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Prevalence of *qnr*, *aac(6′)-Ib-cr*, *qepA*, and *oqxAB* in *Escherichia coli* Isolates from Humans, Animals, and the Environment

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***qnr*, *aac(6′)-Ib-cr*, *qepA*, and *oqxAB* genes were detected in 5.7%, 4.9%, 2.6%, and 20.2% of 1,022 *Escherichia coli* isolates from humans, animals, and the environment, respectively, collected between 1993 and 2010 in China. The prevalence of *oqxAB* in porcine isolates (51.0%) was significantly higher than that in other isolates. This is the first report of *oqxAB*-positive isolates from ducks and geese and as early as 1994 from chickens.**

Quinolone resistance was thought to be acquired only by chromosomal mutations, until plasmid-mediated quinolone resistance (PMQR) was described in 1998 (9). Since then, five major groups of Qnr determinants (QnrA, QnrB, QnrC, QnrD, and QnrS) have been identified (14, 16). Two additional PMQR determinants, Aac(6′)-Ib-cr (13) and quinolone extrusion by QepA or OqxAB (14, 16), have been also described. OqxAB, conferring resistance to quinoxaline-di-*N*-oxide olaquindox (a quinoxaline derivative used as a veterinary growth promoter) was originally identified in an *Escherichia coli* isolate from swine manure (6, 15). PMQR genes are increasingly being identified worldwide in clinical isolates of *Enterobacteriaceae*. However, OqxAB was not recognized as a PMQR determinant until recently. Thus, data on the prevalence and epidemiology of *oqxAB* are limited compared with data on other PMQR genes (16). Here, we report on the prevalence of PMQR genes, including *oqxAB*, in a collection of *E. coli* isolates from humans, animals, and the environment in China.

In total, 1,022 *E. coli* isolates were collected from China between 1993 and 2010. A total of 307 isolates were obtained from feces and urine samples from healthy volunteers or patients, 671

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TABLE 1 PCR primers used to detect plasmid-mediated quinolone resistance genes

Primer	Sequence (5′–3′)	Target	T_m (°C)[a]	Size of product (bp)	Reference or source
qnrA-F qnrA-R	AGAGGATTTCTCACGCCAGG GCAGCACTATKACTCCCAAGG	*qnrA*	57	619	This study
qnrB-F qnrB-R	GGMATHGAAATTCGCCACTG TTTGCYGYYCGCCAGTCGAA	*qnrB*	57	264	Cattoir et al. (2)
qnrC-F qnrC-R	GGGTTGTACATTTATTGAATC TCCACTTTACGAGGTTCT	*qnrC*	57	447	Wang et al. (18)
qnrD-F qnrD-R	CGAGATCAATTTACGGGGAATA AACAAGCTGAAGCGCCTG	*qnrD*	57	582	Cavaco et al. (3)
qnrS-F qnrS-R	GCAAGTTCATTGAACAGGCT TCTAAACCGTCGAGTTCGGCG	*qnrS*	57	428	Cattoir et al. (2)
qepA-F qepA-R	CTGCAGGTACTGCGTCATG CGTGTTGCTGGAGTTCTTC	*qepA*	60	403	Cattior et al. (1)
oqxA-F oqxA-R	GACAGCGTCGCACAGAATG GGAGACGAGGTTGGTATGGA	*oqxA*	62	339	This study
oqxB-F oqxB-R	CGAAGAAAGACCTCCCTACCC CGCCGCCAATGAGATACA	*oqxB*	62	240	This study
aac-F aac-R	TTGCGATGCTCTATGAGTGGCTA CTCGAATGCCTGGCGTGTTT	*aac(6′)-Ib*	57	482	Park et al. (12)

[a] Melting temperature.

