

## Common Somatic O and Heat-Labile Serotypes among *Campylobacter* Strains from Sporadic Infections in the United States

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Somatic O (formerly heat-stable) and heat-labile (HL) serotyping methods are commonly used to type *Campylobacter jejuni* and *Campylobacter coli* isolates. Although both systems are effective, the labor and time required for each have limited their application. These systems can be simplified by reducing the number of antisera used. To find an appropriate panel of antisera, we determined the distribution of common serotypes in the United States among a representative sample of 298 *Campylobacter* isolates. The strains, obtained between July 1989 and June 1990 from persons with sporadic cases of diarrhea, were collected from 19 randomly chosen counties in all geographic (census) regions of the United States. All strains were serotyped by the O and HL systems. By phenotypic methods, 288 *C. jejuni*, 9 hippurate-negative *C. jejuni/C. coli*, and 1 *Campylobacter lari* were identified. Of 57 O antisera, 24 typed 252 (84.6%) strains. Of the 55 HL antisera, 23 serotyped 253 (84.9%) strains. All strains were typeable in the unabsorbed O antisera. In the absorbed HL antisera, four strains were nontypeable and 14 were rough and untypeable. In each geographic region, 9 or more O and HL serotypes were found. Serotypes O:1, O:4, and O:13,16,43,50 and HL 1 were identified in all regions. The combination of both schemes gave greater discrimination than either system alone, but the maintenance of both requires a large resource investment. A serotyping scheme incorporating the 24 most prevalent O and 23 most prevalent HL serotypes could be useful for outbreak support and for surveillance. In the near future, we anticipate using a molecular subtyping method in combination with limited serotyping to distinguish *Campylobacter* strains.

Thermophilic *Campylobacter* spp., particularly *Campylobacter jejuni* and *Campylobacter coli*, have been isolated from a number of sources, including humans, pets, wild birds, and farm and laboratory animals. Environmental sources include foods and untreated surface water. In recent years, much interest has been shown in the pathogenicity of *Campylobacter* species for humans, and multiple studies have found that *C. jejuni/C. coli* is the most common enterobacterial cause of diarrheal disease in developed countries (40).

Numerous typing methods have been developed to differentiate campylobacters for epidemiologic purposes. These typing schemes have provided a mechanism to determine the relatedness of isolates and have identified sources and modes of transmission. The application and evaluation of approximately 35 typing methods or modifications of methods applied to *C. jejuni* have been recently reviewed (29).

At present, the most widely used typing procedures are the somatic O (formerly heat-stable) serotyping scheme of Penner and Hennessy (30) and the heat-labile (HL) serotyping scheme of Lior et al. (23). The molecular basis for the heat-stable antigenic diversity in *C. jejuni* and *C. coli* was found to consist of somatic (O) lipopolysaccharides (24, 33); hence, the term somatic O serotyping is used in lieu of heat-stable serotyping. The HL scheme is based on flagellar or nonflagellar, undetermined antigen (1). The O scheme recognizes 60 serotypes (42 *C. jejuni* and 18 *C. coli*) and employs a passive hemagglutination technique with un-

absorbed antisera. The HL scheme presently recognizes 128 serotypes with antisera prepared from 74 *C. jejuni*, 44 *C. coli*, and 10 *Campylobacter lari* strains (22a). The HL antigens are detected on live cells by testing first with polyvalent, pooled, unabsorbed antisera, second with single, unabsorbed antiserum, and finally with single, absorbed antiserum.

Although both schemes have been used worldwide and both have been effective in differentiating isolates, routine use and application of either scheme is problematic and impractical for most clinical, reference, and research laboratories. Both methods are time and labor intensive. The O scheme occasionally detects high-titer, transient antigens (28), and the use of unabsorbed antisera can produce cross-reacting serotype complexes such as O:4,13,16,43,50 (25, 31). Repeat testing is often required. To identify the 128 serotypes in the HL scheme, 272 antisera must be prepared and carefully monitored (16 antisera pools; 128 single, unabsorbed antisera; and 128 single, absorbed antisera). Multiple absorptions are often necessary, requiring large amounts of media and incubator space (29); resulting titers may be low.

Because of these problems, several methods or simplifications of the O and HL procedures have been suggested (2, 9, 11, 16, 25, 43, 46). These include limiting the number of antisera and combining both serotyping systems (17, 18) or combining one system with biotyping (15) or phage typing (36) or using all three (21, 22). Previous studies indicate that a set of 10 to 12 antisera will type as many as 75% of strains (10, 17, 19, 27, 32, 38, 39). In this report, we identify the most commonly occurring serotypes of *C. jejuni* in the United States and propose establishing a limited set of

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antisera for use in the *Campylobacter* Reference Laboratory at the Centers for Disease Control and Prevention (CDC).

