PHENOTYPIC AND GENOTYPIC IDENTIFICATION OF FLOUROQUINOLONES RESISTANT *ESCHERICHIA COLI* ISOLATED FROM DISEASED CHICKEN IN SUEZ CANAL DISTRICT

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ABSTRACT

A total of 70 Escherichia coli strains were isolated from samples (liver and heart) collected from diseased chickens (n=105) in about 21 commercial farms located in Suez Canal district of Egypt during May-July 2012. We investigated the susceptibility to Flouroquinolones (Ciprofloxacin, Enrofloxacin and Norfloxacin) and 9 other antibacterials (Gentamicin, Streptomycin, Doxycyclin, Amoxicillin, Ampicillin, Cephazolin, Cephradine, Lincomycin, Florfenicole) by disc diffusion method and measured the MIC of ciprofloxacin and enrofloxacin. Resulted flouroquinolones resistant E.coli isolates were then serotyped, and investigated for the prevalence of plasmid-mediated fluoroquinolone resistance (PMQR) genes (qnrA, qnrB, qnrS, aac(6`)-Ib-cr, and qepA) by polymerase chain reaction. Among the E.coli isolates, 72.8% (51/70) were resistant to Ciprofloxacin and Enrofloxacin with MIC ≥ 4 and ≥ 2 (µg/ml) respectively. These

isolates were resistant also to at least 6 other investigated antibacterials in the study for susceptibility/resistance. Among the resistant 51 E.coli strains, 36 (70.5%) were positive for PMQR genes, 27 (52.9%) were positive for at least one gene of the qnr genes investigated in the study, 16 (31.37%) were positive for qnrA, qnrB and qnrS separately, 20 (39.2%) were positive to aac(6`)-Ib-cr, 35 (68.6%) were positive to qepA, while 4 (7.8%) harbored all the 5 investigated genes. The resistance of E. coli to flouroquinolones has been increasing in the past twenty years but unfortunately no enough data about isolates harboring PMQR genes in Egypt either in human or poultry field. Further investigations concerning the antimicrobial resistance problem in human and veterinary field in Egypt is recommended as a surveillance program is a must.

Key words: Flouroquinolones resistance, *E.coli*, qnr, qepA, aac(6`)-Ib-cr, chickens.

INTRODUCTION

Antimicrobials are valuable tools to treat clinical disease and to maintain healthy and productive animals. However, the treatment of whole herds and flocks with antimicrobials for disease prevention and growth promotion has become a controversial practice ***(Van de Boggard, 1999).*** Sick animals are sometimes treated individually, but often whole flocks or herds of animals are treated at once, including animals that are not ill. In addition, antimicrobials are used in the absence of disease to prevent diseases during times when animals may be susceptible to infections. This practice is very common in countries where infections

caused by enteric pathogens are severe on poultry farms. Such misuse and/or inappropriate usage increases the likelihood of selecting for organisms that are resistant to the antibiotic. Of particular concern is the emergence of resistance to frontline antimicrobials, such as the fluoroquinolones, which because of their low toxicity and relatively broad-spectrum coverage are extremely valuable for treating human infections ***(Livermore et al., 2002).***

It was stated by well established evidence that antibiotics can lead to the emergence and dissemination of resistant *E. coli* which can then be passed into people via food or direct contact with infected animals. These resistant microbes may function as a potential source in the transportation of antimicrobial resistance to human pathogens ***(Schroeder et al. 2002).***

Unfortunately, data on the prevalence of antimicrobial-resistant veterinary pathogens are sparse, particularly in developing countries where antimicrobials are overused in veterinary medicine and food animals ***(Ban, 2001).*** Data on the prevalence of antimicrobial-resistant veterinary pathogens are needed for science-based risk assessments focusing on the relative risks concerning use of antimicrobials in animal husbandry ***(Yang et al., 2004).***

