Germany's *E. coli* Outbreak and Water Quality Protection Needs

nce again bean sprouts are implicated in a massive foodborne disease outbreak, this time involving a new strain of *Escharid*

time involving a new strain of *Escherichia coli* (O104:H4). This tragedy is a reminder that other foods and drinking water sources may also be vulnerable to contamination. Minimally, the outbreak in Germany teaches us that: 1) microbes are still evolving; 2) new strains have the potential to affect different populations with increased adverse effects, and 3) infection control efforts in the food industry are also applicable in the water industry.

From indigenous to deadly

E. coli outbreaks are identified more frequently than other bacterial etiology due to the severity of symptoms, which range from mild diarrhea to a severe hemolytic uremic syndrome (HUS, usually only found in young children and the elderly). *E. coli* are naturally present in the feces of all warmblooded animals, and thus, not all strains are harmful. In fact, *E. coli* in the gut is beneficial, as it produces vitamin K and competes with harmful bacteria to prevent intestinal

infections. The problem is that some *E. coli* acquire virulence factors from neighboring bacteria in the environment, creating killer strains from a primarily commensal organism.

Many of us are familiar with the particularly deadly *E. coli* serotype O157:H7; this and other lesser known strains are part of a class of bacteria known as enterohemorrhagic *E. coli* (EHEC). EHEC are moderately invasive in the intestine but produce a potent toxin, known as a *Shiga* toxin (possibly acquired from human pathogenic *Shigella* bacteria). The infectious dose of O157:H7 is very low (10-100), and toxin production is worsened by host iron deficiency and, ironically, antibiotic treatment, as dying bacteria tend to release more toxin.

The O157:H7 strain has been implicated in major drinking water outbreaks from both private and public source waters. From 1982 to 2002, there were 350 documented outbreaks of *E. coli* O157:H7, with 15 percent due to drinking water exposures and 61 percent to food. Produce (i.e., the 2006 US spinach outbreaks) and ground beef (including the original 1992 *E. coli* O157:H7 Jack-in-the-Box outbreak) have been the primary routes of exposure to humans, but water-farm-handler-food transmission routes likely overlap, suggesting the need for more comprehensive controls.

A new threat

E. coli O104:H4 has never been documented before in an outbreak and has only rarely occurred in humans. This new strain

By Kelly A. Reynolds, MSPH, Ph.D.

has emerged with increased virulence, increased antibiotic resistance and increased toxicity. The strain does

produce *Shiga* toxin but is not related to the O157 serotype. Interestingly, over the last five years, the incidence of severe non-O157 infections has been increasing. The million-dollar question is why?

The most recent *E. coli* outbreak was discovered in Germany on May 22, due to a significant increase in patients with bloody diarrhea and severe kidney and neurological symptoms (i.e., paralysis and coma). From May 2 to June 13, there were 3,256

> documented cases, primarily in Germany but also involving over 13 countries in the European Union/European Economic Area.¹ To date, infections have resulted in 35 documented deaths, for a case fatality rate of 1.07. While new cases are expected, the outbreak peak appears to have passed.

> There are key differences in the current *E. coli* outbreak compared to previous events. First, severe *Shiga* toxin-producing *E. coli* (STEC) infections typically occur in children, but the epidemiology of the European

outbreak indicates that most cases are in adult women, specifically women between 20-50 years old and described as being mostly "fit and with healthy lifestyles."

Early evidence pointed to a possible link to cucumbers, tomatoes and leafy salads in northern Germany. It wasn't until June 10 that German authorities confidently linked a variety of bean and seed sprouts as the contaminated vehicle. Further evidence traced the contamination to a farm in the Lower Saxony region in northwestern Germany.²

Authorities now recommend that people in Germany not eat raw bean and seed sprouts of any origin, until further notice. In addition, it is recommended that all food products from the Lower Saxony farm be recalled. Although O104:H4 strains have not been detected at the farm, a package of sprouts from the region tested positive and was found in the home of an infected family. Persons consuming sprouts were nine times more likely to experience bloody diarrhea.³ In addition, adult females are more likely to consume sprouts than males and children – a consistent characteristic with the outbreak's epidemiology.

How did E. coli O104:H7 get here?

The German agency, BfR (Bundesinstitut fur Risikobewertung) is investigating the origin of the O104:H4 outbreak strain. The BfR suspects that the current strain is probably a recombinant of two pathogenic *E. coli* types, including: 1)

the source of the recent outbreak can help to evaluate future control needs. As pointed out in *Food Safety News*, we should not overlook the obvious lesson in Germany's outbreak: "…it is not about the sprouts".

