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Review

Outbreaks Where Food Workers Have Been Implicated in the Spread of Foodborne Disease. Part 4. Infective Doses and Pathogen Carriage

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ABSTRACT

In this article, the fourth in a series reviewing the role of food workers in foodborne outbreaks, background information on the presence of enteric pathogens in the community, the numbers of organisms required to initiate an infection, and the length of carriage are presented. Although workers have been implicated in outbreaks, they were not always aware of their infections, either because they were in the prodromic phase before symptoms began or because they were asymptomatic carriers. Pathogens of fecal, nose or throat, and skin origin are most likely to be transmitted by the hands, highlighting the need for effective hand hygiene and other barriers to pathogen contamination, such as no bare hand contact with ready-to-eat food. The pathogens most likely to be transmitted by food workers are norovirus, hepatitis A virus, *Salmonella, Shigella,* and *Staphylococcus aureus.* However, other pathogens have been implicated in worker-associated outbreaks or have the potential to be implicated. In this study, the likelihood of pathogen involvement in foodborne outbreaks where infected workers have been implicated was examined, based on infectious dose, carriage rate in the community, duration of illness, and length of pathogen excretion. Infectious dose estimates are based on volunteer studies (mostly early experiments) or data from outbreaks. Although there is considerable uncertainty associated with these data, some pathogens appear to be able to infect at doses as low as 1 to 100 units, including viruses, parasites, and some bacteria. Lengthy postsymptomatic shedding periods and excretion by asymptomatic individuals of many enteric pathogens is an important issue for the hygienic management of food workers.

This article is the fourth in a series of several reviewing the role of food workers in foodborne outbreaks. Members of the Committee on Control of Foodborne Illnesses of the International Association for Food Protection analyzed 816 foodborne disease outbreaks in which food workers were implicated as the source of contamination *(80, 194, 195)* and grouped these outbreaks into different types of contamination scenarios. Specifically, this review deals with the doses of pathogens required to infect individuals. These pathogens have been or could be involved in foodborne disease outbreaks through worker infection or contamination. It also describes the incubation period, duration, symptomatic and asymptomatic carriage rates, and persistence of pathogen excretion. For the purposes of this report, ''ill'' is defined as an individual having unambiguous symptoms that alert the worker that handling food and food contact surfaces is not appropriate. ''Asymptomatic'' is defined as the condition of individuals who are not obviously ill but are colonized and shedding pathogens periodically; this can be for a short time before the illness develops (prodrome), subsequent to the illness in the recovery phase, or a long-term carrier. These individuals, their coworkers and management are typically unaware of their condition, and they are capable of contaminating the kitchen environment over a period of time unless they practice meticulous hygiene or use barriers to prevent pathogen contamination. Interestingly, from the review, an almost equal number of outbreaks occurred where workers were asymptomatic (a few were chronic excretors) as those where they were ill. Therefore, it is important to recognize the risks of infected but apparently well employees.

INFECTIVE DOSES FOR FOODBORNE AND OTHER ENTERIC PATHOGENS

High numbers of pathogens can be present in fecal matter, especially during diarrheal episodes, with levels of up to 10^{11} infectious cells or viral particles per ml or g of feces, although levels of 10^5 to 10^9 are more common *(7, 33, 65, 203).* These situations present opportunities for contamination by those preparing food and subsequent illnesses for those who consume the food. Clearly, the more pathogens consumed in a food or transferred through other fecal-oral route scenarios, the more likely an illness will result. Much work has been conducted to determine minimum in-

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TABLE 1. *Quantitative infectious dose data from volunteer studies and pathogen levels found in foods implicated in outbreaks*

Pathogen	Infectious dose from volunteer studies (CFU or no. of virus particles)	Infectious dose from outbreak data and estimates (CFU or no. of virus particles)	Levels in outbreak food samples (CFU/g or CFU/ml)	Pathogen implicated in food worker outbreaks		Reference(s)
				Inferred[a]	Published	
Campylobacter jejuni	500 (1 volunteer)			Rare	Rare	*166*
	800: 10% attack rate, 50% infected; 10^5: 46% attack rate, 85% infected			NA[b]	NA	*14*
	10^3–10^6, 1–60% attack rate			NA	NA	*106*
Clostridium perfringens		>10^8 (estimated)	≥10^6	No	No	*198*
		10^9				*85*
EPEC[c]	1950–1956 studies: 10^6–10^9; 1978 study of outbreak strains: 10^6–10^{10} with up to 50% attack rate for diarrhea in <20 h			Unknown, maybe in developing countries	Unknown, maybe in developing countries	*116*
		10^2 (estimated)				*106*
	1998 studies: 10^9–10^{10} with bicarbonate, 13–44% and 100% attack rates			NA	NA	*106*
ETEC[d]	10^7–10^9 in meal, 30–100% attack rate; 10^9 with bicarbonate, 70% attack rate			Rare in developed countries, more frequent in developing countries	Probable	*106*
EAEC[e]	10^8–10^{10} with bicarbonate, 25–38%, 60% attack rates			Unknown	Unknown	*106*
EIEC[f]		≤10, 10^1–10^3 (estimated)		Unknown, maybe in developing countries	Unknown, maybe in developing countries	*174, 198*
EHEC[g]		<100 (estimated)		Occasional for O157:H7	Occasional for O157:H7	*129*
Escherichia coli O157:H7		10 (estimated), 10–100		Occasional	Occasional	*17, 174*
E. coli O157:H7 in salami		2–45, children most at risk	0.3–0.4	NA	NA	*193*
E. coli O157:H7 in venison jerky		Mean 10^4	3–93	NA	NA	*185*
E. coli O157:H7 in hamburger patties		<13.5–675 (median 67.5 organisms/uncooked patty); median 23 in served undercooked burgers, children most at risk	<0.3–15	NA	NA	*185*
E. coli O157:H7 in pumpkin salad		Mean 31, school children	0.04–0.18	NA	NA	*185*
E. coli O157:H7 in melon given to children		Mean 1,100	43	NA	NA	*185*
E. coli O157:H7 in raw milk cheese		125–500	5–10	NA	NA	*185*
E. coli O111			0.1 in mettwurst	Not known	Not known	*129*

TABLE 1. *Continued*

Pathogen	Infectious dose from volunteer studies (CFU or no. of virus particles)	Infectious dose from outbreak data and estimates (CFU or no. of virus particles)	Levels in outbreak food samples (CFU/g or CFU/ml)	Pathogen implicated in food worker outbreaks		Reference(s)
				Inferred[a]	Published	
Listeria monocytogenes		10^3 (estimated)		No	No	*174*
			0.3 in frankfurters	NA	NA	*127*
Salmonella Typhi		15–20 to 10^3 (estimated)		Occasional but declining	Occasional	*18, 198*
	10^9, 95% attack rate; 10^8, 89% attack rate; 10^7, 50% attack rate; 10^5, 28% attack rate; 10^3, 0% attack rate			NA	NA	*92*
Nontyphoidal *Salmonella*		$<10^1$–10^{11} in 13 outbreaks involving 11 types of food		Frequent	Frequent	*39*
	10^5–10^9 with six serovars in eggnog, 17–100% attack rates			NA	NA	*106*
		Up to 10^6 but could be as low as 10–100 cells		NA	NA	*78*
Salmonella Anatum in egg roll filling			0.03	NA	NA	*68*
Salmonella Cubana in carmine capsules			10^6 dose: 4.57 log CFU, 70.9% attack rate	NA	NA	*38, 58, 111*
Salmonella Eastbourne in chocolate			0.02–2.5	NA	NA	*139*
Salmonella Enteritidis in ice cream		10 for children, 25–50 for adults	0.004–0.46, 65–73 g eaten	NA	NA	*204*
Salmonella Enteritidis in macaroni salad		44,000, 34% attack rate	1,000	NA	NA	*100*
Salmonella Enteritidis in plain rolled egg		11, 67% attack rate	0.135	NA	NA	*100*
Salmonella Heidelberg in Cheddar cheese			0.0018–0.0036	NA	NA	*57*
Salmonella Infantis in ham			23,000	NA	NA	*139*
Salmonella Javiana and Oranienberg in mozzarella cheese			0.0036–0.043	NA	NA	*86*
Salmonella Minnesota in protein dietary supplement			0.11–0.13	NA	NA	*139*
Salmonella Newport in ground beef			0.06–0.23	NA	NA	*56*
Salmonella Newport in hamburger			dose: 1.23 log CFU, 1.07% attack rate	NA	NA	*56, 58*

TABLE 1. *Continued*

Pathogen	Infectious dose from volunteer studies (CFU or no. of virus particles)	Infectious dose from outbreak data and estimates (CFU or no. of virus particles)	Levels in outbreak food samples (CFU/g or CFU/ml)	Pathogen implicated in food worker outbreaks: Inferred[a]	Pathogen implicated in food worker outbreaks: Published	Reference(s)
Salmonella Nima in chocolate			0.043–0.24	NA	NA	*3*
Salmonella Saintpaul, Rubislaw, and Javiana		4–45	0.04–0.45	NA	NA	*113*
Salmonella Schwarzengrund in powdered pancreatin (other serovars found in pancreatin were Eimsbuettel, Brandenburg, Livingstone, and Enteritidis)		Pediatric cystic fibrosis patients (<4 years old, mostly about 1.5 years old and on broad spectrum antibiotics); 31% in one group infected dose <50	<2/100 g (in one infant, 22–44 salmonellae were consumed per day for 36 h before symptoms)	NA	NA	*38, 121*
Salmonella Typhimurium in frozen egg yolk			0.6	NA	NA	*139*
Salmonella Typhimurium in imitation ice cream			dose: 3.79 log CFU; 55.0% attack rate	NA	NA	*4, 58*
Salmonella Typhimurium and Braenderup in frozen dessert			1.5	NA	NA	*139*
Salmonella Typhimurium in grated yam with soup		138,000, 40% attack rate	2,300	NA	NA	*100*
Shigella dysenteriae	<10	≥1 (estimated)		Probable in developing countries	Unknown, maybe in developing countries	*198*
	10^1 in milk, 10% attack rate; 2×10^2 in milk, 50% attack rate; 2×10^3 in milk, 70% attack rate; 10^4 in milk, 83% attack rate					*114*
Shigella flexneri 2a	1.4×10^3 in buffer, 27% attack rate in previously challenged volunteers and 92% in naïve subjects; 1.4×10^2 in buffer, 43% attack rate in naïve subjects			Occasional	Occasional	*107*
	10^2–10^3 in bicarbonate, 43–86% attack rate; 10^2–10^4 in milk, 22–57% attack rate			NA	NA	*106*
Shigella sonnei	500 in milk, 47–55% attack rate			Occasional	Occasional	*106*
Staphylococcus aureus		<1–5 μg enterotoxin produced with $\geq 10^5$ cells	10^5–10^8	Frequent but declining	Frequent	*198*
		144 ± 50 ng enterotoxin		NA	NA	*54*