TABLE 2 Distribution of PMQR genes in 1,022 *E. coli* isolates of human, animal, and environmental origins

Source	No. of isolates	% of PMQR genes (no. of isolates)						
		qnrA	*qnrB*	*qnrS*	*aac(6′)-Ib-cr*	*qepA*	*oqxAB*	PMQR
Humans								
Commensal isolates	52			3.8 (2)			3.8 (2)	7.7 (4)
Diarrheal isolates	42			7.1 (3)		4.8 (2)	9.5 (4)	19.0 (8)
Extraintestinal isolates	213		0.5 (1)	1.4 (3)	5.2 (11)	4.2 (9)	4.7 (10)	15.0 (32)
Total	307		0.3 (1)	2.6 (8)	3.6 (11)	3.6 (11)	5.2 (16)	14.3 (44)
Chickens								
Commensal isolates	16		12.5 (2)	18.8 (3)	43.8 (7)		18.8 (3)	68.8 (11)
Diarrheal isolates	7					57.1 (4)	28.6 (2)	57.1 (4)
Extraintestinal isolates	361		0.8 (3)	0.6 (2)	1.4 (5)	0.3 (1)	19.7 (71)	21.1 (76)
Total	384		1.3 (5)	1.3 (5)	3.1 (12)	1.3 (5)	19.8 (76)	23.7 (91)
Pigs								
Commensal isolates	10			40.0 (4)	10.0 (1)		50.0 (5)	70.0 (7)
Diarrheal isolates	173	2.3 (4)		7.5 (13)	6.9 (12)	5.2 (9)	47.4 (82)	56.1 (97)
Extraintestinal isolates	15						93.3 (14)	93.3 (14)
Total	198	2.0 (4)		8.6 (17)	6.6 (13)	4.5 (9)	51.0 (101)	59.6 (118)
Other animals[a]								
Commensal isolates	69		2.9 (2)	4.3 (3)	5.8 (4)		2.9 (2)	10.1 (7)
Diarrheal isolates	8			12.5 (1)		25.0 (2)		37.5 (3)
Extraintestinal isolates	12						16.7 (2)	16.7 (2)
Total	89		2.2 (2)	4.5 (4)	4.5 (4)	2.2 (2)	4.5 (4)	13.5 (12)
Environment	44		6.8 (3)	20.5 (9)	22.7 (10)		20.5 (9)	36.4 (16)
Total	1,022	0.4 (4)	1.1 (11)	4.2 (43)	4.9 (50)	2.6 (27)	20.2 (206)	27.5 (281)

[a] Other animals include cattle (32 isolates), dogs (6 isolates), ducks (11 isolates), and geese (40 isolates).

isolates were obtained from heart, liver, spleen, blood, or feces samples of diseased or healthy animals (specifically, 384 chickens, 32 cattle, 6 dogs, 11 ducks, 40 geese, and 198 pigs), and 44 isolates were randomly collected from the environment on different farms, including surface soil, sewage, drinking water, and pond water. Each isolate was from a separate specimen.

All isolates were screened for *oqxA* and other PMQR genes [i.e., *qnrA*, *qnrB*, *qnrC*, *qnrD*, *qnrS*, *aac(6′)-Ib-cr*, and *qepA*] by PCR (Table 1). All *oqxA*-positive isolates were also screened for *oqxB* (8). Both strands of the purified PCR products were sequenced, and *qnr* alleles were assigned by referring to the *qnr* gene nomenclature (7). All isolates PCR positive for *aac(6′)-Ib* were further analyzed by digestion with FokI and/or direct sequencing to identify *aac(6′)-Ib-cr*.

