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Review

Heat Resistance of *Listeria monocytogenes*

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ABSTRACT

The heat resistance data on *Listeria monocytogenes* in culture media and foods are summarized. Most heat resistance data for foods have been obtained in dairy, meat, poultry, and egg products. Limited data have been published on seafood, fruits, and vegetables. The methodologies employed have evolved over time; hence data from earlier experiments are not directly comparable to more recent studies. Many factors influence the heat resistance of *L. monocytogenes*. Variation exists among different strains in their ability to withstand heat treatment. In addition, heat resistance is influenced by age of the culture, growth conditions, recovery media, and characteristics of foods such as salt content, a_w, acidity, and the presence of other inhibitors. Listeriae are more heat resistant than most other nonspore-forming foodborne pathogens, and thus, processing recommendations based on data from experiments with *Salmonella* spp. or pathogenic *Escherichia coli* may not be sufficient to eliminate similar numbers of *L. monocytogenes*. The data provided in this review may prove useful for food processors in determining appropriate times and temperatures for producing foods free of vegetative pathogens.

Listeria monocytogenes is a gram-positive, nonspore-forming bacterium that is commonly found in soil, water, and decaying plant material *(137)*. As such, there are many potential routes for contamination of foods with this organism. One characteristic that makes *L. monocytogenes* particularly difficult to control is its ability to grow in foods at refrigeration temperatures.

Although *L. monocytogenes* has been known as a human pathogen since 1929, it was recognized as a foodborne pathogen only relatively recently *(78, 137)*. In the early 1980s, several large outbreaks of listeriosis were traced to consumption of contaminated foods. In 1981, 41 persons in Canada became ill after eating cole slaw containing *L. monocytogenes,* and 18 of these people died *(139)*. Then in 1983, 14 people died of listeriosis, and milk (almost certainly contaminated with *L. monocytogenes* after pasteurization) was epidemiologically linked to the outbreak (although the organism was never isolated from the milk) *(36, 55)*. In 1985 listeriosis (142 cases with 48 deaths) associated with consumption of a Mexican-style cheese occurred in California *(92)*. These outbreaks, with their high mortality rates, drew the attention of microbiologists, the government, and the food industry and stimulated research on the detection, heat resistance, and survival of *L. monocytogenes* in foods. During the late 1990s, outbreaks and recalls of meat products due to the presence of *L. monocytogenes (27, 28)* heightened awareness of this pathogen, fostering a renewed emphasis on control measures.

Thermal processes destroy foodborne pathogens and are one of the primary techniques used to ensure the safety of foods. Therefore, the survival and heat resistance of *L. monocytogenes* under different conditions in many foods have been investigated. However, the data accumulated over the past 20 to 25 years need to be evaluated carefully, because methods have evolved with our changing knowledge of this organism. Recently, the effectiveness of irradiation and high hydrostatic pressure for destruction of foodborne pathogens has been assessed and processes involving combinations of these treatments with heat can produce an additional margin of safety *(61, 65, 84, 115, 122, 134, 142, 150)*. Bacteriocins and some preservatives also have been used in combination with thermal processing to destroy listeriae *(15, 84, 91, 108, 114, 132, 153, 154, 158)*.

Heat resistance of *L. monocytogenes* is influenced by many factors such as strain variation, previous growth conditions, exposure to heat shock, acid, and other stresses, and composition of the heating menstruum. Several researchers have devised models to predict the effects of variations of some of these factors on thermotolerance *(13, 31, 82, 94, 112, 157)*. In addition, the number of surviving cells detected depends on the recovery medium and incubation conditions used. This review summarizes published data on the heat resistance of *L. monocytogenes*; the first three tables include heat resistance data on cells tested in laboratory media under various conditions, while the other 11

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TABLE 1. *Thermal resistance of* L. monocytogenes *in laboratory culture media*

Strain	Medium	*D*-values (min) at temperature (°C) of: 52	54	55	56	58	60	62	64	66	*z*-value (°C)
Scott A[a]	TPB	38.8	16.8		7.4						5.6[h]
LCDC 81-861[a]	TPB	49.1	14.4		10.4						6.0[h]
DA-3[a]	TPB	63.8	22.8		5.7						3.85[h]
Brie-1[a]	TPB	64.1	31.6		16.0						6.6[h]
Scott A[b]	PB				3.0		0.5			0.1	5.9
F5069[b]	PB				4.1		0.7			0.1	6.6
SLU 10[c]	PS					2.2	1.2	0.42			5.6
SLU 110[c]	PS					1.9	0.85	0.46			6.6
SLU 136[c]	PS					1.3	0.81	0.1			6.5
SLU 594[d]	PS					1.7	0.75	0.3			5.3
ATCC 19111[d]	PS					1.8	0.72	0.35			5.6
NCTC 5105[d]	PS					1.9	1.1	0.38			5.7
SLU 92[d]	PS						1.9	1.1	0.5		6.9
SLU 196[d]	PS						3.1	1.3	0.68		6.1
NCTC 7973[e]	PB-B			4.2							
NCTC 7973[e]	PB-C			6.3							
Lm Scott A[f]	TSB	9.9			2.21		0.38		0.15		7.27
Lm 1151[f]	TSB	20.1			3.8		0.88		0.23		7.05
Lm 5S[g]	TSB						4.1			0.4	6.0

[a] Golden et al. *(64)*. Test medium was tryptose phosphate broth (TPB). Survivors were counted on tryptose phosphate agar (TPA) after growth for 3 days at 30°C.

[b] Foegeding and Stanley *(58)*. Test medium was phosphate buffer (PB). Survivors were counted on TPA after growth for 5 days at 35°C.

[c] Sörqvist *(147)*. Test medium was physiological saline (PS). Survivors were counted on blood agar after 5 days at 4°C followed by 7 days at 37°C.

[d] Sörqvist *(148)*. Test medium was PS. Survivors were counted on blood agar after 5 days at 4°C followed by 7 days at 37°C.

[e] Patchett et al. *(127)*. Test medium was phosphate buffer (PB). Cells had been grown in batch (B) or chemostat (C) cultures. Survivors were counted on coryneform agar after growth for 2 days at 30°C.

[f] Casadei et al. *(26)*. Test medium was trypticase soy broth (TSB). Survivors were counted on TSA after growth for 6 days at 30°C.

[g] Quintavilla and Campanini *(133)*. Survivors were counted on TSA with pyruvate after growth for 3 days at 37°C.

[h] Calculated from data in table.

tables summarize data on the heat resistance of *Listeria* spp. in various foods. While not to be construed as process recommendations, these findings may be useful for food processors in determining appropriate times and temperatures for destroying this pathogen.

FACTORS AFFECTING HEAT RESISTANCE

Strain variation. Heat resistance data for different *L. monocytogenes* strains listed in Table 1 demonstrate significant variability. However, results reported from different laboratories cannot usually be directly compared because test media and procedures used for enumerating surviving cells often differ. Under similar experimental conditions, some strains of *L. monocytogenes* are 2.5 to 3 times more heat resistant than other strains when tested in laboratory culture media (Table 1). Golden et al. *(64)* reported that $D_{56°C}$-values in tryptose phosphate broth for *L. monocytogenes* strains Brie-1 (from Brie cheese), LCDC 81-861 (from cabbage implicated in a Canadian outbreak), Scott A (a human isolate from a 1983 milk outbreak), and DA-3 (another human isolate) were 16.0, 10.4, 7.4, and 5.7 min, respectively. In another set of experiments, $D_{57°C}$-values ranged from 6.5 to 26 min for 29 *L. monocytogenes* strains in tryptone soy broth *(101)*.

Data on heat resistance of *L. monocytogenes* in foods (discussed later) also indicate variation among strains. *D*-values reported for two or more strains under similar experimental conditions are tabulated (tables shown later in the text) for: (i) ground chicken *(59)* and pork *(88)*; (ii) sausage *(7)*; (iii) milk *(12, 37, 38, 43, 136)*; (iv) liquid egg yolks *(3, 124)*; (v) salmon and cod *(4);* and (vi) cabbage *(6)* and carrots *(59)*.

