

Review

Outbreaks Where Food Workers Have Been Implicated in the Spread of Foodborne Disease. Part 6. Transmission and Survival of Pathogens in the Food Processing and Preparation Environment

EWEN C. D. TODD,^{1*} JUDY D. GREIG,² CHARLES A. BARTLESON,³ AND BARRY S. MICHAELS⁴

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

MS 08-293: Received 19 June 2008/Accepted 18 July 2008

ABSTRACT

This article, the sixth in a series reviewing the role of food workers in foodborne outbreaks, describes the source and means of pathogen transfer. The transmission and survival of enteric pathogens in the food processing and preparation environment through human and raw food sources is reviewed, with the main objective of providing information critical to the reduction of illness due to foodborne outbreaks. Pathogens in the food preparation area can originate from infected food workers, raw foods, or other environmental sources. These pathogens can then spread within food preparation or processing facilities through sometimes complex pathways and may infect one or more workers or the consumer of foods processed or prepared by these infected workers. The most frequent means of worker contamination is the fecal-oral route, and study results have indicated that toilet paper may not stop transmission of pathogens to hands. However, contact with raw foods of animal origin, worker aerosols (from sneezes), vomitus, and exposed hand lesions also have been associated with outbreaks. Transfer of pathogens has been documented through contaminated fabrics and carpets, rings, currency, skin surfaces, dust, and aerosols and though person-to-person transmission. Results of experiments on pathogen survival have indicated that transmission depends on the species, the inoculum delivery route, the contact surface type, the duration and temperature of exposure, and the relative humidity. Generally, viruses and encysted parasites are more resistant than enteric bacteria to adverse environmental conditions, but all pathogens can survive long enough for transfer from a contaminated worker to food, food contact surfaces, or fellow workers.

A lack of understanding in many food service settings concerning the transmission and growth of pathogens (80, 195, 199) can lead to potential foodborne outbreak situations. Similar issues are associated with food prepared in households and served at home or used in home-catering situations, but problems in these settings are much less likely to be reported (80). Such lack of understanding or ignoring the risks of contamination is illustrated by a hepatitis A virus (HAV) outbreak involving an ice-slush beverage contaminated by an infected employee in a convenience market (30). The utensils for the beverage machine used by the infected worker were kept beside the toilet bowl. This gross error in hygiene should have been identified by the management, and appropriate information should have been relayed to employees. Outbreaks also have been caused by workers changing diapers on infants with diarrhea before going to work. Sick children at home are a risk factor for foodborne illness, and effective hand washing practices

must be taught and adhered to (113). Even healthy family members may transmit pathogens. For instance, a higher carriage rate for β -hemolytic streptococci occurs in children, up to 47% in normal school populations (95). In numerous reports from countries where sanitary facilities are lacking, food has become contaminated by unwashed hands and exposure to temperature abuse. Contamination can be transferred to and from workers through raw food, hands (including dirty fingernails, rings, and other jewelry), clothing, aerosols, fomites, food soil, food packages, and other environmental sources and pathogens can survive for extended periods of time on many surfaces, including skin.

TRANSFER OF CONTAMINATION ASSOCIATED WITH FOOD WORKERS

Raw foods of animal origin (cross-contamination). Contamination during production and processing, or cross-contamination in the kitchen, was reported as a contributing factor in one-third of outbreaks identified in U.S. foodborne disease outbreak data collected by the Centers for Disease Control and Prevention from 1998 to 2002 (60). Types of

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

TABLE 1. Levels of pathogens in raw meat and poultry and in animal feces

Pathogen	Source of contamination	Contamination level (per g or ml)	Reference(s)
<i>Campylobacter</i>	Chicken juice, skin, carcass	10 ³ –10 ⁷ CFU	23, 39, 157
	Cecal contents of broilers	10 ⁵ –10 ⁹ CFU	64
	Retail chicken carcasses	20% with >10 ⁵ CFU	64, 91
	Chicken breast	87% with mean of 1.9 × 10 ³ CFU/fillet	118
	Retail chicken fillet surface	Mean of 1.9 × 10 ³ CFU	118
	Retail chicken fillet deep tissue	Mean of 0.24 CFU	118
	Chicken carcasses	Most chicken carcasses <10 ⁴ –10 ⁶ CFU; 36% of chickens had >5,500 CFU; <5% had 10 ⁵ –10 ⁹ CFU	91
<i>Escherichia coli</i> O157:H7	Whole chicken carcass rinse at rehang	Mean of 2.66 log CFU	14
	After broiler chill and processing	Mean of 0.43 log CFU	14
<i>Salmonella</i>	Ground beef	5 CFU	31
	Cattle and sheep feces (high shedders)	10 ³ –10 ⁵ CFU	153
	Chicken juice, skin, organ, carcass	10 ² –10 ⁶ CFU	73, 81, 91
	Chicken fillets	Most chickens with enrichment-positive samples only; 2 of 101 chickens with 10 ^{3.8} –10 ^{4.5} CFU/carcass by direct enumeration. Chicken fillets: 8.6% were positive with counts ranging from 1–3.81 log MPN/fillet	198

cross-contamination included hand to surface, surface to hand, food to hand, hand to food, and combinations of these. Any surface touched by infected workers while preparing food can easily become contaminated (47, 221). The rate of transfer of microorganisms from raw to cooked food via hands ranges from 0.005 to 100%, although the lower rates are recorded more frequently (34, 39, 221). Humbert et al. (81) recovered *Salmonella* at up to 60, 280, and 960 CFU/g from liver, spleen, and ovaries, respectively, from 4-week-old laying chickens naturally infected with *Salmonella* Enteritidis PT33, and the juice or skin of chicken carcasses may contain *Campylobacter* or *Salmonella* at 10² to 10⁷ CFU/g or ml (Table 1).

Harrison et al. (73) isolated *Campylobacter* from 3% of samples from external food packaging and 34% of samples from whole packaging (outside plus inside wrapping); *Salmonella* was also found on 11% of samples from the whole packaging. These data indicate that workers and consumers may contaminate their hands when handling food packages even before opening them, providing another means of spreading pathogens. Sattar et al. (185) found that the transfer of HAV between objects depended on the drying time on the donor surface. Transfer from metal disc to finger pad was 24.7% after 20 min and 5.3% after 120 min. Similarly, the transfer from finger to metal was 28.4% after 20 min and 7.4% after 120 min. From finger pad to finger pad, transfer was similar (27.0% after 20 min and 8.9% after 120 min). Bidawid et al. (16) found a transfer rate for HAV of 9% from finger pads of adult volunteers to pieces of fresh lettuce and up to 46% from fingers to three different foods; for feline calicivirus the rate was as high as 14% from food to hand (17). However, Chen et al. (34) found that the transfer efficiency of bacteria was much less: 0.3% between hands and lettuce and 1% between hands and spigot. After touching chicken inoculated with 8 to 9 log CFU

of *Enterobacter aerogenes*, hands transferred 2.4 to 5.7 log CFU to a spigot on the hand washing sink (34). Even after hands were washed and dried with a paper towel, 1.9 to 6.5 log CFU still remained. When individuals prepared chicken contaminated with *Salmonella* and *Campylobacter*, the *Salmonella* was isolated more frequently (39), confirming the findings of previous studies in which *Salmonella* survived better than *Campylobacter* on dry surfaces (46). Cogan et al. (39) also found that after cleaning, fewer food preparation surfaces were contaminated with both these pathogens but more kitchen sites were contaminated, i.e., taps, utensils, and condiments, indicating that bacteria are easily transferred during the “cleaning” process.

Approximately 62,000 cases of foodborne *Escherichia coli* O157:H7 infection and 2 million cases of foodborne *Campylobacter* infection are reported each year in the United States (137). Many of these *E. coli* cases are associated with ground beef, and 80% of these *E. coli* cases have been associated with consumption of hamburgers prepared at home (136). Although the *E. coli* O157:H7 contamination rate is <0.3% in ground beef (208), levels as low as 5 CFU/g have been reported from outbreak samples (Table 2), and the infectious dose is considered very low, e.g., 1 to 100 cells (196). Recent work has shown that some cattle can be high shedders of *E. coli* O157:H7 (>10⁴ CFU/g feces; Table 1); therefore, certain batches of ground beef may have higher concentrations of *E. coli* than others. High-shedding sheep (excreting >10⁴ CFU/g) were responsible for an outbreak in Scotland (153). *E. coli* outbreaks from ground beef are continuously reported, so even small amounts of contamination from meat on hands must be considered a risk for transmission. In a survey of almost 20,000 adults, Alterkruse et al. (4) found that 25% of men and 14% of women did not routinely wash their hands with soap after handling raw meat or poultry, but these percentages

TABLE 2. Survival of enteric pathogens on hands and food contact surfaces

Infective agent	Surfaces	Suspending media	Loss (log CFU or %) or half-life	Reference
<i>Campylobacter</i>	On glass slides simulating a work surface and on moist cloth	Not stated	Decimal reduction times on glass, 0.5–24 h at RT ^a	67
	Poultry		Decimal reduction times on raw and cooked meats, ≥3 days at 4°C	67
<i>C. jejuni</i>	Hands	Peptone water with chicken broth and 50% blood	3–7 log CFU loss in 2 min, 6 log CFU loss in 15 min, 6 log CFU loss in 45 min	38
<i>Escherichia coli</i>	Dry inanimate surfaces		Up to 6 days	101
	Fingertips	Mixed culture with <i>Listeria monocytogenes</i> serotype 4b in saline and milk	Saline: 99.95% loss after 15 min, 100.0% loss after 45 min. Milk: 92.05% loss after 15 min, 94.15% loss after 45 min	192
	Fingertips	Suspended in broth culture at 530 CFU/fingertip	99% in 1 h	164
	Fingertips	Broth culture	99.99% after 5 min, >6 log CFU after 90 min	54
	Coins		At 25°C, survived 7, 9, and 11 days on the surfaces of pennies, nickels, and dimes or quarters, respectively	89
<i>Klebsiella aerogenes</i>	Teflon and glass surfaces		At 25°C, survived 4–7 days	89
	Dry inanimate surfaces		1.5 h to 16 mo	101
	Skin, with outbreak strains more resistant than environmental strains	Suspension in Ringer's solution	97.04% after 5 min, 98.82% after 10 min	41
	Contaminated donor fabrics to hands	Broth culture	0.29% transferred and 66% loss on skin after 5 min	120
	Fingertips	Broth culture	99% after 5 min, 3–4 log CFU after 90 min	54
<i>Listeria</i> spp.	Dry inanimate surfaces		1 day to months	101
	<i>L. monocytogenes</i>	Fingertips	Saline	0.45% after 30 min
		Milk	23% after 30 min, 9% after 120 min	192
<i>L. monocytogenes</i> sero-type 4b	Fingertips	Mixed culture with <i>E. coli</i> in saline and milk	Saline: 98.5% after 15 min, 99.55% after 45 min. Milk: 66.05% after 15 min, 85.8% after 45 min	192
		Two <i>L. monocytogenes</i> 4b strains in milk alone	0.0–17.7% after 120 min	192
<i>Salmonella</i>	Dry inanimate surfaces		1 day	101
	Currency notes in Myanmar	Total aerobic bacteria and fecal coliforms	0–2.9 × 10 ⁷ CFU/cm ² of notes. <i>Salmonella</i> , enterotoxigenic <i>E. coli</i> , and <i>Vibrio</i> isolated from notes from butchers and fishmongers	99
<i>Salmonella</i> Anatum	Fingertips	Suspended in broth culture at 530 CFU/fingertip	<i>Salmonella</i> Anatum still present 3 h later	164
<i>Salmonella</i> Enteritidis	Hands to Formica surface, utensils, kitchen surfaces	Egg white and yoke	Survived well for 24 h	83
	Dishcloths from homes of salmonellosis cases	Egg or blood	Survived >1 yr	82
		Natural contamination	12% positive after many days	82
	Coins		At 25°C, survived 1, 2, 4, and 9 days on the surfaces of pennies, nickels, quarters, and dimes, respectively	89
	Teflon and glass surfaces		Up to 17 days	89

TABLE 2. Continued

Infective agent	Surfaces	Suspending media	Loss (log CFU or %) or half-life	Reference
<i>Salmonella</i> Typhi	Dry inanimate surfaces		6 h to 4 wk	101
<i>Salmonella</i> Typhimurium	Dry inanimate surfaces		10 days to 4.2 yr	101
<i>Shigella</i> spp.	Dry inanimate surfaces		2 days to 5 mo	101
<i>Staphylococcus aureus</i>	Glass	Broth diluted with distilled water	At RH ^b 40–60% and 37°C: 49–94% after 60 min, 98–100% after 12 h. At RH 40–60% and RT: 8–11% after 60 min, 29–89% after 12 h	103
	Skin of volunteers	Broth diluted with distilled water	7–39% after 5 h, 72–94% after 12 h	103
	Fingertips	Broth culture	99% after 5 min; 4 log decline after 90 min	54
	Dust exposed to different light intensities	Naturally contaminated sweepings from hospital wards	At RH 50–60%: maximum loss after 10 days of 0.6 log CFU in low daylight, 2 log CFU in sunshine, 0.13–0.64 log CFU in artificial light	109
	Glass coverslip	Serum	0.0–62.0% after 100 min, 8.0–99.5% after 2–3 days	117
		Water	0.0–95.0% after 100 min, 84.0–98.4% after 2–3 days	117
	Dry inanimate surfaces		7 days to 7 mo	101
<i>S. saprophyticus</i>	Contaminated donor fabrics to hands	Broth culture	1.67% transferred and 58% loss on skin after 5 min	120
<i>Streptococcus haemolyticus</i>	Airborne	Broth culture	At RH 0%, 100% in 2 h. At RH 20%, 50% in 2 h	174
<i>S. pyogenes</i>	Dust exposed to different daylight intensities	Naturally contaminated sweepings from hospital wards	0.5 log CFU in low daylight, 1.0 log CFU in sunshine in 10 days	109
	Glass coverslip	Serum	0.0–56.6% in 100 min, 0.0–92.8% in 2–3 days	117
		Water	0.0–91.6% in 100 min, 0.0–99.14% in 2–3 days	117
	Contaminated donor fabrics to hands	Broth culture	0.01–0.02% transferred and 38–77% loss on skin after 5 min	120
<i>Vibrio cholerae</i>	Dry inanimate surfaces		3 days to 6.5 mo	101
	Dry inanimate surfaces		1–7 days	101
Hepatitis A virus	Stainless steel	Fecal suspension	Half-life (h) 5°C 20°C 35°C Low RH (25%) 169 187 65 Medium RH (55%) 151 128 50 High RH (80%) 123 71 21 Ultrahigh RH (95%) 103 51 2	132
	Fingertips	Fecal suspension	70–84% loss in 4 h	131
	Hands	Fecal suspension	5.5–7.7 h	185
	Stainless steel	Fecal suspension	At RH 25%: 5°C, 169 h; 20°C, 187 h; 35°C, 65 h	185
	Stainless steel, copper, polythene, polyvinyl chloride	Phosphate-buffered saline (PBS)	After 8 h at 20°C, there was a 2-log reduction of added virus on stainless steel, polythene, and polyvinyl chloride surfaces compared with a 1-log reduction at 4°C. Virus survival on copper was lower at both temperatures, with reductions of 3 log units at 20°C and 2 log units at 4°C	102
	Dry inanimate surfaces		2 h to 60 days	101
	Aluminum	PBS drying for 3–5 h	0.1-log titer reduction	1

