

and/or turkey to which salt, sugar, sodium nitrite, and spices are normally added. When making frankfurters, the meat/ingredient mixture, commonly referred to as the sausage emulsion, is stuffed into natural or artificial casings, which are then twisted to form sausage links. Frankfurters are typically cooked to an internal temperature of $\geq 70^{\circ}\text{C}$ to (a) coagulate protein, (b) fix the cured color, and (c) destroy certain pathogens and bacteria that would cause spoilage. Although not an absolute requirement, frankfurters and other similar sausages are commonly subjected to natural smoke. Alternatively, a commercially available liquid smoke solution can be added to the sausage emulsion or atomized onto the surface of frankfurters before or during heating. After cooking, frankfurters are cooled, packaged, and refrigerated for shipment to wholesale and retail markets (Figure 16–1). Skinless frankfurters, which are very popular, are produced in a similar manner, except that the artificial casing is mechanically peeled from the sausage after cooking.

Cooking renders the sausages free of *L. monocytogenes*. However, post-processing contamination can occur and in rare instances has led to illness, such as the 1998–1999 multistate outbreak with 101 confirmed cases, including 15 adult deaths and six miscarriages/stillbirths (CDC, 1999). It should be noted that multiplication of *L. monocytogenes* on frankfurters is probably necessary for disease to occur, but data on the dose-response relationship in humans are not available. This and other aspects of the risk analysis of *L. monocytogenes* in frankfurters are discussed below. Several reports dealing with risk assessment and *L. monocytogenes* have been published and contain additional background information (Farber *et al.*, 1996; Hitchins, 1996; Buchanan *et al.*, 1997; Bemrah *et al.*, 1998; FAO/WHO, 2000; Buchanan & Lindqvist, 2000; Ross *et al.*, 2000; FDA, 2001).

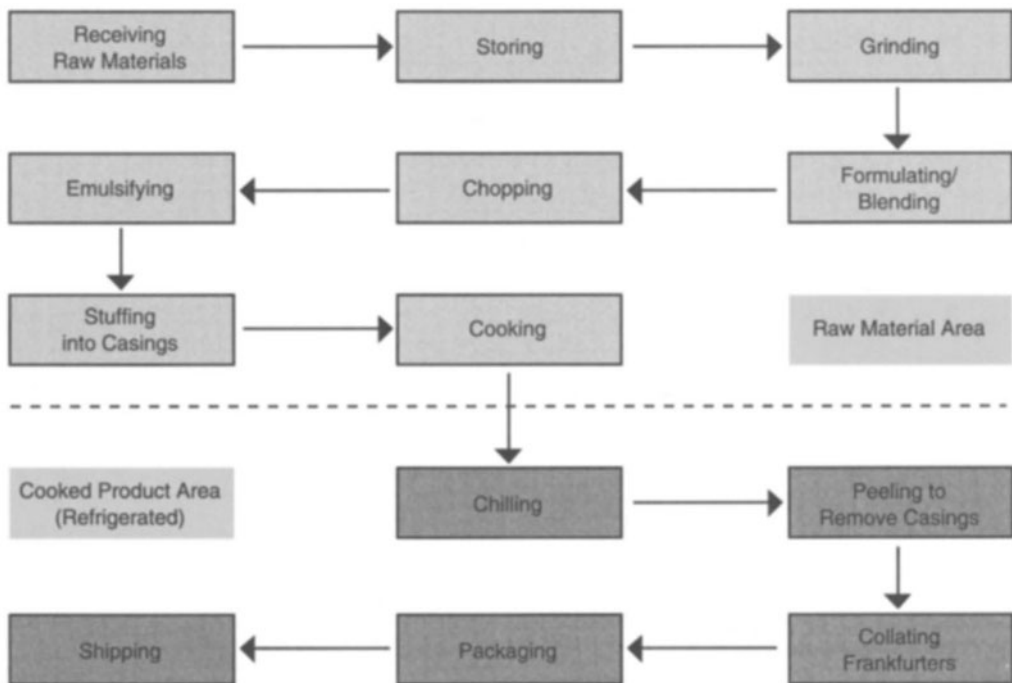


Figure 16–1 Flow diagram for typical frankfurter operation.

16.2 RISK EVALUATION

16.2.1 Hazard Identification

Epidemiologic data suggest that most exposures to *L. monocytogenes* are foodborne (Ciesielski *et al.*, 1988; Broome *et al.*, 1990; Farber & Peterkin, 1991; McLauchlin, 1993; Mead *et al.*, 1999). Although listeriosis occurs infrequently, at somewhere between 2–6 cases annually per million of population, between 20–30% of the cases are fatal (McLauchlin, 1993; Rocourt, 1996; Mead *et al.*, 1999). Thirteen serotypes of *L. monocytogenes* have been identified, but most illnesses have been associated with only three, namely serovars 4b, 1/2a, and 1/2b. Serovar 4b accounts for close to half of all the cases in Europe, while serovars 4b, 1/2a, and 1/2b are more equally represented in North America (Farber & Peterkin, 1991; McLauchlin, 1993; Rocourt, 1996; Rocourt & Bille, 1997). However, the potential for all serovars to cause human illness cannot be discounted.

All *L. monocytogenes* serotypes are considered in this risk evaluation. *L. monocytogenes* is a Gram-positive, non-spore-forming rod. It is facultatively anaerobic, growing at temperatures between –0.4 and 45 °C, pH values 4.39–9.4, a water activity of 0.92 or higher, and a salt concentration of 10% or less (ICMSF, 1996). It is catalase positive, oxidase negative and produces β -hemolytic colonies on blood agar. The organism is widely distributed in nature and can be found on fruits and vegetables, in soil, water, sewage, silage, slaughterhouse waste, milk from healthy and mastitic cows, as well as human and animal feces (Farber & Peterkin, 1991, 1999). *L. monocytogenes* can be found as part of the normal flora of many animal species and humans, and it has been estimated that between 2 and 6% of humans and animals are transient carriers of the organism. Fecal excretion of the organism is not necessarily an indication of disease, and the role of healthy carriers in the epidemiology of listeriosis is still unclear (Rocourt, 1996).

L. monocytogenes is inactivated by heat treatments of 70 °C or above, used in cooking or pasteurization. However, products, such as frankfurters, that are purchased as a pre-cooked sausage are sometimes eaten without further cooking or may be reheated by a variety of methods (frying pan, grilling, simmering water, microwave). The practice of reheating in a microwave oven was associated with one sporadic case of listeriosis in an elderly woman who had cancer (CDC, 1989). Meat products, such as cooked sausages, can be recontaminated between cooking and packaging in the processing facility. During extended refrigerated storage (e.g., ≥ 35 days), the organism could multiply to potentially hazardous levels on the surface of the frankfurters and in the exudate, especially if the product is temperature-abused. The free exudate in the package has a higher concentration of cells than the product itself and may be a source of cross-contamination in the kitchen.

16.2.2 Hazard Characterization

Human listeriosis is now recognized as a disease primarily caused by ingestion of *L. monocytogenes* in food. This also includes secondary transmission between a mother and her fetus or neonate. Despite its widespread presence in the environment, illness due to this organism occurs infrequently. The outcome of listeriosis can be severe, with an estimated case fatality rate of between 20–30% in the segments of the population most at risk, i.e., immunocompromised individuals, pregnant women, the very young (i.e., fetuses and neonates), and the elderly. This spectrum of individuals is estimated to com-

prise 15–20% of the population (Buchanan *et al.*, 1997) and is expected to increase, reflecting the trend in longer life expectancy and an overall aging population.

Unlike the symptoms caused by many foodborne pathogens, diarrhea and other gastrointestinal symptoms are not common symptoms of listeriosis, although the patient may experience malaise and a mild fever. In several foodborne listeriosis outbreaks, patients have exhibited only these mild symptoms (Salamina *et al.*, 1996; Dalton *et al.*, 1997). However, the majority of reported cases have been the invasive type of listeriosis, symptoms of which include meningitis, encephalitis, and septicemia. In pregnant mothers, unrecognized and untreated listeriosis can lead to abortion of the fetus, stillbirth, or premature delivery of a sick child. Serious sequelae, such as mental retardation and hydrocephalus, also have been reported following cases of foodborne listeriosis (Büla *et al.*, 1995).

There are no experimental dose-response data available for *L. monocytogenes* in humans (i.e., the minimum infectious dose (MID) is uncertain). As with many other foodborne pathogens, the MID will depend on factors such as the virulence of the strain, the amount of food consumed, the levels of the organism in the food, and the immune state of the host. Some animal data are available, mainly using the mouse model (Audurier *et al.*, 1980; Golnazarian *et al.*, 1989; FAO/WHO, 2000), but extrapolation of mouse data to the human situation is tenuous at best. While dose-response studies using human volunteers for certain pathogens have sometimes been conducted, this is not feasible with *L. monocytogenes*, since the risk of severe morbidity or mortality is too great. An alternate approach used with RTE foods is to combine epidemiologic data with estimates of the number of organisms present in the incriminated food. However, because outbreaks are an uncommon occurrence and it is difficult to obtain sufficient material in most cases, the amount of reliable data is scant and the information insufficient to draw reliable conclusions. The type of food consumed, perhaps, may influence the dose-response relationship.

When a risk estimate for *L. monocytogenes* is being made, it is important to determine which type of illness is being considered (i.e., mild versus invasive) and for which part of the population (i.e., “normal” versus “high-risk”). Since frankfurters are consumed by all segments of the population, different risk estimates ideally should be used for normal versus high-risk individuals.

Foodborne listeriosis appears generally to follow one of three scenarios. Scenario 1 consists of isolated cases for which information about the food is seldom available. Scenario 2 consists of an outbreak or cluster of cases involving a single lot of contaminated food. Both of these events typically involve errors in food handling that lead to a single lot of food becoming contaminated and an opportunity for multiplication before the food is consumed. Once the implicated food is eliminated, further cases cease to occur.

Scenario 3 consists of an outbreak involving anywhere from a few cases to several hundred cases scattered by time and location. This type of outbreak typically involves an unusually virulent strain that has become established in the food processing environment and contaminates multiple lots of food over days or months of production. Experience in cooked meat and poultry operations indicates that a niche or site within the cooked product environment wherein *L. monocytogenes* becomes established and multiplies is commonly involved. The sites may be impossible to reach and clean with normal cleaning and sanitizing procedures. In fact, the processing environment typically appears visually clean and acceptable. The sites serve as a reservoir from which the pathogen is dispersed during a food processing operation, contaminating food contact surfaces and, thereby, the

food. The niche usually affects only the product being handled (e.g., filled, sliced, conveyed, assembled, packaged) along one packaging line and not the product on an adjacent line. Microbiological testing is necessary to detect the niche. Examples of niches include hollow rollers on conveyors, cracked tubular support rods on equipment, the space between close fitting layers of metal-to-metal or metal-to-plastic, worn or cracked rubber seals around doors, on-off valves and buttons for equipment, and saturated insulation.

In all three scenarios, there is an opportunity for *L. monocytogenes* to multiply in the food before it is consumed. Food processors should establish systems to prevent scenario 3 events and minimize the risk of scenarios 1 and 2.

16.2.3 Exposure Assessment

L. monocytogenes is found as a frequent contaminant of RTE foods (Farber & Peterkin, 1991, 1999). Many different foods have been involved in both sporadic cases and food-borne outbreaks, with the approximate levels detected ranging from 100 to 1×10^9 cfu g⁻¹ (Table 16-1). The meat industry has a long history of providing safe food, including frankfurters, notwithstanding the recent large listeriosis outbreak (CDC, 1999). Qvist & Liberski (1991) found *L. monocytogenes* to be present in four of 67 (6%) frankfurter samples, while Wang & Muriana (1994) detected the organism in seven of 93 (7.5%) packages of retail frankfurters examined. The US Department of Agriculture (USDA) monitoring program for RTE meats after packaging at the manufacturing facility found the prevalence of *L. monocytogenes* in small-diameter cooked sausages to be 5.3, 4.8, 4.1, 3.7, 3.3, 3.5, and 1.8% annually from 1993 to 1999, respectively.

Frankfurter processing involves a step-heating schedule to allow for the formation of the "skin," for setting of the emulsion, and to allow the curing reaction to occur. This step also reduces *L. monocytogenes* numbers. Zaika *et al.* (1990) prepared frankfurters from a sausage emulsion inoculated to contain around 10^8 cfu g⁻¹ of *L. monocytogenes*. After stuffing, all frankfurters were thermally processed (without smoke) according to a standard commercial heating schedule. Zaika and colleagues found that *L. monocytogenes* decreased approximately 1000-fold in frankfurters heated to an internal temperature of 71.1 °C. Hence, based on those data, cooking frankfurters to an internal temperature of 71.1 °C should eliminate higher levels of *L. monocytogenes* ($\sim 10^3$ cfu g⁻¹) than are likely to occur in raw frankfurter emulsions. In addition, post-process pasteurization can eliminate *L. monocytogenes* from the surface of frankfurters that have been contaminated between cooking and packaging. This procedure is currently practiced commercially by certain manufacturers throughout the world.

The cooking step for frankfurters can be controlled in facilities operating with effectively designed and implemented HACCP plans, but preventing contamination of the cooked product during cooling and packaging is much more difficult. Data gathered by the American Meat Institute in 1988 pointed to frankfurters as a likely carrier of *L. monocytogenes* and suggested that uncontrolled environmental conditions before packaging may play a major role in contaminating the finished product (Anon., 1989). Environmental sampling of a turkey frankfurter plant, whose product was linked to a case of listeriosis, found that contamination of the majority of frankfurters occurred at a single point during the peeling process, just prior to packaging (Wenger *et al.*, 1990). In retrospect, a properly applied environmental sampling program (see Chapter 10) might have detected the site and, if corrected, prevented contamination of the final product.

Table 16-1 Foodborne Outbreaks due to *L. monocytogenes*

Location (year)	No. of Cases (deaths)	Perinatal/nonperinatal	No. cfu g ⁻¹	Foods associated	Serotype	Reference
Boston, USA (1979)	20 (5)*	0/20	NK [†]	Raw celery, tomatoes, lettuce [†]	4b	Ho et al., 1986
New Zealand (1980)	29 (9)	22/7	NK	Shellfish, raw fish	most cases 1/2b	Lennon et al., 1984
Maritime Prov., Canada (1981)	41 (17)	34/7	NK	Coleslaw [†]	4b	Schlech et al., 1983
Massachusetts, USA (1983)	49 (14)	7/42	NK	Pasteurized milk [†]	4b	Fleming et al., 1985
California, USA (1985)	142 (48)	93/49	10 ³ - 10 ⁴	Queso blanco fresco cheese	4b	Linnan et al., 1988
Switzerland (1983-1987)	122 (31)	65/57	10 ⁴ - 10 ⁶	Raw milk cheese	4b	Bille, 1990
Connecticut, USA (1989)	9 (1)	2/7	NK	Shrimp [†]	4b	Riedo et al., 1994
United Kingdom (1987-1989)	355 (94) ^s	185/129 ⁱⁱ	< 10 ² - 10 ⁶	Pâté [†]	4b; 4b(x)	McLauchlin et al., 1991
W. Australia (1990)	11** (6)	11/0	8.8 × 10 ³	Pâté [†]	1/2a	Kittson, 1992
Tasmania, Australia (1991)	4(0)	0/4	1.6 × 10 ⁷	Smoked mussels	1/2b	Mitchell et al., 1991
New Zealand (1992)	3 (2)	2/1	NK	Smoked mussels	1/2a	Baker & Wilson, 1993; Brett et al., 1998
France (1993)	39 (12)	31/8	< 100 - 1.0 × 10 ⁴	Pork rilletes (pâté)	4b	Goulet, 1995
France (1992)	279 (85)	92/187	NK	Pork tongue in jelly	4b	Goulet et al., 1993

Italy (1993)	23 (0)	0/23	NK	Rice salad [†]	1/2b	Salamina <i>et al.</i> , 1996
USA (1994)	45 (0)	1/44	2.9×10^{11}	Chocolate milk	1/2b	Dalton <i>et al.</i> , 1997; Proctor <i>et al.</i> , 1995
France (1995)	20 (4)	9/11	NK	Raw milk soft cheese	4b	Goulet <i>et al.</i> , 1995
Sweden (1994–1995)	6 (1) ^{††}	2/4	$< 100 - 2.5 \times 10^6$	Cold-smoked rainbow trout	4b	Ecklow <i>et al.</i> , 1996
Canada (1996)	2 (0)	0/2	2×10^9	Imitation crab meat	1/2b	Farber <i>et al.</i> , 2000
Sweden (NK)	5 (0)	0/5	1.9×10^5	Cold-smoked rainbow trout	1/2a	Miettinen <i>et al.</i> , 1999
Finland (1999)	18 (4)	0/18	$< 100 - 1.1 \times 10^4$	Butter	3a	Lyytikäinen <i>et al.</i> , 1999
USA (1998–1999)	101 (20)	NK	NK	Frankfurters, sliced deli meats	4b	CDC, 1999

* For 2 of these 5 deaths, an underlying disease, not listeriosis was apparently the cause of death.

[†] Not known.

[‡] Foods only epidemiologically linked.

[§] Outcome only known for 252 cases.

^{||} Only 314 patients categorized.

^{**} Includes two sets of twins.

^{††} Not known if death due to listeriosis or leukemia.

Glass & Doyle (1989) reported that this pathogen can proliferate on vacuum-packaged, artificially contaminated (~ 0.01 *L. monocytogenes* cfu g⁻¹) retail frankfurters during storage at 4.4 °C; populations increased by 2–5 log₁₀ on samples that were judged organoleptically acceptable after 4 weeks. In another study, *L. monocytogenes* increased in number from 5×10^2 to 2.1×10^5 MPN g⁻¹ on vacuum-packed frankfurters stored at 4 °C for 20 days. Interestingly, non-inoculated control products contained 1.2×10^2 MPN g⁻¹ after the 20-day storage period, after initially testing negative for the organism (Bunčić, *et al.*, 1991). In a more detailed study of vacuum-packaged all-beef, poultry, or beef/pork frankfurters stored at 5 °C for up to 28 days, McKellar *et al.* (1994) found 40 of 61 (65.6%) frankfurters supported growth of *L. monocytogenes*, with an average increase of 1.26 log₁₀ within a 14-day period. Initial lactic acid bacteria concentrations, as well as phenol and nitrite levels, varied considerably during storage, unlike sodium chloride levels. In addition, average pH levels decreased significantly during storage by 0.19 pH units. Several statistical models were derived in an attempt to describe adequately the growth and death of the organism in all frankfurter samples. Although no one model was found to be sufficient, the best model implicated initial and final lactic acid bacteria counts and initial pH as factors that influence the growth of *L. monocytogenes*.

16.2.4 Risk Characterization

Cooked sausages, of which the frankfurter is but one example, are consumed in large quantities throughout the world. It is estimated that 20 billion frankfurters are consumed each year in the US, for an average of 60 frankfurters per person per year. A retrospective case-control study conducted in 1986–1987 involving 154 listeriosis patients found that sporadic cases were significantly more likely to have eaten either frankfurters that had not been fully reheated or chicken meat that was still pink (Schwartz *et al.*, 1988). It was estimated that 20% of sporadic cases were linked to the two food products. A subsequent case-control study identified as potential causes Mexican-style and feta cheeses, foods purchased from the deli section of grocery stores and, again, undercooked chicken, but not frankfurters (Schuchat *et al.*, 1992).

The population of the US in 1998 was 270 million. From 1996–1999, the estimated incidence of listeriosis from the FoodNet system was 5, 5, 6, and 5 cases per million per year, respectively (CDC, 2000) or about 1,350 cases per year. If it is assumed that 1% (13.5 cases) of the total listeriosis cases might be due to frankfurters and there are 20×10^9 servings of frankfurters per year, then that equals 13.5 cases for every 20×10^9 servings among the total population.

Approximately 20% of the US population (54 million) is immunocompromised. If it is assumed that this high-risk population consumes frankfurters at the same rate as the general population (i.e., about 60 frankfurters per year), then the total number of frankfurters consumed by this subpopulation would be $(54 \times 10^6) \times 60 = 3240 \times 10^6$ or 3.24×10^9 . If it is assumed that listeriosis is restricted to this immunocompromised population and 1% (13.5) of the total listeriosis cases might be due to frankfurters, then there would be 13.5 cases for every 3.24×10^9 servings among the high risk population.

The risk associated with frankfurters, however, is likely to be related to growth of *L. monocytogenes* and handling/preparation practices. Between 1993–1999, the prevalence of *L. monocytogenes* among the lots sampled by USDA-Food Safety and Inspection Service declined from 5.3 to 1.8%. Using a single dose approach (Buchanan *et al.*, 1997), one could assume that all cases of listeriosis are associated with the proportion of frank-

furters having a high level of *L. monocytogenes* (e.g., $\geq 1 \times 10^3$ cfu g⁻¹). Although there are no data for the proportion of frankfurters that may have this level, it will be assumed that 5% contain *L. monocytogenes* but only 10% of those have a high level (i.e., $\geq 1 \times 10^3$ cfu g⁻¹) when purchased. The total number of contaminated frankfurters having a high level of *L. monocytogenes* purchased by the high-risk population then would be $3.24 \times 10^9 \times 0.05 \times 0.10 = 16.2 \times 10^6$.

If it is further assumed that the method of handling and reheating is inadequate to eliminate the high level of *L. monocytogenes* in 10% of the servings, then the frequency of exposure to a high level of *L. monocytogenes* would be $16.2 \times 10^6 \times 0.10 = 1.62 \times 10^6$. If 1% of all listeriosis is associated with eating franks, then the 13.5 cases would more likely result from the estimated 1.62×10^6 franks having a high level of *L. monocytogenes* that are handled and prepared in such a manner that illness can result among susceptible individuals.

