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Prevalence, antimicrobial resistance and virulence genes of *Escherichia coli* isolated from retail meat in Tamaulipas, Mexico

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ABSTRACT

Objectives: The aim of this study was to determinate the prevalence of *Escherichia coli* and its resistance to antimicrobials and the presence of virulence genes in retail samples of beef and pork in several locations in Tamaulipas, Mexico.
Methods: A total of 106 samples (54 beef and 52 pork) collected from August 2013 to March 2014 were analysed to detect *E. coli* isolates. The *E. coli* isolates were then analysed for detection of virulence factors and antimicrobial resistance genes. Antimicrobial susceptibility to 16 antimicrobial agents was also determined.
Results: A total of 158 *E. coli* isolates were obtained, among which 3 (1.9%) harboured the virulence gene *stx1*, 28 (17.7%) harboured *stx*2 and 34 (21.5%) harboured *hlyA*. High phenotypic resistance was observed in almost all isolates, since 146 (92.4%) showed a multiresistant phenotype with resistance to cefalotin (92%), ampicillin (92%), cefotaxime (78%), nitrofurantoin (76%) and tetracycline (75%). The antimicrobial resistance genes *tet*(A) and *tet*(B) were detected in 56% of isolates, *strA* in 9.6%, *aadA* in 17% and *aac(3)-IV* in only 0.6% of strains.
Conclusions: Based on these results, it can be concluded that retail beef and pork meat may play a role in the spread of antimicrobial-resistant *E. coli* strains in this region.
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1. Introduction

Foodborne diseases typically present as diarrhoeic episodes and affect 550 million patients every year. The World Health Organization (WHO) estimates that 1 in 10 people become sick annually by consuming contaminated foods. Many of these diseases are frequently associated with consumption of contaminated meat that has not been adequately cooked [1], e.g. with pathogens such as *Salmonella* spp. and *Escherichia coli* [2–5]. In addition, in recent times *E. coli* has also taken relevance as a model for the dissemination of drug resistance in bacterial populations and as an indicator of the selective pressure of indiscriminate antibiotic use in animal production [6–9], making it a reference model in worldwide monitoring programmes of drug resistance [2], which may help to establish strategies to reduce the risk to the population [10]. In Mexico, prevalence studies of *E. coli* in meat are scarce; therefore, we do not have ready available information about the level of drug resistance and the distribution of virulence genes [11–14]. The aim of this study was to determinate the prevalence of *E. coli* and its resistance to antimicrobials and the presence of virulence genes in retail samples of beef and pork in several locations in Tamaulipas, Mexico.

2. Materials and methods

2.1. Sample collection

From August 2013 to March 2014, a total of 106 meat samples (54 beef and 52 pork) were purchased randomly from 55 supermarkets and retail stores (butcheries) located in 11 cities of Tamaulipas, Mexico. Five supermarkets from each city were randomly sampled. In each store, one ground beef sample and one ground pork sample were purchased randomly, in packing from ca. 500 g presentation. All samples collected were aseptically manipulated, were labelled and were stored individually on ice for transport to the laboratory in the Centro de Biotecnología Genómica of the Instituto Politécnico Nacional (Reynosa, Mexico).

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2.2. Isolation and identification of Escherichia coli

Microbiological analysis was performed according to the national Mexican standard for pathogen detection in foods [15]. Portions (25 g) were obtained for each sample and were homogenised for 2 min. Following homogenisation, samples were cultured on eosin–methylene blue (EMB) agar (BD Becton Dickinson & Co., Mexico State, Mexico) plates. After 18–24 h of incubation at 37 °C, presumptive colonies with characteristics corresponding to *E. coli* morphology were selected. From each sample, six colonies were individually inoculated in Difco™ tryptic soy agar (BD Becton Dickinson & Co.) and were incubated for 24 h a 37 °C to obtain a pure culture (six isolates per beef sample and six isolates per pork sample). Standard biochemical tests were applied to confirm the identity of *E. coli*, including lactose fermentation, citrate metabolism, methyl red–Voges-Proskauer, urease production and indole production.