Among the 1,022 *E. coli* isolates, PMQR genes were present in 281 (27.5%) isolates; *qnr*, *aac(6′)-Ib-cr*, *qepA*, and *oqxAB* were detected alone or in combination in 58 (5.7%), 50 (4.9%), 27 (2.6%), and 206 (20.2%) isolates, respectively. None of the isolates carried *qnrC* or *qnrD*. The detected *qnr* genes included 1 *qnrA1*, 3 *qnrA3*, 1 *qnrB2*, 1 *qnrB4*, 7 *qnrB9*, 2 *qnrB10*, 35 *qnrS1*, and 8 *qnrS2* genes. PMQR genes were detected in isolates from chickens (23.7%), ducks (27.3%), geese (15.0%), pigs (59.6%), humans (14.3%), dogs (50.0%), and the environment (36.4%). In this study, *oqxAB* was the most common PMQR gene and was found as early as 1994 from chickens, whereas *qnrA*, *qnrB*, *qnrS*, *aac(6′)-Ib-cr*, and *qepA* emerged in 2004 from pigs, in 2007 from humans, in 2003 from pigs, in 2003 from pigs, and in 2003 from chickens, respectively. Notably, 42 isolates in this study were positive for two PMQR genes, while 9 isolates were positive for three PMQR genes. Isolates with more than one PMQR gene were commonly isolated from the environment (25.0%; 11/44).

The prevalence of PMQR genes in animal intestinal isolates was 45.6% (129/283), which was significantly higher than those in the animal extraintestinal isolates (23.7%) and human isolates (14.3%) ($P < 0.005$). The prevalence of *oqxAB* in animal isolates was 27.0% (181/671), which was significantly higher than that in human isolates (5.2%) ($P < 0.005$). A surprisingly high prevalence of *oqxAB* (39.0%) was recently detected in *E. coli* isolates from animals, farmworkers, and the environment in Guangdong province during 2002 (19). The prevalence of *oqxAB* in China was significantly higher than those previously reported for Denmark, Sweden (1.8%), and South Korea (0.4%) (14). In this study, the prevalence of *oqxAB* in pigs (51.0%; 101/198) was significantly higher than those in chickens (19.8%; 76/384) and other animals (4.5%; 4/89) ($P < 0.005$) (Table 2). Olaquindox was commonly used as a therapeutic and preventive antibiotic in swine in China and was allowed at a concentration of 50 ppm in feed for pigs below 35 kg. However, olaquindox was forbidden in poultry and aquaculture since 2001 (10), which may explain the relatively low prevalence of *oqxAB* in chickens, ducks, geese, cattle, and dogs.

TABLE 3 Results on conjugative transfer experiments and QRDR status[d]