TABLE 2. *Continued*

Infective agent	Surfaces	Suspending media	Loss (log CFU or %) or half-life	Reference
Adenovirus	Glazed ceramic tile	PBS drying for 3–5 h	0.6-log titer reduction	1
	Cloth	PBS drying for 3–5 h	1.6-log titer reduction	1
	Aluminum	20% feces drying for 3–5 h	0.1-log titer reduction	1
	Glazed ceramic tile	20% feces drying for 3–5 h	0.4-log titer reduction	1
	Cloth	20% feces drying for 3–5 h	0.8-log titer reduction	1
	Dry inanimate surfaces		7 days to 3 mo	101
	Aluminum	PBS drying for 3–5 h	2.1-log titer reduction	1
	Glazed ceramic tile	PBS drying for 3–5 h	2.3-log titer reduction	1
	Cloth	PBS drying for 3–5 h	3.3-log titer reduction	1
	Aluminum	20% feces drying for 3–5 h	2.4-log titer reduction	1
Astrovirus	Glazed ceramic tile	20% feces drying for 3–5 h	3.5-log titer reduction	1
	Cloth	20% feces drying for 3–5 h	3.2-log titer reduction	1
Rotavirus	Dry inanimate surfaces		7–90 days	101
	Aluminum	PBS drying for 3–5 h	1.0-log titer reduction	1
	Glazed ceramic tile	PBS drying for 3–5 h	1.2-log titer reduction	1
	Cloth	PBS drying for 3–5 h	0.6-log titer reduction	1
	Aluminum	20% feces drying for 3–5 h	0.8-log titer reduction	1
	Glazed ceramic tile	20% feces drying for 3–5 h	0.3-log titer reduction	1
	Cloth	20% feces drying for 3–5 h	1.0-log titer reduction	1
	Stainless steel, rough plastic, smooth plastic, glass	Fecal suspension	RH 25%, 0.03–0.05 log unit/day; RH 50%, 0.05–0.07 log unit/day; RH 85%, 1.6–2.4 log unit/day (99.9% reduction in 48 h)	183
	Fingertips	10% fecal suspension	At 20, 60, and 260 min after inoculation, 43, 57, and 93%, respectively	6
	Norovirus and feline calicivirus	Dry inanimate surfaces		6–60 days
Dry inanimate surfaces			8 h to 7 days	101
Computer mouse, keyboard keys, telephone wire, telephone receiver, telephone buttons, brass disks representing faucets and door handle surfaces		Feline calicivirus in culture medium with fetal bovine serum	90%: 0–4 h on computer keys, mouse, brass, and telephone wire; 4–8 h on telephone receiver; 12–24 h on telephone buttons; nondetectable at 72 h	36
Metal disc			89%	130
Strawberries			99%	130
<i>Entamoeba histolytica</i>	Lettuce		80%	130
	Ham		57%	130
	Nail region of hands	Fecal suspension	Survival up to 45 min	5
	Fingers and thumbs	Fecal suspension	≥95% in 10 min	194

^a RT, room temperature.

^b RH, relative humidity.

varied by population. Men take more risks than women, younger people take more risks than older ones, whites take more risks than blacks, and individuals with higher incomes are not as careful as are those with lower incomes. In a case-control study, individuals who prepared food in case, as opposed to control, households were less likely to wash hands and work surfaces after handling raw ground meat (136). Hands or contaminated work surfaces are potential vehicles of cross-contamination from raw meat to ready-to-eat (RTE) foods, and the authors estimated that hand washing could have prevented 34% of the *E. coli* infections. The risks of contamination were confirmed by Wachtel et al. (214), who found that when hamburger patties containing

E. coli O157:H7 are formed, the organisms are transferred to both hands and cutting boards and to lettuce subsequently put on the boards. A 15-s water rinse did not remove significant amounts of pathogens from the boards, whether the boards were washed immediately after contamination or after overnight room-temperature storage.

Diseases linked to *Campylobacter* and *E. coli* O157:H7 tend to be seasonal and related mostly to young children, who are exposed to environmental sources such as water, to elevated ambient temperatures, and to contaminated food (116). However, the most likely reservoirs for these organisms in food processing and preparation settings are poultry and ground beef, respectively (23, 91, 93, 206,

212). Levels of *Campylobacter* in chickens can reach 10^9 CFU per carcass but mostly are $<10^4$ CFU (Table 1). *E. coli* O157:H7 has been detected in cattle feces at concentrations of 4 to 10^7 CFU/g, but usual levels are <10 to 100 CFU/g (69). Decontamination steps at slaughterhouses may explain the low prevalence of beef contamination; $<0.5\%$ of ground beef samples were positive for *E. coli* O157:H7 during the last decade, and 0.24% were positive in 2007 (208). Nevertheless, because of the high volume of ground beef consumed, outbreaks and cases within families continue (13), as illustrated by a large recall of product from a meat packer following an outbreak in Colorado in 2007 that was associated with retail-purchased ground beef (212). Poor worker hygiene could result in infection after direct contact with raw meat and poultry or indirect contact with contaminated food contact surfaces.

Cogan et al. (39) found that cleaning with hot water and detergent did little to remove *Salmonella* and *Campylobacter* from chicken preparation areas in kitchens; hypochlorite was much more effective for destroying these organisms. In two other studies, one in Ireland and the other in Puerto Rico, of meal preparation involving chicken, the researchers noted that cross-contamination was widespread. In the Irish study, *Campylobacter*, *E. coli*, *Salmonella*, and *Staphylococcus aureus* from chicken were found on dishcloths, refrigerator handles, oven handles, counter tops, draining boards, and preparers' hands (63). *Campylobacter* and *S. aureus* were the pathogens most frequently found both on surfaces and on hands. During preparation of chicken salad in Puerto Rico, only 25% of workers washed their hands with soap and water before handling the chicken and after the chicken was ready but before handling the lettuce and tomatoes (48). Only 55% of those who used the same cutting board for the chicken and the vegetables washed the cutting board with soap and water, and 13% used the same knife to cut both chicken and vegetables without cleaning the knife between uses. *Salmonella* was isolated from the chicken and refrigerator handles, *S. aureus* was found on all the food and the contact surfaces (chicken breasts, lettuce and tomatoes, refrigerator and freezer, counter tops, cutting boards, and knives), and *Listeria* spp. were found on all areas excepts the counter tops. *Campylobacter* cross-contamination in kitchens also was studied under typical kitchen scenarios, and *Campylobacter* transfer from naturally contaminated chicken parts was quantified (119). The mean transfer rates from chicken legs and fillets to hands were 2.9 and 3.8%, respectively. The transfer from legs to the plate (0.3%) was significantly lower ($P < 0.01$) than the transfer from fillets to the cutting board and knife (1.1%). The average transfer rates from hands or kitchen utensils to RTE foods ranged from 2.9 to 27.5%.

Fischer et al. (55) found that although most consumers are knowledgeable about the importance of preventing cross-contamination and using adequate heating to prevent foodborne illness, this knowledge is not necessarily translated into behavior. Potentially risky behaviors were observed in the domestic food preparation environment during a study of volunteers in The Netherlands who prepared chicken salad. Eighteen of the participants made errors in

food preparation that could potentially result in cross-contamination with *Campylobacter*, and seven participants allowed raw meat juices to come in contact with the final meal. Precautions that food workers should apply to reduce the risk of cross-contamination and infection by *E. coli* and *Campylobacter* include (i) storing ground beef products at temperatures $<40^\circ\text{F}$, (ii) using a thermometer to check the temperature of cooked beef to make sure it exceeds 160°F to kill pathogenic bacteria, (iii) storing or handling raw meats so blood or juices do not drip or contact RTE food, (iv) washing and sanitizing surfaces and utensils after preparing raw meats, and (v) washing hands after handling raw meats. *Salmonella* is more frequently associated with food worker outbreaks because it grows in more environmental niches than does *Campylobacter* and survives longer under adverse conditions (Table 2).

Listeria spp. do not normally colonize the human body. Because they are transient in the gastrointestinal system, *Listeria* organisms are more likely to be transferred to food workers through contact with raw foods. In a study of 44 food establishments, Kerr et al. (97) found that 12% of workers had low levels of *Listeria* contamination (usually *Listeria monocytogenes*), as indicated by palm print cultures before and after washing. Almost all of the workers did not wash their hands sufficiently to remove the *Listeria* (they did not use soap, washed for too short a time, or used a dirty hand towel). In two cases, the washing and drying process actually contaminated the hands; *Listeria* was not recovered from the prewashing palm prints. Various modeling scenarios of transfer from comminuted chicken contaminated with *L. monocytogenes* to ham slices have been developed using the hand with and without gloves and with and without washing. Results indicated that the highest risk corresponded to the use of the same gloves to handle contaminated meat and then sliced ham compared with the safer method of using different gloves to handle each product (163). Hand washing followed by bare hand contact with food was not sufficient to assure low risk of *L. monocytogenes* contamination. A combination of gloves and proper hand washing was the most effective procedure; only 250 of the 100,000 slices were simulated to be contaminated (0.25%). Even when gloves are used, hands should be properly washed with soap or with an antimicrobial product (65, 159, 163). Gill and Jones (61) found that transfer of marker bacteria from gloves was enhanced by surface wetness. According to Nel et al. (149), individuals working with raw meat in moist environments can expect contamination from zoonotic pathogens both from direct contact and from deboning equipment, especially soiled knives and conveyor belts, which are rarely cleaned. Pathogens can survive and some bacteria can multiply on these surfaces to sufficient numbers that can be transferred to food (82) and cause infection. If a *Listeria*-contaminated meat product is put through a slicing machine, the pathogen could spread to other products and to the delicatessen environment after attachment or could form a biofilm (98, 213).

Hands. Hand transfer has been identified as a significant mode of transmission for pathogens, but there are lim-

ited precise data confirming this scenario. Pether and Scott (165) found that even with high levels of *Salmonella* in convalescent patients, it was difficult to detect the pathogen through direct transfer from fingers to agar plates. However, when the patients used a finger-rinse technique and the culture was subsequently enriched, the detection rate increased to 9 in 30 attempts before hand washing and 1 in 30 attempts after hand washing. Unlike *Salmonella*, *Shigella*, and some viruses, several bacterial pathogens such as *Campylobacter*, *E. coli* O157:H7, and *Vibrio cholerae* are not transmitted easily from infected food workers through the fecal-oral route (66, 73, 136, 202, 203). This finding is contrary to what one would expect from organisms with low infective doses, such as *E. coli* and *Campylobacter*. The reasons are unclear but may be associated with the relatively few asymptomatic carriers in the community, although in surveys up to 13% of apparently well individuals excreted *Campylobacter* (94). Another factor may be that these organisms were excreted in stools for only relatively short periods of time, typically <10 days for *E. coli* (168) and <7 days for *Campylobacter* (94), although longer periods have been noted. 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 (191). Healthy carriers of *E. coli* O157:H7 and *E. coli* O157:H⁻ have been documented (15), but few large population studies have been conducted to determine the carriage rate for healthy adults, which is assumed to be low. In northern Italy, verotoxin-producing *E. coli* O157 was found in 4 individuals (1.1%) in a survey of 350 farm workers on 276 dairy farms and 50 abattoir employees (189). On a repeat examination of the positive farm workers 35 to 84 days later, all the workers were negative but the wife of one worker was positive for the same strain; all strains carried some virulence genes linked to disease. None of these persons had diarrhea, and these individuals could be considered short-term asymptomatic carriers who may have developed immunity. The infections were assumed to come from the association of these workers with cattle, although the workers did not drink raw milk. This prevalence of verotoxin-producing *E. coli* O157 is relatively high compared with that reported from other studies conducted in Canada and Switzerland. Secondary spread of *E. coli* O157:H7 is most likely after an outbreak situation, either food-borne or community, e.g., Orr et al. (154), Bruce et al. (20), and David et al. (44).

Once the food worker is infected, many factors affect the transmission of enteric pathogens, including the concentration and frequency of the infectious agent, survival of the pathogen on hands and in the environment, concentration deposited in the food, and degree of temperature abuse (for bacteria). Transfer can occur (i) directly from person to person (2, 43, 68, 155), (ii) indirectly in two stages from person to contact surface and from contact surface to person (71, 129, 170, 188), and (iii) indirectly from person to food and from food to person (11, 21, 22).

