Journal of Food Protection, Vol. 71, No. 12, 2008, Pages 2582–2595

Review

Outbreaks Where Food Workers Have Been Implicated in the Spread of Foodborne Disease. Part 5. Sources of Contamination and Pathogen Excretion from Infected Persons

EWEN C. D. TODD,[1]* JUDY D. GREIG,[2] CHARLES A. BARTLESON,[3] AND BARRY S. MICHAELS[4]

[1]*Department of Advertising Public Relations and Retailing, Michigan State University, East Lansing, Michigan 48824, USA;* [2]*Public Health Agency of Canada, Laboratory for Foodborne Zoonoses, 160 Research Lane, Unit 206, Guelph, Ontario, Canada N1G 5B2;* [3]*Bartleson Food Safety Consultants, P.O. Box 11983, Olympia, Washington 98508-1983, USA; and* [4]*The B. Michaels Group, Inc., 487 West River Road, Palatka, Florida 32177, USA*

MS 08-295: Received 17 June 2008/Accepted 18 July 2008

ABSTRACT

In this article, the fifth in a series reviewing the role of food workers in foodborne outbreaks, background information on the routes of infection for food workers is considered. Contamination most frequently occurs via the fecal-oral route, when pathogens are present in the feces of ill, convalescent, or otherwise colonized persons. It is difficult for managers of food operations to identify food workers who may be excreting pathogens, even when these workers report their illnesses, because workers can shed pathogens during the prodrome phase of illness or can be long-term excretors or asymptomatic carriers. Some convalescing individuals excreted *Salmonella* for 102 days. Exclusion policies based on stool testing have been evaluated but currently are not considered effective for reducing the risk of enteric disease. A worker may exhibit obvious signs of illness, such as vomiting, but even if the ill worker immediately leaves the work environment, residual vomitus can contaminate food, contact surfaces, and fellow workers unless the clean-up process is meticulous. Skin infections and nasopharyngeal or oropharyngeal staphylococcal or streptococcal secretions also have been linked frequently to worker-associated outbreaks. Dermatitis, rashes, and painful hand lesions may cause workers to reduce or avoid hand washing. Regardless of the origin of the contamination, pathogens are most likely to be transmitted through the hands touching a variety of surfaces, highlighting the need for effective hand hygiene and the use of barriers throughout the work shift.

In this article, the fifth in a series on food worker–associated outbreaks, the ways that pathogens can enter the food environment are discussed. In previous articles, the different outbreaks, the factors contributing to them, and the infective doses and carriage of the implicated pathogens have been described *(46, 119–121).* Pathogens that can infect food workers have multiple sources, and infected workers in turn become potential sources of contamination in food processing and preparation facilities. The pathogens then can become a part of the transient or resident flora. According to Snyder *(116),* transient pathogen sources include (i) fecal contamination on hands that remains after using the toilet, changing diapers, or cleaning up after pets; (ii) raw products (e.g., meat, poultry, fish, or unwashed fruits and vegetables); and (iii) infected cuts and boils that are touched or picked, or an infected fingernail. Transient pathogens are excreted in feces and various body fluids or tissues by persons infected or colonized by these pathogens. When diarrhea results in many liquid stools per day, hands easily become contaminated because billions of pathogen cells are present *(6).* However, many colonized individuals may be asymptomatic, or symptoms may be so mild that they are considered part of a normal digestive upset. Although usually there are fewer pathogen cells per gram of solid stool than of liquid stool, the colonized individuals may erroneously think they are not a threat during food preparation and may become careless in their hygiene habits, allowing fecal contamination of food and the environment, including food contact surfaces. For viral pathogens, especially noroviruses and rotaviruses, vomitus can contain a concentration of particles similar to or greater than that in liquid diarrhea *(24),* and transmission and subsequent illness has resulted from cleanup procedures. Urine has occasionally been a means of pathogen transmission, typically only for organisms that invade the organs and blood supply, such as hepatitis A virus (HAV).

Resident sources of pathogens are less common and include permanent inhabitants on the epidermal skin layer, often protected in cracks and crevices where they cannot be removed easily through normal washing procedures. Although most microflora do not cause foodborne illness, *Staphylococcus aureus* can be a resident skin pathogen, multiplying in moist, warm areas such as the groin and frequently residing in the nasopharynx, from where it contaminates the skin on a regular basis through hand contact. This organism can be released though perspiration, aerosols from sneezing, and saliva onto food or food contact surfaces, cutlery, etc. Food workers also frequently develop

* Author for correspondence. Tel: 517-355-8371; Fax: 517-432-2589; E-mail: todde@msu.edu.

small cuts or burns in food preparation settings, and these wounds can become infected. Infected areas typically contain millions of *S. aureus* cells or group A streptococci, which can cause heavy contamination of foods handled by these workers or can cause impetigo. Staphylococcal nasal carriage seems to predispose people, particularly children, to the development of impetigo *(71).* Long-term or permanent carriers of pathogens such as *Salmonella* Typhi and some parasites also may be considered resident pathogen sources because shedding can occur on an intermittent basis over months or years. Injuries and infections reduce the inclination of workers to wash and dry their hands thoroughly and frequently. Quantitative data, where available, accentuate the importance of potential pathogen transmission though contaminated excretions and are discussed in detail in the following sections.

FECES AS A SOURCE OF CONTAMINATION FROM INFECTED AND COLONIZED WORKERS

Fecal sources from ill persons. Digested food is mainly liquid before reaching the colon, where water and nutrients are absorbed, leaving a semisolid stool. If the colon is damaged by enteric infections or toxins, the absorption of water is less efficient, resulting in watery stools with a moisture content of 50 to 90%. Bouts of diarrhea make it more likely that moist feces will penetrate toilet paper, contaminating hands and making effective cleanup more difficult. Levels of various pathogens in stool are listed in Table 1. *Salmonella* at up to 10^7 CFU/g can be found in feces of ill or early convalescent persons, who continue excreting for days or even weeks after the illness has resolved *(126)*; infants can excrete this organism for much longer periods (6 to 8 months). Up to 10^9 HAV particles per ml or g of feces can occur, with concentrations highest before symptoms are apparent *(95).* Norovirus levels have ranged from 10^4 to 10^{10} DNA copies per g of feces from symptomatic patients *(20).* However, shedding can occur before the main symptoms are apparent, as demonstrated by Graham et al. *(44)* in dosed volunteers. Ozawa et al. *(98)* analyzed data from 55 norovirus outbreaks and 35 sporadic cases in Japan and found that genotype 4 (GII/4) strains were predominant and occurred with the highest viral load of all genotypes (mean values, 7.96×10^9 copies per g of stool). These authors also found that a large number of symptomatic food workers were infected with this strain. However, symptomatic and asymptomatic individuals for all genotypes had about the same viral load, 10^7 to 10^8 copies per g of stool, which confirms the importance of transmission by individuals who are infected but not ill. The magnitude of seroconversion was highest in individuals who vomited. Ward et al. *(128, 129)* found that up to 10^{11} viral particles were excreted from persons infected with rotavirus but only 10^6 to 10^7 particles were infectious, as demonstrated by cell culture. The concentration of protozoan parasites *Giardia* or *Cryptosporidium* in feces of infected persons can range from 10^5 to 10^7 cells per g; numbers are even higher for HAV (10^8 particles) and rotavirus (10^7 to 10^{12} particles) *(41).* Because 100 to 200 g of feces are produced per individual per day, Gerba *(41)* determined that there could be as many as 10^{14} enteric pathogen cells in a single bowel movement of 100 g from an infected person. Because pathogens can be present in feces at levels of 10^4 to 10^{11} units per g *(6),* 0.1 mg of fecal material on the skin, which is barely noticeable, might contain up to 10^6 infectious bacterial cells, parasitic oocysts, or viral particles under diarrheic conditions. Gibson et al. *(42)* stated that the average American excretes 100 to 500 g of feces each day, and 0.1 g of residual fecal material remains on undergarments on a regular basis. This continual daily source of fecal contamination must be removed or excluded before workers touch food or food contact surfaces.

