Food Safety Revisited

Niels Frimodt-Møller and Anette M. Hammerum
National Center for Antimicrobials and Infection Control, Statens Serum Institut, Copenhagen, Denmark

(See the major articles by Kieke et al., on pages 1200–8, and Jalava et al., on pages 1209–16.)

The destiny of nations depends on how they nourish themselves.

Anthelme Brillat-Savarin

Two papers in this issue of the Journal of Infectious Diseases focus on food safety from 2 different angles. First, in an elegant work from Finnish investigators [1], the culprit behind an epidemic caused by Yersinia pseudotuberculosis was uncovered: grated carrots. The same serotype and pulsed-field gel electrophoresis (PFGE) type of Y. pseudotuberculosis was isolated from patients and from the farm where the carrots were grown and processed. The epidemic, which involved an estimated 558 patients, had its root in 1 kitchen preparing food for some 7400 individuals, mostly children in day-care centers or schools. The grated carrots were used in a raw salad without further processing. This first-time isolation of Y. pseudotuberculosis from a contaminated food ingredient resulted from a rapid and relevant reaction from health authorities, as well as sound epidemiological and microbiological work, for which the authors are to be congratulated.

Although there have been few published reports on this subject, it is not the first time that Y. pseudotuberculosis has caused epidemics: they have been reported from Russia [2], Japan [3, 4], Canada [5], and even previously in Finland [6]. Several food ingredients have been incriminated—for example, carrots [3], lettuce [2], other vegetables [3], and even pickled products [4]. Y. pseudotuberculosis has also been isolated from water and meat products [4]. The bacterium has been found in a range of animals, such as pigs, sheep, deer, hares, rabbits, wild boar, jackal, rodents, birds, and others [7–13]. In these animals, the organism can be cultured from tonsils, tongues, and all larger organs, as well as from the gut [11]. The animal reservoir probably explains how carrot plants can be contaminated on the ground or at any stage of storage if the carrots are not secluded from contact with animals or their feces. Another mode of contamination could be by handling of the products by personnel having contact with animals.

With such common occurrence in animals and foods, one wonders why so relatively few cases or epidemics are reported. Is it possible that the organism is overlooked in feces cultures in routine laboratories?

The second article on the issue of food safety revisits the problem of the selection of antibiotic resistance in enterococci by the use of antibiotic growth promoters. In a comprehensive study, the investigators from Minnesota isolated Enterococcus faecium from poultry meat and feces from both healthy vegetarians and hospital patients [14]. The isolates were investigated for resistance to streptogramins using both phenotypic and genotypic methods. Furthermore, in a novel approach, inducible resistance toward several streptogramins was determined by spectrophotometric growth measurements. The results showed significant correlation with having streptogramin-resistant enterococci isolated from the gut and eating poultry meat [14].

Antibiotic growth promoters have been an issue in Europe since the early 1990s, and European investigators have expended a great deal of effort in elucidating the risk for humans of using antibiotic growth promoters on such a broad scale as was the case in Europe, where almost all feeds sold on the market contained some antibiotic growth promoter. Most research has focused on vancomycin-resistant E. faecium (VRE) and/or streptogramin-resistant E. faecium (SRE); in some cases, the bacteria have been resistant to both groups of antibiotics. With the use in feed of avoparcin or virginiamycin (the gly-

Received 5 July 2006; accepted 5 July 2006; electronically published 27 September 2006.

Potential conflicts of interest: none reported.

Reprints or correspondence: Dr. Niels Frimodt-Møller, National Center for Antimicrobials and Infection Control, Statens Serum Institut, Artillerivej 5, 2300 Copenhagen S, Denmark (nfm@ssi.dk).

The Journal of Infectious Diseases 2006;194:1191–3
© 2006 by the Infectious Diseases Society of America. All rights reserved.
0022-1899/2006/19409-0001$15.00
copeptide and streptogramin used in animal husbandry), VRE or SRE was selected [15], and there was a high prevalence of VRE or SRE in poultry before and at slaughter [16], the farms were constantly contaminated, and VRE and SRE spread to new flock [17], VRE was found in poultry meat in food stores [18], and there was a prevalence of up to ~20% of VRE in feces from nonhospitalized European individuals [19]. VRE from human infections showed the same PFGE types as strains found in pigs [20, 21]. In addition, VRE and SRE (from poultry and pigs) in milk ingested by volunteers survived the acidity of the stomach and reached the gut, multiplied, and remained there for 14 days [22], and VRE and SRE transconjugants could be detected in feces 2 days after the ingestion of both a VRE/SRE donor strain and a recipient Enterococcus faecium strain in human volunteers without antibiotic treatment [23]. The fina worrying step in the risk chain was provided by the report from Michigan several years ago about the isolation from a patient of a vancomycin-resistant Staphylococcus aureus containing the vanA element from E. faecalis [24]. More evidence that antibiotic growth promoters confer resistance has now been provided [14].

Both articles [1, 14] are examples of how the industrialization of food production in our modern societies carries and even amplifies the risk for unaware consumers. If we are unable to change the mores of industrialized society (life is short, we are all in a hurry, etc.), how can we reduce or remove the risk?

The answer for the SRE and VRE is easy: ban antibiotic growth promoters! This has worked in Europe without serious consequences for the well-being of the animals, their breeders, or consumers [25]. In Denmark, isolation of VRE and SRE has been greatly reduced over the past 10 years (http://www.danmap.dk). VRE and SRE are still present in the poultry flocks albeit in lower numbers, but they can be found with selective enrichment methods [26]. This means that VRE and SRE are still present and that the reintroduction of growth promoters would easily increase the level of resistant bacteria in poultry flocks.

For Y. pseudotuberculosis, it may not be that simple. A number of steps could be envisaged, but we do not have the solution, because it depends on the particular setting. Some suggestions could be given for the production line: grow carrots in glass houses, to avoid contact with animals; store them out of range of animals; encourage and enforce hand hygiene and hygienic measures for the machinery involved; develop sensitive microbiological methods for the detection of zoonotic bacteria at all levels; and implement methods to decrease numbers of bacteria on the products (e.g., heating, UV light, and radiation). If specific reservoirs are identified, the application of hazard analysis and critical control point during production must be addressed. For kitchens the size of the Finnish example, some of the same suggestions could be used there as well, and, if these are not possible, it might be considered whether there should be regulations against certain meals—for example, prohibiting the use of raw vegetables, nonboiled eggs, or raw oysters—when food distribution exceeds a certain number or type of recipients (e.g., immunocompromised individuals). Finally, both articles are fin examples of the need for this type of high-class epidemiological and microbiological research to demonstrate the (mis-)conduct of modern human society.

References