Inadequate hand washing by food workers was cited as a contributory factor in 31% of outbreaks occurring in

Washington State from 1990 to 1999 (40). Food workers have been observed to wash and dry hands but then wipe their clean hands on their dirty pants (128). A lack of effective barriers such as gloves, hand-held utensils, or deli papers was noted in outbreak reports implicating hands in the transmission of pathogens. The majority of the food worker-associated outbreaks reviewed by Greig et al. (66) and Todd et al. (202, 203) involved transmission of the pathogen to food by food workers' hands; hand contact was described as a factor in 40% of the 816 outbreaks, and the investigators specifically mentioned that the food worker was not wearing gloves in 1.3% of the outbreaks. Bare hand contact may have contributed to more outbreaks if gloves had been worn inconsistently, but data on wearing of gloves were not recorded during the investigations. Investigators also noted that functioning and readily accessible sink and toilet facilities with adequate supplies frequently were lacking.

Researchers have identified street vendors as the source of fecally contaminated foods, particularly in developing countries. These vendors are independent entrepreneurs with limited supervision or government oversight, and toilets, toilet paper, and clean water for hand washing often are not readily available. During a dysentery epidemic in Zambia, *Shigella dysenteriae* was transmitted person to person and from prepared foods sold by vendors (207). *Shigella* can survive for over 3 h on fingertips (84). In a multivariate analysis, HAV was associated with cross-border travel to Mexico by Hispanic children who ate food from a taco stand or a street vendor and who ate salad and/or lettuce during travel (216). In Accra, Ghana, most street vendors surveyed exhibited good hygienic behavior, but only 18% of them associated diarrhea with germs (141). Mesophilic bacteria were detected in 356 of 511 foods sampled (69.7%): 28 samples contained *Bacillus cereus* (5.5%), 163 contained *S. aureus* (31.9%), and 172 contained *Enterobacteriaceae* (33.7%). *Shigella sonnei* and enteroaggregative *E. coli* were isolated from macaroni, rice, and tomato stew, and *Salmonella* Arizonae was isolated from soup. The authors recommended that these food vendors should be educated in food hygiene, especially on the causes of diarrhea, the transmission of diarrheal pathogens, the handling of equipment and cooked food or other RTE foods such as fruit, hand washing practices, and environmental hygiene. Fecal coliform bacteria and *E. coli* were isolated from 48 and 17%, respectively, of market-vended beverages sold in Guatemala (193). There was a significant decrease in fecal coliform levels in samples from control vendors who were asked to use hand washing soap and sanitizers for equipment and containers. These situations illustrate that street-vended food often can be contaminated with pathogens when this food is prepared under unhygienic conditions and where temperature control is difficult to maintain. Health Canada (75) advised travelers that although eating food purchased from street vendors can enhance a cross-cultural experience, many of these vendors lack adequate sanitary facilities and proper refrigeration, increasing the risk for travelers' diarrhea.

Some operations and specific foods involving bare

hand contact are more likely to be vehicles for infection or intoxication, e.g., various types of vegetable, meat, and fruit salads associated with such activities as tearing lettuce; making guacamole or salsa; mixing ingredients; slicing, deboning, grating, or shredding meats; removing the casings from sausages and hams and handling these products while slicing; peeling and deveining shrimp; shelling eggs; and making sandwiches. Even minimal hand contact with moist surfaces such as tomato and pineapple slices, cut melon, lettuce, and mashed potatoes can result in significant pathogen transmission (40, 87, 100, 138). Garnishing food by placing boiled shrimp on top of a tossed salad, putting parsley into cooked food, and picking fresh basil demonstrate how even small operations can transmit pathogens and cause outbreaks (108, 112). Other actions less frequently thought of as hazardous but associated with outbreaks include handling of baked goods such as buns and cooked pasta products, preparing frosting for cakes, dipping donuts in glaze, mixing milk from powder, reconstituting orange juice from concentrate, using whipping cream and mousse ingredients for desserts, and using water and ice for beverages (66, 202, 203). Some activities resulted in both hands and arms being immersed in foods, followed by thorough mixing, which can result in widespread distribution of the pathogen in the food product. An estimate can be made of the pathogen level on hands that can lead to outbreaks. For example, a cake decorator came to work 1 day after she started developing gastroenteritis from a norovirus infection. She contaminated icing with the virus while mixing in the sugar water and subsequently infected an estimated 1,000 people (104). The infectious dose for norovirus is estimated to be between 10 and 100 particles (9, 29, 106, 204). The decorator wore long artificial nails, which would collect at least 0.1 mg of fecal material during the course of a working day. Because there may be 10^5 to 10^{11} viral particles per g (9), the load under the nails could be 10^1 to 10^7 , which is sufficient to contaminate the icing and infect 1,000 persons. Another cake-associated outbreak affecting as many as 2,700 people was caused by one or more ill employees applying strawberry filling by direct hand contact to 46 wedding cakes (57). The investigation revealed that cutting boards and utensils were not properly sanitized, employees did not wear gloves or use them correctly, and some employees wiped their hands on soiled uniforms.

Most microorganisms causing foodborne illness survive long enough on hands and surfaces for transfer to food or fellow workers. Although more attention may be paid to hygiene and cleanliness in most commercial operations, at times pathogens from personnel or food sources will contaminate the food preparation environment, including RTE food. Because transmission is much more efficient from wet hands, hand drying after washing has been strongly advocated to reduce cross-contamination (120, 127, 142, 156), and drying is best done with towels from a hands-free towel dispenser (72, 74).

Fingernails (long and artificial). Long, polished, chipped, and/or artificial nails can increase overall micro-

bial counts on hands by trapping fecal matter and food particles (107). McGinley et al. (135) measured the bacterial load on different parts of the hand. The subungual spaces (beneath the fingernails) had an average of 5.39 log CFU compared with 2.55 to 3.53 log CFU for other hand sites; *Staphylococcus epidermidis*, *Staphylococcus haemolyticus*, and *Staphylococcus hominis* were the most frequently isolated species. Other bacteria recovered from subungual spaces included gram-negative bacilli (*Pseudomonas* and coryneforms) and yeasts. Long and/or dirty fingernails have been implicated in fecal transmission of pathogens to food, as previously illustrated by the outbreak involving a cake decorator. When fingernails poke through toilet paper, fecal contamination occurs and normal washing is largely ineffective without the use of a fingernail brush (110, 111, 144). Bacteria and viruses are known to persist under fingernails (162, 182). This persistence obviously occurred in the cake-associated outbreak, where the decorator wore long artificial fingernails and decorated 80 cakes without using gloves after she became infected with a norovirus (104). The role of fingernails was less clear in a 1974 outbreak when 107 people became infected with HAV after eating cold sandwiches (107). The food worker stated that she cleaned her long fingernails with a nail-file or toothpick regularly, but she frequently touched her face with her hands and snacked on food ingredients. Complete removal of fecal material with toothpicks or files is not possible. Unfortunately, certain food handling activities often improperly rely on fingernail use for rapid task execution, such as shelling hard-boiled eggs, deveining shrimp, separating sliced meats and vegetables for sandwich making, or picking up chopped ingredients from a cutting board.

Clothing, rings, and other jewelry. Macintosh and Hoffman (120) compared the transfers of *Staphylococcus saprophyticus*, *E. coli*, *Pseudomonas aeruginosa*, *Klebsiella aerogenes*, *Streptococcus pyogenes*, and *Serratia marcescens* from a so-called donor fabric to hands. The organisms with the lowest inoculum (*S. saprophyticus* at 4.5×10^5 CFU/cm²) had the highest transfer rate per square centimeter (1.67%), and the organism with the highest inoculum (*S. pyogenes* at 3.9×10^7 CFU/cm²) had the lowest transfer rate per square centimeter (0.007%). The same transfer phenomenon was observed for fabric-to-fabric transfer and by other researchers (145, 148). However, all transfer scenarios were enhanced when either the donor fabric or the hands were moist (127). Rusin et al. (178) found greater transfer efficiency for gram-positive bacteria, gram-negative bacteria, and phage from phone receivers and faucets to hands than for other surfaces. However, for all three organism types, the inoculum size on the phone receiver or the faucet was about 3 to 5 log CFU less than that on all other surfaces.

Contamination of clothing with vomitus can result in person-to-person transmission, as occurred during an outbreak of norovirus among football players (12). During the game, several players on the visiting team suddenly began to vomit and have diarrhea after having contracted norovirus infection from eating boxed lunches contaminated by

a food worker. They continued to play despite their illnesses, and it was not possible for players on both teams to avoid contact with feces and vomitus through their hands and clothing. Two days later, some of the players on the home team also had similar gastrointestinal symptoms. Another report of likely clothing transmission was where a food worker infected hospital staff and patients with norovirus after the worker prepared a salad (113). The clothes that she wore to work had been contaminated when she was taking care of a child at home who had gastroenteritis. In a similar scenario, a food worker at a London hospital apparently contaminated turkey sandwiches, which led to salmonellosis in staff members and one patient (121). She also had been caring for a sick child but did not develop symptoms herself until after most of those infected had become ill. Laboratory analysis of stool specimens of the affected individuals revealed two strains of *Salmonella* Virchow, one from the baby and the other from the worker, indicating the occurrence of two outbreaks at about the same time. The worker probably contaminated the turkey meat during its preparation (kitchen hygiene was poor), but it is not clear whether the infective strains came from the turkey, a likely source for *Salmonella* Virchow. Food workers often eat meals prepared on-site and become ill along with other consumers. This episode demonstrates the importance of strain typing and molecular epidemiology, because multiple strains can be responsible for point-source outbreaks but may not have been identified in the past.

Although lower bacterial counts are typically found on women's skin than on men's skin, the presence of rings and other jewelry can increase total microbial counts on hands (79, 88, 123, 181, 190). However, in a more recent study of hand contamination as determined by the "glove juice" technique, wearing of a single plain finger ring did not increase the total bacterial load on the hands of health care workers nor was it associated with an increased rate of carriage of *S. aureus* or nonfermentative gram-negative rods (53). However, plain rings were associated with an increased rate of *Enterobacteriaceae* carriage. In addition to holding food debris and both resident and transient pathogens (25, 26, 122, 161, 210, 211), rings and other jewelry also can hold caustic industrial sanitizers and disinfectants and/or food allergens. These chemical compounds can be irritating and/or sensitizing and can react with metals in rings and watch bracelets (92, 160), resulting in allergic contact dermatitis (3, 56, 62, 122, 140, 205) and producing possible infection and colonization by enteric pathogens (126, 167). The resulting dermatitis or infection can then inhibit frequent and vigorous hand washing (105, 135). Rings and stones (precious, semiprecious, or glass) also can fall into food and become a physical hazard in addition to a personal loss (85). One person developed chronic eczema on the hand from handling foods such as raw meat and poultry and tomatoes, resulting in burning and stinging on the skin contact area (122). Although this condition may be rare, food workers are exposed to these types of foods on a continual basis and some may develop eczema. The condition itself does not contaminate foods unless the skin becomes infected, but it discourages any action that will cause

discomfort, including washing and drying of the affected areas.

AEROSOLS, FOMITES, SOIL, AND OTHER FORMS OF ENVIRONMENTAL CONTAMINATION

Aerosols. Aerosol transmission of norovirus has been documented in several scenarios, including outbreaks in hospital wards, residential institutions, and airplanes following vomiting incidences (29, 76, 186, 219). Patrons and staff in food service settings have been infected when a person vomited in another area of the establishment (125, 158). In an outbreak at a restaurant, Marks et al. (125) found an inverse correlation between the attack rate and the distance from a person who had vomited. Aerosols and direct contact with soiled clothing played a role in transmission between the two affected football teams already discussed (12). For pathogens resistant to stomach acids, vomitus that contaminates the food preparation area, sinks, restrooms, wash station, and food worker clothing (113) is an excellent transfer medium because of its thixotropic (sticky) nature. Fecally contaminated environmental surfaces have been associated with norovirus transmission in institutional outbreaks (183). Astroviruses, noroviruses, and rotaviruses were detected at multiple swab sites in a hospital pediatric unit in the United Kingdom (58). Toilet taps were the most contaminated items. Intermittently during the study, fecal samples from selected patients in the unit contained all three virus types.

Flushing toilets can disperse enteric pathogens into the restroom area, as demonstrated by experiments conducted by Barker and Jones (9). When 10^{10} CFU of *S. marcescens* and 10^{10} PFU of phage, as surrogates for bacterial and viral pathogens, respectively, present in diarrheal stools, were added to the sides and water of a toilet bowl, there was a 2- to 3-log reduction in the number of organisms in the bowl water after the first flush and a further 2-log reduction after the second flush. Both the bacteria ($1,370$ CFU/m³) and the phage ($2,420$ PFU/m³) were found in the air after the first flush, but almost twice as many virus particles as bacteria were detected. Sequential flushing resulted in further distribution of the organisms into the air, although the numbers declined after each flush. When the toilet bowl water was disinfected and neutralized before flushing, the number of bacteria released into the air was greatly reduced. Organisms were also found on the toilet seat, on a shelf behind the toilet, and in the cistern. The recess under the rim of the toilet bowl was heavily colonized. This area had previously been found to be where *Salmonella* persisted in domestic homes after a family member had recently suffered an attack of salmonellosis with acute diarrhea (8). Gerba et al. (59) also found that a persistent fraction of seeded bacteria was attached to the porcelain surface of the toilet and concluded that subsequent elution of these organisms was responsible for continuing residual contamination in the toilet bowl water. Closing the toilet lid had little effect on reduction of bacteria released into the air and persisting in the immediate environment because of the gaps between the top of the porcelain rim and the seat and

between the seat and the lid (9, 139). Thus, with up to 10^{11} virus particles in the bowl water after acute diarrhea, there is a substantial risk that pathogens such as norovirus will be distributed in the area by flushing and could infect subsequent toilet users and cleaners directly through inhalation or ingestion of the aerosolized droplets or through contamination of hands touching contaminated surfaces hours or even days later (10). Adding disinfectant to the bowl with diarrheic stools or vomitus before flushing may prevent further spread and reduce the chance of infection by subsequent toilet users. However, high numbers of enteric bacteria also can be found in urinals, on toilet seats, tap handles, and the inside handle of an entrance door, and in wash basin overflow areas (139).

