Fluoroquinolone-resistant *Salmonella* sp. in Carcasses

To the Editor: Fluoroquinolone (FQ)-resistant *Salmonella* has been isolated from patients in Taiwan (*1–7*). Recently, a report further indicated that several patients were infected with *Salmonella enterica* serovar Schwarzengrund with high-level FQ resistance (*1*). *S.* Schwarzengrund has never been isolated from food animals in Taiwan.

We report the isolation of FQ-resistant strains from pork and broiler carcasses sampled from 2000 to 2003: 27 in 2000, 3 in 2001, 4 in 2002, and 2 in 2003. These isolates made up 18.85% of the 191 *Salmonella* strains obtained from pork and broiler carcasses in the study period. Of these isolates, 16 FQ-resistant *S.* Schwarzengrund strains were further analyzed to elucidate the possible mechanism of FQ resistance. Ciprofloxacin MIC levels in these isolates ranged from 4 to 16 μg/mL, and all had high-level nalidixic acid resistance (≥1,024 μg/mL). All of the 16 investigated strains displayed mutations possibly associated with high-level FQ resistance. The mutation sites included 2 sites (Ser83Phe and Asp87Gly) in the quinolone resistance–determining region (QRDR) of *gyrA*, 2 sites (Thr57Ser and Ser80Arg) in the QRDR of *parC*, and 1 site (Ser458Pro) in the QRDR of *parE*, respectively. Four strains had mutations in the QRDR of *gyrA* and *parC* only but not in the QRDR of *parE* (Table).

In conclusion, high-level FQ resistance was detected in *S.* Schwarzengrund isolated from pork and chicken in Taiwan. Specific mutation sites of *gyrA*, *parC*, and *parE* were associated with high-level FQ resistance in all the isolates investigated. Our results warrant further investigation of the public health consequences of FQ use in food animals in Taiwan.

Table. Characteristics of ciprofloxacin-resistant *Salmonella enterica* serovar Schwarzengrund strains from carcasses*†

Strain no.	Origin*	Year isolated	Antimicrobial drug resistance profile	Quinolone MICs (μg/mL)				Substitutions in QRDR‡		
				NAL	FLU	ENR	CIP	*gyrA*	*parC*	*parE*
A5	B, M	2000	CmSxtTc	1,024	512	32	8	Ser83Phe; Asp87Gly	Thr57Ser; Ser80Arg	Ser458Pro
A16	P, E	2000	ApCmNSxtTc	2,048	512	32	8	Ser83Phe; Asp87Gly	Thr57Ser; Ser80Arg	Ser458Pro
A17	P, E	2000	ApCmNSxtTc	2,048	512	32	16	Ser83Phe; Asp87Gly	Thr57Ser; Ser80Arg	Ser458Pro
A18	P, E	2000	ApCmNSxtTc	2,048	512	32	16	Ser83Phe; Asp87Gly	Thr57Ser; Ser80Arg	Ser458Pro
A19	P, E	2000	ApCmCnNSxtTc	1,024	512	32	8	Ser83Phe; Asp87Gly	Thr57Ser; Ser80Arg	Ser458Pro
A20	P, E	2000	ApCmNSxtTc	2,048	512	32	8	Ser83Phe; Asp87Gly	Thr57Ser; Ser80Arg	Ser458Pro
A29	B, S	2000	CmNSxtTc	1,024	512	32	8	Ser83Phe; Asp87Gly	Thr57Ser; Ser80Arg	Ser458Pro
A36	B, S	2000	ApCmSxtTc	1,024	512	32	8	Ser83Phe; Asp87Gly	Thr57Ser; Ser80Arg	Ser458Pro
A41	P, S	2000	ApCmCnNSxtTc	1,024	512	32	16	Ser83Phe; Asp87Gly	Thr57Ser; Ser80Arg	Ser458Pro
A45	P, S	2000	ApCmNSxtTc	1,024	512	32	16	Ser83Phe; Asp87Gly	Thr57Ser; Ser80Arg	
A51	P, S	2000	ApCmCnNSxtTc	1,024	512	16	4	Ser83Phe; Asp87Gly	Thr57Ser; Ser80Arg	
A56	B, M	2000	ApCmCnNSxtTc	2,048	512	64	16	Ser83Phe; Asp87Gly	Thr57Ser; Ser80Arg	
A61	P, S	2000	CmSxtTc	1,024	512	32	8	Ser83Phe; Asp87Gly	Thr57Ser;Ser 80Arg	Ser458Pro
A62	P, S	2000	ApCmCnSxtTc	2,048	512	64	16	Ser83Phe; Asp87Gly	Thr57Ser; Ser80Arg	
B16	P, E	2001	ApCmCnCroTc	2,048	512	32	16	Ser83Phe; Asp87Gly	Thr57Ser; Ser80Arg	Ser458Pro
B73	P, N	2003	ApCmCnNSxtTc	2,048	512	32	16	Ser83Phe; Asp87Gly	Thr57Ser; Ser80Arg	Ser458Pro

