with regard to molecular typing, and Angela Huber and Marion Lindermayer for expert technical assistance. We dedicate this paper to Jürgen Heesemann, 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

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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 lipoooligosaccharides (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 particular 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*

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

*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 plecercoid larvae of various diphyllobothroid 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 sparganosis 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

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