Pathogen responsible for the German *E. coli* outbreak has distinguishing features

 The strain of *E. coli* responsible for the recent German foodborne outbreak shows some characteristics that distinguish it from the strains that have caused illness in the US. First, except for the recent cases that are tied to tourists who have recently returned from that area in Germany, the German strain O104:H4, has only been traced to one previous foodborne illnesses in the US—“a [1994] outbreak of bloody diarrhea associated with consumption of raw milk in Montana.” The most common outbreak strain found in the US is *E. coli* O157:H7, which is a Shiga-toxin producing strain and is considered an adulterant when found in meat samples.

 *E. coli* O104:H4 is not among the other six Shiga-toxin producing strains that have been identified as responsible for previous *E. coli-based* outbreaks. At present none of these six strains nor *E. coli* O104:H4 is considered an adulterant and there are no US regulations requiring packers to test for them.

 A team led by Helge Karch at the Institute for Hygiene at the University of Munster has conducted a detailed analysis of the *E. coli* O104:H4 outbreak in Germany. The results of their analysis were published online in the June 23, 2011 issue of *Lancet Infections Diseases*.

 As of June 23, 2011, the outbreak has caused nearly 2,800 cases of identified illness including 849 cases of hemolytic uremic syndrome (HUS)—a kidney disease—and 39 deaths. According to a New York Times article by Gina Kolata (<http://www.nytimes.com/2011/06/23/health/research/23ecoli.html?ref=health>), said “at least 100 people in Germany will need kidney transplants or will have to undergo dialysis for the rest of their lives.”

 Unlike other *E. coli* outbreaks, the Lancet article reports, “the particular features of this outbreak are the predominant involvement of adult women and common severe neurological complications, such as encephalopathy and epileptic seizures.” Most outbreaks have a greater impact on children and the elderly, but the identification of bean sprouts as the source of the *E. coli* strain in this case explains the larger number of women affected by this outbreak because they eat more sprouts than other groups in the general population.

 “In the past 15 years, only a single *E. coli* O104:H4 has been isolated from a patient with hemolytic uremic syndrome in Germany. A single additional case of the syndrome associated with *E. coli* O104:H4 has also been reported from a woman in Korea.”

 Karch’s team analyzed 80 stool samples from a wide area in Germany. Their analysis showed that all of the cases belonged to *E. coli* serotype O104:H4 and were from a single clone so there is ultimately a single source of the bacteria. One of the characteristics of this particular bacteria is that it is a combination of the normal O104:H4 that has the gene for “Shiga-toxin 2…and the aggregative (so-called stacked-brick) adherence pattern on intestinal epithelial cells.”

 The authors theorize that the stacked-brick adherence pattern allowed the *E. coli* cells to stick to the walls of the intestines in greater numbers than in other adherence patterns. This then could have allowed an increased amount of Shiga-toxin to make its way into the bloodstream, producing the larger percentage of HUS cases than is typical of other outbreaks.

 The study authors write, “our data do not permit us to state if the Shiga-toxin-producing and [stacked-brick adherence pattern] traits…combined recently, or if this pathogen is coming to light now because of conditions suitable for its spread.”

 There are a number of lessons that can be learned from this study. First, early identification of the characteristics of the strain responsible for an outbreak of bloody diarrhea is crucial because in the German case, “the outbreak strain…would have been missed by standard diagnostic procedures focused on the O157:H7 strain.”

 Second, the identification of any antibiotic resistance in an outbreak strain needs to be completed so that if antibiotics are administered, they are ones that the pathogen is susceptible to. Otherwise the antibiotics may suppress competing microbiota, making room for the pathogen to multiply rapidly.

 Third, “the value of standing collections of well characterized pathogens with known provenance” shorten up the time required to identify the various characteristics of an outbreak-causing pathogen.

 The authors conclude, “although we lack an explanation for increased virulence, this outbreak tragically shows that blended virulence profiles in enteric pathogens introduced into susceptible populations can have serious consequences for infected people.”

*Daryll E. Ray holds the Blasingame Chair of Excellence in Agricultural Policy, Institute of Agriculture, University of Tennessee, and is the Director of UT’s Agricultural Policy Analysis Center (APAC). Harwood D. Schaffer is a Research Assistant Professor at APAC. (865) 974-7407; Fax: (865) 974-7298;* *dray@utk.edu**and**hdschaffer@utk.edu**;*[*http://www.agpolicy.org*](http://www.agpolicy.org/)*.*

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