*QRDR, quinolone resistance–determining region; B, broiler; M, middle Taiwan; P, pork; E, east Taiwan; S, south Taiwan; N, north Taiwan.
†Antimicrobial agents are ampicillin (Ap), chloramphenicol (Cm), ciprofloxacin (CIP), enrofloxacin (ENR), flumequine (FLU), gentamicin (Cn), ceftriaxone (Cro), nalidixic acid (NAL),neomycin (N), trimethoprim/sulfamethoxazole (Sxt), and tetracycline (Tc).
‡No *gyrB* substitutions were detected.

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Cocirculation of Dengue Serotypes, Delhi, India, 2003

To the Editor: Delhi, in the northern part of India, has had outbreaks of dengue caused by various dengue virus types in 1967, 1970, 1982, 1988, and 1996 (*1*–*5*). In 1988, for the first time, a few cases of dengue hemorrhagic fever (DHF) were seen (*4*). Subsequently, we reported the largest outbreak of DHF/dengue shock syndrome (DSS) in Delhi in 1996 and confirmed dengue virus type 2 as the etiologic agent (*5*).

We report the results of virologic testing of samples received at the All India Institute of Medical Sciences from patients with suspected dengue fever or denguelike illness from Delhi and its adjoining areas during a 2003 outbreak of dengue. According to the World Health Organization (*6*), 2,185 laboratory-confirmed cases were reported during this outbreak.

Of the blood samples received by the virology laboratory, 42 were received on ice from patients with acute denguelike illness. Serum was separated aseptically and stored at –70°C. The standard method of virus cultivation, which used the C6/36 clone of the *Aedes albopictus* cell line, was followed with some modifications (*7*). On days 5 and 10, harvested cells were tested by an indirect immunofluorescence assay (IFA) using monoclonal antibodies to dengue virus types 1–4 (provided by the Centers for Disease Control and Prevention, Atlanta, Georgia, USA, during the 1996 outbreak). If IFA results were negative for dengue viruses on first passage, a second passage was made, and cells were again harvested on days 5 and 10 for IFA. The 4 dengue virus types (obtained from the National Institute of Virology, Pune, India) were included as positive controls, and uninfected C6/36 cells were kept as negative controls.

Dengue virus could be isolated in C6/36 cells from 8 (19%) of 42 samples processed for virus isolation (Table). Of the 8 isolates, two each were identified as dengue virus types 1 and 2, three as type 3, and one as type 4. All but one isolate were from patients with uncomplicated dengue fever. One dengue type 2 isolate was obtained from a 7-year-old boy with secondary dengue infection and DHF/DSS. The ages of culture-positive patients ranged from 5 to 62 years, with a median of 22 years. These patients were equally distributed between children (<12 years) and adults. The male-to-female ratio for these 8 patients was 5:3. The duration of fever at the time of viral isolation was 1–5 days, with a median of 3 days.

All previous outbreaks in Delhi have occurred during the monsoon (rainy) season between August and November and subsided with the onset of winter. We recently reported the results of serologic testing during the 2003 outbreak, which also occurred from September to November, with a peak in mid-October 2003 (*8*). This outbreak was

Table. Culture-positive dengue patients*

Age (y)/sex	Dengue type isolated	Secondary infection (anti–dengue IgG antibodies + by ELISA)	Duration of fever (d)
9/M	DENV-1	Yes	4
25/F	DENV-3	No	4
7/M	DENV-2	Yes	5
7/F	DENV-4	No	1
40/F	DENV-1	Yes	3
62/M	DENV-2	Yes	3
39/M	DENV-3	Yes	2
5/M	DENV-3	No	3

*ELISA, enzyme-linked immunosorbent assay; IgG, immunoglobulin G.