Because not all strains have been tested under comparable conditions, it is not possible to conclude that one particular strain is the most heat resistant. Strain Scott A that has been widely used in research appears to be intermediate in its thermal resistance. Data on *Listeria innocua* indicate that it is more heat resistant than some strains of *L. monocytogenes* when the two organisms are tested under identical conditions and may be a suitable model for estimating the thermal tolerance of *L. monocytogenes (45, 58, 85, 130, 131)*. For example, Foegeding and Stanley (58) found that two strains of *L. innocua* had $D_{56°C}$-values of 6.3 min, compared with a $D_{56°C}$-value of 3.0 min for *L. monocytogenes* Scott A and 4.1 min for strain F5069.

Age of microorganisms. Cells in the stationary phase of growth appear to be the most resistant to thermal stress.

TABLE 2. *Effects of heat shock and growth temperature on the thermal resistance of* L. monocytogenes *in culture media*

Strain	Medium	Heat shock/growth condition	*D*-values (min) at temperature (°C) of:						*z*-value (°C)
			50	52	55	57.8	60	65	
F5069[a]	TSYE	No heat shock		37.9		9.4			
F5069[a]	TSYE	48°C, 30 min		49.8		10.3			
F5069[a]	TSYE	48°C, 15 min				11.7			
Scott A[b]	TSYE	No heat shock	49.4		8.81		1.58	0.28	6.6
Scott A[b]	TSYE	48°C, 10 min	96.9		20.0		3.32	0.54	6.7
Scott A[c]	TSYE	No heat shock, aerobic			8.89				
Scott A[c]	TSYE	No heat shock, anaerobic			12.0				
Scott A[c]	TSYE	48°C, 10 min, aerobic			18.7				
Scott A[c]	TSYE	48°C, 10 min, anaerobic			26.4				
13-249[d]	TPB	No heat shock, log					0.6		
13-249[d]	TPB	No heat shock, stationary					2.22		
13-249[d]	TPB	46°C, 30 min, log					1.7		
13-249[d]	TPB	46°C, 30 min, stationary					4.01		
NCTC 7973[e]	PB	Growth at 10°C; batch			3.1				
NCTC 7973[e]	PB	Growth at 10°C; chemostat			3.9				
NCTC 7973[e]	PB	Growth at 30°C; batch			4.2				
NCTC 7973[e]	PB	Growth at 30°C; chemostat			6.3				
Scott A[f]	BHI-A	Growth at 10°C					0.83		
Scott A[f]	BHI-A	Growth at 37°C					1.11		
Scott A[f]	BHI-L	Growth at 10°C					0.95		
Scott A[f]	BHI-L	Growth at 37°C					1.28		

[a] Bunning et al. *(18).* Survivors were counted on TSYE (trypticase soy with yeast extract) agar after growth for 7 days at 25°C.
[b] Linton et al. *(95).* Log phase cells tested in TSYE broth. Survivors were counted on TSYE agar after growth for 2 days at 37°C.
[c] Linton et al. *(96).* Log phase cells tested in TSYE broth. Survivors were counted on TSYE agar after aerobic or anaerobic growth for 2 days at 37°C.
[d] Jørgensen et al. *(79).* Strain 13-249 was grown to log or stationary phase, heat shocked (46°C, 30 min) or not, and inoculated into TPB pH 7.0. Survivors were counted on TSYE agar with pyruvate after growth for 5 days at 30°C.
[e] Patchett et al. *(127).* Test medium was PB. Cells were grown in batch or chemostat cultures. Survivors were counted on coryneform agar after growth for 2 days at 30°C.
[f] Juneja et al. *(83).* Test medium was BHI (brain heart infusion) adjusted to pH 7 with acetic (A) or lactic (L) acids. Survivors were counted on TSYE agar with pyruvate after growth for 4 days at 30°C.

$D_{60°C}$-values measured for stationary-phase cells of *L. monocytogenes* strain 13-249 were nearly four times higher than those for log-phase cells (2.22 min compared to 0.6 min) (Table 2) *(79).* In other experiments, $D_{56°C}$-values for strain Scott A in stationary phase were about eight times higher than for log-phase cells (8.6 min compared to 1.0 min) (Table 3) *(97).*

Growth conditions. Temperature and the composition of the growth medium, whether a food or laboratory culture broth, affect rates of growth and the synthesis of cellular constituents that determine the thermal tolerance of bacterial cells.

Some components present in foods and culture media can protect bacterial cells from thermal damage by stabilizing membranes or other cellular structures or by stimulating the production of stress-related proteins *(137).* The osmotic environment in which cells are grown can be a major determinant of their heat resistance. *L. monocytogenes* cells grown in 1.5 M NaCl have a greatly elongated shape and increased heat resistance compared to those grown at a normal NaCl level of 0.09 M. Growth in tryptic phosphate broth containing 0.09, 0.5, 1.0, or 1.5 M NaCl followed by testing in media with the same salt concentration resulted in $D_{60°C}$-values of 1.6, 2.5, 7.4, and 38.1 min, respectively (Table 3) *(81).* Similar results were observed in ground beef with added salt (Table 4).

Cells of strain Scott A grown in a fat-rich medium (butter and cream) were four to eight times as resistant to

TABLE 3. *Effects of sugar, salt, and other stresses on the thermal resistance of* L. monocytogenes *Scott A in culture media*

Stress	Conditions		*D*-values (min) at temperature (°C) of:				
			56	60	62.8	65.6	68.3
Sucrose[a], water activity	0.98			2.0	0.74	0.36	0.15
	0.96			2.9	0.97	0.52	0.30
	0.94			5.6	3.0	1.1	0.47
	0.92			7.6	5.3	3.1	1.6
	0.90			8.4	5.9	3.8	1.9
NaCl[b]	Growth	Heating					
	0.09 M	0.09 M		0.4			
	0.09 M	0.5 M		0.5			
	0.09 M	1.0 M		1.15			
	0.09 M	1.5 M		3.3			
	0.5 M	0.5 M		0.63			
	1.0 M	1.0 M		1.85			
	1.5 M	1.5 M		9.53			
	1.5 M	0.09 M		0.95			
Acid; pH[c]	Acetic acid; pH 7.0			1.11			
	Acetic acid; pH 5.4			1.22			
	Lactic acid; pH 7.0			1.28			
	Lactic acid; pH 5.4			1.14			
Other stresses[d]	Control; log phase		1.0				
	Stationary phase		8.6				
	Starvation 138–156 h; log phase		13.6				
	4–8% ethanol; log phase		4.1				
	HCl to pH 4.5; log phase		8.8				
	500 ppm H_2O_2; log phase		2.9				

[a] Sumner et al. *(152).* Cells were tested in phosphate buffer with sucrose added to produce solutions with the indicated water activities. Survivors were estimated after incubation in tryptose broth with pyruvate for up to 2 weeks.

[b] Jørgensen et al. *(81).* Cells were grown in media containing 0.09, 0.5, 1.0, or 1.5 M NaCl and then inoculated into test media containing 0.09, 0.5, 1.0, or 1.5 M salt. Survivors were counted on tryptone soy agar after growth for 5 days at 30°C.

[c] Juneja et al. *(82).* Test medium was brain heart infusion broth with lactic or acetic acids added to achieve a pH of 7.0 or 5.4. Survivors were counted on TSYE agar with pyruvate after growth for 4 days at 30°C.

[d] Lou and Yousef *(97).* Cells were tested in TSBYE under the conditions indicated. Control cells were unstressed and in early log phase of growth. Survivors were counted on TSYE agar after growth for 3 days at 35°C.

heat at 60°C as cells grown in trypticase soy broth (Tables 1 and 9) *(26).* However, starvation (growth in a minimal medium for some time prior to testing) also significantly enhanced thermal resistance, as did the addition of HCl, ethanol, or H_2O_2 to growth media (Table 3) *(97, 102).* It appears that many stressful conditions encountered during growth enhance resistance to other stresses, including heat.