The first quinolone, nalidixic acid (possessing a naphthyridone core), was introduced into clinical use in 1962 ***(Ball, 2000)***. In the mid-1980s, ciprofloxacin, a fluoroquinolone (with a quinolone core) that had a wider spectrum of in vitro antibacterial activity, particularly against

gram-negative bacteria, first became available clinically. Since then, newer agents with increased antimicrobial activity against gram-positive pathogens have been developed, but the activity of ciprofloxacin against gram-negative pathogens has been largely unsurpassed ***(Hooper, 2005).*** In the decades that have elapsed since the introduction of fluoroquinolones, resistance of the *Enterobacteriaceae* to these agents has become common, widespread, and generally nonclonal. ***(Colodner et al., 2001).***

It is known that there are at least two mechanisms of resistance to these synthetic antimicrobial agents. The most common mechanism is mutational alteration in the so-called quinolone resistance-determining regions of the drug targets ***(Hawkey, 2003).*** The second is the reduction of fluoroquinolone accumulation by active export of the drugs via over expression of chromosomal efflux pumps either alone ***(Poole, 2005)***, or associated with decreased production or qualitative alteration of outer membrane porins ***(Ruiz, 2003).*** Recently, three additional plasmid-mediated low-level resistance mechanisms have been reported: the qnr proteins that protect type II topoisomerases from quinolones ***(Martìnez-Martìnez et al., 1998; Robicsek et al., 2006a)***; aac(6`)-Ib-cr, a variant aminoglycoside acetyltransferase that modifies ciprofloxacin ***(Robicsek et al., 2006b)***; and a plasmid-mediated efflux pump, qepA ***(Yamane et al., 2007).***

Although normally commensal in nature, certain strains of *Escherichia coli* are associated with a variety of infections in humans and animals ***(Bertschinger, 1999)***. In chickens they may cause infections

of the respiratory tract and soft tissues, resulting in colibacillosis, air sacculitis, and cellulitis ***(Gross, 1991)***. Colibacillosis in poultry farms is usually primary or a secondary infection with viral diseases. The morbidity and mortality are approximately 25% and 5%, respectively. Poultry farms often use ciprofloxacin or different other antimicrobials to treat infected chicken flocks ***(Xu, 2001)***.

Aim of work: The poultry reservoir of plasmid-mediated quinolone resistance (PMQR) is still controversial and little information is available on the prevalence of these resistance determinants in developing countries. The aim of this study was to identify, characterize and investigate prevalence of the PMQR in a collection of clinical multi-resistant *Escherichia coli* isolated from diseased chickens with failure of treatment, collected in May–July 2012, in Egypt.

MATERIALS AND METHODS

Collection of samples: Tissues were collected aseptically according to clinical findings and post mortem examinations of diseased chickens suspected with colibacillosis with no response to antibiotic treatment and exhibiting perihepatitis, pericarditis, airsaculitis and pneumonia. A total of 105 samples (liver, heart) were aseptically collected from 21 different commercial broilers farms distributed in Ismailia, Port Said, Suez and North Sinai in the period from May to July 2012. All samples were directly transported to the laboratory and processed as soon as possible.

Isolation and Identification: Was done according to ***Koneman et al. (1992)***. The collected and prepared samples in were enriched in Buffer Pepton Water (BPW) (Lab, UK) by incubation at 37°C for 18-24 hours. After enrichment, a loopful from the incubated broth was streaked directly onto Eosin- Methylene –Blue Agar, EMB (Lab, UK) and incubated at 37°C for 18-24 hours. Colonies producing metallic sheen were subjected for further biochemical procedures as using Gram's staining, detection of motility, oxidase test, indole test, methyl-red test, Voges-Proskauer test, citrate utilization test, urea hydrolysis test, sugar fermentation (glucose, lactose, sucrose, mannital and salicin), H_2S production and gelatine liquefaction.