TABLE 1. *Continued*

Pathogen	Infectious dose from volunteer studies (CFU or no. of virus particles)	Infectious dose from outbreak data and estimates (CFU or no. of virus particles)	Levels in outbreak food samples (CFU/g or CFU/ml)	Pathogen implicated in food worker outbreaks		Reference(s)
				Inferred[a]	Published	
Streptococcus pyogenes (group A)		≤10^3 (estimated)		Occasional, declining	Occasional	*174, 198*
Streptococcus group D		>10^7 (estimated)		Rare	Rare	*198*
Streptococcus faecalis subsp. *liquefaciens*				Not known	Not known	*139*
Vibrio cholerae O1 and O139				Likely occasional in endemic areas in developing countries	Likely in developing countries	*74*
	10^8–10^{11} with no buffering, 10^3–10^4 with bicarbonate buffer or mixed with food, 60–100% attack rates with 10^5	Typical outbreak dose estimated to be 10^2–10^3; higher risk for achlorohydric individuals		NA	NA	*106, 147*
Vibrio cholerae non-O1		10^6–10^{11} (estimated)		Not known	Not known	*174*
	10^6–10^9, 67–100% attack rate			NA	NA	*106*
	Most pathogenic strain: 10^6–10^9, 83% attack rate; 10^3–10^8 CFU/g stool at peak excretion			NA	NA	*134*
Vibrio parahaemolyticus			10^3–10^4	Not known	Not known	*68*
	10^5–10^7 (Kanagawa phenomenon positive)			NA	NA	*147*
	10^7–10^9, 50–100% attack rate			NA	NA	*106*
Vibrio vulnificus		Very low in susceptible populations		Not known	Not known	*147*
Yersinia enterocolitica	10^9 for 1 volunteer	10^8–10^9 (estimated)		Occasional	Occasional	*40*
		10^2, 10^6		NA	NA	*174*
Hepatitis A virus		10^1–10^2 (estimated)		Frequent	Frequent	*198*
Norovirus		≤100 (estimated)		Frequent and increasing	Frequent	*24*
		10–100		NA	NA	*69*
	82% of volunteers became infected; of these infections, 68% resulted in illness, whereas the remaining 32% were asymptomatic			NA	NA	*76*
	50–62% of volunteers at doses from ≤10^4 to 10^8 in individuals with a epithelial binding gene (Se+); those without (Se−) even at 10^8 dose did not develop diarrhea			NA	NA	*120*

TABLE 1. *Continued*

Pathogen	Infectious dose from volunteer studies (CFU or no. of virus particles)	Infectious dose from outbreak data and estimates (CFU or no. of virus particles)	Levels in outbreak food samples (CFU/g or CFU/ml)	Pathogen implicated in food worker outbreaks: Inferred[a]	Pathogen implicated in food worker outbreaks: Published	Reference(s)
Norovirus (cont'd)	Confirmed Lindesmith findings but with higher infection rates in Se+ individuals			NA	NA	*93*
Rotavirus	25–50% symptomatic with a dose of 10^1–10^4	10^1–10^2 (estimated)		Occasional	Not known but possible	*2, 198*
	93% infectivity with 10^3 dose					*205*
		10				*43*
Cryptosporidium parvum	10–100 oocysts, 9–1,042 oocysts	1–30 oocysts	<56 oocysts	Rare	Rare	*28, 106, 131, 198*
	Infection but no symptoms = 30 oocysts; ID_{50} = 132 oocysts[h]; 100% infection = 1,000 oocysts			NA	NA	*51*
Cyclospora cayetanensis		Assumed to be low		Not known but probable	Not known	*88*
	200–49,000 oocysts did not cause symptoms in 7 volunteers			NA	NA	*1*
Entamoeba coli	1 cyst	1 cyst (estimated)		Not known	Not known	*106, 198*
Giardia lamblia	10 cysts	1 cyst (estimated)		Rare	Rare	*27, 198*
	10 cysts (0% receiving 1 cyst, 36.4% receiving 10–25 cysts, and all who received 100 cysts became infected)			NA	NA	*163*

[a] Our inferences based on various sources.
[b] NA, not applicable.
[c] EPEC, enteropathogenic *E. coli*.
[d] ETEC, enterotoxigenic *E. coli*.
[e] EAEC, enteroaggregative *E. coli*.
[f] EIEC, enteroinvasive *E. coli*.
[g] EHEC, enterohemorrhagic *E. coli*.
[h] ID_{50}, median infective dose.

fectious doses for foodborne pathogens from volunteer studies and counts in food following an outbreak, but accurate determination with this approach is not possible because in theory one infectious unit of a bacterium, virus, or parasite has the potential to cause an intestinal infection. A few bacterial cells (or even one) can multiply rapidly under grossly abusive time-temperature conditions; therefore, even low levels of pathogen contamination in a food can result in ingestion of large numbers of organisms. In volunteer studies, levels of organisms given to small numbers of healthy adults tended to be high and often not all of these individuals showed symptoms. For instance, Hornick et al. *(91)* stated that 200 cells of *Shigella* spp., 10^5 cells of *Salmonella* Typhi, 10^8 to 10^{10} cells of *Escherichia coli,* and 10^7 cells of *Vibrio cholerae* are sufficient to cause diarrheal infections with about a 20 to 30% attack rate in volunteers, but the infectious dose was dependent on the pH of the inoculum. In contrast, much lower levels of pathogens were occasionally found in leftover food associated with outbreaks, but we do not know the actual amounts eaten by affected individuals, the precise pathogen levels in those food portions, or the immune status of those affected. Nor do we know the values of the same parameters for those who ingested food but were not ill. We reviewed the literature to record what is known about doses, both low and high, in causing illness through volunteer study data and pathogen levels found in foods implicated in outbreaks (Table 1). These types of data can determine an infectious dose with an attack rate only in a defined population or the concentration of the pathogen only in the food consumed. More detailed discussion of infectious doses can be found in the *Hazard Characterization for Pathogens in Food and Water: Guidelines (59).* Whether volunteer experiments, especially those carried out many decades ago, are valid reproductions of real-life scenarios is frequently questioned *(39).* For instance, in one study the recipients of the *Salmonella* Typhi cultures delivered in milk were typically healthy adult males, and despite the fact that this organism is widely assumed to cause infection at a much lower dose than that required by other salmonellae, the median infectious dose (10^7 CFU) was still relatively high when the cultures were ingested *(92).* The Quailes and Zermat strains chosen may not have been be typical of most typhoid fever strains, but they were chosen because they contained the Vi (virulence) antigen. Conversely, it is rarely possible to determine an attack rate with a known dose in an outbreak situation. In some cases, very low levels have been found in food samples tested after outbreaks (Table 2). Stomach acids are one of the body's defense mechanisms against gastrointestinal infections. Gastric fluid consists of HCl and pepsin and can kill bacteria within 15 min when the pH is less than 3.0 *(67).* Individuals with a reduced amount of stomach acid or who are taking antacids or other medications, particularly to counteract gastroesophageal reflux disease, would require lower doses of pathogens to become infected. When the pH is raised above 4.0, bacterial overgrowth may occur. Acquired hypochlorhydria (low gastric acid output) can result from atrophic gastritis, malnutrition, and other conditions frequent in the community *(191),* such as celiac disease, Addison disease, asthma, eczema, diabetes mellitus, chronic hives, psoriasis, rosacea, and osteoporosis, and iatrogenic hypochlorhydria can be caused by gastric surgery or by drugs that inhibit acid secretion. Tennant et al. *(191)* demonstrated experimentally that 2.5-fold more *Yersinia enterocolitica,* 5.4-fold more *Salmonella* Typhimurium, and 13.6-fold more *Citrobacter rodentium* survived passage through the stomachs of hypochlorhydric mice (≈pH 7) than through the stomachs of hyperchlorhydric mice (<pH 3.6). Buffering of a dose for volunteers, such as with sodium bicarbonate, typically yields a higher attack rate *(58, 76).* The fat content of the food matrix also may protect the organism from stomach acids and is an important factor in outbreaks related to foods such as chocolate, tahini, and hamburgers whether individuals have low stomach acidity or not (Table 2). Thus, it is impossible to determine the exact minimum infectious doses for individuals or even populations, but lower infectious doses can be expected for high-risk people, such as those who are young, old, on medication, or in areas with a high rate of malnutrition.

In general, we expect that pathogens with very low infectious doses could be more easily transmitted by infected food workers. In contrast, we anticipate that certain pathogens requiring relatively large numbers to infect healthy populations would be less likely to be involved in outbreaks associated with food workers. However, because rapid growth under abusive time-temperature conditions can result in ingestion of large numbers of organisms, even organisms with high infectious doses can cause disease. Organisms apparently requiring large numbers of cells for colonization include *Clostridium perfringens,* enteropathogenic *E. coli* (EPEC), enterotoxigenic *E. coli* (ETEC), enteroaggregative *E. coli* (EAEC), *Listeria monocytogenes,* some strains of nontyphoidal *Salmonella, Staphylococcus aureus, Streptococcus* group D, *V. cholerae,* and *Vibrio parahaemolyticus.* No evidence has directly implicated *C. perfringens* or *Listeria* in food worker–related outbreaks. However, *C. perfringens* is frequently found in food environments where there is raw meat and poultry. Bryan and Kilpatrick *(20)* found that on visits to a roast beef sandwich restaurant on three successive days, many pieces of equipment, e.g., slicer, knife, scales, thermometers, towels, environment, work table countertop, and fan, were contaminated with spores of *C. perfringens.* All nine stool specimens collected from seven workers and 4 of 10 hand-rinse cultures were positive for *C. perfringens.* Although the strains were not typed to establish associations, it is likely that workers and food environments are frequently contaminated with this pathogen, and outbreak opportunities are limited only by preventing temperature abuse of cooked meat and poultry. *L. monocytogenes* has been implicated in the contamination of delicatessen meats at both the processing and the retailing steps, and bacterial transfer has occurred from slicing machines *(102, 142),* but no direct link with the fecal-oral route involving food workers has been established despite the fact that this pathogen has been found occasionally in stools *(81, 173).*

Similarly, *V. parahaemolyticus* never has been linked

TABLE 2. *Epidemiological features of foodborne pathogens transmitted by infected food workers*