2.3. Detection of virulence genes

Bacterial DNA for PCR was obtained by suspending bacterial colonies from a 24-h culture from tryptic soy agar plates in 500 μL of sterile water and boiling at 95 °C for 15 min, followed by centrifugation at 13 000 × *g* for 3 min. PCR analyses were performed using specific primers to the major enterohaemolysin/Shiga toxin-producing *E. coli* virulence genes that encode Shiga toxin Stx1–Stx2 and HlyA [16]. The PCR reaction mixture contained a final concentration of 1× buffer, 25 mM $MgCl_2$, 10 mM dNTPs, 10 mM primers, 5 U *Taq* DNA polymerase and sterile water in a final volume of 25 μL. The PCR amplification conditions were initial denaturation at 95 °C for 1 min, followed by 30 cycles of denaturation at 95 °C for 45 s, annealing at 53 °C for 45 s and extension at 72 °C for 45 s, and a final cycle of amplification at 72 °C for 7 min. PCR products were evaluated in 2.5% agarose gels with SYBR Gold (Invitrogen, Paisley, UK) at 100 V for 45 min. Negative controls (samples without a DNA template) and positive controls (samples with DNA from the collection of the Instituto Politécnico Nacional) were included in all PCR assays. DNA bands were visualised and photographed under ultraviolet light.

2.4. Antimicrobial susceptibility testing

Susceptibility testing to 16 antimicrobial agents was performed by the agar disk diffusion method according to Clinical and Laboratory Standards Institute (CLSI) guidelines [17]. The antimicrobial agents used included tetracycline (TET; 30 μg), amoxicillin/clavulanic acid (AMC; 30 μg), ciprofloxacin (CIP; 5 μg), amikacin (AMK; 30 μg), ampicillin (AMP; 10 μg), levofloxacin (5 μg), cefalotin (CEP; 30 μg), cefotaxime (CTX; 30 μg), ceftriaxone (30 μg), chloramphenicol (CHL; 30 μg), gentamicin (GEN; 10 μg), netilmicin (NET; 30 μg), nitrofurantoin (NIT; 300 μg), cefepime (30 μg), trimethoprim/sulfamethoxazole (25 μg) and streptomycin (STR; 30 μg). *Escherichia coli* strains were evaluated based on the diameter of the clear zone of inhibition around each antimicrobial disk. The results were interpreted in accordance with criteria provided by the CLSI and were classified as susceptible, intermediate or resistant. These antimicrobials are representative of the major classes of antimicrobial drugs that are important both to veterinary and human medicine. *Staphylococcus aureus* ATCC 29213 and *E. coli* ATCC 25922 were used as control strains.

2.5. Detection of antimicrobial resistance genes

The presence of genes associated with resistance to tetracycline [*tet*(A) and *tet*(B)], β-lactams (bla_{TEM}, bla_{NDM-1} and bla_{SHV}) and aminoglycosides [*strA*, *aadA*, *aac(3)-IV*] were detected by PCR [18,19]. PCR was performed on bacterial lysates as described previously. PCR was performed in a 25 μL reaction mixture containing 1× buffer, 25 mM $MgCl_2$, 10 mM dNTPs, 10 mM primers, 5 U *Taq* DNA polymerase and sterile water in a final volume of 25 μL. PCR amplification conditions were 95 °C for 1 min, followed by 30 cycles at 95 °C for 45 s, 59–42 °C for 45 s and 72 °C for 45 s, with a final amplification cycle at 72 °C for 7 min. Appropriate positive and negative controls were included in each PCR run. PCR products were electrophoresed in 2.7% agarose gels with SYBR Gold at 100 V for 45 min.

2.6. Statistical analysis

Data were analysed using IBM SPSS Statistics v.24.0 (IBM Corp., Armonk, NY). Univariate analysis was performed for calculation of the difference in prevalence by the χ^2 test. The level of significance was set at $P < 0.05$. Descriptive statistics (estimation of proportions) were used to summarise the prevalence of *E. coli* and antimicrobial susceptibility patterns of the isolates.

3. Results

3.1. Prevalence of Escherichia coli

In total, 636 strains were isolated from the 106 meat samples (324 stains from 54 beef samples and 312 strains from 52 pork samples) from the 11 cities of Tamaulipas included in this work. From the 106 total meat samples collected, 59 (55.7%) were

Table 1
Prevalence of *Escherichia coli* strains and virulence factors detected in meat samples from Tamaulipas, Mexico.

