Foodborne Illness Acquired in the United States

To the Editor: The updated estimates of foodborne illness in the United States reported by Scallan et al. probably overestimate the occurrence of illness caused by unspecified agents because they did not account for the apparent sensitivity of the population survey to the occurrence of norovirus (*1*,*2*). The number of illnesses attributed to unspecified agents was derived from the simultaneous processes of extrapolation and subtraction: extrapolation from the population survey to create a base of diarrheal illnesses and subtraction of known agents from this base. Scallan et al. averaged illness rates from 3 successive population surveys to come up with a rate of 0.6 episodes of acute gastroenteritis per person per year. However, the individual rates were 0.49 (2000–2001), 0.54 (2002–2003), and 0.73 (2006–2007). The 2006–2007 survey was conducted at the time of widespread norovirus activity. The estimated rate of population illness was strongly correlated with the number of confirmed and suspected norovirus outbreaks reported to the Centers for Disease Control and Prevention Foodborne Disease Outbreak Surveillance System during each of the survey periods (300, 371, and 491, respectively; R^2 0.97, $p<0.0001$). No other known agents were correlated with the population survey rates, and the total numbers of outbreaks were inversely correlated with the population survey data.

The strength of the correlation between norovirus outbreaks and survey results suggests that the population survey is sensitive to norovirus activity and that norovirus may account for much of what is considered to be unspecified. The fact that the highest observed population rate was ≈50% greater than the lowest rate suggests that annual variation in norovirus activity may account for a considerable proportion of what otherwise seems to be unspecified. More thorough and timely investigation and reporting of outbreaks could facilitate the development of models to evaluate the number of illnesses and update them annually.

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To the Editor: The report by Scallan et al. provides a valuable update regarding estimated annual illnesses, hospitalizations, and deaths caused by recognized foodborne pathogens, most of which cause diarrheal disease, in the United States (*1*). However, absent from this study, and from most previous reviews of foodborne illness, was attention to possible extraintestinal disease, especially antimicrobial drug–resistant infections caused by food-source *Escherichia coli* and associated resistance elements.

A growing body of molecular and epidemiologic evidence suggests that a substantial fraction of extraintestinal *E. coli* infections in humans, particularly those involving antimicrobial drug–resistant strains, might be caused by *E. coli* from food animals (*2*). Extraintestinal pathogenic and antimicrobial drug–resistant *E. coli* commonly contaminate retail meat products (*3*,*4*); rates of contamination and resistance associated with "no antibiotics" production methods, labeling, and markets are lower (*4*). In a study of women with acute urinary tract infection, frequent consumption of chicken and pork was associated with isolation of antimicrobial drug–resistant *E. coli* from urine (*5*).

Extraintestinal *E. coli* infections, which include urinary tract infections and sepsis, are more common and result in more hospitalizations and deaths than do infections caused by the classic foodborne pathogens. For example, each year in the United States, an estimated 40,000 deaths are associated with sepsis caused by extraintestinal *E. coli* infection (*6*); only <1,400 deaths are caused by all major classic foodborne pathogens combined (*1*). Therefore, if even a modest fraction (e.g., 5%–10%) of all extraintestinal *E. coli* infections in humans are of foodborne origin—which seems highly plausible, considering the molecular evidence (*2*)—the extent of associated disease may equal or exceed that attributable to the classic foodborne pathogens as estimated by Scallan et al. Greater recognition of this possibility by the public health system is needed so that appropriate attention can be devoted to this neglected, invisible foodborne disease threat.

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In Response: We welcome the suggestions by Hedberg (*1*) and Johnson (*2*) on how future estimates of foodborne illness in the United States could be improved (*3*,*4*). We agree with Johnson that our estimates of foodborne illness probably reflect underrecognition of the extent of extraintestinal disease (*2*). Our estimates of foodborne illness caused by major pathogens included 7 pathogens that cause conditions other than gastroenteritis. However, our estimates of foodborne illness caused by unspecified agents relied on reports of acute gastroenteritis. We did not include illness caused by known foodborne agents that do not typically cause gastroenteritis, such as ciguatoxins and some mushroom toxins, for which insufficient data were available to estimate agent-specific episodes of illness (*4*). We agree that urinary tract infections with *Escherichia coli*, most of which come from the patient's own gut flora and for which the original source may be food, may fall into this category and that the number of such illnesses is large (*5*). Moreover, there are probably unknown or unrecognized agents in the food supply that cause illness other than gastroenteritis. We recognize the need to think of ways to include more of these agents—known and unspecified—in future estimates.