Pathogen	Incubation period (days unless otherwise noted)	Duration of illness (days unless otherwise noted)	Carriage rates reported	Postsymptomatic shedding (days unless otherwise noted)	% of individuals who excrete the pathogen but are asymptomatic	Clinical source of pathogen[a]	Reference(s)
Aeromonas spp.		Dysentery-like syndrome can last weeks				F	*198*
			England: 5.7–6.0% of GI cases,[b] 3.2–5.2% of controls			F	*196*
			London: 4.2% of unselected hospital stool samples			F	*132*
			Nigeria: 2.5% in wards and clinics		Nigeria: 1.0% in wards and clinics		*52*
			Nigeria: 3% of diarrheal children (≤5 yr old), 1.9% of healthy children (≥6 yr old)			F	*199*
			Mumbai: 1.8% of diarrheal cases			F	*44*
Campylobacter jejuni	2–5 (1–10)	2–5 (≥10)				F	*36*
	2–5	2–10					*26*
	2–5	7–10					*198*
				2–3 wk; if untreated, relapses occur in 20% of patients		F	*126*
			England: 4.2–12.2% of GI cases, 0.5–0.9% of controls			F	*196*
	2–5 (11)	<1–7 wk	5–11% of diarrheal cases	≤7 wk	1.3–13%	F	*99*
			Puget Sound: 3.6% of diarrheal cases			F	*186*
		3–6	South Africa: 31% of diarrheal children 0–8 mo; 38% of diarrheal children 9–24 mo		Asymptomatic children: 5% (0–8 mo old), 40% (9–24 mo old)	F	*16*
			U.S. hospitals: 2.33% of diarrheal cases			F	*180*
			Belgium children with GI: 0 to <8 mo, 31%; 8–24 mo, 38–40%			F	*41*
			Thai children with GI: 10–22% from <6 to 60 mo, mean 18% (*C. coli* and *C. jejuni*)			F	*189*
					Melbourne: 0.1%	F	*87*

TABLE 2. *Continued*

Pathogen	Incubation period (days unless otherwise noted)	Duration of illness (days unless otherwise noted)	Carriage rates reported	Postsymptomatic shedding (days unless otherwise noted)	% of individuals who excrete the pathogen but are asymptomatic	Clinical source of pathogen[a]	Reference(s)
C. jejuni (cont'd)				Norway: 16% of cases excreted *Campylobacter* for 15–69, median 31		F	*98*
			Seoul: 0.4% of child diarrheal cases			F	*103*
			Israel: 0.0–0.6% of military diarrheal cases			F	*35*
			Israel: 6.3% of child diarrheal cases (Bedouin)		Israel: 4.5% of Bedouin children	F	*12*
			Netherlands: 1.3% in GI cases		Netherlands: 0.6%	F	*46*
			Asymptomatic rate: nonpregnant adults, <1%; pregnant adults, 0.2–1.6%			F	*126*
			Mumbai: 1.5% of diarrheal cases			F	*44*
			North India (*Campylobacter* sp.): 13.5% of diarrheal cases, 0.6% of asymptomatic controls			F	*95*
					Adelaide: 9–77%	F	*104*
Enterohemorrhagic *Escherichia coli* (STEC, VTEC)[c]	1–8	5–10				F, Shiga toxin in B, stool, kidneys	*26*
	2–10	5–10					*36*
E. coli O157:H7		4–10, longer for sequelae		Prolonged carriage is uncommon but can occur		F	*26*
		2–6 (1–8); 3 wk in one-third of children					*68*
			England: 0.0–0.1% of GI cases, 0.0% of controls			F	*196*
				Argentinian day-care centers: O157:H7, 23–30; O26:H11, 31–37; O145:NM, 19. U.S. day-care center: O157:H7, 11–57. German day-care center: O157:H7, 2–62		F	*130*

TABLE 2. *Continued*

Pathogen	Incubation period (days unless otherwise noted)	Duration of illness (days unless otherwise noted)	Carriage rates reported	Postsymptomatic shedding (days unless otherwise noted)	% of individuals who excrete the pathogen but are asymptomatic	Clinical source of pathogen[a]	Reference(s)
E. coli (cont'd)					Ontario outbreak: 31% exposed to raw milk source were asymptomatic; 53% of the asymptomatic children had laboratory evidence of infection	F	*50*
				Typically <10		F	*160*
			U.S. hospitals: 0.57% in northern states; 0.13 in southern states (mean 0.39%)			F	*180*
			Puget Sound: 0.6% of diarrheal cases			F	*186*
			Calgary: 0.6%, verotoxin detected in 2.1% of diarrheal cases			F	*161*
	3–4 (2–8)			Canadian outbreak: 14% excreted for 10–39 after symptoms stopped		F	*150*
E. coli non-O157 VTEC, STEC			England: 0.2–0.4% of GI cases, 0.5–1.0% of controls			F	*196*
			VTEC Netherlands: 0.3% in GI cases		VTEC Netherlands: 0.2%	F	*46*
			Kenya: 0.1% (food workers by PCR only)			F	*153*
			VTEC Mexico City: 8.6% of child diarrheal cases		VTEC Mexico City: 1.2% (children)	F	*156*
			Lagos: 6% (50% children) of diarrheal cases			F	*148*
EAEC[d]			Seoul: 15% of child diarrheal cases			F	*103*
			Israel: 14.8% of child diarrheal cases (Bedouin)		Israel: 9.2% of children (Bedouin)	F	*12*
			Kenya: 6.3% of diarrheal cases of food workers		Kenya: 2.0% (food workers)	F	*153*
			England: 2.9–5.1% of GI cases, 0.5–1.8% of controls			F	*196*

TABLE 2. *Continued*

Pathogen	Incubation period (days unless otherwise noted)	Duration of illness (days unless otherwise noted)	Carriage rates reported	Postsymptomatic shedding (days unless otherwise noted)	% of individuals who excrete the pathogen but are asymptomatic	Clinical source of pathogen[a]	Reference(s)
EIEC[e]	0.5–3	3 to >7	Mexico City: 1% of child diarrheal cases			F	*26, 198*
			Seoul: 0.4% of child diarrheal cases			F	*103*
EPEC[f]	0.5–3	3–14				F	*36*
			England: 0.1% of GI cases, 0.2% of controls			F	*196*
			Seoul: 6% of child diarrheal cases			F	*103*
			Mumbai: 20.7% of diarrheal cases			F	*44*
			Kenya: 0.8% (food workers by PCR only)			F	*153*
			Mexico City: 9.3% of child diarrheal cases		Mexico City: 1.2% (children)	F	*156*
ETEC[g]	1–3	3 to >7				F	*26*
	≥1 (0.5–3)						*198*
	0.5–3	3–14					*36*
			England: 1.6–1.9% of GI cases, 0.0% of controls			F	*196*
			Seoul: 22% of child diarrheal cases			F	*103*
			Mumbai: 11% of diarrheal cases			F	*44*
			Israel: 0.6% of child diarrheal cases (Bedouin)		Israel: 1.6% of children (Bedouin)	F	*12*
			Mexico City: 13.3% of child diarrheal cases		Mexico City: 2.5% (children)	F	*156*
			Israel: 15–32% of military diarrheal cases			F	*35*
Listeria monocytogenes	0.25–2 (GI type)	Variable, many weeks				F	*26*
	2–6 (10) wk (invasive type)						*36*
							198
				0.6–3.4% with no known exposure to *Listeria*; 0–21% with likely exposure to *Listeria*		F	*154*

TABLE 2. *Continued*

Pathogen	Incubation period (days unless otherwise noted)	Duration of illness (days unless otherwise noted)	Carriage rates reported	Postsymptomatic shedding (days unless otherwise noted)	% of individuals who excrete the pathogen but are asymptomatic	Clinical source of pathogen[a]	Reference(s)
L. monocytogenes (cont'd)			Austria: 0.8% of stools of healthy persons; 3.6% by PCR, 1.15% by culture			F	*81*
			New York State: 0.12% of diarrheal cases; 1 isolate from a stool sample from a person with invasive listeriosis			F	*173*
Salmonella Typhi and Paratyphi	Typhi: 8–14; Paratyphi: 1–10	Many days to weeks		Chronic asymptomatic carriers occur		F, U, B	*36*
				More women carriers than men (3:1)		F	*90*
	3–60 (7–14)		Calcutta: 37.3% with typhoid fever symptoms			F	*162*
			2–4% chronic carriage, women almost twice that of men, higher in older age groups			F	*21*
				Salmonella Paratyphi B: 6 yr in one case		F	*60*
				Salmonella Typhi: carrier in outbreak infected >7 yr		F	*209*
	Volunteers: 4–11 but usually 7–9, depending on *Salmonella* Typhi dose					F	*71*
Nontyphoidal *Salmonella*					>50% of convalescent patients stop excreting at <5 wk, and 90% stop within 9 wk; 0.7% are excretors at 12 mo; 0.5% of asymptomatic persons can excrete at 12 mo	F	*21*
			England: 1.1–5.0% of GI cases, 0.2–0.4% of controls			F	*196*
			UK: 0.15% of children; Tokyo: 0.15%			F	*38*

TABLE 2. *Continued*

Pathogen	Incubation period (days unless otherwise noted)	Duration of illness (days unless otherwise noted)	Carriage rates reported	Postsymptomatic shedding (days unless otherwise noted)	% of individuals who excrete the pathogen but are asymptomatic	Clinical source of pathogen[a]	Reference(s)
Salmonella Enteritidis and other serotypes	1–3	4–7				F	*26*
	0.25–2 (1–2)	1–2 (longer with sequelae					*198*
	0.5–3	3–5					*36*
				Outbreaks: median duration 5 wk	Developed countries: 0.23% (adults); 0.98% (children). Food workers in developed countries: 1.7%, maximum 10.3%. Developing countries: 1.19% (adults); 2.4% (children). Food workers in developed countries: 6.21%, maximum 18.7%	F	*31*
				4–22 wk in infants <12 mo old		F	*6*
			Sweden: 5.4% of diarrheal cases; 4.6% of healthy cases; 73% of asymptomatic contacts with index cases			F	*96*
			Puget Sound: 1.5% of diarrheal cases			F	*186*
			Netherlands: 0.4% of GI cases		Netherlands: 0.3%	F	*46*
				Bangkok food workers: 2–4% shedding after 7–90 days; reinfection with another serotype (21–24%)	Bangkok: 4.7%, 10% in food workers	F	*179*
				12 days–5 wk			*178*

TABLE 2. *Continued*

Pathogen	Incubation period (days unless otherwise noted)	Duration of illness (days unless otherwise noted)	Carriage rates reported	Postsymptomatic shedding (days unless otherwise noted)	% of individuals who excrete the pathogen but are asymptomatic	Clinical source of pathogen[a]	Reference(s)
Salmonella Enteritidis and other serotypes (cont'd)			U.S. hospitals: 1.82%			F	*180*
			Kenya: 16% of diarrheal cases (food workers)			F	*153*
			Israel: 0.0–0.7% of military diarrheal cases			F	*35*
			Seoul: 0.4% of child diarrheal cases			F	*103*
					Mexico: 3.7% (children)	F	*156*
			Mumbai: 4.1% of diarrheal cases			F	*44*
					Melbourne: 0.4%	F	*87*
Shigella spp.	0.5–6 (1.5–3)					F	
	1–2	4–7					*26*
	0.5–2						*198*
	0.5–4	4–7					*36*
	3–6.1 (mean 5.2) for *S. dysenteriae* in volunteers						*114*
			England: 0.1–0.8% of GI cases, 0.0% of controls			F	*196*
		Mean 7.1 (2–90)		<4 wk to months			*25*
			U.S. hospitals: 1.06%			F	*180*
			Mumbai: 1.7% of diarrheal cases			F	*44*
			Mexico City: 7.5% of child *Salmonella* and *Shigella* diarrheal cases			F	*9*
			Mexico City: 2.6% of child diarrheal cases			F	*156*
			Seoul: 1.7% of child diarrheal cases			F	*103*
			S. sonnei: 17-mo carrier state in an institutionalized mentally disabled teen despite antibiotic treatment. *S. flexneri*: 16-mo carrier state in a correctional inmate who was a volunteer for dose-response studies. Colonization site in both cases was the colon with 10^4–10^8 CFU/g feces excreted				*115*

