0 (0)	11.1 (8)	_____
Ciprofloxacin	43.4 (43)	30 (3)	48.6 (35)	_____
Erythromycin	9.1 (9)	10 (1)	12.5 (9)	_____
Nalidixic acid	48.4 (48)	30 (3)	59.7 (43)	_____

* (number of isolates)