### MATERIALS AND METHODS

**Bacterial strains.** Between July 1989 and June 1990, a group of 298 *Campylobacter* isolates was systematically collected to evaluate the serotype distribution and antimicrobial resistance patterns of *Campylobacter* strains throughout the United States. These isolates came from persons with diarrheal illness who resided in one of 19 counties representing all nine standard census regions of the United States plus California. Within a census region, participating counties were randomly chosen from among those that had reported >25 cases of campylobacteriosis in 1987 and 1988. Each selected site was requested to submit to CDC *Campylobacter* isolates from up to the first five persons reported to the county health department with a *Campylobacter* infection during each month of the study period. For analysis, the Southeast and E. South Central regions were combined into a single entity identified as the Southeast region.

The isolates were identified by biochemical tests by the submitting laboratory and sent to CDC for confirmation and further testing. Identification was confirmed by cellular morphology and motility by dark-field examination, hippurate hydrolysis by the ninhydrin tube test, and confirmation of negative results by the gas-liquid chromatography hippurate hydrolysis method (26). By phenotypic tests, 288 *C. jejuni*, 9 hippurate-negative *C. jejuni/C. coli*, and 1 *C. lari* were identified.

**Serotyping procedures.** All strains were serotyped by the O and HL systems as described previously (28). Strains were tested in each scheme after three to five subcultures. Strains that were nonreactive or only weakly reactive in a single O antiserum and strains that were nontypeable, rough, or weakly reactive in a single HL antiserum were subcultured an additional five times and retested. A total of 63 and 170 repeat tests were performed for the O and HL schemes, respectively.

The panel of O antisera included serotypes O:1 to O:56 and contained two O:5 antisera, one prepared to a *C. jejuni* strain (O:5<sup>+</sup>) and one prepared to a *C. coli* strain (O:5<sup>-</sup>). The HL antisera panel included serotypes HL 1 to HL 60, except for HL 3, 37, and 43, for which no serotype strains have been designated, and HL 42, 51, 58, and 59, for which no antisera were available at CDC. J. L. Penner kindly serotyped five strains with a panel of 60 O antisera, and H. Lior kindly serotyped 19 strains with a panel of 128 HL antisera.

### RESULTS

**Somatic O serotyping.** All strains were typeable in the 57 unabsorbed O antisera, with 74 serotypes identified. Of the 298 strains, 143 (48.0%) reacted in one antiserum, 84 (38.3%) reacted in two antisera, 24 (8.0%) reacted in three antisera, and 17 (5.7%) reacted in four or more antisera.

Among the 57 O antisera, 12 antisera (O:1, 2, 3, 4, 5<sup>-</sup>, 5<sup>+</sup>, 8, 13, 16, 17, 43, and 50) identified 186 (62.4%) strains. Six additional antisera (O:6, 7, 15, 19, 25, and 38) increased the number of serotyped strains to 220 (73.8%). Twenty-four antisera identified 252 (84.6%) strains. The most frequently identified serotypes and the common cross-reacting antigens are listed in Table 1.

Sixty strains reacted in O antisera of the 4,13,16,43,50 complex and were identified as serotype O:4 (27 strains),

TABLE 1. Occurrence of common somatic O and HL serotypes in the United States among 298 *Campylobacter* strains

| Serotype                             | No. of strains |
|--------------------------------------|----------------|
| <b>O<sup>a</sup></b>                 |                |
| 1 or 1,8.....                        | 34             |
| 13,16,43,50.....                     | 33             |
| 8 or 8,17.....                       | 29             |
| 4.....                               | 27             |
| 5 <sup>-</sup> ,5 <sup>+</sup> ..... | 24             |
| 2.....                               | 22             |
| 3.....                               | 17             |
| 6,7,25(29) <sup>b</sup> .....        | 14             |
| 19.....                              | 10             |
| 15,38.....                           | 10             |
| 10,17.....                           | 9              |
| 11.....                              | 9              |
| 23,36.....                           | 7              |
| 21 or 21,29.....                     | 7              |
| <b>HL<sup>c</sup></b>                |                |
| 1.....                               | 34             |
| 4.....                               | 29             |
| 9.....                               | 22             |
| 36.....                              | 22             |
| 2.....                               | 19             |
| 33.....                              | 16             |
| 8.....                               | 12             |
| 6.....                               | 11             |
| 13.....                              | 10             |
| 7.....                               | 9              |
| 49.....                              | 9              |
| 11.....                              | 8              |
| 16.....                              | 8              |
| 24.....                              | 7              |
| 5.....                               | 6              |
| 41,60.....                           | 6              |
| 70.....                              | 5              |
| 17.....                              | 4              |
| 28.....                              | 4              |
| 32.....                              | 4              |
| 38.....                              | 4              |
| 40.....                              | 4              |

<sup>a</sup> O:5<sup>-</sup> and O:25 are *C. coli* serotypes. Others listed are *C. jejuni* serotypes.