Thus, the probability (P) that the high-risk population would acquire listeriosis from frankfurters containing high levels of *L. monocytogenes* would be $P = 13.5$ cases per 1.62×10^6 servings (i.e., approximately one case per 120,000 servings) or $P = 8.3 \times 10^{-6}$.

Since numerous assumptions have been made and reliable data are lacking, it is impossible to validate this estimate or to calculate attendant uncertainties. For example, the estimate assumes that all the strains of *L. monocytogenes* detected on frankfurters are equally virulent, which is unlikely. The estimate does suggest, however, that the risk of acquiring listeriosis from frankfurters on a per serving basis is exceptionally low, even among the high-risk population. Experience indicates that when frankfurters are contaminated with a highly virulent strain, cases may occur among the high-risk population (CDC, 1999).

The draft risk assessment conducted jointly by the US Food and Drug Administration and the USDA estimated the median number of cases of listeriosis per serving of frankfurters to be 3.0×10^{-6} , 5.0×10^{-8} and 5.9×10^{-9} for the perinatal, elderly, and intermediate age-based populations, respectively (FDA, 2001). The perinatal population consisted of fetuses and neonates (16 weeks after fertilization to 30 days after birth) with exposure occurring in utero from contaminated food eaten by the pregnant mother. The elderly group included individuals who were 60 or more years of age. The intermediate age group included the remaining population, including susceptible individuals not included as perinatal or elderly, such as cancer, AIDS, and transplant patients, for whom there was insufficient data for them to be considered as a separate population.

On a per serving basis, reheated frankfurters were of low risk relative to 19 other categories of foods studied, ranking 15th out of 20 for the three population groups. Frankfurters consumed without reheating were of high risk, ranking first or second among the 20 categories for the three population groups, assuming that from 1–14% of frankfurters are consumed without reheating. Overall, frankfurters ranked number 4 or 5 among the 20 food categories for the three population groups when the median estimates were used to predict the relative risk for listeriosis on a per annual consumption basis, again assuming that 1–14% of frankfurters are consumed without reheating (FDA, 2001).

16.3 RISK MANAGEMENT

16.3.1 Acceptable Level of Consumer Protection

In most industrialized countries, the annual incidence of listeriosis is between 2–6 cases per million of the population (Rocourt & Bille, 1997). For the risk characterization,

an estimate of 1% of the total cases of listeriosis was used for the cases that might be associated with frankfurter consumption, i.e., 13.5 cases. An example of a public health goal could be to reduce the number of cases attributable to the consumption of frankfurters by some value (e.g., 50% reduction to no greater than 6.75 cases per million of the population).

16.3.2 Establishing a Food Safety Objective

Due to its widespread presence in the environment, eradication of *L. monocytogenes* from the food supply is impossible. There is general agreement that when the organism is ingested in low numbers, even susceptible individuals have little chance of developing listeriosis. A realistic food safety objective (FSO) must therefore be established that would, as far as possible, recognize that it is not possible with current technology to eliminate contamination of foods with *L. monocytogenes*. When total prevention is not possible, measures must be put into place to control recontamination to an acceptable level. To achieve these goals, it is essential that GHP and HACCP programs that are specific to the control of *L. monocytogenes* be applied at all stages of manufacture, storage, transport, and retail. In addition, research should continue to develop additional barriers to control the growth of *L. monocytogenes* in meat and poultry products. This might include the addition of food additives and/or the use of a competitive flora (McMullen & Stiles, 1996).

It is currently recognized that on a daily basis, humans are consuming *L. monocytogenes* in foods at levels of at least 100 cfu g⁻¹ and not becoming ill. For example, data from Denmark show an overall contamination rate for cold-smoked salmon products of around 25–32%, with 4–5% of those samples containing greater than 100 cfu g⁻¹ (Huss, 1997). Even with the large amounts of Danish cold-smoked salmon being consumed worldwide, there have been no recorded cases of listeriosis due to this product. In addition, countries such as Canada have established an action level for *L. monocytogenes* of 100 cfu g⁻¹ for low risk products and have not seen an increase in the baseline levels of human listeriosis. This is also consistent with the estimated dose-response curve for *L. monocytogenes* from Buchanan *et al.* (1997), which includes an estimate for immunocompromised individuals, is reasonably conservative, and allows for the possibility of a single cell causing serious illness (Figure 16–2). Finally, epidemiologic data indicate that foods involved in listeriosis outbreaks are those in which the organism has multiplied and in general have contained levels well in excess of 100 cfu g⁻¹ (see Table 16–1). Thus, on the basis of epidemiologic and prevalence data, the following FSO is proposed:

The concentration of *L. monocytogenes* in frankfurters should not exceed 100 cfu g⁻¹ at the time of consumption.

This proposal is consistent with an earlier recommendation from the International Commission on Microbiological Specifications for Foods (ICMSF) (ICMSF, 1994). It also is compatible with a conclusion of the World Health Organization (WHO)/Food and Agriculture Organization (FAO) risk assessment for *L. monocytogenes* in RTE foods that a more strict tolerance of “not detected in 25 g” does not provide a higher level of protection (Ross *et al.*, 2000). Indeed, the incidence rate for listeriosis in the US, which has maintained the “negative in 25 g” tolerance, is not lower than other industrialized countries that have applied a tolerance of 100 cfu g⁻¹.

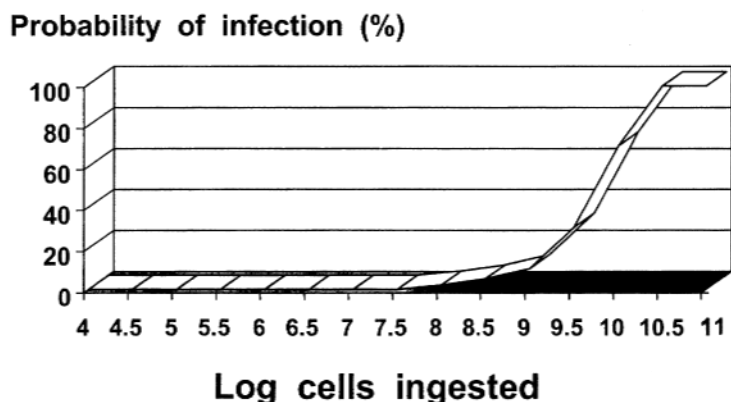


Figure 16-2 Estimated dose response curve for *Listeria monocytogenes*.

16.3.3 Control Measures

It is anticipated that industry can easily meet the proposed FSO for the product at the time of manufacture. However, if frankfurters are recontaminated after the commercial cooking, by the time they are consumed, there is potential for concentrations to exceed 100 g^{-1} , especially with a long refrigerated shelf-life, no additional barriers to growth, and/or temperature abuse. To reduce the incidence of listeriosis (e.g., to fewer than 5 cases per million population) by specifically reducing the number of cases due to frankfurters (e.g., from 13.5 to no greater than 6.75 cases), then other risk management options need be considered.

A flow diagram for a typical frankfurter operation is shown in Figure 16-1. HACCP systems and GHP procedures must be used to achieve the expected level of control and meet the FSO. It is important to consider available control measures in the context of the steps used in manufacture. In this example, controlling *L. monocytogenes* so the concentration will not exceed 100 cfu g^{-1} when frankfurters are consumed will involve combinations of the following control measures:

- *Controlling initial levels in raw materials*—Handling raw materials during storage and preparation to minimize an increase in numbers due to contamination or growth.
- *Reducing levels during cooking of frankfurters in the manufacturing process*—Cooking to eliminate *L. monocytogenes* in the raw meat emulsion. Establishing a performance criterion for cooking that results in process criteria (i.e., critical limits) that can be incorporated into the HACCP plan.
- *Preventing recontamination between cooking and packaging*—Minimizing recontamination between cooking and packaging by adopting GHP measures, e.g., separating raw from cooked product, and an environmental management and monitoring program.
- *Reducing levels in cooked product after packaging (in-pack pasteurization)*—Applying commercially feasible pasteurization processes for pasteurizing the product after packaging.
- *Preventing increase in levels between packaging and preparation for serving*—Controlling the increase of *L. monocytogenes* that subsequently may occur with

recontamination and growth in packaged frankfurters during storage and distribution. Examples of control measures might include: adding safe, acceptable additives to control growth of *L. monocytogenes* on frankfurters, using code-dating practices and improved chill chain management to preclude an unacceptable increase in *L. monocytogenes*, or freezing the product.

- *Reducing levels prior to consumption*—Improving storage and handling practices and inactivating *L. monocytogenes* through effective cooking and reheating prior to consumption. These outcomes could be achieved as control measures in food service operations or through education of consumers, particularly the more susceptible population and their health care providers.

16.3.4 Performance Criteria

A performance criterion is the required outcome of one or more control measures at a step or combination of steps applied to achieve an FSO (see Chapter 3). Performance criteria are usually applied at steps where hazards can either be reduced or where hazards may increase.

To arrive at a performance criterion for the control measures needed to meet an FSO for frankfurters, the equation from chapter 3 can be used:

$$H_0 - \Sigma R + \Sigma I \leq \text{FSO}$$

$$H_0 - \Sigma R + \Sigma I \leq 2.0$$

where:

FSO = Food safety objective

H_0 = Initial level of the hazard

ΣR = Total (cumulative) reduction of the hazard from processing, etc.

ΣI = Total (cumulative) increase of the hazard

FSO, H_0 , R, and I are expressed in $\log_{10}$ units.

Manufacturers of frankfurters should assume that *L. monocytogenes* will be present in all species and types of raw meat and poultry. This is evident from reports in the literature and results from surveys such as the USDA baseline studies for ground beef, ground turkey, and ground chicken (Table 16-2) (USDA, 1995a; USDA, 1995b; USDA, 1996). Data of this nature can be used to establish the initial concentration (H_0). Another option would be to generate similar data from raw emulsions collected at the facility prior to cooking.

The USDA baseline studies reported the prevalence of *L. monocytogenes* in ground beef, chicken, and turkey to be in the range of 18–35% when analyzing 25 g samples. Further analysis of the positive samples yielded low numbers of *L. monocytogenes*. It can be concluded from the data that concentrations of 100 g^{-1} or fewer would be expected under normal conditions. Further reducing the likelihood of higher concentrations is the effect of blending and emulsification that occurs in the manufacturing process. These steps disperse the cells more uniformly throughout the meat emulsion and result in an overall lower number g^{-1} with little likelihood of localized pockets of higher concentrations.

16.3.4.1 Controlling Initial Levels in Raw Materials

Consideration should be given to multiplication of *L. monocytogenes* that might occur as raw meat and poultry is stored and prepared for cooking. It is important to note that

Table 16-2 USDA Baseline Survey Results for Ground Beef, Ground Turkey, and Ground Chicken

	Ground beef (563 samples)	Ground turkey (165 samples)	Ground chicken (162 samples)
Prevalence based on 25 g samples	18%	23%	35%
Geometric mean for positive samples only	3.9/g	3.8/g	2.24/g
95% confidence level	2.2–7.2/g	0.4–2.9/g	1.06–4.7/g

Samples found positive by the qualitative method were further analyzed to determine the number of *L. monocytogenes* g⁻¹ of ground meat. The results for the positive samples were as follows:

Number of <i>L. monocytogenes</i> /g	Ground beef (99 samples)	Ground turkey (52 samples)	Ground chicken (59 samples)
< 0.03	45.2	82.5	56.1
0.03–0.29	0.0	0.0	0.0
0.3–2.9	30.2	6.7	29.3
3.0–29.9	15.0	0.0	10.5
30–299.9	9.6	10.9	3.2
300 or higher	3 samples had > 110/g	0.0	0.9

Note: These data indicate, for example, that in the case of ground beef, 563 samples were analyzed using 25 g each. Only 99 samples were found to contain *L. monocytogenes*. Upon further analysis of the 99 samples, 90.4% had fewer than 30 cfu gram⁻¹. From an overall perspective, only 3 of the total 563 samples had greater than 110 *L. monocytogenes* gram⁻¹, the upper limit of detection used for testing.

In the case of ground turkey, 165 samples were analyzed using 25 g each. Only 52 were found to contain *L. monocytogenes*. Upon further analysis of the 52 samples, 89.2% had fewer than 30 cfu gram⁻¹. From an overall perspective, the highest number detected among the 165 samples was a sample containing 93 cells gram⁻¹.

the samples for the baseline studies were collected, packaged, and shipped in refrigerated containers to the laboratory. Samples were considered acceptable for analysis if they were received at the laboratory by the day after collection and with a temperature of 10 °C or less. Hence, the samples were at least one day older than when collected and some temperature abuse may have occurred.

To further place the possibility of multiplication during preparation into perspective, the data in Table 16-3, derived from the USDA Pathogen Modeling Program, indicate that from 4–9 days at 4 °C would be required for *L. monocytogenes* to increase 10-fold in meat or poultry with no added salt (0.5%) (Buchanan & Whiting, 1996). Food MicroModel (Ver 3.02) similarly predicts that it would take around 6 days for a 10-fold increase of *L. monocytogenes* (4 °C, 0.5% NaCl, pH 6.5). Longer times would be required in raw materials containing added salt. For example, the mechanically deboned poultry meat used for manufacturing frankfurters typically contains added salt and sodium nitrite and is chilled to well below 4 °C before shipping to the frankfurter manufacturing plant. The values in Table 16-3 include the lag phase estimated for each set of parameters. The length of the lag phase that actually occurs in raw meat under commer-

Table 16-3 Days for a 1 Log₁₀ (10-Fold) Increase in *L. monocytogenes* at 4 °C and pH 6.0

Sodium chloride (%)	Days for 10-fold increase			
	Aerobic conditions (e.g., meat at top of container)		Anaerobic conditions (e.g., meat below surface of container)	
	No NaNO ₂ Added	150 µg g ⁻¹ NaNO ₂ added	No NaNO ₂ Added	150 µg g ⁻¹ NaNO ₂ added
0.5	5.3	8.9	4.1	8.8
1.0	5.6	9.4	4.5	9.6
1.5	6.0	10.0	4.9	10.5
2.0	6.4	10.6	5.3	11.5
2.5	6.8	11.4	5.8	12.4
3.0	7.3	12.3	6.2	13.4

cial conditions has not been determined, but the raw meat and/or poultry used for the manufacture of frankfurters is normally less than 7 days from the date of slaughter.

From the data in Table 16-2 it is evident that the numbers of *L. monocytogenes* are very low in raw meat and poultry. Furthermore, the data in Table 16-3 indicate that significant multiplication during preparation is unlikely. For this example, it will be assumed that the initial concentration may under certain circumstances increase 10-fold prior to commercial cooking. Thus, in this example, the initial number in the raw materials prior to cooking is assumed to be no more than 1000 g⁻¹ (i.e., H₀ = 3).

16.3.4.2 Reducing Levels during Cooking of Frankfurters in the Manufacturing Process

If it is assumed that initial numbers of *L. monocytogenes* in the raw materials could be as high as 1000 g⁻¹ (i.e., H₀ = 3), then a performance criterion for the reduction step (i.e., cooking) to meet the FSO would, theoretically, only need to be a 1 log₁₀ reduction as given below and illustrated in Figure 16-3a.

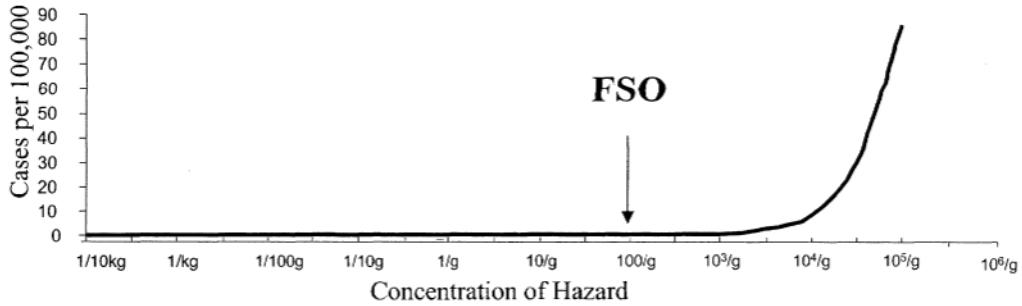
$$H_0 - \Sigma R + \Sigma I \leq \text{FSO}$$

$$3 - \Sigma R + 0 \leq 2.0$$

$$\Sigma R = 1$$

However, if any viable cells remain after cooking, by the time the frankfurters are consumed, there is the potential for concentrations to exceed 100 g⁻¹, especially with a long refrigerated shelf-life, no additional barriers to growth, and/or if temperature abuse occurs. Therefore, to ensure that survivors do not remain and multiply during distribution and storage, it is necessary to apply a more stringent cooking process. For this example, it will be assumed that a 6 log₁₀ reduction (R = 6) during cooking will achieve the desired result, given an initial concentration of no greater than 1000 g⁻¹ (H₀ = 3). Thus, a 6 log₁₀ reduction would result in a final concentration of ≤ 1 cfu kg⁻¹ after cooking. This could be expressed as a performance criterion:

The concentration of *L. monocytogenes* after cooking shall be ≤ 1 cfu kg⁻¹.



1) FSO could be met with only 1 $\log_{10}$ reduction:

$$\begin{array}{c} \leftarrow H_0 \\ \Sigma R = 1 \end{array}$$

2) Cooking during manufacture (6 $\log_{10}$ reduction, $\Sigma R = 6$) is applied to reduce levels to meet a performance criterion of $\leq 1 \text{ kg}^{-1}$ ($\leq 10^{-3}$ per g)

$$\begin{array}{c} \leftarrow H_0 \\ \Sigma R = 6 \end{array}$$

Figure 16-3a Control measure: cooking.

This is demonstrated in the following equation and illustrated in Figure 16-3a.

$$H_0 - \Sigma R + \Sigma I \leq 1/\text{kg} (\leq 10^{-3}/\text{g})$$

$$3 - \Sigma R + 0 \leq -3$$

$$\Sigma R = 6$$

If the initial number is less than 1000 g^{-1} through raw material selection and/or preventing any increase during handling and storage (e.g., $\leq 100 \text{ g}^{-1}$), then a 5 $\log_{10}$ reduction would be adequate to meet the performance criterion of $\leq 1 \text{ kg}^{-1}$.

16.3.4.3 Prevent Recontamination Following Cooking

L. monocytogenes is a very hardy organism and can be recovered from different locations in the plant environment, depending on the level of control. In the absence of an effective control program, the organism can persist for prolonged periods in frankfurter production environments. During a 1989 investigation, the same strain of *L. monocytogenes* was recovered from a patient, from leftover frankfurters in the patient's refrigerator, and 4 months later from the frankfurter peeler in the manufacturing facility (Wenger *et al.*, 1990). In another outbreak in the US associated with frankfurters, it was reported that removing a refrigeration unit resulted in new or increased contamination of production equipment (CDC, 1999). This outbreak involved cases from August 2, 1998 through January 16, 1999, suggesting that multiple lots of product were implicated. Attempts to verify that the strain had become established in the environment, however, were unsuccessful. Thus, even if process criteria are met for cooking, recontamination of processed

product is a possibility unless proper attention has been paid to the GHPs that are targeted toward control of *L. monocytogenes*.

Certain measures that fall within the realm of GHP have been found to be important for control of *L. monocytogenes* in cooked meat and poultry operations. The measures include plant layout, equipment design, maintaining equipment, cleaning and sanitizing procedures that are specific to listeriae control, maintaining a clean and dry environment, use of enclosed steam to sanitize equipment as a scheduled routine procedure, and low temperature storage (Tompkin *et al.*, 1999).

Two factors determine the effectiveness of a listeriae control program: routine environmental testing and the response to a positive finding. Without an environmental testing program, it is not possible to assess control. Furthermore, in the event a positive product contact sample is detected, corrective actions should be initiated to eliminate the source of contamination, thereby minimizing the risk of product contamination. To verify control, plants should implement an environmental monitoring program for *L. monocytogenes* or an indicator, such as *L. innocua* or *Listeria spp.* The program must be specific to each plant and should detail the areas to be sampled, the analytic method to be used, the frequency of sampling, and the action to be taken when *Listeria spp.* are detected (Tompkin *et al.*, 1992, 1999).

An effective monitoring program to assess control of the cooked product environment should consider the following strategies:

- preventing the establishment of *L. monocytogenes* in niches or other sites that can lead to contamination of RTE foods,
- implementing a sampling program that can assess in a timely manner whether the environment in which RTE foods are produced is under control,
- responding to each positive product contact surface sample as rapidly and as effectively as possible, and
- verifying that the problem has been corrected and providing data (e.g., tabulated, trends in graphical form) to facilitate short- and long-term assessment of control.

Experience in frankfurter operations indicates that sites within the cooked product environment wherein *L. monocytogenes* becomes established and multiplies (i.e., niches) are important contributing factors to product contamination. The sites serve as a reservoir from which the pathogen is dispersed and contaminates product contact surfaces. Furthermore, an environmental sampling program, such as described in Chapter 11, is necessary to assess whether the environment is under control and to detect the presence of a niche.

Response to a positive product contact sample (e.g., a sponge sample collected from a conveyor belt used in a frankfurter operation) is best when the following items are considered. Assuming an effective control program is in place, the primary source of *L. monocytogenes* in frankfurter operations is contamination from one or more niches. In general, contamination flows downstream with the process flow, much like a river. To regain control, it is necessary to locate the source where growth and contamination are occurring. This can best be accomplished by creating a map of the cooked product rooms and the equipment layout (see Chapter 11). The results of samples collected from each piece of equipment should be recorded on the map, including both positive and negative results. The layout map should then be reviewed for patterns. Which sites are more frequently positive? Where in the process flow do the first positives occur? It is important when seeking the source to analyze all the sponge samples separately and not as com-

posites. In addition, sampling should be aggressive both in terms of location and frequency throughout the period of operation. Unfortunately, niches can rarely be detected unless the equipment is operating and product is being produced. While pursuing the source of contamination, consideration must be given to the possibility that a niche may not be involved (contamination could result, e.g., from someone touching the floor or other unclean surface and returning to handle exposed product).