Fecal contamination of fingers and fingernails occurs more often in individuals with diarrhea, and Lin et al. *(76),* using ground beef or artificial feces as fecal matter surrogates, found that decontamination of the fingernail region was very difficult. Hand washing and alcohol gel had little effect, but use of a fingernail brush provided appreciable reductions. With $10^{-4.7}$ g of fecal contamination per hand and a pathogen load of 10^9 to 10^{11} units per g, as under diarrheal conditions, a 2- to 3-log reduction by washing and sanitizing would be insufficient to prevent a food worker from transmitting the pathogen to surfaces. Although fewer organisms probably are excreted when workers are asymptomatic or recovering from infection, any laxness in hand hygiene, such as not cleaning nails or only rinsing the hands, could lead to contamination of the food environment.

Sattar et al. *(112)* found that environmental surfaces remained contaminated with norovirus for 7 days after institutional outbreaks. Transfer of enteric bacteria from well individuals is illustrated by a study of postdefecation cleaning practices during which left and right hand fecal coliform counts were assessed for 90 individuals in Bangladesh *(55).* No toilet paper was used for cleaning; instead, traditional usage of the left hand and a small amount of water was used. Fecal coliform counts of the left hand averaged 3.93 log CFU, and those for the right hand counts averaged 2.99 log CFU. This degree of contamination on hands is related to the sanitation of latrines, which was critical for child survival *(54).* The authors speculated that the sitting area provided an opportunity for pathogens to transfer to hands and then into the mouth and was associated with the lack of water to clean the latrines. In rural communities in northern India, *Campylobacter* infection was significantly higher in persons who did not wash their hands with soap after perianal cleansing following defecation (10.2 versus 4.3%) *(59).*

In Santiago, Chile, where typhoid fever is endemic, family members of ill children belonging to a lower socioeconomic group were more likely to be carriers of bacterial enteric pathogens, such as enteropathogenic *Escherichia coli, Shigella* spp., *Salmonella,* and *Campylobacter* (13.8%), than were those individuals in a higher socioeconomic group (8.3%) *(3).* In homes with frequent carriers of these pathogens, unhygienic habits such as poor toilet care, inadequate hand washing, and children eating unwrapped candies were noted. The women who prepared the meals often were the carriers, and 39.5% of individuals who

TABLE 1. *Levels of pathogens in clinical specimens and body excretions*

Pathogen	Source of contamination	Contamination levels (per g or ml)	Reference
Salmonella	Feces while ill or during early convalescence	10^5–10^7 CFU	*126*
	Feces in late excretion period (infants excrete longer than do adults)	10^0–10^3 CFU	
	Feces during convalescence	6×10^3 CFU 15 days after illness	*85*
		5×10^2 to 6×10^7 CFU (median, 6.0×10^6 CFU) <10 days after illness; 1.3×10^2 to 1.6×10^9 CFU (median, 1.0×10^5 CFU) 10–19 days after illness; 0×10^6 to 3.5×10^6 CFU (median, 2.5×10^4 CFU) 20–25 days after illness; 7.0×10^1 to 1.8×10^5 CFU (median, 1.4×10^2 CFU) 6–35 days after illness; 2.0×10^0 to 3.5×10^4 CFU (median, 5.5×10^3 CFU) 42–50 days after illness; 0×10^4 to 6×10^4 CFU (median, 2.5×10^4 CFU) 69–102 days after illness	*103*
	Pus in infected lesions	10^7–10^8 CFU (median) for intra-abdominal and anorectal and soft tissue infections (one sample with almost exclusively *S. aureus* and two with beta hemolytic streptococci)	*70*
S. aureus and *Streptococcus pyogenes*	Saliva in a sneeze from carriers	Typical person infected with streptococci: saliva, 10^0–10^6 CFU; <100 CFU/154 cm^2 1.5–9.5 ft (0.5–2.9 m) from sneeze source. One carrier sneezed twice (days 1 and 6): saliva, 3.2×10^6 to 7.5×10^6 CFU; 23–500 CFU/154 cm^2 1.5–9.5 ft from sneeze source	*48*
S. pyogenes	Saliva in a cough from carriers	10^3–10^6 CFU (1 of 20 persons infected with streptococci coughed 6 CFU/154 cm^2 9.5 ft [2.9 m] from cough source; most of the other 19 persons did not cough any streptococci)	*48*
Enteroviruses (e.g., coxsackie virus, echovirus, poliovirus)		10^3–$10^{7.5}$ infectious particles; $10^{8.2}$ infectious particles	*41*
Hepatitis A virus	Feces, highest numbers before symptoms begin	10^9 virions	*95*
		10^8 infectious particles	*41*
Norovirus Group G-I	Feces while ill	2.2×10^4 to 2.9×10^{10} copies/g of feces; median, 8.4×10^5 copies/g	*20*
Norovirus Group G-II		2.5×10^4 to 7.7×10^{10} copies/g of feces; median, 3.0×10^8 copies/g	*20*
Norovirus Group G-I		GI 2.79×10^7 copies/g of feces	*98*
Norovirus Group G-I/4		GI/4 2.02×10^8 copies/g of feces	*98*
Norovirus Group G-II		GII, 3.81×10^8 copies/g of feces	*98*
Norovirus Group G-II/4		GII/4 7.96×10^9 copies/g of feces	*98*
Rotavirus	Feces and vomitus while ill	10^{11} particles excreted but only 10^6–10^7 infectious	*129*
		100 times more virus in vomitus than in feces	*24*
		8×10^9 to 10×10^9 infectious particles	*4*
		$>10^{12}$ infectious particles	*8*
		10^{10}–10^{12} infectious particles in feces	*41*
Cryptosporidium spp.		10^8–10^9 oocysts in a single bowel movement	*16*
		10^6–10^7 oocysts; 3×10^9 oocysts/day	*41*
Giardia lamblia or *G. intestinalis*		$<10^9$ cysts daily in stools	*16*
		1×10^6 to 5×10^6 cysts	*41*

cooked in families with children suffering from typhoid fever had *E. coli* on their hands compared with 16.1% for those who had no children ill with *Salmonella* Typhi infection. Khan et al. *(64)* found that the risk of children developing diarrhea from *Shigella* within these families was high whether the members were ill or not. The above data indicate the levels of contamination that can result from a lack of toilet paper and clean water and how easily one hand can contaminate the other, increasing the risk of both symptomatic and asymptomatic infections.