			MIC (μg/ml)[a]							QRDR[c] mutation(s) in:	
Strain	PMQR determinant(s)	Specimen	NAL	OLA	CIP	NOR	OFX	LVX	MDR phenotype[b]	*gyrA*	*parC*
U027	*aac(6′)-Ib-cr*	Human urine	512	8	0.25	1	0.5	0.25	AMP, TET, SXT, CHL, GEN, CEF , AMK	S83L	WT
T-U027	*aac(6′)-Ib-cr*		4	8	0.016	0.06	0.03	0.03	AMP, TET, SXT, CHL, GEN		
U054	*aac(6′)-Ib-cr*	Human urine	>1024	4	64	256	32	16	AMP, TET, SXT, CHL, GEN, CEF, CAZ, ATM	S83L, D87N	S80I
T-U054	*aac(6′)-Ib-cr*		4	8	0.016	0.06	0.03	0.03	AMP		
U072	*aac(6′)-Ib-cr*	Human urine	>1024	4	1	8	2	0.5	AMP, TET, SXT, GEN, STR, CEF	S83L	S80I
T-U072	*aac(6′)-Ib-cr*		8	4	0.016	0.06	0.03	0.03	AMP, TET, SXT, STR, CEF		
U175	*aac(6′)-Ib-cr*	Human urine	>1024	16	128	256	32	32	AMP, TET, SXT, CHL, STR, CEF, CAZ, ATM, AMK	S83L, D87N	S80I
T-U175	*aac(6′)-Ib-cr*		4	4	0.008	0.06	0.03	0.03	AMP, TET, SXT, CHL, STR, CEF		
U220	*aac(6′)-Ib-cr*	Human urine	>1024	8	256	256	32	16	AMP, TET, SXT, GEN, STR, CEF, CAZ	S83L, D87N	S80I
T-U220	*aac(6′)-Ib-cr*		4	8	0.016	0.06	0.03	0.03	AMP, TET, SXT, STR, CEF		
U242	*aac(6′)-Ib-cr*	Human urine	>1024	16	128	256	32	16	AMP, TET, CEF	S83L, D87N	S80I
T-U242	*aac(6′)-Ib-cr*		4	8	0.016	0.06	0.06	0.03	AMP, TET		
U015	*qepA*	Human urine	>1024	16	128	>256	32	16	AMP, TET, SXT, GEN, STR, CEF, CAZ, CTX, ATM, AMK	S83L, D87N	S80I
T-U015	*qepA*		4	8	0.03	0.25	0.03	0.016	AMP, TET, SXT, GEN, CEF, CTX, ATM , AMK		
U155	*qepA*	Human urine	>1024	4	128	256	16	16	AMP, TET, SXT, CHL, GEN, STR, CEF, CAZ, CTX, ATM	S83L, D87N	S80I
T-U155	*qepA*		4	8	0.06	0.25	0.06	0.03	AMP, GEN, CEF, CAZ, CTX, ATM		
U222	*qepA*	Human urine	>1024	8	>256	>256	64	32	AMP, TET, SXT, CHL, GEN, STR, PIP, CEF, CTX, AMK	S80L, D87N	S80I
T-U222	*qepA*		4	8	0.016	0.125	0.03	0.016	AMP, SXT, CHL, GEN, PIP, CEF, AMK		