When equipment has been identified as the source, the following steps can be effective. First, dismantle the equipment, collecting samples of suspicious material as this is being done. Second, replace obvious defective parts (e.g., hollow rollers) and clean and sanitize the equipment as it is being reassembled. If this procedure fails to eliminate the cause (or source) of contamination, it may be necessary to remove sensitive electronics, oil, and grease, and heat the equipment. This can be accomplished by placing the equipment in an oven and heating to an internal temperature of 71 °C with high humidity. Alternatively, the equipment can be shrouded with a tarp and steam can be injected into the space until the equipment reaches an internal temperature of 71 °C. Internal temperatures can be monitored with strategically placed thermocouples.

In frankfurter operations the following are examples of identified sources of contamination:

- rubber gaskets around the doors and other openings to the brine chill system
- saturated insulation on pipes carrying refrigerant to the brine chill system
- peelers that remove artificial casings before packaging
- casing removal systems
- hollow rollers on conveyors
- on/off valves and buttons for various equipment
- within complex collating equipment
- hollow support rods on the frames of equipment.

It is difficult to assign a performance criterion to recontamination, as any recontamination has the potential to reach high numbers through growth during the subsequent distribution and storage.

To ensure that the performance criterion of $\leq 1 \text{ kg}^{-1}$ (i.e., $\leq 10^{-3}$ per g) is not exceeded due to recontamination, there must be no recontamination and “I” in the following equation must be zero. This is illustrated in Figure 16–3b.

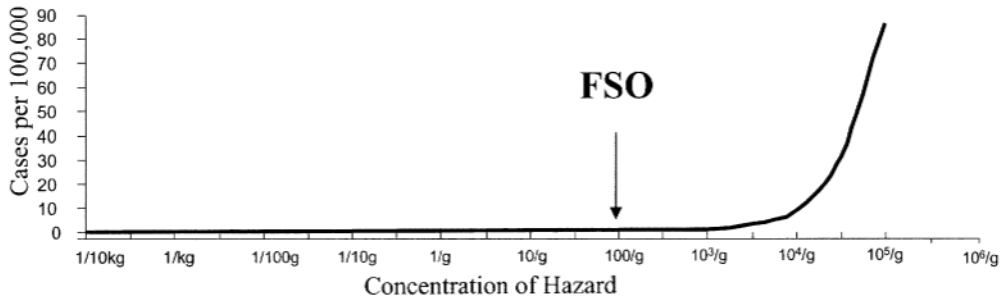
$$H_0 - \Sigma R + \Sigma I \leq 1 \text{ kg}^{-1} (\leq 10^{-3} \text{ per g})$$

$$3 - 6 + \Sigma I \leq -3$$

$$\Sigma I = 0$$

16.3.4.4 Reducing Levels in Cooked Product after Packaging (In-Package Pasteurization)

Experience indicates that recontamination after cooking is the most common reason for the presence of *L. monocytogenes* in packaged, cooked sausages, such as frankfurters. If the increase in concentration due to recontamination is assumed to be as high as 10 g^{-1} , then an in-package pasteurization treatment could be applied as a means to achieve a 4 log reduction and still meet the performance criterion of $\leq 1 \text{ kg}^{-1}$ (i.e., 10^{-3} per g). As demonstrated in the following equation, if cooking provides an initial 6 log reduction and recontamination results in 10 cfu g^{-1} , then an in-package pasteurization treatment could be used to provide a final 4 log reduction and meet the performance criterion of $\leq 1/\text{kg}$.



Preventing recontamination between cooking and packaging through effective GHPs and an environmental sampling program

$$H_0 - \Sigma R + \Sigma I \leq \text{Performance criterion (1 kg}^{-1} \text{ or } \leq 10^{-3} \text{ per g)}$$

$$3 - 6 + \Sigma I \leq -3$$

$$\Sigma I \leq 0$$

Figure 16-3b Control measure: preventing recontamination.

Hence, the combined reduction steps for cooking and in-package pasteurization result in a 10 log reduction (i.e., $\Sigma R = 10$). This is illustrated in Figure 16-3c.

$$H_0 - \Sigma R + \Sigma I \leq 1 \text{ kg}^{-1} (\leq 10^{-3} \text{ per g})$$

$$3 - \Sigma R + 4 \leq -3$$

$$\Sigma R = 10 \text{ (Cooking 6D, In-pack pasteurization 4D)}$$

16.3.4.5 Preventing Increase in Levels between Packaging and Preparation for Serving

In this and the next example, an in-package pasteurization treatment is not applied. If the increase in concentration due to recontamination is again assumed to be as high as 10 g^{-1} (i.e., a 4 log₁₀ increase from 1 kg^{-1}) and if the FSO of 100 g^{-1} at time of consumption is to be met, there can be no greater than a 10-fold increase during storage and distribution before the food is consumed (see also Figure 16-3d).

$$H_0 - \Sigma R + \Sigma I \leq \text{FSO}$$

$$3 - 6 + 4 \leq 2$$

$$\Sigma I \leq 1$$

If recontamination occurs, then an increase of 10-fold or more must be controlled by adding inhibitory ingredients, freezing the product for distribution, and/or establishing use by date coding that are within the time for a 10-fold increase.

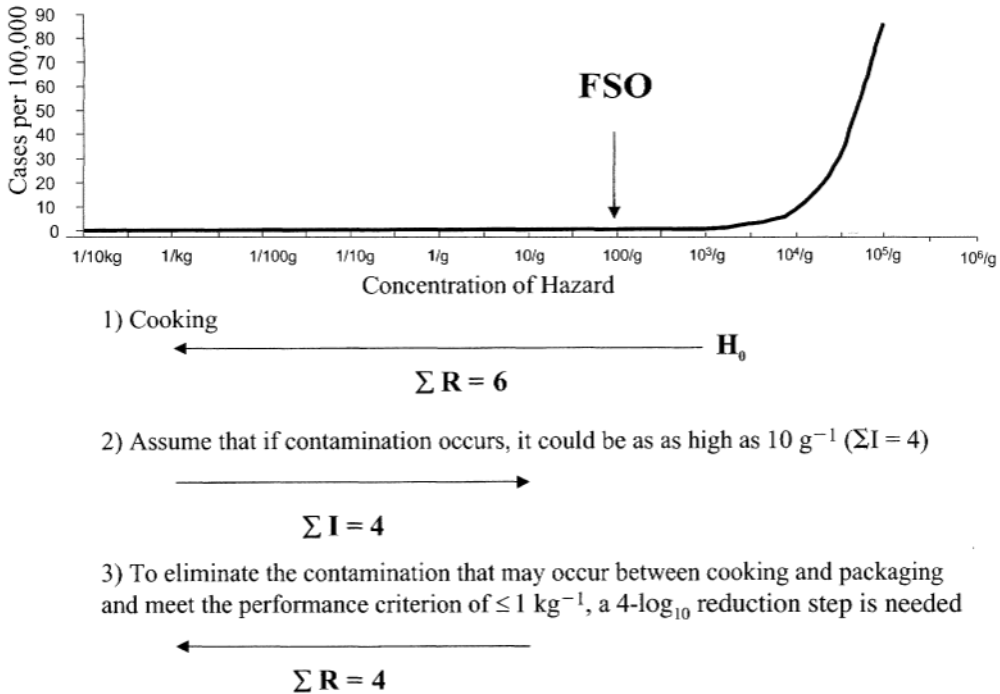


Figure 16-3c Control measure: reducing levels using in-package pasteurization.

16.3.4.6 Reducing Levels Prior to Consumption

If the product is recontaminated with *L. monocytogenes* to a level of 10 g^{-1} ($I=4$) and growth does occur during subsequent distribution and storage, then the handling and preparation procedures become very important for susceptible consumers. If it is assumed that there could be a 5 log_{10} increase due to growth ($I=5$) between when the frankfurters are cooked in the factory and when they are reheated for serving, then the combined increase after cooking would be $\Sigma I = 4 + 5$ for a total increase of 9 log_{10} . At the time of preparation, the frankfurters would have a level of 10^6 g^{-1} . This could be represented as follows (see also Figure 16-3e).

$$H_0 - \Sigma R + \Sigma I \leq \text{FSO}$$

$$3 - 6 + 9 \leq \text{FSO}$$

$$H_0 - \Sigma R + \Sigma I \leq 6$$

Therefore, to meet the required FSO of $\leq 100 \text{ g}^{-1}$, an additional reduction step during preparation for serving would be required prior to consumption, as follows.

$$H_0 - \Sigma R + \Sigma I \leq \text{FSO}$$

$$3 - \Sigma R + 9 \leq 2$$

$$\Sigma R \leq 10$$

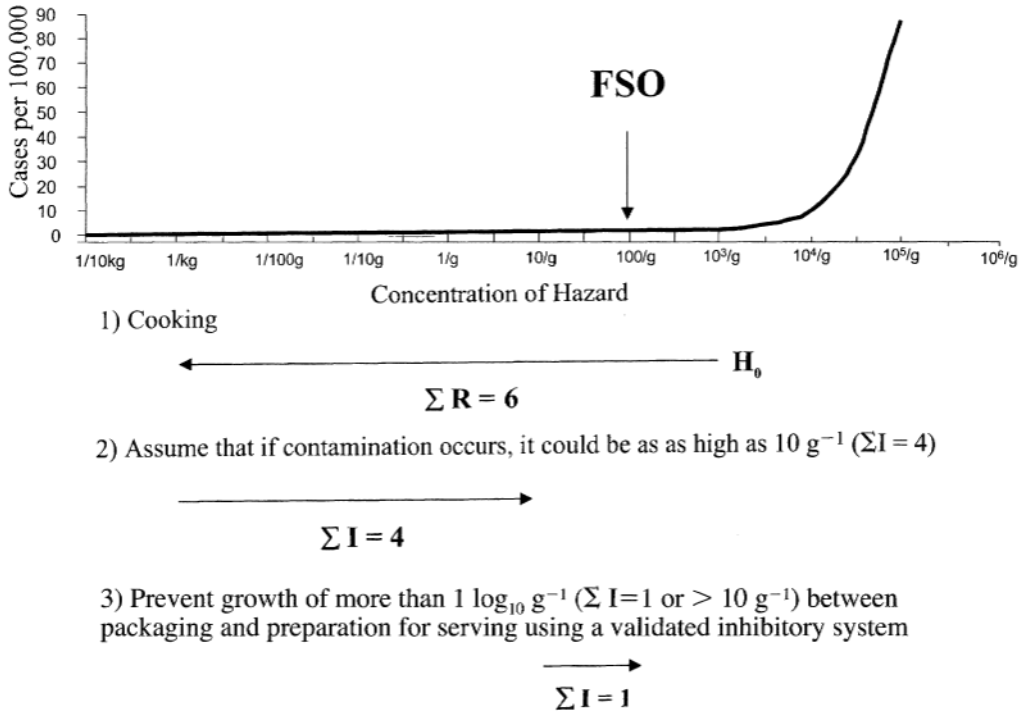


Figure 16-3d Control measure: use of inhibitor(s) to prevent growth after packaging.

After taking account of the $6 \log_{10}$ reduction (i.e., $\Sigma R = 6$) that occurs when the frankfurters were cooked during manufacture, the reduction during preparation for serving must be at least as follows (Figure 16-3e).

$$\Sigma R \leq 4$$

The final reheating before serving then must reduce the level of *L. monocytogenes* by $4 \log_{10}$ to ensure that the frankfurters will meet the FSO of 100 g^{-1} at the time they are consumed.

16.4 PROCESS AND PRODUCT CRITERIA

Each manufacturer must determine the parameters for cooking that will provide the desired product quality and cost, and, in this example, ensure that the performance criterion (i.e., $< 1 \text{ kg}^{-1}$) is met for cooking. The parameters adopted by one manufacturer may differ from those adopted by others. This is due to differences in equipment, desired product quality, type of raw materials (e.g., beef, pork, chicken and/or turkey), type of casing (e.g., natural, artificial), method of applying smoke (e.g., natural, liquid), fat content, and other factors. To meet the performance criterion requires knowledge of the heat sensitivity of *L. monocytogenes*, particularly in frankfurter emulsions.

Historically, the minimum internal requirement set by USDA for cooked sausage was 64.4°C . For frankfurters, industry typically applied processes to achieve at least 68.3°C . Since about 1980–1985, many processors have increased the temperature to 71°C , or

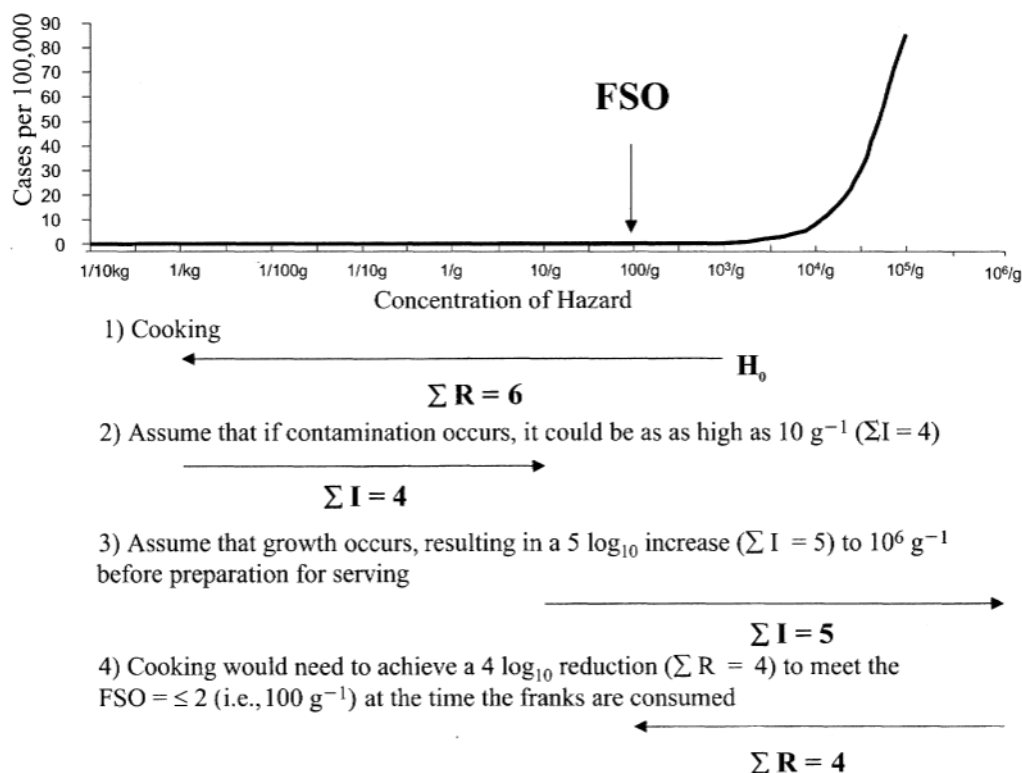


Figure 16-3e Control measure: reducing levels prior to consumption.

higher, to achieve greater consistency in product quality and refrigerated shelf-life. Even with the minimum requirement of 64.4°C , however, there was no evidence that cooked sausages were implicated as a source of foodborne illness due to survival of enteric pathogens, probably due to the low concentration of enteric pathogens present initially and their sensitivity to heat. In the case of *L. monocytogenes*, the data in Table 16-4 indicate that survival also is unlikely to occur. Even with extensive testing for the pathogen since 1988, there has been no documented incident in which *L. monocytogenes* surviving the heat process was the underlying reason for a contaminated cooked sausage.

The thermal process can be validated by various means. One could be to conduct thermal destruction tests in the laboratory using frankfurter emulsion inoculated with a mixture of *L. monocytogenes* isolates from meat operations. Another may be to perform in-house challenge experiments with nonpathogenic surrogate organisms, such as *L. innocua*. Various items, such as provided in Chapter 3, should be considered when conducting such studies to validate a thermal process. A third approach to validation is to review the published literature for data on thermal resistance. For example, numerous studies have been published that report thermal destruction values for *L. monocytogenes* in various meat and poultry products (ICMSF, 1996; Farber & Peterkin, 1999; Zaika *et al.*, 1990; Fain *et al.*, 1991).

The data summarized in Table 16-4 provide lethality values for *L. monocytogenes* in meat, poultry, and seafood cooked to temperatures within the range of 60 – 65°C . A number of factors influence the rate of death, but from the data, it can be concluded that even

Table 16-4 Reported Thermal Destruction Values for *L. monocytogenes* in Meat, Poultry, and Seafood Products

Product	Temp (°C)	D value* (min.)	Reference
Ground meat with cure mix	64	2.28	Farber (1989)
Ground meat	64	<1.01	
Beef	65	0.93	Mackey <i>et al.</i> (1990)
Chicken leg	65	0.53	
Chicken breast	65	0.52	
Buffer	65	0.29	Boyle <i>et al.</i> (1990)
Meat slurry	65	0.75	
Lean beef	62.8	0.6	Fain <i>et al.</i> (1991)
Fatty beef	62.8	1.2	
Beef steak	63.9	2.2	Gaze <i>et al.</i> (1989)
Chicken breast	63.9	1.6–1.8	
Ham—control culture	60	1.8	Carlier <i>et al.</i> (1996)
Ham—heat-shocked culture	60	3.5	
Ham—"resistant" culture	60	1.0	
Mussels	62.2	1.85	Bremer & Osborne (1995)
Crabmeat	60	1.3–2.6	Harrison & Huang (1990)
Lobster meat	62.8	1.1	Budu-Amoako <i>et al.</i> (1992)
Salmon	65	0.9–1.2	Embarek & Huss (1993)
Cod	65	0.27–0.28	
Cured sausage blend of 66% pork and 33% beef	63.9	3.3	Farber & Brown (1990)
Cured sausage blend of 66% pork and 33% beef	30 min. at 47.8 °C, then heat to 63.9 °C	4.2	
Cured sausage blend of 66% pork and 33% beef	60 min. at 47.8 °C, then heat to 63.9 °C	4.7	
Cured sausage blend of 66% pork and 33% beef	120 min. at 47.8 °C, then heat to 63.9 °C	8.0	

*D value = time in minutes at a specified temperature for a one log₁₀ (i.e., ten-fold) reduction in the number of *L. monocytogenes*.

these lower temperatures are effective for killing *L. monocytogenes* in a wide variety of foods. Such data can be used to arrive at alternative thermal processes for sausage products, such as frankfurters, and to consider the disposition of products in the event of a deviation. The adequacy of a thermal process based on an internal temperature of 70 °C can be determined from the data in Table 16-5.

An example of a default process criterion to achieve a 6 log₁₀ reduction for in-pack pasteurization can be found in the draft Code of Practice developed by a Belgium-Dutch Chilled Meals Working Group, which recommends an internal heat treatment of 70 °C for 2 minutes for elimination of *L. monocytogenes* in chilled, long-life pasteurized foods (Lund & Notermans, 1993). The advantage in using a performance criterion rather than a default process criterion is that it allows flexibility in how the performance criterion is achieved. This would facilitate the development of new technology that may offer novel ways to achieve the same end result.

Table 16-5 Reported D values for *L. monocytogenes* at 70 °C

Product	D value (min)*	Reference
Beef	0.14	Mackey <i>et al.</i> (1990)
Chicken leg	0.11	
Chicken breast	0.13	
Meats (predicted value)	0.13	Boyle <i>et al.</i> (1990)
Meat slurry	0.23	
Beef slurry (pH 5.9)	0.23	
Phosphate buffer (pH 7.2)	0.15	Gaze <i>et al.</i> (1989)
Chicken	0.16, 0.20	
Beef steak	0.20, 0.14	
Turkey meat emulsion	0.35	Fain <i>et al.</i> (1991)
Salmon, cod	0.13–0.17	Embarek <i>et al.</i> (1993)
Sucrose solution, a_w 0.98	0.15 @ 68.3C	Sumner <i>et al.</i> (1991)
Sucrose solution, a_w 0.96	0.30 @ 68.3C	
Raw milk	0.05 @ 68.9C	Bradshaw <i>et al.</i> (1985)
Raw milk	0.015 @ 71. 7C	

*D value = time in minutes at a specified temperature for a one $\log_{10}$ (i.e., ten-fold) reduction in the number of *L. monocytogenes*.

Product criteria that are intended to prevent growth after packaging could be validated by performing challenge studies using surface-inoculated frankfurters to assess the likely increase (ΣI) of *L. monocytogenes* to $\geq 100 \text{ g}^{-1}$ before the recommended use-by date. Challenge studies could involve, for example, using commercial product inoculated and packaged in a laboratory with a mixture of five isolates of *L. monocytogenes* and then stored at temperatures to which the product would be exposed during storage and distribution. Many factors should be considered when conducting such studies (see Chapter 3). Predictive modeling also can provide an estimate of the pathogen's behavior and possible growth or inactivation (McKellar *et al.*, 1994; Armitage, 1997; McClure *et al.*, 1997; McDonald & Sun, 1999; Buchanan & Whiting, 1996).

Examples of additives that have been found effective for inhibiting *L. monocytogenes* include sodium lactate, sodium acetate, and sodium diacetate (Schmidt, 1993; Schmidt & Leistner, 1993; Qvist *et al.*, 1994; Wederquist *et al.*, 1994, 1995; Blom *et al.*, 1997; Tompkin, 1999; Juncher *et al.*, 2000; Hoogenkamp & Wales, 2000).