City	*E. coli* prevalence [*n* (%)]	Other species [*n* (%)]	*P*-value	Virulence factors [*n* (%)]				*P*-value
				hlyA	*stx1*	*stx2*	*hlyA* + *stx2*	
Altamira	13 (8.2)	47 (9.8)	N/S	0	0	5 (17.9)	0	N/S
Hidalgo	22 (13.9)	38 (7.9)	N/S	0	0	13 (46.4)	0	<**0.05***
Nuevo Laredo	39 (24.7)	21 (4.4)	<**0.05***	25 (73.5)	0	3 (10.7)	2 (66.7)	N/S
Miguel Alemán	8 (5.1)	52 (10.9)	N/S	2 (5.9)	0	0	0	N/S
Ciudad Mante	18 (11.4)	42 (8.8)	N/S	4 (11.8)	0	2 (7.1)	1 (33.3)	N/S
Matamoros	8 (5.1)	52 (10.9)	N/S	0	1 (33.3)	2 (7.1)	0	N/S
Rio Bravo	14 (8.9)	46 (9.6)	N/S	1 (2.9)	1 (33.3)	0	0	N/S
Reynosa	14 (8.9)	46 (9.6)	N/S	2 (5.9)	1 (33.3)	3 (10.7)	0	N/S
Tampico	8 (5.1)	52 (10.9)	N/S	0	0	0	0	N/S
Ciudad Victoria	10 (6.3)	50 (10.5)	N/S	0	0	0	0	N/S
Valle Hermoso	4 (2.5)	32 (6.7)	N/S	0	0	0	0	N/S
Total	158 (100)	478 (100)		34 (100)	3 (100)	28 (100)	3 (100)	

N/S, not significant ($P > 0.05$).
* Statistically significant ($P < 0.05$).

positive for *E. coli*, comprising 29/59 (49.2%) beef samples and 30/59 (50.8%) pork samples. Among the 636 strains from the 11 cities, 158 (24.8%) were confirmed as *E. coli*, comprising 74/158 strains (46.8%) from 29 beef samples and 84/158 strains (53.2%) from 30 pork samples. Most of the *E. coli* isolates were obtained from Nuevo Laredo, with 5/5 beef samples and 4/5 pork samples being positive for *E. coli*, representing 8.5% of all positive samples (9/106) and 24.7% of all *E. coli* strains (39/158) ($P > 0.05$) (Table 1). The city with the lowest percentage of *E. coli* isolates detected was Valle Hermoso, with 2/4 beef samples and 0/3 pork samples, representing 1.9% of all positive samples (2/106) and 2.5% of all *E. coli* strains (4/158) (Fig. 1; Table 1).

3.2. Detection of virulence genes

The presence of the virulence genes *stx1*, *stx2* and *hlyA* was tested by PCR analysis in all 158 *E. coli* strains, among which 65 (41.1%) were positive for one of these genes, comprising 32 from beef samples and 33 pork samples.