TABLE 2. *Continued*

Pathogen	Incubation period (days unless otherwise noted)	Duration of illness (days unless otherwise noted)	Carriage rates reported	Postsymptomatic shedding (days unless otherwise noted)	% of individuals who excrete the pathogen but are asymptomatic	Clinical source of pathogen[a]	Reference(s)
Shigella spp. (cont'd)			Israel: 6–20% of military diarrheal cases			F	*35*
			Bangkok: up to 52% of mucoid or bloody diarrheal cases			F	*122*
			Netherlands: 0.0% in GI cases		Netherlands: 0.0%	F	*46*
Staphylococcus aureus	1–6 h	1–2				N, S, T	*26*
	0.5–8 h	1					*36*
	1–8 h (mean 4.4 h)	mean 20 h					*89*
			England: 0.1–0.4% of GI cases, 0.1–0.2% of controls (fecal counts $>10^6$ CFU/g)			F	*196*
			Perineal carriage rate: 25%			S	*10*
			U.S. national survey: 32.4%, 56.5% of these with enterotoxin genes			N	*109*
			Nasopharynx carriage rates 14–64% in 14 studies, median 27.5%; 20–75% carry intermittently, 10–35% carry persistently (18% over 8 yr)			N	*201*
			Santiago food workers: 34.3%, 54.3% producing enterotoxin			N	*55*
			UK hospital admissions: nose, 30–60%; stool, 10–33%			N, F	*158*
Streptococcus pyogenes				21–60	20–65%	S, T	
Vibrio cholerae O1	1–3	3–7				F	*26*
	0.25–5				4%		*198*
	1–5 (1.5)						
	Hours to 5 days	3–4					*36*
	0.25–5	3–9 with treatment, 0.9% case fatality rate	Mumbai: 33% of diarrheal cases			F	*44*
	Singapore outbreak: 4–203 h, median 38 h					F	*72*

TABLE 2. *Continued*

Pathogen	Incubation period (days unless otherwise noted)	Duration of illness (days unless otherwise noted)	Carriage rates reported	Postsymptomatic shedding (days unless otherwise noted)	% of individuals who excrete the pathogen but are asymptomatic	Clinical source of pathogen[a]	Reference(s)
V. cholerae O1 (cont'd)				Nigerian convalescent patients discharged with tetracycline: 9 of 13 after 2 wk, 4 of 13 after 3–6 mo, 2 of 13 after 7 mo (reinfection possible)		F	*200*
V. cholerae non-O1	10 h (range 5.5–96 h) with 10^5–10^9 dose in volunteers	21 h (range 3.5–48 h) in volunteers			Asymptomatic carriage rates, about 4% in persons with high-risk activities such as eating oysters in New Orleans or going on pilgrimage to Mecca		*134*
	Outbreak1: 11.5 h (range 5.25–37.5 h)	18–24 h					*136*
	Outbreak 2: 20–30 h	<24 h					*136*
Vibrio spp.			England: <0.1% of GI cases, 0.0% of controls			F	*196*
Yersinia enterocolitica	1–2	1–3 wk				F	*26*
	3–7	1–21					*36*
		Months with chronic enterocolitis					*165*
			England: 0.8–3.4% of GI cases, 2.4–3.1% of controls (*Yersinia* spp.)			F	*196*
	2–11						*141*
		Mean 8.4 (4–10)	Santiago: 1.1–1.9% of <4-yr-old diarrheal children. Maryland: 2% of <2-yr-old diarrheal children. Montréal: 2.8% of 2-yr-old diarrheal children. Bangladesh: 0.06% of <7-yr-old diarrheal children	Santiago: intermittent carriage by asymptomatic children up to 14 wk with pathogenic strains	Santiago: 0.2–6% with pathogenic strains	F	*135*
			Mumbai: 0.1% of diarrheal cases			F	*44*
			Netherlands: 0.4% of GI cases		Netherlands: 0.8%	F	*46*

TABLE 2. *Continued*

Pathogen	Incubation period (days unless otherwise noted)	Duration of illness (days unless otherwise noted)	Carriage rates reported	Postsymptomatic shedding (days unless otherwise noted)	% of individuals who excrete the pathogen but are asymptomatic	Clinical source of pathogen[a]	Reference(s)
Y. enterocolitica (cont'd)			Nigeria: 1.5% of diarrheal patients, highest in children 1–10 yr old (7.5%)				*146*
					Belgium: 81% of children in an outbreak	F	*202*
Enteroviruses			U.S.: 10% in soiled diapers; 30–40% in summer months				*65*
Hepatitis A virus	10–50 (28)	2–3 wk (occasionally months)		21 (180)		F, U (antibodies in serum)	*26*
	10–50 (mean 30)	1–2 wk (months)		≤5 mo			*198*
	15–50	1–2 wk					*36*
			U.S.: 9.7/100,000 people	30–45	Italy: 8.2%		*65*
				6 mo after diagnosis of infection in premature infants			*138*
Hepatitis E virus	15–45	≤5 mo		≤8 wk		F (antibodies in serum)	*164*
	2–8 wk	≤8 wk					
	22–60, mode 40	about 3 wk					*37*
Adenovirus (enteric)		3	Worldwide: 4–17% of diarrheal children. Buenos Aires: 3.3% of diarrheal cases, mainly children		Buenos Aires: 0.8% of asymptomatic persons	F	*133*
Adenovirus types 40, 41			England: 1.8–3.1% of GI cases, 0.1% of controls			F	*196*
Norovirus (caliciviruses, Norwalk-like virus)	0.6–3.2 (1–2)					F, V	*26*
	1–3	0.5–2.5		2 to >14			*105*
	0.5–2						*24*
	1–2	1–2.5					*198*
	1–2	0.5–2.5					*36*
	15–77 h	Mean or median 12–60 h					*108* (based on Kaplan criteria)
	Volunteers: 15 h (peaked at 25–72 h) after virus given				Volunteers: viral antigen in stool was detected ≤2 wk after virus given	F	*76, 145*
	Outbreak: 2–61 h (median 34 h)	Outbreak: 21–180 h		≥14			*75*
		0.5–3.5	6.8% of hospitalized diarrheal patients	≥14		F	*69*

TABLE 2. *Continued*

Pathogen	Incubation period (days unless otherwise noted)	Duration of illness (days unless otherwise noted)	Carriage rates reported	Postsymptomatic shedding (days unless otherwise noted)	% of individuals who excrete the pathogen but are asymptomatic	Clinical source of pathogen[a]	Reference(s)
Norovirus (caliciviruses, Norwalk-like virus) (cont'd)	1–2						*42*
	10–51 h			2 wk		F	*164*
			England: 1.1–1.5% of GI cases, 0.2% of controls (calicivirus)			F	*196*
		Elderly: 3–4, up to 19		Variable but 70% in elderly patients		F	*73*
			Netherlands: 16.1% in GI cases		Netherlands: 5.2%	F	*46*
			England: 6.5–7.0% of GI cases, 0.1–0.5% of controls			F	*196*
Rotavirus	1–3	4–8				F, V	*26*
	1–3	4–8					*198*
	1–3	Up to 7					*36*
	11 h to 6 days	5		≤2 wk		F	*164*
			England: 0.2–0.3% of GI cases, 0.0% of controls			F	*196*
			Seoul: 47% of child diarrheal cases			F	*103*
			Melbourne: 82.4% of child diarrheal cases			F	*8*
			Mexico City: 28.3% child diarrheal cases		Mexico City: 29.9% (children), 20.5% (adults)	F	*9*
			Israel: 4.3% child diarrheal cases (Bedouin)		Israel: 0.8% of children (Bedouin)	F	*12*
			Netherlands: 7.3% of GI cases		Netherlands: 0.7%	F	*46*
			Virginia: 25% in children with enteritis; 11.6% in adults with enteritis		Virginia: 8.3% (children), 0.0% (adults)		*167*
					Children in the community: 16% for ages 1 mo to 12 yr. Children in daycare: 12–30%. Patients without diarrhea in South Africa: 13%. Patients without diarrhea in Europe and Australia: 38–48%		*84*
			U.S.: 10.4% annual rate for infection in children	8–23			*65*

TABLE 2. *Continued*

Pathogen	Incubation period (days unless otherwise noted)	Duration of illness (days unless otherwise noted)	Carriage rates reported	Postsymptomatic shedding (days unless otherwise noted)	% of individuals who excrete the pathogen but are asymptomatic	Clinical source of pathogen[a]	Reference(s)
Rotavirus (cont'd)					Paris, hospitalized children ≤2 yr old: asymptomatic rotaviral infection and disease affected 2% of neonates, 20% of children 1–6 mo old, and 37% of children 7–24 mo old. Virus carriage occurred in 27, 19, and 14% of those children, respectively	F	*32*
Astrovirus	10–70 h	2–9		≥6		F	*198*
	2–4	<4		1 wk			*164*
			1 of 16 volunteers developed vomiting and diarrhea; 10 of 16 developed rises in serum antibody against astrovirus			F	*110*
			England: 2.0–3.0% of GI cases, 0.2% of controls			F	*196*
			Netherlands: 2.0% of GI cases		Netherlands: 0.6%	F	*46*
Adenovirus					Melbourne: 0.1%	F	*87*
Adenovirus (enteric)		3	Worldwide: 4–17% of child diarrheal cases. Buenos Aires: 3.3% of diarrheal cases, mainly children		Buenos Aires: 0.8% of asymptomatic persons	F	*133*
Adenovirus types 40, 41			England: 1.8–3.1% of GI cases, 0.1% of controls			F	*196*
Cryptosporidium spp.	2–10	Weeks to months				F	*26*
	2–4	1–4 wk					*198*
				≤5 wk			*181*
	1–12	4–21					*36*
	2–12 (median 7)	20–35					*176*
	5–22 (mean 9, median 6.5) in volunteers	2.5–3.5 in volunteers			U.S.: 15% in persons with past infections. Tropical developing countries: almost 100%	F	*51*