The use of toilet paper, however, does not guarantee there will be no fecal contamination. Hutchinson *(57)* found

that when solid stools containing ''scant growth'' of *Shigella sonnei* from a convalescent patient were touched by fingers wrapped with a double thickness of toilet paper, the pathogen reached the bare fingertips in four of five attempts. When the experiment was repeated with loose stools with heavier growth from patients with acute or mild shigellosis, contamination of fingertips occurred on every occasion. Even more revealing is the work of Michaels et al. *(89),* who collected data from volunteers on four consecutive days employing coprostanol as a marker for fecal contamination. Coprostanol is formed by the microbial breakdown of cholesterol in the human intestine and has been used as a human fecal biomarker *(107).* Volunteers were instructed to use six stacks of toilet paper, each with six sheet layers, to clean after each defecation during each day of the study. The amount of coprostanol in each layer of toilet paper was measured as was the level of coprostanol per gram of feces for that test day. The last sheet in each stack closest to the hand frequently had coprostanol contamination, indicative of finger contamination (15 of 24 individuals, 63%), despite the fact that penetration through the entire stack of six sheets occurred much less frequently (7 of 24 individuals, 29%). This finding indicates that fingers tend to become contaminated by both poke through the toilet paper and, more importantly, circumvention of the barrier protection afforded by the paper, which becomes contaminated, and transfer of the fecal matter to the last toilet sheet. The fecal mass on the paper from the fingers, based on the amount of coprostanol detected, was calculated, and the geometric mean quantities of fecal material detected on hands was estimated at approximately $10^{-5.6}$ g. However, on one occasion the fecal mass was found to be much higher at >1 mg. From the work of Feachem et al. *(35)* linking enteric bacterial counts to fecal mass, the results of Michaels et al. *(89)* indicate that $10^{3.4}$ to $10^{4.4}$ fecal coliforms could be present on hands when initially contaminated. Previous investigators had shown lower levels of hand contamination ranging from 5 to 2,000 CFU (fecal coliforms, *Enterobacteriaceae,* or *E. coli*) *(25, 26, 49, 55, 68).* Nevertheless, any fecal contamination on the hands has the potential to harbor pathogens if they are present in the gut, and these studies indicate the limited effectiveness of toilet paper and the quantity and frequency of hand contamination during toilet paper use.

Carriage rates before, during, and after illness and in asymptomatic individuals are important to determine. Unfortunately, information is lacking for several of these parameters for pathogens potentially associated with food workers *(121).* Carriage rates range between <1 and 36%, and shedding can occur many days before symptoms appear, making exclusion of excreting employees from the food environment very difficult. This difficulty is compounded by the fact that many pathogens survive well in the environment, and reinfection can occur in endemic regions.

Asymptomatic fecal excretion. Worker health status is not necessarily linked to risk of contamination. Medus et al. *(85)* noted that 64 (53%) of 121 *Salmonella*-positive food workers in 19 foodborne outbreaks in Minnesota reported not having recent gastrointestinal symptoms, and Sattar et al. *(113)* stated that possibly >90% of those individuals infected with viruses remain asymptomatic while discharging infective particles into their surroundings. Graham et al. *(44)* found that after inoculation of 50 volunteers with norovirus in buffered saline with bicarbonate to neutralize stomach acidity, 82% became infected, 68% were symptomatic (vomiting and/or watery diarrhea), and 32% were asymptomatic; the overall infection rate was higher than previously recognized, with a high rate of subclinical infection, prolonged virus shedding, and a significant correlation of magnitude of antibody responses with severity of symptoms. Although early studies indicated that norovirus shedding began with onset of symptoms and did not extend beyond 72 h, Graham et al. found that after inoculation of the 50 volunteers, virus first appeared in the stool at 15 h, and excretion peaked at 3 days and continued for at least 7 days.

In outbreak scenarios, those involving asymptomatic workers are as frequent as those involving workers who were ill *(46, 119, 120),* and excretion of pathogens can occur both before the illness and after the patient has recovered. During HAV infection, as many as 10^9 viral particles per gram of feces may be shed from >24 h to 2 weeks before symptoms develop *(43, 95).* Peak fecal excretion occurs before the onset of jaundice and other symptoms and then declines after jaundice appears. HAV excretion, however, can continue for many weeks after recovery and for up to several months in children. A food worker was still shedding Norwalk-like virus 10 days after resolution of illness following an outbreak, indicating that the recommendations of exclusion of workers for 48 to 72 h after recovery may not be sufficient to prevent transmission *(99).* This finding was supported by a study of 99 individuals infected with Norwalk-like (noro) viruses *(110)*; the virus was detected in 26% of the patients up to 3 weeks after the onset of symptoms; the highest proportion of long-term shedders (38%) were children less than 1 year of age. Long-term shedding was not associated with increased severity of disease or prolonged duration of clinical signs. Children have been identified as long-term excretors of enteric viruses, and these infected children may infect parents in the home, as demonstrated in a recent survey *(46, 119, 120).*

Hutchinson *(57)* found *S. sonnei* on the hands of 49% of young children in nursery schools in infected communities after these children had used the toilet, and over one-third of these children touched their faces and mouths or sucked their fingers. She also found that toilet seats in schools, nurseries, and private homes in these same communities were a means of transmission; 32% of these seats were contaminated with *S. sonnei.* Mothers in developing countries frequently carry *E. coli* as a result of caring for infants *(9, 84, 87).* In Thailand, enterotoxigenic *E. coli* was found on the hands of 6 of 42 mothers and of 37 of 50 children *(30).* These infected women typically prepared the meals for the family. Pether and Scott *(103)* examined stools of patients convalescing from *Salmonella* infections

from 6 to 102 days after symptoms ceased. As expected, stool counts tended to decrease over time but not in a consistent way. Median values for days after illness were 6.0×10^6 CFU at < 10 days, 1.0×10^5 CFU at 10 to 19 days, 2.5×10^4 CFU at 20 to 25 days, 1.4×10^2 CFU at 26 to 35 days, 5.5×10^3 CFU at 42 to 50 days, and 2.5×10^4 CFU at 69 to 102 days. The specimens that remained positive between 42 and 102 days had higher median counts than did some earlier specimens, indicating that an on-going colonization within the intestines may occur, allowing a continual supply of organisms for excretion, although all patients were asymptomatic. However, the counts of all patients diminished over time. The authors discounted concern about these numbers, assuming that the infectious dose was relatively high and that hand washing would remove any organisms present on hands after defecation. However, the large number of reports of salmonellosis outbreaks associated with food workers call this reasoning into question *(46).*