A uniform procedure has not been developed to validate code-dating practices to ensure the safety of perishable foods with extended shelf-life (e.g., frankfurters and other cooked meat or poultry products). As discussed in Chapter 3, a number of factors must be considered when validating the effectiveness of one or more steps along the food chain. The following factors should be considered when performing a validation study for code-dating frankfurters:

- physiological state of the inoculum (e.g., 24 h broth culture)
- method of inoculation (surface)
- inoculum level (per package or per g)
- source and number of strains

- virulence of the strains (outbreak strains should be avoided in most laboratories)
- temperature(s) of storage
- natural competitive flora (commercial v. pilot plant product)
- product formulation (fermentable carbohydrate and rate of acid production).

16.5 GHP AND HACCP

Certain aspects of GHP must be controlled to minimize recontamination of cooked products, as has been discussed in this chapter and Chapter 11. In this example, a performance criterion of $\leq 1 \text{ kg}^{-1}$ was established for the cooked product and for in-package pasteurization to ensure that the FSO of $\leq 100 \text{ g}^{-1}$ is met. This led to the establishment of process criteria that could be adopted as critical limits in the HACCP plan. The critical limits typically consist of a holding time and internal temperature, their selection being influenced by the cooking system (e.g., continuous or batch oven). If, for example, 71°C is adopted, the critical limit for cooking frankfurters during their manufacture could be stated as “an internal temperature of 71°C ”, a hold time not being necessary.

With a properly designed and implemented HACCP plan, there would be no benefit to testing the cooked product for further validation of the process or to verify that each lot has been cooked correctly. If an event occurs requiring such testing, then a sampling plan of $n = 5$, $c = 0$, $m = 0/25$ could be used. This would provide 95% confidence of detecting a positive lot if the mean count was 0.03 cfu g^{-1} or greater.

16.6 ACCEPTANCE CRITERIA FOR FINAL PRODUCT

16.6.1 Organoleptic

No organoleptic criteria are applicable to assess the likely presence of *L. monocytogenes* in frankfurters.

16.6.2 Chemical and Physical

No chemical or physical criteria are applicable to assess the likely presence of *L. monocytogenes* in frankfurters.

16.6.3 Microbiological

When it is known that a product has been pasteurized in the pack, sampling final product would provide no added value and is not recommended. Likewise, for those plants that are using a validated kill step for cooking and where an effective environmental sampling program documents that the risk of recontamination is being controlled, there is little value in testing end product. The reason is that a comprehensive management system can maintain frequencies of contamination to less than 0.5%. Under these circumstances, the frequency of defective units is too low for detection with any practical sampling plan (see Chapters 6, 7, and 17).

Where product is crossing international borders and nothing is known about the product or the manufacturing process, end-product testing may be appropriate. The ICMSF has provided guidance on sampling plans for *L. monocytogenes* in a variety of foods and conditions (van Schothorst, 1996). For frankfurters that have not been in-pack pasteur-

ized and, if contaminated, growth could occur (e.g., no added inhibitors), ICMSF sampling case 12 ($n = 20$, $c = 0$) would apply. When case 12 is applied and 25 g analytical units are used in a two-class sampling plan, which would be typical for *L. monocytogenes*, there is 95% probability that a positive lot would be detected when the mean concentration is 1 cfu in 186 g or greater. This assumes a log-normal distribution and standard deviation of 0.8. Thus, the sampling plan performance would be adequate to detect lots that exceed an FSO of $\geq 100 \text{ g}^{-1}$. If the frankfurters are intended specifically for highly susceptible individuals (e.g., hospitals), then case 15 would apply ($n = 60$, $c = 0$). When case 15 is applied and 25 g analytical units are used, there is a 95% probability that a positive lot would be detected when the mean concentration is 1 cfu in 526 g or greater.

16.7 REFERENCES

- Anonymous (1989). 600, 000 pounds of turkey hot dogs recalled by Plantation. *Food Chem News* 32, 52–53.
- Armitage, N. H. (1997). Use of predictive microbiology in meat hygiene regulatory activity. *Int J Food Microbiol* 36, 103–109.
- Audurier, A., Pardon, P. & Martin, C. (1980). Experimental infection of mice with *Listeria monocytogenes* and *L. innocua*. *Annals Microbiol (Paris)* 131B, 47–57.
- Baker, M. & Wilson, N. (1993). Microbial hazards of seafood: a New Zealand perspective. *Communic Diseases New Zealand* 93, 12–15.
- Bemrah, N., Sanaa, M., Cassin, M. H. *et al.* (1998). Quantitative risk assessment of human listeriosis from consumption of soft cheese made from raw milk. *Prevent Vet Med* 37, 129–145.
- Ben Embarek, P. K. & Huss, B. H. H. (1993). Heat resistance of *Listeria monocytogenes* in vacuum packaged pasteurized fish fillets. *Int J Food Microbiol* 20, 85–95.
- Bille, J. (1990). Epidemiology of human listeriosis in Europe, with special reference to the Swiss outbreak. In *Foodborne Listeriosis*, pp. 71–74. Edited by A. J. Miller, J. L. Smith & G. A. Somkuti. Society for Industrial Microbiology. New York: Elsevier.
- Blom, H., Nerbrink, E., Dainty, R. *et al.* (1997). Addition of 2.5% lactate and 0.25% acetate controls growth of *Listeria monocytogenes* in vacuum-packed, sensory-acceptable servelat sausage and cooked ham stored at 4°C. *Int J Food Microbiol* 38, 71–76.
- Boyle, D. L., Sofos, J. N. & Schmidt, G. R. (1990). Thermal destruction of *Listeria monocytogenes* in a meat slurry and in ground beef. *J Food Sci* 55, 327–329.
- Bradshaw, J. G., Peeler, J. T., Corwin, J. J. *et al.* (1985). Thermal resistance of *Listeria monocytogenes* in milk. *J Food Prot* 48, 743–745.
- Bremer, P. J. & Osborne, C. M. (1995). Thermal death times of *Listeria monocytogenes* in green shell mussels (*Perna canaliculus*) prepared for hot smoking. *J Food Prot* 58, 604–608.
- Brett, M. S. Y., Short, P. & McLauchlin, J. (1998). A small outbreak of listeriosis associated with smoked mussels. *Int J Food Microbiol* 43, 223–229.
- Broome, C. V., Gellin, B. & Schwartz, B. (1990). Epidemiology of listeriosis in the United States. In *Foodborne Listeriosis*, pp. 61–65. Edited by A. J. Miller, J. L. Smith & G. A. Somkuti. Society for Industrial Microbiology, New York: Elsevier.
- Buchanan, R. L. & Whiting, R. C. (1996). *USDA Pathogen Modeling Program*, version 5.1. Philadelphia, PA: Agricultural Research Service, U. S. Department of Agriculture.
- Buchanan, R. L., Damert, W. G., Whiting, R. C. & van Schothorst, M. (1997). The use of epidemiologic and food survey data to estimate a conservative dose-response relationship for *Listeria monocytogenes* levels and incidence of listeriosis. *J Food Prot* 60, 918–922.
- Buchanan, R. & Lindqvist, R. (2000). *Topic 1: Hazard Characterization of Listeria monocytogenes in Ready-to-Eat Foods*. Report of the Joint FAO/WHO Expert Consultation on Risk Assessment of Microbiological Hazards in Foods. Rome: FAO Headquarters.

- Budo-Amoako, E. S., Toora, C., Walton, R. F. *et al.* (1992). Thermal death times for *Listeria monocytogenes* in lobster meat. *J Food Prot* 55, 211–213.
- Büla, C., Bille, J. & Glauser, M. P. (1995). An epidemic of food-borne listeriosis in western Switzerland: description of 57 cases involving adults. *Clin Infect Diseases* 20, 66–72.
- Bunčić, S., Paunovic, L. & Radisic, D. (1991). The fate of *Listeria monocytogenes* in fermented sausages and in vacuum-packaged frankfurters. *J Food Prot* 54, 413–417.
- Carlier, V., Augustin, J. C. & Rozier, J. (1996). Heat resistance of *Listeria monocytogenes* (phagovar 2389/2425/3274/2671/47/108/340): D- and z-values in ham. *J Food Prot* 59, 588–591.
- CDC (Centers for Disease Control and Prevention) (1989). Listeriosis associated with consumption of turkey franks. *MMWR Morb Mortal Wkly Rep* 38, 267–268.
- CDC (Centers for Disease Control and Prevention) (1999). Update: Multistate outbreak of Listeriosis—United States, 1998–1999. *MMWR Morb Mortal Wkly Rep* 47, 1117–1118.
- CDC (Centers for Disease Control and Prevention) (2000). Preliminary FoodNet data on the incidence of food-borne illnesses—selected sites, United States, 1999. *MMWR* 49, 201–205.
- Ciesielski, C. A., Hightower, A. W., Parsons, S. K. *et al.* (1988). Listeriosis in the United States: 1980–1982. *Arch Intern Med* 148, 1416–1419.
- Dalton, C. B., Austin, C. C., Sobel, J. *et al.* (1997). An outbreak of gastroenteritis and fever due to *Listeria monocytogenes* in milk. *New Engl J Med* 336, 100–105.
- Ecklow, A., Danielsson-Tham, M.-L., Ericsson, H. *et al.* (1996). Listeriosis in the provinces of Värmland: the first reported outbreak of food-borne listeriosis in Sweden. In *Proceedings of the Symposium "Food Associated Pathogens"*, pp. 222–223. Uppsala, Sweden.
- Fain Jr., A. R., Line, J. A., Moran, A. B. *et al.* (1991). Lethality of heat to *Listeria monocytogenes* Scott A: D-value and z-value determinations in ground beef and turkey. *J Food Prot* 54, 756–761.
- FAO/WHO (Food and Agriculture Organization/World Health Organization) (2000). *Joint FAO/WHO Expert Consultation on Risk Assessment of Microbiological Hazards in Foods*. FAO Food and Nutrition Paper 71. Rome: FAO.
- Farber, J. M. (1989). Thermal resistance of *Listeria monocytogenes* in foods. *Int J Food Microbiol* 8, 285–291.
- Farber, J. M. & Brown, B. E. (1990). Effect of prior heat shock on heat resistance of *Listeria monocytogenes* in meat. *Appl Environ Microbiol* 56, 1584–1587.
- Farber, J. M., Cai, Y. & Ross, W. H. (1996). Predictive modeling of the growth of *Listeria monocytogenes* in CO₂ environments. *Int J Food Microbiol* 32, 133–144.
- Farber, J. M., Daley, E., Mackie, M. T. *et al.* (2000). A small outbreak of listeriosis potentially linked to the consumption of imitation crab meat. *Lett Appl Microbiol* 31, 100–104.
- Farber, J. M. & Peterkin, P. I. (2000). *Listeria monocytogenes*. In *The Microbiological Safety and Quality of Food*, pp. 1178–1232. Edited by B. M. Lund, A. C. Baird-Parker & G. W. Gould. Gaithersburg, MD: Aspen Publishers, Inc.
- Farber, J. M. & Peterkin, P. I. (1991). *Listeria monocytogenes*, a food-borne pathogen. *Microbiol Rev* 55, 476–511.
- FDA (US Food and Drug Administration) (2001). *Draft Assessment of the Relative Risk to Public Health from Foodborne Listeria monocytogenes among Selected Categories of Ready-to-Eat Foods*. Center for Food Safety and Applied Nutrition. Washington, DC: US Department of Health and Human Services.
- Fleming, D. W., Cochi, S. L., MacDonald, K. L. *et al.* (1985). Pasteurized milk as a vehicle of infection in an outbreak of listeriosis. *New Engl J Med* 312, 404–407.
- Gaze, J. E., Brown, G. D., Gaskell, D. E. *et al.* (1989). Heat resistance of *Listeria monocytogenes* in homogenates of chicken, beef steak and carrot. *Food Microbiol* 6, 251–259.
- Glass, K. A. & Doyle, M. P. (1989). Fate of *Listeria monocytogenes* in processed meat products during refrigerated storage. *Appl Environ Microbiol* 55, 1565–1569.
- Golnazarian, C. A., Donnelly, C. W., Pintauro, S. J. *et al.* (1989). Comparison of infectious dose of *Listeria monocytogenes* F5817 as determined for normal versus compromised C57B1/6J mice. *J Food Prot* 52, 696–701.
- Goulet, V. (1995). Investigation en cas d'épidémie de listériose. *Méd et Maladie Infect* 25, Spécial, 184–190.
- Goulet, V., LePoutre, A., Rocourt, J. *et al.* (1993). Épidémie de listériose en France; bilan final et résultats de l'enquête épidémiologique. *Bull Épidémiol Hebdomaire* 4, 13–14.

- Goulet, V., Jacquet, C., Vaillant, V. *et al.* (1995). Listeriosis from consumption of raw-milk cheese. *Lancet* 345, 1581–1582.
- Harrison, M. A. & Huang, Y. W. (1990). Thermal death times for *Listeria monocytogenes* (Scott A) in crabmeat. *J Food Prot* 53, 878–880.
- Hitchins, A. D. (1996). Assessment of alimentary exposure to *Listeria monocytogenes*. *Int J Food Microbiol* 30, 71–85.
- Ho, J. L., Shands, K. N., Friedland, G. *et al.* (1986). An outbreak of type 4b *Listeria monocytogenes* infection involving patients from eight Boston hospitals. *Arch Intern Med* 146, 520–524.
- Hoogenkamp, H. & Wales, J. (2000). Acetate and lactate blends fight bacteria with extra vigour. *Meat Int* 10, 24–25.
- Huss, H. H. (1997). Control of indigenous pathogenic bacteria in seafood. *Food Control* 8, 91–98.
- ICMSF (International Commission on Microbiological Specifications for Foods) (1994). Choice of sampling plan and criteria for *Listeria monocytogenes*. *Int J Food Microbiol* 22, 89–96.
- ICMSF (International Commission on Microbiological Specifications for Foods) (1996). *Microorganisms in Foods 5. Characteristics of Microbial Pathogens*. Gaithersburg, MD: Aspen Publishers, Inc.
- Juncher, D., Vestergaard, C. S., Søltfoft-Jensen, J. *et al.* (2000). Effects of chemical hurdles on microbiological and oxidative stability of a cooked cured emulsion type meat product. *Meat Sci* 55, 483–491.
- Kittson, E. (1992). A case cluster of listeriosis in Western Australia with links to pâté consumption. In *Proceedings of the 11th International Symposium "Problems of Listeriosis,"* pp. 39–40. Copenhagen, Denmark.
- Lennon, D., Lewis, B., Mantell, C. *et al.* (1984). Epidemic perinatal listeriosis. *Pediat Infect Diseases* 3, 30–34.
- Linnan, M. J., Mascola, L., Lou, X. D. *et al.* (1988). Epidemic listeriosis associated with Mexican-style cheese. *New Engl J Med* 319, 823–828.
- Lund, B. M. & Notermans, S. H. W. (1993). Potential hazards associated with REPFEDS. In *Clostridium botulinum: Ecology and Control in Foods*, pp. 279–303. Edited by A. H. W. Hauschild & K. L. Dodds. New York: Marcel Dekker, Inc.
- Lyytikäinen, O., Ruutu, P., Mikkola, J. *et al.* (1999). An outbreak of listeriosis due to *Listeria monocytogenes* serotype 3a from butter in Finland. *Eurosurveillance Wkly* 11 March.
- Mackey, B. M., Pritchett, C., Norris, A. *et al.* (1990). Heat resistance of *Listeria*: strain differences and effects of meat type and curing salts. *Lett Appl Microbiol* 10, 251–255.
- McClure, P. J., Beaumont, A. L., Sutherland, J. P. & Roberts, T. A. (1997). Predictive modeling of growth of *Listeria monocytogenes*: The effects on growth of NaCl, pH, storage temperature and NaNO₂. *Int J Food Microbiol* 34, 221–232.
- McDonald, K. & Sun, D-W. (1999). Predictive food microbiology for the meat industry: a review. *Int J Food Microbiol* 52, 127.
- McKellar, R. C., Moir, R. & Kalab, M. (1994). Factors influencing the survival and growth of *Listeria monocytogenes* on the surface of Canadian retail frankfurters. *J Food Prot* 57, 387–392.
- McLauchlin, J. (1993). Listeriosis and *Listeria monocytogenes*. *Environ Policy & Practice* 3, 201–214.
- McLauchlin, J., Hall, S. M., Velani, S. K. *et al.* (1991). Human listeriosis and pâté: a possible association. *Brit Med J* 303, 773–775.
- McMullen, L. M. & Stiles, M. E. (1996). Potential for use of bacteriocin-producing lactic acid bacteria in the preservation of meats. *J Food Prot Suppl*, 64–71.
- Mead, P. S., Slutsker, L., Dietz, V. *et al.* (1999). Food-related illness and death in the United States. *Emerging Infect Diseases* 5, 607–625.
- Miettinen, M. K., Siitonen, A., Heiskanen, P. *et al.* (1999). Molecular epidemiology of an outbreak of febrile gastroenteritis caused by *Listeria monocytogenes* in cold-smoked rainbow trout. *J Clin Microbiol* 37, 2358–2360.
- Mitchell, D. L., Misrachi, A., Watson, A. J. *et al.* (1991). A case cluster of listeriosis in Tasmania. *Listeria* in smoked mussels in Tasmania. *Communic Disease Intell* 15, 427.
- Proctor, M. E., Brosch, R., Mellen, J. W. *et al.* (1995). Use of pulsed-field gel electrophoresis to link sporadic cases of invasive listeriosis with recalled chocolate. *Appl Environ Microbiol* 61, 3177–3179.
- Qvist, S. & Liberski, D. (1991). *Listeria monocytogenes* in frankfurters and ready-to-eat sliced meat products. *Danish Veterinaertidsskr* 74, 773–774, 776–778.

- Qvist, S., Sehested, K. & Zeuthen, P. (1994). Growth suppression of *Listeria monocytogenes* in a meat product. *Int J Food Microbiol* 24, 283–293.
- Riedo, F. X., Pinner, R. W., Tosca, M. de L. *et al.* (1994). A point-source foodborne listeriosis outbreak: Documented incubation period and possible mild illness. *J Infect Disease* 170, 693–696.
- Rocourt, J. (1996). Risk factors for listeriosis. *Food Control* 7, 195–202.
- Rocourt, J. & Bille, J. (1997). Foodborne listeriosis. *World Health Stats Quarterly* 50, 67–73.
- Ross, T., Todd, E. & Smith M. (2000). *Exposure Assessment of Listeria monocytogenes in Ready-to-Eat Foods*. Preliminary Report for Joint FAO/WHO Expert Consultation on Risk Assessment of Microbiological Hazards in Foods. Rome: FAO.
- Salamina, G., Dalle Donne, E., Niccolini, A. *et al.* (1996). A foodborne outbreak of gastroenteritis involving *Listeria monocytogenes*. *Epidemiol & Infection* 117, 429–436.
- Schlech, W. F., III, Lavigne, P. M., Bortolussi, R. A. *et al.* (1983). Epidemic listeriosis—evidence for transmission by food. *New Engl J Med* 308, 203–206.
- Schmidt, U. (1993). Behaviour of *Listeria monocytogenes* on sliced bruhwurst sausage. *Mitteilungsblatt der Bundesanstalt für Fleischforschung* 119, 16–20.
- Schmidt, U. & Leistner, L. (1993). Behavior of *Listeria monocytogenes* in unpackaged sliced bruhwurst sausage. *Fleischwirt* 73, 733–740.
- Schuchat, A., Deaver, K. A., Wenger, J. D. *et al.* (1992). Role of foods in sporadic listeriosis. I. Case-control study of dietary risk factors. *J Am Med Assoc* 267, 2041–2045.
- Schwartz, B., Ciesielski, C. A., Broome, C. V. *et al.* (1988). Association of sporadic listeriosis with consumption of uncooked hot dogs and undercooked chicken. *Lancet* 2 (8614), 779–782.
- Sumner, S. S., Sandros, T. M., Harmon, M. C. *et al.* (1991). Heat resistance of *Salmonella typhimurium* and *Listeria monocytogenes* in sucrose solutions of various water activities. *J Food Sci* 56, 1741–1743.
- Tompkin, R. B. (1999). *Petition to USDA-FSIS Seeking Approval for Use of Increased Concentrations of Sodium Diacetate and Sodium Acetate in Meat and Poultry Products*. Letter dated March 24. USDA-FSIS, Hearing Clerk's Office, Washington, DC.
- Tompkin, R. B., Christiansen, L. N., Shaparis, A. B. *et al.* (1992). Control of *Listeria monocytogenes* in processed meats. *Food Austral* 44, 370–376.
- Tompkin, R. B., Scott, V. N., Bernard, D. T. *et al.* (1999). Guidelines to prevent post-processing contamination from *Listeria monocytogenes*. *Dairy, Food & Environ San* 19, 551–562.
- USDA (US Department of Agriculture) (1995a). *Nationwide Raw Ground Chicken Microbiological Survey. March 1995–May 1995*. Washington, DC: Microbiology Division, Food Safety and Inspection Service, US Department of Agriculture.
- USDA (US Department of Agriculture) (1995b). *Nationwide Raw Ground Turkey Microbiological Survey. January 1995–March 1995*. Washington, DC: Microbiology Division, Food Safety and Inspection Service, US Department of Agriculture.
- USDA (US Department of Agriculture) (1996). *Nationwide Federal Ground Beef Microbiological Survey. August 1993–March 1994*. Washington, DC: Microbiology Division, Food Safety and Inspection Service, US Department of Agriculture.
- van Schothorst, M. (1996). Sampling plans for *Listeria monocytogenes*. *Food Control* 7, 203–208.
- Wang, C. & Muriana, P. M. (1994). Incidence of *Listeria monocytogenes* in packages of retail franks. *J Food Prot* 57, 382–386.
- Wederquist, H. J., Sofos, J. N. & Schmidt, G. R. (1994). *Listeria monocytogenes* inhibition in refrigerated vacuum packaged turkey bologna by chemical additives. *J Food Sci* 59, 498–500, 516.
- Wederquist, H. J., Sofos, J. N. & Schmidt, G. R. (1995). Culture media comparison for the enumeration of *Listeria monocytogenes* in refrigerated vacuum packaged turkey bologna made with chemical additives. *Lebens Wissensch Technol* 28, 455–461.
- Wenger, J. D., Swaminathan, B., Hayes, P. S. *et al.* (1990). *Listeria monocytogenes* contamination of turkey franks: Evaluation of a production facility. *J Food Prot* 53, 1015–1019.
- Zaika, L. L., Palumbo, S. A., Smith, J. L. *et al.* (1990). Destruction of *Listeria monocytogenes* during frankfurter processing. *J Food Prot* 53, 18–21.

