

with regard to molecular typing, and Angela Huber and Marion Linder Mayer for expert technical assistance. We dedicate this paper to Jürgen Heeseemann, the first head of the German Consiliary Laboratory for Diphtheria, on the occasion of his 60th birthday.

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***Campylobacter jejuni* HS:23 and Guillain-Barré Syndrome, Bangladesh**

To the Editor: Guillain-Barré syndrome (GBS) is an acute peripheral neuropathy triggered by a preceding infectious illness. Gastroenteritis caused by *Campylobacter jejuni* is the most frequently reported antecedent event (1). In Japan, South Africa, China, and Mexico, *Campylobacter* strains with certain Penner heat-stable (HS) serotypes, including HS:19 and HS:41, are overrepresented among isolates from GBS case-patients, compared with isolates from enteritis case-patients (2,3). Several studies indicate that *C. jejuni* HS:19 and HS:41 have a clonal population structure and suggest that these serotypes might have unique virulence properties that are intricately linked to development of GBS (4). However, data from the United Kingdom and the Netherlands suggest that such virulence properties may not be restricted to specific HS serotypes because many other serotypes can be cultured from patients with GBS (5). We report a non-HS:19 and non-HS:41 *C. jejuni* serotype and sequence type (ST)–3219 that are overrepresented among isolates from GBS patients in Bangladesh.

We conducted a prospective case-control study of the serotype and genotype of *C. jejuni* associated with GBS in Bangladesh. Case-patients were 97 persons with GBS admitted to Dhaka Medical College Hospital, Bangabandhu Sheikh Mujib Medical University, and Dhaka Central Hospital during July 2006–June 2007. All fulfilled the diagnostic criteria for GBS of the National Institute of Neurological Disorders and Stroke of the US National Institutes of Health (Bethesda, MD, USA) (6). The control group comprised 97 patients with other neurologic diseases, matched with

case-patients by sex, age, and date of admission to the hospital. A second control group comprised 97 healthy family members of case-patients. Up to 3 stool samples were cultured from each case-patient and control.

Campylobacter strains were presumptively identified with Gram stain, oxidase, and hippurate hydrolysis tests and confirmed with a *C. jejuni* species-specific PCR. Serotyping was performed at the National Laboratory for Enteric Pathogens, Canadian Science Centre for Human and Animal Health, Winnipeg, Manitoba, Canada. All strains were serotyped according to the HS serotyping schemes of Penner et al. (7). To determine the class of lipooligosaccharides (LOS) locus in each of the *C. jejuni* strains, genomic DNA was isolated by using the DNeasy tissue kit (QIAGEN, Venlo, the Netherlands). PCR analysis was performed with primer sets specific for classes A, B, C, D, and E (8).

We isolated *C. jejuni* from fecal samples of 10 case-patients. *Campylobacter* strains were not isolated from the control groups ($p < 0.001$). Serotyping of the 10 GBS-related strains showed 4 different HS serotypes. *C. jejuni* HS:23 was found in 5 (50%) strains; HS:19, in 2 (20%); HS:55 and HS:21, in 1 strain each. One strain was untypeable according to the HS typing scheme. In a collection of clinical *C. jejuni* isolated during the same period from patients with enteritis, HS:23

was encountered in 9 (28%) of 32 patients. Serotypes previously associated with GBS were HS:1, HS:2, HS:4, HS:4/50, HS:5, HS:10, HS:13/65, HS:16, HS:19, HS:23, HS:35, HS:37, HS:41, HS:44, and HS:64 (5,9).

Nine (90%) of the *C. jejuni* isolates from the case-patients had the class A or class B LOS, which are highly associated with the presence of ganglioside-mimicking structures in LOS (10). Godschalk et al. found that 14 (82%) of 17 GBS-associated isolates possessed a class A/B/C locus (8). Parker et al. (10) found that all GBS-related strains and 64% of the other clinical and environmental isolates belonged to LOS class A/B/C loci. The expression of ganglioside-mimicking structures in *Campylobacter*; LOS is considered essential for the induction of autoantibodies that lead to GBS. Godschalk et al. (8) demonstrated that specific genes involved in *C. jejuni* LOS biosynthesis are crucial for the induction of antiganglioside antibodies that lead to GBS.

We performed multilocus sequence typing to examine the overall genomic variation among 10 GBS-related *C. jejuni* strains. We identified 6 different STs among the GBS-related *C. jejuni* strains (Table). However, ST-3219 has a new combination of alleles and was identified in 4 strains. Concordantly, the analysis demonstrated that *C. jejuni* isolates with serotype HS:23 were all ST-3219. Of particu-

lar interest, ST-985 (BD-67) shared 5 alleles (*aspA*, *uncA*, *glnA*, *glyA*, *pgm*) with ST-3219 (Table).

