

INFECTIOUS DISEASE

A germy world—food-borne infections in 2009

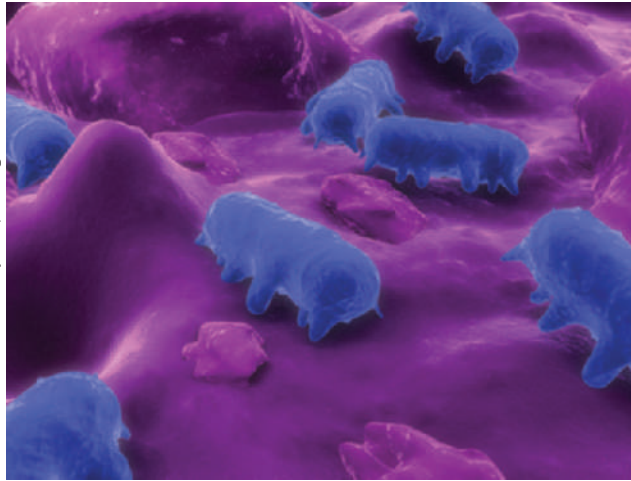
Lawrence R. Schiller

The recent outbreak of *Salmonella typhimurium*, associated with peanut butter and products containing it in the US and Canada, highlights our ongoing susceptibility to food-borne infections despite advanced food production systems. As the globalization of food resources continues, how can we control outbreaks and minimize their effect on health?

We live in a germy world. We have more bacterial cells in and on our bodies than we do human cells, and every day we come into contact with countless micro-organisms: in the air that we breathe, on the surfaces we touch, in the water we drink, and in the food we eat. Multicellular organisms evolved in this environment, and have developed sophisticated mechanisms and barriers that prevent bacterial invasion and damage. Most of the time these mechanisms and barriers work well, but sometimes they are weak or, in some cases, micro-organisms evolve counterstrategies, and we become ill.

All living humans are the descendents of a small group of ancestors who lived about 50,000 years ago.¹ At that time, humans subsisted as hunter-gatherers and lived in relatively small, mobile groups, the numbers of which were limited by the scarce resources available within local areas. In that era, the population of *Homo sapiens* is estimated to have been fewer than 1 million, and may have been as few as 20,000.¹ Pathogens no doubt lurked within these communities and caused significant illness, but the spread of disease was limited because any given group lived largely in isolation from others.

The first fixed human settlements date back merely 24,000 years, and villages and cities only developed approximately 10,000 years ago, as agriculture and animal husbandry began to replace the hunter-gatherer lifestyle.² The population exploded when food became more plentiful and certain. For the first time, distribution of food and water and removal of waste became important issues; these systems



© HAAP Media Ltd, a subsidiary of Jupiterimages

created potential pathways for the distribution of pathogens. Moreover, large numbers of susceptible hosts lived in close proximity to one another. Epidemics were facilitated by this close proximity of hosts and became routine events, killing tens of thousands, until improvements in sanitation during the past 200 years substantially reduced their occurrence; as a result, the human population increased from under 1 billion to over 6 billion. Despite this improved sanitation, fecal-oral transmission of disease is still prevalent, even in the Western world, and as many as 9,000 Americans die each year from food-borne illnesses.³

Distribution of food has changed from a regional system to a global network, which facilitates the spread of food-borne diseases. I can remember a time 50 years ago when fresh fruits and vegetables were only available 'in season,' and canned or frozen food supplies were the only available options for much of the year. Efficient transportation has now made virtually every fresh food available all year round; this globalization

of food sources has made cases of imported disease more likely. Outbreaks of *Cyclospora* from imported raspberries,⁴ hepatitis A from green onions,⁵ salmonellosis from peppers,⁶ and *Escherichia coli* from meat⁷ and fruit juice⁸ have all been reported in the US.

The industrialization of food production and processing has also caused problems. In the past, local, small-scale production meant that any particular incident of food contamination infected only small amounts of food and relatively small numbers of individuals were exposed to pathogens. The centralization of food

production has increased the potential for crosscontamination during processing, and broadened the distribution of contaminated products to millions of people.

The outbreak of *Salmonella typhimurium* associated with peanut butter and products containing it that took place in the US and Canada between September 2008 and January 2009 is a good example of this problem. A reported 529 people from 43 American states and 1 person from Canada were infected, and the contamination may have contributed to 8 deaths.⁹ Prompt recognition of the outbreak by the Centers for Disease Control's PulseNet surveillance staff and local public-health partners led to identification of the source of the outbreak—a peanut processing plant in Georgia.⁹ This plant was reportedly guilty of many lapses in good manufacturing practice that might have led to contamination. What might have been only a local problem was magnified as this plant produced peanut butter and other peanut products that were distributed to over 2,000 food companies in

the US and 23 other countries.¹⁰ Over 400 peanut-containing products were recalled because of potential contamination.¹⁰

What can be learned from this experience? About 40,000 laboratory-confirmed cases of salmonellosis are reported each year, with *S. typhimurium* being the most commonly identified serotype.¹¹ However, only an estimated 3% of salmonella infections are laboratory-confirmed; therefore, about 1.2 million cases probably occur each year.¹¹ During the 2003–2007 period, an average of 18 *S. typhimurium* outbreaks were reported to the US Center for Disease Control each year.⁹ Salmonellosis, therefore, remains prevalent in the US. The recent salmonellosis outbreak is the second linked to peanut butter in the US; the first occurred in 2006–2007 and was associated with a different strain of bacteria.¹² The roasting of peanuts should kill any pathogenic organism, which indicates that contamination of products that contain peanuts might occur after the roasting process. *Salmonella* can persist indefinitely in peanut butter, and temperatures used to manufacture other food products that contain peanut butter might not kill the pathogens. The salmonellosis outbreak in Georgia highlights the importance of vigilant surveillance of disease. Had the surveillance network not been in place or had it malfunctioned, the outbreak might have continued much longer and endangered an even greater number of individuals.