TABLE 2. *Continued*

Pathogen	Incubation period (days unless otherwise noted)	Duration of illness (days unless otherwise noted)	Carriage rates reported	Postsymptomatic shedding (days unless otherwise noted)	% of individuals who excrete the pathogen but are asymptomatic	Clinical source of pathogen[a]	Reference(s)
Cryptosporidium spp. (cont'd)			England: 0.4–13% of GI cases, 0.0% of controls			F	*196*
			Iran: 4–7% in healthy people; 50% in immunodeficient people; 11.5–20% in hemodialysis patients; 27.5% in renal transplant patients			F	*175*
					Peru: 70–76%	F	*152*
			Naples: 4.4% in immunocompetent children; 4.8% in immunodeficient children		Naples: 6.4% in immunocompetent children; 22.0% in immunodeficient children		*159*
			U.S.: 3.8% in all age groups; 8–26% of children in day care: 1–3% of children not in day care. Developed countries: 0.6–20%	7–15 (2 mo)	Day care: 27–50%		*65*
			Netherlands: 2.0% in GI cases		Netherlands: 0.1%	F	*46*
			Israel: 6.6% of child diarrheal cases (Bedouin)		Israel: 1.5% of children (Bedouin)	F	*12*
					Melbourne: 0.4%	F	*87*
					Venezuela: 71.4%	F	*30*
			14 studies: 1.1–9.1% in developed and 7.7–11.1% in developing countries. Children mean: 1.8% in developed countries, 4.9% in developing countries				*140*
			Developed countries: 2.2% (14% with HIV). Developing countries: 6.1% (24% with HIV)		Developed countries: 0.2% (0.0% in persons with HIV). Developing countries: 1.5% (5% in persons with HIV)	F	*82*
Cyclospora cayetanensis (7–14 day sporulation time needed after excretion to be infective)	1–14 (7)	Weeks to months				F	*26*
					Haiti and Peru: 72–94%	F	*152*
			Venezuela: 6.1% (only 2 symptomatic infants)		Venezuela: 5.2%	F	*30*

TABLE 2. *Continued*

Pathogen	Incubation period (days unless otherwise noted)	Duration of illness (days unless otherwise noted)	Carriage rates reported	Postsymptomatic shedding (days unless otherwise noted)	% of individuals who excrete the pathogen but are asymptomatic	Clinical source of pathogen[a]	Reference(s)
C. cayetanensis (cont'd)			Netherlands: 0.0% of GI cases		Netherlands: 0.0%	F	*46*
			Peru: 1.1% (children)		Lima: 68%	F	*124*
Entamoeba histolytica	2–3 days to 1–4 wk	Weeks to months		Years	10%	F	*26*
	2–4 wk						*36*
			Netherlands: 0.1% of GI cases		Netherlands: 0.0%	F	*46*
			Mexico City: 70.3% of child diarrheal cases		Mexico City: 43.7% (children)	F	*156*
					Melbourne: 1.5% (*Entamoeba* spp.)	F	*87*
			Israel: 0.0–0.9% of military diarrheal cases			F	*35*
			India: 7.4% in symptomatic and asymptomatic persons in a survey of a slum				*171*
					Food workers: Tacoma (Wash.) food service and retail: 1.2%. San Francisco: restaurants, 2.4%; restaurants and hotels, 2.9%. Chicago large hotel on two occasions: 7.1–15.0%. Prisoner mess force: 7.2%. Leningrad retail shops: 22.8%		*97*
Giardia lamblia (*G. intestinalis*)	1–2 wk	Days to weeks				F	*26*
	3–25 (7)	1–2 wk (months or years)		Months			*27*
	7	1–2 wk to months					*198*
	1–3 wk						*36*
			England: 0.4–1.0% of GI cases; 0.3–0.5% of controls			F	*196*
			Mexico City: 33.3% of child diarrheal cases		Mexico City: 20.0% (children)	F	*156*
			Mexico City: 11.6% of child diarrheal cases		Mexico City: 7.0% (children), 2.3% (adults)	F	*9*
			Netherlands: 5.0% of GI cases		Netherlands: 4.9%	F	*46*
			Israel: 16.0% of child diarrheal cases (Bedouin)		Israel: 23.0% of children (Bedouin)	F	*12*

TABLE 2. *Continued*

Pathogen	Incubation period (days unless otherwise noted)	Duration of illness (days unless otherwise noted)	Carriage rates reported	Postsymptomatic shedding (days unless otherwise noted)	% of individuals who excrete the pathogen but are asymptomatic	Clinical source of pathogen[a]	Reference(s)
G. lamblia (*G. intestinalis*) (cont'd)					San Francisco restaurant and hotels: 3.5%		*97*
					Melbourne: 1.6%	F	*87*
			Israel: 0.7–2.5% of military diarrheal cases			F	*35*
			U.S.: 3.8% of all age groups, 8–26% of children in day care, 1–3% of children not in day care. Developed countries: 29–54%				*65*
					Jerusalem: 37%	F	*94*
			Naples: 11.9% in immunocompetent children, 12.1% in immunodeficient children		Naples: 6.0% in immunocompetent children; 8.0% in immunodeficient children		*159*
					35% of children in day care	F	*151*
				5–41 in inoculated volunteers		F	*163*

[a] F, feces; U, urine; B, blood; N, nasal secretions; S, skin or skin lesions; T, throat; V, vomitus.
[b] GI, gastroenteritis.
[c] STEC, Shiga toxin–producing *E. coli*; VTEC, verotoxigenic *E. coli*.
[d] EAEC, enteroaggregative *E. coli*.
[e] EIEC, enteroinvasive *E. coli*.
[f] EPEC, enteropathogenic *E. coli*.
[g] ETEC, enterotoxigenic *E. coli*.

to worker outbreaks or person-to-person transmission, but new evidence indicates that some *V. parahaemolyticus* strains have infected persons with doses $\leq 10^3$ CFU *(68).* Potentially, therefore, infection with *V. parahaemolyticus* could result from worker errors in food service establishments because of the low dose required to cause serious illness in susceptible populations. However, there has been no record of this, possibly because this organism does not typically colonize humans, occurs in select marine environments, and is not a routine part of surveillance activities with prepared food items.

Toxigenic *V. cholerae* O1 and O139 serotypes are infective at a dose of 10^3 to 10^4 organisms; a non-O1 strain that does not produce cholera toxin but produces a heat-stable enterotoxin, NAG-ST *(155),* is able to colonize the intestinal tract but only at a much higher dose (10^6 CFU). Food workers have been implicated many times in cholera outbreaks *(53).* Examples include rice served at functions in African settings where cholera patients or victims have been present *(184),* cooked shellfish at a restaurant in Thailand *(72),* and cold meals served on airplanes during flights in the Middle East and Latin America *(187).* In each case, hygiene was poor and there were opportunities for pathogen growth in food. Although we lack any information on other vibrios implicated in food worker outbreaks, such outbreaks may occur, especially because variants of *V. cholerae* may be confused with other *Vibrio* species, such as *Vibrio mimicus* and *Vibrio alginolyticus (207).*

Much lower infective doses seem to be associated with *Campylobacter* (500 to 800 CFU; Table 1). Volunteer studies are not permitted for enteroinvasive *E. coli* (EIEC) and enterohemorrhagic *E. coli* (EHEC) because of disease severity, but it is assumed from outbreak data that few cells are required to cause illness (<100 or even <10 CFU for *E. coli* O157:H7 in some situations). However, similar types of pathogens such as *Shigella dysenteriae* and *Shigella flexneri* 2a have been given to volunteers to determine infective doses. The lowest dose to cause an infection for an antibiotic-resistant pandemic strain of *S. dysenteriae* was 200 CFU, the mean incubation period was 5.2 days, the presentation was clinical colitis, and at the height of excretion stools contained 10^6 to 10^{10} CFU/g *(114).* The lowest infectious dose was estimated at <500 CFU for *Shigella sonnei,* <140 CFU for *S. flexneri,* and <10 CFU for virulent strains of *S. dysenteriae,* but in some volunteer studies with *S. flexneri* the inocula were buffered to prevent any reduction of the dose by stomach acid *(107).* EIEC is assumed to have the same low infective dose as *S. dysenteriae,* e.g., <10 CFU *(198),* but this assumption is based on similarity of infective mechanisms rather than any direct observations.

We expected to find evidence of worker-associated outbreaks of *Campylobacter,* EIEC, EHEC, and shigellae infections. Many documented outbreaks of shigellae infection have involved food workers *(80, 194, 195).* However, *Campylobacter* and *E. coli* O157:H7, even with their low infective doses, have rarely been recorded as transmitted by food workers and thus leading to outbreaks. Greig et al. *(80)* found only five documented *Campylobacter* and three documented *E. coli* O157:H7 worker-associated outbreaks, despite the fact that *Campylobacter* is the leading cause of bacterial diarrhea in the United States, New Zealand, and other developed countries *(198).* In a 1997 study of 30,000 diarrheal stool samples, *E. coli* O157:H7 was the fourth most prevalent bacterial enteric pathogen, and person-to-person transmission of *E. coli* O157:H7 is not considered uncommon *(180).* There are many opportunities for food workers to be infected with these two organisms. The reasons for low worker involvement in outbreaks are not apparent, especially because *Campylobacter* can remain viable in stool specimens for ≥7 days after patient recovery *(99),* and *E. coli* O157:H7 can remain viable for ≥10 days *(160).* Perhaps the low rate of worker involvement in outbreaks is due to the fact that there are relatively few asymptomatic carriers in the community, although up to 13% of apparently healthy individuals in surveys have been found to excrete *Campylobacter jejuni* or *Campylobacter coli (99).* Healthy carriers of *E. coli* O157:H7 and O157:H- also have been documented *(11),* but few large population studies have been conducted to determine the carriage rates for healthy adults, which is assumed to be low. In a limited survey in northern Italy, verotoxigenic *E. coli* (VTEC) O157 was found in 4 (1.1%) of 350 farm workers on 276 dairy farms and in 50 abattoir employees *(177),* but not all VTEC strains necessarily cause disease *(83).* Secondary spread of *E. coli* O157:H7 is most likely after an outbreak, either as a foodborne or community infection *(19, 123, 150).*

Campylobacter frequently enters the food service environment via raw poultry but tends to die off rapidly, especially in dry, warm conditions and will not grow in many foods. EHEC survives better and can grow in most ready-to-eat (RTE) foods. However, although animals are the normal reservoir of *E. coli* O157:H7, the meat slaughtering and processing industry takes extensive precautions to prevent contamination of meat and poultry products. Thus, contamination is rare in raw foods of animal origin, although *E. coli* occasionally will enter the kitchen through ground beef. In a recent outbreak of *E. coli* infection in Belgium, ice cream was implicated, and the same *E. coli* strains (O145 and O26, both EHEC) were found in the fecal samples of patients and in ice cream from one of the birthday parties, in fecal samples taken from calves, and in samples of soiled straw from the farm at which the ice cream was produced *(45).* Researchers postulated that cross-contamination occurred through a worker who was involved in the production of the ice cream and had contact with the animals; he was not trained or properly instructed in hygienic issues. Except for their relative rarity, *E. coli* strains have characteristics similar to those of *Salmonella,* the second most frequently documented pathogen in food worker–associated outbreaks *(80),* and therefore, there is no obvious reason why *E. coli* O157 or other EHEC should not be implicated more frequently through worker errors in typical food service settings around the world. EPEC, ETEC, and EAEC require large numbers to cause diarrhea (10^6 to 10^8 CFU) and are not likely to be involved in worker transmission, but these pathogens are endemic only in developing countries, where outbreaks may not yet have been investigated and published.