C023	*qnrA1*, *oqxAB*, *aac(6′)-Ib-cr*	Pig feces	>1024	128	8	32	16	8	AMP, TET, SXT, GEN, STR	S83L	S80I
T-C023	*qnrA1*, *aac(6′)-Ib-cr*		16	8	0.25	1	0.5	0.25	AMP, GEN		
C040	*qnrA3*, *aac(6′)-Ib-cr*	Pig feces	32	2	≤0.125	0.5	0.25	0.25	AMP, TET, SXT, GEN, STR	WT	WT
T-C040	*qnrA3*, *aac(6′)-Ib-cr*		16	4	0.125	0.5	0.25	0.125	AMP, TET, SXT		
C041	*qnrA3*, *aac(6′)-Ib-cr*	Pig feces	16	2	≤0.125	0.5	0.25	0.25	AMP, TET, SXT, GEN, STR	WT	WT
T-C041	*qnrA3*, *aac(6′)-Ib-cr*		16	4	0.25	0.5	0.25	0.125	AMP, TET, SXT		
C042	*qnrA3*, *aac(6′)-Ib-cr*	Pig feces	16	2	≤0.125	0.5	0.5	0.25	AMP, TET, SXT, GEN, STR	WT	WT
T-C042	*qnrA3*, *aac(6′)-Ib-cr*		16	4	0.125	0.5	0.25	0.125	AMP, TET, SXT		
C053	*qnrS1*	Pig feces	8	8	≤0.125	0.25	0.5	0.25	AMP, CHL	WT	WT
T-C053	*qnrS1*		32	4	0.25	0.5	1	0.5	AMP, CHL		
C058	*qnrS1*	Pig feces	16	32	≤0.125	0.25	0.5	0.25	AMP, TET, SXT	WT	WT
T-C058	*qnrS1*		32	4	0.25	0.5	1	0.5	AMP		
C111	*qnrS1*	Pig feces	256	8	0.5	1	4	2	AMP, TET, SXT, CHL, STR, PIP	S83L	WT
T-C111	*qnrS1*		32	4	0.25	0.5	1	0.5	AMP, TET, SXT, CHL, PIP		
C112	*qnrS1*	Pig feces	256	8	0.5	1	8	2	AMP, TET, SXT, CHL, STR, PIP	S83L	WT
T-C112	*qnrS1*		32	4	0.25	0.5	1	0.5	AMP, TET, SXT, CHL, PIP		
C113	*qnrS1*	Pig feces	256	16	0.5	1	4	4	AMP, TET, SXT, CHL, STR, PIP	S83L	WT
T-C113	*qnrS1*		32	4	0.25	0.5	1	0.5	AMP, TET, SXT, CHL, PIP		
C194	*qnrS1*	Pig feces	16	32	≤0.125	0.25	0.5	0.25	AMP, TET, GEN, STR, FOF	WT	A56T
T-C194	*qnrS1*		32	4	0.125	0.5	1	0.5	AMP		
C261	*qnrS1*	Human feces	32	4	≤0.125	0.25	0.5	0.25	AMP, TET, SXT	WT	WT
T-C261	*qnrS1*		32	4	0.25	0.5	1	0.5	TET, SXT		
C263	*qnrS1*	Dog feces	16	8	≤0.125	0.5	0.5	0.5	AMP, TET, SXT, GEN, STR, PIP	WT	WT
T-C263	*qnrS1*		16	4	0.125	0.5	1	0.5	AMP		