Antibiogram: All isolated *E.coli* strains (n=70) were tested for susceptibility to different antimicrobials; Ciprofloxacin (5µg, HiMedia, India), Enrofloxacin (5 µg, HiMedia, India)), Norfloxacin (10 µg, HiMedia, India), Gentamicin (10 µg, HiMedia, India), Ampicillin (10 µg, HiMedia, India), Amoxicillin (25 µg, HiMedia, India), Florfenicole (30 µg, HiMedia, India), Cephradine (30 µg, Oxoid, UK), Cephazoline (30 µg, Oxoid, UK), Lincomycin (2 µg, HiMedia, India), Streptomycin (10 µg, HiMedia, India) and Doxycycline (10 µg, HiMedia, India) by the Disc Diffusion Method on Mueller Hinton Plates (Lab, UK) according to ***NCCLS (2002).*** The Minimum Inhibitory Concentration (MIC) of Ciprofloxacin and Enrofloxacin were measured using Broth Macrodilution Method for all *E. coli* isolates outlined by ***NCCLS (2002)*** with Mueller-Hinton broth (Lab, UK). All susceptibility results were interpreted according to the **NCCLS** interpretive standards **(2007).**

Isolates showing MIC ≥ 4 (μg/ml) for ciprofloxacin and ≥ 2 for Enrofloxacin (Bayer recommendations) were considered resistant. *E. coli* ATCC 25922 was used as quality control organism in antimicrobial susceptibility experiments.

Serotyping: Serotyping of the resistant *E.coli* isolates was done at the Bacteriology Unit in Reference Lab. for veterinary Quality control on Poultry production (RLQP)-Dokki, Egypt (accredited for Iso 17025). *E. coli* strains were biochemically confirmed and submitted to slide agglutination tests using polyvalent and monovalent antisera against serogroups and serotypes O26, O25, O103, O111, O78, O114, O145, O185, O91, O86, O44 and O86. Commercially available antisera were used.

Isolation and identification of resistance genes by PCR: All resistant *E.coli* isolates (n=51) were screened by PCR amplification for the transferable quinolone resistance determinants qnrA, qnrB, qnrS ***(Robicsek et al., 2006c)***, aac-(6`)-Ib-cr ***(Lunn et al., 2010)*** and qepA ***(Cattoir, 2008)*** in Biotechnology Unit, RLQP (accredited for Iso. 17025). Bacterial DNA was extracted using QIAamp DNA Mini Kit (Qiagen, Germany, Cat.no. 51304). PCR was carried out in a final volume of 25 μl containing 6 μl template DNA, 1 μl (20 Pmol) of each primer, 12.5 μl of EmeraldAmp® MAX (Germany, Cat.no. RR320A) and 4.5 μl ddH2O. All samples were subjected to primary denaturation and Taq polymerase activation at 95 □ C for 10 min. and final extension cycle at 72 □ C for 10 min. The primers, cycling conditions and product sizes were summarized in Table (1).

Table (1): Primers and cycling conditions of the genes PMQR genes

Gene	Primers	Amplified segment (bp)	Secondary denaturation	Annealing	Extension
qepA	CGTGTTGCTGGAGTTCTTC	403	94°C 45 sec.	50°C 45 sec	72°C 45 sec
	CTGCAGGTACTGCGTCATG				
qnrA	ATTTCTCACGCCAGGATTTG	516	94°C 45 sec.	53°C 45 sec	72°C 60 sec
	GATCGGCAAAGGTTAGGTCA				
qnrB	GATCGTGAAAGCCAGAAAGG	469			
	ACGATGCCTGGTAGTTGTCC				
qnrS	ACGACATTCGTCAACTGCAA	417			
	TAAATTGGCACCCTGTAGGC				
aac(6')-Ib-cr	CCCGCTTTCTCGTAGCA	113	94°C 30 sec.	52°C 30 sec	72°C 30 sec
	TTAGGCATCACTGCGTCTTC				

RESULTS

The number and percentage of resistant *E.coli* isolates in relation to the geographic area and time of sample collection were summarized in Table (2).