In volunteer studies, some serovars of *Salmonella* have infectious doses that appear large (10^5 to 10^{10} CFU), but data from outbreaks suggest a lower range but with considerable variability ($<10^1$ to 10^9 CFU). Carmine dye in capsules containing 4.57 log CFU *Salmonella* Cubana had a 70.9% attack rate when given to susceptible patients *(58, 111),* and an outbreak of *Salmonella* Typhimurium infection had an attack rate of 55.0% when imitation ice cream was eaten with an average dose of 3.79 log CFU *(4, 58).* In contrast, outbreaks involving fatty products have had much lower contamination levels, e.g., chocolate bars with <10 CFU *Salmonella* Napoli per g *(78),* chocolate balls with 2.5 CFU *Salmonella* Eastbourne per g *(58),* both with unknown attack rates, and hamburger with 6 to 23 CFU *Salmonella* Newport per g and a low attack rate (1%) *(56, 58).* Other examples are shown in Table 1. Different *Salmonella* serotypes are widespread globally and occupy niches allowing them to reside in food processing and preparation environments awaiting adequate conditions for contamination and/or growth in food. Many of the documented outbreaks occurred with low doses because of the protective characteristics of the specific foods such as oils or fats in cheese, chocolate, ice cream, and egg-based foods; fats and oils coating the bacterial cells reduce the opportunities for the gastric fluid to inactivate the organisms. However, apart from egg-based mayonnaise salad items, these types of products have not been associated with outbreaks implicating infected food workers.

Multi-ingredient RTE items are much more likely to be associated with food worker errors because these items require more handling during preparation. A Japanese ministerial directive advises restaurants and caterers to freeze portions of both raw food and cooked dishes for at least 2 weeks so they can be examined if an outbreak occurs. Kasuga et al. *(100)* evaluated the analyses of 39 salmonellosis outbreaks associated with schools, hospitals, restaurants, caterers, and confectioneries where the food items had been preserved. Levels in foods ranged from 0.135 to 5.0×10^7 CFU/g, with doses estimated at 1.1×10^1 to 7.5×10^9 CFU. Food workers were not specifically mentioned as the cause of the outbreaks (implicated foods were mostly egg dishes contaminated with *Salmonella* Enteritidis), but some worker transmission probably occurred. Five separate outbreaks (with three serotypes) were listed as arising from contaminated grated yam diluted with soup. The doses for these infections ranged from 1.38×10^5 to 7.5×10^8 CFU, with attack rates of 21.9 to 100% (Table 2). Although these numbers seem to be very precise, there may have been an uneven distribution of the *Salmonella* in the foods (less likely in liquids), and die-off could have occurred during the storage time, which brings some uncertainty to the results. However, from these data we can propose scenarios where food workers infected with *Salmonella* could contaminate RTE foods with low levels of the pathogen, which would then increase after temperature abuse to higher concentrations, e.g., to 10^1 to 10^3 CFU/g or higher, which would be sufficient to cause an outbreak (Table 1) *(100).*

Workers may be long-term carriers of pathogens. Although chronic *Salmonella* Typhi carriers are rare (only 1 of 800 volunteers tested for many years chronically excreted the organism, and he had gall bladder disease *(90)*), they can excrete as many as 10^{11} CFU/g feces *(92)* and have been responsible for several outbreaks *(190, 209).* The issue of excretors of enteric bacterial pathogens has been further discussed by Cruickshank and Humphrey *(38),* but they focused more on transient carriers. They mentioned studies indicating *Salmonella* carrier rates of 0.2 to 5%; the most extensive study in Japan revealed rates of only 0.15%. These authors quoted estimates for carriers in the United States and United Kingdom to be 200,000 and 50,000, respectively.

S. aureus should not be associated with worker outbreaks when these outbreaks are mostly linked to low pathogen doses. An enterotoxin dose of ≤ 1.0 μg in contaminated food produces symptoms of staphylococcal intoxication, but this toxin level is typically reached only when *S. aureus* populations exceed 10^5 CFU/g *(198).* Occasionally levels are very high, as occurred in a large outbreak in Brazil where up to 3 mg probably was ingested *(48).* The many documented outbreaks associated with *S. aureus* reflect the high nasal carriage rate in the population *(55, 109).* This carriage, in turn, allows frequent contamination of the hands and arms and may result in heavily loaded infected skin lesions that act as foci for spreading the pathogen by hand contact during preparation and handling of RTE foods. Staphylococcal growth in food must occur for enterotoxin to be produced, and therefore sufficient time and temperature abuse are required.

The infectious dose for group A streptococci (beta-hemolytic *Streptococcus pyogenes*) is probably quite low ($<10^3$ CFU); for group D streptococci (*Streptococcus faecalis, Streptococcus faecium, Streptococcus durans, Streptococcus avium,* and *Streptococcus bovis*), it is probably $>10^7$ CFU *(198).* Group A streptococci have been implicated in outbreaks after workers have sneezed on foods, which were then improperly stored.

Y. enterocolitica has only occasionally been implicated in food worker–associated outbreaks; Greig et al. *(80)* listed seven episodes. This small number of outbreaks may be due to the high recorded infective dose (10^6 to 10^9 CFU) and the fact that the organism is more infective at lower temperatures, such as 22°C, rather than at 37°C *(165).* Although *Y. enterocolitica* is a ubiquitous microorganism, the majority of isolates recovered from asymptomatic carriers, food, and environmental samples are nonpathogenic *(61).* However, in a Washington State outbreak associated with tofu, two of the employees preparing the tofu were asymptomatically infected with the outbreak strain *(22).*

Infections with hepatitis A virus (HAV) occur globally, mainly through person-to-person spread, but outbreaks also have been associated with food and water contaminated by human sewage or by infected individuals. According to Greig et al. *(80),* HAV was the third most frequently reported pathogen in outbreaks associated with food workers, after norovirus and *Salmonella.* In the United States, the United Kingdom, northern Europe, and Japan, caliciviruses such as noroviruses and sapoviruses (e.g., Sapporo virus) are the most common cause of sporadic acute gastrointestinal illness in patients of all age groups except infants and toddlers, in whom rotaviruses predominate *(137)*. Norwalk agent, named after the location of the first documented outbreak in Norwalk, Ohio, was confirmed as an infectious agent through administration of fecal filtrates to volunteers by Dolin et al. *(49)*. Sapporo virus was first detected during a gastroenteritis outbreak in a home for infants in Sapporo, Japan in 1977 and now occurs globally. This virus plays an important role in outbreaks of infantile gastroenteritis and is less important in foodborne outbreaks, although such infections have been documented *(34)*. The lowest infectious doses for HAV and norovirus, both well established as pathogens transmitted by food workers, are unknown but estimated to be 10 to 100 virus particles *(198)*. However, not all those who ingest norovirus particles are affected. When volunteers were given dilutions of a stool filtrate, 82% became infected; of these infections, 68% resulted in illness and the remaining 32% were asymptomatic *(76)*. Lindesmith et al. *(120)* discovered that the infectious doses depended on the genetic makeup of volunteers; 50 to 62% of the volunteers developed diarrhea when they were given doses from $\leq 10^4$ to 10^8 and had an epithelial binding gene (Se+), whereas volunteers who did not have the gene (Se−) remained well with doses up to 10^8. This work was confirmed by Hutson et al. *(93)*, who found even higher rates of infection for those with the Se gene. Because persons infected with these viruses often excrete 10^5 to 10^{12} infectious particles per ml of diarrheal feces *(65)* and the infective dose is apparently very low, contamination of hands, food, and nonfood contact surfaces or utensils through fecal transfer or aerosolization of vomitus onto food or surfaces can easily lead to infection of workers and patrons in food service establishments. However, not all excreted particles are necessarily infectious *(206)*. As analytical methods improve for norovirus detection, more outbreaks will be identified as caused by this pathogen, and more persons will be identified as carrying norovirus in their stools. O'Neill et al. *(149)* developed a sensitive nested reverse transcriptase PCR method with a 10- to 1,000-fold increase in sensitivity over other PCR protocols and electron microscopic methods and found that a positive diagnosis could be made in 30 of 31 gastroenteritis outbreaks investigated. This improved diagnostic ability will allow researchers to separate cases of norovirus infection associated with outbreaks in hotels, restaurants, hospitals, and nursing homes from nonoutbreak cases of diarrhea such as those in elderly patients in hospital wards. For infected individuals in outbreaks, the positive diagnostic rate ranged from 11.5 to 100%.

The same situation applies to rotaviruses, also transmitted by the fecal-oral route, with the feces of an infected person containing 8 to 10×10^9 infectious particles per ml, where only 10 to 100 particles are required for transmission of infection *(2)*. Bishop *(13)* reported even higher numbers, with clinically infectious persons shedding $>10^{12}$ rotavirus particles per g or ml, and the virus appears to retain infectivity for many months. Although 14 foodborne rotavirus disease outbreaks have been documented *(172)*, these were in New York State in the 1980s and 1990s and involved cold foods, salads, shepherd's pie, strawberry shortcake, hamburger, brownies, and ice served in food service establishments and camps. Seven of these outbreaks were confirmed. Sattar et al. *(172)* suggested that the quality of surveillance in New York was the reason these outbreaks were detected, whereas other outbreaks in the United States may have occurred but were not recognized; at least some of these outbreaks may have been misdiagnosed as norovirus or multiple pathogen disease outbreaks. For instance, in an outbreak aboard a naval ship, six enteric viruses (three norovirus genotypes, a sapovirus, and a rotavirus) were isolated from those individuals who ate salad and developed diarrhea *(64)*. Another foodborne outbreak occurred in 2000 in the District of Columbia when college students were infected after eating tuna or chicken salad sandwiches; the cooks also were infected and may have been the source of the virus *(23)*. Outbreaks associated with school meals in Japan have been reported *(172)*. Mead et al. *(128)* stated that an estimated 39,000 foodborne rotavirus infection cases occur each year in the United States, more than nine times the number of estimated HAV infection cases *(4, 168)*. Because several food worker–associated HAV infection outbreaks have been noted in the literature *(80)*, it is surprising that there is no evidence for more rotavirus infection outbreaks initiated by workers in the United States. The lack of documentation for foodborne rotavirus infection outbreaks and sporadic cases is not easily explained, although diarrhea most frequently occurs in the winter months in young children who are not involved in food preparation. Adults tend to have immunity, but volunteer and epidemiological studies have revealed that adults can develop diarrhea when exposed, as in a college student outbreak *(23)*. Teachers and family members of sick children also can be infected, as illustrated by outbreaks in closed communities such as a kibbutz and schools *(63, 125)*.