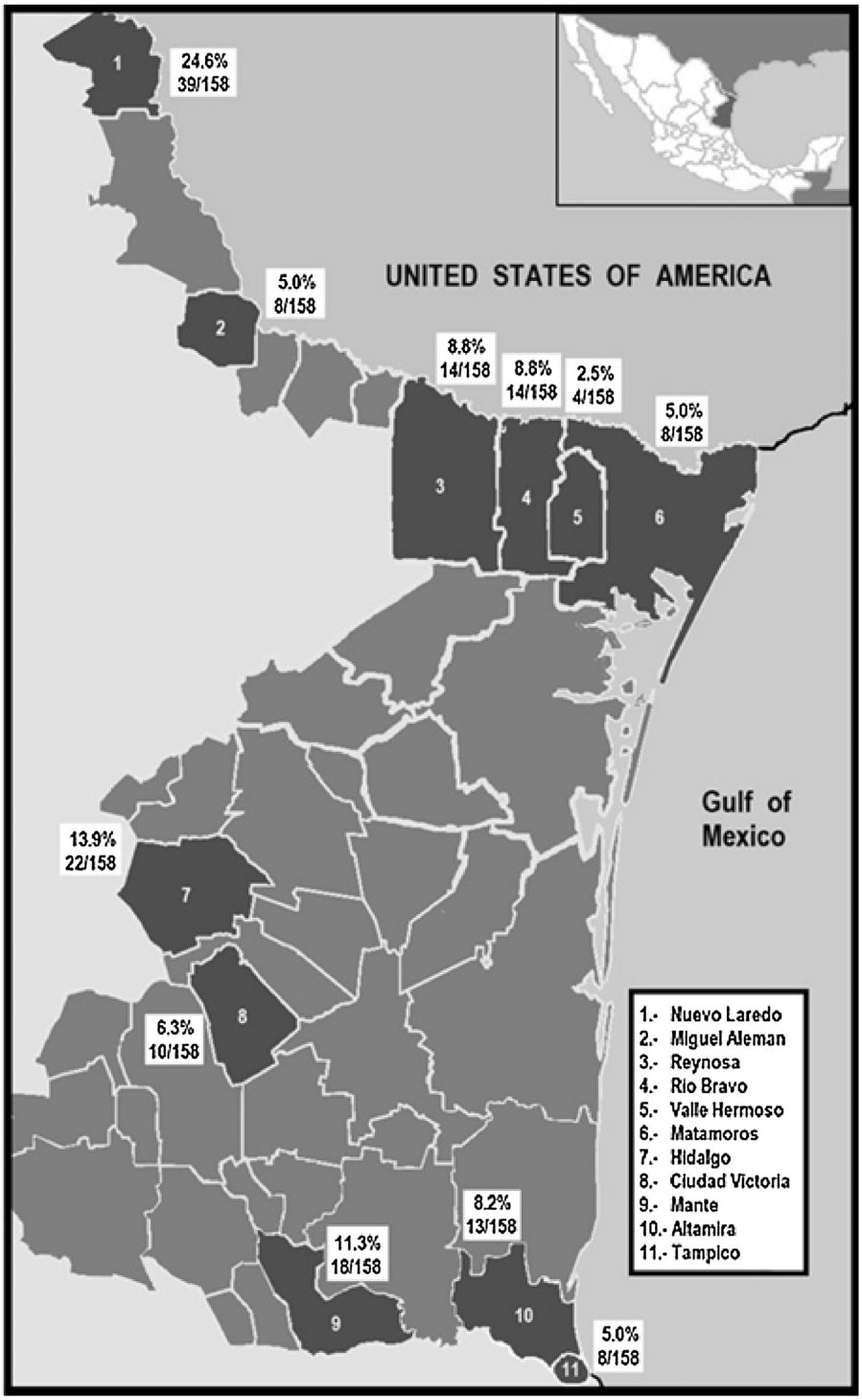

Fig. 1. Prevalence of *Escherichia coli* isolated from meat samples in supermarkets of Tamaulipas, Mexico. The number and percentage of *E. coli* strains detected is showed in each included city.

The *stx1* gene was detected in 3 (1.9%) of the strains tested, the *stx*2 gene was detected in 28 strains (17.7%) and the *hlyA* gene was the most prevalent being detected in 34 strains (21.5%). Only three strains (1.9%) contained both *stx2* and *hlyA*. None of the strains contained all three virulence genes (Table 1).