Hedberg noted that rates of acute gastroenteritis in the 3 FoodNet population surveys correlate with the number of reported foodborne norovirus outbreaks and suggested that we may have consequently underestimated illnesses caused by norovirus and overestimated illnesses caused by unspecified agents (*1*). Although such an association between FoodNet survey findings and reported norovirus outbreaks is possible, it should be treated with caution. The annual number of foodborne norovirus outbreaks reported during our study was probably influenced by improvements in diagnosis and surveillance. The steady increase in suspected and confirmed foodborne norovirus outbreaks during 1998–2003 was accompanied by a decrease in foodborne outbreaks of unknown etiology, suggesting that the higher number of norovirus outbreaks reported during 2006–2007 resulted, at least in part, from improved diagnosis. In addition, nonfoodborne outbreaks caused by a new norovirus strain increased during 2002–2003 and were not reflected in the population survey by an increased rate of gastroenteritis (*6*). The surveys varied in how questions were worded and ordered, especially the 2006–2007 survey compared with earlier surveys, and higher rates of acute gastroenteritis might be related to these variations. Moreover, the 2006–2007 increase in gastroenteritis reported by the surveys was driven by increases in diarrhea rather than vomiting (the latter is more suggestive of norovirus illness). The limited number of comparison data points—only 3—and the fact that the data were from different populations (all states vs. 10 FoodNet sites) during slightly different time periods (FoodNet surveys were conducted over 12 months but not in 1 calendar year) also warrant conservative assessment of the association between annual rates of acute gastroenteritis and number of foodborne norovirus outbreaks. Because of uncertainties in both measures, we preferred a rank-based method; the Spearman ρ gives an exact p value of 0.33.

To estimate average annual number of illnesses, we used data from several years (2000–2008) and probability distributions. The fraction of acute gastroenteritis estimated to be attributable to norovirus was wide (6%–26%), so it probably encompassed year-to-year variations in incidence. Not only is norovirus estimated to be the most common known cause of foodborne illness, but it is the illness for which we have the least surveillance information. Better data for norovirus are needed. Studies to examine the association between norovirus activity and reported rates of acute gastroenteritis of unknown cause could be useful. Although the most recent FoodNet survey of gastroenteritis was conducted in 2006, more surveys await funding.

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Comment on Zoonoses in the Bedroom

To the Editor: In response to Chomel and Sun (*1*), we would like to correct potentially misleading representations of risk factors for parasitic diseases. The authors correctly described risk for Chagas disease from exposure to infected insect vectors but included Chagas disease in the table, "Zoonoses acquired by close contact with pet, 1974–2010." The bloodborne protozoan that causes Chagas disease is transmitted not by contact with an infected mammal but by contact with a vector insect that has bitten an infected mammal (*2*).

For some parasitic zoonoses, contact with pets may not be a major source of infection. Molecular studies indicate that risk for human infection with *Giardia* and *Cryptosporidium* spp. from dogs and cats may be lower than previously believed. Infections with these parasites are usually with species-specific genotypes. Human infections with assemblages C, D (dog specific), and F (cat specific) of *G. duodenalis* have not been confirmed. Infections with assemblages A or B have been reported for humans and other animal species, including dogs and cats, but no direct transmission has been documented (*3*,*4*). Most human cryptosporidial infections are caused by *C. hominis* and *C. parvum* (*5*); a smaller percentage are caused by *C. canis* and *C. felis.*

Human infection with *Toxocara canis* or *T. cati* occurs when embryonated eggs are ingested; however, embryonation requires 2–4 weeks in the environment, suggesting that the risk from eggs in pet fur may be less than risk from exposure to eggs in contaminated soil. Other more serious zoonotic parasitic disease risks from contact with pet feces, including toxoplasmosis, are mentioned only briefly, if at all.

Physicians need information that accurately communicates zoonotic parasitic disease risks to their patients. However, inaccurate or overstated risk communication can also lead to unnecessary prevention efforts and misdirected concerns about dogs and cats as sources of disease.

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