The temperature during growth affects lipid biosynthesis, composition of membranes, and protein synthesis, and thereby influences the ability of *L. monocytogenes* to withstand thermal inactivation *(83).* Growth temperature also affects the pH at which maximum heat resistance occurs *(123).* Several experiments provide evidence that cells grown at higher temperatures are more heat resistant than those grown at lower temperatures *(121, 145, 146).* When *L. monocytogenes* was grown in liver sausage slurry, cells grown at 19°C had a $D_{60°C}$-value of 0.8 min, while those grown at 37°C had a $D_{60°C}$-value of 1.6 min *(7).* Other data in Table 2 demonstrate that *L. monocytogenes* strains grown at 10°C were more heat sensitive than those grown at 30°C *(127)* or at 37°C *(83).* Therefore, mild temperature abuse of refrigerated foods could increase thermal resistance of any resident *L. monocytogenes.*

Test conditions. Results from numerous studies indicate that *L. monocytogenes* is more resistant to heat when tested in foods than when it is suspended in laboratory media *(8, 26, 58, 79).* Heat resistance varies depending on the food. For example, *L. monocytogenes* was found to be significantly more resistant to heat when mixed into a beef homogenate than in chicken or carrot homogenates *(59).*

During pasteurization of foods and media containing nisin, a bacteriocin active against *L. monocytogenes,* heat acted synergistically with nisin to inactivate nisin-resistant strains *(113).* The effect of nisin in different foods is discussed later.

The rate at which cells are heated during testing influences their survival. When cells of *L. monocytogenes* in pork were heated slowly (1.3°C/min), they exhibited a greater heat resistance than when heated rapidly (2.2 to 8.0°C/min) (Table 4) *(88).* Similar results were obtained when cells were heated with careful monitoring in tryptic

TABLE 4. *Thermal resistance of* L. monocytogenes *in ground meat and poultry*

Meat	Conditions	*D*-values (min) at temperature (°C) of:										*z*-value (°C)
		50	51.7	54.4	55	57.2	60	62	62.8	64/65	70	
Beef[a] <7% fat	Cooked, Scott A						8.32	4.2		2.19	0.2	5.98
Beef[a] <7% fat	Cooked, NCTC 11994						6.27	2.9		2.21	0.14	5.98
Beef[b]	Raw	85			21		3.8			0.93	0.14	7.2
Beef[c] 1.25% fat	Meat slurry						2.54			0.75	0.23	
Beef[d] 20% fat	Inoculated			22.4		15.7	4.47		2.56			7.9
Beef[d] 20% fat	Contaminated			12.5		3.41	1.62		0.73			7.9
Beef[e] 30.5% fat	High fat		71.1			5.8			1.2			6.3
Beef[e] 2% fat	Low fat		81.3			2.6			0.6			5.17
Beef[f] 2.35% fat	Vacutainers	36.1			3.53		0.33					4.9[l]
Beef[f] 2.35% fat	Vacuum bags	43.5			3.14		0.24					4.4[l]
Beef[g] 3.1% fat	pH 7.2, 0% lactate				2.94							
Beef[g] 3.1% fat	pH 7.2, 2.4% lactate				2.39							
Beef[g] 3.1% fat	pH 7.2, 4.8% lactate				2.17							
Beef[g] 3.1% fat	pH 5.7, 0% lactate				3.93							
Beef[g] 3.1% fat	pH 7.2, 2.4% lactate				2.5							
Beef[g] 3.1% fat	pH 7.2, 4.8% lactate				2.05							
Beef[h], 0 M NaCl	Growth 0.09 M NaCl						1.15					
Beef[h], 0 M NaCl	Growth 1.5 M NaCl						1.95					
Beef[h], 1.5 M NaCl	Growth 0.09 M NaCl						3.98					
Beef[h], 1.5 M NaCl	Growth 1.5 M NaCl						10.8					
Beef[i] pH 5.4	Log, no heat shock						3.07					
Beef[i] pH 5.4	Log, heat shocked						3.83					
Beef[i] pH 5.4	Stationary no heat shock						8.65					
Beef[i] pH 5.4	Stationary heat shocked						7.88					
Beef[i] pH 6.2	Log, no heat shock						4.03					
Beef[i] pH 6.2	Log, heat shocked						9.51					
Beef[i] pH 6.2	Stationary no heat shock						12.53					
Beef pH 6.2[i]	Stationary heat shocked						12.23					
Beef, pH 5.64[j]	Heating: >10°C/min						6.7			1.4		6.9
Beef, pH 5.64[j]	Heating: 0.6°C/min						6.4			1.6		6.5
Beef, pH 5.64[j]	Heating: 0.3°C/min						7.1			1.7		6.6
Beef, pH 6.0[k]	C: heat only						1.5			0.88	0.47	19.8[l]
Beef, pH 6.0[k]	P: heat only						1.63			0.56	0.37	15.6[l]
Beef, pH 6.0[k]	C: heat + irradiation						0.77			0.26	0.13	13.0[l]
Beef, pH 6.0[k]	P: heat + irradiation						0.73			0.28	0.09	11.0[l]
Beef, pH 6.0[k]	C: heat, irradiation, storage						0.96			0.35	0.19	14.2[l]
Beef, pH 6.0[k]	P: heat, irradiation, storage						0.96			0.36	0.22	15.6[l]
Chicken[a]	Cooked, Scott A						5.29	2.51		1.56	0.16	6.72
Chicken[a]	Cooked, NCTC 11994						5.02	2.21		1.84	0.2	7.39
Chicken leg[b]	Raw	179			14		5.6			0.53	0.11	6.7
Chicken breast[b]	Raw	100			13		8.7			0.52	0.13	6.3
Chicken breast[p]	Raw										0.133	
Pork[m]	Fresh: Scott A							6.5				
Pork[m]	Fresh; ATCC 19111							7.7				
Pork[m]	Old; Scott A							4.3				
Pork[m]	Old; ATCC 19111							5.2				
Pork[m]	Heating 1.3°C/min							9.2				
Pork[m]	Heating 2.2°C/min							6.2				
Pork[m]	Heating 8.0°C/min							5.5				
Pork[n]	Alone	109			9.8		1.14					5.05
Pork[n]	With soy hulls	114			10.2		1.7					5.45
Pork[o]	With pressure	0.37										
		0.63										

[a] Gaze et al. *(59)*. Survivors were counted after growth on TSAYE for 2 days at 30°C.

[b] Mackey et al. *(101)*. Bacteria were *L. monocytogenes* strain 5, isolated from prepacked chicken. Survivors were counted after growth on tryptose agar for 2 days at 37°C.

[c] Boyle et al. *(8)*. Bacteria were Strain Scott A. Survivors were counted on CN agar with pyruvate and horse blood.

[d] Schoeni et al. *(140)*. Bacteria were a five-strain mixture (inoculated) or Scott A (naturally contaminated—meat from an experimentally infected cow). Survivors were counted after growth on tryptose agar for 4 days at 30°C.