A case-control study to determine risk factors for gastroenteritis attributable to norovirus, Sapporo-like virus (SLV), and rotavirus showed different risk factors for the three pathogens *(47)*. For norovirus gastroenteritis, having a household member with gastroenteritis, contact with a person with gastroenteritis outside the household, and poor food-handling hygiene were associated with illness (17, 56, and 47% risk, respectively). For SLV gastroenteritis, contact with a person with gastroenteritis outside the household was associated with a higher risk of illness (60%). For rotavirus gastroenteritis, contact with a person with gastroenteritis outside the household and poor food-handling hygiene were associated with a higher risk of illness (86 and 46%, respectively). The authors concluded that transmission of these viral pathogens occurs primarily person to person, and for norovirus gastroenteritis, foodborne transmission seems to play an important role. Unlike rotavirus and SLV gastroenteritis, norovirus gastroenteritis is not limited to the youngest age groups, and de Wit et al. *(47)* stated that this lack of specificity could explain why hygiene during food preparation and having a household gastroenteritis contact had a higher impact on norovirus gastroenteritis than on SLV gastroenteritis. These authors also considered that undetected asymptomatic rotavirus and SLV infections may occur at older ages through these routes. Elderly individuals are prone to SLV infections for reasons similar to those that account for such infections in infants *(112)*.

Astroviruses, which are less well studied than norovirus and rotaviruses, have been associated with outbreaks of acute gastrointestinal illness, mainly by person-to-person spread, in daycare centers, military bases, maternal-care facilities, and hospital wards *(137)*. Glass et al. *(70)* reviewed eight surveys of children's stool specimens for astrovirus, which was detected by enzyme immunoassay, and the prevalence ranged from 2.5 to 10% for those children with diarrhea compared with 0.7 to 2.4% for those without diarrhea (controls). Because enzyme immunoassays are less sensitive than other methodologies such as real-time PCR, the actual prevalence of infection is probably much higher. In one study in a day-care center, all the children carried the virus and remained excretors for many days, even weeks, although they were asymptomatic. Children and immunocompromised individuals are most likely to be infected, and astrovirus can be transmitted to adult family members via sick children. Outbreaks have been associated with consumption of oysters, food supplied by schools, and drinking water; however, <1% of astrovirus infections are considered foodborne *(70)*. Adenovirus types 40 and 41 and astroviruses also have been implicated in gastroenteritis, but there are no known outbreaks associated with food workers infected with SLV, adenoviruses, or astroviruses.

Clinical infections and outbreaks of hepatitis E have been recorded predominantly in countries where the disease is endemic, including eastern and central Asia, Mexico, and parts of Africa *(208)*. Hepatitis E virus (HEV) is the most common cause of acute hepatitis in adults in parts of Asia and Africa, where large outbreaks have been associated with sewage-contaminated drinking water. Wild and domestic animals may serve as a reservoir for HEV in endemic areas, causing human infection from water sources polluted by animal wastes. HEV can be detected 2 weeks before the onset of liver enzyme elevations, and shedding ends when the enzyme level returns to normal, about a month later *(37)*. If food worker–associated outbreaks of HEV infection were to occur, they would be in endemic regions, but no such outbreaks have been documented.

The protozoan parasites *Cryptosporidium parvum, Cyclospora cayetanensis,* and *Giardia lamblia* (=*Giardia intestinalis*) all have very low infectious doses and have been implicated in food worker–associated outbreaks, although infrequently, at least in published reports. Persons at increased risk for infection include those who (i) contact infected wild and domestic animals, (ii) ingest contaminated recreational (e.g., lake, river, pool, or hot tub) or drinking water, (iii) have with close contacts with infected persons (e.g., in the same family or household or in day-care settings), or (iv) are infected with the human immunodeficiency virus *(28, 151)*. *Cyclospora* was isolated from 0.1 to 0.5% of stools in clinical laboratories in the United States and England, respectively, mainly from people who had recently traveled abroad *(151)*. Giardiasis can be contracted from drinking recreational water contaminated either by sewage or by wild animals, but as for most protozoan parasites, person-to-person spread is also well documented *(79)*. In prisoner volunteer studies, one cyst did not infect two healthy adults but 10 cysts did infect two additional persons, as did higher numbers of cysts *(163)*. Diapering of infected infants led to one outbreak of giardiasis in a home and another in a day-care center *(194)*. Transmission of various protozoan parasites is feasible via the fecal-oral route because persons infected with *Cryptosporidium* have been reported to shed 10^8 to 10^9 oocysts in a single bowel movement, whereas those with giardiasis can shed $\leq 10^9$ cysts daily in stools *(27)*. Therefore, risk factors for food workers include travel to regions where these organisms are endemic, contact with diarrheic children, and ingestion of contaminated water and food. Other protozoan parasites with the potential to infect food workers are *Balantidium coli, Dientamoeba fragilis, Entamoeba histolytica, Isospora belli,* and any of the >1,000 species of Microsporidia, but it remains to be seen whether there will be any documented foodborne outbreaks arising from these organisms through contamination of the food supply by infected staff.

In Table 1, we categorized the likelihood of each of the pathogens listed in published reports as implicated in food worker–associated outbreaks. For those pathogens for which outbreak information is available *(80, 194, 195)*, we listed such outbreaks as frequent, occasional, or rare and whether they seem to be declining or increasing. For those where we suspect there are situations where they could occur, especially in developing countries where reports are less frequently published, we are more vague in our description, e.g., not known but probable, likely occasional in endemic areas in developing countries, unknown but maybe in developing countries, and rare in developed countries but more frequent in developing countries. Over time and with more investigative reports, we may see these outbreak frequencies defined more specifically. However, this information provides managers of food operations and local food inspectors ideas about the most likely locations of concern for pathogens being transmitted through food workers.

CARRIAGE AND SHEDDING OF PATHOGENS IN ILL AND ASYMPTOMATIC INDIVIDUALS

Table 2 lists the incubation period, duration of illness, carriage rates, length of postsymptomatic shedding, and rate of pathogen excretion by asymptomatic individuals for many enteric pathogens. Incubation periods range from hours (e.g., *S. aureus*) to many weeks (e.g., HAV). The longer the incubation period, the more likely infected persons will excrete the pathogen. Also, the longer a food worker or family member has gastrointestinal symptoms, the more opportunities exist for fecal contamination in a food preparation setting. When paid sick leave is limited or not available, food handlers may work while ill without reporting their condition to management or may deny mild symptoms such as loose stools *(195)*. Gastroenteritis symptoms may last many days or even weeks or months, as in cases of infection with *Salmonella* Typhi, *Shigella* spp., HAV, HEV, and the protozoan parasites.

Postsymptomatic shedding may be of long duration for

Campylobacter, Salmonella, Shigella, V. cholerae, Yersinia, the enteric viruses and parasites. In a given population, the pathogen carriage rates for those with diarrhea can range from <1 to >70%, with certain populations more at risk (typically young children and those living in tropical developing countries) than others. However, some of the long carriage periods may be due to reinfection in endemic areas *(143).* Chalker and Blaser *(31)* reviewed the intestinal carriage of nontyphoidal *Salmonella* in healthy populations, which ranged from 0.0 to 1.35% (mean, 0.27%) in developed countries and 0.0 to 6.5% (mean, 1.5%) in developing countries. However, the rate of carriage in food workers was higher with a mean of 1.7% in developed countries (up to 18.7% in one study) and a mean of 6.2% in developing countries (up to 10.3% in another study). These authors also estimated that the average duration of *Salmonella* excretion in convalescing patients was about 5 weeks. This estimate was based on the work of Buchwald and Blaser *(21),* who found that >50% of convalescent patients stopped excreting in less than 5 weeks, but a few continued to excrete for up to 12 months (both convalescing and apparently asymptomatic persons). Of 151,452 workers studied in public service and food production in Shenzhen, Guangdong, China, in 2003, 455 (0.30%) were positive for *Salmonella* and 210 (0.14%) were positive for *Shigella* spp. *(118).* These rates were lower for both pathogens than the rates obtained in a study conducted in 1998 (0.49 and 0.37%, respectively), indicating improvement in hygienic practices in the intervening years. Individuals may become infected with *E. histolytica* when hand washing is not properly practiced during food service operations, but outbreaks of disease associated with this protozoan have not been recorded.

In specific studies, pathogens with the highest prevalences in diarrheic populations were *Campylobacter* (31%) in South African children, ETEC (22%) in Korean children, EPEC (20.7%) in Mumbai, *V. cholerae* (33%) in Mumbai, norovirus (16.1%) in individuals with gastrointestinal disease in The Netherlands, and *Entamoeba* (70.3%) and *Giardia* (up to 33.3%) in Mexican children (Table 2). In gastrointestinal outbreak settings, the average patient-positive rate for norovirus was 34% (221 of 647) when patients were tested by a nested reverse transcriptase PCR method compared with 0.4% (2 of 532) control fecal specimens from non-outbreak hospitalized patients *(149).* However, these two control patients were repeatedly positive. In a large survey of infectious intestinal disease cases in England, Tompkins et al. *(196)* found that 6.5 to 7.0% of cases and 0.1 to 0.5% of controls had Norwalk-like viruses in their stools, as determined by electron microscopy. As methodology improved, so did the prevalence rate. In The Netherlands, de Wit et al. *(46)* found that norovirus was detected by reverse transcriptase PCR in 16.1% of individuals with gastrointestinal disease and in 5.2% of healthy individuals. Svraka et al. *(188)* reviewed the etiological role of viruses in 941 gastroenteritis outbreaks in The Netherlands from 1994 to 2005. Noroviruses were detected as the causative agent in 735 (78.1%) of the outbreaks, and rotaviruses, adenoviruses, and astroviruses were responsible for 46 (4.9%), 9 (1.0%), and 5 (0.5%) of the outbreaks, respectively. Foodborne transmission was associated with 6.6% of the norovirus infection outbreaks and occurred in food service settings in >8.2% of these outbreaks.

