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Shiga Toxin-producing *Escherichia coli*, Idaho

To the Editor: Data collected from expanded surveillance study suggest that more than half of Idaho Shiga toxin-producing *Escherichia coli* (STEC) illnesses are caused by non-O157 serotypes. Using data from a regional medical center whose stool culture protocol included Shiga toxin testing, we predicted Idaho's STEC incidence to be significantly higher if non-O157 STEC *E. coli* were routinely detected by immunoassay. Recent findings suggest that the prediction was accurate in an expanded surveillance area.

Several studies have shown an increased incidence of non-O157 STEC infections in the United States. For example, a community hospital in Virginia detected non-O157 serotypes in 31% of patients with STEC from 1995–2002 (1). A 1998 Nebraska study that analyzed 30,000 diarrheal stool samples found that non-O157 and O157:H7 STEC were equally prevalent (2). Additionally, findings from a Connecticut study of laboratory-confirmed cases (3), STEC surveillance results from Montana (4), and a recent study from Michigan (5) indicate that non-O157 serotypes

comprise a substantial percentage of STEC cases.

In other countries, nonculture-based methods are routinely used for STEC detection (6). However, *E. coli* O157:H7 culture methods remain the focus in the United Kingdom, Canada, and the United States (6). Reliance on culture methods can result in misleading interpretations of STEC prevalence. For example, 93% of STEC infections in Canada are reported to be *E. coli* O157:H7, yet a Manitoba 1992 study showed that when toxin assays were used, 35% of the recovered STEC isolates were non-O157 serotypes (6).

Analysis of reported non-O157 STEC cases in Idaho showed a similar trend. From 2002–2004, 66% of Idaho's non-O157 cases originated in Health District 7, where >70% of stool cultures are screened by enzyme immunoassay (EIA) for Shiga toxin (Premier EHEC, Meridian Bioscience, Cincinnati, OH, USA). This rate was disproportionately higher than that of the remaining 6 health districts, which primarily use culture methods to screen for *E. coli* O157:H7. We hypothesized that this disproportion was due to differences in stool culture protocol. To test this premise, we conducted enhanced surveillance for 16 months in a “low” STEC incidence area, Health District 5. A total of 2,065 stools submitted for culture were screened for Shiga toxin by EIA. With this approach, reported non-O157 STEC incidence rose from <1 case/year/100,000 population to 11 cases/year/100,000 population. Additionally, 56% of recovered STEC isolates were non-O157 serotypes, mirroring the proportion of non-O157 detected in District 7. Notably, this appears to be the endemic rate for District 5 because no non-O157 STEC outbreaks or matching pulsed-field gel electrophoresis patterns were detected during the surveillance period. Although our study captured only a portion of stool cultures in Idaho, our findings

demonstrated increased prevalence of non-O157 STEC in the region when nonculture methods were used.

Two barriers cited for not routinely screening diarrheal stools for Shiga toxin are cost and perception of low non-O157 STEC incidence. While toxin testing is more expensive than culture testing, the potential effects of misdiagnosis may outweigh cost concerns. A study estimating the financial repercussions of *E. coli* O157 infections in the United States suggested that annual cost associated with this pathogen is \$405 million, with the cost per case varying from \$26 for those who do not seek medical care to \$6.2 million for a patient with fatal hemolytic uremic syndrome (HUS) (7). Non-O157 STEC infections have been an important cause of HUS in many countries. For example, a 3-year prospective study in Germany and Austria reported that non-O157 serotypes comprised 90 (43%) of 207 STEC isolates from stools of 394 pediatric patients with HUS (8). Further, a 6-year Danish study of 343 registered STEC patients found that 76% of STEC and 48% of HUS cases were attributable to non-O157 serotypes (9). In the United States, continued reliance on O157 STEC culturing hinders our ability to determine the financial effects and the

proportion of HUS cases attributable to non-O157 STEC.

Some evidence suggests that the testing focus may be changing in the United States. We used US Census Bureau population statistics to translate reported O157:H7 and non-O157 STEC cases for each state into incidence data. Despite widespread variation in STEC testing and incidence among states, there has been a significant statistical decline in the proportion of *E. coli* O157:H7 among total STEC cases every year since 2001 (Figure; $p < 0.001$) (10). Consistent with this trend, the incidence of non-O157 STEC in the United States has increased (10). This may indicate that more laboratories are adopting Shiga toxin testing protocols, as we are advocating in Idaho. Our findings suggest that perceptions of low non-O157 STEC incidence in Idaho are probably artifactual and due to overemphasis on culture methods for O157 STEC. Our ongoing EIA-based surveillance highlights the need for continued investigation of the epidemiology of non-O157 STEC disease. We conclude that O157 STEC culturing has limited usefulness in areas like the Idaho health districts investigated, where non-O157 serotypes accounted for 55% of STEC illnesses. The true involvement of non-

O157 in STEC disease will remain obscured as long as screening methods focus on traditional culture methods.