E. coli O157:H7 in Frozen Raw Ground Beef Patties

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17.1 INTRODUCTION

This chapter is concerned with *Escherichia coli* O157:H7 in frozen raw ground beef patties. The information follows the principles outlined in earlier chapters. The unique severity of the pathogen and its impact among children under five years of age warrant special consideration. In this example, the merits of microbiological testing will be explored as a potential control measure to enhance the safety of ground beef. A significant portion of this material was published as a discussion paper (Tompkin & Bodnaruk, 1999). This example draws primarily on data from the US. The information may be applicable in other countries with similar prevalence rates in ground beef and where undercooking is a risk factor.

Ground beef is an important raw agricultural commodity. In 1992 in the US, the types of cattle from which ground beef was derived were steers, 39.1%; heifers, 19.2%; cows, 34%; and bulls, 7.7%. Imported boneless manufacturing beef composed 15% of the total US ground beef supply. Imports into the US account for almost half of the world trade in fresh, chilled, and frozen beef. Ninety percent of the beef imported into the US is from Australia, New Zealand, and Canada. Other sources include Costa Rica, Honduras, and the Dominican Republic. The annual average quantity of beef imported into the US has been over 600,000 tons (USDA, 1994). Annual per capita consumption was estimated to be about 11.8–12.7 kg from 1982–1998 (AMI, 2000). Ground beef is consumed in the form of hamburger and as a major ingredient in a variety of foods.

E. coli O157:H7 is among the more newly recognized foodborne pathogens, being first identified during an outbreak in 1982 in which ground beef was implicated. It has since been a steadily increasing cause of foodborne illness worldwide. It is recognized that there are numerous serotypes of enterohemorrhagic *E. coli* (EHEC), some of which may be more important in certain regions of the world than *E. coli* O157:H7 (WHO, 1997; Acheson, 2000).

Since 1982, ground beef has become recognized as a major vehicle for transmission of *E. coli* O157:H7 in the US. In general, the pathogen is deposited on the surface of beef carcasses during the slaughtering process. After chilling, the carcasses are cut into large portions for sale as bone-in or boneless beef cuts (e.g., round, loin, rib, chuck). During this

process, the larger portions are trimmed of excess fat and other tissue. The trimmings commonly are used in the manufacture of raw ground beef and a wide variety of ready-to-eat products (e.g., sausages). The same process of trimming meat and fat occurs at other steps along the food chain, with much of it being used for ground beef. Wherever it may occur along the food chain, the process of trimming and subsequent grinding distributes the pathogen throughout the ground meat. The most common scenario leading to illness has involved undercooking, survival of the pathogen, and subsequent infection, particularly among the more susceptible consumers. Cross-contamination in kitchens and food service establishments from ground beef to other ready-to-eat foods also has occurred.

It is extremely difficult to compare data from different countries on incidence and prevalence due to the use of different methods of sampling/reporting. However, data for different countries indicate that *E. coli* O157:H7 is an international concern, with the prevalence in meat ranging from around 0.1%–5% and prevalence in cattle ranging from around 1.5%–28% (Table 17–1).

17.2 RISK EVALUATION

17.2.1 Hazard Identification

E. coli is a species of Gram-negative, facultatively anaerobic, rod-shaped bacteria commonly found in the lower part of the intestine of warm-blooded animals. *E. coli* O157:H7 is a particular serotype of the group referred to as enterohemorrhagic *E. coli* (EHEC). This is a subgroup of the verocytotoxigenic *E. coli* (VTEC) that has been shown to cause human illness. VTECs produce verotoxins, or shiga-like toxins, that are closely related to the toxin produced by *Shigella dysenteriae* (Cassin *et al.*, 1998). Most EHEC isolates are acid-tolerant, capable of surviving in acid foods and during passage through the stomach (Benjamin & Datta, 1995; Arnold & Kaspar, 1995; Leyer *et al.*, 1995; Cheville *et al.*, 1996).

Table 17–1 Incidence of *E. coli* O157:H7 Infection and Prevalence in Meat and Cattle in Various Countries

Country	Incidence HUS/VTEC*†	Prevalence (%) in Meat	Prevalence (%) in Cattle
Argentina	HUS 7.8 per 100,000	0.4 bovine meat	1.5
Australia	HUS 2.79 per 100,000		
Canada	VTEC 3.0 per 100,000		
Denmark	VTEC 0.1 per 100,000	0.13 minced beef	1.4
Germany		0.7 beef	
		5.0 minced beef	
Japan		0.3 carcass	1.4
Netherlands	30 per year	0.08 beef/pork	11.1
England and Wales	VTEC 1.29 per 100,000	1.5 beef	15.6
US	0.74 per 100,000	1.0	5
			28‡

*HUS = hemolytic uremic syndrome

†VTEC = verocytotoxigenic *E. coli*

‡using improved isolation methods

Cattle appear to be the main reservoir of *E. coli* O157:H7. Contamination of carcasses during slaughter is the primary route that ultimately leads to contamination of ground beef. Other foods (e.g., lettuce, sprouts, fruit juices, vegetables, raw milk) and water also have been implicated as vehicles of transmission. Person-to-person is an important mode of transmission, particularly in day care centers. Direct contact with animals carrying the organism is also a recognized source of infection (WHO, 1997).

17.2.2 Hazard Characterization

E. coli O157:H7 infection can result in moderate to severe disease or death, with most deaths occurring in children under five years of age and in the elderly (AGA, 1994; Tarr, 1994). Studies conducted in FoodNet sites in the US suggest that from 13–27 cases of infection occur in the community for each confirmed case that is reported (Mead *et al.*, 1999). Within the FoodNet sites, the annual laboratory-confirmed case rate per 100,000 population has been from 2.1–2.8 in the four years of 1996–1999 (CDC, 2000).

The time to onset of symptoms following exposure ranges from three to nine days, with an average of four. The duration of illness ranges from two to nine days; however, with complications, the illness may last many months and lead to permanent damage or even death. Symptoms may include hemorrhagic colitis: grossly bloody diarrhea, severe abdominal pain, vomiting, but no fever; hemolytic uremic syndrome (HUS): prodrome of bloody diarrhea, acute nephropathy, seizures, coma, and death; and thrombotic thrombocytopenic purpura: similar to HUS but also fever and central nervous system disorder (ICMSF, 1996).

The potent toxins of *E. coli* O157:H7 can initially cause a particularly severe form of human disease, hemorrhagic colitis. About 10% of these patients can go on to develop HUS, characterized by acute renal failure, hemolytic anemia, and thrombocytopenia, which is particularly serious in young children and elderly people. On average, 2–7% of patients with HUS die, but in some outbreaks among the elderly, the mortality rate has been as high as 50% (WHO, 1997). Long-term renal dysfunction occurs in about 10–30% of survivors of HUS. The estimate for the prevalence of HUS in North America is about 3/100,000 children under five years of age per year (Mahon *et al.*, 1997).

From outbreak data, it appears that fewer than 100 cells can cause disease among consumers (AGA, 1994), particularly those at greater risk (e.g., less than five years of age) (Mahon *et al.*, 1997). In the 1993 northwestern US outbreak involving undercooked hamburgers, it was estimated that as few as a dozen cells may have caused illness among children (Tarr, 1994).

There are estimated to be about 73,480 cases of illness attributed to *E. coli* O157:H7 per year in the US, of which 85% are estimated to be foodborne. The number of hospitalizations and deaths due to foodborne transmission of this pathogen was estimated to be 1,843 and 52 per year, respectively (Mead *et al.*, 1999). These values differ significantly from the earlier estimates of Mahon *et al.* (1997). The more recent estimate revealed more cases, but fewer deaths. The estimated costs of medical treatment and lost productivity range from \$216–580 million/year (AGA, 1994).

17.2.3 Exposure Assessment

The source of *E. coli* O157:H7 on beef carcasses is the hide and intestines of the cattle being slaughtered. Prevalence among cattle being held for slaughter is similar to or

slightly higher than the prevalence on the external surface of hides of recently slaughtered animals (Hancock, 1998).

The percent of cattle with *E. coli* O157:H7 in their feces was initially reported to be typically less than 5% (Hancock, 1998). A later study involving a more sensitive analytical method found 28% of the cattle entering slaughtering plants to be actively shedding *E. coli* O157:H7 or nonmotile *E. coli* O157 in their feces during July and August, the months of highest prevalence. Eleven percent of the hide surfaces also were positive (Elder *et al.*, 2000).

Studies have shown that colonization of cattle is of short duration (1–2 months); long-term carriers have not been found. The typical pattern of shedding in a herd followed over time is one of epidemics of shedding interspersed with longer periods with rare or non-shedding animals. These epidemics occur mainly during warm weather, suggesting that environmental proliferation may play an important role in the epidemiology of *E. coli* O157:H7 (Hancock, 1998). It is important to note that *E. coli* O157:H7 does not cause any adverse effects in cattle. Its presence in a herd or individual animal can be detected only by microbiological testing.

While some cross-contamination may occur from carcass-to-carcass through contact with common equipment and workers' hands, there is no published evidence that *E. coli* O157:H7 has ever become established and multiplied in a slaughtering/chilling/fabrication operation and contaminated subsequent lots of beef.

Technology is currently available that can:

- minimize carcass contamination during the slaughtering process,
- reduce the likelihood of microbial attachment to exposed tissues, and
- decontaminate carcasses, such as with steam, hot water, or organic acid sprays prior to chilling.

Collectively, the above control measures can significantly reduce, but not eliminate, the likely presence of enteric pathogens on raw beef. Systems for decontamination will likely involve multiple control measures during slaughtering, evisceration, and chilling (Dorsa *et al.*, 1996; Dorsa, 1997; Castillo *et al.*, 1998; Nutsch *et al.*, 1998; Sofos & Smith, 1998; Sofos *et al.*, 1999; Bacon *et al.*, 2000).

It is reasonable to assume that when present, *E. coli* O157:H7 is on the surface of carcasses, not in internal tissues. Rapid chilling of adequately spaced carcasses should retard *E. coli* O157:H7 growth on carcass surfaces. Surface dehydration during chilling is an additional factor that can restrict growth.

After chilling, it is not likely that *E. coli* O157:H7 will multiply during subsequent fabrication because the lower temperature limit for multiplication of *E. coli* O157:H7 is 7–8 °C (ICMSF, 1996). Furthermore, estimates for growth under optimum laboratory conditions in broth indicate that:

- at 10 °C, the time to increase 10-fold is estimated to be 73 h, and
- at 15 °C, the time to increase 10-fold is estimated to be 25 h (Buchanan & Whiting, 1996).

Thus, in controlled chilling/fabrication operations, the concentration of *E. coli* O157:H7 should not increase. Conversely, in operations that do not address chilling/fabrication as important factors requiring control, some degree of multiplication may occur.

Epidemiologic data for outbreaks reported each year by the Centers for Disease Control and Prevention (CDC) indicate the risk of *E. coli* O157:H7 from beef continues

to be associated with consumers who have not changed their handling/cooking habits to control the risk of the pathogen. These consumers do not understand the risks and/or do not have the knowledge to deal with a pathogen such as *E. coli* O157:H7. Some may be aware of the risk and have chosen to ignore recommendations for proper handling and cooking of ground beef. A 1996 survey indicated that 19.7% of the population consumed pink (undercooked) hamburger at some time during the previous 12 months (CDC, 1998a).

In its report of FoodNet for 1997, CDC stated: "In contrast with previous investigations, hamburgers eaten at fast food restaurants were not associated with infection, suggesting that recent changes in that industry may have reduced *E. coli* O157:H7 infections from that source" (CDC, 1998b). This is a conservative assessment because a review of the outbreaks reported each year by CDC shows no reported outbreaks from the larger fast food operations between 1993–2000. This verifies that the control measures adopted in those operations have been effective in managing the risk of *E. coli* O157:H7 in ground beef.

The improved controls in fast food operations do not appear to have carried through to other areas. A case-control study conducted at FoodNet sites has determined that undercooked ground beef continues to be a principal food source of *E. coli* O157:H7 infections (CDC, 1998b). Outbreaks have continued to occur in other food service establishments (e.g., restaurants, schools) and among consumers who prepare ground beef at home, on camping trips, or in other settings. Ground beef is the primary vehicle, not roasts, steaks, etc.

This information suggests that a continuing effort should be directed toward educating food handlers and consumers in proper handling and cooking of ground beef before serving. A goal should be established to reduce the percentage of consumers who consume rare ground beef from approximately 20% to some lower value. School systems and other food service establishments should consider additional training/education and other options (e.g., precooked beef products).

17.2.3.1 Survey Results from USDA-FSIS

The US Department of Agriculture (USDA)-Food Safety and Inspection Service (FSIS) established a sampling and testing program for ground beef in 1994 to:

- stimulate industry actions to reduce the presence of *E. coli* O157:H7 in raw ground beef (i.e., to encourage industry to institute and maintain effective control measures),
- encourage industry to sample and test raw ground beef routinely,
- find and remove from commerce product containing *E. coli* O157:H7, and
- expand the agency's information base and understanding in the control of *E. coli* O157:H7 (FSIS, 1994).

Results of the sampling and testing program are summarized in Table 17–2. The methods of sampling and analysis have been modified since 1995 to increase sensitivity of detection. During 1995–1997, a single 25 g sample was analyzed. Since the beginning of fiscal year 1998, five 65 g samples have been analyzed.

During the 1990s, industry implemented new control measures to reduce the occurrence of salmonellae and *E. coli* O157:H7 in ground beef. Due to the increased sensitivity of the methods used by FSIS, it is not possible to assess the effectiveness of changes implemented by industry or to determine whether the prevalence of *E. coli* O157:H7 in ground beef has, in fact, increased since the inception of the sampling program.

Table 17-2 Results from the FSIS Sampling Program Beginning with Fiscal Year 1995

<i>Fiscal Year</i>	<i>No. Samples</i>	<i>No. Positive</i>	<i>% Positive</i>
1995*	5291	3	0.057
1996*	5326	4	0.075
1997*	5919	2	0.034
1998†	7529	14	0.19
1999†	8710	29	0.33
2000‡	>4,000	38	0.95

*25 g analytical units; †325 g analytical units, ‡325 g and improved isolation method (i.e., immunomagnetic beads to concentrate cells). The results for FY2000 are through August 11, 2000.

17.2.3.2 Data from Ground Beef Implicated in Illness and Lots Found Positive by USDA-FSIS

As ground beef has been implicated in outbreaks, samples from some implicated lots have been collected and analyzed. The ability to detect additional positive samples from across implicated lot(s) indicates a relatively high prevalence and concentration. In a few instances, a quantitative analysis was performed to determine the concentration of *E. coli* O157:H7. For example, in the 1993 outbreak that occurred in the northwestern US, frozen ground beef patties produced at a particular facility on November 19 and 20, 1992 were found positive. Positive samples were found in lots 4 and 9–17 in the production of November 19 and in lot 7 in the production of November 20. Quantitative analysis of some of the positive lots conducted by FSIS and CDC resulted in most probable number values in the range of 1–4 cells g⁻¹, with a single high value of 15 g⁻¹ (FSIS, 1993).

Other evidence suggesting a relatively high prevalence and concentration of *E. coli* O157:H7 in certain lots of ground beef includes:

- large numbers of cases among consumers,
- multiple outbreaks involving consumers in different locations, and
- ability to detect *E. coli* O157:H7 with just a single sample from an implicated lot.

Follow-up sampling of positive lots of ground beef detected at retail stores has led to a positive sample from coarse ground beef used.

Data from the USDA-FSIS and lots implicated in outbreaks suggest that the vast majority of ground beef has a very low prevalence and concentration of *E. coli* O157:H7. In rare instances, a very small proportion of the lots contains a relatively high prevalence and concentration. It is because of this kind of skewed distribution in the number of bacteria that a log-normal distribution function is often used in microbiology. A hypothetical log-normal distribution of counts with a standard deviation would give the curve shown in Figure 17-1. Baseline studies on the numbers of bacteria in raw meat and poultry in the US and Canada provide further support to the general applicability of a log-normal distribution for the microbial population in these foods (FSIS, 1996; CFIA, 2000). When a log-normal distribution is assumed for these data, it is estimated that the standard deviation would be between 1.3 and 0.55. Figure 17-2 shows an estimated log-normal distribution (mean = 1.2, sigma = 0.8) from counts obtained in the baseline study of the number of Biotype I *E. coli* in raw ground beef in the US compared to the

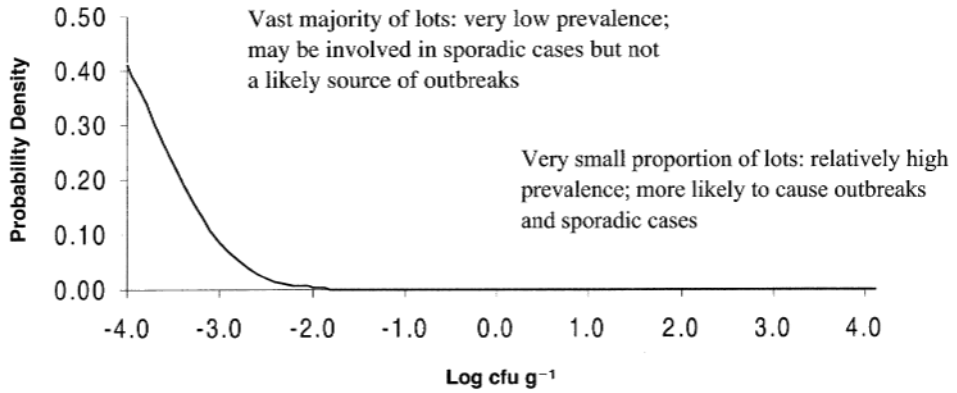


Figure 17-1 Illustration of the likely distribution in prevalence and concentration of *E. coli* O157:H7 in ground beef in North America.

hypothetical distribution for the counts of *E. coli* O157:H7 in raw ground beef. To consider the uses and limitations of sampling for *E. coli* O157:H7 in raw ground beef, we have assumed that the counts for this organism also have a log-normal distribution with a standard deviation value of 0.8, but a mean concentration $< 1.2 \text{ g}^{-1}$. Although hypothetical, such a distribution of counts would be consistent with a prevalence of 1% positive in 325 g samples taken.

17.2.3.3 Lots with Low Prevalence and Concentration

The vast majority of ground beef has a very low prevalence and, presumably, low concentration of *E. coli* O157:H7. For example, the results of the FSIS sampling program suggest a background prevalence of about 1% or less when analyzing 325 g. This prevalence is likely influenced by the prevalence of *E. coli* O157:H7 that occurs in cattle at the time of slaughter and also reflects the level that may be normally achievable from slaugh-

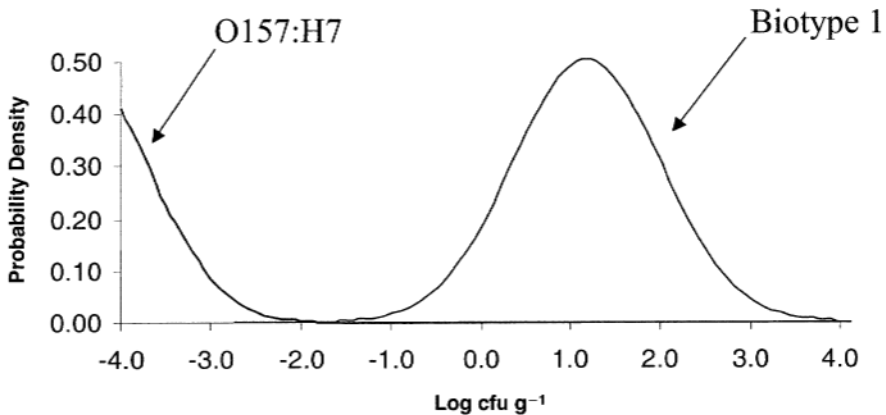


Figure 17-2 Comparison of hypothetical distribution in prevalence and concentration of *E. coli* O157:H to fitted distribution for *E. coli* Biotype I (mean = 1.2, sigma = 0.8) taken from the U.S. Nationwide Raw Beef Microbiological Survey Aug 1993–Mar 1994.

ter to the manufacture of ground beef with existing technology. These lots have such a low prevalence of contamination that defective lots cannot be detected with any degree of confidence through routine microbiological testing. Possibly, these lots may be involved in sporadic cases, but seldom with outbreaks.

Considering the low prevalence, it is questionable that the FSIS sampling program alone, with 5,000–7,000 samples/year, would have a measurable impact on the number of cases/100,000/year attributable to *E. coli* O157:H7 in ground beef. While perhaps ineffective in ensuring consumer protection, the FSIS program does send a strong message to industry and consumers as to its expectations. This has been one factor that has resulted in the implementation of improved control measures in slaughtering operations.

It would be impractical to implement a routine sampling plan to detect and reject contaminated lots with a $\leq 1\%$ prevalence of contamination. For example, if the prevalence rate was 0.7%, the number (n) of samples required to detect the pathogen with a 95% probability would be 428 units. Even sampling to provide a 90% confidence level would require 329 sample units. Additional information describing the difficulty of detecting positive lots with low prevalence of contamination is evident in Table 17–3.

