The Detection of and Response to a Foodborne Disease Outbreak: A Cautionary Tale

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(See the Major Article by Fernandes et al on pages 903–9.)

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Fernandes et al [1] provide convincing evidence that an outbreak of Campylobacter (species not provided) infection occurred in the population of a small island in England in late September and early October 2011. The investigators conclude that a combination of descriptive epidemiology, genomic epidemiology, and environmental investigation identified the likely source of the outbreak: milk pasteurized using a malfunctioning milk pasteurizer. They are to be congratulated for their effort to identify the source of the outbreak, take necessary corrective action to ensure it does not happen again, and share their investigative findings with the scientific and public health communities. However, I believe that there is a cautionary tale to be told with this investigation and report—namely, that modern laboratory science can enhance foodborne disease surveillance and outbreak detection, but overreliance on it to solve many foodborne disease outbreaks poses a real shortcoming in our evolving public health practice.

Almost 40 years ago, I had the good fortune to be appointed the director of infectious disease epidemiology activity at the Minnesota Department of Health. At that time, foodborne disease outbreaks that we investigated often related to situations where, for example, grandma’s potato salad was left in the hot sun for hours at the church picnic and the subsequent Staphylococcus intoxication cases were readily apparent among the picnic attendees. The primary epidemiologic tools that we had in our foodborne disease investigation kit were a simple case-control study methodology with simple 2-by-2 statistical analysis tables and no-memory calculators. Our laboratory support might include serotyping of Salmonella and Shigella strains and the use of animal challenge studies to determine the presence or absence of enterotoxins. There was no molecular characterization of the involved pathogens; no laboratory test methods for routine testing of Campylobacter, norovirus, or hepatitis A virus; and no multivariate analysis run on a computer. Despite our crude tools, at least by today’s standards of epidemiology, we solved some very complicated and challenging foodborne outbreaks.

Over the course of the next 40 years, the Minnesota Department of Health made foodborne disease surveillance and outbreak investigation a priority. We pioneered a number of the epidemiologic methods used today to investigate foodborne disease, particularly those involving outbreaks related to mass-produced foods with low-level contamination and which are disseminated around the world. These pose among the greatest public health challenges for detection and intervention. We also pioneered the early use of laboratory-based molecular characterization of pathogens such as Escherichia coli O157:H7 and Salmonella species to assist in both detecting and defining outbreaks [2–5]. In the mid-1990s we formed Team Diarrhea, a now well-known group of public health graduate students devoted to the rapid and comprehensive interview and investigation of cases of likely foodborne disease reported through our state-wide active disease surveillance system. Over these past 40 years, the Minnesota Department of Health has led many foodborne disease outbreaks of international importance and published widely on this work in the leading medical journals.

The purpose of the brief history of the Minnesota Department of Health activities is to serve as a foundation for my...
comments on the article by Fernandes et al. I believe this article really was the telling of the story of how whole-genome sequencing (WGS) solved a milk-borne Campylobacter outbreak and allowed for an intervention to be put in place. If this is the conclusion intended by the authors, that would be unfortunate and should serve as a cautionary tale for others tempted to substitute a new laboratory tool for critical and timely shoe-leather epidemiology. Since 1996, when the Centers for Disease Control and Prevention started a national molecular subtyping system, PulseNet, it has been possible to share information about specific pathogen identification and relatedness quickly across public health agencies around the world. Most of the early testing used pulsed-field gel electrophoresis (PFGE) to establish a unique molecular fingerprint of the pathogen isolate and share it via the Web. More recently, the precision of laboratory tests has been greatly enhanced with WGS, as it is now possible to differentiate clonal pulsed-field gel (PFG) clusters into distinct genetic lineages that are different and not related, particularly as they might be associated with a specific food source. In other words, with PFGE analysis we could sometimes specifically identify a distinct genetic lineage, but sometimes not. When we couldn’t, we would lump “apples and oranges” together and misclassify specific strains of a pathogen as the same when they were not. This can make any epidemiologic analysis problematic.

WGS is a powerful tool for use in our foodborne disease investigations, but only when it is needed and when the lack of isolate relatedness is the reason for the investigation to potentially be unable to identify the implicated food item(s) in a timely manner. For example, in the outbreak investigation described by Fernandes et al, I believe that WGS analysis of isolates from the cluster of Campylobacter cases with onset of illness between 29 September and 5 October 2011 was unnecessary and wasted potentially valuable response time. Of note, children from two schools accounted for 52% of the cases. We have long known how to quickly and effectively conduct an epidemiologic case-control study of such a cluster with a high degree of certainty that such cases are related [6]. Fortunately for the affected community, it appears that the Campylobacter contamination of the milk was sporadic and not ongoing. Had the contamination been ongoing, the delay in conducting the case-control study with the initial cases reported, determining the source, and taking corrective action to warn the community to not drink the milk and to stop distributing the contaminated milk would have been unfortunate. It would have also meant additional, preventable cases occurring that could be directly related to the lack of timeliness of the investigation.

Unfortunately, it has been our experience over the recent months that an increasing number of foodborne disease outbreak investigations have been delayed waiting for WGS data to become available. For some of these investigations, the complexity of the isolates involved made the differentiation of clonal PFG clusters into distinct genetic lineages an important part of the investigation. But for many of the outbreak investigations, the WGS of isolates was unnecessary to either identify outbreak-associated cases or implicate the responsible food item. In these situations, using this new powerful laboratory tool to define cases did not help, but rather compromised the investigation. This should be a cautionary tale to all who are involved in foodborne disease outbreak investigations. A powerful new laboratory tool is only as good as it is appropriately applied to the situation.

Note

Potential conflict of interest. Author certifies no potential conflicts of interest. The author has submitted the ICMJE Form for Disclosure of Potential Conflicts of Interest. Conflicts that the editors consider relevant to the content of the manuscript have been disclosed.

References