Table (2): Sample collection and *E.coli* isolation in relation to geographic area and time of collection

	Area				Time			Total
	Ismailia	Port Said	Suez	North Sinai	May	June	July	
No. of farms	15	1	2	3	7	5	9	21
No. of samples	75	5	10	15	35	25	45	105
No. of Isolated *E.coli*	53	4	2	11	20	20	30	70
No. of resistant *E.coli*	36	4	2	9	18	13	20	51
Percentage of resistant *E.coli*	67.9	100	100	81.8	90	65	66.6	72.8

Antimicrobial resistance pattern: The 70 *E.coli* isolates showed a susceptibility/resistance pattern against ciprofloxacin, enrofloxacin, norfloxacin as Flouroquinolones in addition to different other antimicrobial types, summarized in Table (3).

Table (3): The susceptibility and resistance pattern of *E.coli* Isolates against flouroquinolones and other antimicrobials by Disc Diffusion Method

No.	Cip5	Enr5	Nor10	G10	FFC30	Amp10	Am25	S10	Dox10	L2	CE30	KZ30
S	2	2	0	24	0	1	1	9	0	0	0	0
I	17	6	11	6	0	0	0	5	52	0	0	0
R	51	62	59	40	70	69	69	56	18	70	70	70

Cip5:ciprofloxacin 5µg, Enr5:enrofloxacin 5µg, Nor10:norfoxacin 10µg, G10:gentamicin10µg,
FFC30:florfenicole 30µg Amp10:ampicillin 10µg, Am25:amoxicillin 25µg, S10:streptomycin 10µg,
Dox10:doxycyclin 10µg, L2:lincomycin 2µg CE30:cepheradine 30µg, KZ30: cephrazoline 30µg.
S: susceptible, I: intermediate, R: resistant **(according to NCCLS, 2007)**

Two *E.coli* isolates were susceptible to both ciprofloxacin and enrofloxacin with MIC of 0.312 (µg/ml) and also to gentamicin, but resistant to all other antimicrobials used in the study. 51 *E.coli* isolates (72.8%) were found resistant to ciprofloxacin and enrofloxacin with MIC range of 4-32 and 8-32 (µg/ml) respectively in addition to at least 6 other antimicrobials.

Serotyping: *E.coli* serotypes of resistant isolates were summarized in Table (4).

Table (4): different serotypes of resistant *E.coli* isolates

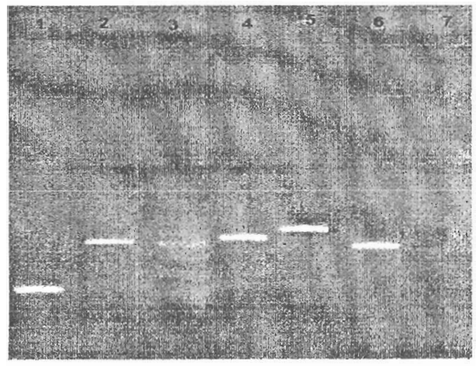
serotype	Poly I					Poly II		Poly III				un-typable	A typical *E.coli*
	O44:K74	O185:K-	O26:K60	O91:K-	O114:K90	O86:K61	O111:K58	O145:K-	O78:K80	O103:K-	O25:K11		
No. of isolates	4	6	5	2	5	3	3	2	3	4	2	11	1

Screening for the Flouroquinolones resistance genes: Among the total of 51 resistant isolates, PMQR determinants were present in 36 (70.5%), with qnr, aac(6′)-Ib-cr, and qepA being detected alone or in combination in 27 (52.9%), 20 (39.2%), and 35 (68.6%) strains, respectively. Among the twenty seven strains positive for qnr genes, 16 were positive for qnrA, qnrB and qnrS separately. Four strains (7.8%) were positive for all investigated genes while 4 other strains harboured qepA alone (the only strains that harboured only one gene). 20 strains carried both aac(6′)-Ib-cr and qepA coexisted. Detailed information on these PMQR determinant-positive isolates is given in Table (5).