(Continued on following page)

TABLE 3 (Continued)

			MIC (μg/ml)[a]							QRDR[c] mutation(s) in:	
Strain	PMQR determinant(s)	Specimen	NAL	OLA	CIP	NOR	OFX	LVX	MDR phenotype[b]	*gyrA*	*parC*
C389	*qnrS1*	Chicken feces	32	8	≤0.125	0.25	0.5	0.5	AMP, TET, SXT, FOF	WT	WT
T-C389	*qnrS1*		32	4	0.25	0.5	1	0.5	TET, SXT		
U033	*qnrS1*	Human urine	>1024	4	16	128	64	32	AMP, TET, GEN, PIP	S83L, D87N	S80I
T-U033	*qnrS1*		16	8	0.5	1	1	0.5	AMP, TET, GEN, PIP		
U116	*qnrS1*	Human urine	>1024	16	32	128	64	64	AMP, TET, GEN, PIP	S83L, D87N	S80I
T-U116	*qnrS1*		32	8	0.25	0.5	1	0.5	AMP, TET, GEN, PIP		
U145	*qnrS1*	Human urine	>1024	32	64	256	128	64	AMP, TET, GEN, CEF	S83L, D87N	S80I
T-U145	*qnrS1*		32	4	0.25	0.5	1	0.5	AMP, TET		
C193	*qnrS1*, *oqxAB*	Pig feces	64	64	0.25	0.5	2	1	AMP, TET, SXT, GEN, STR, FOF	WT	A56T
T-C193	*qnrS1*		32	4	0.25	0.5	1	0.5	AMP, TET, SXT		
C544	*qnrS1*, *oqxAB*	Chicken feces	64	128	0.25	1	2	0.5	AMP, TET, SXT, CHL, GEN, STR, PIP, CEF, CTX, AMK, FOF	WT	WT
T-C544	*qnrS1*		32	4	0.125	0.5	1	0.5	AMP, TET, SXT, CHL, STR		
C052	*qnrS1*, *oqxAB*	Pig feces	32	128	0.25	0.5	1	0.5	AMP, TET, SXT, CHL, GEN, STR, PIP	WT	WT
T-C052	*qnrS1*		32	4	0.125	0.5	1	0.5	AMP, CHL		
C054	*qnrS1*, *oqxAB*	Pig feces	32	64	0.25	0.5	1	0.5	AMP, TET, SXT, CHL, GEN, STR	WT	WT
T-C054	*qnrS1*		32	8	0.125	0.5	1	0.5	AMP, CHL		
C055	*qnrS1*, *oqxAB*	Pig feces	32	64	≤0.125	0.5	1	0.5	AMP, TET, SXT, CHL, GEN, STR	WT	WT
T-C055	*qnrS1*		16	4	0.125	0.5	1	0.5	AMP, CHL		
C594	*qnrS1*, *oqxAB*	Dust	32	128	≤0.125	0.5	1	0.5	TET, SXT, CHL	WT	WT
T-C594	*qnrS1*, *oqxAB*		64	32	0.25	1	1	0.5	TET, SXT, CHL		
C709	*qnrS1*, *oqxAB*	Dust	32	128	0.25	0.5	1	0.5	AMP, TET, AMK, SXT, CHL	WT	WT
T-C709	*qnrS1*, *oqxAB*		64	32	0.25	1	2	0.5	TET, SXT, CHL		
C056	*qnrS1*, *oqxAB*, *aac(6′)-Ib-cr*	Pig feces	128	128	1	2	2	0.5	AMP, TET, SXT, CHL, GEN, STR, FOF	WT	WT
T-C056	*oqxAB*, *aac(6′)-Ib-cr*		32	256	0.03	0.125	0.06	0.03	AMP, TET, SXT, CHL, GEN		
C578	*qnrS1*, *aac(6′)-Ib-cr*	Duck feces	32	32	0.5	1	1	0.5	AMP, TET, SXT	WT	WT
T-C578	*qnrS1*, *aac(6′)-Ib-cr*		32	8	0.5	1	1	0.5	AMP, TET, SXT		
C197	*qnrS2*, *aac(6′)-Ib-cr*	Pig feces	32	4	0.25	1	0.5	0.25	AMP, TET, SXT, STR	WT	WT
T-C197	*qnrS2*, *aac(6′)-Ib-cr*		32	4	0.5	1	1	0.5	AMP, TET, SXT		
C265	*oqxAB*, *aac(6′)-Ib-cr*	Chicken liver	>1024	128	8	32	8	4	AMP, TET, SXT, CHL, GEN, CEF	S83L, D87N	S80R
T-C265	*oqxAB*, *aac(6′)-Ib-cr*		32	64	0.03	0.125	0.06	0.03	AMP, TET, SXT, CHL		
C324	*oqxAB*, *aac(6′)-Ib-cr*	Chicken liver	>1024	256	32	64	16	8	AMP, TET, SXT, CHL, GEN, CEF, CAZ	S83L, D87N	S80I
T-C324	*oqxAB*, *aac(6′)-Ib-cr*		16	128	0.016	0.125	0.06	0.03	AMP, TET, SXT, CHL		
C327	*oqxAB*, *aac(6′)-Ib-cr*	Chicken liver	>1024	512	64	128	32	16	AMP, TET, SXT, CHL, GEN, CEF, ATM, AMK, FOF, NIT	S83L, D87G	S80I
T-C327	*oqxAB*, *aac(6′)-Ib-cr*		16	128	0.016	0.125	0.06	0.03	AMP, TET, SXT, CHL		
C034	*oqxAB*	Pig feces	>1024	128	8	32	16	8	AMP, TET, SXT, PIP, GEN, STR	S83L, D87Y	S80I
T-C034	*oqxAB*		32	64	0.016	0.125	0.06	0.03	TET, GEN		
C671	*oqxAB*	Chicken feces	>1024	128	16	32	16	8	AMP, TET, SXT, CHL, STR, CEF	S83L, D87N	S80I
T-C671	*oqxAB*		32	64	0.03	0.125	0.06	0.03	AMP, TET, SXT, PIP, CEF		
U080	*oqxAB*	Human urine	>1024	256	64	128	32	16	AMP, TET, SXT, CHL, STR	S83L, D87N	S80I
T-U080	*oqxAB*		32	64	0.03	0.125	0.06	0.03	AMP, TET, SXT, CHL		
J53 Az^{r}			4	8	0.008	0.016	0.03	0.016			

[a] CIP, ciprofloxacin; LVX, levofloxacin; NAL, nalidixic acid; NOR, norfloxacin; OFX, ofloxacin; OLA, olaquindox.