<sup>b</sup> ( ), presence of antigen varies.

<sup>c</sup> HL serotype includes strains that reacted in two or more antisera; for example, HL 1 includes HL 1,2; 1,7; 1,24; etc. See text for other combinations of HL antigens. Of 298 strains, 4 were nontypeable and 14 were rough and untypeable for HL antigens.

O:16 (2 strains), O:16,50 (10 strains), and O:4 complex (21 strains that reacted in other combinations of antisera in the 4,13,16,43,50 complex). The O:3 antigen was detected on some strains of the 4 complex serotype. For the purpose of this study, these 60 strains were identified as serotype O:4 (27 strains) and serotype O:13,16,43,50 (33 strains) (Table 1).

Thirty-four strains were identified as serotype O:1 or 1,8 and reacted in antisera O:1 (8 strains), O:1,8 (24 strains), O:1,11 (1 strain), and O:1,8,44 (1 strain).

The 29 strains that were identified as serotype O:8,17 reacted in O:8 only (22 strains), O:8,44 (1 strain), or O:8,17 (6 strains).

Fourteen strains reacted in antisera of the 6,7,25 complex. O antigens 8 and 29 were occasionally demonstrated on strains of this complex. For the purpose of this study, these 14 isolates were identified as O:6,7,25.

Twenty-four strains were identified as serotype O:5<sup>-</sup>,5<sup>+</sup>, with 1 strain reacting in O:5<sup>-</sup>; 1 strain reacting in O:5<sup>-</sup>,31; 4 strains reacting in O:5<sup>+</sup>; 1 strain reacting in O:5<sup>+</sup>,27; and 17 strains reacting in O:5<sup>-</sup>,5<sup>+</sup> antisera.

TABLE 2. Geographic distribution of *Campylobacter* O and HL serotypes in the United States

| Region                      | No. of strains | No. of different serotypes |    | Most common serotypes <sup>a</sup>                         |                              |
|-----------------------------|----------------|----------------------------|----|--|------------------------------|
|                             |                | O                          | HL | O  | HL                           |
| New England                 | 17             | 9                          | 10 | 8  | 1; 2                         |
| Mid Atlantic                | 31             | 13                         | 14 | 1; 2; 3; 8; 13,16,43,50                                    | 1; 4; 33; 36                 |
| East North Central          | 45             | 13                         | 19 | 1; 2; 3; 4; 5 <sup>-</sup> ,5 <sup>+</sup> ; 6,7,25; 10,17 | 4; 6; 9; 13; 24; 33; 36      |
| South East                  | 27             | 12                         | 15 | 1,8; 3; 5 <sup>-</sup> ,5 <sup>+</sup> ; 13,16,43,50       | 9; 36                        |
| West North Central          | 32             | 17                         | 18 | 5 <sup>-</sup> ,5 <sup>+</sup> ; 8; 13,16,43,50            | 1; 4; 36                     |
| West South Central          | 21             | 12                         | 12 | 4; 13,16,43,50; 19   | 1; 4                         |
| Mountain                    | 34             | 19                         | 16 | 1; 3; 4; 5 <sup>-</sup> ,5 <sup>+</sup> ; 13,16,43,50; 28  | 1; 8; 9; 11; 36              |
| Pacific (except California) | 73             | 21                         | 28 | 1; 2; 4; 6,7,25; 8; 13,16,43,50; 15; 19; 21,29; 23,36; 54  | 1; 2; 4; 5; 6; 8; 11; 49; 84 |
| California                  | 18             | 12                         | 11 | 2; 4; 8  | 2; 4                         |

<sup>a</sup> Three or more strains per serotype.

Strains designated as serotype O:15,38 reacted in antiserum O:15 (two strains), O:15,38 (seven strains), and O:15,42 (one strain).

The 7 strains identified as O:21,29 reacted in antiserum O:21 only (one strain) or O:21,29 (six strains). The antigens 15 and 18 were occasionally detected on strains of serotype O:21,29.

Nine strains were identified as serotype O:11; the O:8 antigen was detected on four of these strains.

The nine strains identified as O:10,17 reacted in antiserum O:10 (two strains), O:17 (one strain), or O:10,17 (six strains).

**HL serotyping.** Of the 298 strains, 280 were typeable and 4 were nontypeable in the 55 HL antisera. Fourteen strains were rough and untypeable. Seventy-one serotypes were identified among the typeable strains. Of the 280 typeable strains, 228 (81.4%) reacted in one antiserum, 49 (17.5%) reacted in two antisera, and 3 (1.1%) reacted in three antisera.