Table (5): Prevalence of PMQR Genes among *Escherichia coli* resistant Isolates (n=51)

	Positive for PMQR Genes							Negative for PMQR genes
	qnr genes	qnrA	qnrB	qnrS	aac(6′)-Ib-cr	qep A	All genes together	
No. of resistant strains	27	16	16	16	20	35	4	15
percentage of resistant strains	52.9	31.37	31.37	31.37	39.2	68.6	7.8	29.5
MIC range: Ciprofloxacin	4-32	4-32	4-32	4-32	4-32	4-32	16-32	8-16
Enrofloxacin	8-32	8-32	8-32	8-32	8-32	8-32	16-32	8-16

Lane 1: aac(6′)Ib-cr 113 bp
Lane 2: qnrS 417 bp
Lane 3: Marked DNA Molecular Weight Marker Gel Pilot 100 bp ladder (cat. no. 239035), QIAGEN (USA).
Lane 4: qnrB 469 bp
Lane 5: qnrA 516 bp
Lane 6: qepA 403 bp
Lane 7: Negative control



Agarose Gel Showing Bands of Amplified PCR Products of *PMQR* Genes of *E. coli* Isolated from diseased chickens

DISSCUSION

Antibacterial resistance is a global problem especially in developing countries like Egypt. Our investigation took advantage of the repeated and high sounded complains from commercial broilers farms suffering from treatment failure of their flocks and admitting samples to our laboratory for sensitivity test. The emergence of strains showing resistance to several quinolones is a public health concern. Before the early 1990s, clinical isolates of *E. coli* rarely showed resistance to quinolones. However, since then the frequency of resistance has significantly increased worldwide ***(Hopkins et al., 2005).*** In this study, 72.8% of *E. coli* isolates had high resistance to some flouroquinolones in addition to multiresistance to other antimicrobials. Resistance to 7 quinolone antimicrobial agents was observed in more than 50% of the *E.coli* isolates from chickens during 2000-2008 in China by ***Chen et al. (2011)*** and considered that prevalence ratio very high.

Different serotypes were identified among resistant *E.coli* isolates (about 11 serotypes). Despite that avian pathogenic *E. coli* most commonly belongs to O1, O2, or O78 and typically possesses virulence factors ***(Mellata et al., 2003)***, we detected other serotypes of mostly O185, O44, O114, O26, O103. These findings agree with those found by ***Fatma Yousseff et al. (2008)*** who isolated also different serotypes of *E.coli* from layers and broilers in Ismailia, Egypt (O78, O26, O1, O2, O157 and O111) showing multiresistance to danofloxacin, oxytetracycline, ampicillin and amoxicillin and ***Fatma et al. (2012)*** who identified about

6 serotypes of *E.coli* (O18, O114, O78, O103, O86 and O12) isolated from breeders and broilers poultry farms in Mansoura, Egypt, with evidence of multiresistant O78 and O114 to streptomycin, flumequine and nalidexic acid. ***Mai Kandil et al. (2011)*** isolated O78:K80, O86:K61 from chicken meat, O26:K60 from mastitic cow while O114:K58 from cow meat from different sources in Egypt. It is interesting to indicate that O86:K61 identified in current study (3/51) was isolated and identified also from urine of haemolytic urimic humans in Egypt (12/110) by ***Osman et al. (2012)***.

Our findings of 36 (70.5%) resistant *E.coli* strains from diseased chickens positive for PMQR genes in comparison to ***Ma et al. (2009)*** findings; of 34.7 and 66.7% from food producing animals and companion animals respectively, were considered high and can be related to the difference in the health status of hosts; as we collected samples from diseased chickens suffering from treatment failure.