Many factors may influence whether pathogen ingestion will lead to symptomatic or asymptomatic infections. Noda et al. *(97)* conducted a statistical analysis of norovirus outbreaks in Japan and found that the attack rate in oyster-associated outbreaks was significantly higher than that in food worker–associated outbreaks, but the number of cases was greater in the latter (median, 40 versus 17 for the oyster-linked outbreaks). Most of the facilities involved in the food worker–associated outbreaks were large restaurants such as hotels that served food for parties and schools. The authors concluded that the attack rate in foodborne outbreaks of norovirus infection may be influenced by differences in implicated foods (sewage-contaminated oysters versus food worker–contaminated restaurant items), susceptibility of the host for norovirus infection, and pathogenicity of the strains. They also noted that a single norovirus genotype was responsible for most of the food worker–associated outbreaks (typically GII/4 or GII/3), whereas multiple norovirus genotypes were frequently involved in the oyster-associated outbreaks (accumulated over time in the oyster beds). The attack rate in 27 outbreaks associated only with GII/4 was lower than that in 136 other outbreaks, suggesting that GII/4 noroviruses cause asymptomatic infection more frequently than do other norovirus genotypes.

Well-investigated norovirus outbreaks sometimes demonstrate the complicated nature of infection spread, which can depend on prolonged asymptomatic shedding by some infected persons, the environmental stability of the implicated strains, and the lack of lasting immunity in persons who have been infected previously *(15).* One norovirus outbreak that occurred at a family reunion in West Virginia in 2006 involved attendees from Florida, Maryland, New York, Pennsylvania, Virginia, and West Virginia *(18).* The investigation indicated that a combination of person-to-person and foodborne transmission of two strains of norovirus, likely introduced by persons from two different states and subsequently into at least two food items, was the probable cause of these illnesses, highlighting the challenge of investigating and controlling norovirus outbreaks.

The carriage rate for *Salmonella* in England was determined by testing more than 2,800 stool specimens; 5% of ill individuals who presented to general practitioners were carriers compared with 1.1% of those individuals in the community that had loose stools or vomiting and 0.4% of those with no apparent illness *(123).* To determine the carriage rate for *Salmonella* in food workers, 331,644 fecal specimens were collected from workers in food factories, hotels, restaurants, and supermarkets in one region in Japan *(94)*; 0.032% of these samples contained *Salmonella,* and the most common serovars were Infantis, Corvallis, Enteritidis, and Agona. There were only two distinct pulsed-field gel electrophoresis (PFGE) profiles among the 16 *Salmonella* Corvallis isolates, indicating that the sources of infection for the many different types of food workers were not extensive. In Japan, these same serovars have been associated with broiler chickens and eggs. In the same community, a survey of diarrheal stools from clinical patients revealed a higher rate of infection (1.25%) but not the same serovar distribution (mainly *Salmonella* Enteritidis). This research indicates that relatively few asymptomatic food workers may carry *Salmonella,* but the reservoir is probably animal sources such as poultry or eggs, which workers contact while preparing food. In an outbreak in Jordan, *Salmonella* Enteritidis infection was traced to one food worker in a hospital *(65)*; 183 of 619 hospital employees and patients met the case definition after eating mashed potatoes for lunch. No diarrhea was reported by kitchen employees up to 3 months before the outbreak. However, the investigation revealed that 11 kitchen employees were positive for the same *Salmonella* serovar, but only 1 of them was responsible for preparing the mashed potatoes on the day of the lunch. Although the potatoes were mechanically mashed, this worker had mixed the potatoes with milk by hand and wore no gloves. He was apparently asymptomatic at the time but was the first patient to present to the emergency room about 16 h after the meal. Because the potatoes were prepared on the morning of the luncheon, there would have been only a few hours for *Salmonella* growth after the mashing.

Three percent of typhoid fever survivors become permanent carriers, harboring the organisms in the gallbladder, biliary tract, or rarely the intestine or urinary tract *(12).* Horwood and Minch *(56)* found *E. coli* on the hands of 13 of 34 food workers from foodservice establishments; those individuals without *E. coli* were waitresses and others not intimately handling food. de Wit and Kampelmacher *(25)* proposed that *E. coli* and fecal coliform counts increase as human involvement with moist foods of animal origin increases, because bacterial counts on the hands of more than 250 food workers and non–food workers revealed that hand contamination was more related to the type and nature of raw animal products they handled than to toilet hygiene. A survey of the hands of food processing plant workers revealed *E. coli* at >20 CFU per hand sample taken before and after defecation and hand washing (4 and 25%, respectively) *(26).* However, the average level of *E. coli* before and after collection of positive samples was about the same: 2.30 log CFU per sample. These quantitative data indicate that toilet use itself is not the only source of *E.*

coli for workers. The authors concluded that asymptomatic *Salmonella* excretors have only a low risk of transferring *Salmonella* from their stools by their hands to food. Contamination of hands with *Salmonella* is more likely to result from inconsistent hand washing during preparation of meat *(69).*

VOMIT AND URINE AS SOURCES OF CONTAMINATION FROM INFECTED AND COLONIZED WORKERS

Vomitus. Vomiting is the forceful expulsion of stomach contents through the mouth and is a symptom of a number of intestinal diseases caused by emetic toxins produced by infectious or toxigenic agents acting on the vomiting center of the brain. Nausea usually precedes vomiting. The onset of vomiting may be rapid and uncontrollable, often occurring during or shortly after meal time, which is indicative of a short incubation period for an emetic toxin in the food or the end of the prodrome stage for noroviruses. Noroviruses frequently cause projectile vomiting, and electron microscopic examination has revealed that a minimum of 10^6 virus particles per ml may be present in vomitus *(51).* Cotterill et al. (*24*) reported that rotavirus concentration in vomitus can be 100 times that in feces and that Norwalk virus is also found in vomitus. These findings suggest why vomiting is an important means of transmission for enteric viruses. The causative agent may be isolated from gastric contents, but acidic gastric secretions may destroy the pathogen before it can be isolated. Environmental cleaning and disinfection are vital for preventing disease transmission.