If the defect level is 0.5% and 30 sample units are tested, there is an 86% probability that all 30 samples will be found negative and the lot will be accepted. Even with 100 sample units, there is a 61% probability that all 100 samples will be found negative and the lot will be accepted. Clearly, microbiological sampling and testing are very ineffective for detecting lots with low prevalence of contamination.

17.2.3.4 Lots with Relatively High Prevalence

Experience indicates that a very small proportion of lots of ground beef have a relatively high prevalence (e.g., 5% or higher) and, presumably, higher concentration of cells (e.g., 1–10 g^{-1}). In these lots, it has been possible to find additional positive samples when resampled. Published information does not explain how these lots acquire a higher prevalence or concentration of *E. coli* O157:H7. It is strongly suspected that contamination results when trimmings from one or a few carcasses are introduced into the grinding process with other trimmings and cause a “comet-like” effect as described in Chapter 10 and Figure 10–4c. It is probable that these lots would more likely cause outbreaks as well as sporadic cases, if the product is undercooked or other foods are contaminated.

Due to the higher prevalence of contamination in these lots, it may be possible to apply a statistically valid sampling plan and, to the extent possible, exclude them from the mar-

Table 17–3 Probability of Accepting a Defective Lot with Indicated Proportion of Defective Sample Units

% defective	No. of sample units			
	15	30	60	100
0.1	0.99	0.97	0.94	0.90
0.5	0.93	0.86	0.74	0.61
1	0.86	0.74	0.55	0.37
2	0.74	0.55	0.30	0.13
5	0.46	0.21	0.05	0.01

ket. Since it is not possible to anticipate which lots may be in this category, the intent of the sampling plan would be to detect them as they are produced.

17.2.4 Risk Characterization

The annual per capita consumption of ground beef in the US was estimated to be approximately 12 kg from 1982–1998 (AMI, 2000). If it is assumed that all the ground beef is consumed as hamburger patties weighing 125 g each, this would result in 96 servings/person/year. Since the total population in the US in 1998 was approximately 270 million, the total number of servings on a national basis would be about 26×10^9 servings per year. If the USDA survey data for FY 2000 are used, it can be assumed that the prevalence rate for *E. coli* O157:H7 in ground beef is about 1%. This prevalence rate was determined using five analytical units, each weighing 65 g. A positive result for any of the five analytic units was reported as a positive 325 g.

Data are not available for 125 g samples; however, for this example it will be assumed that the prevalence rate in 125 g samples is 0.7%. Given that assumption, it can be estimated that the number of raw hamburger patties containing *E. coli* O157:H7 each year is 26×10^7 (i.e., 1% of 26×10^9 servings). While a survey indicated that 19.7% of the population had consumed pink (undercooked) ground beef at some time during the previous 12 months, it is not likely that this occurred with each serving. For example, individuals purchasing hamburgers in fast food operations would very likely receive a hamburger that is well done and with no evidence of undercooking. This would significantly reduce the extent of exposure to undercooked hamburgers containing *E. coli* O157:H7. If it is assumed that undercooked hamburgers were consumed 5% of the time among the respondents of the survey, then, it can be estimated that the number of undercooked servings that contained *E. coli* O157:H7 before cooking would be about 26×10^5 [i.e., $5\% \times 19.7\% \times (26 \times 10^7)$]. If it is further assumed that the concentration of *E. coli* O157:H7 in the patties before cooking is 1 g^{-1} (H_0), then the number of cells would be 125/patty and their distribution within the patties would influence whether they are killed during cooking. It has been reported that an expected D value for *E. coli* O157:H7 in ground beef with 30.5% fat is 0.47 min at 62.8 °C (Line *et al.*, 1991). Cooking beef to this temperature typically yields a medium-rare (i.e., not raw) appearance. It is not likely that *E. coli* O157:H7 with an initial level of 1 g^{-1} would survive in a patty that is cooked to 62.8 °C unless, perhaps, all the cells are at the site of slowest heating within the patty. Thus, it may be possible for patties to appear undercooked (medium-rare), but have no viable cells remaining. As with many of the assumptions in this example, data are not available for the frequency of undercooking. Thus, it will be assumed for this example that 10% of the undercooked patties are insufficiently cooked to kill all the cells that are present. This leads to an estimate of approximately 26×10^4 patties/year potentially being consumed in the US that contain viable *E. coli* O157:H7.

The extent to which these patties would lead to illness would be influenced by many factors, in particular the susceptibility of the consumer. It is not likely that the most highly sensitive population, namely children less than five years of age and the elderly, would consume hamburger at a rate of 96 servings/year. In addition, these estimates assume that all the ground beef is consumed as patties. These estimates also ignore the fact that a significant portion of the patties are cooked in USDA inspected facilities and

in fast food operations that apply control measures to ensure a minimum internal temperature of 70 °C.

17.3 RISK MANAGEMENT

17.3.1 Level of Consumer Protection

The exposure assessment revealed that about 25% of the *E. coli* O157:H7 outbreaks during 1982–1997 and 33% of illnesses were attributable to ground beef (FSIS, 1998). An estimate for sporadic cases is not available. Using those data, and assuming that there are 62,458 foodborne cases of *E. coli* O157:H7 per year, then the number of cases attributable to ground beef would be approximately 20,611 cases/year. An example of a public health goal for this food-pathogen combination would be to reduce or eliminate the number of cases attributable to ground beef. A 25% reduction in the number of cases attributed to ground beef, for example, would mean a reduction of 5,152 cases/year.

17.3.2 Establishing a Food Safety Objective

In this example, it is difficult to establish a clear relationship between the level of *E. coli* O157:H7 consumed and the probability of illness. However, outbreak data (AGA, 1994; Tarr, 1994) and risk estimates (Cassin *et al.*, 1998) suggest that fewer than 100, to as low as a dozen or so cells, can cause illness among consumers, particularly those at greater risk. Therefore, to establish a food safety objective (FSO) for this example, it is important for the value to be necessarily conservative to reflect the degree of uncertainty, the relatively low infective dose, and the severity of the illness. An example of an FSO would be:

The concentration of *E. coli* O157:H7 in raw ground beef shall not exceed 1 cfu 250 g⁻¹.

Since a concentration of 1 cfu 250 g⁻¹ also can be expressed as $-2.4 \log_{10}$ cfu g⁻¹, the FSO also could be stated as the concentration of *E. coli* O157:H7 in raw ground beef shall not exceed $-2.4 \log_{10}$ cfu g⁻¹. The FSO is equivalent to no greater than 1 cell per two patties with each weighing 125 g, a common weight for commercially manufactured ground beef patties.

The proposed FSO should provide a margin of safety considering the thermal destruction that occurs even in undercooked patties. This FSO is only applicable to ground beef for sale in retail stores or for use in food service establishments. Ground beef for manufacturing products cooked in commercial establishments with Hazard Analysis Critical Control Point (HACCP) systems need not meet the proposed FSO because the required cooking processes have been validated to ensure destruction of at least 10^5 g⁻¹ salmonellae or *E. coli* O157:H7.

17.4 CONTROL MEASURES

17.4.1 Good Hygienic Practices (GHP)/HACCP

A generic model HACCP plan has been developed for teaching the principles of HACCP to those involved in the manufacture of frozen ground beef patties (Bernard *et*

al., 1997). The document discusses the strengths and limitations of GHP and HACCP for control of enteric pathogens in ground beef and provides guidance that may facilitate meeting the FSO by manufacturers of ground beef.

17.4.2 Controlling Initial Number (H_0)

The only means to control the concentration of enteric pathogens in raw ground beef is through raw material selection. The options would be limited to (1) supplier assurance programs and (2) pretesting trimmings before use. In addition, maintaining an effective time-temperature control program is needed to prevent growth of *E. coli* O157:H7 on carcasses during chilling/cutting and through grinding/freezing the ground beef patties.

17.4.2.1 Control through Supplier Assurance Programs

Control through supplier assurance programs entails selecting suppliers who have implemented interventions to minimize carcass contamination (e.g., enclosing bung in plastic bag and tying, an improved method of hoof and hide removal, use of steam vacuum on areas likely to be contaminated) during the slaughtering process and/or to decontaminate carcasses (e.g., hot water or steam pasteurization, organic acid spray) at strategic steps in the slaughtering process. As described earlier, it is likely that these control measures can significantly reduce, but will not likely eliminate, the presence of enteric pathogens (Dorsa *et al.*, 1996; Dorsa, 1997; Castillo *et al.*, 1998; Nutsch *et al.*, 1998; Sofos & Smith, 1998; Sofos *et al.*, 1999; Bacon *et al.*, 2000).

Suppliers should be audited following the guidelines in Chapter 4 and the results used as a basis for approval. Familiarity with the current use of interventions and their effectiveness should be incorporated into the audit. In addition, certain interventions (e.g., steam pasteurization, hot water sprays) may have been validated to achieve a minimum level of reduction under specified conditions of operation.

17.4.2.2 Control through an Incoming Raw Materials Screening Program

Control through an incoming raw materials screening program consists of using microbiological tests of incoming raw materials to identify and select the better, more reliable suppliers and to eliminate suppliers who exercise less control of microbial quality (Bernard *et al.*, 1997; Eisel *et al.*, 1997; Scanga *et al.*, 2000). These tests could involve indicator microorganisms as a general measure of GHP and good manufacturing practices.

Trimmings to be used for ground beef could be pretested for *E. coli* O157:H7 by various sampling plans (AMSA, 1999; Scanga *et al.*, 2000). One option would be to have the supplier sample the trimmings and have them analyzed while the raw materials are in transit. Slaughtering operations that use their own in-house trimmings to manufacture frozen ground beef patties could sample the trimmings before grinding.

Producers of ground beef should consider how much reliance to place on the interventions employed by suppliers and whether it is necessary, or effective, to test for *E. coli* O157:H7. If testing is used as a control measure, then consideration must be given to testing raw materials (i.e., trimmings) or finished product (i.e., frozen patties).

To meet the required FSO through raw material selection, the concentration of *E. coli* O157:H7 in the trimmings would have to be controlled so the ground beef will meet the FSO (i.e., $FSO = -2.4 \log_{10}$ or not exceed 1 per 250 g). This can be expressed in the following simple equation (described fully in Chapter 3).

$$H_0 - \Sigma R + \Sigma I \leq \text{FSO}$$

$$H_0 - 0 + 0 \leq -2.4$$

$$H_0 \leq -2.4$$

Thus, the initial concentration of *E. coli* O157:H7 in the frozen ground beef patties must be $-2.4 \log_{10} \text{ cfu g}^{-1}$ and, if present, there must be no growth ($\Sigma I = 0$).

There has been only one report of beef with a concentration of *E. coli* O157:H7 as high as 1,000/g. That occurred in a very small operation (i.e., a locker plant) that processed individual animals for local customers (Todd *et al.*, 1988). Such operations would be less able to exercise control over the slaughtering/chilling/fabrication process. In larger commercial operations, however, lower numbers of *E. coli* O157:H7 in trimmings and, ultimately, in ground beef would be expected. This would reflect the fact that few carcasses would carry the pathogen and some dilution of the pathogen would occur as trimmings are mixed with the large quantities of negative product. For these reasons, it may be assumed that the concentration of *E. coli* O157:H7 in trimmings from these operations would not likely exceed 100 g^{-1} . Even in the outbreak that occurred in the northwestern US, the highest concentration detected was 15 g^{-1} . Therefore, it is reasonable to assume that the initial level is unlikely to exceed 100 g^{-1} (i.e., $H_0 = 2$).

17.4.3 Reducing Levels (ΣR)

If an initial concentration of 100 g^{-1} is assumed as a worst case, then a performance criterion can be established for the reduction necessary to achieve the FSO of $-2.4 \log_{10} \text{ cfu g}^{-1}$ (1 cell per 250 g) at the time the patties are consumed. This can be expressed as follows:

$$H_0 - \Sigma R + \Sigma I \leq \text{FSO}$$

$$2 - \Sigma R + 0 \leq -2.4$$

$$\Sigma R \leq 4.4$$

Thus, the performance standard for cooking the frozen patties could be stated as:

a 4.4D reduction of *E. coli* O157:H7 in the coldest area of the patty.

If the initial level (H_0) can be controlled to a lower level (e.g., $H_0 = 1$), then the performance criterion could be met with a milder cooking process (e.g., $\Sigma R \leq 3.4$).

The only means available to the ground beef producer to ensure that the patties will be cooked sufficiently to meet the FSO in restaurants, institutions, hotels, and other commercial operations would be to provide cooking instructions. This also would be necessary for consumer package labeling, but it is less certain whether consumers follow these instructions. In some regions of the world, regulations specify minimum cooking temperatures for ground beef. In the US, the Food Code (FDA, 1999) recommends that ground beef be cooked to an internal temperature of 66°C for 1 min, 68°C for 15 s or 70°C for < 1 s. These time-temperature relationships provide a 6.5D or greater reduction for salmonellae, a pathogen of comparable heat resistance to *E. coli* O157:H7. Many states have adopted these recommendations. Food service operations that comply with this requirement would be applying a validated cooking process that would exceed the above performance standard. One reason for the greater thermal destruction required in the Food Code is that a value of 1000 g^{-1} was assumed for the estimated initial concen-

tration of *Salmonella* and *E. coli* O157:H7 in ground beef ($H_0 = 3$), and a minimum 6D reduction was desired ($\Sigma R = 6$).

17.4.4 Options Involving Research and Education

Information from the above risk evaluation could lead to several additional risk management options. Examples could include:

- support research that may lead to on-farm control measures (e.g., competitive exclusion, vaccine, control of water supply for cattle). This option should be viewed as a long-term endeavor.
- support research that may lead to additional in-plant control measures to minimize contamination during slaughter and/or decontamination of carcasses.
- provide education to food handlers, particularly those in higher risk situations (e.g., non-fast food service establishments, homes), for proper handling and preparation of ground beef for serving.
- encourage the use of a thermometer and discourage reliance on appearance for estimating doneness when cooking ground beef.
- evaluate the barriers to acceptance of irradiation of ground beef and assess how they may be overcome.
- support research on new technologies to reduce the level of *E. coli* O157:H7 in ground beef while retaining acceptable quality.

17.5 ACCEPTANCE CRITERIA

17.5.1 Organoleptic Criteria

The presence of *E. coli* O157:H7 in raw ground beef cannot be assessed by organoleptic evaluation and, thus, meaningful organoleptic criteria cannot be established.

17.5.2 Chemical and Physical Criteria

The presence of *E. coli* O157:H7 in raw ground beef cannot be assessed by chemical or physical determinations and, thus, meaningful chemical or physical criteria cannot be established.

17.5.3 Microbiological Criteria

The following considers the merits of testing frozen ground beef patties to verify that the FSO has been met by the manufacturer.

While it may not be possible to detect with confidence lots with a low prevalence of contamination, it may be possible to detect some lots of higher prevalence that are more likely to be associated with outbreaks. Thus, one risk management option may be to implement a routine sampling program specifically intended to detect those lots of relatively higher prevalence and, perhaps, concentration. Rare as those lots may be, they are more likely to be associated with illness. It must be emphasized, however, that such a sampling and testing program cannot guarantee the safety of lots that test negative!

The decision to test the frozen ground beef patties as a risk management option can be influenced by many factors, such as:

- the potential public health impact, particularly to young children, in the event *E. coli* O157:H7 is present.
- the intended customer (e.g., food service or home) and intended use (i.e., likelihood of undercooking).
- the effectiveness of product labeling and supporting education to reduce the likelihood of mishandling and undercooking.
- costs associated with illness and liability should product be implicated.

17.5.3.1 Choice of Sampling Plan

The sampling plans recommended in Chapter 8 consider risk to the consumer, including the effect certain microbial agents may have on certain sensitive populations. *E. coli* O157:H7 has been placed into a classification that requires the most stringent sampling plans (i.e., cases 13–15) and 15, 30, or 60 sample units from a lot.

The decision to select 15, 30, or 60 samples depends on whether the hazard would be expected to decrease, not change, or increase between when a food is sampled and when it is consumed (NAS-NRC, 1969; Foster, 1971; NRC, 1985; ICMSF, 1986).

E. coli O157:H7 cannot multiply in frozen ground beef patties; extended holding times above 7–8 °C are needed for even a 10-fold increase. Thus, case 15 does not apply.

Since cooking will kill *E. coli* O157:H7, this suggests that a sampling plan of $n = 15$, $c = 0$ (case 13) would be appropriate. However, approximately 20% of consumers continue to consume rare ground beef. While it would be expected that partial cooking may reduce the number of *E. coli* O157:H7, cooking to a rare state cannot be depended on to eliminate the hazard. This leads to a sampling plan of $n = 30$, $c = 0$ (case 14) as being appropriate for raw ground beef. Recovery of *E. coli* O157:H7 from frozen ground beef implicated in outbreaks is evidence that freezing cannot be relied upon to eliminate the pathogen, further supporting selection of case 14.

It is important to reemphasize the limitations of testing food for the purpose of detecting and eliminating positive lots. For example, even with the proposed sampling plan of $n = 30$, $c = 0$, there is a 21% probability of accepting a lot when the proportion of positive sample units throughout the lot is 5%. There is a 74% probability of accepting a lot when the proportion of positive sample units is 1% (see Table 17–3).

17.5.3.2 Lot

The National Academy of Sciences-National Research Council Salmonella Committee (NAS-NRC, 1969) defined a lot as:

- an identifiable and identified unit of production, distinguishable from other units, that has been packaged
- within a determined period of time
- without major interruptions of flow, shutdowns, or other changes (such as the use of different sources of raw materials) that could be expected to cause one portion of the lot to differ significantly in integrity from another and that is
- complete and
- accessible for inspection and testing.

The ICMSF has defined a lot as follows:

In the commercial sense, a lot is a quantity of food supposedly produced under identical conditions, all packages of which would normally bear a lot number that identifies the production during a particular time interval, and usually from a particular line, retort, or other critical processing unit. Statistically, a lot is considered as a collection of units of a product from which a sample is drawn to determine acceptability of the lot (ICMSF, 1986).

In this example, the maximum size of a lot consists of all the ground beef produced on common equipment from cleanup to cleanup. Smaller lots could be established (e.g., hourly).

17.5.3.3 Sample and Analytical Units

A sample unit is a representative sample collected at random from a lot that is a small-scale replica of the lot itself and from which a smaller analytical unit may be drawn for analysis.

- The sample unit is one frozen patty.
- The analytical unit is 25 g.

Ideally, the sample units would be submitted to a certified laboratory and analyzed by an official method, or its equivalent, for *E. coli* O157:H7. The method can be adjusted for analysis of 15 analytical units into a single 375 g composite sample. Research has validated that up to fifteen 25 g analytical units of ground beef or trimmings can be composited without significant loss in *E. coli* O157:H7 detection sensitivity (Silliker & Nickelson, 1995). This permits testing at a more reasonable cost and makes it more practical to screen lots to detect those of higher prevalence. The statistical implications of the proposed sampling plan are discussed in the following section.

17.5.3.4 Disposition of Positive Lots

The definition of a lot, the amount of product in a lot, and means of disposition are important considerations in the disposition of a positive lot. In this example, a lot consists of all product produced on common equipment from cleanup to cleanup, including rework. Rework consists of deformed, broken, or underweight patties. Rework can be returned to the grinder or blender on a frequent basis during operation, with the portion remaining at the end of production being discarded or handled separately. None would be added into the next day's production. Possible risk management options for positive lots include diverting the ground beef for use in products to be cooked in a facility that has an effective control system (i.e., GHP, HACCP) to ensure proper handling and destruction of the pathogen. Cooking can provide a substantial margin of safety that *E. coli* O157:H7 will be killed. It is recommended, however, that such lots not be used for the manufacture of fermented meats or in high temperature-short time processes, such as for links, patties, or meatballs.

17.6 STATISTICAL IMPLICATIONS OF THE PROPOSED SAMPLING PLAN

One premise of this example is that sampling can be used to screen out some, but not all, lots that exceed the FSO. A major weakness in this premise is that contamination is

more likely to be heterogeneous as illustrated in Figures 10–4b and c. The following discussion assumes, however, that contamination is randomly distributed (i.e., log-normal distribution and standard deviation of 0.8).

The proposed sampling plan involves analysis of 25 g from each of 30 sample units from a lot. A negative result provides 95% confidence that the concentration of *E. coli* O157:H7 in the lot is no more than 1 cell per 250 g (NAS-NRC, 1969; Foster, 1971). In addition, there is a 95% probability that if sampling and testing were extended indefinitely, the positive units found would not exceed 10%, equivalent to a lot average of fewer than one cell in 250 g (NAS-NRC, 1969). The underlying principles for relating the performance of a given sampling plan are explained in Legan *et al.* (2001) and in Chapter 7.

The sampling plans originally proposed by the NAS (NAS-NRC, 1969) and the Interagency-Industry Committee (Foster, 1971) were intended to be applied to suspect lots of food. They were to be used to supplement, not replace, existing routine tests being performed by companies to assess the acceptability of their products. Some companies, however, have adopted the sampling plans for end-product testing where the conditions of operation warranted concern. Similarly, the ICMSF has recommended the use of the appropriate sampling plans as a means to assess incoming lots of food at port-of-entry when no history of the conditions of production and storage is known.

Experience with the foregoing sampling plans has been favorable. Rarely has a food been found to cause foodborne illness, such as salmonellosis, when a lot was sampled and found acceptable. Conversely, foods implicated in foodborne illness normally have been found positive with the sampling plans. For about 30 years, sampling plans involving 15, 30, and 60 sample units from a lot have been accepted and applied by both industry and certain control authorities.