The common pairs of antigens seen in this study were HL 41,60 (six strains); HL 6,38 (five strains); HL 1,2, HL 1,7, and HL 1,24 (three strains each); and HL 2,9, HL 2,33, and HL 6,50 (two strains each). Of the 23 additional strains that reacted with two antisera, each serotype was identified only once. The three strains reacting in three antisera each were identified as serotypes HL 1,2,33, HL 1,7,36, and HL 4,5,50.

The 11 most common antisera in the HL system (HL 1, 2, 4, 6, 7, 8, 9, 13, 33, 36, and 49) identified 193 (64.8%) strains. Of these 193 strains, 153 (79.3%) reacted in only one antiserum. Six additional antisera (HL 5, 11, 16, 24, 41, and 60) identified 228 (76.5%) strains. As many as 253 (84.9%) strains were serotyped by as few as 23 HL antisera (Table 1). Included in Table 1 are the numbers of strains identified in the 23 common serotypes. Only one pair of antigens, HL 41, 60, was among the common serotypes. Our final panel of common HL antisera will not include serotype HL 17; high-quality reagents were not available.

**Geographic distribution.** Between 17 and 73 strains were serotyped from each of the nine geographic regions (Table 2). In each geographic region, 11 or more O and HL serotypes were identified (Table 2). Serotypes O:1, O:4, O:13,16,43,50, and HL 1 were identified in all regions, but these serotypes were not necessarily the most common in all regions (Table 2). No single, predominant serotype was unique to one region. O serotypes 17, 28, 54, and 21,29 are common serotypes in certain regions (Table 2); however, these serotypes were also found in fewer numbers in other geographic locations.

**Relationship of O and HL schemes.** Strains of the same O

serotype usually consisted of multiple HL serotypes and vice versa. The five most frequently occurring O serotypes with their corresponding HL serotypes are given in Table 3. Likewise, the five most commonly identified HL serotypes and their parallel O serotypes are shown in Table 4. Most strains of one O serotype have a dominant corresponding HL serotype and vice versa. For example, strains of serotype O:1 were usually serotype HL 33, and HL 4 strains were most often O:2 (Tables 3 and 4).

**Correlation of serotypes from sporadic cases and outbreaks.** The predominate serotypes among *C. jejuni* isolates from humans associated with 15 outbreaks occurring in the United States from 1978 to 1989 and investigated by CDC (3, 4, 7, 27a, 28, 35, 44) are shown in Table 5. The serotypes O:2:HL

TABLE 3. HL serotype for the five most commonly occurring O serotypes

| O serotype (no. of strains)         | HL serotype <sup>a</sup>                      | No. of strains |
|-------------------------------------|---|----------------|
| 1 or 1,8 (34)                       | 33  | 14             |
|                                     | 2   | 6              |
|                                     | Rg  | 4              |
|                                     | 1,2; 2,33                                     | 2 each         |
| 13,16,43,50 (33)                    | 4; 10; 42; 2,9; 9,33; 1,2,33                  | 1 each         |
|                                     | 7   | 6              |
|                                     | 36  | 6              |
|                                     | 1   | 5              |
|                                     | 1,7   | 3              |
| 8 or 8,17 (29)                      | 7; 16   | 2 each         |
|                                     | 2; 4; 1,24; 7,16; 7,26; 24,53; 1,7,36; NT; Rg | 1 each         |
|                                     | 49  | 9              |
|                                     | 2   | 6              |
|                                     | 1; 4; 33                                      | 2 each         |
| 4 (27)                              | 10; 36; 1,49; 2,9; 2,36; 2,49; 4,49; Rg       | 1 each         |
|                                     | 1   | 15             |
|                                     | 24  | 4              |
|                                     | 17  | 3              |
|                                     | 1,24  | 2              |
| 5 <sup>-</sup> ,5 <sup>+</sup> (24) | 55; 1,2; Rg                                   | 1 each         |
|                                     | 9   | 18             |
|                                     | Rg  | 2              |
|                                     | 32; 52; 9,32; 9,52                            | 1 each         |

<sup>a</sup> Rg, rough and untypeable; NT, nontypeable.