Vomitus can contaminate the environment, rest rooms, hand washing stations, and food worker clothing *(22, 31, 77),* and contamination of food may occur directly via aerosolized particles or hands. Noroviruses also can be transmitted to other individuals via aerosols produced during vomiting *(14, 51, 81).* Aerosol transmission resulted in infection of individuals that had simply walked through an emergency department while a vomiting patient was being examined *(114).* If an individual vomits within the kitchen area, fellow workers may become infected through inhalation of the agent or by hand-to-mouth transfer from contaminated environmental surfaces. The ease of airborne transmission of norovirus also was demonstrated in an outbreak at a restaurant where no food source was implicated but analysis of the attack rate revealed an inverse correlation with the distance from a person who had vomited *(81).* Although vomiting usually precedes unexpected bowel movements, vomiting and diarrhea may occur at the same time, making it difficult to determine which body fluid resulted in the transmission. This difficulty was demonstrated when a worker at a catering company frosted many cakes during a 6-h shift, although he had five episodes of diarrhea and two of vomiting at the beginning of his shift *(73).* Residual vomitus or feces on his hands after cleaning up from an attack was the likely source of the norovirus as he mixed the frosting with his hands. His actions infected nine other employees, who in turn caused another 3,000 illnesses from consumption of the cakes they prepared. In another scenario, a concert attendee vomited four times in an auditorium waste bin, on the carpet, and in the adjacent men's toilet. Before the next day's performance, the area was cleaned with a vacuum and a spillage compound without disinfectant. The next day, school children who sat on the same level of the auditorium where the infected antendee had been seated contracted norovirus infections from the viral particles still remaining on the seats and other areas of the auditorium *(31).* The staff who had worked on the upper tiers that same evening or had helped clean up after the vomiting and the family members who accompanied the infected antendee also were infected. The most likely cause of the 300-case outbreak was inadequate cleaning and disinfection of hard surfaces, carpets, and soft furnishings contaminated by vomit, leading to aerosols. In another outbreak, a kitchen assistant vomited into a kitchen sink; he tried to disinfect it with a chlorine-based sanitizing powder but failed to inform his supervisor *(101).* Boiled potatoes for a potato salad were cooled in the same sink the next day by another staff member, and the contaminated salad infected 56 wedding guests.

Another more recent example illustrates the importance of preventing vomiting while working in the kitchen environment and the importance of appropriate cleanup if vomiting occurs. In Michigan in 2006, a norovirus infection outbreak occurred in late January and early February 2006 *(19).* At least 364 restaurant patrons became ill with gastroenteritis after dining at a restaurant where employees had reported to work while ill. Low-level transmission occurred in the week before 28 January, with seven patrons who dined at the restaurant between 21 and 27 January meeting the case definition. From 21 January to 3 February, exposure to the virus likely occurred by contact with contaminated surfaces and objects. On 28 January, a line cook vomited at home before reporting to work at 11:00 a.m. and then vomited again into a waste bin beside the frontline workstation at approximately 2:00 p.m. while preparing antipasti platters, pizzas, and salads. After vomiting, he remained on site (but off the cooking line) and left work at 4:15 p.m. This person also reported to work on 29 January from 11:00 a.m. to 4:30 p.m. while still experiencing loose stools. Because of the open physical layout of the restaurant, no barrier impeded airborne spread of the virus from the kitchen to the main dining area. Direct foodborne transmission also was likely, as demonstrated by Bidawid et al. *(7),* who found that 46, 18, and 13% of surrogate feline calicivirus was transferred from contaminated fingerpads to ham, lettuce, and metal disks, respectively. The antipasti platter was one of many dishes that the infected line cook prepared, and statistical analysis revealed a significant association between the platter and the ill persons. Attack rates increased after the cook vomited and among employees who worked on 28 January, with a higher percentage of line cooks becoming ill compared with servers. Other environmental contamination probably contributed to transmission. The investigation revealed deficiencies in employee hand washing practices, cleaning and sanitizing of food and nonfood contact surfaces, temperature monitoring and maintenance of potentially hazardous food (even though

this issue would not impact the multiplication of norovirus directly), and maintenance of hand-sink stations for easy accessibility and proper use. A quaternary ammonium–based sanitizer that was ineffective against norovirus was used to clean the restaurant. Management was advised by the local health department to disinfect the restrooms and all surface areas within at least a 25-ft (7.6 m) radius of the vomiting site with a bleach solution. These actions were effective for preventing further illnesses.

Urine. Urine has been occasionally reported as a source of some pathogens. An HAV infection outbreak at a U.S. Navy base occurred when salad was intentionally contaminated by a cook with a personality disorder. As a mark of insubordination, he deliberately urinated into the salad during its preparation while he was still asymptomatic and transmitted his infectious HAV particles to the men eating the salad *(62). Salmonella* Typhi and *Salmonella* Paratyphi also can be detected in urine during systemic infection *(11),* highlighting the necessity of effective hand hygiene even for food workers who use the toilet only for urination.

SKIN AS A SOURCE OF CONTAMINATION FROM INFECTED AND COLONIZED WORKERS

Skin infections and secretions. Skin, the largest organ of the body, may have a large exposed area for abrasions, cuts, and punctures to occur, and these wounds can develop into infected boils and lesions. Boils and other deep lesions are typically caused by invasive infections by *S. aureus* carrying Panton-Valentine leucocidin genes *(53).* Extensive involvement of hands and arms is difficult to avoid during food preparation and food service, and this involvement has led to contamination, mainly by *S. aureus,* and subsequent outbreaks. Work in the kitchen environment can result in rough skin and small cuts, which may become infected. Boils also can occur through rubbing of skin by clothing or removal of hair follicles. These boils tend to occur on parts of the body other than the hands but act as reservoirs for *S. aureus* hand contamination through touching. Although workers tend to ignore these minor irritations, if the sores or boils are on the hands or arms, affected workers often reduce or stop hand washing to reduce the pain and inconvenience of removing bandages. As the infection progresses, pus can spread infectious organisms over skin surfaces and increase the risk of food contamination.

Open cuts, sores, and eczematous or excoriated lesions were associated with 25 outbreaks (37% caused by staphylococci and 24% caused by streptococci) reviewed by Greig et al. *(46).* The pathogens were carried by food workers who in turn contaminated custards, ham and cheese sandwiches, and potato, macaroni, egg, rice, tuna, and chicken salads. A home-catered social in Saskatchewan in 1988 resulted in 49 cases of *S. aureus* infection. The same phage type was isolated from potato salad, six patients, and six food workers; the same strain was isolated from an infected cut of a worker who mixed the salad with bare hands *(118).* In Georgia in 2000, PFGE analysis revealed that *S. aureus* from the food, ill patrons, and a plastic bandage from a worker's finger were indistinguishable *(74).* An outbreak of group A streptococcal pharyngitis at a school banquet was traced to contaminated macaroni and cheese. The same strain of *Streptococcus pyogenes* was isolated from affected individuals and a food worker's hand wound obtained in a fistfight 2 weeks earlier *(34).* Culture-positive purulent lesions can contain different pathogens, including enterococci, at up to 2×10^8 CFU/ml of fluid *(70)* (Table 1). *S. aureus* counts from persons with infected skin lesions may reach up to 10^5 CFU/cm^2 *(96),* but direct contact with food alone is insufficient for an infective dose or for toxin production in food, and a subsequent growth period is required to reach the high numbers necessary to produce toxin. However, the presence of infected open cuts and sores is not a prerequisite for contamination of food *(52).* Symptomless carriage of *S. aureus* and *S. pyogenes* in the nares and on the skin is common, and small numbers of these pathogens can continually contaminate surfaces, including food. Typically, temperature abuse of the food during storage is an additional requirement, and Bryan *(13)* found that such abuse was a contributory factor in 40% of foodborne outbreaks of *S. aureus* infection, where the storage time was sufficient for the organism to produce a heat-stable enterotoxin.