<u>An understanding of</u>

Shiga-toxin producing strains, generally from cattle, and 2) enteroaggregative *E. coli* (EaggEC) typically linked to human hosts. Because the O104:H4 strain is rare, there are still many uncertainties about its origin. What we know is that animal and human bacteria readily exchange information in the environment and, in this case, formed a more dangerous microbe.⁴ DNA profiling tells us that the new strain is more highly related to other human strains as opposed to animals.⁵ These related strains have enteroaggregative properties, meaning that they stick better to the gut and produce increased diarrhea in infected hosts. A highly related version was first identified in Germany in 2001; however, the earlier strain caused fewer than five documented cases worldwide.

Another problem with the new strain is that it is resistant to eight classes of antimicrobial agents, including penicillins and streptomycin. The reality is that *E. coli* is common in feces and the environment, and has the fastest generation time of any culturable bacteria (20 minutes). Thus, rapid evolution is inevitable. Throw some antibiotics into the environment, as we've done for decades, and you get new, antibiotic-resistant bacterial strains and other virulence factors.

Toxin-producing strains of bacteria have approximately a 10-percent hospitalization rate. O104:H4 raises the bar to about 33 percent of documented cases resulting in hospitalization.⁶ Another interesting difference is the prevalence of neurological symptoms in the current outbreak.⁶ In previous *E. coli* outbreaks, neurological symptoms (ranging from confusion to seizures to comas) occurred in a small percentage of patients with hemolytic uremic syndrome (HUS), compared to nearly half in the current outbreak.

Preventing E. coli in drinking water

While the German *E. coli* outbreak involved produce, drinking water is also vulnerable to fecal contamination. An understanding of the source of the recent outbreak can help to evaluate future control needs. As pointed out in *Food Safety News*, we should not overlook the obvious lesson in Germany's outbreak: "...it is not about the sprouts".⁷ Although this article points toward a need to examine, and reform, the whole food system, the same could be said for the drinking water system. New pathogens continue to emerge, environmental routes that include human and animal reservoirs persist, and drinking water distribution systems are increasingly vulnerable. Sprouts are the culprit today but other environmental transmission routes are possible in the future.

The *Toronto Star* recently published an article related to what Europe can learn from the *E. coli* drinking water outbreak in Ontario, Canada that sickened approximately 2,500 people in Walkerton, a town of 5,000 residents, resulting in seven deaths in May of 2000.⁸ A lack of oversight related to water quality guidelines, slow response time and cuts in environmental regulatory resources, were all sighted as some of the regulatory deficiencies that advanced the Canadian outbreak. Since 2000, Ontario has increased the number of water and food inspectors and invested tens of millions of dollars in resources to improve water quality in the region.

Budget cuts are a reality in the US and around the world and environmental regulatory agencies are often early to the chopping block. Whether we are talking about food safety or water quality, the outbreak in Germany reminds us that now is **not** the time to let our guard down.

References

1. *ProMED Digest*. Epidemiological update, 12 Jun 2011, 11:00; Monday, June 13, 2011. Vol. 2011 (257).

2. WHO, Regional Office for Europe, *International Health Regulations*. WHO update, Saturday, June 11, 2011. http://www.euro.who.int/en/what-we-do/health-topics/emergencies/international-health-regulations/news/ news/2011/06/ehec-outbreak-update-14.

3. Marler, Bill. Sprout Seeds likely source of 3330 Illnesses and 36 Deaths in *E. coli* Outbreak. *Food Poison Journal*. June 13, 2011.

4. Raymond, R. Is Germany's Outbreak Source Human, not Cow? *Food Safety News*. June 09, 2011. http://www.foodsafetynews.com/2011/06/is-germanys-outbreak-source-human-not-cow/.

5. *ProMED Digest*. Enteroaggregative phenotype. June 10 2011. Vol. 2011 (251).

6. *Science Insider*. June 6, 2011. American Association for the Advancement of Science (AAAS). http://news.sciencemag.org/scienceinsider/2011/06/ sprouts-so-far-innocent-of-e-col.html.

7. Kim, E.J. It's Not About the Sprouts. *Food Safety News*. June 11, 2011. http://www.foodsafetynews.com/2011/06/its-not-about-the-sprouts/.

8. Benzie, R. *E. coli Outbreak in Europe Recalls Lessons from Walkerton*. June 2, 2011. http://www.thestar.com/news/canada/article/1001836–e-colioutbreak-in-europe-recalls-lessons-from-walkerton.

About the author

◆ Dr. Kelly A. Reynolds is an Associate Professor at the University of Arizona College of Public Health. She holds a Master of Science Degree in public health (MSPH) from the University of South Florida and a doctorate in microbiology from the University of Arizona. Reynolds has been a member of the WC&P Technical Review Committee since 1997. She can be reached via email at reynolds@u.arizona.edu

Reprinted with permission of Water Conditioning & Purification International ©2020. *Any reuse or republication, in part or whole, must be with the written consent of the Publisher.*