Our findings of a *C. jejuni* HS:23 serotype and ST-3219 that is highly prevalent among GBS-related *C. jejuni* strains from Bangladesh are consistent with previous observations that specific LOS types and serotypes are overrepresented among GBS-related *C. jejuni* strains. These observations support the hypothesis that, although a great variety of *C. jejuni* serotypes can be isolated from GBS patients in some geographic areas, specific clonal serotypes and multilocus types are prevalent in GBS patients in other places. The association of GBS with *C. jejuni* LOS class A/B/C is the only consistent finding when universal collections of GBS-associated strains are considered.

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Table. Serotyping and multilocus sequence typing analysis of *Campylobacter jejuni* strains associated with Guillain-Barré syndrome, Bangladesh*

Strain	Year	Disease	LOS class	Penner type(s)†	ST	Allele, no.						
						<i>aspA</i>	<i>glnA</i>	<i>gltA</i>	<i>glyA</i>	<i>pgm</i>	<i>tkt</i>	<i>uncA</i>
BD-07	2006	GBS	A	HS:19	22	1	3	6	4	3	3	3
BD-10	2006	GBS/MFS	B	HS:23	3219	10	27	33	19	10	5	7
BD-22	2006	GBS	B	HS:23	3219	10	27	33	19	10	5	7
BD-27	2006	GBS	A	UT	587	1	2	42	4	90	25	8
BD-34	2006	GBS	B	HS:23	3219	10	27	33	19	10	5	7
BD-39	2006	GBS	A	HS:19	660	1	3	6	4	54	91	3
BD-67	2007	GBS/MFS	B	HS:23	985	10	27	89	19	10	132	7
BD-74	2007	GBS/MFS	B	HS:23	3219	10	27	33	19	10	5	7
BD-75	2007	GBS	A	HS:55	587	1	2	42	4	90	25	8
BD-94	2007	GBS	E	HS:21	2109	4	7	10	4	10	7	1

*LOS, lipooligosaccharides; ST, sequence type; GBS, Guillain-Barré syndrome; HS, heat stable; MFS, Miller-Fisher syndrome; UT, untypeable.

†Penner HS serotypes.

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Enzootic Sparganosis in Guangdong, People's Republic of China

To the Editor: Sparganosis is a worldwide parasitic zoonosis caused by infection with spargana, the plerocercoid larvae of various diphylobothroid tapeworms belonging to the genus *Spirometra* (1–3). Sparganosis poses a serious threat to human health; the spargana invade mainly the brain, eye, abdominal cavity, spinal cord, and subcutaneous tissues; can damage local tissues; and can cause blindness, paralysis, and even death (4,5).

In the People's Republic of China, sparganosis has emerged as an important foodborne parasitic disease, with ≈1,000 human cases reported in 22 provinces during 1927–2007. Guangdong Province has the most cases (6).

Persons in Guangdong Province eat frog meat and place frog poultices made from raw frog meat on open wounds and lesions, which facilitates human infection with spargana. To assess the risk for human infection with sparganosis in this province and to strengthen public food safety awareness, we conducted a comprehensive investigation of spargana infection in frogs, the second intermediate host of *Spirometra*.

By necropsy we examined for spargana 544 frogs (446 *Rana nigromaculata* and 98 *R. tigrina*) from Yunfu, Maoming, and Zhanjiang in western Guangdong Province during October 2007–October 2008 (7). Of these 544 frogs, 455 were wild, and 89 were aquacultured. Spargana were found in 27.3% (124/455) of examined wild frogs; of these, 30.0% (107/357) were *R. nigromaculata*, significantly more ($p < 0.05$) than the 17.3% (17/98) that were *R. tigrina*. This finding suggests that *R. nigromaculata* is the main intermediate host of *Spirometra* in western Guangdong Province.

We found 719 spargana in infected wild frogs. The number of worms per frog ranged from 1 to 41, with an average of 5.8 worms per infected frog. No spargana were found in 89 aquacultured *R. nigromaculata* frogs.

The examined wild frogs looked normal and healthy and had no obvious symptoms. During necropsy, we detected local edema, muscle bleeding, and fragile tissues in the tissues invaded by spargana. We also found cysts in some tissues that contained 1 or a few worms. Spargana dissected from host tissue were flat, white worms, which continuously crept in the normal saline. These worms ranged from 2 mm to 115 mm long and from 1 mm to 2 mm wide.

Frogs are the second intermediate hosts of *Spirometra* spp.; pigs, mice, and humans become infected as paratenic hosts by ingesting *Spirometra* larvae in cyclops or frogs (8,9). Because persons in Guangdong