TABLE 4. *(Continued)*

[e] Fain et al. *(44).* Strain used was Scott A. Survivors were counted after growth on TPA with dextrose and iron and a sheeps' blood overlay for 2 days at 35°C.
[f] Doherty et al. *(35).* Strain NCTC 11994 was mixed with beef and heated in vacutainers or vacuum bags. Survivors were counted after growth on Palcam agar with polymixin B, acriflavin, and ceftazidine for 2 days at 30°C.
[g] McMahon et al. *(108).* Strain NCTC 11994 was mixed with beef and pH adjusted beef containing added lactate and heated in vacutainers. Survivors were counted after growth on Palcam agar with polymixin B, acriflavin, and ceftazidine for 3 days at 30°C.
[h] Jørgensen et al. *(81).* Strain Scott A was grown in media containing 0.09 or 1.5 M NaCl and then inoculated into beef containing 1.5 M or no added salt. Survivors were counted after growth on TSA for 5 days at 30°C.
[i] Jørgensen et al. *(79).* Strain 13-249 (isolated from ham) was grown to log or stationary phase, heat shocked (46°C, 30 min) or not, and inoculated into beef at pH 5.4 or 6.2. Survivors were counted after growth on TSAYE with pyruvate for 5 days at 30°C.
[j] Hansen and Knøchel *(66).* A serotype 1 strain was mixed with minced beef and heated at various rates. Survivors were counted after growth on TSAYE with pyruvate for 5 days at 30°C.
[k] Grant and Patterson *(65).* Two strains, C (CRA 433) and P [P(10)4], in cooked beef were exposed to heat alone, to heat after irradiation at 0.8 kGy, and to heat after irradiation at 0.8 kGy and storage at 2 to 3°C for 14 days. Survivors were counted after growth on TPA with pyruvate for 3 days at 30°C.
[l] Calculated from data in table.
[m] Kim et al. *(88).* Bacteria were inoculated into raw pork obtained within 3 days of slaughter (fresh) or after 3 months storage in a freezer (old). ATCC 19111 was also tested after heating at different rates. Survivors were counted after growth on TSAYE for 2 days at 37°C.
[n] Ollinger-Snyder et al. *(120).* Survivors were counted after growth on McBride *Listeria* agar for 2 days at 35°C.
[o] Murano et al. *(115).* Three strains exposed to hydrostatic pressure (414 MPa) while heating. Survivors were counted after growth on TSAYE for 2 days at 35°C.
[p] Murphy et al. *(118).* Survivors were counted after growth on TSAYE with rifampicin and streptomycin for 4 to 6 days at 37°C.

phosphate broth. At heating rates of ≤5°C/min, inactivation was less than the predicted kill at final temperatures of 50 to 64°C *(149).* Slow heating processes appear to allow some adjustment by the cells so that they exhibit a greater resistance to killing temperatures, possibly through the production of heat shock proteins. This may be a concern in foods cooked slowly, particularly if pieces of food are sufficiently large to impede heat transfer.

During testing, *L. monocytogenes* cells are exposed to high temperatures for specific times, and then samples containing listeriae are added to some type of growth medium and incubated under certain conditions to enumerate surviving cells to determine the effectiveness of the heat treatment. Because differences in recovery conditions affect the number of cells counted (and therefore, the *D*-values calculated), they are briefly described in the footnotes to the tables. One should take these different conditions into account when interpreting reported data on heat resistance.

Various selective and nonselective media and incubation conditions have been used for recovery. Initially a nonselective cold-enrichment procedure was used to select for *Listeria* spp. while suppressing competitive microflora unable to grow at lower temperatures *(6).* Some researchers also reported that cold enrichment enhanced recovery of heat-damaged cells *(30, 104, 148),* while others reported that higher temperatures increased recovery from milk *(110),* as well as growth on blood agar and trypticase soy agar (TSA) *(100).*

Some selective media, containing compounds that inhibit non-*Listeria* spp., were developed for enumerating *L. monocytogenes* survivors at higher incubation temperatures. However, many selective media, including some that are commonly used, are not satisfactory for recovery of heat-injured cells *(128, 143).* In one comparison study, media containing phenylethanol, acriflavin, potassium tellurite, polymyxin B sulfate, or 5% NaCl were detrimental to the recovery of injured cells *(143).* Other researchers compared selective and nonselective media following heat treatment and found that *Listeria* counts were higher on the nonselective medium *(34, 44, 63, 71, 95, 96, 136).* One approach that combines the advantages of selective and nonselective media involves culturing heat-treated listeriae on nonselective agar for several hours to allow repair, followed by an overlay with a selective medium *(2, 21, 156).*

Other incubation conditions can also influence recovery. The time needed to repair injured cells varies with incubation temperature and different media *(21, 100, 106, 110).* Sensitivity of heat-injured cells to oxygen is another factor influencing recovery of survivors; some researchers use an anaerobic incubation system or oxygen scavengers to encourage growth and repair of injured cells *(60, 74, 89, 90, 109, 129, 151).*

If injured *L. monocytogenes* are not counted, the lethality of a thermal process may be overestimated. Moreover, because heat-injured cells may have the opportunity to repair in rich media or foods, particularly during cold storage, they remain a potential public health threat. Hoffmans et al. *(74)* have recently reviewed methods to recover heat-injured *L. monocytogenes* and discuss possible repair of injured cells and subsequent growth in foods.

Environmental stresses. Heat shock, the short-term exposure of cells to temperatures above those for optimum growth, results in increased heat resistance *(1, 18, 19, 22, 47, 48, 53, 79, 90, 95, 96).* Data in Tables 2, 4, 5, and 6 indicate that the degree of enhanced thermal resistance is strain dependent and also varies with the length of the heat shock, the pH of the medium, and the growth phase of the cells. This increased heat tolerance may persist for several hours or longer during refrigerated storage *(48, 80).* Heat

TABLE 5. *Thermal resistance of* L. monocytogenes *in cured meats*

Meat	Conditions	*D*-values (min) at temperature (°C) of: 51.7	54.4/55	57.2	60	62.8/63	64	66	77	88	*z*-value (°C)
Sausage[a]	23% beef; 77% pork	44.4	20.1	11.2	9.13						10.0
Sausage[b]	Pork only				7.3	3.0		1.0			6.8
Sausage[c]	Pork liver; Scott A			8.91	2.42	1.12					6.2
Sausage[c]	Pork liver; V7				1.0						
Sausage[c]	Pork liver; HO-VJ-S				1.6						
Sausage[d]	Summer sausage							2.08	0.84	0.37	29.3[e]
Sausage[f]	No heat shock						3.3				
Sausage[f]	Heat shock, 30 min						4.2				
Sausage[f]	Heat shock, 60 min						4.7				
Sausage[f]	Heat shock, 120 min						8.0				
Ham[g]	No heat shock		17.8		1.82						5.05
Ham[g]	Heat shocked		19.2		3.48						6.74

[a] Schoeni et al. *(140).* Bacterial inoculum was a five-strain mixture. Survivors were counted on tryptose agar after growth for 4 days at 30°C.

[b] Quintavilla and Campanini *(133).* Survivors were counted by the most probable number method after incubation for 7 days in *Listeria* enrichment broth at 28°C.

[c] Bhaduri et al. *(7).* Survivors were counted on tryptose agar after growth for 3 days at 37°C.

[d] Roering et al. *(135).* A three-strain mixture was used: 101M, F6854, and CLIP23485. Survivors were counted on BHI agar after growth for 1 day at 37°C.

[e] Calculated from data in table.

[f] Farber and Brown *(48).* A 10-strain mixture was tested in sausage (33% beef, 66% pork) with and without previous heat shock at 48°C. Survivors were counted on tryptose agar after growth for 7 days at 25 to 30°C.

[g] Carlier et al. *(22).* A French outbreak strain was used. Cells were heat shocked at 42°C for 60 min. Survivors were counted on Palcam agar after growth for 3 days at 37°C.

shock effects can be a significant factor in bulk foods heated slowly, in food receiving marginal heat treatment, or when ingredients are preheated before the final mix is processed.

Increased heat tolerance can be induced by short-term exposure (shock) to high salt or solute levels. When grown in 0.09 mol/liter NaCl and then exposed to test media lontaining 0.5, 1.0, or 1.5 mol/liter NaCl, *L. monocytogenes* cells exhibited a 1.3- to 8-fold increase in heat resistance (Table 3) *(81).* Similar results were observed with minced beef (Table 4). A model has been developed to predict the survival of *L. monocytogenes* at 50 to 60°C in culture media containing 0 to 4% NaCl *(93).* Downshock (growth in 1.5% NaCl followed by heating in 0.09% NaCl) significantly reduced the increased resistance usually observed when cells were heated at the high salt concentration. Juneja and Eblen *(82)* developed a model for the thermal resistance of *L. monocytogenes* as affected by pH, NaCl, and sodium pyrophosphate in a model beef gravy (proteose peptone, beef extract, yeast extract, and soluble starch). At pH 8.0, 0.3% sodium pyrophosphate decreased the heat resistance at 55°C at both 0 and 6% NaCl. However, this decreased resistance did not occur at lower pH values, where heat resistance appeared to be affected by interactions between NaCl, pH, and phosphate levels. In most cases, salt appeared to have a protective effect, resulting in higher heat resistance, but this was not always the case.