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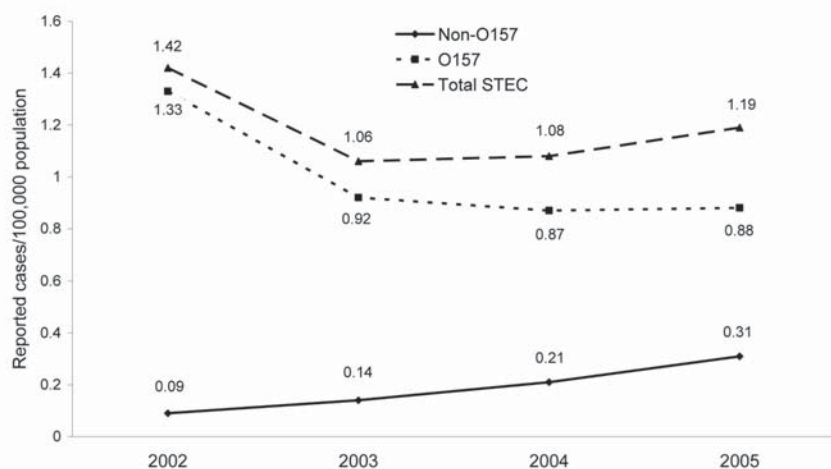


Figure. Shiga toxin-producing *Escherichia coli* (STEC) incidence trends, United States, 2002-2005.

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Imported Chikungunya Infection, Italy

To the Editor: Chikungunya virus (CHIKV) infection is a self-limiting illness characterized by fever, headache, weakness, rash, and arthralgia. Some patients have prolonged weakness or arthralgia lasting several months. In 2006, several Indian Ocean states and India had an outbreak of CHIKV infection (1,2). During the epidemic's peak, some European and American travelers returning from these areas were infected (3–6).

Because the foci of *Aedes albopictus*, 1 of the 2 main vectors of CHIKV, are now in Italy and many travelers visit CHIKV-epidemic areas, surveillance for imported cases is mandatory in Italy (7). From July to September 2006, a total of 17 confirmed cases of CHIKV infection were observed in travelers at 5 Gruppo di Interesse e Studio delle Patologie di

Importazione (GISPI) centers (Italian network of Institutes of Infectious and Tropical Diseases). Serologic diagnosis was performed with a hemagglutination-inhibition test and confirmed by a plaque-reduction neutralization test (8). Demographic and epidemiologic characteristics of these patients are reported in the Table.

Cases were distributed throughout the year with a peak from March to May 2006 (n = 10). Nine patients (53%) were men. Median age was 43 years (range 31–66 years). Several reasons for travel were reported: tourism (64.6%), visits to relatives or friends (11.8%), business (11.8%), and missionary work (5.9%). One patient was a resident in the disease-epidemic area. The median exposure time in the CHIKV-endemic area for the 15 travelers was 15 days (range 9–93 days) (missionary and resident patients were excluded). The median delay before being seen at a clinic after return was 2 days (range 0–73 days). Only 7 patients (41.2%) were hospitalized. The remainder were outpatients.

All patients had fever; arthralgia (88.2%, n = 15), weakness (70.6%, n = 12), headache (11.8%, n = 2), diarrhea (11.8%, n = 2), and gum bleeding and epistaxis (5.9%, n = 1) were other reported symptoms. The median duration of fever was 5 days (range 2–12 days). Only 7 of 16 patients (43.8%) were still febrile when first seen. Physical examination showed diffuse macular erythematous rash in 13 patients (76.5%), a similar rate to that reported among French travelers (4). Hepatomegaly was found in 2 patients (11.8%), splenomegaly in 2 (11.8%), and peripheral lymphadenopathy in 2 (11.8%).

Twelve acute-phase patients were admitted to the hospital for blood testing within 3 days of the initial examination. In contrast with results of other studies, leukopenia and thrombocytopenia were uncommon in our study. Leukopenia (leukocyte count $\leq 4,000/\mu\text{L}$) was present in 4 patients

(33.3%) and thrombocytopenia (platelet count $\leq 150,000/\mu\text{L}$) in 1 patient (8.3%). This finding may help distinguish CHIKV infection from dengue fever (4). Anemia (hemoglobin level $\leq 12 \text{ g/dL}$) was found in only 1 patient (8.3%). Alanine aminotransferase (ALT) and aspartate aminotransferase (AST) determination were available for 12 patients. ALT and AST levels were elevated ($>40 \text{ IU/L}$) in 5 (41.7%) and 2 (16.7%) patients, respectively. Seven (46.7%) of 15 patients fully recovered within 1 month; 8 patients (53.3%) reported persistent arthralgia.

Because the GISPI network provides regional coverage only, the number of imported CHIKV cases in all of Italy in 2006 was likely higher. Moreover, most patients probably did not seek medical care, and when they did, physicians may have failed to recognize the disease because of lack of familiarity with it or limited diagnostic facilities. Differential diagnosis with other arthropodborne viruses of the *Alphavirus* genus (Ross River, Barmah Forest, o'nyong nyong, Sindbis, and Mayaro viruses) is difficult, but these are comparatively rare. In contrast, dengue and CHIKV epidemics may overlap, and potential patients should be screened for both.

The potential risk for introduction and establishment of CHIKV reservoirs in areas with mosquito vectors was discussed in March 2006 by a multidisciplinary European expert panel (9). In Italy, *A. albopictus* was first recorded in 1990; it has since quickly spread across the country. Scattered foci are now reported in almost all regions, mainly along the coastal plains, from the sea to the inlands, up to an altitude of $\approx 500\text{--}600 \text{ m}$ (7).

The ability of *A. albopictus* to colonize new areas and its adaptability to the mild Italian climate allow vector populations to be active throughout the year (10). The patient is thought to be viremic for only 6–7 days (shortly before and during the febrile period) (6). We were unable to directly assess