In Calcutta, 37.3% of those individuals with diarrhea had typhoid fever symptoms, and *Shigella* spp. were found in 52% of cases in Bangkok in which the affected individual presented with mucoid or bloody diarrhea (Table 2). In contrast, the prevalence of *E. coli* O157:H7 and *Y. enterocolitica* in gastrointestinal cases is typically <1 and 1 to 2%, respectively. However, in England in a very large case-control study, the low prevalence of *Yersinia* spp. was equal in healthy and ill persons *(196),* but this information may be incomplete because many *Yersinia* strains are nonpathogenic. VTEC and Shiga toxin–producing *E. coli* has been found in 8.6% of children with diarrhea in Mexico City and 6% of individuals with diarrhea (50% of the affected children) in Seoul. A very large range in rotavirus prevalence was found in gastrointestinal cases, from 0.2% in England to 82.4% (children) in Melbourne. Military personnel in Israel experienced gastrointestinal discomfort caused by several types of infections, but many soldiers remained on duty; 15 to 32% of the soldiers were infected with ETEC, up to 20% were infected with *Shigella,* and 16% were infected with *Giardia (35). Cryptosporidium* infection in Iran is related to the health status of the individual and ranges from 4 to 7% for healthy persons to 50% for those who were immunodeficient. Postsymptomatic shedding can last for many days or weeks for *Campylobacter, E. coli* O157, *Salmonella, Shigella, Yersinia,* HAV, *Cryptosporidium, Entamoeba,* and *Giardia,* and shedding lasts the longest in children *(175).* Protozoan parasites remain viable in the bowel for extended periods; after cessation of diarrhea, *Cryptosporidium* oocysts can be excreted for up to 50 days, and *Giardia* oocysts can be excreted even longer *(27). Salmonella* may be present equally in diarrheal and apparently healthy persons, and people with close contact with index cases can have a high carriage rate without any symptoms *(96),* all of which is a concern for food worker carriage and transmission. Apparently recovered individuals with nontyphoid *Salmonella* enterocolitis may relapse 3 weeks later, depending on disruption of antibiotic regimes or inappropriate treatment *(144).* Some lengthy carriage times for pathogens such as *V. cholerae* may be attributable to reinfection in endemic regions *(200),* perpetuating a low level of infection in a community. Johnstone and Iverson *(97)* reported carriage rates for *E. histolytica* in food workers ranging from 1.2 to 15% in the United States. This rate was even higher in Leningrad (now St. Petersburg, Russia), with a rate of >22%. Travel abroad and familial spread in households were at least some of the sources of infection for food workers. However, these studies were conducted many decades ago, and the rates are unlikely to be as high today in U.S. food establishments. However, these results can be used as indications of levels likely to be encountered in regions with poor hygiene.

Asymptomatic cases may originate during outbreaks, as demonstrated during an *E. coli* O157 infection episode where 14% of the individuals excreted the organism for up to 39 days after cessation of symptoms and during a yersiniosis outbreak in a Belgium day-care center in which 81% of the children were asymptomatic (Table 2). Most of the high rates were in young children under poor hygienic conditions, but high rates also were found in closed communities such as military bases. The risk of foodborne contamination is less where certain enteric pathogens are more prevalent in infants and young children and adults develop immunity. However, in some situations children have infected parents who in turn contaminated food. Of particular concern is *Salmonella,* which was isolated from the stools of 16% of food workers with diarrhea in Kenya and 10% of asymptomatic food workers in Thailand *(153, 179).* In the United States, Buchwald and Blaser *(21)* estimated that 200,000 individuals may be excreting *Salmonella* at any one time and many of these excretors would be food workers.

S. aureus and beta-hemolytic streptococci are unique among the pathogens transmitted by food workers because they can asymptomatically colonize the nasopharynx and throat for extended periods or indefinitely in high concentrations and can be regularly transferred to hands and arms though hand-to-face contact. Wounds on the skin also may be infected, providing a supplemental source of contamination. In a review of 131 staphylococcal disease outbreaks in the United States between 1977 and 1981, Holmberg and Blake *(89)* found that 67% of the apparently healthy food workers harbored the same phage type of *Staphylococcus* as found in the implicated food. Katzenell et al. *(101)* reviewed 18 outbreaks, and 15 of them had links to infected food handlers or their children who had pharangitis. Most of the foods were RTE cold items, including cabbage, chicken, egg, potato and tuna salads, mousse with cream, and rice souse.

In a Chilean study, 34% of 102 food workers from 19 restaurants in Santiago were colonized with *S. aureus,* and 54% of the strains were enterotoxigenic, mostly producing type A toxin *(55)*; 19 of the 102 workers had the potential to contaminate RTE food and cause patron illnesses, especially in facilities where up to 75% of the workers were colonized. These carriage rates are very similar to those in a U.S. national survey conducted in 2001 and 2002 *(109)* (Table 2). Other pathogens with relatively high asymptomatic fecal carriage rates include *V. cholerae* (4% in those exposed to high risk activities), norovirus (5.2% in the general population in The Netherlands), rotavirus (>20% in Mexico City [both children and adults] and up to 27% of neonates in Paris), *Cryptosporidium* (up to 76% of persons in Peru and Venezuela), *Cyclospora* (up to 94% of the population in Peru and Haiti), *Giardia* (23% of Bedouin children in Israel), and *Entamoeba* and *Giardia* (43.7 and 20.0% of children in Mexico City, respectively). All these pathogens demonstrate the potential for person-to-person and person-to-environment transmission where food and infected food workers may play a role during an outbreak.

Healthy individuals may be continually exposed to infected workers in food production and preparation operations because these workers may stay at work over many days. Thomas et al. *(192)* found that employees who worked in what was termed high-risk settings in both rural and urban settings in British Columbia (where workers' duties increase the likelihood of transmission of gastrointestinal disease to others) were 1.4 times more likely to discontinue working while ill (7 of 14 food workers, 0 of 2 day-care workers, and 5 of 22 health care workers) than were ill employees (92 of 185) in perceived low-risk settings. The period off work ranged from 0.5 to 12 days, with a median of 1 day. However, 50% of workers in this study did not take time off, including two workers who returned to work while still experiencing symptoms. Reasons given for staying at work were health condition not serious enough to stay home, unable to afford to take time off work, and employer depends on them or there was no one who could cover for them. These findings agree with the conclusions of Aronsson et al. *(5),* who found that one-third of the persons in a Swedish survey reported that they had gone to work two or more times during the preceding year despite thinking they should have taken sick leave. The areas where people reported to work most consistently when ill were in the care and education sectors (nursing and midwifery professionals, registered nurses, nursing home aides, compulsory school teachers, and preschool or primary educators), which had faced personnel cutbacks at the time. If employees return to work too soon or continue to work while ill, other employees and patrons are at a greater risk of illness, as illustrated by an outbreak of viral gastroenteritis in a nursing home *(168).*

Stewart et al. *(182, 183)* found that sickness presenteeism accounted for four times more lost productive time than absenteeism. Presenteeism is defined as lost productivity that occurs when employees come to work but perform below the normal output because of some kind of illness. When workers come to work sick, they perform below par and can infect others, which can contribute to further absenteeism and/or presenteeism *(117).* Although the costs associated with employee absenteeism have long been studied, the costs of presenteeism have yet to be fully evaluated. Levin-Epstein *(117)* noted that nearly 40% of employers surveyed in 2004 reported that presenteeism was a problem in their organizations. In other studies, researchers determined that paid sick leave policies reduced the rate of contagious infections in the workplace by isolating sick workers at home, failure to take time off to regain one's health actually led to longer absences because health worsened, and as an illness spread within the workplace additional workers were affected, raising the total employee absence time. These general workforce conclusions are particularly applicable to the food industry, where patrons and fellow workers are exposed to pathogens and paid sick leave is a rarity for employees.

CONCLUSION

The lowest pathogen doses causing infection in food workers and others varies by organism and can be strain dependent. Other factors affecting the infectious dose are the effectiveness of the stomach acid barrier, which may be compromised with certain preexisting medical conditions and acid lowering medicines, and the protective effects of certain types of foods (e.g., those with high fat content).

Data from volunteer studies and outbreak investigations often are inconsistent (Table 1). Viral pathogens, predominantly norovirus, caused 33% of outbreaks and 41% of cases of foodborne disease in the United States from 1998 to 2002 *(66).* Mead et al. *(128)* estimated that annually in the United States 9,200,000 norovirus, 3,900 rotavirus, 3,900 astrovirus, and 4,170 HAV infections occur as a result of consumption of contaminated food. These outbreaks continue to occur, as illustrated by the fact that approximately 1,500 restaurant patrons became ill after dining at three restaurants in 2006 in Syracuse, New York, Lansing, Michigan, and Indianapolis, Indiana *(29, 77, 169, 170).* In a Michigan outbreak, at least 364 people became ill with gastroenteritis after dining at a restaurant where employees had reported to work while ill. Also in 2006, Michigan health authorities received 144 reports of suspected or confirmed norovirus outbreaks, compared with 34 in 2005 *(29).* The strain of circulating norovirus in an area may be a major determinant of outbreak occurrence. Of the five genogroups of norovirus, genogroup II is the predominant human strain in most communities *(33, 119, 157).* The DNA viral load of genogroup II is >100-fold higher than that of genogroup I in fecal specimens of patients with norovirus-associated gastroenteritis *(33).* This increased cDNA (a double-stranded DNA version of an mRNA molecule) viral load may account for the higher transmissibility of genogroup II strains through the fecal-oral route. In 2006 in Michigan, norovirus genogroup II was identified in 97% of the 89 confirmed outbreaks, and genogroup I was identified in the remaining 3% of the outbreaks. From 2000 to 2004, the predominant genogroup found in calicivirus outbreaks in the United States was genogroup II (79%) followed by genogroup I (19%) and sapovirus (2%) *(15).*

Although norovirus outbreaks are increasing, the opposite is true for streptococcal and staphylococcal episodes, which are less frequently reported currently than in previous decades *(80, 194, 195).* We are not aware of data demonstrating a decrease in the carriage level of these two pathogens in food workers *(109),* so the risk of food contamination remains. However, it is unclear whether the fewer reported outbreaks are due to better hygienic practices, better refrigeration of food, less effort directed toward identifying these pathogens during outbreaks, or a combination of factors. Although there may be reduced testing for these two pathogens, it is more likely there are fewer complaints because of the short incubation times during local outbreaks, which can occur at wedding receptions or family gatherings. The most logical explanation is that better attention to personal hygiene among food workers reduces contamination as does the use of gloves or other utensils and the installation of sneeze guards and other barriers to contamination. One of the successes in reduction of outbreaks is the improvement in proper cooling procedures limiting pathogen growth in RTE food, especially foods such as potato salad or sliced ham or roast beef, where bare hand contact may have occurred. Cooling procedures are now explicitly targeted in many jurisdictions. Recently, state and federal food regulations *(197)* give specific instructions for rapidly cooling potentially hazardous food susceptible to temperature abuse (placing food in shallow pans, stirring food in a container on an ice bath, inserting ice sticks into the food, adding ice as an ingredient, using rapid cooling equipment, allowing loosely covered or uncovered foods in pans in a cooler if protected from overhead contamination, and use of prechilled ingredients).

Communication between food service managers and employees is vital. Information about the risks of pathogen infection and transmission must be provided to all employees *(197).* If a worker displays symptoms of an enteric infection, the manager should be informed and allowed to make a decision concerning when the employee should be sent home and when the employee should return to work. Unfortunately, only about 15% of food service workers have paid sick days, and the risk of excreting norovirus particles extends to weeks after symptoms cease *(62).* Indeed, the postsymptomatic excretion time for many enteric pathogens can be long, which poses a problem for managers and health authorities as they attempt to set reasonable policies. Because a large proportion of infected workers may be asymptomatic but still have millions of infectious organisms in their stools, the only solution is proper hygiene and creation of barriers against pathogen transmission to foods. Although hand washing does not eradicate the risk of transmission of illness to fellow workers and patrons, it will reduce the number of viral particles or bacterial cells on hands and in the food preparation environment, whether the infected workers go home or stay at work. The sources of pathogen contamination and how pathogens are excreted from infected persons is the topic of the next article in this series of food worker–associated outbreaks and their prevention.

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