Loosli (114) and Robertson et al. (174) indicated that dust in hospital wards and army barracks could become highly contaminated with pathogens associated with diseases of the respiratory tract, particularly streptococcal infections. Dispersion of these microorganisms into the air from floors, bedclothes, and clothing at the time of floor sweeping, bedmaking, and dressing resulted in a general contamination of the whole ward environment. Streptococci disseminated through the air as dustborne particles were deposited on furniture (e.g., bedside tables, nurses' desks, chairs, instruments, tables, and carts), food, and toys; the skin, hands, and clothes of the patients and hospital personnel (nurses, doctors, attendants, and visitors); and again on the floor and bed surfaces. Respiratory tract infections may be acquired from inhalation of these dustborne organisms or by direct transfer from a dusty surface to the nose and mouth by the hands.

Mills et al. (146) found that when surgeons were appropriately gowned and booted, wore a paper hood covering the head and neck, and double-gloved after hand scrubbing and then performed a mock hip joint operation, they transmitted twice as many *S. epidermidis* and other normal skin flora onto the operating table when they were sweating as when they performed under nonsweating conditions. Sweating was defined as beads of perspiration on the forehead after they exercised on a stationary bicycle. The authors speculated that the organisms could have been transferred by droplets or exfoliated skin flakes from exposed parts of the forehead, seepage through the hood or mask dripping directly onto the exposed area, escape through holes in the gloves or gowns, or tracking onto the forearm of the gown at the interface of gloves and gown, but these possibilities were not explored further. However, the authors raised concerns about staphylococcal infections encouraged by hot and other sweat-inducing conditions, because *S. aureus* and *S. epidermidis* are the organisms most responsible for joint replacement infections. Ritter et al. (173) found that airborne contamination increased when doors were left open and five people were present in the operating theatre. Wearing of a surgical mask had no effect on the overall operating room environmental contamination. Clearly, if infections can occur under operating room conditions, they are more likely to occur in hot kitchen environments with multiple people working together, airflows not designed for containment, hot employees tending

to wipe sweat off with arms and hands, and many opportunities to contaminate food contact surfaces with staphylococci.

Fomites and soil in food operations. During and after illness, enteric viruses are shed in large numbers in body secretions, including blood, feces, urine, saliva, and nasal fluid. Fomites (inanimate objects or substances capable of transferring microorganisms from one individual to another) become contaminated with virus by direct contact with body secretions or fluids and by contact with soiled hands. Bacteria and viruses are transmitted by large and small droplets generated when talking, sneezing, coughing, or vomiting (10, 70). Shaking a contaminated fomite such as a blanket or vacuuming a soiled carpet may disturb airborne pathogens that have settled (19). Once a fomite is contaminated, the transfer of infectious organisms may readily occur between inanimate and animate objects, or vice versa, and between two separate fomites. Fomites may be covered with a thin layer of undesirable organic matter, which affects transfer and survival of pathogens. In the food preparation and processing environments, these objects tend to be utensils and equipment covered in food soil. The microorganisms are protected from environmental stress and death by mucus, sputum, feces, blood, serum, or more likely food materials such as milk, albumin, or chicken broth (38, 51, 83, 117, 177, 192, 197). The buffering effects of these substances contribute to the long-term survival of organisms on food and household items, including improperly cleaned utensils and fabrics, where there are added factors of temperature, humidity, and bacterial competition. Although most bacteria are killed during dry conditions, any survivors, even among non-sporeformers, can revive when moisture is again present (197, 215). This revival was recently illustrated by large outbreaks of salmonellosis arising from chocolate crumb and peanut butter ingredients (32, 217). Some of these surviving organisms may be difficult to culture because of stress conditions and are thus called viable but not culturable (176). Organisms dried on environmental surfaces may remain protected over extended periods (51, 83, 117, 192, 197). *S. aureus* survives well in dust (109, 147), on fabrics (103, 133, 147), on tile or glass surfaces (117, 169), and on floor materials (7). *Salmonella* Enteritidis in minimally cooked scrambled eggs derived from infected shell eggs can survive for 24 h on a Formica surface (83). Other pathogens can survive even longer, e.g., *Enterococcus faecalis* for 5 days on hospital bed rails and *Enterococcus faecium* on counter tops for more than 7 days (152); both species were recoverable from hospital bed rails (after 24 h) and telephone handsets (after 60 min). Managers of food service operations should not re-serve food, particularly to or from patients in medical isolation or quarantine or patients who are highly immunocompromised, because pathogens have the ability to survive on dry surfaces and on the outside of packaging (Table 1) (73, 209). Various microorganisms also can remain viable on stainless steel, ceramic, and glass surfaces; e.g., *E. coli* survived for 60 days on stainless steel (83, 134, 169).

Contamination from community environments. Fecal and other human contamination is relatively prevalent in our society, as demonstrated in a 5-year study of hygiene in shopping centers, daycare operations, offices, restaurants, theatres, and airports and on personal items in four U.S. cities (171). Total and fecal coliforms were detected on 20 and 7% of the surfaces in these facilities, respectively. Evidence of mucus, saliva, sweat, and urine was found on up to 15% of the sites. When contamination of surfaces used by the public was simulated using an invisible fluorescent tracer, contamination from these surfaces was transferred to 86% of exposed individual's hands. Five office volunteers were sampled 20 min after they arrived home after work, and the tracer was found on volunteers' hands, backpacks, keys, purses, doorknobs, light switches, counter tops, and kitchen appliances. A study of United Kingdom households revealed that diaper changing occurred mainly in living rooms, and evidence of fecal contamination was found in 12% of living room samples and 15% of bathroom samples (42). Fecal contamination also was present on taps and soap dispensers in kitchens, although washing hands with soap after diaper changing reduced the risk of contamination.

Soil on surfaces can protect microbes from environmental stress and assist in transfer of pathogens within the food plant or kitchen. Soil type, degree of wetness, contamination level, contact time, and soil characteristics such as texture, smoothness, stickiness, and absorbency are important determinants of transfer potential. Surface tension of liquids and surface free energy will influence the tenacity of soils that cling to or release from surfaces. Teixeira et al. (200) found that surface hydrophobicity and roughness are key factors in determining the extent of adhesion by *Salmonella* Typhimurium to surfaces. Stainless steel was colonized to the greatest extent followed by marble and, at almost to the same extent, granite. All three materials are commonly used for work benches in kitchens in many countries. When a liquid with high surface tension is present on a surface, it will more easily transfer to surfaces of lower energy, allowing a liquid to spread completely over a surface. A textured surface tends to hold liquid contaminants better than smooth surfaces. Higher pressures tend to increase contact area and transfer, such as during hand slicing of meat (213). Microbial adhesion properties and survival also are important. Pathogen species and even strains may have different adhesion characteristics. In a study of the adherence of *Salmonella* Enteritidis (4 strains) and *L. monocytogenes* (10 strains) to stainless steel 304, marble, granite, glass, two kinds of silestone, and polypropylene from a bowl and a cutting board, intrinsic factors such as cell envelopes, adhesins, cell wall proteins, and extracellular polymers were more critical than the contact surfaces (201). The smaller the agent, e.g., a virus compared with a parasite, the more likely it can lodge in small surface scores or imperfections on the contact surface or skin. However, the presence of antimicrobials, including cleaning compounds, will impact survival and transfer. Bidawid et al. (16) found that when fingertips contaminated with HAV were sanitized with alcohol sanitizer (65 or 75% ethanol concentrations) before lettuce was touched, transfer

of the virus was reduced to 0.3 to 0.6% compared with 9% transfer from unwashed fingertips.

S. aureus, *Pseudomonas*, *Bacillus* spp., and enterococci have been isolated from the mouse, mouse pad, and keyboard of computers in health care settings (124, 179). Keyboards and other office equipment should not be touched by persons caring for patients or persons working with food unless there has been effective hand washing or the equipment is covered with a protective sleeve that can be easily cleaned and disinfected. Thus, keyboards should be disinfected daily or when visibly soiled or if they become contaminated with blood, other body excretions, or food particles. Norovirus can be consistently transferred from fingers to melamine surfaces, taps, door handles, and telephone receivers, with sequential transfers to up to seven clean surfaces (10). In a study in which a bacteriophage was used as a model enteric virus, approximately 10^7 PFU was applied to hands of volunteers or door handles (172). After the volunteers touched these surfaces, at least 14 people could be sequentially contaminated by horizontal spread from touching the same door handle, and successive transmission from one person to another could be followed up to the sixth contact person. The phage was reisolated after 24 h from the hands of individuals, even after normal activities and hand cleaning. Contact transmission may occur from surfaces to many users over long periods of time because viruses can remain stable for weeks or even years under favorable conditions (27). Rotavirus in fecal matter can survive on nonporous surfaces (steel, plastic, and glass) for days at low to medium relative humidity (5 to 50%), but its survival on cardboard, paper currency, and cloth was very limited based on experimental contamination studies (183). However, pathogens can survive on coins for several days (89), and *E. coli* (including enterotoxigenic *E. coli*), *Salmonella*, and *Vibrio* have been recovered from currency notes found in butcher shops and at fishmongers in Myanmar (99). The level of contamination of paper currency may depend upon how long the notes have been in circulation; paper currency in Nigeria and the Philippines was withdrawn after notes were found to be heavily contaminated with microorganisms, including cysts and ova of intestinal parasites (143). In a limited U.S. study, 94% of 1-dollar bills yielded at least one bacterial isolate, and 7% of the bills were contaminated with either *S. aureus* or *Klebsiella pneumoniae* (166). These data indicate that foodborne pathogens can be transferred to currency (coins and paper) when hands are not properly washed and dried, and some pathogens can survive on currency and contaminate other users. Effective hand washing must be stressed when currency has been handled and before food is touched in food service establishments, particularly where RTE foods are being served.

SURVIVAL OF PATHOGENS

Hands. The survival of pathogens on hands is outlined in Table 2. The soil matrix, relative humidity, and temperature all influence pathogen survival. Death rates are different for viruses and bacteria and depend on factors associated with specific virus groups. Naked viruses such as

norovirus, rhinovirus, and enterovirus are more stable on skin than are viruses with envelopes, such as the influenza virus (183). Declines can be rapid on hands, but most pathogens that cause foodborne illness survive long enough on hands and contact surfaces to allow some transfer to food or fellow workers. *Salmonella* can survive for several hours on fingertips, but hand washing followed by drying with paper towels effectively reduced the risk of transmission to food (164). *S. aureus* has adapted to drying, survives well on the skin (103), is difficult to remove, and can be dispersed through shedding of skin squames (150). *S. aureus* has a continual source because it can be resident in the anterior nares and the urogenital tract, areas frequently touched by fingers, and is transient under fingernails (147, 150). *S. aureus* also can colonize wounds, and pus spread over the skin can become a major source of contamination. Some agents such as enteroviruses are resistant to physicochemical inactivation and must be removed by vigorous friction applied during hand washing or hand drying (187).

Food preparation environment. A pathogen's opportunity for infectivity depends partially upon its ability to survive on surfaces, including foods. Herrmann and Cliver (78) noted a 2- to 3-log reduction in the titer of coxsackievirus A9 inoculated into raw ground beef during storage at 4°C for 14 days. This storage period is longer than that usually used in either domestic or commercial settings, but the persistence of the enteric virus indicates that raw ground beef contaminated by an infected handler could be a vehicle for transmission if the beef were eaten uncooked, e.g., as steak tartare. Cliver et al. (37) studied the potential for enteric virus survival in low-moisture foods developed for space flights by mimicking contamination by a food worker during final packaging. The foods tested were of many types, including bacon, cheese sandwiches, spaghetti, and banana pudding. Each food was freeze-dried, inoculated with viruses, sealed under vacuum in plastic pouches, and stored at room temperature or 5 or 12°C. The enteric virus types were reovirus, poliovirus types 1 and 2, and echovirus type 6. Although reoviruses were not capable of persisting more than 1 day in these foods, the enteroviruses showed great stability, persisting up to 2 weeks at room temperature and up to 2 months at refrigeration temperatures. Interactions between temperature, pH, and protein and salt content also influenced virus survival in the low-moisture foods.

Kramer et al. (101), Sattar et al. (184), and Rzezutka and Cook (180) reviewed data indicating that pathogens causing gastroenteritis can survive on surfaces for several hours or days, especially on moist surfaces (Table 2). Rusin et al. (178) sampled volunteers' hands after they touched surfaces contaminated with *Micrococcus luteus*, *Serratia rubidea*, and phage PRD-1. Activities included wringing out a dishcloth or sponge, turning off a faucet, cutting up a carrot, making hamburger patties, holding a phone receiver, and removing laundry from the washing machine. Transfer efficiencies for the phone receiver and faucet were 38 to 65% and 27 to 40%, respectively. A study in Ohio of 70 food service operations revealed that 86% harbored enteric bacteria on cutting boards and other food prepara-

tion surfaces. These bacteria were found on door handles of coolers and freezers in 57.1% of establishments and on hand washing faucets in 52.9% of the operations (96). Paulson (159) found that when gloved hands were in contact for 5 to 10 s with surfaces such as cutting boards and door knobs contaminated with feline calicivirus at 5.9 log particles, 4.7 to 5.4 log particles were recovered from hands. The highest bacterial transfer rates from fomites to hands were found with hard nonporous surfaces. Clay et al. (36) found that feline calicivirus can survive sufficiently long on fomites (computer and telephone compound material and metals) for transfer of this norovirus from person to person. Norovirus survived in carpets and toilet facilities for more than 1 day and infected concert hall attendees (52). Survival time for virus on carpets may be substantially longer, possibly at least 12 days, based on reports of illnesses from carpet removers in a hospital ward (33). Pathogens tend to survive longer on surfaces such as ceramic tile, steel, dust, glass, and plastic than on hands (Table 2). Even with low transfer rates, the numbers of bacteria transferred to the hands were still high (up to 10⁶ cells). Transfer of bacteria from the fingertips to lips is similar to that observed from hard surfaces to hands.