3.3. Antimicrobial resistance and detection of resistance-related genes

In the phenotypic resistance tests to antimicrobials, 92.4% of *E. coli* strains were resistant to at least four different antimicrobials. Most of the strains exhibited multidrug resistance patterns to seven, eight or nine antibiotics, simultaneously (48.7%; $n = 77$). A total of 133 different phenotypic resistance patterns were detected, examples of which are shown in Table 4. Of the 158 *E. coli* strains, 145 (91.8%) were resistant to CEP and 143 (90.5%) were resistant to AMP, followed by 123 (77.8%) to CTX, 120 (75.9%) to NIT and 107 (67.7%) to TET. On the other hand, 147 strains (93.0%) were susceptible to NET, 143 (90.5%) to CIP, 138 (87.3%) to AMK and 129 (81.6%) to GEN. No significant statistical associations were found between resistance phenotypes and meat type (Table 2). The presence of genes related to antibiotic resistance was also analysed. Of the 107 *E. coli* strains resistant to TET, one or both of the tested tetracycline resistance genes [*tet*(A) and *tet*(B)] were detected in only 60 strains (56.1%) (Table 3). Of the 62 strains resistant to STR, 6 (9.7%) harboured only *strA*, 11 (17.7%) harboured *aadA* and 21 (33.9%) harboured both *strA* and *aadA*. The presence of one or both genes was detected in 13/23 isolates (56.5%) from beef and in 25/39 isolates (64.1%) from pork. Among the 29 GEN-resistant strains, the *aac(3)-IV* gene was detected in only 1 strain (3.4%). However, in the 62 strains resistant to STR, *aac(3)-IV* was detected in 2 isolates (3.2%) (1 from a beef sample and 1 from a pork sample). In the 143 strains resistant to AMP and the 86 strains resistant to AMC, bla_{TEM} was the most prevalent, being present in 18/143 (12.6%) AMP-resistant strains and in 11/86 (12.8%) AMC-resistant strains. bla_{SHV} was detected in only 3 strains from beef samples resistant to AMP (2/64) and AMC (1/37), whereas none of

Table 2
Prevalence of phenotypic resistance to antimicrobials in *Escherichia coli* isolated from meat samples from Tamaulipas, Mexico.

Antimicrobial group	Antimicrobial agent	Phenotypic resistance [*n* (%)]			*P*-value
		Overall ($N = 158$)	Beef ($n = 74$)	Pork ($n = 84$)	
Aminoglycosides	Streptomycin	62 (39.2)	23 (31.1)	39 (46.4)	N/S
	Netilmicin	11 (7.0)	5 (6.8)	6 (7.1)	N/S
	Amikacin	20 (12.7)	12 (16.2)	8 (9.5)	N/S
	Gentamicin	29 (18.4)	16 (21.6)	13 (15.5)	N/S
Cephalosporins	Cefalotin	145 (91.8)	67 (90.5)	78 (92.9)	N/S
	Cefotaxime	123 (77.8)	54 (73.0)	69 (82.1)	N/S
	Cefepime	88 (55.7)	40 (54.1)	48 (57.1)	N/S
	Ceftriaxone	72 (45.6)	35 (47.3)	37 (44.0)	N/S
β-Lactams	Ampicillin	143 (90.5)	64 (86.5)	79 (94.0)	N/S
	Amoxicillin/clavulanic acid	86 (54.4)	37 (50.0)	49 (58.3)	N/S
Nitrofurans	Nitrofurantoin	120 (75.9)	56 (75.7)	64 (76.2)	N/S
Amphenicols	Chloramphenicol	36 (22.8)	16 (21.6)	20 (23.8)	N/S
Quinolones	Levofloxacin	41 (25.9)	20 (27.0)	21 (25.0)	N/S
Sulfonamides	Sulfamethoxazole/trimethoprim	76 (48.1)	30 (40.5)	46 (54.8)	N/S
Tetracyclines	Tetracycline	107 (67.7)	45 (60.8)	62 (73.8)	N/S
Fluoroquinolones	Ciprofloxacin	15 (9.5)	6 (8.1)	9 (10.7)	N/S

N/S, not significant ($P > 0.05$).

Table 3
Prevalence of genes related to antimicrobial resistance in *Escherichia coli* isolated from meat samples from Tamaulipas, Mexico.

Antimicrobial group	Phenotype resistance	Gene	Prevalence [*n* (%)]
Tetracyclines	Tetracycline (107/158)	*tet*(A)	26/107 (24.3)
		tet(B)	12/107 (11.2)
		tet(A) + *tet*(B)	22/107 (20.6)
Aminoglycosides	Streptomycin (62/158)	*strA*	6/62 (9.7)
		aadA	11/62 (17.7)
		strA + *aadA*	21/62 (33.9)
		aac(3)-IV	2/62 (3.2)
		aadA + *aac(3)-IV*	–
	Gentamicin (29/158)	*strA*	2/29 (6.9)
		aadA	6/29 (20.7)
		strA + *aadA*	11/29 (37.9)
		aac(3)-IV	–
		aadA + *aac(3)-IV*	1/29 (3.4)
β-Lactams	Ampicillin (143/158)	bla_{TEM}	18/143 (12.6)
		bla_{NDM-1}	5/143 (3.5)
		bla_{SHV}	1/143 (0.7)
		$bla_{TEM} + bla_{SHV}$	1/143 (0.7)
	Amoxicillin/clavulanic acid (86/158)	bla_{TEM}	11/86 (12.8)
		bla_{NDM-1}	4/86 (4.7)
		bla_{SHV}	–
		$bla_{TEM} + bla_{SHV}$	1/86 (1.2)