Food safety can be difficult in practice and involves consumers as well as producers. As we have evolved from hunter-gatherers who might endanger only our families or clans with contaminated foods to a global population with complex, industrialized food production that can place millions at risk, improved management of the entire food chain has become necessary. At the farm level, good agricultural practices can reduce the contamination of produce and livestock. Food processing must include sufficient safeguards to ensure that contaminants are not introduced during manufacture and that the integrity of systems are checked at critical junctures. Clean transport and maintenance of a cold chain (when necessary) are essential, and proper handling, cooling

and preparation of food at stores and restaurants should be mandatory. In the home clean preparation, avoidance of cross-contamination, thorough cooking and chilling can all reduce the chance of pathogenic, food-borne bacteria being ingested.

Adequate inspection and oversight of these steps is as important as epidemiological surveillance to minimize outbreaks of disease. Although this task is daunting, we must all support efforts to produce a safe food supply. In the US, the FDA and the Department of Agriculture have primary responsibility for regulating interstate commerce of food products. State and local regulations also govern food processing, but enforcement varies from place to place; we must harmonize and strengthen this regulatory framework. Experience has proven once again that we cannot leave this obligation to food manufacturers alone.

Digestive Health Associates of Texas, Baylor University Medical Center, Dallas, TX, USA.

Correspondence: Digestive Health Associates of Texas, Baylor University Medical Center, 712 North Washington Avenue, Suite 200, Dallas, TX 75246, USA
lrsmd@aol.com

doi:10.1038/nrgastro.2009.40

Competing interests

The author declared no competing interests.

As we have evolved
... improved
management of
the entire food
chain has become
necessary

1. Harpending, H. C. *et al.* Genetic traces of ancient demography. *Proc. Natl Acad. Sci. USA* **95**, 1961–1967 (1998).
2. Bocquet-Appel, J.-P. & Bar-Yosef, O. (eds) *The Neolithic Demographic Transition and its Consequences* (Springer, New York, 2008).
3. Mead, P. S. *et al.* Food-related illness and death in the United States. *Emerg. Infect. Dis.* **5**, 607–625 (1999).
4. Ho, A. Y. *et al.* Outbreak of cyclosporiasis associated with imported raspberries, Philadelphia, Pennsylvania, 2000. *Emerg. Infect. Dis.* **8**, 783–788 (2002).
5. Wheeler, C. *et al.* An outbreak of hepatitis A associated with green onions. *N. Engl. J. Med.* **353**, 890–897 (2005).
6. Centers for Disease Control. Outbreak of *Salmonella* serotype Saintpaul infections associated with multiple raw produce items—United States, 2008. *MMWR Morb. Mortal. Wkly Rep.* **57**, 929–934 (2008).
7. Hussein, H. S. Prevalence and pathogenicity of Shiga toxin-producing *Escherichia coli* in beef cattle and their products. *J. Anim. Sci.* **85** (Suppl. 13), E63–E72 (2007).
8. Vojdani, J. D., Beuchat, L. R. & Tauxe, R. V. Juice-associated outbreaks of human illness in the United States, 1995 through 2005. *J. Food Prot.* **71**, 356–364 (2008).
9. Centers for Disease Control. Multistate outbreak of *Salmonella* infections associated with peanut butter and peanut butter-containing products—United States, 2008–2009. *MMWR Morb. Mortal. Wkly Rep.* **58**, 85–90 (2009).
10. U.S. Food & Drug Administration. Peanut product recalls: *Salmonella typhimurium*. <http://www.fda.gov/oc/opacom/hottopics/salmonellatyp.html> (2009).
11. Voetsch, A. C. *et al.* FoodNet estimate of the burden of illness caused by nontyphoidal *Salmonella* infections in the United States. *Clin. Infect. Dis.* **38** (Suppl. 3), S127–S134 (2004).
12. Centers for Disease Control. Multistate outbreak of *Salmonella* serotype Tennessee infections associated with peanut butter—United States, 2006–2007. *MMWR Morb. Mortal. Wkly Rep.* **21**, 521–524 (2007).

TRANSPLANTATION

Steroid use in HCV-infected liver transplant recipients

Bruno Roche and Didier Samuel

The recurrence of hepatitis C in HCV-infected, orthotopic liver transplant recipients is a major problem that can influence the survival of both grafted tissue and patients. Yet the effects of immunosuppression strategies, in particular those of steroids, on disease recurrence remain unclear.

The effect of corticosteroids on the recurrence of hepatitis C in patients who have undergone orthotopic liver transplantation (OLT) is unclear, and seems to differ according to how they are used. We know that corticosteroids increase serum

viral load when administered to patients with chronic hepatitis C.¹ Several studies have shown that use of corticosteroid boluses (high doses given intravenously) to treat rejection episodes after OLT can be harmful to HCV-infected liver transplant