While it may be possible to rationalize the use of 30 sample units for determining the acceptability of a lot, the definition of a lot and the procedure for how and when samples should be collected from a lot are more difficult to justify on a statistical basis. Admittedly, the proposal provides for sampling frequencies that consider the need for procedures to be reasonable in a commercial operation. Confounding the issue is the uncertain distribution of *E. coli* O157:H7 throughout an operating day from cleanup to cleanup. The following from The Committee on *Salmonella* provides some guidance (NAS-NRC, 1969) on this concern.

The matter of *Salmonella* control is complicated by the question of distribution of salmonellae when they do occur in a food product. If this defect is random, it is not controlled by time; i.e., there is an equal opportunity for contamination to occur at any stage of the operation. If, on the other hand, contamination was limited to a certain segment of time during processing, then the defect would not be randomly distributed throughout the lot. If distribution is random, regular sampling procedures will detect the organism; if nonrandom, there is virtually no assurance of control unless the regulatory nature of time is known.

As previously indicated, in sampling a lot, random sampling is recommended. A random sample is one in which any individual tested is as likely to detect contamination as any other. This does not mean that one sample at one given time gives a measure of control of finished product, but rather that selecting samples at random during processing gives information on the lot. It is important that the lot be so defined that the foregoing requirements are met (NAS-NRC, 1969).

In conclusion, the sampling plan proposed above may be useful for screening out lots with a high prevalence of *E. coli* O157:H7 that exceed the FSO ($-2.4 \log_{10}$ cfu g⁻¹). How-

Table 17-4 The Relationship between the Number of 25 g Samples That Would Need To Test Negative To Control a Given Concentration (See Also Figure 17-3)

No. of 25 g samples testing negative	Prevalence (%) needed to detect positives at 95% probability	Log concentration per 25 g controlled at 95% probability	Concentration controlled at 95% probability
60	4.8	-1.33	1/526 g
50	5.8	-1.26	1/455 g
40	7.2	-1.17	1/370 g
30	9.5	-1.05	1/278 g
25	11	-0.97	1/233 g
20	14	-0.87	1/185 g
15	18	-0.73	1/135 g
10	26	-0.52	1/83 g
5	45	-0.10	1/32 g
3	63	-0.27	1/13 g

ever, it is also true that the sampling plan will have a relatively poor ability to detect positive lots where the level of the hazard is not random or is much lower. Another way of explaining this is to consider the likely defect rates. When the mean count is around $-2.4 \log_{10} \text{ cfu g}^{-1}$, the defect rate would be around 10% (sigma 0.8). The current defect rate measured in the FSIS sampling program is closer to 1%, which would equate to a mean count of around $-4.5 \log_{10} \text{ cfu g}^{-1}$, assuming a similar standard deviation (see Table 17-4 and Figure 17-3). Hence, it is important to again reemphasize the limitations of testing

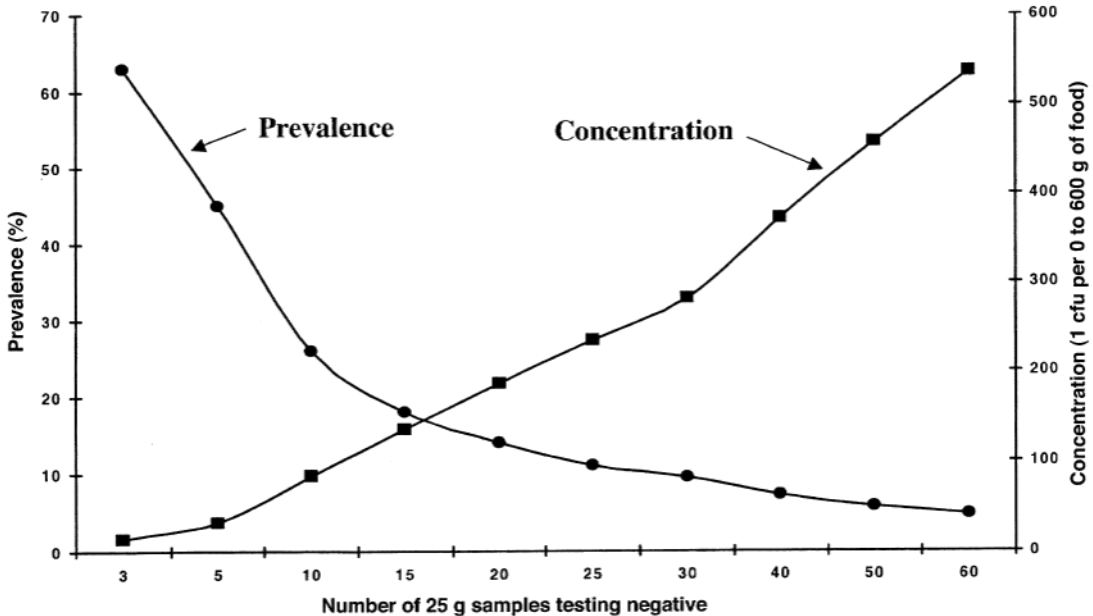


Figure 17-3 Relationship between the number of 25 g samples testing negative and the concentration and prevalence needed before the lot is rejected with 95% probability (assuming sigma = 0.8).

food for the purpose of detecting and eliminating positive lots with a low defect rate. Even with the proposed sampling plan of $n = 30$, $c = 0$, there is a 74% probability of acceptance when the proportion of positive sample units is 1%.

17.7 REFERENCES

- Acheson, D. W. K. (2000). How does *Escherichia coli* O157:H7 testing in meat compare with what we are seeing clinically? *J Food Prot* 63, 819–821.
- AGA (American Gastroenterological Association) (1994). *Consensus Conference Statement. E. coli O157:H7 Infections: An Emerging National Health Crisis*, July 11–13, 1994.
- AMI (American Meat Institute) (2000). *Meat & Poultry Facts 1999*. Washington, DC: American Meat Institute.
- AMSA (American Meat Science Association) (1999). *The Role of Microbiological Testing in Beef Food Safety Programs. The Scientific Perspective. Consensus of the 1999 Symposium*. Kansas City, MO: The American Meat Science Association.
- Arnold, K. W. & Kaspar, C. W. (1995). Starvation and stationary phase induced acid tolerance in *Escherichia coli* O157:H7. *Appl Environ Microbiol* 61, 2037–2039.
- Bacon, R. T., Belk, K. E., Sofos, J. N. *et al.* (2000). Microbial populations on animal hides and beef carcasses at different stages of slaughter in plants employing multiple-sequential interventions for decontamination. *J Food Prot* 63, 1080–1086.
- Benjamin, M. M. & Datta, A. R. (1995). Acid tolerance of enterohemorrhagic *Escherichia coli*. *Appl Environ Microbiol* 61, 1669–1672.
- Bernard, D. T., Cole, W. R., Gombas, D. E. *et al.* (1997). Frozen, raw beef patties for food service. *Dairy, Food & Environ San* 17, 427–431.
- Buchanan, R. L. & Whiting, R. C. (1996). *USDA Pathogen Modeling Program*, version 5.1. Philadelphia, PA: Agricultural Research Service–US Department of Agriculture.
- Cassin, M. H., Lammerding, A. M., Todd, E. C. D. *et al.* (1998). Quantitative risk assessment for *Escherichia coli* O157:H7 in ground beef hamburgers. *Int J Food Microbiol* 41, 21–44.
- Castillo, A., Lucia, L. M., Goodson, K. J. *et al.* (1998). Comparison of water wash, trimming, and combined hot water and lactic acid treatments for reducing bacteria of fecal origin on beef carcasses. *J Food Prot* 61, 823–828.
- CDC (Centers for Disease Control & Prevention) (1998a). Multistate surveillance for food-handling, preparation, and consumption behaviors associated with foodborne diseases: 1995 and 1996 BRFSS food-safety questions. *MMWR Morb Mortal Wkly Rep* 47/No. SS-4.
- CDC (Centers for Disease Control & Prevention) (1998b). *FoodNet 1997 Surveillance Results*. <http://www.cdc.gov/ncidod/dbmd/foodnet/foodnet.htm>
- CDC (Centers for Disease Control & Prevention) (2000). Preliminary FoodNet data on the incidence of food-borne illnesses—selected sites, United States, 1999. *MMWR* 49, 201–205.
- CFIA (Canadian Food Inspection Agency) (2000). Canadian Microbiological Baseline Survey of Chicken Broiler and Young Turkey Carcasses, June 1997–May 1998. Canadian Food Inspection Agency, Poultry Inspection Programs, Food of Animal Origin, Ottawa, Ontario. (available through website: <http://www.inspection.gc.ca/english/anima/meavia/mmopmmhv/chap19/baseline-e.pdf>).
- Cheville, A. M., Arnold, K. W., Buchreiser, C. *et al.* (1996). *rpoS* regulation of acid, heat, salt tolerance in *Escherichia coli* O157:H7. *Appl Environ Microbiol* 62, 1822–1824.
- Dorsa, W. J. (1997). New and established carcass decontamination procedures commonly used in the beef-processing industry. *J Food Prot* 60, 1146–1151.
- Dorsa, W. J., Cutter, C. N., Siragusa, G. R. & Koohmaraie, M. (1996). Microbial decontamination of beef and sheep carcasses by steam, hot water spray washes, and a steam-vacuum sanitizer. *J Food Prot* 59, 127–135.
- Eisel, E. G., Linton, R. H. & Muriana, P. M. (1997). A survey of microbial levels for incoming raw beef, environmental sources, and ground beef in a red meat processing plant. *Food Microbiol* 14, 273–282.
- Elder, R. O., Keen, J. E., Siragusa, G. R. *et al.* (2000). Correlation of enterohemorrhagic *Escherichia coli* O157 prevalence in feces, hides and carcasses of beef cattle during processing. *Proc Natl Acad Sci* 97, 2999–3003.

- FDA (Food and Drug Administration) (1999). *Food Code*. Washington, DC: US Department of Health and Human Services, Public Health Service, Food and Drug Administration.
- Foster, E. M. (1971). The control of salmonellae in processed foods: A classification system and sampling plan. *JAOAC* 54, 259–266.
- FSIS (Food Safety and Inspection Service) (1993). *Report on the Escherichia coli O157:H7 Outbreak in the Western States*. Washington, DC: US Department of Agriculture.
- FSIS (Food Safety and Inspection Service) (1994). Microbiological testing program for *Escherichia coli* O157:H7 in raw ground beef. FSIS Notice 50–94 (12–23–94), US Department of Agriculture, Washington, DC.
- FSIS (Food Safety and Inspection Service) (1996). Nationwide Federal Plant Raw Ground Beef Microbiological Survey. August 1993–March 1994. US Department of Agriculture, Science and Technology Division, Microbiology Division, Washington, DC.
- FSIS (Food Safety and Inspection Service) (1998). *Preliminary Pathways and Data for a Risk Assessment of E. coli O157:H7 in Beef*. Prepared by the *E. coli* O157:H7 risk assessment team. October 28, Washington, DC.
- Hancock, D. D. (1998). *A Summary of E. coli O157:H7 Research at Washington State University*. Handout from Governor's Conference on Ensuring Meat Safety, Developing a Research and Outreach Agenda on *E. coli* O157:H7. February 9–10, Lincoln, NB. Pullman, WA: Washington State University.
- ICMSF (International Commission on Microbiological Specifications for Foods) (1986). *Microorganisms in Foods 2. Sampling for Microbiological Analysis: Principles and Specific Applications*, 2nd edn. Toronto: University of Toronto Press.
- ICMSF (International Commission on Microbiological Specifications for Foods) (1996). Intestinally pathogenic *Escherichia coli*. In *Microorganisms in Foods 5. Characteristics of Microbial Pathogens*, pp. 126–140. Gaithersburg, MD: Aspen Publishers, Inc.
- Legan, J. D., Vandeven, M. H., Dahms, S. & Cole, M. B. (2001). Determining the concentration of microorganisms controlled by attributes sampling plans. *Food Control* 12, 137–147.
- Leyer, G. J., Wang, L.-L. & Johnson, E. A. (1995). Acid adaptation of *Escherichia coli* O157:H7 increases survival in acidic foods. *Appl Environ Microbiol* 61, 3752–3755.
- Line, J. E., Fain, A. R., Moran, A. B. *et al.* (1991). Lethality of heat to *Escherichia coli* O157:H7: D-value and z-value determinations in ground beef. *J Food Prot* 54, 762–766.
- Mahon, B. E., Griffin, P. M., Mead, P. S. & Tauxe, R. V. (1997). Hemolytic uremic syndrome surveillance to monitor trends in infection with *Escherichia coli* O157:H7 and other Shiga toxin-producing *E. coli*. *Emerging Infect Disease* 3, 409–412.
- Mead, P. S., Slutsker, L., Dietz, V. *et al.* (1999). Food-related illness and death in the United States. *Emerging Infect Disease* 5, 607–625.
- NAS-NRC (National Academy of Sciences-National Research Council) (1969). *An Evaluation of the Salmonella Problem*. Publication No. 1683, Washington, DC: Committee on Salmonella, NAS-NRC.
- NRC (National Research Council) (1985). *An Evaluation of the Role of Microbiological Criteria for Foods and Ingredients*. Subcommittee on Microbiological Criteria. Washington, DC: National Academy Press.
- Nutsch, A. L., Phebus, R. K., Riemann, M. J. *et al.* (1998). Steam pasteurization of commercially slaughtered beef carcasses: evaluation of bacterial populations at five anatomical locations. *J Food Prot* 61, 571–577.
- Scanga, J. A., Grona, A. D., Belk, K. E. *et al.* (2000). Microbiological contamination of raw beef trimmings and ground beef. *Meat Sci* 56, 145–152.
- Silliker, J. H. & Nickelson, R. (1995). *Methods for Sampling and Compositing Fresh Red Meat Products for Analysis of Pathogenic and Indicator Bacteria*. Final Report to the National Cattleman's Beef Association (addendum of September 6, 1995). Englewood, CO: National Cattleman's Beef Association.
- Sofos, J. N., Belk, K. E. & Smith, G. C. (1999). Processes to reduce contamination with pathogenic microorganisms in meat. In *Congress Proceedings*, 7-L2 pp. 597–606. 45th International Congress of Meat Science and Technology, August 1–6, Yokohama, Japan.
- Sofos, J. N. & Smith, G. C. (1998). Nonacid meat decontamination technologies: Model studies and commercial applications. *Int J Food Microbiol* 44, 171–188.
- Tarr, P. I. (1994). *Testimony to Washington State Senate, Department of Agriculture*. January 20, 1994.
- Todd, E. C. D., Szabo, R. A., Peterkin, P. *et al.* (1988). Rapid hydrophobic grid membrane filter-enzyme-labeled antibody procedure for identification and enumeration of *Escherichia coli* O157 in foods. *Appl Environ Microbiol* 54, 2536–2540.

- Tompkin, R. B. & Bodnaruk, P. W. (1999). A proposed food safety management plan for *E. coli* O157:H7 in ground beef. In *The Role of Microbiological Testing in Beef Food Safety Programs*. Edited by C. Calkins and M. Koochmaraie. Kansas City, MO: American Meat Science Association.
- USDA (US Department of Agriculture–Animal and Plant Health Inspection Service–Centers for Epidemiology and Animal Health) (1994). *Escherichia coli* O157:H7 *Issues and Ramifications*. Fort Collins, CO: US Department of Agriculture, Animal and Plant Health Inspection Service, Centers for Epidemiology and Animal Health.
- WHO (World Health Organization) (1997). *Prevention and Control of Enterohaemorrhagic Escherichia coli (EHEC) Infections* (WHO/FSF/FOS/97.6). Geneva: Food Safety Unit, Programme of Food Safety and Food Aid, World Health Organization.

Glossary

Acceptance criteria (for a food operation)

Statements of conditions that differentiate acceptable from unacceptable food operations.

Acceptance criteria (for lot acceptance)

Statements of conditions that differentiate acceptable from unacceptable lots (batches) of food.

Control (verb)

To take all necessary actions to ensure and maintain compliance with established criteria.

Control (noun)

The state wherein correct procedures are being followed and criteria are being met.

Control measure

Any action and activity that can be used to prevent or eliminate a food safety hazard or reduce it to an acceptable level.

Corrective action

Any action to be taken when the results of monitoring at the critical control point indicate a loss of control.

Critical control point (CCP)

A step at which control can be applied and is essential to prevent or eliminate a food safety hazard or reduce it to an acceptable level.

Critical limit

A criterion which separates acceptability from unacceptability.

Default criterion

A conservative criterion established to ensure the safety of a food under worst-case conditions.

Deviation

Failure to meet a critical limit.

Dose-response assessment

The determination of the relationship between the magnitude of exposure (dose) to a chemical, biological, or physical agent and the severity and/or frequency of associated adverse health effects (response).

Expert panel

A group of individuals who collectively have knowledge or experience with a hazard or food and the conditions that can lead to foodborne illness, and who have the ability to provide advice based on available scientific information.

Exposure assessment

The qualitative and/or quantitative evaluation of the likely intake of biological, chemical, and physical agents via food, as well as exposures from other sources if relevant.

Flow diagram

A systematic representation of the sequence of steps or operations used in the production or manufacture of a particular food item.

Food operation

A location along the food chain where food is handled or prepared for commercial reasons.

Food safety objective (FSO)

A statement of the maximum frequency and/or concentration of a microbiological hazard in a food at the time of consumption that provides the appropriate level of protection.

Hazard Analysis Critical Control Point (HACCP)

A system that identifies, evaluates, and controls hazards which are significant for food safety.

HACCP plan

A document prepared in accordance with the principles of HACCP to ensure control of hazards that are significant for food safety in the segment of the food chain under consideration.

Hazard

A biological, chemical, or physical agent in, or condition of, food with the potential to cause an adverse health effect.

Hazard analysis (in HACCP)

The process of collecting and evaluating information on hazards and conditions leading to their presence to decide which are significant for food safety and therefore should be addressed in the HACCP plan.

Hazard characterization

The qualitative and/or quantitative evaluation of the nature of the adverse health effects associated with the hazard. For the purpose of microbiological risk assessment the concerns relate to microorganisms and their toxins.

Hazard identification

The identification of biological, chemical, and physical agents capable of causing adverse health effects and which may be present in a particular food or group of foods.

Monitor

The act of conducting a planned sequence of observations or measurements of control parameters to assess whether a CCP is under control.

Performance criterion

The required outcome of a step, or combination of steps, that contribute to assuring a food safety objective is met.

Process criteria

The control parameters of a step, or combination of steps, that can be applied to achieve a performance criterion.

Product criteria

A parameter of a food that can be used to assess the acceptability of a lot or consignment.

Qualitative risk assessment

A risk assessment based on data which, while forming an inadequate basis for numerical risk estimations, nonetheless, when conditioned by prior expert knowledge and identification of attendant uncertainties permits risk ranking or separation into descriptive categories of risk.

Quantitative risk assessment

A risk assessment that provides numerical expressions of risk and indication of the attendant uncertainties.

Risk

A function of the probability of an adverse health effect, and the severity of that effect, consequential to a hazard(s) in food.

Risk analysis

A process consisting of three components: risk assessment, risk management, and risk communication.

Risk assessment

A scientifically based process consisting of the following steps: (i) hazard identification, (ii) hazard characterization, (iii) exposure assessment, and (iv) risk characterization.

Risk characterization

The process of determining the qualitative and/or quantitative estimation, including attendant uncertainties, of the probability of occurrence and severity of known or potential adverse health effects in a given population based on hazard identification, hazard characterization, and exposure assessment.

Risk communication

The interactive exchange of information and opinions throughout the risk analysis process concerning hazards and risks, risk-related factors and risk perceptions, among

risk assessors, risk managers, consumers, industry, the academic community, and other interested parties, including the explanation of risk assessment findings and the basis of risk management decisions.

Risk estimate

Output of risk assessment.

Risk management

The process, distinct from risk assessment, of weighing policy alternatives, in consultation with all interested parties, considering risk assessment and other factors relevant for the health protection of consumers and for the promotion of fair trade practices, and, if needed, selecting appropriate prevention and control options.

Safe food (the Codex document gives this as the definition for food safety – not safe food)

Food that does not cause harm to the consumer when it is prepared and/or eaten according to its intended use.

Sensitivity analysis

A method used to examine the behavior of a model by measuring the variation in its outputs resulting from changes in its inputs.

Step

A point, procedure, operation, or stage in the food chain including raw materials, from primary production to final consumption.

Tolerable level of risk (TLR)

The level of risk proposed following consideration of public health impact, technological feasibility, economic implications, and that which society regards as reasonable in the context of and in comparison with other risks in everyday life.

Transparent

Characteristics of a process where the rationale, the logic of development, constraints, assumptions, value judgements, decisions, limitations, and uncertainties of the expressed determination are fully and systematically stated, documented, and accessible for review.

Uncertainty analysis

A method used to estimate the uncertainty associated with model inputs, assumptions and structure/form.

Validation

Obtaining evidence that the elements of the HACCP plan are effective.

Verification

The application of methods, procedures, tests, and other evaluations, in addition to monitoring to determine compliance with the HACCP plan.

Objectives and Accomplishments of the International Commission on Microbiological Specifications for Foods

HISTORY AND PURPOSE

The International Commission on Microbiological Specifications for Foods (ICMSF, the Commission) was formed in 1962 through the action of the International Committee on Food Microbiology and Hygiene, a committee of the International Union of Microbiological Societies (IUMS). Through the IUMS, the ICMSF is linked to the International Union of Biological Societies (IUBS) and to the World Health Organization (WHO) of the United Nations.

In the 1960s, there was growing recognition of foodborne disease and greatly increased microbiological testing of foods. This, in turn, created unforeseen problems in international trade in foods. Different analytical methods and sampling plans of doubtful statistical validity were being used. Furthermore, analytical results were interpreted using different concepts of biological significance and acceptance criteria, creating confusion and frustration for both the food industry and regulatory agencies.