During regular food operations, bacteria and viruses can be transferred throughout the preparation environment. In various studies, relative humidity has been one of the most significant determinants for bacterial survival. In general, survival is highest at low relative humidity followed by very high relative humidity. The intermediate range (around 50%) is most detrimental to the majority of bacterial species (50, 133, 134, 147). The same results have been found for various viruses (49, 115). Typically, HAV and rotavirus are stabilized at low relative humidity, and enteroviruses are stabilized at higher humidities (27). This difference may explain the uneven survival of feline calicivirus in different foods: 1% in strawberries compared with 20% in lettuce and 43% in ham after 30 min (130). Microorganisms from the human intestinal tract and enteric pathogens have adapted to moist conditions and die or grow slowly in a dry environment (35, 220). Human skin bacteria and *S. aureus* are adapted to a dryer existence (134); consequently *S. aureus* will grow in low-moisture food that would not support the growth of other food pathogens. This organism will grow at low water activities (a_w) of 0.85 to 0.99 but will not produce enterotoxin until at least an a_w of 0.87 (86). This ability may be one reason why *S. aureus* and *K. pneumoniae* survive better on fingertips than do *P. aeruginosa* and *E. coli*, and strains recently isolated from hospital settings were more resistant on skin than were stored culture-collection strains (54). Survival of other transient organisms on skin such as *Campylobacter jejuni*, *E. faecalis*, *K. aerogenes*, and *S. pyogenes* is lower than that of a skin resident such as *S. saprophyticus* (24, 28, 38, 120, 152). Depending on the initial dose in the aqueous medium (broth, saline, or blood) and the hand or environmental surface type, there was sometimes a 2- to 5-log die-off in the first 5 to 10 min on contact surfaces (particularly of *Campylobacter*), which continued progressively until the organism could not be detected.

A risk-based approach to understanding the dynamics of contamination and transfer starts from the principle that pathogens are continuously introduced into homes and work places through people, food, water, surfaces of inanimate materials, animals such as pets and insects, and air. Inadequate disposal of human and animal excreta and contact with raw foods of animal or plant origin (e.g., carcasses or manure applied to crops) increase this risk. Sites where moisture remains or where stagnant water accumulates, such as sinks, toilets, drains, other standing water, and cleaning cloths, can support microbial growth and become a source of infection. The risk of exposure for individuals, both food workers and consumers, can be assessed by determining the frequency that pathogens occur, the potential for pathogen occurrence on hands, hand and food contact surfaces, laundry, and other reservoirs, and the potential for pathogen transfer (18). Thus, there are multiple issues involved in contamination, transfer of pathogens, and risk of illness that require expert knowledge and perceptive oversight. Individual employees cannot be expected to understand all the food safety issues associated with producing or preparing a food. However, owners and managers of food operations must understand the issues and provide the essential areas of expertise, either themselves or through hired consultants, to make the workplace as free from contamination sources as possible. Otherwise, unsatisfactory inspection reports, outbreaks, or food recalls are to be expected.

Managerial policies in food establishments and employee errors have been identified as major risk factors in outbreaks. In one example, ill employees in a Minnesota establishment prepared salads and infected 220 patrons at eight banquets with norovirus. A new policy of sick leave without pay had been initiated by new managers 1 month previously, and the workers did not want to lose wages (218). In another scenario in Washington State in 1990, bad management practices resulted in 143 patrons becoming ill with norovirus (40). A food worker called in sick at a fast food facility, but the manager demanded that the worker report for duty because he suspected the employee was lying. Upon arrival, the worker was ill but was asked by the manager to prepare lettuce and tomato dishes before going home. The worker used his bare hands. The manager had violated company policy that required all ill employees be excluded from work and did not enforce requirements for proper hand washing, but the work demands were considered paramount. Management should recognize that contamination spread through improper hygiene can be costly. In one norovirus outbreak at a hospital, the expenses were estimated over 3 months at more than \$650,000 to halt the infection from spreading among patients, staff, and visitors (90). These costs included extra cleaning supplies, staff sick leave, diagnostic tests, replacement staff and salaries, and lost revenue from closed beds. Terminal disinfection cost \$96,000, and to eliminate any remaining viruses all disposable supplies in infected areas were removed and replaced with fresh ones at an expense of more than \$53,000. For a management policy in food service operations to be successful, a well-trained certified kitchen manager must set

an example for employees, although even this approach will not prevent ill employees from working (77). Effective hand washing, no bare hand contact with RTE food, and use of other barriers to pathogen contamination are the topics of the next paper in this series of food worker outbreaks and their prevention.

ACKNOWLEDGMENTS

The authors appreciate the critical reviews of 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) and Ruth L. Petran (Corporate Scientist, Food Safety, Ecolab Inc., St. Paul, MN), who gave useful comments and new material to improve the manuscript.

REFERENCES

1. Abad, F. X., A. Bosch, and R. M. Pinto. 1994. Survival of enteric viruses on environmental fomites. *Appl. Environ. Microbiol.* 60: 3704–3710.
2. Adams, B. G., and T. J. Marrie. 1982. Hand carriage of aerobic gram-negative rods by health care personnel. *J. Hyg. (Lond.)* 89: 23–31.
3. Adams, R. M. 1990. Occupational skin disease, 2nd ed. W. B. Saunders, Philadelphia.
4. Alterkruse, S. F., S. Yang, B. B. Timbo, and F. J. Angulo. 1999. A multi-state survey of consumer food-handling and food-consumption practices. *Am. J. Prev. Med.* 16:216–221.
5. Andrews, J. 1934. The retention of *Endamoeba histolytica* cysts under finger-nails. *Am. J. Trop. Med.* s1–14:439–441.
6. Ansari, S. A., S. A. Sattar, V. S. Springthorpe, G. A. Wells, and W. Tostowaryk. 1988. Rotavirus survival on human hands and transfer of infectious virus to animate and nonporous inanimate surfaces. *J. Clin. Microbiol.* 26:1513–1518.
7. Ayliffe, G. A., B. J. Collins, and E. J. L. Lowbury. 1967. Ward floors and other surfaces as reservoirs of hospital infection. *J. Hyg. Camb.* 65:515–536.
8. Barker, J., and S. F. Bloomfield. 2000. Survival of *Salmonella* in bathrooms and toilets in domestic homes following salmonellosis. *J. Appl. Microbiol.* 89:137–144.
9. Barker, J., and M. V. Jones. 2005. The potential spread of infection caused by aerosol contamination of surfaces after flushing a domestic toilet. *J. Appl. Microbiol.* 99:339–347.
10. Barker, J., I. B. Vipond, and S. F. Bloomfield. 2004. Effects of cleaning and disinfection in reducing the spread of norovirus contamination via environmental surfaces. *J. Hosp. Infect.* 58:42–49.
11. Bean, N. H., and P. M. Griffin. 1990. Foodborne disease outbreaks in the United States, 1973–1987: pathogens, vehicles, and trends. *J. Food Prot.* 53:804–817.
12. Becker, K. M., J. N. MacCormack, C. L. Moe, and K. L. Southwick. 2000. Transmission of Norwalk virus during football game. *N. Engl. J. Med.* 343:1223–1227.
13. Belson, K., and K. Fahim. 6 October 2007. After extensive beef recall, Topps goes out of business. *New York Times*. Available at: <http://www.nytimes.com/2007/10/06/us/06topps.html>. Accessed 5 February 2008.
14. Berrang, M. E., J. S. Bailey, S. Alterkruse, B. Patel, W. Shaw, R. J. Meinersmann, and P. J. Cray. 2007. Prevalence and numbers of *Campylobacter* on broiler carcasses collected at rehang and post-chill in 20 U.S. processing plants. *J. Food Prot.* 70:1556–1560.
15. Bettelheim, K. A. 2001. Enterohaemorrhagic *Escherichia coli* O157:H7: a red herring? *J. Med. Microbiol.* 50:201–202.
16. Bidawid, S., J. M. Farber, and S. A. Sattar. 2000. Contamination of foods by food handlers: experiments on hepatitis A virus transfer to food and its interruption. *Appl. Environ. Microbiol.* 66:2759–2763.
17. Bidawid, S., N. Malik, O. Adegbunrin, S. A. Sattar, and J. M. Farber. 2004. Norovirus cross-contamination during food handling and

- interruption of virus transfer by hand antisepsis: experiments with feline calicivirus as a surrogate. *J. Food Prot.* 67:103–109.
18. Bloomfield, S. F., and E. A. Scott. 2003. Developing an effective policy for home hygiene: a risk-based approach. *Int. J. Environ. Health Res.* 13(Suppl. 1):S57–S66.
 19. Boone, S. A., and C. P. Gerba. 2007. Significance of fomites in the spread of respiratory and enteric viral disease. *Appl. Environ. Microbiol.* 73:1687–1696.
 20. Bruce, M. G., M. B. Curtis, M. M. Payne, R. K. Gautom, E. C. Thompson, A. L. Bennett, and J. M. Kobayashi. 2003. Lake-associated outbreak of *Escherichia coli* O157:H7 in Clark County, Washington, August 1999. *Arch. Pediatr. Adolesc. Med.* 157:1016–1021.
 21. Bryan, F. L. 1972. Emerging foodborne problems. II. Factors that contribute to outbreaks and their control. *J. Milk Food Technol.* 35: 632–638.
 22. Bryan, F. L. 1978. Factors that contribute to outbreaks of foodborne disease. *J. Food Prot.* 41:816–827.
 23. Bryan, F. L., and M. P. Doyle. 1995. Health risks and consequences of *Salmonella* and *Campylobacter jejuni* in raw poultry. *J. Food Prot.* 58:326–344.
 24. Burtenshaw, J. M. L. 1938. The mortality of the haemolytic *Streptococcus* on the skin and on other surfaces. *J. Hyg.* 38:575–586.
 25. Campolmi, P., P. Lombardi, T. Lotti, and A. Sertoli. 1982. Immediate and delayed sensitization to garlic. *Contact Dermatitis* 8:352–353.
 26. Carmichael, A. J., I. S. Foulds, and C. Y. Tan. 1988. Allergic contact dermatitis from potato flesh. *Contact Dermatitis* 20:64–65.
 27. Carter, M. J. 2005. Enterically infecting viruses: pathogenicity, transmission and significance for food and waterborne infection. *J. Appl. Microbiol.* 98:1354–1380.
 28. Casewell, M. W., and N. Desai. 1983. Survival of multiply-resistant *Klebsiella aerogenes* and other gram-negative bacilli on finger-tips. *J. Hosp. Infect.* 4:350–360.
 29. Caul, E. O. 1994. Small round structured viruses: airborne transmission and hospital control. *Lancet* 343:1240–1242.
 30. Centers for Disease Control. 1990. Foodborne hepatitis A—Alaska, Florida, North Carolina, Washington. *Morb. Mortal. Wkly. Rep.* 39: 228–232.
 31. Centers for Disease Control and Prevention. 1993. Update: multi-state outbreak of *Escherichia coli* O157:H7 infections from western United States 1992–1993. *Morb. Mortal. Wkly. Rep.* 42:258–263.
 32. Centers for Disease Control and Prevention. 2007. Multistate outbreak of *Salmonella* serotype Tennessee infections associated with peanut butter—United States, 2006–2007. *Morb. Mortal. Wkly. Rep.* 56:521–524.
 33. Cheesbrough, J. S., L. Barkess-Jones, and D. W. Brown. 1997. Possible prolonged environmental survival of small round structured viruses. *J. Hosp. Infect.* 34:325–326.
 34. Chen, Y., K. M. Jackson, F. P. Chea, and D. W. Schaffner. 2001. Quantification and variability analysis of bacterial cross-contamination rates in common food service tasks. *J. Food Prot.* 64:72–80.
 35. Christian, J. H. B., and W. J. Scott. 1953. Water relations of salmonellae at 30°C. *Aust. J. Biol. Sci.* 6:565–573.
 36. Clay, S., S. Maherchandani, Y. S. Malik, S. Yashpal, and S. M. Goyal. 2006. Survival on uncommon fomites of feline calicivirus, a surrogate of noroviruses. *Am. J. Infect. Control* 34:41–43.
 37. Cliver, D. O., K. D. Kostenbader, and M. R. Vallenias. 1970. Stability of viruses in low moisture foods. *J. Milk Food Technol.* 33: 484–491.
 38. Coates, D., D. N. Hutchinson, and F. J. Bolton. 1987. Survival of thermophilic campylobacters on fingertips and their elimination by washing and disinfection. *Epidemiol. Infect.* 99:265–274.
 39. Cogan, T. A., S. F. Bloomfield, and T. J. Humphrey. 1999. The effectiveness of hygiene procedures for prevention of cross-contamination from chicken carcasses in the domestic kitchen. *Lett. Appl. Microbiol.* 29:354–358.
 40. Conference for Food Protection. 1 February 2002. Preventing contamination from hands, section 3-301.11. Report of the Council III Bare Hand Contact Committee. Conference for Food Protection no. 00-01-07. Available at: <http://www.foodprotect.org/doc/III-27a%20Bare%20Hand%20contact%20report.doc>. Accessed 8 April 2008.
 41. Cooke, E. M., A. S. Edmondson, and W. Starkey. 1981. The ability of strains of *Klebsiella aerogenes* to survive on the hands. *J. Med. Microbiol.* 14:443–450.
 42. Curtiss, V., A. Biran, K. Deverell, C. Huges, K. Bellamy, and B. Drasar. 2003. Hygiene in the home: relating bugs and behaviour. *Soc. Sci. Med.* 57:657–672.
 43. D'Alessio, D. J., J. A. Peterson, C. R. Dick, and E. C. Dick. 1976. Transmission of experimental rhinovirus colds in volunteer married couples. *J. Infect. Dis.* 133:28–36.
 44. David, S. T., L. MacDougall, K. Louie, L. McIntyre, A. M. Pacagnella, S. Schleicher, and A. Hamade. 2004. Petting zoo-associated *Escherichia coli* O157:H7—secondary transmission, asymptomatic infection, and prolonged shedding in the classroom. *Can. Commun. Dis. Rep.* 30:173–180.
 45. Davis, J. G., J. R. Blake, D. J. White, and C. M. Woodall. 1969. The types and numbers of bacteria left on hands after normal washing and drying by various methods. *Med. Officer* 31:235–238.
 46. DeBoer, E., and M. Hahne. 1990. Cross contamination with *Campylobacter jejuni* and *Salmonella* spp. from raw chicken products during food preparation. *J. Food Prot.* 53:1067–1068.
 47. de Wit, J. C., G. Broekhuizen, and E. H. Kampelmacher. 1979. Cross-contamination during the preparation of frozen chickens in the kitchen. *J. Hyg. (Lond.)* 83:27–32.
 48. Dharod, J. M., R. Perez-Escamilla, S. Paciello, K. Venkitanarayanan, A. Bermudez-Millan, and G. Damio. 2007. Critical control points for home prepared ‘chicken and salad’ in Puerto Rican households. *Food Prot. Trends* 27:544–552.
 49. Dixon, G. J., R. W. Sidwell, and E. McNeil. 1966. Quantitative studies on fabrics as disseminators of viruses. II. Persistence of poliomyelitis virus on cotton and wool fabrics. *Appl. Microbiol.* 14: 183–188.
 50. Dunklin, E. W., and T. T. Puck. 1948. The lethal effect of relative humidity on air-borne bacteria. *J. Exp. Med.* 87:87–101.
 51. Elmros, T. 1977. Survival of *Neisseria gonorrhoeae* on surfaces. *Acta Dermato-venereol.* 57:177–180.
 52. Evans, M. R., R. Meldrum, W. Lane, D. Gardner, C. D. Ribeiro, C. I. Gallimore, and D. Westmoreland. 2002. An outbreak of viral gastroenteritis following environmental contamination at a concert hall. *Epidemiol. Infect.* 129:355–360.
 53. Fagernes, M., E. Lingaas, and P. Bjark. 2007. Impact of a single plain finger ring on the bacterial load on the hands of healthcare workers. *Infect. Control Hosp. Epidemiol.* 28:1191–1195.
 54. Filho, P. P. G., M. Stumpf, and C. L. Cardoso. 1985. Survival of gram-negative and gram-positive bacteria artificially applied on the hands. *J. Clin. Microbiol.* 21:652–653.
 55. Fischer, A. R. H., A. E. I. De Jong, E. D. Van Asselt, R. De Jonge, L. J. Frewer, and M. J. Nauta. 2007. Food safety in the domestic environment: an interdisciplinary investigation of microbial hazards during food preparation. *Risk Anal.* 27:1065–1082.
 56. Fisher, A. A., and F. Stengel. 1977. Allergic occupational hand dermatitis due to calf’s liver. An urticarial “immediate” type hypersensitivity. *Cutis* 19:561–565.
 57. Friedman, D. S., D. Heisey-Grove, F. Argyros, E. Berl, J. Nsubuga, T. Stiles, J. Fontana, R. S. Beard, S. Monroe, M. E. McGrath, H. Sutherland, R. C. Dicker, A. DeMaria, and B. T. Matyas. 2005. An outbreak of norovirus gastroenteritis associated with wedding cakes. *Epidemiol. Infect.* 133:1057–1063.
 58. Gallimore, C. I., C. Taylor, A. R. Genney, A. J. Cant, A. Galloway, M. Iturriza-Gomara, and J. J. Gray. 2006. Environmental monitoring for gastroenteric viruses in a pediatric primary immunodeficiency unit. *J. Clin. Microbiol.* 44:395–399.
 59. Gerba, C. P., C. Wallis, and J. L. Melnick. 1975. Microbiological hazards of household toilets: droplet production and the fate of residual organisms. *Appl. Environ. Microbiol.* 30:229–237.
 60. Gerner-Smidt, P., and J. M. Whichard. 2007. Foodborne disease trends and reports. *Foodborne Pathog. Dis.* 4:1–4.