TABLE 4. O serotypes for the five most commonly occurring HL serotypes

| HL serotype <sup>a</sup><br>(no. of strains) | O serotype                                    | No. of<br>strains |
|--|---|-------------------|
| 1 (34)                                       | 4   | 18                |
|  | 13,16,43,50                                   | 10                |
|  | 1,8   | 3                 |
|  | 8   | 2                 |
|  | 8,17  | 1                 |
| 4 (29)                                       | 2   | 21                |
|  | 3; 8; 31; 1,8; 8,17; 8,44; 13,16,43,50; 23,36 | 1 each            |
| 9 (22)                                       | 5 <sup>-</sup> ,5 <sup>+</sup>                | 20                |
|  | 31  | 1                 |
|  | 1,8   | 1                 |
| 36 (22)                                      | 3   | 14                |
|  | 13,16,43,50                                   | 6                 |
|  | 8; 3,43                                       | 1 each            |
| 2 (19)                                       | 1,8   | 8                 |
|  | 8   | 7                 |
|  | 1; 1,11; 8,17; 13,16,43,50                    | 1 each            |

<sup>a</sup> HL serotype includes strains that reacted in two or more antisera, for example, HL 1 includes HL 1,2; 1,7; 1,24, etc. See text for other combinations of HL antigens.

4 and O:23,36:HL 5 were the most frequently encountered serotypes and were identified in 11 (73.3%) of the 15 outbreaks. All serotypes associated with outbreaks were also common serotypes found in sporadic cases in this study, except for serotypes O:27 and HL 77. Only three sporadic case isolates were serotype O:27, and none were serotype HL 77.

## DISCUSSION

The O and HL serotyping schemes are useful epidemiologic tools for differentiating *C. jejuni* and *C. coli* isolates (5, 22, 29). Although these typing schemes show a large diversity of serotypes within both species, only a limited number of serotypes are found frequently. The dominant serotypes are similar around the world, with the exception of a few

TABLE 5. Predominant O:HL serotype of *C. jejuni* isolates from humans in 15 outbreaks occurring in the United States, 1978 to 1989

| Serotype (O:HL) | No. of<br>outbreaks |
|-----------------|---------------------|
| 2:4 .....       | 7 <sup>a,b</sup>    |
| 23,36:5 .....   | 5 <sup>b,c</sup>    |
| 4:1 .....       | 1 <sup>d</sup>      |
| 16:1 .....      | 1                   |
| 16:13 .....     | 1 <sup>c</sup>      |
| 27:9 .....      | 1                   |
| 19:77 .....     | 1                   |
| 1,8:2 .....     | 1                   |

<sup>a</sup> Serotypes O:2:HL 4 and O:27:HL 9 were predominant in a food-borne outbreak (4).

<sup>b</sup> Includes serotypes O:2:HL 4 and O:23,36:HL 5 in a multistrain outbreak (3).

<sup>c</sup> Two serotypes, O:23,36:HL 5 and O:16:HL 13, were isolated from humans in a waterborne outbreak (44).

<sup>d</sup> Four other serotypes were identified in a waterborne outbreak. O:4:HL 1 was predominant (27a).

serotypes found more frequently in developing countries (29). In a previous study (28), we demonstrated that as few as 10 antisera from either the O or HL scheme serotyped over 70% of 157 isolates from sporadic cases in the United States, which occurred primarily in the Midwest. We observed similar results in this study with strains collected from all nine regions of the United States. Twelve O and 10 HL antisera identified approximately 65% of 298 strains from sporadic cases (Table 1). Approximately 85% of strains were serotyped with 24 O and 23 HL antisera.

The 24 most common O and 23 most common HL serotypes in this study (Table 1) were primarily the same as those found to be common serotypes in the O and HL schemes (31a). Discrepancies between the findings of this study and the previously identified common serotypes in the O and HL schemes included O:10, 15, 17, 25, and 38 and HL 13, 16, 24, 33, and 49. These serotypes were among the more common serotypes identified in the present study, but were not among the 24 most common O serotypes identified by Penner or the 15 most common HL serotypes from humans identified by Lior (31a).

Several laboratories have identified a large variety of O:HL combinations (17, 18, 28). Strain discrimination is obviously greater with a combination of the two systems. The value of combining the two schemes has been demonstrated in several epidemic investigations. Application of the HL scheme to strains of the same O serotype was useful in distinguishing strains from cows incriminated in an outbreak caused by raw milk outbreak (17), strains from goats in an outbreak caused by goat milk (17), and strains from humans with exposure to a common source (18). Likewise, strains of the same HL serotype were distinguished by the O system in two milk-borne outbreaks (15, 17).

Intestinal carriage of *Campylobacter* spp. by healthy animals in the United States is common. Many of the O and HL serotypes identified in the United States in dogs (8); farm animals, including poultry, cattle, sheep, pigs, goats, and horses (5, 12, 41, 45); laboratory animals, including ferrets, hamsters, cats, dogs, and nonhuman primates (34, 42); and domestic and wild birds (47) were among the common serotypes identified in the human enteritis cases in this study. Our data support the previous suggestion that healthy animals as well as those with diarrhea represent a potential reservoir for human *Campylobacter* infections (8, 12, 42, 45, 47).