Skin infections on hands, however, do not necessarily prove that these lesions are the source of an outbreak. Phage typing of the skin may indicate a different strain from that in the implicated food. In one scenario, both *S. aureus* and *Salmonella* were found in leftover turkey but could not be traced to skin infections or colonized anterior nares of food workers *(86).* In another outbreak, 36 persons rapidly became ill with *S. aureus* intoxication after eating vanilla slices (thick vanilla-flavored custard in puff pastry) from a bakery *(36).* One of these persons developed five *S. aureus*–infected skin lesions on her legs 1 day after she ate the contaminated food. Investigators surmised that her legs became contaminated with diarrhea, and the pathogen subsequently invaded the skin. In another situation, although a food worker had severe cellulitis of his hands, he could not be linked to an outbreak of *Streptococcus* pharyngitis *(33).*

Molecular and toxin typing can help determine the cause of an outbreak, even when multiple bacterial strains have been isolated. After a staphylococcal food poisoning outbreak affecting 10 of 356 students who attended a high school breakfast, 27 *S. aureus* isolates producing different enterotoxin types were recovered from 7 patients, 2 food workers, and food leftovers *(130).* Based on genotyping results and enterotoxin typing, three distinct strains were identified. Two of these strains were found in specimens from patients and a hand lesion of a food worker, indicating the precise source of the contamination. However, not all outbreaks involving infected cuts are caused by staphylococci or streptococci. A food handler who had painful skin lesions on his hands was associated with an outbreak of HAV infection lasting several weeks *(127).* He prepared bread products and sandwiches for an English village shop while wearing visibly soiled bandages on his hands, which prevented him from washing properly. Viral particles were

likely excreted onto his hands during his mild illness while he continued to work preparing the bakery items.

Dermatitis and other skin diseases. Impetigo can be caused by either *S. aureus* or *Streptococcus* spp. In streptococcal cases, *S. pyogenes* colonizes the skin and can be transferred from one lesion to another and from one mucous membrane surface to another *(28).* The lesions eventually develop a characteristic honey-colored crust and heal without scarring. These lesions most commonly occur on the face or extremities. Colonization of the mucous membranes of the nose or throat occurs about 2 to 3 weeks after *S. pyogenes* appears on the skin, and the bacteria can survive an average of 10 days on the skin before any infected lesions appear. Occupational skin diseases (contact dermatitis being most frequently reported) account for approximately 40% of all occupational illnesses according to the U.S. Bureau of Labor Statistics, and contact dermatitis accounts for more than 90% of workers' compensation claims for occupational skin disease *(82, 83).* Occupations where hands are frequently immersed in water, as in food industries, have an increased potential for skin problems *(75, 82).* Age, sex, and skin allergy history also are important for food workers.

Women are more likely to have an atopic history and/or become sensitized to allergens than are men. Older individuals generally have dry, thin skin that is prone to cracking, resulting in more frequent cases of irritation and pain, which discourage hand washing. Atopic dermatitis (an allergic hypersensitivity such as eczema that affects various parts of the body) results in increased risk for sensitization and irritation by hand sanitizers, disinfectants, and metals such as chromium and nickel in stainless steel watches or jewelry. Atopy, which tends to have a strong hereditary component, also can be associated with food allergens, antimicrobial ingredients in soaps, allergens associated with gloves and antimicrobials, alcohol in instant hand sanitizers or hand antiseptics *(10, 38, 39, 125, 136),* glove powders, and pyrogens from sterile latex gloves *(72, 131, 133).*

Animal testing has revealed that several components are needed for sensitization of the skin leading to atopic diseases, including skin damage, an allergen potentiator, and occlusion of the skin *(66).* In the food processing and service environment, known allergens are present, and the skin can be damaged with brushes, cut, scratched, scalded, or burned or can come into contact with caustic chemicals. When hands are placed in gloves, the occlusion provided is conducive to the development of allergic contact dermatitis *(105, 106).* These issues highlight the importance of skin health in the overall control of transmission of human pathogens from food workers *(88).*

Sweat. Sites rich in sweat glands are well-recognized secondary niches of *S. aureus (29).* Although contamination of food by sweat has not been identified directly in an outbreak report, perspiration can allow normal skin microflora, including *S. aureus,* to be transferred to food. Staphylococci can migrate through perspiration to contaminate hands, jewelry, and work surfaces *(1, 2, 58, 90).* Moist hands facilitate the transfer of microorganisms, and sweat can transport potential pathogens colonizing the skin *(100).* In a hot kitchen environment, droplets of sweat containing *S. aureus* or *S. pyogenes* from the face and nose may directly contaminate food being prepared or indirectly contaminate food after perspiration is wiped away with the hands. If temperature abuse occurs, foodborne illness can result *(21).*

NASOPHARYNGEAL AND OROPHARYNGEAL SECRETIONS AS SOURCES OF CONTAMINATION FROM INFECTED AND COLONIZED WORKERS

S. aureus. Enterotoxigenic staphylococci have been isolated from about 30% of the population and 12 to 58% of food workers in different parts of the world *(2, 102, 104).* An estimated 25 to 50% of food workers are cutaneous or nasopharyngeal carriers of *S. aureus,* and 15 to 20% of these strains are enterotoxigenic *(50, 91, 109).* After an outbreak, these percentages may be higher *(27).* Food workers can harbor strains that produce both high amounts of toxin and more than one enterotoxin *(108).* Reina et al. *(109)* found that strains isolated from the skin were more enterotoxigenic than those from the respiratory tract or other clinical specimens; 61.7% of these strains produced one or more enterotoxins (mostly enterotoxins C and B). The evidence pointing to food workers as the source of many outbreaks of staphylococcal infection is compelling. Matching staphylococci phage types were obtained from the food, the affected individual, or the handler in 27% of outbreaks from 1977 to 1981 in the United States, and 67% of food workers were asymptomatic carriers of the same phage type as that in the implicated food *(52).* In a study of 358 outbreaks and sporadic cases of staphylococcal food poisoning in the United Kingdom between 1969 and 1990, strains from 79% of the incidents produced staphylococcal enterotoxin A or enterotoxin A plus another enterotoxin *(132).* The level of *S. aureus* in the implicated food ranged from none detected to 1.5×10^{10} CFU/g (median, 3.0×10^{7} CFU/g).

Hatakka et al. *(50)* studied the carriage rates of *S. aureus* in airline catering staff in Finland and found prevalences in noses and on hands of 29 and 9%, respectively. Forty-six percent of the strains isolated from workers were enterotoxigenic; 6% from their hands and 12% from their nasal cavities. PFGE results indicated that the same strain tended to be found in noses and hands, but detection of carriers is best done through nasal cavity sampling *(50).* The most prevalent enterotoxin producer was type A, which accounted for 34.9% of all the enterotoxigenic strains. The level of toxin production was somewhat lower than that found by Reali *(108),* who found that 76 to 80% of strains from nasal swabs of healthy carriers and from fecal and/or urine specimens and 100% of isolates from infected wounds and cutaneous and urogenital infections were toxin producers. However, the level of toxin production was similar to that in a more recent study of food workers in Santiago conducted by Figueroa et al. *(37),* who found that 54% of the strains were enterotoxigenic, with producers of enterotoxin A dominating. In another survey of food workers in

Botswana, 57.5% were positive for *S. aureus (78).* Of the 204 *S. aureus* strains isolated, 30.9, 44.6, and 24.5% were from the hand, nasal cavity, and face, respectively, and 21% of the isolates were enterotoxigenic, a lower percentage than in the Chilean and Finnish studies. These data indicate that many food workers are carriers of enterotoxigenic *S. aureus,* and screening of these workers and administration of antibiotics or removal of these workers from the food preparation area is impractical.