Table 4
Representative examples of phenotypic characteristics of multidrug-resistant *Escherichia coli* isolated from meat samples collected in Tamaulipas, Mexico.

Resistance phenotype	No. of isolates	
	Beef (n = 74)	Pork (n = 84)
STR–TET	4	–
CEP–CTX–AMP–NIT	6	1
FEP–CEP–CTX–AMP	1	–
STR–CEP–SXT–TET	–	1
CEP–AMP–NIT–TET–AMC	3	1
FEP–CEP–CTX–AMP–NIT	2	–
FEP–CEP–AMP–TET–AMC	–	1
CEP–CTX–SXT–AMK–AMP–AMC	1	–
FEP–CEP–CTX–AMP–CRO–NIT–TET	1	1
FEP–CEP–CTX–SXT–AMP–CHL–NIT	–	2
FEP–CEP–CTX–AMP–CRO–NIT–AMC	3	3
FEP–CEP–CTX–SXT–AMP–NIT–TET–AMC	–	1
FEP–CEP–CTX–SXT–AMP–CHL–NIT–TET	1	1
STR–FEP–GEN–CTX–AMK–AMP–CRO–TET	–	2
STR–FEP–CEP–CTX–SXT–AMP–CRO–NIT–TET	–	2
STR–FEP–CEP–CTX–SXT–AMP–NIT–TET–AMC	1	1
NET–CEP–CTX–SXT–AMP–CRO–NIT–TET–AMC	1	–
LVX–CEP–CTX–SXT–AMP–CRO–CHL–NIT–TET–AMC	–	1
LVX–FEP–CEP–CTX–SXT–AMP–CRO–NIT–TET–AMC–CIP	–	1
STR–FEP–CEP–CTX–SXT–AMP–CRO–CHL–NIT–TET–AMC	1	1
FEP–CEP–CTX–SXT–AMK–AMP–CRO–CHL–NIT–TET–AMC	–	1
STR–NET–FEP–CEP–GEN–CTX–SXT–AMP–CRO–NIT–TET–AMC	–	1
STR–FEP–CEP–GEN–CTX–SXT–AMK–AMP–CHL–NIT–TET–AMC	1	–
LVX–FEP–CEP–GEN–CTX–SXT–AMK–AMP–CHL–NIT–TET–AMC	1	–
STR–LVX–FEP–CEP–CTX–SXT–AMP–CHL–NIT–TET–AMC–CIP	–	1
STR–FEP–CEP–GEN–CTX–SXT–AMK–AMP–CRO–NIT–TET–CIP	1	–
STR–LVX–FEP–CEP–CTX–SXT–AMP–CRO–CHL–NIT–TET–AMC–CIP	1	–
STR–LVX–NET–FEP–CEP–GEN–CTX–SXT–AMP–CHL–NIT–TET–CIP	1	–

STR, streptomycin; TET, tetracycline; CEP, cefalotin; CTX, cefotaxime; AMP, ampicillin; NIT, nitrofurantoin; FEP, cefepime; SXT, trimethoprim/sulfamethoxazole; AMC, amoxicillin/clavulanic acid; AMK, amikacin; CRO, ceftriaxone; CHL, chloramphenicol; GEN, gentamicin; NET, netilmicin; LVX, levofloxacin; CIP, ciprofloxacin.

the strains from pork samples harboured bla_{SHV}. On the other hand, bla_{NDM-1} was not detected in strains from beef samples but was present in 9 strains from pork samples (9/158; 5.7%).