Previous serotyping studies indicate that certain animals, i.e., poultry and cattle, are more important reservoirs for human infections than are other hosts, such as sheep and pigs (45). Pigs commonly carry *C. coli*, but only 3 to 5% of *Campylobacter* isolates from human gastroenteritis are attributed to *C. coli* (20). The nine hippurate-negative *C. jejuni/C. coli* strains in this study (presumably *C. coli*) represent only 3% of isolates, and all nine reacted in *C. coli*-specific heat-stable typing sera (O:26, O:30, O:34, O:46, and O:47, one strain each; O:28, four strains). These findings suggest that *C. coli* disease in the United States occurs infrequently and that pigs are an uncommon source for human disease. Five of the nine hippurate-negative strains, representing four serotypes, were isolated from persons in one state of the Mountain region.

The distribution of multiple serotypes in all regions was not surprising considering the mode of *Campylobacter* transmission is via the fecal-oral route from numerous animals, animal products, or vehicles contaminated with animal waste. The predominant source for sporadic cases in the United States is poultry (6, 13), with other important sources

being pets, unpasteurized milk, and untreated surface water such as creeks, ponds, and high mountain streams (6, 14, 37). Strains of multiple serotypes have been isolated from each of these sources. The serotypes from these sources are similar to those usually associated with human cases, including the serotypes found to be most prevalent in this study.

Serotypes associated with milk-borne and waterborne outbreaks and small clusters of cases in the United States are diverse (5) (Table 5) and are usually among the common serotypes found in sporadic cases. Two serotypes were predominant in the outbreaks investigated in the United States. These may represent more virulent or easily transmitted strains.

Because strains from sporadic cases and outbreaks are similar and because we intend to simplify serotyping, we will limit the panel of antisera used for epidemiologic purposes to the 24 most common O and 23 most common HL serotypes.

Genetic typing methods for *Campylobacter* that employ polymerase chain reaction and pulsed-field gel electrophoresis are under investigation at CDC and other institutions. It may be more cost effective to maintain one, or part of one, system and augment this with a more generic, molecular subtyping method. We would like to evaluate this approach and compare its utility with that of traditional serotyping.