***S. pyogenes* (group A streptococci and beta-hemolytic streptococci).** The nasopharynx (area between the nose and throat) and the oropharynx (oral part of the pharynx reaching from the soft palate to the level of the hyoid bone) are potential breeding areas for microbial pathogens. *S. aureus* and *S. pyogenes* may reside in these areas without causing any indication of colonization, and coughs, sneezes, and wiping of the nose or mouth can transfer these bacteria to the hands. Even if cultures of these areas are positive for *S. pyogenes* or *S. aureus,* it is difficult to determine during outbreak investigations exactly how food has been contaminated. The organisms may originate from sputum or respiratory droplets or by direct hand contact by food workers. Persons whose nasal passages are colonized by beta-hemolytic streptococci are more likely to transmit infection than those with positive throat but negative nasal cultures, and such carriers disseminate the streptococci in highly contaminated nasal secretions that reach the environment, chiefly via the hands when blowing noses but also through contaminated handkerchiefs, clothing, and bedding *(48).* One chronic carrier had 2.4×10^7 CFU from a nose blow into a handkerchief compared with 3.8×10^4 CFU in his saliva *(48).* Bar-Dayan et al. *(5)* elucidated the differences between the symptoms of patients with endemic airborne streptococcal pharyngitis and those of patients with epidemic foodborne streptococcal pharyngitis. The patients with foodborne streptococcal pharyngitis had a significantly higher frequency of sore throat, fever, pharyngeal erythema, tonsillar enlargement, and submandibular lymphadenopathy and a lower frequency of coryza and cough compared with the patients with endemic airborne streptococcal pharyngitis. Although both foodborne and airborne streptococcal infection caused upper respiratory tract infection, the clinical manifestation of foodborne streptococcal pharyngitis was more severe and more confined to the pharynx than that of the more endemic airborne disease. Involvement of the nasal mucosa and bronchial tree was more common in cases of airborne streptococcal pharyngitis than in cases of the foodborne disease.

Experimental observations revealed that saliva in a cough of one carrier contained *S. pyogenes* at 10^3 to 10^6 CFU/ml and delivered 6 CFU/154 cm^2 at a distance of 9.5 ft (2.9 m), but most coughs from and loud talking by carriers spread few streptococci into the environment *(48).* Although sneezing is generally recognized as a risk factor and sneeze guards are put around food displays, few instances have been reported in which sneezing has been the direct cause of foodborne illness, and no reported foodborne illnesses have been associated with coughing *(117).* However, sneezes did allow the beta-hemolytic streptococci from 7 of 20 carriers to reach as far as 9.5 ft, and the organisms were persistent enough in the air to be cultured up to 16 min later *(47).* High numbers of *S. pyogenes* cells can be expelled in large droplets during a sneeze, settling in a zone of up to 1.5 ft (0.5 m) from the carrier source. A single sneeze by a nasal carrier of *S. pyogenes* is capable of expelling up to 5.0×10^7 CFU, whereas a sneeze by a throat carrier can expel around 1×10^5 CFU and a cough may expel 40,000 to 50,000 CFU *(48).* When outbreaks have occurred, they typically have been associated with streptococcal infection of a food worker, as demonstrated in the following two episodes. Egg salad served at a charity luncheon in Baltimore in 1957 infected 600 people, who contracted streptococcal pharyngitis *(33).* It was assumed that a woman who cooked and peeled the boiled eggs, at least in part, contaminated them with *S. pyogenes* by sneezing, although sneezing was not observed. A similar situation occurred in 1990, when egg salad served for lunch at a military base caused 61 cases of streptococcal infection *(79).* The same strain of *S. pyogenes* was isolated from the salad and the worker who had prepared the food while ill. The egg salad was stored in a refrigerator with a door that did not close properly, resulting in inadequate cooling of the salad. Sneezing or throat secretions were associated with the egg salad contamination. Following initial contamination, temperature abuse allows exponential growth of organisms. In their review of 18 streptococcal foodborne outbreaks, Katzenell et al. *(63)* found that cold salads were the main vehicle of infection, and the outbreaks occurred either in warm climates or during summer months in more equitable regions where there were opportunities for rapid growth. Almost all of these salads were prepared 24 h in advance of the meal time, and some of the foods were kept out of the refrigerator for several hours before they were served, providing opportunities for growth. In one experiment, streptococci isolated from throats of patients with pharyngitis were grown at room temperature on a medium containing eggs; these bacteria multiplied by 8 log units in 40 h. In outbreaks for which the data have been published, food workers typically had sore throats and/or pharyngitis either before or during the meal preparation; one of the workers had an infected lesion. Asymptomatic carriage was rare. Although the risk of food contamination is high when food is touched by colonized food workers *(13),* it is not always easy to discover how a particular worker becomes infected and how that worker then contaminated the food, causing illness. An effective approach to preventing transmission of staphylococcal and streptococcal infections is training food employees to recognize infected lesions and utilize double waterproof barriers, such as a bandage and finger cot to cover an infected lesion on a finger, and to follow good personal hygiene practices *(124).* Community-acquired methicillin-resistant *S. aureus* (MRSA) is spread by direct contact, in aerosols, and on fomites. However, occasionally MRSA is linked to food. In one serious hospital outbreak in The Netherlands, 21 hematology patients (77.8%) developed clinical disease, and 5 died *(67).* Subsequently, MRSA was detected in food and in the throat of

one health care worker who prepared food for the hematology patients. Food contaminated by this worker most likely caused the first case of MRSA septicemia, and the outbreak strain probably was transmitted to the surgical unit by a colonized nurse. Another outbreak investigation of MRSA infections revealed that a worker at a convenience store was associated with three cases of MRSA infection that occurred after consumption of pork and coleslaw *(61).* The worker apparently became colonized while visiting an elderly relative and remained culture positive without showing signs of illness for more than 8 months.

Work exclusion criteria. Postsymptomatic workers may continue to excrete pathogens, but at lower rates, for days, weeks, and occasionally years. Although shedding duration can be measured only by a regular stool-testing regimen, this approach often is ineffective, costly, and not recommended, especially for individuals hospitalized for >3 days *(55, 92, 111, 115)*. An outbreak involving an airline caused 290 cases of salmonellosis during a period of 6 days *(135).* Unfortunately, the infected employee was not identified during regular inspection and testing of the establishment. Stool testing and exclusion of workers has been an issue for many decades, and recommendations differ among jurisdictions. Winter *(134)* recognized the limitations of annual testing of food workers for pathogens but understood that some form of certification for workers within the European Economic Community was necessary. A better approach would be for employees in large companies to be examined by a nurse or physician and certified to be free from infectious agents such as *Salmonella* Typhi, HAV, *S. pyogenes,* and *Mycobacterium tuberculosis.* Winter *(134)* found that potential employees may be rejected (5 to 50% of them) because of decayed teeth, infested hair, dirty body and clothes, and dirty or bitten fingernails. Pether and Scott *(103)* examined the stools of convalescent carriers of different *Salmonella* serovars and suggested that although some of their fingers were contaminated, the clinically well food worker with formed stools should be allowed back at work without further examination of fecal specimens.