61. Gill, C. O., and T. Jones. 2002. Effects of wearing knitted or rubber gloves on the transfer of *Escherichia coli* between hands and meat. *J. Food Prot.* 65:1045–1048.
62. Goh, C. L. 1989. An epidemiological comparison between occupational and non-occupational hand eczema. *Br. J. Dermatol.* 120: 77–82.
63. Gorman, R., S. Bloomfield, and C. C. Adley. 2002. A study of cross-contamination of food-borne pathogens in the domestic kitchen in the Republic of Ireland. *Int. J. Food Microbiol.* 76:143–150.
64. Gormley, F. J., M. MacRae, K. J. Forbes, I. D. Ogden, J. F. Dallas, and N. J. C. Strachan. 2008. Has retail chicken played a role in the decline of human campylobacteriosis? *Appl. Environ. Microbiol.* 74:383–390.
65. Green, L. R., V. Radke, R. Mason, L. Bushnell, D. W. Reimann, J. C. Mack, M. Motsinger, T. Stigger, and C. A. Selman. 2007. Factors relating to food worker hand hygiene practices. *J. Food Prot.* 70: 661–666.
66. Greig, J. D., E. C. D. Todd, C. A. Bartleson, and B. Michaels. 2007. Outbreaks where food workers have been implicated in the spread of foodborne disease. Part 1. Description of the problem, methods and agents involved. *J. Food Prot.* 70:1752–1761.
67. Griffiths, P. L., and R. W. Park. 1990. Campylobacters associated with human diarrhoeal disease. *J. Appl. Bacteriol.* 69:281–301.
68. Gwaltney, J. M., Jr., P. B. Moskalski, and J. O. Hendley. 1978. Hand-to-hand transmission of rhinovirus colds. *Ann. Intern. Med.* 88:463–467.
69. Gyles, C. L. 2007. Shiga toxin-producing *Escherichia coli*: an overview. *J. Anim. Sci.* 85(Suppl. E):E45–E62.
70. Hamburger, M., Jr., and O. H. Roberson. 1948. Expulsion of group A hemolytic streptococci in droplet nuclei by sneezing, coughing, and talking. *Am. J. Med.* 4:690–701.
71. Hare, R., and M. Ridley. 1958. Further studies on the transmission of *Staph. aureus*. *Br. Med. J.* 11:69–73.
72. Harrison, W. A., C. J. Griffith, T. Ayers, and B. Michaels. 2003. Bacterial transfer rates and cross-contamination potential associated with paper towels dispensing. *Am. J. Infect. Control* 31:387–392.
73. Harrison, W. A., C. J. Griffith, D. Tennant, and A. C. Peters. 2001. Incidence of *Campylobacter* and *Salmonella* isolated from retail chicken and associated packaging in South Wales. *Lett. Appl. Microbiol.* 33:450–454.
74. Hattula, J. L., and P. E. Stevens. 1997. A descriptive study of the handwashing environment in a long-term care facility. *Clin. Nurs. Res.* 6:363–374.
75. Health Canada. 13 March 2007. Gastrointestinal illness while travelling. Public Health Agency of Canada. Available at: http://www.phac-aspc.gc.ca/tmp-pmv/travel/gastro_e.html. Accessed 17 June 2008.
76. Hedberg, C. W., and M. T. Osterholm. 1993. Outbreaks of food-borne and waterborne viral gastroenteritis. *Clin. Microbiol. Rev.* 6: 199–210.
77. Hedberg, C. W., S. J. Smith, E. Kirkland, V. Radke, T. F. Jones, C. A. Selman, and the EHS-Net Working Group. 2006. Systematic environmental evaluations to identify food safety differences between outbreak and nonoutbreak restaurants. *J. Food Prot.* 69: 2697–2702.
78. Herrmann, J. E., and D. O. Cliver. 1973. Degradation of coxsackievirus type A9 by proteolytic enzymes. *Infect. Immun.* 7:513–517.
79. Hoffman, P. N., E. M. Cooke, M. R. McCarville, and A. M. Emmerston. 1985. Micro-organisms isolated from skin under wedding rings worn by hospital staff. *Br. Med. J.* 290:206–207.
80. Holmberg, S. D., and P. A. Blake. 1984. Staphylococcal food poisoning in the United States. New facts and old misconceptions. *JAMA* 251:487–489.
81. Humbert, F., J. J. Carramifiana, F. Lalande, and G. Salvat. 1997. Bacteriological monitoring of *Salmonella enteritidis* carrier birds after decontamination using enrofloxacin, competitive exclusion and movement of birds. *Vet. Rec.* 141:297–299.
82. Humphrey, T. 2001. The spread and persistence of *Campylobacter* and *Salmonella* in the domestic kitchen. *J. Infect.* 43:50–53.
83. Humphrey, T. J., L. W. Martin, and A. Whitehead. 1994. Contamination of hands and work surfaces with *Salmonella enteritidis* PT4 during the preparation of egg dishes. *Epidemiol. Infect.* 113:403–409.
84. Hutchinson, R. I. 1956. Some observations on the method of spread of Sonne dysentery. *Mon. Bull. Minist. Health Public Health Lab. Serv.* 15:110–118.
85. Hyman, F. N., K. C. Klontz, and L. Tollefson. 1993. Eating as a hazard to health: preventing, treating dental injuries caused by foreign objects in food. *J. Am. Dent. Assoc.* 124:65–69.
86. International Commission on Microbiological Specifications for Foods. 2003. Microorganisms in foods. 5. Characteristics of microbial pathogens. Kluwer Academic–Plenum Publishers, New York.
87. Iversen, A. M., C. L. Bartlett, W. D. Cubitt, M. Gill, and D. A. McSwiggan. 1987. Two outbreaks of foodborne gastroenteritis caused by a small round structured virus: evidence of prolonged infectivity in a food handler. *Lancet* ii:556–558.
88. Jacobson, G., J. E. Thiele, J. H. McCune, and L. D. Farrell. 1985. Handwashing: ring-wearing and number of microorganisms. *Nurs. Res.* 34:186–188.
89. Jiang, X., and M. P. Doyle. 1999. Fate of *Escherichia coli* O157:H7 and *Salmonella* Enteritidis on currency. *J. Food Prot.* 62:805–807.
90. Johnson, C., T. Perl, H. Qiu, J. Ticehurst, C. Dickson, P. Rosenbaum, P. Lawson, A. Stokes, C. Lowenstein, M. Kaminsky, S. Cosgrove, and K. Green. 31 August 2007. Norwalk virus: “cruise ship” illness challenging and costly to hospitals, too. *Sci. Daily*. Available at: <http://www.sciencedaily.com/releases/2007/08/070829162751.htm>. Accessed 5 September 2007.
91. Jørgensen, F., R. Bailey, S. Williams, P. Henderson, D. R. A. Wareing, F. J. Bolton, J. A. Frost, L. Ward, and T. J. Humphrey. 2002. Prevalence and numbers of *Salmonella* and *Campylobacter* spp. on raw, whole chickens in relation to sampling methods. *Int. J. Food Microbiol.* 76:151–164.
92. Kanerva, L., T. Sipiläinen-Malm, T. Estlander, A. Zitting, R. Jolanki, and K. Tarvainen. 1994. Nickel release from metals, and a case of allergic contact dermatitis from stainless steel. *Contact Dermatitis* 31:299–303.
93. Kapperud, G., J. Lassen, S. M. Ostroff, and S. Aasen. 1992. Clinical features of sporadic *Campylobacter* infections in Norway. *Scand. J. Infect. Dis.* 24:741–749.
94. Karmali, M. A., and P. C. Fleming. 1979. *Campylobacter* enteritis. *Can. Med. Assoc. J.* 120:1525–1532.
95. Karoui, R., H. A. Majeed, A. M. Yousof, M. A. A. Moussa, S. D. Iskander, and K. Hussain. 1982. Hemolytic streptococci and streptococcal antibodies in normal schoolchildren in Kuwait. *Am. J. Epidemiol.* 116:709–721.
96. Kassa, H., B. Harrington, M. Bisesi, and S. Khuder. 2001. Comparisons of microbiological evaluations of selected kitchen areas with visual inspections for preventing potential risk of foodborne outbreaks in food service operations. *J. Food Prot.* 64:509–513.
97. Kerr, K. G., D. Birkenhead, K. Seale, J. Major, and P. M. Hawkey. 1993. Prevalence of *Listeria* spp. on the hands of food workers. *J. Food Prot.* 56:525–527.
98. Keskinen, L. A., E. C. D. Todd, and E. T. Ryser. 2008. Transfer of surface-dried *Listeria monocytogenes* from stainless steel blades to roast turkey breast. *J. Food Prot.* 71:176–181.
99. Khin, N. O., P. P. Win, A. M. Han, and T. Aye. 1989. Contamination of currency notes with enteric pathogens. *J. Diarrhoeal Dis. Res.* 7:92–94.
100. Khuri-Bulos, N. A., M. A. Khalaf, A. Shenhabi, and K. Shami. 1994. Foodhandler-associated *Salmonella* outbreak in a university hospital despite routine surveillance cultures of kitchen employees. *Infect. Control Hosp. Epidemiol.* 15:311–314.
101. Kramer, A., I. Schwebke, and G. Kampf. 2006. How long do nosocomial pathogens survive on inanimate surfaces? *BMC Infect. Dis.* 6: 130–138. Available at: <http://www.biomedcentral.com/1471-2334/6/130>. Accessed 8 April 2008.
102. Kukavica-Ibrulj, I., A. Darveau, J. Jean, and I. Fliss. 2004. Hepatitis A virus attachment to agri-food surfaces using immunological, vi-