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#### REFERENCES

- Alm, R. A., P. Guerry, M. E. Power, H. Lior, and T. J. Trust. 1991. Analysis of the role of flagella in the heat-labile Lior serotyping scheme of thermophilic campylobacters by mutant allele exchange. *J. Clin. Microbiol.* **29**:2438-2445.
- Bar, W., G. Fricke, and H. Goossens. 1989. Distribution of serotypes and biotypes of thermophilic campylobacters in the Federal Republic of Germany: a comparison with other countries. *Zentralbl. Bakteriol. Parasitenkd. Infektionskr. Hyg. Abt. 1 Orig.* **271**:127-134.
- Birkhead, G., R. L. Vogt, E. Heun, C. M. Evelt, and C. M. Patton. 1988. A multiple-strain outbreak of *Campylobacter* enteritis due to consumption of inadequately pasteurized milk. *J. Infect. Dis.* **157**:1095-1097.
- Blaser, M. J., and C. M. Patton. 1985. *Campylobacter* enteritis associated with foodborne transmission: new serotyping data. *Am. J. Epidemiol.* **121**:625-626.
- Blaser, M. J., J. L. Penner, and J. G. Wells. 1982. Diversity of serotypes in outbreaks of enteritis due to *Campylobacter jejuni*. *J. Infect. Dis.* **146**:826.
- Deming, M. S., R. V. Tauxe, P. A. Blake, S. E. Dixon, B. S. Fowler, T. S. Jones, E. A. Lockamy, C. M. Patton, and R. K. Sikes. 1987. *Campylobacter* enteritis at a university: transmission from eating chicken and from cats. *Am. J. Epidemiol.* **126**:526-534.
- Finch, M. J., and P. A. Blake. 1985. Foodborne outbreaks of campylobacteriosis: the United States experience. *Am. J. Epidemiol.* **122**:262-268.
- Fox, J. G., M. C. Claps, N. S. Taylor, K. O. Maxwell, J. I. Ackerman, and S. B. Hoffman. 1988. *Campylobacter jejuni/coli* in commercially reared beagles: prevalence and serotypes. *Lab. Anim. Sci.* **38**:262-265.
- Fricke, C. R., M. M. Alemohammad, and R. W. A. Park. 1987. A study of factors affecting the sensitivity of the passive haemagglutination method for serotyping *Campylobacter jejuni* and *Campylobacter coli* and recommendations for a more rapid procedure. *Can. J. Microbiol.* **33**:33-39.
- Fricke, C. R., and R. W. A. Park. 1989. A two-year study of the distribution of "thermophilic" campylobacters in human, environmental and food samples from the Reading area with particular reference to toxin production and heat-stable serotype. *J. Appl. Bacteriol.* **66**:477-490.
- Fricke, C. R., J. Uradzinske, M. M. Alemohammad, R. W. A. Park, C. Whelan, and R. W. A. Girdwood. 1986. Serotyping of campylobacters by co-agglutination on the basis of heat-stable antigens. *J. Med. Microbiol.* **21**:83-86.
- Harris, N. V., R. J. Kimball, P. Bennett, Y. Johnson, D. Wakely, and C. M. Nolan. 1987. *Campylobacter jejuni* enteritis associated with raw goat's milk. *Am. J. Epidemiol.* **126**:179-186.
- Harris, N. V., N. S. Weiss, and C. M. Nolan. 1986. The role of poultry and meats in the etiology of *Campylobacter jejuni/coli* enteritis. *Am. J. Public Health* **76**:407-411.
- Hopkins, R. S., R. Olmstead, and G. R. Istre. 1984. Endemic *Campylobacter jejuni* infections in Colorado: identified risk factors. *Am. J. Public Health* **74**:249-250.
- Hutchinson, D. N., F. J. Bolton, D. M. Jones, E. M. Sutcliffe, and J. D. Abbott. 1987. Application of three typing schemes (Penner, Lior, Preston) to strains of *Campylobacter* spp. isolated from three outbreaks. *Epidemiol. Infect.* **98**:139-144.
- Illingworth, D. S., and C. R. Fricke. 1987. Rapid serotyping of campylobacters based on heat-stable antigens using a combined passive haemagglutination/co-agglutination technique. *Lett. Appl. Microbiol.* **5**:61-63.
- Jones, D. M., E. M. Sutcliffe, and J. D. Abbott. 1985. Serotyping of *Campylobacter* species by combined use of two methods. *Eur. J. Clin. Microbiol.* **4**:562-565.
- Kajiser, B., and E. Sjogren. 1985. *Campylobacter* strains in Sweden. *Acta Pathol. Microbiol. Immunol. Scand. Sect. B* **93**:315-322.
- Karmali, M. A., J. L. Penner, P. C. Fleming, A. Williams, and J. N. Hennessy. 1983. The serotype and biotype distribution of clinical isolates of *Campylobacter jejuni* and *Campylobacter coli* over a three-year period. *J. Infect. Dis.* **147**:243-246.
- Karmali, M. A., and M. B. Skirrow. 1984. Taxonomy of the genus *Campylobacter*, p. 1-20. In J. P. Butzler (ed.), *Campylobacter* infections in man and animals. CRC Press, Boca Raton, Fla.
- Khakhria, R., and H. Lior. 1992. Extended phage-typing scheme for *Campylobacter jejuni* and *C. coli*. *Epidemiol. Infect.* **108**:403-414.
- Lior, H. 1989. Les *Campylobacter* marqueurs epidemiologiques. *Med. Malad. Infect.* **19**:18-24.
- Lior, H. Personal communication.
- Lior, H., D. L. Woodward, J. A. Edgar, L. J. Laroche, and P. Gill. 1982. Serotyping of *Campylobacter jejuni* by slide agglutination based on heat-labile antigenic factors. *J. Clin. Microbiol.* **15**:761-768.
- Mandatori, R., and J. L. Penner. 1989. Structural and antigenic properties of *Campylobacter coli* lipopolysaccharides. *Infect. Immun.* **57**:3506-3511.
- Mills, S. D., R. V. Congi, J. N. Hennessy, and J. L. Penner. 1991. Evaluation of a simplified procedure for serotyping *Campylobacter jejuni* and *Campylobacter coli* which is based on the O antigen. *J. Clin. Microbiol.* **29**:2093-2098.
- Morris, G. K., M. R. El Sherbeeny, C. M. Patton, H. Kodaka, G. L. Lombard, P. Edmonds, D. G. Hollis, and D. J. Brenner. 1985. Comparison of four hippurate hydrolysis methods for identification of thermophilic *Campylobacter* spp. *J. Clin. Microbiol.* **22**:714-718.
- Munroe, D. L., J. F. Prescott, and J. L. Penner. 1983. *Campylobacter jejuni* and *Campylobacter coli* serotypes isolated from chickens, cattle, and pigs. *J. Clin. Microbiol.* **18**:877-881.
- Patton, C. M. Unpublished data.
- Patton, C. M., T. J. Barrett, and G. K. Morris. 1985. Comparison of the Penner and Lior methods for serotyping *Campylobacter* spp. *J. Clin. Microbiol.* **22**:558-565.
- Patton, C. M., and I. K. Wachsmuth. 1992. Typing schemes—are current methods useful? p. 110-128. In I. Nachamkin, M. J.