Decreasing a_w values and increasing solute concentrations are correlated with greater heat resistance in *L. monocytogenes.* When strain Scott A was exposed to solutions of various sucrose concentrations, the $D_{65.6°C}$-values increased from 0.36 to 3.8 min and the *z*-value increased from 7.6 to 12.9°C as a_w decreased from 0.98 to 0.90 (Table 3) *(152).* Other solutes, including 3 M NaCl or KCl and 1 M xylose, α-methylglucoside, mannose, glucose, galactose, lactose, sucrose, glycerol, or mannitol, significantly reduced thermal injury of *L. monocytogenes* in phosphate buffer held at 52°C for 1 h *(144).* However, some other solutes (ammonium chloride and fructose) did not protect the cells but rather increased cell death at 52°C.

The protective effect of solutes may have practical implications in foods. The addition of salt, glucose, and curing salts to meat reportedly increases the thermal resistance of *L. monocytogenes (140, 158).* When added to liquid whole eggs (Table 10) and egg yolks (Table 11), sucrose and NaCl significantly increased *D*-values. For example, *L monocytogenes* heated in egg yolks with no added solutes had a $D_{64.4°C}$-value of 0.44 min, while cells heated in yolks with 10% sucrose or 10 or 20% NaCl had $D_{64.4°C}$-values of 0.97, 6.11, and 27.3 min, respectively *(124).*

Acid shock (short-term exposure of cells to media at pH 4.0) slightly increased $D_{58°C}$-values for some strains of *L. monocytogenes* (Table 7) *(51).* However, *L. monocytogenes* was more heat sensitive when heated in more acidic cabbage juice (pH 4.6 versus 5.6) (Table 14) *(6).* Juneja and Eblen *(82)* found that the heat resistance of *L. monocytogenes* was reduced at pH 4.0 compared to pH 8.0. The $D_{55°C}$-value was 5.35 min at pH 4.0 with no added NaCl or sodium pyrophosphate compared to a $D_{55°C}$-value of 25.55 min at pH 8.0 under the same conditions. When 6%

TABLE 6. *Thermal resistance of* L. monocytogenes *in whole milk*

Strain	Conditions	*D*-values (min) at temperature (°C) of: 55/55.2	56	57.8/58	60	62.7/62.8	63/63.3	66.1	68.9	71.7	*z*-value (°C)
Scott A[a]	Homogenized				2.1						
Scott A[b]	Raw	8.2		2.3							
Scott A[c]	Raw			4.83			0.33	0.12	0.05	0.015	6.2
Scott A[d]	Raw			5.5			0.52		0.067		6.3
Scott A[c]	Sterile			4.26			0.58	0.165	0.053	0.033	6.5
				4.97				0.23	0.097	0.045	6.6
Scott A[d]	Sterile			4.8			0.84		0.12		7.0
BS-9[d]	Raw			7.26			0.65		0.055		5.8
BS-9[d]	Sterile			6.8			1.13		0.152		6.7
SE-31[d]	Raw			8.81			0.77		0.047		5.3
SE-31[d]	Sterile			7.34			0.83		0.103		6.8
V7 and F6861[e]	Sterile			2.2–2.75							
ATCC 19111[f]	Sterile					0.65					
ATCC 19113[f]	Sterile					0.4					
ATCC 19115[f]	Sterile					0.4					
F5027[f]	Sterile					0.35					
F5069[f]	Sterile	24				1.0				0.05	4.3
F5069[g]	Sterile					0.6					
F5069[h]	Sterile									0.05	
F5069[i]	Sterile			5.52		0.64		0.28–0.3	0.085–0.14		7.3
F5069[i]	Sterile; intracellular			7.16		0.92		0.28–0.3	0.065–0.15		5.6
NCTC 98635S[j]	Sterile		9.6		1.5		0.4				5.0
NCTC 98635R[j]	Sterile		10.5		1.7		0.4				5.0
NCTC 11994S[j]	Sterile		10.1		1.5		0.4				5.0
NCTC 11994R[j]	Sterile		11.4		1.8		0.5				5.1

[a] Holsinger et al. *(75).* Survivors were counted on tryptose agar after growth for 2 days at 37°C.
[b] Kamau et al. *(86).* Survivors were counted on TSAYE after growth for 2 days at 35°C in the presence of 9.5% CO_2.
[c] Bradshaw et al. *(10, 11).* Survivors were counted on TSAYE after growth for 2 days at 37°C.
[d] Bradshaw et al. *(12).* Survivors were counted on TSAYE after growth for 7 days at 25°C.
[e] Farber et al. *(49).* Survivors were counted on tryptose agar after growth for 7 days at 30°C.
[f] Donnelly and Briggs *(37).* Survivors were counted on TPA with iron after growth for 2 days at 37°C.
[g] Knabel et al. *(90).* Survivors were counted on TSAYE after growth for 21 days at 25°C.
[h] Bunning et al. *(19).* Survivors were counted on TSAYE after growth for 7 days at 25°C.
[i] Bunning et al. *(20).* Survivors were counted on TSAYE after growth for 7 days at 25°C.
[j] Rowan and Anderson *(136).* Strains were morphologically R (rough) or S (smooth). Survivors were counted on TSAYE after growth for 2 days at 37°C.

NaCl was added, the $D_{55°C}$-value was 27.24 min at pH 8.0 but only 12.49 min at pH 4.0. When *L. monocytogenes* was grown to stationary phase in tryptic soy broth acidified with HCl, Mazzotta *(103)* found that thermal resistance decreased with decreasing pH, from a $D_{60°C}$-value of 2 min at pH 7.0 to 0.7 min at pH 5.0, 0.4 min at pH 4.0, and 0.2 min at pH 3.0. A similar decrease was seen with acid-adapted cells, although the *D*-values were higher.

HEAT RESISTANCE STUDIES IN FOODS

Meat and poultry products. *D*-values for *L. monocytogenes* in uncured meat and poultry are shown in Table 4; Table 5 contains data for *L. monocytogenes* in cured meat. These data indicate that listeriae can be eliminated by thermal processing, but the effectiveness of different heating regimens is affected by characteristics of the meat (e.g., age and fat content), heat source, rate of heating, and exposure of the bacteria to stresses such as acid, heat shock, and preservatives (Tables 4 and 5). Murphy et al. *(119)* recently determined the heat resistance of *L. innocua* strain M1 in chicken breast meat. The $D_{60°C}$-value of 5.02 min and $D_{65°C}$-value of 1.71 min, with a *z* of 6.3°C, were similar to data obtained for *L. monocytogenes* in chicken by Gaze et al. *(59),* supporting the use of this strain for verification of processes designed to kill *L. monocytogenes.*

Most experiments have been done with only one product, so it is not possible to state that listeriae survive heating in one type of meat better than in another. Results from two experiments in which listeriae were heated in chicken and in beef under similar conditions were inconsistent *(59, 101).* Data from experiments with cooked versus raw beef and chicken *(59)* suggest that listeriae may be more heat resistant in cooked meat. This may have implications for some postpackaging pasteurization processes.