- rological and thermodynamic assays. *J. Appl. Microbiol.* 97:923–934.
103. Lacey, R. W., V. G. Alder, and W. A. Gillespie. 1970. The survival of *Staphylococcus aureus* on human skin. An investigation using mixed cultures. *Br. J. Exp. Pathol.* 51:305–313.
 104. Lane, K. M., M. Scarborough, and M. Park. 2001. Outbreaks of foodborne disease in Georgia, 2000. *Ga. Epidemiol. Rep.* 17(6):1–3.
 105. Larson, E., J. J. Leyden, K. J. McGinley, G. L. Grove, and G. H. Talbot. 1986. Physiologic and microbiologic changes in skin related to frequent handwashing. *Infect. Control* 7:59–63.
 106. LeBaron, C. W., N. P. Furutan, J. F. Lew, J. R. Allen, V. Gouvea, C. Moe, and S. S. Monroe. 1990. Viral agents of gastroenteritis. Public health importance and outbreak management. *Morb. Mortal. Wkly. Rep. Recomm. Rep.* 39(RR-5):1–24.
 107. Levy, B. S., R. E. Fontaine, C. A. Smith, J. Brinda, G. Hirman, D. B. Nelson, P. M. Johnson, and O. Larson. 1975. A large food-borne outbreak of hepatitis A. Possible transmission via oropharyngeal secretions. *JAMA* 234:289–294.
 108. Lew, J. F., C. A. Bopp, M. E. Dance, M. J. Gillenwater, R. I. Glass, P. M. Griffin, T. Mercatante, and D. L. Swerdlow. 1991. An outbreak of shigellosis aboard a cruise ship caused by a multiple-antibiotic-resistant strain of *Shigella flexneri*. *Am. J. Epidemiol.* 134:413–420.
 109. Lidwell, O. M., and E. J. Lowbury. 1950. The survival of bacteria in dust. III. The effect of light on the survival of bacteria in dust. *J. Hyg. Camb.* 48:28–37.
 110. Lin, C. M., F. M. Wu, M. P. Doyle, B. S. Michaels, and K. Williams. 2002. Influence of fingernail length and type on removing *Escherichia coli* from the nail regions using different hand washing interventions. *J. Food Prot.* 65:117.
 111. Lin, C. M., F. M. Wu, H. K. Kim, M. P. Doyle, B. S. Michael, and L. K. Williams. 2003. A comparison of hand washing techniques to remove *Escherichia coli* and caliciviruses under natural or artificial fingernails. *J. Food Prot.* 66:2296–2301. Erratum in *J. Food Prot.* 2004, 67:430.
 112. Lin, F. Y., J. M. Becker, E. F. Becker, T. Cramton, C. Groves, R. M. Helfrich, E. Israel, B. P. Lim, D. S. Swetter, and J. B. Robbins. 1988. Restaurant-associated outbreak of typhoid fever in Maryland: identification of carrier facilitated by measurement of serum Vi antibodies. *J. Clin. Microbiol.* 26:1194–1197.
 113. Lo, S. V., A. M. Connolly, D. Joynson, S. R. Palmer, P. D. Thomas, and D. Wright. 1994. The role of the pre-symptomatic food handler in a common source outbreak of food-borne SRSV gastroenteritis in a group of hospitals. *Epidemiol. Infect.* 113:513–521.
 114. Loosli, C. G. 1947. Dust and its control as a means of disinfection of air. *Am. J. Public Health* 37:353–359.
 115. Loosli, C. G., O. H. Robertson, and T. T. Puck. 1943. The production of experimental influenza in mice by inhalation of atmospheres containing influenza virus dispersed as fine droplets. *J. Infect. Dis.* 72:142–153.
 116. Louis, V. R., I. A. Gillespie, S. J. O'Brien, E. Russek-Cohen, A. D. Pearson, and R. R. Colwell. 2005. Temperature-driven *Campylobacter* seasonality in England and Wales. *Appl. Environ. Microbiol.* 71:85–92.
 117. Lowbury, E. J. L., and J. Fox. 1953. The influence of atmospheric drying on the survival of wound flora. *J. Hyg. Camb.* 51:203–214.
 118. Luber, P., and E. Bartelt. 2007. Enumeration of *Campylobacter* spp. on the surface and within chicken breast fillets. *J. Appl. Microbiol.* 102:313–318.
 119. Luber, P., S. Brynestad, D. Topsch, K. Scherer, and E. Bartelt. 2006. Quantification of *Campylobacter* species cross-contamination during handling of contaminated fresh chicken parts in kitchens. *Appl. Environ. Microbiol.* 72:66–70.
 120. Macintosh, C. A., and P. N. Hoffman. 1984. An extended model for the transfer of micro-organisms via the hands: differences between organisms and the effect of alcohol disinfection. *J. Hyg.* 92:345–355.
 121. Maguire, H., P. Pharoah, B. Walsh, C. Davison, D. Barrie, E. J. Threlfall, and S. Chambers. 2000. Hospital outbreak of *Salmonella virchow* possibly associated with a food handler. *J. Hosp. Infect.* 44:261–266.
 122. Maibach, H. 1976. Immediate hypersensitivity in hand dermatitis: role of food-contact dermatitis. *Arch. Dermatol.* 112:1289–1291.
 123. Mallison, G. F. 1979. CDC expert gives handwashing tips. *Hosp. Infect. Control* 6:59–61.
 124. Man, G. S., M. Olapoju, M. V. Chadwick, P. Vuddamalay, A. V. Hall, A. Edwards, and J. R. Kerr. 2002. Bacterial contamination of ward-based computer terminals. *J. Hosp. Infect.* 52:314–315.
 125. Marks, P. J., I. B. Vipond, D. Carlisle, D. Deakin, R. Fey, and E. O. Caul. 2000. Evidence for airborne transmission of Norwalk-like virus (NLV) in a hotel restaurant. *Epidemiol. Infect.* 124:481–487.
 126. Marples, M. J. 1965. The ecology of the human skin. Charles C Thomas, Bannerstone House, Springfield, IL.
 127. Marples, R. R., and A. G. Towers. 1979. A laboratory model for the investigation of contact transfer of micro-organisms. *J. Hyg. Camb.* 82:237–248.
 128. Martin, T. A., R. M. Biery, V. Gibson, and G. L. Hoff. 1985. Food-borne streptococcal pharyngitis, Kansas City, Missouri. *Am. J. Epidemiol.* 122:706–709.
 129. Matousek, W. J. 1921. Contamination of the hands and other objects in the spread of contagious diseases. *JAMA* 76:1490.
 130. Mattison, K., K. Karthikeyan, M. Abebe, N. Malik, S. A. Sattar, J. M. Farber, and S. Bidawid. 2007. Survival of calicivirus in foods and on surfaces: experiments with feline calicivirus as a surrogate for norovirus. *J. Food Prot.* 70:500–503.
 131. Mbithi, J. N., J. R. Boulet, S. A. Sattar, and V. S. Springthorpe. 1992. Survival of hepatitis A virus on human hands and its transfer on contact with animate and inanimate surfaces. *J. Clin. Microbiol.* 30:757–763.
 132. Mbithi, J. N., V. S. Springthorpe, and S. A. Sattar. 1991. Effect of relative humidity and air temperature on survival of hepatitis A virus on environmental surfaces. *Appl. Environ. Microbiol.* 57:1394–1399.
 133. McDade, J. J., and L. B. Hall. 1963. An experimental method to measure the influence of environmental factors on the viability and the pathogenicity of *Staphylococcus aureus*. *Am. J. Hyg.* 77:98–108.
 134. McDade, J. J., and L. B. Hall. 1964. Survival of gram-negative bacteria in the environment. I. Effect of relative humidity on surface-exposed organisms. *Am. J. Hyg.* 80:192–204.
 135. McGinley, K. J., E. L. Larson, and J. J. Laeyden. 1988. Composition and density of the microflora in the subungual space of the hand. *J. Clin. Microbiol.* 26:950–953.
 136. Mead, P. S., L. Finelli, M. A. Lambert-Fair, D. Champ, J. Townes, L. Hutwagner, T. Barrett, K. Spitalny, and E. Mintz. 1997. Risk factors for sporadic infection with *Escherichia coli* O157:H7. *Arch. Intern. Med.* 157:204–208.
 137. Mead, P. S., L. Slutsker, V. Dietz, L. F. McCaig, J. S. Bresee, C. Shapiro, P. M. Griffin, and R. V. Tauxe. 1999. Food-related illness and death in the United States. *Emerg. Infect. Dis.* 5:607–625.
 138. Meara, J. 1987. Viral gastroenteritis and food handlers—melon off the menu. *Lancet* ii:865.
 139. Mendes, M. F., and D. J. Lynch. 1976. A bacteriological survey of washrooms and toilets. *J. Hyg. (Lond.)* 76:183–190.
 140. Meneghini, C. L. 1985. Sensitization in traumatized skin. *Am. J. Ind. Med.* 8:319–321.
 141. Mensah, P., D. Yeboah-Manu, K. Owusu-Darko, and A. Ablordey. 2002. Street foods in Accra, Ghana: how safe are they? *Bull. W.H.O.* 80:546–554.
 142. Merry, A. F., T. E. Miller, G. Findon, C. S. Webster, and S. P. W. Neff. 2001. Touch contamination levels during anaesthetic procedures and their relationship to hand hygiene procedures: a clinical audit. *Br. J. Anaesth.* 87:291–294.
 143. Michaels, B. 2002. Handling money and serving ready-to-eat food. *Food Service Technol.* 2:1–3.
 144. Michaels, B., V. Gangar, C.-M. Lin, and M. Doyle. 2003. Use limitations of alcoholic instant hand sanitizer as part of a food service hand hygiene program. *Food Service Technol.* 3:71–80.
 145. Michaels, B., B. Smith, and M. Pierson. 2001. Pathogenic and in-