- Blaser, and L. S. Tompkins (ed.), *Campylobacter jejuni*: current status and future trends. American Society for Microbiology, Washington, D.C.
30. Penner, J. L., and J. N. Hennessy. 1980. Passive hemagglutination technique for serotyping *Campylobacter fetus* subsp. *jejuni* on the basis of soluble heat-stable antigens. *J. Clin. Microbiol.* **12**:732-737.
  31. Penner, J. L., J. N. Hennessy, and R. V. Congi. 1983. Serotyping of *Campylobacter jejuni* and *Campylobacter coli* on the basis of thermostable antigens. *Eur. J. Clin. Microbiol.* **2**:378-383.
  - 31a. Penner, J. L., and H. Lior. Personal communication.
  32. Prescott, J. F., and O. S. Gellner. 1984. Intestinal carriage of *Campylobacter jejuni* and *Salmonella* by chicken flocks at slaughter. *Can. J. Comp. Med.* **48**:329-331.
  33. Preston, M. A., and J. L. Penner. 1987. Structural and antigenic properties of lipopolysaccharides from serotype reference strains of *Campylobacter jejuni*. *Infect. Immun.* **55**:1806-1812.
  34. Russell, R. G., J. I. Sarmiento, J. Fox, and P. Panigrahi. 1990. Evidence of reinfection with multiple strains of *Campylobacter jejuni* and *Campylobacter coli* in *Macaca nemestrina* housed under hyperendemic conditions. *Infect. Immun.* **58**:2149-2155.
  35. Sacks, J. J., S. Lieb, L. M. Baldy, S. Berta, C. M. Patton, M. C. White, W. J. Bigler, and J. J. Witte. 1986. Epidemic campylobacteriosis associated with a community water supply. *Am. J. Public Health* **76**:424-428.
  36. Salama, S. M., F. J. Bolton, and D. N. Hutchinson. 1990. Application of a new phagetyping scheme to campylobacters isolated during outbreaks. *Epidemiol. Infect.* **104**:405-411.
  37. Schmid, G. P., R. E. Schaefer, B. D. Plikaytis, J. R. Schaefer, J. H. Bryner, L. A. Wintermeyer, and A. F. Kaufmann. 1987. A one-year study of endemic campylobacteriosis in a midwestern city: association with consumption of raw milk. *J. Infect. Dis.* **156**:218-222.
  38. Sjogren, E., K. Alestig, and B. Kaijser. 1989. *Campylobacter* strains from Swedish patients with diarrhoea, distribution of serotypes over a five year period. *Acta Pathol. Microbiol. Immunol. Scand.* **97**:221-226.
  39. Sjogren, E., M. Johnny, and B. Kaijser. 1989. The serotype distribution of *Campylobacter* in patients with diarrhoea in Kuwait. *FEMS Microbiol. Lett.* **48**:237-239.
  40. Tauxe, R. V. 1992. Epidemiology of *Campylobacter jejuni* infections in the United States and other industrialized nations, p. 9-19. In I. Nachamkin, M. J. Blaser, L. S. Tompkins (eds.), *Campylobacter jejuni*: current status and future trends. American Society for Microbiology, Washington, D.C.
  41. Taylor, D. N., M. Brown, and K. T. McDermott. 1982. Waterborne transmission of *Campylobacter enteritis*. *Microb. Ecol.* **8**:347-354.
  42. Taylor, N. S., M. A. Ellenberger, P. Y. Wu, and J. G. Fox. 1989. Diversity of serotypes of *Campylobacter jejuni* and *Campylobacter coli* isolated in laboratory animals. *Lab. Anim. Sci.* **39**:219-221.
  43. Thompson, C. J., and K. A. Bettelheim. 1989. Applications of a new method for devising bacteriological serotyping schemes. *J. Clin. Microbiol.* **27**:2391-2392.
  44. Vogt, R. L., H. E. Sours, T. Barrett, R. A. Feldman, R. J. Dickinson, and L. Witherell. 1982. *Campylobacter enteritis* associated with contaminated water. *Ann. Intern. Med.* **96**:292-296.
  45. Warner, D. P., J. H. Bryner, and G. W. Beran. 1986. Epidemiologic study of campylobacteriosis in Iowa cattle and the possible role of unpasteurized milk as a vehicle of infection. *Am. J. Vet. Res.* **47**:254-258.
  46. Wong, K. H., S. K. Skelton, C. M. Patton, J. C. Feeley, and G. Morris. 1985. Typing of heat-stable and heat-labile antigens of *Campylobacter jejuni* and *Campylobacter coli* by coagglutination. *J. Clin. Microbiol.* **21**:702-707.
  47. Yogasundram, K., S. M. Shane, and K. S. Harrington. 1989. Prevalence of *Campylobacter jejuni* in selected domestic and wild birds in Louisiana. *Avian Dis.* **33**:664-667.