Age of the meat may affect thermal destruction of listeriae: $D_{62°C}$-values for *L. monocytogenes* cells inoculated into fresh pork were about 1.5 times greater than those for cells inoculated into 3-month old ground pork (Table 4)

TABLE 7. *Effects of heat shock, growth temperatures, and lactoperoxidase on thermal resistance of* L. monocytogenes *in whole milk*

Strain	Conditions	*D*-values (min) at temperature (°C) of: 52.2	55.2	56	57.8/58	60	62.8/63	71.7	*z*-value (°C)
F5069[a]	Growth 37°C, aerobic						0.6		
F5069[a]	Growth 37°C, anaerobic						2.5[b]		
F5069[a]	Growth 43°C, aerobic						1.53[b]		
F5069[a]	Growth 43°C, anaerobic						4.05		
NCTC 98635S[c]	Growth 37°C			9.6		1.5	0.4		5.0
NCTC 98635S[c]	Growth 42.8°C			24.5		3.7	1.1		5.1
NCTC 98635R[c]	Growth 37°C			10.5		1.7	0.4		5.0
NCTC 98635R[c]	Growth 42.8°C			29.1		4.4	1.4		5.2
NCTC 11994S[c]	Growth 37°C			10.1		1.5	0.4		5.0
NCTC 11994S[c]	Growth 42.8°C			26.5		3.9	1.2		5.1
NCTC 11994R[c]	Growth 37°C			11.4		1.8	0.5		5.1
NCTC 11994R[c]	Growth 42.8°C			31.5		4.8	1.4		5.2
F5069[d]	No heat shock							0.05	
F5069[d]	Heat shock: 48°C, 15 min							0.077	
V7 and F6861[e]	No acid shock				2.45				
V7 and F6861[e]	Acid shock—24 hr				3.0				
V7 and F6861[e]	No acid shock				2.17				
V7 and F6861[e]	Acid shock—4 hr				2.66				
V7 and F6861[e]	No acid shock				2.75				
V7 and F6861[e]	Acid shock—instantly				3.90				
Scott A[f]	No additions	30.2	8.2		2.3				
Scott A[f]	With H_2O_2	29.4	11.1		2.6				
Scott A[f]	With lactoperoxidase	10.7	1.6		0.5				

[a] Knabel et al. *(90).* Cells were grown at 37°C or 43°C prior to testing. Survivors were counted on TSAYE after aerobic or anaerobic growth for 21 days at 25°C.

[b] *D*-values were estimated from graph.

[c] Rowan and Anderson *(136).* Strains were morphologically R (rough) or S (smooth) and were grown at 37 or 42.8°C prior to testing. Survivors were counted on TSAYE after growth for 2 days at 37°C.

[d] Bunning et al. *(19).* Survivors were counted on TSAYE after growth for 7 days at 25°C.

[e] Farber et al. *(49).* Survivors were counted on tryptose agar after growth for 7 days at 30°C.

[f] Kamau et al. *(86).* Survivors were counted on TSAYE after growth for 2 days at 35°C in the presence of 9.5% CO_2.

(88). The reason for this is unknown but may involve the formation of lipid oxides in meat during storage and the toxicity of these oxides to *Listeria* cells.

High fat (30.5%) ground beef was more protective of *L. monocytogenes* at 57.2 and 62.8°C than low fat (2%) beef, as indicated by higher *D*-values (Table 4) *(44).* Increasing the pH of raw ground beef from 5.6 to 5.9 to 6.2 increased $D_{60°C}$-values for *L. monocytogenes (66, 79),* while an increase to pH 7.2 reportedly decreased $D_{55°C}$-values *(108).*

Preservatives or other compounds added to meat can also affect the apparent thermal tolerance of *L. monocytogenes.* Sodium lactate caused a small decrease in thermal tolerance of listeriae in ground beef *(108)* and a slight decrease in ground pork *(159).* When lactate and monolaurin were injected into beef roasts, the thermotolerance of *L. monocytogenes* injected into the roasts was reduced *(154).* Soy hulls added to ground pork caused a small increase in thermal resistance of *L. monocytogenes* that could add 2 min to the recommended cooking time for this mixed soy-pork product *(120).*

Slow heating (1.3°C/min) of inoculated ground pork samples allowed survival of more *L. monocytogenes* than rapid heating (8.0°C/min) *(88).* More survivors were also detected in pork that was heated aerobically rather than anaerobically. This increase in thermal resistance was not observed when ground beef was heated slowly. The low pH of the beef (5.6) also may prevent the induction of thermotolerance during slow heating *(66).*

Heat shock increases thermotolerance of listeriae in foods. When exponential phase *L. monocytogenes* cells were mixed with minced beef and exposed to 46°C for 30 min prior to testing, $D_{60°C}$-values more than doubled in heat-shocked cells at pH 6.2 *(79).*

Osmotic up-shock enhanced thermal resistance of *L. monocytogenes* in minced beef. Transfer of listeriae (grown in 0.09 M NaCl) to minced beef containing 1.5 M NaCl increased thermotolerance at 60°C by nearly fourfold. However, osmotic down-shock (from growth at 1.5 M NaCl to testing in beef with no added NaCl) resulted in a $D_{60°C}$-value of 1.95 compared to a $D_{60°C}$-value of 10.8 for cells grown and tested in 1.5 M NaCl *(81).*

Microwave cooking of meat is convenient, but several researchers report that *L. monocytogenes* can survive cooking at recommended times or to recommended temperatures in microwave ovens *(50, 70, 99).* The problem with uneven heating in microwave ovens may be offset if recommended standing times after cooking are followed. After cooking

stuffed chickens containing 10^7 CFU listeriae/g stuffing for the recommended time, temperatures measured in stuffing varied from 52 to 78°C, and listeriae levels had decreased by less than tenfold. However, after the recommended cooking time plus the recommended standing time of 20 min, temperatures in the stuffing were 72 to 85°C, and most samples were *Listeria*-free. Some experiments demonstrated substantial reductions in numbers of listeriae when meat was maintained at 70°C for 2 min *(33)*.

Salt (sodium chloride) content of soups, stews, and mixed food dishes can also impact the effectiveness of microwave cooking. A surface-heating phenomenon was observed in beef broth containing 0.3% sodium. Surface temperatures of the broth were as much as 22°C higher than those measured at 1.27 cm below the surface. These temperature variations were not observed in milk, which has a much lower sodium content *(73)*. The effects of microwave radiation on foodborne pathogens have been reviewed elsewhere *(72)*.

Conventional cooking methods do not always destroy listeriae. When applied to the surface of chicken breasts, *L. monocytogenes* was difficult to eradicate by either dry heat *(25)* or moist heat *(24, 69)*. Cooking to internal temperatures of 65.6 and 71.1°C resulted in a 2- to 3-log kill, while cooking to temperatures above 73.8°C caused a 4- to 5.5-log kill. When inoculated internally and on the surface of vacuum-packaged beef roasts pumped with brines, *L. monocytogenes* was detected at levels of 10^3 CFU/g after one and two cooking periods to an internal temperature of 62.8°C *(154)*. After one cooking period, viable *L. monocytogenes* were isolated from surface and interior samples, whereas after a second cooking, only bacteria inoculated internally survived.

In another study, *L. monocytogenes* was spread on precooked beef loin chunks that were then vacuum packaged and heated at 85°C for 16 min. *L. monocytogenes* populations declined by 10,000-fold on the surface and 1,000,000-fold in broth from meat cooked in the package *(32)*. *L. monocytogenes* cells also survived on precooked roasts that were surface inoculated with 10 ml of a 10^9 CFU/ml suspension of *L. monocytogenes* and heated to 91 or 96°C for 3 or 5 min. Even after subsequent storage at 4 or 10°C for up to 56 days, survivors were detected in samples from each treatment *(67)*.

Other preservation methods, such as irradiation and hydrostatic pressure, may be used in combination with thermal processes to ensure the safety of meat. In one study *Listeria* populations decreased 1 log in ground pork after 109 min at 50°C *(120)*. However, this mild heat treatment along with high hydrostatic pressure (414 MPa) resulted in *D*-values of 0.37 to 0.63 min and extended the shelf life of the meat by about 3 weeks *(115)*. Irradiation of minced cook-chill roast beef at a dose of 0.8 kGy sensitized *L. monocytogenes* cells to heat, thereby reducing decimal reduction times two- to fourfold *(65)*. This heat sensitivity persisted for up to 2 weeks at refrigeration temperatures, which suggests that irradiation of cook-chill products would allow easier inactivation of *L. monocytogenes* during reheating.