- dicator bacteria associated with handwashing and drying contact surfaces. *J. Food Prot.* 64(Suppl. A):95.
146. Mills, S. J. C., D. J. Holland, and A. E. Hardy. 2000. Operative field contamination by the sweating surgeon. *Aust. N.Z. J. Surg.* 70:837–839.
 147. Minor, T. E., and E. H. Marth. 1976. Staphylococci and their significance in food. Elsevier Scientific Publishing Company, Amsterdam.
 148. Montville, R., and D. W. Schaffner. 2003. Inoculum size influences bacterial cross contamination between surfaces. *Appl. Environ. Microbiol.* 69:7188–7193.
 149. Nel, S., J. F. R. Lues, E. M. Buys, and P. Venter. 2004. The personal and general hygiene practices in the deboning room of a high throughput red meat abattoir. *Food Control* 15:571–578.
 150. Noble, W. C. 1962. The dispersal of staphylococci in hospital wards. *J. Clin. Pathol.* 15:552–558.
 151. Noble, W. C. 1975. Dispersal of skin microorganisms. *Br. J. Dermatol.* 93:477–485.
 152. Noskin, G. A., V. Stosor, I. Cooper, and L. R. Peterson. 1995. Recovery of vancomycin-resistant enterococci on fingertips and environmental surfaces. *Infect. Control Hosp. Epidemiol.* 16:577–581.
 153. Omisakin, F., M. MacRae, I. D. Ogden, and N. J. C. Strachan. 2003. Concentration and prevalence of *Escherichia coli* O157 in cattle feces at slaughter. *Appl. Environ. Microbiol.* 69:2444–2447.
 154. Orr, P., B. Lorencz, R. Brown, R. Kielly, B. Tan, D. Holton, H. Clugstone, L. Lugtig, C. Pim, S. Macdonald, G. Hammond, M. Moffatt, J. Spika, D. Manuel, W. Winther, D. Milley, H. Lior, and N. Sinuff. 1992. An outbreak of diarrhea due to verotoxin-producing *Escherichia coli* in the Canadian Northwest Territories. *Scand. J. Infect. Dis.* 26:675–684.
 155. Parry, M. F., J. H. Hutchinson, N. A. Brown, C. H. Wu, and L. Estreller. 1980. Gram-negative sepsis in neonates: a nursery outbreak due to hand carriage of *Citrobacter diversus*. *Pediatrics* 65:1105–1109.
 156. Patrick, D. R., G. Findon, and T. E. Miller. 1997. Residual moisture determines the level of touch-contact-associated bacterial transfer following hand washing. *Epidemiol. Infect.* 119:19–25.
 157. Patterson, M. F. 1995. Sensitivity of *Campylobacter* spp. to irradiation in poultry meat. *Lett. Appl. Microbiol.* 20:338–340.
 158. Patterson, W., P. T. Fryers, J. Green, and P. Haswell. 1997. Outbreak of small round structured virus gastroenteritis arose after kitchen assistant vomited. *Commun. Dis. Rep.* 27:R101–R103.
 159. Paulson, D. S. 1996. To glove or to wash: a current controversy. *Food Qual.* June–July:60–64.
 160. Peltonen, L. 1979. Nickel sensitivity in the general population. *Contact Dermatitis* 5:27–32.
 161. Peltonen, L., G. Wickstrom, and M. Vaahtoranta. 1985. Occupational dermatoses in the food industry. *Derm. Beruf Umwelt* 33:166–169.
 162. Pereira, M. L., L. S. do Carmo, E. J. dos Santos, and M. S. Bergdoll. 1994. Staphylococcal food poisoning from cream-filled cake in a metropolitan area of south-eastern Brazil. *Rev. Saude Publica* 28:406–409.
 163. Pérez-Rodríguez, F., E. C. D. Todd, A. Valero, E. Carrasco, R. M. García, and G. Zurera. 2006. Linking quantitative exposure assessment and risk management using the food safety objective concept: an example with *Listeria monocytogenes* in different cross-contamination scenarios. *J. Food Prot.* 69:2384–2394.
 164. Pether, J. V. S., and R. J. Gilbert. 1971. The survival of *Salmonella* on finger-tips and transfer of the organisms to food. *J. Hyg. Camb.* 69:673–681.
 165. Pether, J. V. S., and R. J. D. Scott. 1983. *Salmonella* carriers; are they dangerous? A study to identify finger contamination with salmonellae by convalescent carriers. *J. Infect.* 5:81–88.
 166. Pope, T. W., P. T. Ender, T. M. Koroscil, W. K. Woelk, and M. A. Koroscil. 2001. Contaminated currency: the true return on the dollar, session 220C, paper 3-332. Presented at the 101st General Meeting of the American Society for Microbiology, Orlando, FL, 20 to 24 May 2001.
 167. Price, P. B. 1938. The bacteriology of normal skin: a new quantitative test applied to a study of the bacterial flora and the disinfectant action of mechanical cleansing. *J. Infect. Dis.* 63:301–308.
 168. Raffaelli, R. M., M. Paladini, H. Hanson, L. Kornstein, A. Agasan, S. Slavinski, D. Weiss, G. F. Fennelly, and J. T. Flynn. 2007. Child care-associated outbreak of *Escherichia coli* O157:H7 and haemolytic uremic syndrome. *Pediatr. Infect. Dis. J.* 26:951–953.
 169. Rathmachers, B., and M. Borneff. 1977. Development of a new test method for surface disinfection procedures. IV. Natural bacterial dying rates and their modification by environmental factors. *Zentbl. Bakteriol. Hyg. I Abt. Orig. B* 165:43–59.
 170. Reed, S. E. 1975. An investigation of the possible transmission of rhinovirus colds through indirect contact. *J. Hyg. (Lond.)* 75:249–258.
 171. Reynolds, K. A., P. A. Watt, S. A. Boone, and C. P. Gerba. 2005. Occurrence of bacteria, and biochemical markers on public surfaces. *Int. J. Environ. Health Res.* 15:225–234.
 172. Rheinbaben, F., S. Schunemann, T. Gross, and M. H. Wolff. 2000. Transmission of viruses via contact in a household setting: experiments using bacteriophage straight phiX174 as a model virus. *J. Hosp. Infect.* 46:61–66.
 173. Ritter, M. A., H. Eitzen, M. L. V. French, and J. B. Hart. 1975. The operating room environment as affected by people and the surgical face mask. *Clin. Orthopaed. Res.* 111:147–150.
 174. Robertson, O. H., E. W. Dunklin, Jr., W. Lester, and T. T. Puck. 1948. The influence of relative humidity on the survival of respiratory pathogens dispersed into the environment. *Trans. Assoc. Am. Phys.* 61:280–287.
 175. Roth, R. R., and W. D. James. 1988. Microbial ecology of the skin. *Ann. Rev. Microbiol.* 42:441–464.
 176. Rowan, N. J. 2004. Viable but non-culturable forms of food and waterborne bacteria: quo vadis? *Trends Food Sci. Technol.* 15:462–467.
 177. Rubin, H. E. 1985. Protective effect of casein toward *Salmonella typhimurium* in acid-milk. *J. Appl. Bacteriol.* 58:251–255.
 178. Rusin, P., S. Maxwell, and C. Gerba. 2002. Comparative surface-to-hand and fingertip-to-mouth transfer efficiency of gram-positive bacteria, gram-negative bacteria, and phage. *J. Appl. Microbiol.* 93:585–592.
 179. Rutala, W. A., M. S. White, M. F. Gergen, and D. J. Weber. 2006. Bacterial contamination of keyboards: efficacy and functional impact of disinfectants. *Infect. Control Hosp. Epidemiol.* 27:372–377.
 180. Rzezutka, A., and N. Cook. 2004. Survival of human enteric viruses in the environment and food. *FEMS Microbiol. Rev.* 28:441–453.
 181. Salisbury, D. M., P. Hutfilz, L. M. Treen, G. E. Bollin, and S. Gautam. 1997. The effect of rings on microbial load of health care workers' hands. *Am. J. Infect. Control* 25:24–27.
 182. Samadi, A. R., M. I. Huq, and Q. S. Ahmed. 1983. Detection of rotavirus in handwashings of attendants of children with diarrhoea. *Br. Med. J. (Clin. Res. Ed.)* 15:188.
 183. Sattar, S. A., N. Lloyd-Evans, V. S. Springthorpe, and R. C. Nair. 1986. Institutional outbreaks of rotavirus diarrhoea: potential role of fomites and environmental surfaces as vehicles for virus transmission. *J. Hyg. (Lond.)* 96:277–289.
 184. Sattar, S. A., V. S. Springthorpe, J. Tetro, R. Vashon, and B. Keswick. 2002. Hygienic hand antiseptics: should they not have activity and label claims against viruses? *Am. J. Infect. Control* 30:355–372.
 185. Sattar, S. A., J. Tetro, S. Bidawid, and J. Farber. 2000. Foodborne spread of hepatitis A: recent studies on virus survival, transfer and inactivation. *Can. J. Infect. Dis.* 11:159–163.
 186. Sawyer, L. A., J. J. Murphy, J. E. Kaplan, P. F. Pinsky, D. Chacon, S. Walmsley, L. B. Schonberger, A. Phillips, K. Forward, C. Goldman, J. Brunton, R. A. Fralick, A. O. Carter, W. G. Gary, Jr., R. I. Glass, and D. E. Low. 1988. 25- to 30-nm virus particle associated with a hospital outbreak of acute gastroenteritis with evidence for airborne transmission. *Am. J. Epidemiol.* 127:1261–1271.
 187. Schürmann, W., and H. J. Eggers. 1985. An experimental study on the epidemiology of enteroviruses: water and soap washing of po-

- liovirus 1-contaminated hands, its effectiveness and kinetics. *Med. Microbiol. Immunol. (Berl)*. 174:221–236.
188. Scott, E., and S. F. Bloomfield. 1990. The survival and transfer of microbial contamination via cloths, hands and utensils. *J. Appl. Bacteriol.* 68:271–278.
 189. Silvestro, L., M. Caputo, S. Blancato, L. Decastelli, A. Fioravanti, R. Tozzoli, S. Morabito, and A. Caprioli. 2004. Asymptomatic carriage of verocytotoxin-producing *Escherichia coli* O157 in farm workers in Northern Italy. *Epidemiol. Infect.* 132:915–919.
 190. Simmons, B. P. 1983. CDC guidelines for the prevention and control of nosocomial infections. Guideline for hospital environmental control. *Am. J. Infect. Control* 11:97–120.
 191. Slutsker, L., A. A. Ries, K. D. Greene, J. G. Wells, L. Hutwagner, and P. M. Griffin. 1997. *Escherichia coli* O157:H7 diarrhea in the US: clinical and epidemiological features. *Ann. Intern. Med.* 126:505–513.
 192. Snelling, A. M., K. G. Kerr, and J. Heritage. 1991. The survival of *Listeria monocytogenes* on fingertips and factors affecting elimination of the organism by hand washing and disinfection. *J. Food Prot.* 54:343–348.
 193. Sobel, J., B. Mahon, C. E. Mendoza, D. Passaro, F. Cano, K. Baier, F. Racioppi, L. Hutwagner, and E. Mintz. 1998. Reduction of fecal contamination of street-vended beverages in Guatemala by a simple system for water purification and storage, handwashing, and beverage storage. *Am. J. Trop. Med. Hyg.* 59:380–387.
 194. Spector, B. K., and F. Buky. 1934. Viability of *Endamoeba histolytica* and *Endamoeba coli*: effect of drying. *Public Health Rep.* 49:379–385.
 195. St. Louis, M. E., J. D. Porter, A. Helal, K. Drame, N. Hargrett-Bean, J. G. Wells, and R. V. Tauxe. 1990. Epidemic cholera in West Africa: the role of food handling and high-risk foods. *Am. J. Epidemiol.* 131:719–728.
 196. Strachan, N. J. C., M. P. Doyle, F. Kasuga, O. Rotariu, and I. D. Ogdén. 2005. Dose response modelling of *Escherichia coli* O157 incorporating data from foodborne and environmental outbreaks. *Int. J. Food Microbiol.* 103:35–47.
 197. Strange, R. E., and C. S. Cox. 1976. Survival of dried and airborne bacteria, p. 111–154. In T. R. G. Gray and J. R. Postgate (ed.), *Survival of vegetative microbes*. Cambridge University Press, New York.
 198. Straver, J. M., A. F. W. Janssen, A. R. Linnemann, M. A. J. S. van Boekel, R. R. Beumer, and M. H. Zwietering. 2007. Number of *Salmonella* on chicken breast file at retail level and its implications for public health risk. *J. Food Prot.* 70:2045–2055.
 199. Swerdlow, D. L., G. Malenga, G. Begkoyian, D. Nyangulu, M. Toole, R. J. Waldman, D. N. Puh, and R. V. Tauxe. 1997. Epidemic cholera among refugees in Malawi, Africa; treatment and transmission. *Epidemiol. Infect.* 118:207–214.
 200. Teixeira, P., J. C. Lima, J. Azeredo, and R. Oliveira. 2007. Colonisation of bench cover materials by *Salmonella typhimurium*. *Food Sci. Technol. Int.* 13:5–10.
 201. Teixeira, P., S. Silva, F. Araújo, J. Azeredo, and R. Oliveira. 2007. Bacterial adhesion to food contacting surfaces, p. 13–20. In A. Méndez-Vilas (ed.), *Communicating current research and educational topics and trends in applied microbiology*. Microbiology series 1, vol. 1. Available at: <http://www.formatex.org/microbio/pdf/Pages13-20.pdf>. Accessed 7 May 2008.
 202. Todd, E. C. D., J. D. Greig, C. A. Bartleson, and B. Michaels. 2007. Outbreaks where food workers have been implicated in the spread of foodborne disease. Part 2. Description of outbreaks by size, severity, and settings. *J. Food Prot.* 70:1975–1993.
 203. Todd, E. C. D., J. D. Greig, C. A. Bartleson, and B. Michaels. 2007. Outbreaks where food workers have been implicated in the spread of foodborne disease. Part 3. Factors contributing to outbreaks and description of outbreak categories. *J. Food Prot.* 70:2199–2217.
 204. Todd, E. C. D., J. D. Greig, C. A. Bartleson, and B. Michaels. Outbreaks where food workers have been implicated in the spread of foodborne disease. Part 4. Infective doses and pathogen carriage. *J. Food Prot.*, in press.
 205. Tosti, A., and L. Guerra. 1988. Protein contact dermatitis in food handlers. *Contact Dermatitis* 19:149.
 206. Tuttle, J., T. Gomez, M. P. Doyle, J. G. Wells, T. Zhao, R. V. Tauxe, and P. M. Griffin. 1999. Lessons from a large outbreak of *Escherichia coli* O157:H7 infections: insights into the infectious dose and method of widespread contamination of hamburger patties. *Epidemiol. Infect.* 122:185–192.
 207. Tuttle, J., A. A. Ries, R. M. Chimba, C. U. Perera, N. H. Bean, and P. M. Griffin. 1995. Antimicrobial-resistant epidemic *Shigella dysenteriae* type 1 in Zambia: modes of transmission. *J. Infect. Dis.* 171:371–375.
 208. U.S. Department of Agriculture, Food Safety and Inspection Service. 2007. Microbiological results of raw ground beef products analyzed for *Escherichia coli* O157:H7. Available at: http://www.fsis.usda.gov/Science/ground.beef.E.coli_Testing_results/index.asp. Accessed 15 October 2007.
 209. U.S. Food and Drug Administration. 2005. Food Code 2005. U.S. Department of Health and Human Services, Public Health Service, U.S. Food and Drug Administration, College Park, MD.
 210. Van der Valk, P. G., E. Snater, W. Verbeek-Gijsbers, P. Duller, and P. C. van de Kerkhof. 1996. Out-patient treatment of atopic dermatitis with crude coal tar. *Dermatology* 193:41–44.
 211. Veien, N. K., T. Hattel, O. Justesen, and A. Norholm. 1983. Causes of eczema in the food industry. *Derm. Beruf Umwelt* 31:84–86.
 212. Vogt, R. L., and L. Dippold. 2005. *Escherichia coli* O157:H7 outbreak associated with consumption of ground beef, June–July 2002. *Public Health Rep.* 120:174–178.
 213. Vorst, K. L., E. C. D. Todd, and E. T. Ryser. 2006. Transfer of *Listeria monocytogenes* during mechanical slicing of turkey breast, bologna, and salami. *J. Food Prot.* 69:619–626.
 214. Wachtel, M. R., J. L. McEvoy, Y. Luo, A. M. Williams-Campbell, and M. B. Solomon. 2003. Cross-contamination of lettuce (*Lactuca sativa* L.) with *Escherichia coli* O157:H7 via contaminated ground beef. *J. Food Prot.* 66:1176–1183.
 215. Webb, S. J. 1968. Effect of dehydration on bacterial recombination. *Nature* 217:1231–1234.
 216. Weinberg, M., J. Hopkins, L. Farrington, L. Gresham, M. Ginsberg, and B. P. Bell. 2004. Hepatitis A in Hispanic children who live along the United States–Mexico border: the role of international travel and food-borne exposures. *Pediatrics* 114:e68–e73.
 217. Werber, D., J. Dressman, F. Feil, U. van Treck, G. Fell, S. Ethelberg, A. M. Hauri, P. Roggentin, R. Prager, I. S. Fisher, S. C. Behnke, E. Bartelt, E. Weise, A. Ellis, A. Siitonen, Y. Andersson, H. Tschape, M. H. Kramer, and A. Ammon. 2005. International outbreak of *Salmonella Oranienburg* due to German chocolate. *BMC Infect. Dis.* 5:7.
 218. White, K. E., M. T. Osterholm, J. A. Mariotti, J. A. Korlath, D. H. Lawrence, T. L. Ristinen, and H. B. Greenberg. 1986. A foodborne outbreak of Norwalk virus gastroenteritis, evidence for post-recovery transmission. *Am. J. Epidemiol.* 124:120–126.
 219. Widdowson, M. A., R. Glass, S. Monroe, R. S. Beard, J. W. Bateman, P. Lurie, and C. Johnson. 2005. Probable transmission of norovirus on an airplane. *JAMA* 293:1859–1860.
 220. Winfield, M. D., and E. A. Groisman. 2003. Role of nonhost environments in the lifestyles of *Salmonella* and *Escherichia coli*. *Appl. Environ. Microbiol.* 69:3687–3694.
 221. Zhao, P., T. Zhao, M. P. Doyle, J. R. Rubino, and J. Meng. 1998. Development of a model for evaluation of microbial cross-contamination in the kitchen. *J. Food Prot.* 61:960–963.