[b] Multidrug resistance (MDR) phenotype abbreviations are as follows: AMK, amikacin; AMP, ampicillin; ATM, aztreonam; CAZ, ceftazidime; CEF, cephalothin; CHL, chloramphenicol; CTX, cefotaxime; FOF, fosfomycin; GEN, gentamicin; NIT, nitrofurantoin; PIP, piperacillin; STR, streptomycin; SXT, trimethoprim-sulfamethoxazole; TET, tetracycline.

[c] QRDR, quinolone resistance-determining region; S83L, mutation of the amino acid at codon 83 from S to L (etc.); WT, wild type (i.e., no mutation).

[d] The “T-” prefix indicates a transconjugant.

In these 281 PMQR-positive isolates, 89 isolates with distinct PMQR genes or sources (specifically, 27 humans, 24 chickens, 24 pigs, 9 environmental sources, and 5 other animals) were selected for conjugation experiments using J53 Az^r (i.e., azide resistant) as the recipient strain (17). Transconjugants were selected on tryptic soy agar plates containing sodium azide (100 μg/ml) and tetracycline (20 μg/ml), chloramphenicol (50 μg/ml), gentamicin (8 μg/ml), or amoxicillin (100 μg/ml). A total of 41 transconjugants were successfully obtained at a frequency of 10^{-7} to 10^{-3} cells per recipient. Nine (22.5%) transconjugants carrying *oqxAB* were successfully obtained from 40 OqxAB-producing isolates. Cotransfer of resistance to ampicillin, tetracycline, trimethoprim-sulfamethoxazole, and chloramphenicol was observed in 36 (87.8%), 30 (73.2%), 26 (63.4%), and 20 (48.8%) of the 41 transconjugants, respectively. MICs of 41 *E. coli* isolates (including 15 *oqxAB*-positive and 26 *oqxAB*-negative isolates) were determined by the broth microdilution method according to CLSI guidelines (4, 5). The isolates with *oqxAB* had olaquindox MICs of ≥64 μg/ml. Transconjugants carrying *oqxAB* showed 4- to 32-fold increases in olaquindox MICs compared with those of the recipient. This is consistent with the *oqxAB* genotype, suggesting that *oqxAB* has a role in olaquindox resistance, as reported by other studies (6, 19). Transfer of the *qnr* gene can elevate ciprofloxacin MICs by 16- to 64-fold relative to those of the recipient, which is greater than the effects of *qepA* and *aac(6′)-Ib-cr* (Table 3).

The quinolone resistance-determining regions (QRDRs) of the *gyrA* and *parC* genes in PMQR-positive isolates were sequenced to confirm the mutations as previously described (11). Of the 41 *E. coli* isolates, 17 (41.5%) had wild-type *gyrA* and *parC* genes, and these isolates had ciprofloxacin MICs ranging from ≤0.125 to 1 μg/ml. Mutations in both *gyrA* (S83 and D87) and *parC* (S80) were detected in 16 (39.0%) isolates, with ciprofloxacin MICs of 8 to >256 μg/ml. In the absence of *oqxAB*, olaquindox MICs in the isolates without QRDRs mutations were similar to those of isolates with up to three mutations (4 to 32 μg/ml), suggesting that the QRDR mutations do not affect olaquindox susceptibility (Table 3).

In conclusion, *oqxAB* was prevalent and widespread in *E. coli* isolates from humans, animals, and the environment in China. This study is the first report on the occurrence of *oqxAB* in isolates from ducks and geese and as early as 1994 from chickens.

Nucleotide sequence accession numbers. The sequences of the *qnr* genes found in this study were deposited in GenBank under accession numbers JF773308 to JF773350.

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