In this environment, ICMSF was founded to: (1) assemble, correlate, and evaluate evidence about the microbiological safety and quality of foods; (2) consider whether microbiological criteria would improve and assure the microbiological safety of particular foods; (3) propose, where appropriate, such criteria; and (4) recommend methods of sampling and examination.

Thirty years later, the primary role of the Commission remains to give guidance on: (1) appraising and controlling the microbiological safety of foods and (2) microbiological quality, since this influences consumer acceptance and losses due to spoilage. Meeting those objectives assists international trade, national control agencies, the food industry, international agencies concerned with humanitarian food distribution, and consumer interests.

FUNCTIONS AND MEMBERSHIP

The ICMSF provides basic scientific information through extensive study and makes recommendations without prejudice based on that information. Results of the studies are

published as books, discussion documents, or refereed papers. Major publications of the Commission are listed in Appendix D.

The ICMSF functions as a working party, not as a forum for the reading of papers. Meetings consist largely of discussions within subcommittees, debating to achieve consensus, editing draft materials, and planning. Most work is done between meetings by the Editorial Committee and members, sometimes with the help of nonmember consultants.

Since 1962, 33 meetings have been held in 19 countries (Australia, Brazil, Canada, Denmark, Dominican Republic, Egypt, England, France, Germany, Italy, Mexico, South Africa, Spain, Switzerland, The Netherlands, the United States, the former USSR, Venezuela, and the former Yugoslavia). During its meetings, Commission members frequently participate in symposia organized by microbiologists or public health officials of the host country.

Currently, the membership consists of 15 food microbiologists from 9 countries, with combined professional interests in research, public health, official food control, education, product and process development, and quality control, from government laboratories in public health, agriculture, and food technology; from universities; and from the food industry (see Appendix C). The ICMSF is also assisted by consultants, specialists in particular areas of microbiology who are critical to the success of the Commission (see Appendix C for lists of the consultants, contributors, and reviewers of this volume). New members and consultants are selected for their expertise, not as national delegates. All work is voluntary without fees or honoraria.

Three Subcommissions (Latin American, South-East Asian, and Balkan and Danubian) promote ICMSF's activities among food microbiologists in their regions and facilitate communication worldwide (see Appendix C).

The ICMSF raises its own funds to support its meetings. Support has been obtained from government agencies, WHO, IUMS, IUBS, and the food industry (over 80 food companies and agencies in 13 countries). Grants for specific projects and seminars/conferences have been provided by a variety of sources. Some funds are received from the sale of its books.

RECENT PROJECTS

Microorganisms in Foods 5: Characteristics of Microbial Pathogens (1996) is a thorough, but concise, review of the literature on growth, survival, and death responses of foodborne pathogens. It is intended as a quick reference manual to assist in making decisions in support of Hazard Analysis Critical Control Plans (HACCP) and to improve food safety. (Detailed publication information on the major documents mentioned here is available in Appendix D.)

Microorganisms in Foods 6: Microbial Ecology of Food Commodities (1998) updates and extends an earlier volume in the ICMSF series (1980). For 16 commodity areas, Volume 6 describes the initial microbial flora and the prevalence of pathogens, the microbiological consequences of processing, typical spoilage patterns, episodes implicating those commodities with foodborne illness, and measures to control pathogens.

Discussion Documents Prepared for the Joint Food and Agriculture Organization (FAO) and World Health Organization (WHO) Food Standards Programme, Codex Alimentarius Commission

1. Establishment of Sampling Plans for Microbiological Safety Criteria for Foods in International Trade

2. Discussion of Sampling Plans for *L. monocytogenes*, *Salmonella*, *Campylobacter* and Verocytotoxin-Producing *E. coli* in Foods in International Trade
3. Recommendations for the Future Management of Microbiological Hazards for Foods in International Trade
4. Principles for the Establishment of Food Safety Objectives and Related Control Measures

ICMSF's recommendations for sampling foods and acceptance criteria for *Listeria monocytogenes* were subsequently published as "Sampling plans for *Listeria monocytogenes*" (1994, *Int J Food Microbiol* 22, 89–96), as was "Establishment of microbiological safety criteria for foods in international trade" (1997, *World Health Stats Quarterly*, 50, 119–123).

At the request of the Secretariat of Codex, the ICMSF developed recommendations for revision of the document Principles for the Establishment and Application of Microbiological Criteria for Foods, originally published in the Procedural Manual of Codex.

Addressing the need for a scientific basis in risk assessment, a working group of the ICMSF published "Potential application of risk assessment techniques to microbiological issues related to international trade in food and food products" (1998, *J Food Prot* 61, 1075–1086).

PAST AND FUTURE

For almost 25 years, the ICMSF's major efforts were devoted to methodology. This resulted in improved comparisons of microbiological methods and better standardization (17 refereed publications). Among many significant findings, it was established that when analyzing for salmonellae, analytical samples could be bulked (composited) into a single test with no loss of sensitivity. This made practical the collection and analysis of the large number of samples recommended in some sampling plans.

With the rapid development of alternative methods and rapid test kits, and the ever expanding list of biological agents involved in foodborne illness, the Commission reluctantly discontinued its program of comparison and evaluation of methods in 1986, recognizing that issues of methodology were being addressed effectively by other organizations.

A long-term objective of the Commission has been to enhance the microbiological safety of foods in international commerce. This was initially addressed through books that recommended uniform analytical methods (ICMSF, 1978) and sound sampling plans and criteria (ICMSF, 1974, 1978, 1986). The Commission then developed a two volume set on the microbial ecology of foods (ICMSF, 1980a, b) intended to familiarize analysts with processes used in the food industry and microbiological aspects of foods submitted to the laboratory. Knowledge of the microbiology of the major food commodities and the factors affecting the microbial content of these foods helps analysts to interpret analytical results.

At an early stage, the Commission recognized that no sampling plan can ensure the absence of a pathogen in food. Testing foods at ports-of-entry or elsewhere in the food chain cannot guarantee food safety. This led the Commission to explore the potential value of HACCP for enhancing food safety. A meeting in 1980 with the WHO led to a report on the use of HACCP for controlling microbiological hazards in food, particularly in developing countries (ICMSF, 1982). The Commission then developed a book on the

principles of HACCP and procedures for developing HACCP plans (ICMSF, 1988), covering the importance of controlling the conditions of producing/harvesting, preparing, and handling foods. Recommendations were given in that volume for the application of HACCP from production/harvest to consumption, together with examples of how HACCP can be applied at each step in the food chain.

The Commission next recognized that a major weakness in the development of HACCP plans is the process of hazard analysis. It has become more difficult to be knowledgeable about the many biological agents recognized as responsible for foodborne illness. ICMSF (1996) summarizes important information about the properties of biological agents commonly involved in foodborne illness and serves as a quick reference manual when making judgments on the growth, survival, or death of pathogens.

Subsequently, the Commission updated its volume on the microbial ecology of food commodities (ICMSF, 1998).

The current book, *Microorganisms in Foods 7: Microbiological Testing in Food Safety Management* (2001), illustrates how systems such as HACCP and Good Hygienic Practices (GHP) provide greater assurance of safety than microbiological testing, but also identifies circumstances in which microbiological testing still plays a useful role.

We believe that the original objectives of the Commission are still relevant today. The European Union, the many other political changes occurring throughout the world, the growth of developing countries seeking export markets, and the increased trade in foods worldwide, as evidenced by the passage of the General Agreement on Tariffs and Trade (GATT) and the North American Free Trade Agreement (NAFTA) all point to the continuing need for the independent recommendations on food safety, such as those of the Commission. It is essential that import/export policies be established as uniformly as possible and on a sound scientific basis. The Commission's overall goal will continue to be to enhance the safety of foods moving in international commerce. The Commission will continue to strive to meet this goal through a combination of educational materials, promoting the use of food safety management systems using microbiological food safety objectives, HACCP, and GHP, and recommending sampling plans and microbiological criteria where they have been developed according to Codex principles and offer increased assurance of microbiological safety. The future success of ICMSF will continue to depend upon the efforts of members, support from consultants who generously volunteer their time, and those who provide the financial support so essential to the Commission's activities.

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Publications of the ICMSF

BOOKS

- Food and Agriculture Organization & International Atomic Energy Agency/ICMSF (1970). *Microbiological Specifications and Testing Methods for Irradiated Foods*. Technical Report Series No. 104, Vienna: International Atomic Energy Agency.
- ICMSF (1978). (Reprinted 1982, 1988 with revisions). *Microorganisms in Foods 1. Their Significance and Methods of Enumeration*, 2nd edn. Toronto: University of Toronto Press. (ISBN 0-8020-2293-6).
- ICMSF (1980). *Microbial Ecology of Foods. Volume 1. Factors Affecting Life and Death of Microorganisms*, New York: Academic Press. (ISBN 0-12-363501-2).
- ICMSF (1980). *Microbial Ecology of Foods. Volume 2. Food Commodities*, New York: Academic Press. (ISBN 0-12-363502-0).
- ICMSF (1986). *Microorganisms in Foods 2. Sampling for Microbiological Analysis: Principles and Specific Applications*, 2nd edn. Toronto: University of Toronto Press. (ISBN 0-8020-5693-8) (available outside North America from Blackwell Scientific Publications, Ltd., Osney Mead, Oxford OX2 0EL, UK) (First edition: 1974; revised with corrections, 1978).
- ICMSF (1988). *Microorganisms in Foods 4. Application of the Hazard Analysis Critical Control Point (HACCP) System to Ensure Microbiological Safety and Quality*. Oxford: Blackwell Scientific Publications (ISBN 0-632-02181-0). Also published in paperback under the title *HACCP in Microbiological Safety and Quality* (1988) (ISBN 0-632-02181-0).
- ICMSF (1996). *Microorganisms in Foods 5. Characteristics of Microbial Pathogens*. Gaithersburg, MD: Aspen Publishers Inc. (ISBN 0-412-47350-X).
- ICMSF (1998). *Microorganisms in Foods 6. Microbial Ecology of Food Commodities*. Gaithersburg, MD: Aspen Publishers Inc. (ISBN 0-8342-1825-9). Gaithersburg, MD: Aspen Publishers Inc.
- (Aspen Publishers Inc., 200 Orchard Ridge Dr, Suite 200, Gaithersburg MD 20878, USA, 800-638-8347, <http://www.aspenpublishers.com>. Outside the USA, contact Plymbridge Ltd., Plymouth, UK, tel. (44) 1752 202 301.)

WHO PUBLICATIONS

- ICMSF (authors: J. H. Silliker, A. C. Baird-Parker, F. L. Bryan, J. C. Olson, Jr., B. Simonsen, & M. van Schothorst)/WHO (1982). *Report of the WHO/ICMSF meeting on Hazard Analysis: Critical Control Point System in Food Hygiene*. WHO/VPH/82.37. Geneva: World Health Organization. (also available in French).
- Christian, J. H. B. (1983). *Microbiological Criteria for Foods*. (Summary of recommendations of FAO/WHO expert consultations and working groups 1975-1981). WHO/VPH/83.54. Geneva: World Health Organization.
- ICMSF (authors: B. Simonsen, F. L. Bryan, J. H. B. Christian, T. A. Roberts, J. H. Silliker, & R. B. Tompkin) (1986). *Prevention and Control of Foodborne Salmonellosis through*

Application of the Hazard Analysis Critical Control Point System. Report, International Commission on Microbiological Specifications for Foods (ICMSF). WHO/CDS/VPH/86.65. Geneva: World Health Organization.

OTHER ICMSF TECHNICAL PAPERS

- Thatcher, F. S. (1963). The microbiology of specific frozen foods in relation to public health: Report of an international committee. *J Appl Bacteriol* 26, 266–285.
- Simonsen, B., Bryan, F. L., Christian, J. H. B., Roberts, T. A., Tompkin, R. B., & Silliker, J. H. (1987). Report from the International Commission on Microbiological Specifications for Foods (ICMSF). Prevention and control of foodborne salmonellosis through application of hazard analysis critical control point (HACCP). *Int J Food Microbiol* 4, 227–247.
- ICMSF (1994). Choice of sampling plan and criteria for *Listeria monocytogenes*. *Int J Food Microbiol* 22, 89–96.
- ICMSF (1997). Establishment of microbiological safety criteria for foods in international trade. *World Health Stats Quarterly* 50, 119–123.
- ICMSF (1998). Potential application of risk assessment techniques to microbiological issues related to international trade in food and food products. *J Food Prot* 61, 1075–1086.
- ICMSF [M. van Schothorst, Secretary] (1998). Principles for the establishment of microbiological food safety objectives and related control measures. *Food Control* 9, 379–384.

TRANSLATIONS

- Thatcher, F. S. & Clark, D. S. (1973). *Microorganisms in Foods 1. Their Significance and Methods of Enumeration*. (in Spanish, translated by B. Garcia). Zaragoza, Spain: Editorial Acribia.
- ICMSF (1981). *Microorganismos de los Alimentos 2. Métodos de Muestreo para Análisis Microbiológicos: Principios y Aplicaciones Específicas*, (in Spanish, translated by J.A. Ordonez Pereda. & M.A. Diaz Hernandez). Zaragoza, Spain: Editorial Acribia.
- ICMSF (1983). *Ecología Microbiana de los Alimentos 1. Factores que Afectan a la Supervivencia de los Microorganismos en los Alimentos*, (in Spanish, translated by J. Burgos Gonzalez et al.). Zaragoza, Spain: Editorial Acribia.
- ICMSF (1984). *Ecología Microbiana de los Alimentos 2. Productos Alimenticios*, (in Spanish, translated by B. Sanz Perez et al.). Zaragoza, Spain: Editorial Acribia.
- ICMSF (1988). *El Sistema de Análisis de Riesgos y Puntos Críticos. Su Aplicación a las Industrias de Alimentos*, (in Spanish, translated by P.D. Malmenda & B.M. Garcia). Zaragoza, Spain: Editorial Acribia.
- ICMSF (1996). *Microorganismos de los Alimentos: Características de los Patógenos Microbianos*, (in Spanish, translated by M. R. Vergés). Zaragoza, Spain: Editorial Acribia.

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- Bartram, M. T. (1967). International microbiological standards for foods. *J Milk & Food Technol* 30, 349–351.
- Saa, C. C. (1968). The Latin American Subcommittee on Microbiological Standards and Specifications for Foods. *Revista de la Facultad de Química y Farmacia* 7, 8.

- Cominazzini, C. (1969). The International Committee on Microbiological Specifications for Foods and its contribution to the maintenance of food hygiene. (In Italian). *Croniche Chimico* 25, 16.
- Saa, C. C. (1969). El Comité Internacional de Especificaciones Microbiológicas de los Alimentos de la IAMS. *Revista de la Facultad de Química y Farmacia* 8, 6.
- Mendoza, S. & Quevedo, F. (1971). Comisión Internacional de Especificaciones Microbiológicas de los Alimentos. *Boletín del Instituto Bacteriológico de Chile* 13, 45.
- Thatcher, F. S. (1971). The International Committee on Microbiological Specifications for Foods. Its purposes and accomplishments. *IAOAC* 54, 836–814.
- Clark, D. S. (1977). The International Commission on Microbiological Specifications for Foods. *Food Technol* 32, 51–54, 67.
- Clark, D. S. (1982). International perspectives for microbiological sampling and testing of foods. *J Food Prot* 45, 667–671.
- Anonymous (1984). International Commission on Microbiological Specifications for Foods. *Food Lab Newsl* 1, 23–25. (Box 622, S-751 26 Uppsala, Sweden)
- Quevedo, F. (1985). Normalización de alimentos y salud para América Latina y el Caribe. 3. Importancia de los criterios microbiológicos. *Boletín de la Oficina Sanitaria Panamericana* 99, 632–640.
- Bryan, F. L. & Tompkin, R. B. (1991). The International Commission on Microbiological Specifications for Foods (ICMSF). *Dairy, Food & Environ San* 11, 66–68.
- Anonymous (1996). The International Commission on Microbiological Specifications for Foods (ICMSF): update. *Food Control* 7, 99–101.

Table of Sources

CHAPTER 2

Figure 2–1. *Source:* Data from Center for Disease Control and Prevention, Atlanta, Georgia.

Figure 2–2. *Source:* Data from Federal Institute for Health Protection of Consumers and Veterinary Medicine (FAO/WHO Collaborating Centre for Research and Training in Food Hygiene and Zoonoses), Berlin, Germany and International Commission on Microbiological Specifications for Food, University of Toronto Press, Toronto, Canada.

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Table 2–1. Courtesy of World Health Organization, 2000, Geneva, Switzerland and the Food and Agriculture Organization of the United Nations from “Exposure Assessment of *Listeria Monocytogenes* in Ready-to-Eat Foods, *Preliminary Report for Joint FAO/WHO Expert Consultation on Risk Assessment of Microbiological Hazards in Foods*, 2000.

Table 2–2. *Source:* Data from Center for Disease Control and Prevention, 2000a, Annual Reports from the Center for Disease Control and Prevention, Preliminary FoodNet Data on the Incidence of Foodborne Illnesses—Selected Sites, United States, 1999, *Morbidity and Mortality Weekly Reports*, Vol. 49, pp. 201–205, Table 2–2.

CHAPTER 3

Figure 3–1. *Source:* Data from Center for Disease Control and Prevention, Atlanta, Georgia.

Figure 3–2. *Source:* Data from Center for Disease Control and Prevention, Atlanta, Georgia.

Figure 3–3. *Source:* Reprinted from Center for Disease Control and Prevention, Atlanta, Georgia.

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Table 3–1. *Source:* Reprinted with permission from R. B. Tompkin and T. V. Kueper, Microbiological Considerations in Developing New Foods. How Factors Other Than Temperature Can be Used to Prevent Microbiological Problems, In *Microbiological Safety of Foods in Feeding Systems*, pp. 100–122, ABMPS Report No. 125, © 1982, Advisory Board on Military Personnel Supplies, National Research Council, National Academy Press.

Table 3–2. *Source:* Reprinted from United States Department of Agriculture, 1996, Pathogen Reduction; Hazard Analysis and Critical Control Point (HACCP) Systems; Final Rule, *Federal Register*, Vol. 61, pp. 38806–38989.

CHAPTER 4

Figure 4–1. *Source:* Adapted with permission from CAC (Codex Alimentarius Commission) 2000. Proposed Draft Framework for Determining the Equivalency of Sanitary Measures Associated

with Food Inspection and Certification Systems. *CCFICS/CX/FICS 00/6, Attachment 1*. Joint FAO/WHO Food Standards Programme, Codex Committee on Food Import and Export Inspection and Certification Systems, Food and Agriculture Organization of the United Nations.

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Table 5–1. Courtesy of International Commission on Microbiological Specifications for Foods, *Microorganisms in Foods 2, Sampling for Microbiological Analysis: Principles and Specific Applications, 2nd Ed.*, Table 25, p. 178 © 1986, University of Toronto Press, Toronto, Canada.

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CHAPTER 9

Exhibit 9-1. Courtesy of International Commission on Microbiological Specifications for Foods, *Microorganisms in Foods 2, Sampling for Microbiological Analysis: Principles and Specific Applications, 2nd Ed.*, Table 11, p. 77, © 1986, University of Toronto Press, Toronto, Canada.

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Table 9-4. Source: Data from R. M. Cannon and R. T. Roe, *Livestock Disease Surveys: A Field Manual for Veterinarians*, Australian Bureau of Animal Health, Department of Primary Industry, Canberra, Australia, 1982, Australian Government Publishing Service.

CHAPTER 10

Figure 10-1. Courtesy of International Commission on Microbiological Specifications for Foods, *Microorganisms in Foods 2, Sampling for Microbiological Analysis: Principles and Specific Applications, 2nd Ed.*, Figure 5, p. 82, © 1986, University of Toronto Press, Toronto, Canada.

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Table 13-3. Source: Reprinted from Food and Safety Inspection Service, 1996, Pathogen Reduction; Hazard Analysis and Critical Control Point (HACCP) Systems; Final Rule, *Federal Register*, Vol. 61, pp. 38806-38989.

CHAPTER 15

Table 15-1. Source: Reprinted from *Food Control*, Vol. 12, No. 3, J. D. Legan, M. H. Vandeven, S. Dahms, M. B. Cole, Determining the Concentration of Microorganisms Controlled by Attributes Sampling Plans, pp. 137-147, © 2001, with permission from Elsevier Science.

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CHAPTER 17

Table 17-1. *Source:* Data from World Health Organization (WHO), 1997, Prevention and Control of Enterohaemorrhagic *Escherichia coli* (EHEC) Infections (WHO/FSF/FOS/97.6). Food Safety Unit, Programme of Food Safety and Food Aid, World Health Organization, Geneva.

Table 17-2. *Source:* Reprinted from Food Safety and Inspection Service, 1993, Report on the *Escherichia coli* O157:H7 Outbreak in the Western States, United States Department of Agriculture, May 21, Washington, DC.

APPENDIX B

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APPENDIX D

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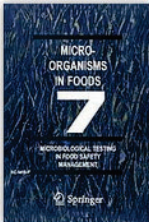
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Series:	Microorganisms in foods, 7
Physical Description:	xiii, 362 pages : illustrations ; 27 cm.
ISBN:	9780834219953, 9780306472626, 0834219956, 0306472627
OCLC Number / Unique Identifier:	47996226
Subjects:	Aliments Normes Food Microbiology Food Microbiology standards Food Standards HACCP Show more
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Description

xiii, 362 p. : ill. ; 27 cm.

ISBN

0834219956

LC classification	QR115 .I46 2002
Related names	Tompkin, R. B. (R. Bruce)
LC Subjects	Food--Microbiology. Food--Standards.
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