Correspondence

Table 1. Taco Dressing That Contained Escherichia coli, Diarrheagenic E. coli Pathotypes (DEPs), and Non-O157 Shiga Toxin–Producing Escherichia coli (STEC)

<table>
<thead>
<tr>
<th>Taco dressing</th>
<th>No. of E. coli–positive samples/total no. of samples</th>
<th>DEP identified (no. of samples)</th>
<th>Gene profile (no. of DEPs samples)</th>
<th>DEP CFU/g per sample</th>
</tr>
</thead>
<tbody>
<tr>
<td>Green chili sauce</td>
<td>29/49</td>
<td>...</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>Red chili sauce</td>
<td>38/62</td>
<td>STEC (4)</td>
<td>stx1-stx2 (3)</td>
<td>1.3, 1.7, and 12.5 × 10^3</td>
</tr>
<tr>
<td></td>
<td></td>
<td>stx2 (1)</td>
<td>7.2 × 10^3</td>
<td></td>
</tr>
<tr>
<td>Pico de gallo</td>
<td>11/18</td>
<td>aEPEC (1)</td>
<td>eaeA</td>
<td>8.3 × 10^3</td>
</tr>
<tr>
<td>Guacamole</td>
<td>5/8</td>
<td>...</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>Coriander and onion</td>
<td>9/9</td>
<td>STEC (2)</td>
<td>stx1-stx2 (1)</td>
<td>5.0 × 10^2</td>
</tr>
<tr>
<td></td>
<td></td>
<td>stx2 (1)</td>
<td>1.0 × 10^2</td>
<td></td>
</tr>
<tr>
<td>Coriander</td>
<td>14/14</td>
<td>...</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>Onion</td>
<td>10/13</td>
<td>...</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>Lettuce</td>
<td>4/5</td>
<td>ETEC (1)</td>
<td>lt</td>
<td>1.5 × 10^6</td>
</tr>
</tbody>
</table>

NOTE. aEPEC, atypical pathogenic E. coli; eaeA, intimin; ETEC, enterotoxigenic E. coli; lt, heat-labile enterotoxin; stx, Shiga toxin.

Non–O157 Shiga Toxin–Producing Escherichia coli Is the Most Prevalent Diarrheagenic E. coli Pathotype in Street-Vended Taco Dressings in Mexico City

To the Editor—We read with great interest the article by McPherson et al [1] that addressed the risk factors for Shiga toxin–producing Escherichia coli (STEC) infection—in particular, food consumption—which have not been previously examined in Australia. Apparently, this is the first study to have examined risk factors for non-O157 STEC infections in both pediatric and adult populations. The study compared case patients infected with O157 and non-O157 STEC with control subjects, revealing that risk factors were different between these patients. Furthermore, negative associations were observed particularly with home-grown vegetables, fruits, and herbs for both O157 and non-O157 STEC infections. Although outbreaks of O157 STEC infection have been associated with consumption of raw vegetables [2, 3], little is known about the presence of non-O157 STEC [4] in these food items worldwide.

In many developing countries, including Mexico, street-vended food is a major source of fast, ready-to-eat meals for a large proportion of the population. In Mexico, street-vended food has been found to be contaminated with diarrheagenic E. coli pathotypes [5] and has been associated with travelers’ diarrhea [6]. Consequently, we analyzed 178 street-vended taco dressings (chili sauces, coriander-onion, pico de gallo, guacamole, and lettuce) collected in Mexico City (Table 1). One hundred twenty (67%) of 178 samples were contaminated with E. coli, and 8 (7%) of them were positive for diarrheagenic E. coli pathotypes by polymerase chain reaction [7], of which 6 (75%) of the 8 belonged to the STEC pathotype; all were found to be serotype O157 negative by the Oxoid agglutination test (Table 1). Four of the non-O157 STEC strains were resistant to ampicillin, and 1 was also resistant to chloramphenicol; all strains were susceptible to trimethoprim-sulfamethoxazole, ciprofloxacin, and cefotaxime. In Mexico City, non-O157 STEC strains have been associated with both cases of community diarrhea [8, 9] and acute diarrhea that required hospitalization in children [10]. It seems that street-vended taco dressings could be a potential vehicle of transmission for non-O157 STEC strains in Mexico City, given that all taco dressings harbored STEC levels of >1 × 10^3 organisms/g (Table 1)—well above the estimated infectious dose of 50–100 CFU for O157:H7 [11]. However, to understand the epidemiology of non-O157 STEC in Mexico, studies such as the one described by McPherson et al [1] are urgently needed.

Acknowledgments

Potential conflicts of interest. All authors: no conflicts.

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References

figure 1. kinetics of (1→3) β-d-glucan levels in a patient with pneumocystis jiroveci pneumonia and human immunodeficiency virus infection (white bars) and in a renal transplant recipient with P. jiroveci pneumonia (gray bars). both patients responded to the treatment and survived the infection.

10. Estrada-Garcia T, Lopez-Saucedo C, Thomp-

p. jiroveci and human immunodeficiency virus infection

2. Watanabe Y, Ozasa K, Mermin JH, et al. Fac-


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Clinical Infectious Diseases 2010; 50:656–1

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doi: 10.1086/649888

Serum (1→3) β-D-Glucan as a Noninvasive Adjunct Marker for the Diagnosis and Follow-Up of Pneumocystis jiroveci Pneumonia in Patients with HIV Infection

To the Editor—We read with great interest the Brief Report by Watanabe et al [1] about the value of the (1→3) β-D-glucan (BG) assay as an adjunct for the diagnosis of Pneumocystis jiroveci pneumonia (PJP) in patients with AIDS. We congratulate the authors because their report has a large study population (111 patients with PJP and a control group with 425 patients). However, we would appreciate your taking into account the following observations.

First, we wonder whether the report is of a prospective study or a retrospective analysis of test performance in those patients with confirmed PJP.

Second, when using a control group, it is important to define accurately the risk factors of the matched control group, because the possible development of PJP is dependant on the host risk(s) for disease.

From our point of view, in this setting, the control group should include human immunodeficiency virus (HIV)–positive patients with CD4+ cell counts ≤200 cells/μm3 or a CD4+ cell percentage ≤14% with a clinical respiratory infection. These characteristics are not described by the authors in their control group.

Third, we would like to remark that the accuracy of a diagnostic test is defined by calculating the cutoff value, the sensitivity, the specificity, and the positive and negative predictive values. In their study, Watanabe et al [1] only report the sensitivity and specificity. In the clinical settings in which BG is used, negative predictive value is high, and it is consequently important to rule out the diagnosis of PJP and other invasive fungal diseases.

Fourth, bacterial pneumonia is a common respiratory infection in this subpopulation, with a 20% rate of positive blood culture results. Both gram-positive and gram-negative bacteremias have been reported to be the source of false-positive BG results [2]. The administration of some antibiotics may also be a cause of BG reactivity [3]. We miss these pertinent data in the cohort assessed by Watanabe et al [1], because both are possible confounding factors.

Fifth, Watanabe et al [1] state that serum BG levels are not suitable for monitoring the response to treatment and that they do not always return to normal levels during treatment. We agree that BG does not return to normal levels during the course of treatment, because 3 weeks is not enough time to achieve a serological cure, which usually requires several weeks after the end of treatment [4]. Our group [4] and others [5, 6] have reported that the kinetics of measured BG (Fungitell; Associates of Cape Cod) suggest that decreasing levels of BG correspond to a favorable response to treatment (Figure 1), whereas increasing levels are associated with treatment failure [4].

Therefore, we believe that prospective