4. Discussion

According to these results, a greater prevalence of *E. coli* was observed in cities from Northern Tamaulipas, bordering with the USA. There are no previous reports in this area to compare the results of the present study with. As far as we know, this is the first work performed in this area of Tamaulipas. The presence of *E. coli* in retail meat indicates low sanitary quality management and a potential risk to consumer health. Although it is considered that cooking meat destroys *E. coli* that might be present, situations such as undercooking, low handler hygiene, and cross-contamination of cooked food with raw meat and surfaces or utensils in contact with raw meat can lead to further distribution of *E. coli* strains. The presence of a high quantity of *E. coli* can indicate low-quality practices, although it does not always represent a health risk since *E. coli* strains comprise a varied group of pathogenic and non-pathogenic serotypes. Shiga toxin-producing *E. coli* (STEC) and enterohaemorrhagic *E. coli* (EHEC) are strains that are considered a high health risk because they can cause diarrhoea and serious conditions such as haemolytic–uraemic syndrome (HUS) and, in some cases, even death [20]. A common characteristic of all EHEC strains is the production of an EHEC-specific plasmid-mediated haemolysin encoded by *hlyA* [21] and at least one Shiga-like toxin encoded by *stx1* or *stx2* [22]. Livestock is considered a reservoir for STEC strains, with the possible route of transmission to humans being beef contaminated with faecal matter at some point in the processing route [23,24]. In several countries, STEC have been detected in beef and pork retail products (in addition to other beef products) by detecting *stx1* and *stx2*. In the samples included in the current study, these genes were detected alone or together in 31 (19.6%) of the isolated strains (Table 1). This prevalence is similar to that reported by Minh et al. in Japan (58/270; 21.5%) [25], Park et al. in South Korea (17%) [26] and Ateba and Mbwe in South Africa (23.7%) [27]. Regarding the prevalence of each gene, *stx1* and *stx2* was identified in 1.9% and 17.7% of the analysed strains, respectively. This high predominance of *stx2* has been observed in some other studies, such as that by Li et al. in China who reported prevalences of 4.9% and 27.6% for *stx1* and *stx2*, respectively [28]. Similar results have also been reported by Minh et al. (6.6% and 14.8% for *stx1* and *stx2*, respectively) and Ateba and Mbwe (6.2% and 17.5% for *stx1* and *stx2*, respectively) [25,27]. These findings are relevant because some epidemiological studies have indicated that strains carrying *stx2* are potentially more virulent and are more frequently related to HUS than those carrying *stx1* or even those carrying both *stx1* and *stx2* [29,30]. Treatment of EHEC infections with antibiotics may worsen the illness, presumably by breaking up the bacteria, causing the release of more toxins and increased toxin production [23,31]. However, early administration using some antimicrobials is effective [32]. Unfortunately, inappropriate antimicrobial use has contributed to the increase in antimicrobial resistance [31,33] and some strains have the ability to transfer antibiotic resistance to others, posing a challenge in the treatment of infectious diseases.

Of all 158 analysed strains, 146 (92.4%) were resistant to at least 4 (and up to 13) antibiotics. This drug resistance may be considered high in comparison with similar reports from other areas. For instance, Sheikh et al. from Canada (with pork, beef, poultry and turkey samples) and Llorente et al. from Buenos Aires (with beef samples), reported a multidrug resistance prevalence of 28.1% and 27.8%, respectively [2,34], both of which are quite below the prevalence in the current study (92.4%). In a review of human and food samples from beef, pork and poultry in the USA from 1950–2002, Tadesse et al. reported a prevalence of multidrug resistance of 54% (59.1% in beef and 53.7% in pork) [35]. Similar findings were

reported by Skockova et al. from the Czech Republic (with samples of beef, pork, poultry and deer) with a multidrug resistance prevalence of 45.2% [36], which although higher are still lower compared with the current findings (92.4%). Looking for similar reports from Mexico, we could only find the work of Canizalez-Roman et al. from Sinaloa, apparently being the first report of this kind of study in Mexico [16]. In that work, several different kinds of food (raw and processed) were analysed for drug resistance to nine antibiotics and 66% of the *E. coli* strains were resistant to one or more antibiotics and 39.2% of strains were multidrug-resistant. These prevalence findings are still low compared with 92.4% multidrug resistance reported in the current study; however, in the present study 16 antimicrobial agents were tested, which may affect the comparison of the findings.