Since the first plasmid-mediated quinolone resistance gene qnrA was reported in 1998 ***(Martînez-Martînez et al., 1998)***, three major groups of qnr genes, qnrA, qnrB, and qnrS, have been identified in various enterobacterial species with a wide range of prevalence ***(Cheung et al., 2005; Jeong et al., 2005; Paauw et al., 2006; Chen et al., 2006; Wu et al., 2007)***. The overall qnr prevalence ranged from 0.2% to 50%, depending on the strain selection criteria used in each study. In our study the frequency of qnr genes (at least one of them) was 52.9% which is considered high compared to 26.6% found by ***Hassan et al. (2012)*** in ESBL producing *E.coli* clinical isolates from human in Egypt.

Lascols et al. (2007), Cesaro et al. (2008) and Kuo et al. (2009) have shown that ciprofloxacin resistance (MIC ≥ 4 µg/ml) is closely related to the number of topoisomerase mutations in *gyrA* and *parC*, and further to the additional harbouring of *qnr* gene in *E. coli* strains. Similar results were found in this study that all of the resistant *E.coli* isolates, harbouring at least one of the 5 genes investigated, resisted ciprofloxacin and enrofloxacin with MIC rang of 4-32 and 8-32 µg/ml respectively, including also the resistant isolates harbouring no genes (with MIC 8-16 µg/ml for both ciprofloxacin and enrofoxacin) and suggesting to have a number of topoisomerase mutations in gyrA and ParC.

The aac(6′)-Ib gene encodes a common aminoglycoside acetyltransferase responsible for resistance to aminoglycoside antibiotics such as kanamycin, amikacin and tobramycin ***(Vakulenko and Mobashery,2003).*** The aac(6′)-Ib-cr is a variant of the aac(6′)-Ib gene, in which twelve base pairs at the 5′ end are different, and which harbours mutations at codons 102 (Trp102Arg) and 179 (Asp179Tyr). Consequently, the variant enzyme acetylates ciprofloxacin and norfloxacin, conferring slightly higher MICs with a 2-to 4-fold increase ***(Robicsek et al., 2006b).*** This may explain why the resistant *E.coli* isolates in our study carring aac(6′)-Ib-cr were multiresistant to ciprofloxacin, enrofloxacin and gentamicin. The aac(6′)-Ib-cr gene, which is capable of modifying ciprofloxacin and reducing its activity, seems to have recently emerged, but in our study it was even more prevalent than the qnr A or B or S genes with prevalence of 39.2% which is high compared to 23.3% found by ***Hassan et al.(2012)*** in human

E.coli clinical isolates from Egypt, and 1% resulted by ***Gosling et al. (2012)*** in ciprofloxacin resistant *E.coli* isolates from Turkey in England, when both *qnrA* and *aac(6 ')-Ib-cr* are present in the same cell, the level of resistance is increased fourfold more than that conferred by *qnrA* alone ***(Robicsek et al.,2006b).*** We could not confirm that genetic combination effect on MIC from our findings as the same MIC range was recorded for qnr genes found separately or combined with aac(6')-Ib-cr (4-32µg/ml).

The recently identified qepA gene was detected in *E. coli* isolates from pigs ***(Liu et al., 2008)***, chickens ***(Wu et al., 2009)***, companion and food producing animals ***(Ma et al. , 2009)*** and human ***(Xi et al., 2009)*** in China, and in various Gram-negative bacteria isolated in other countries of Asia ***(Chen et al., 2006, Yamane et al., 2007)***, Europe ***(Bogaerts et al., 2007; Périchon et al., 2007)*** and *E.coli* isolated from human in Egypt ***(Ghada IIelaly et al., 2010).*** Their prevalence findings ranged from 0.2-15.8%. Our finding of the prevalence of qepA gene (68.6%) in resistant *E. coli* isolates was considered high in comparison to other countries. Unfortunately, due to the low data available about the flouroquinolone resistance in *E.coli* isolated from Egypt, we could not confirm our results in resistant *E.coli* isolated from chickens as ***Ghada IIelay et al. (2010)*** and ***IIassan et al. (2012)*** detected only qepA gene in only 4.4% (2/45) and 6.6% (2/30) respectively of human flouroquinolones resistant *E.coli.*

In conclusion, the results obtained in this study showed that flouroquinolones resistance and PMQR determinants were highly prevalent in *E.coli* strains from diseased chickens in Egypt and call for a national wide surveillance program to monitor microbial trends and antimicrobial resistance patterns in both medical practice and animal husbandry.