Numerous researchers reported that addition of curing salts to beef and pork enhances the thermotolerance of *L. monocytogenes* by two- to eightfold *(47, 101, 140, 158, 159)*. Further experiments determined that this protective effect was primarily due to NaCl and not due to the additional fat often added to sausages or to sodium nitrite, sodium lactate, or sodium erythorbate *(101, 158, 159)*. Dextrose and a phosphate mixture also enhanced the thermal resistance of *L. monocytogenes (158),* but kappa-carrageenen, added to pork with curing salts, lessened the protective effects of the salts *(159)*. Drying of slices of marinated beef at 60°C for 10 h during production of beef jerky reduced *L. monocytogenes* populations by 6 logs *(68)*.

Mazzotta and Gombas (unpublished data) found that the $D_{62°C}$-value for an *L. monocytogenes* strain responsible for an outbreak of listeriosis from hot dogs was 1.8 min in hot dog batter, compared with 3.2 min for a composite of clinical and meat isolates. Additional data on thermal resistance of *L. monocytogenes* in various types of sausage are presented in Table 5. Such data may be used to estimate the feasibility of a postpackaging pasteurization step to ensure the safety of sausages.

Several factors influence the effectiveness of thermal processing. As noted previously, *L. monocytogenes* was more resistant during slow rather than rapid heating *(133)*. This may be of concern for meat products that are heated slowly. Also, heat shock at 48°C for 30 min *(47)* or 120 min *(48)* increased thermal resistance of *L. monocytogenes* in cured meats. For *L. monocytogenes* in ham, a heat shock of 42°C for 60 min approximately doubled *D*-values measured at 60°C *(22)*. The fate of *L. monocytogenes* in cured meats has been studied during various processing techniques. Heating of "beaker sausage" to a temperature of 62.8°C and heating of pepperoni to 51.7°C for 4 h after drying eliminated all *L. monocytogenes* present *(62)*. The process for cooking frankfurters in a smokehouse (70 min to reach an internal temperature 160°F or 71.1°C) resulted in a 3-log reduction of *L. monocytogenes (160)*. In a sausage emulsion processed in a smokehouse (without smoke) all listeriae (initial count approximately 10^9/g) were destroyed when the meat was heated to 155°F *(126)*.

Cooking ham to minimum standards, i.e., a core temperature of 58.8°C, did not completely destroy listeriae in hams containing 10^5 *L. monocytogenes*/g, although it was effective for low levels (<10 CFU/g) of contaminants *(23)*. Therefore it was recommended that hams be cooked to a minimum core temperature of 65°C.

Milk and dairy products. Thermal inactivation studies on *L. monocytogenes* have been conducted in raw, sterile, homogenized, skim, and whole milk (Tables 6 to 8). Data from experiments in which several types of milk were tested under the same conditions demonstrated that thermal resistance of listeriae was higher in raw compared to sterile milk at temperatures below 63°C *(11, 12)* and in skim compared to whole milk at temperatures below 63°C *(11)*. However, differences in *D*-values were not always consistent over a range of temperatures (Table 6).

A recent predictive model demonstrated that the sur-

TABLE 8. *Thermal resistance of* L monocytogenes *(Lm) and* L. innocua *(Li) in skim milk*

Strain	Conditions	*D*-values (min) at temperature (°C) of:									
		55	56	60	61.8/62	62.8	65/65.3	65.7/66	67.5/68	69.5/70	71.7
Lm F5027[a]	Sealed tube				0.4						
Lm 19115[a]	Sealed tube				0.1						
Lm F5069[a]	Sealed tube				0.4						
Lm F5069[c]			9.0	1.0				0.1			
Lm F5069[d]					0.64			0.13	0.058	0.023	
Lm V7[b]		4.65		1.05		0.33	0.1			0.023	0.016
		4.5		0.95							
Lm Scott A[b]		4.35		0.97							
Lm California[b]		4.02		0.95							
Li PFEI[c]			15.0	3.0				0.2			
Li M1[d]					0.64			0.16	0.077	0.022	
Li M1[e]	Batch						0.22		0.068	0.027	
Li M1[e]	Continuous						0.20		0.065	0.027	

[a] Donnelly et al. *(38).* Survivors were counted on TPA with iron and esculin after growth for 2 days at 37°C.
[b] El-Shenawy et al. *(43).* Survivors were counted on tryptose agar after growth for 3 days at 35°C.
[c] Foegeding and Stanley *(58).* Survivors were counted on TPA after growth for 5 days at 35°C.
[d] Fairchild and Foegeding *(45).* Surviving *L. monocytogenes* F5069 were counted on TPA after growth for 7 days at 35°C; surviving *L. innocua* M1 were counted on TSA containing rifampin and streptomycin (TSA+) after growth for 7 days at 35°C.
[e] Fairchild et al. *(46).* Cells were tested by the sealed tube method (batch) or after treatment in a laboratory scale pasteurizer (continuous flow). Survivors were counted on TSA+ after growth for 7 days at 35°C.

vival curve of *L. monocytogenes* at different temperatures was affected by milkfat concentrations of 0 to 5% *(29).* However, an older model did not find that milkfat was a significant factor in the thermal inactivation of *L monocytogenes (37).*

Because fat content affects thermal inactivation of *L. monocytogenes* in meat and fish, survival of listeriae in high fat dairy products during heating was also investigated. At temperatures of 66.1 and 68.9°C, *D*-values measured for strain Scott A in cream were higher than those in whole milk *(11).* A comparison of thermal inactivation of *L. monocytogenes* in half cream (13.36% fat), double cream (55.62% fat), and butter (82.7% fat) revealed that *D*-values were generally lowest in butter, although differences were not always significant (Table 9) *(26).* However, when listeriae were grown in cream or butter and then inoculated into the same product, $D_{60°C}$-values were significantly higher for cells grown in the high fat products as compared to those grown in laboratory culture media *(26).*

Various components of ice cream mix appear to confer thermal stability on *Listeria* spp. Although specific components of the mixture were not identified as protective for *L. innocua,* $D_{60°C}$-values in an ice cream mix were 2.4-fold higher than those measured in whole milk *(105).* In some experiments with variable amounts of some ice cream components, high fructose corn syrup solids and stabilizers containing guar gum and carrageenan appeared to exert a protective effect on *L. monocytogenes (75).* However, guar gum did not exert a strong protective effect on listeriae in milk during pasteurization *(131).* Pasteurization at 79.4°C for 2.6 s was reported to destroy completely 10^5 CFU/ml *L. monocytogenes* in ice cream mix *(11).*

There may be some basis for a difference in thermal inactivation between raw and sterile milk because raw milk

TABLE 9. *Thermal resistance of* L. monocytogenes *in cream and butter*

Dairy product	*D*-values (min) at temperature (°C) of:									*z*-value (°C)
	52/52.2	56	57.8	60	63.3	64	66.1	68	68.9	
Raw cream[a]	28.5		3.97		0.51		0.24		0.1	6.8
Sterile cream[a]	29.7		4.34		0.48		0.29		0.13	7.1
Half cream[b]	43.3	5.04		0.78		0.20		0.15		6.2
Half cream[c]	105.1	23.1		1.58		0.33		0.13		5.32
Double cream[b]	58.1	6.06		0.65		0.21		0.16		6.08[b]
Double cream[c]	71.7	8.54		1.01		0.34		0.13		5.83
Butter[b]	23.7	4.13		0.55		0.19		0.11		6.67
Butter[c]	44.6	6.58		1.24		0.42		0.19		6.71

[a] Bradshaw et al. *(11).* Tests with Scott A. Survivors were counted on TSAYE after growth for 2 days at 37°C.
[b] Casadei et al. *(26).* Tests with Scott A. Survivors were counted on TSA after growth for 6 days at 30°C.
[c] Casadei et al. *(26).* Tests with strain 1151. Survivors were counted on TSA after growth for 6 days at 30°C.