Generally, pathogen-negative stool samples, either preemployment or from an employee recovering from a diarrheal illness, are not necessary conditions of employment or return to work, with the exception of typhoid and paratyphoid infections and infection caused by verocytotoxin-producing *E. coli* (VTEC) *(32).* However, typhoid and paratyphoid fevers and HAV infection need special consideration because of the severity of the illnesses and because individuals can continue to carry and excrete the etiologic agents for long periods after recovery, with a consequent risk of food contamination. There is no consistent approach among different jurisdictions and associations for stool testing and worker exclusions. Typically, three negative stool specimens are required before an infected food employee can return to work. When VTEC infection, including *E. coli* O157:H7 infection, is identified in a food worker, the worker should be excluded from work until the bowel habit has been normal for 48 h and two negative fecal samples taken 48 h apart have been obtained. Symptomless contacts of a person with HAV infection can continue food handling, but workers who have symptoms of hepatitis, have been in an outbreak or have been associated with family members suffering from HAV infection, or have traveled to regions where HAV is endemic would be excluded from work until they have a medical release based on laboratory testing. The Food Marketing Institute *(40)* has published similar recommendations but also stated that the single most critical way that food workers can prevent the spread of HAV is to wash hands thoroughly and often upon arriving at work, after using the toilet, after breaks, and at many other times during food preparation. Disorders such as Crohn's disease or ulcerative colitis are not a barrier to employment as a food worker, even though such disorders may result in diarrhea. Such workers must be made aware of the need to seek medical advice and notify their managers if any change from their normal bowel habit occurs, because such a change must be assumed to be infectious until proven otherwise *(32).* According to the Food Code *(124),* an employee is required to report *Salmonella, Shigella, E. coli* O157:H7, and HAV infections or illnesses such as diarrhea, vomiting, fever, jaundice, or sore throat with fever. Employees are required to provide written medical documentation that they are free from the four specified infectious agents via stool testing *(17).* After an outbreak in Michigan, new guidelines were produced recommending that foodservice workers with suspected norovirus not return to work until they are asymptomatic for 48 to 72 h, longer than the previously recommended 24 h *(17).* Thus, as new data from investigations of outbreaks are generated, they may influence exclusion policies.

CONCLUSION

Enteric organisms from fecal sources are excreted during an infection, whether the individual is symptomatic or not, but the infection exists over a limited time period (usually hours to weeks). Many of these enteric pathogens are of concern to food workers, e.g., *Salmonella, Shigella,* and noroviruses. These pathogens can contaminate the hands after defecation or from touching fecally soiled clothing or surfaces. Changing messy diapers, cleaning after episodes of vomiting or diarrhea by family members, and contact with sick or healthy pets also are well-established risk factors. Michaels et al. *(89)* found that fecal matter as detected by coprostanol can reach the fingers despite the use of up to six layers of toilet paper under conditions of normal perianal cleaning. Thus, the use of toilet paper is no guarantee of uncontaminated hands, and the situation is worse for individuals with diarrhea, especially when the individual has long or artificial fingernails, which are notoriously difficult to clean. Infected workers that are excreting a pathogen also risk infecting their colleagues during shared operational activities. Contamination levels may be higher when workers handle raw foods of animal origin than after toileting. Handling of raw foods can occur in a meat department of a supermarket or in a restaurant, where *Campylobacter, Salmonella,* and *E. coli* O157:H7 can be common contaminants.

Similar scenarios may occur through handling crops,

foods, or food ingredients contaminated with animal manure, animal intestines, sewage, nonpotable water, etc., which may occur during harvesting, hunting, or butchering. Lues and Van Tonder *(80)* found that both aprons and hands of food workers may be vehicles of cross-contamination, but there was no statistical correlation between the organisms on hands and those on aprons, indicating other sources of contamination for aprons. The impact of environmental sources in food environments, including raw foods of animal origin, were further discussed by Todd et al. *(122).*

Parasites tend to be overlooked as a source of foodborne infections, particularly in developed countries where they rarely cause outbreaks. However, *Cryptosporidium, Giardia,* and *Cyclospora* all have caused foodborne outbreaks, although the role of the food preparer in transmitting these agents has not always been clear. Carriage rates of both diarrheic and healthy populations can be >5% *(123),* providing many opportunities for fecal contamination of foods and contact surfaces. No known outbreaks of *Entamoeba histolytica* infection have been reported, but this organism is found worldwide and especially where hygiene is poor. Other parasites that can be spread in food are the obligate parasitic eukaryotic microsporidia. Fresh produce such as berries, sprouts, and leafy greens can contain as high as 10^3 spores of *Enterocytozoon* spp. *(60).* These organisms probably come from contaminated irrigation water.

Carriers of bacterial or viral pathogens were originally infected after they were exposed to a pathogen source, such as during a foodborne or waterborne outbreak or through other infected persons such as convalescent patients and other carriers. From 1979 to 1995, at least 450,000 persons ≥20 years of age were hospitalized for gastroenteritis in the United States *(93).* This number excludes ill children, from whom siblings, parents, friends, caregivers, and teachers can acquire the infective agent and in turn become asymptomatic excretors.

As an indication of the likelihood of spread of pathogens by food workers, Clayton and Griffith *(23)* found that on average workers in the United Kingdom washed their hands adequately on only 9% of occasions after they had touched their faces or hair and on only 25% of those instances in which they touched potentially contaminated objects. In the United States, Green et al. *(45)* observed that workers made hand washing attempts (i.e., removed gloves if worn and placed hands in running water, used soap, and dried hands) after only 27% of activities such coughing, sneezing, working with raw animal products, or handling dirty equipment, utensils, or cloths. These data are in contrast to the confidence expressed by Pether and Scott *(103),* who considered convalescent *Salmonella* carriers a minimal risk for contaminating food. More recent data have indicated that worker hygiene is lax at times and that newer agents such as noroviruses have lower infectious doses than do salmonellae. These data give us reasons to consider the risks that occur in food preparation venues where there are opportunities for fecal and nasal contamination of hands on a regular basis and a lack of adequate hand washing. The next article in this series on food worker infections and hygiene *(122)* will cover the transmission and survival of pathogens in the food processing and preparation environment.

ACKNOWLEDGMENT

The authors appreciate the critical review by Shirley B. Bohm (Consumer Safety Officer, Retail Food Protection, Center for Food Safety and Applied Nutrition, U.S. Food and Drug Administration, College Park, Md.), who offered helpful comments and new information to improve the manuscript.

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