TET and AMP are antibiotics that are widely used in similar published studies, thus some comparison of results can be made; on the other hand, CTX, NIT and CEP are not frequently included. Sheikh et al. reported resistance to TET of 20.5% (16.4% in beef and 31.7% in pork) and to AMP of 7.2% (5.5% in beef and 12.2% in pork) [2]. Tadesse et al. reported the most co-resistance to TET and STR (29.7%), TET and AMP (18.8%) and TET, AMP, STR and a sulfonamide (19.9%) [35]. On the other hand, Llorente et al. reported a resistance prevalence of 28.1% to AMP, STR, AMK and TET, although they did not give information about resistance to each individual antimicrobial [34]. In the same way, Skockova et al. also reported AMP and TET as the antibiotics with the most resistant strains (29% and 25.8%, respectively) [36]. In a report by Canizalez-Roman et al. from Mexico, they indicate that the main resistance in the strains was to TET (34%), CTX (30%) and AMP (29%) [16]. Unfortunately, these percentages were estimated in general for all the food samples included in the study, so we cannot compare them directly with the results in the meat samples in the current study.

In this work, a high prevalence of *E. coli* strains resistant to CEP, AMP, CTX, NIT and TET was found; therefore, it was of particular interest to search for the presence of genes related to drug resistance to these antimicrobials. Of the 158 *E. coli* isolates, 107 (67.7%) were phenotypically resistant to TET, of which 60 (38.0%) harboured one or both TET resistance-related genes. However, in total 81 (51.3%) of the 158 strains harboured one or both genes *tet*(A) and *tet*(B). One interesting finding is that of the 81 isolates with *tet*(A) and/or *tet*(B), 6 (7.4%) had intermediate resistance to TET and 15 (18.5%) were susceptible to TET. For β-lactam-related antibiotics, strains resistant to AMP and AMC were tested for the presence of bla_{TEM}, bla_{SHV} and bla_{NDM-1}. Of the 143 isolates with phenotypic resistance to AMP, 25 (17.5%) had at least one of the *bla* genes, and in this case *bla* genes were only detected in phenotypically resistant strains. However, when the presence of *bla* genes in strains resistant to AMC was searched for, of the 86 phenotypically resistant strains only 16 (18.6%) harboured one of the *bla* genes. In this case, *bla* genes were also found in four strains with intermediate resistance to AMC and four strains were AMC-susceptible. Regarding aminoglycosides, the presence of *strA*, *aadA* and *aac(3)-IV* was according to phenotypic resistance to STR.

The presence of antibiotic resistance genes in *E. coli* strains and their effect on phenotypic resistance are the result of a complex dispersion system. Schmid et al. reported extended-spectrum β-lactamase (ESBL)-producing *E. coli* isolates from farms on which β-lactam antibiotics were not used, suggesting that the presence of such isolates may be due to the use of other different classes or antibiotics that may also select ESBL-producing strains as well [37]. According to Jacoby and Sutton, resistance determinants against aminoglycosides, tetracycline, sulfonamides and cephalosporins are often situated on the same plasmid [38]. Plasmids and transposons carrying multiple antimicrobial resistance genes can also carry genes for virulence and metabolic functions, e.g. Tn*1691* specifies resistance to some antibiotics (STR, sulfonamides and CHL) [36]. This could indicate that there are factors other than veterinary medicine leading to the retention of antibiotic resistance determinants in cattle. Some authors indicate that even the air may be a vehicle for the transfer of elements of genetic resistance to antibiotics in bacteria [37–39].

5. Conclusions

To our knowledge, this study is the first report on the prevalence and antimicrobial resistance in *E. coli* strains from beef and pork samples in Tamaulipas, Mexico. The *E. coli* prevalence was 24.8% (158/636), indicating a low sanitary quality management. Coupled with this, the presence of virulence factors in a high percentage of strains (41.1%) as well as the high multidrug resistance detected to β-lactams, aminoglycosides and tetracycline may represent a health risk for beef consumers because of inadequate handling of meat.

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Competing interests

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Ethical approval

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