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التعريف الظاهري والجيني للميكروب القولوني المقاوم للفلوروكينولون المعزول من الدجاج المصاب في منطقة قناة السويس

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تم عزل عدد 70 سلالة من الميكروب القولوني من عينات (الكبد و القلب) التي تم جمعها من الدجاج المصاب (عدد = 105) في حوالي 21 مزرعة تجارية تقع في منطقة قناة السويس في مصر خلال الفترة من شهر مايو إلي يوليو 2012. تم اختبارالميكروبات من حيث حساسيتها لبعض أفراد مجموعة الفلوروكينولون (سيبروفلوكساسين ، انروفلوكساسين ، نورفلوكساسين) وبعض المضادات الحيوية الأخرى (جنتاميسين ، الستربتوميسين ، دوكسي سايكلين ، أموكسيسيلين ، الأمبيسلين ،سيفازولين، سيفرادين ، لينكومايسين ، فلورفينيكول) و قياس التركيز الأدني المثبط للميكروب من المضاد البكتيري سيبروفلوكساسين و انروفلوكساسين . تلي ذلك تصنيف سيرولوجي للمعزولات التي تم التعرف الظاهري علي مقاومتها للفلوروكينولون ، وكذلك الكشف علي مدي انتشار الجينات المحملة علي البلازميد والمسئولة عن المقاومة البكتيرية للفلوروكينولون (qnrA, qnrB, qnrS, aac(6`)-Ib-cr, and qepA)، من خلال تفاعل البلمرة المتسلسل. بين المعزلات القولونية كانت 72.8 ٪ (70/51) مقاومة لل سيبروفلوكساسين و انروفلوكساسين وكذلك مقاومة

لـ 6 على الأقل من المضادات الحيوية الأخرى قيد الدراسة . بين المعزولات المتعددة المقاومة للمضادات الحيوية وعددها 51 ، كانت 36 (70.5 ٪) إيجابية للجينات المحملة علي البلازميد، حيث كانت 27 (52.9 ٪) إيجابية ل جين واحد على الأقل من الجينات قيد التحقيق في الدراسة، 16 (31.37 ٪) كانت ايجابية لل qnrA ، qnrB و qnrS كلاًعلى حدي ، 20 (39.2 ٪) إيجابية للجين aac(6`)-Ib-cr ، بينما 35 (68.6٪) كانت ايجابية ل qepA ، في حين 4 (7.8 ٪) كانت ايجابية لجميع الجينات. مقاومة الميكروب القولوني للفلوروكينولون في مصر قد تزايد في السنوات العشرين الماضية ولكن للأسف لا بيانات كافية حول المعزلات ومدي إحتواءها علي الجينات المحملة علي البلازميد والقابلة للإنتقال بين أفراد الميكروب الواحد أو بين اجيال هذا الميكروب أو إلي ميكروبات أخري قد تكون أكثر خطورة في إصابتها سواء في مجال الدواجن أو الإنسان. مطلوب مزيد من البحث بشأن مشكلة مقاومة الميكروبات للمضادات الحيوية سواء علي المستوي البيطري أو البشري في مصر ويوصى ببرنامج لمسح وتعقب هذه المشكلة علي مستوي القطر المصري.

الكلمات الدالة : المقاومة البكتيرية للفلوروكينولون' الدواجن ، الميكروب القولوني ، جينات المقاومة المحملة علي البلازميد qnrA, qnrB, qnrS, aac(6`)-Ib-cr, and qepA