TABLE 10. *Thermal resistance of* L. monocytogenes *in liquid whole eggs*

Strain	Additives	*D*-values (min) at temperature (°C) of: 51	55.5/56	57	60	61	63	65/66	*z*-value (°C)
Scott A[a]	None			3.2	1.95		0.49		7.3
Scott A[b]	None			3.7	1.8		0.55		7.2
Scott A[c]	None				2.1				
Scott A[d]	None	14.3	5.3		1.3			0.11	7.0
Scott A[f]	None		3.4		1.3				8.36
HAL 957E1[c]	None				1.5				
F5069[d]	None	22.6	7.1		1.4			0.2	7.2
ATCC 19111[f]	None	21.3	6.2		1.3			0.06	5.9
NCF-U2K3[d]	None	22.0	8.1		1.6			0.11	6.4
NCF-F1KK4[d]	None	21.5	8.0		1.7			0.12	6.6
Five strains[e]	None		7.5				0.42		6.0
Scott A[f]	Nisin		2.17		1.4				
Scott A[f]	Nisin, 2 h				1.1				
HAL 957E1[c]	10% NaCl						5.5		
Scott A[c]	10% NaCl						13.7		
Scott A[f]	10% NaCl		99.2		27.8			2.33	5.47
Scott A[f]	10% NaCl, nisin						7.4		
Scott A[f]	10% NaCl, nisin, 2 hr						2.6		
Scott A[c]	10% sucrose					1.9			
HAL 957E1[c]	10% sucrose					1.2			

[a] Muriana et al. *(117).* Tested in a flow injection system. Survivors were counted after growth on TSA for 4 days at 35°C.

[b] Muriana et al. *(117).* Tested in capillary tubes. Survivors were counted after growth on TSA for 4 days at 35°C.

[c] Bartlett and Hawke *(3).* Tested in capillary tubes. Survivors were counted after growth on TSAYE for 72 h at 35°C.

[d] Foegeding and Stanley *(57).* Tested in capillary tubes. Survivors were counted after growth on TPA (Difco) with 0.5% each ferric citrate and esculin for 5 days at 35°C.

[e] Michalski et al. *(111).* Tested in capillary tubes. Plated on TSA for 72 h at 30°C, then replica plated onto Palcam agar and counted after 24 h at 30°C.

[f] Knight et al. *(91).* Tested in capillary tubes with and without 10 μg nisin/ml and 10% NaCl. In some experiments cells were preincubated with nisin for 2 h prior to heating. Survivors were counted after growth on TSAYE for 2 days at 30°C.

contains some intact bovine phagocytes that may harbor *L. monocytogenes* cells *(41, 98).* However, reports from several studies in which milk containing phagocytes with listeriae was heated revealed that the phagocytes did not protect the bacterial cells from the effects of pasteurization *(17, 20, 41, 98).* Several studies reported that *L. monocytogenes* or *L. innocua* at concentrations of 10^4 to 10^5 CFU/ml cannot survive pasteurization under recommended time-temperature conditions *(12, 43, 52, 130).* Other reports indicated that at least some listeriae could survive pasteurization when the initial population exceeds 10^5 CFU/ml *(41, 54).*

Other factors were found to affect thermal resistance of listeriae in milk. Preheating to 43 or 48°C or growth at higher temperatures (39 or 43°C) increased the thermal resistance of *L. monocytogenes* and allowed the organism to survive minimum conditions that are normally lethal (Table 7) *(19, 49, 53, 90, 136).* Acid shock of listerial cells also increased heat resistance (Table 7) *(49).*

Investigations on the survival of *L. monocytogenes* during the manufacture of nonfat dry milk *(42),* cottage cheese *(138),* and mozzarella cheese *(87)* revealed that this organism can survive these processes if the inoculum is high or processing temperatures are too low. Cooking the curd at 57.2°C for 30 min was sufficient to kill nearly all the listeriae in cottage cheese. However, a few cells were recovered by cold enrichment. Early steps in the making of mozzarella cheese allowed survival of listeriae. However, *L. monocytogenes* was later destroyed during stretching of the curd at 66°C for 5 min or at 77°C for 1 min.

Other preservative methods—both chemical and physical—can enhance the listericidal effects of heat. Heating *L. monocytogenes* to 52.2 or 55.2°C shortly after activation of the lactoperoxidase system in milk decreased *D*-values from 30.2 to 10.7 min and from 8.2 to 1.6 min, respectively *(86).* High pressure treatment of UHT bovine milk *(142)* and ewe's milk *(61)* enhanced inactivation of *L. monocytogenes* at mildly elevated temperatures (50°C). High voltage pulsed electric fields applied to whole, 2% fat, and skim milk at 50°C caused a 4-log reduction of listeriae in milk *(134).*

Egg and egg products. Thermal resistance data from experiments with liquid whole eggs, egg yolks, and egg whites are summarized in Tables 10 to 12. *Listeria* strains exhibit some variability in thermal death times when heated in liquid whole egg (Table 10) *(3, 56, 111)* and egg yolk (Table 11) *(3, 111, 124). L. monocytogenes* and *L. innocua* are as much as eightfold more heat resistant than *Salmonella* spp. (including Enteritidis, Typhimurium, and Senftenberg) when tested in eggs under the same experimental conditions *(107, 117, 124, 141).* Heat resistance data for

TABLE 11. *Thermal resistance of* L. monocytogenes *and* L. innocua *(LI) in liquid egg yolks*

Strain	Additives	*D*-values (min) at temperature (°C) of: 60	61.1	62.2	63/63.3	64.4	66.7/67	*z*-value (°C)
Scott A[a]	None		1.56		0.92	0.82		11.45
Scott A 2045[a]	None		0.94		0.35	0.21		5.08
2284[a]	None		2.3		1.28	0.66		6.35
ST.L.[a]	None		0.92		0.38	0.46		9.7
V-7[a]	None		0.7		0.43	0.19		6.22
LI 2430[a]	None		2.29		1.12	0.69		6.43
Five strains[b]	None	1.34	0.89	0.58				6.06
Five strains[a]	None		1.41		0.81	0.44		6.7
Five strains[c]	None				0.35		0.08	5.0
Five strains[a]	10% sucrose		2.05		1.05	0.97	0.46	8.9[d]
Five strains[a]	10% NaCl				10.5	6.11	2.39	5.4
Five strains[c]	10% NaCl				7.9		2.4	10
Five strains[c]	5% NaCl, 5% sucrose				3.75		0.67	6.0
Five strains[a]	10% NaCl, 5% sucrose				21.3	8.26	4.58	5.4[d]
Five strains[a]	20% NaCl					27.3	13.3	7.4
Scott A[e]	10% NaCl				18.5			
HAL 957E1[e]	10% NaCl				8.2			
Scott A[e]	10% sucrose				2.1			
HAL 957E1[e]	10% sucrose				0.8			

[a] Palumbo et al. *(124).* Survivors were counted after growth on TSA for 48 h at 37°C.
[b] Schuman and Sheldon *(141).* Survivors were counted after growth on BHI for 48 h at 37°C.
[c] Michalski et al. *(111).* Tested in capillary tubes. Plated on TSA for 72 h at 30°C, then replica plated onto Palcam agar and counted after 24 h at 30°C.
[d] Calculated from data in table.
[e] Bartlett and Hawke *(3).* Survivors were counted after growth on TSAYE for 72 h at 35°C.

Salmonella in different products, including eggs, have recently been reviewed *(40).*

Supplementing liquid whole eggs with 10% sodium chloride greatly increased $D_{60°C}$-values for strain Scott A from 1.3 to 27.8 min (Table 10) *(91).* Large increases in *D*-values were also observed for a five-strain mixture of *L. monocytogenes* suspended in egg yolk with 10% NaCl (Table 11) *(111, 124).* However, addition of 10% sucrose to egg yolks caused only a small increase in heat resistance *(124).* Addition of salt or sugar to egg yolks caused a time lag before *L. monocytogenes* died. A similar lag phenomenon has been observed in cured meats *(7, 101)* and ice cream mixes *(75).*

Egg whites from freshly laid eggs have a pH of about 8.2. During refrigerated storage, pH levels gradually rise. In some experiments *(125),* thermal resistance of *L. monocytogenes* at 56.6°C increased as the pH of egg whites rose from 8.2 to 9.3. However, other experiments *(141)* indicated that the pH of egg whites (8.2 versus 9.1) had little effect on *D*-values at 56.7 or 58.3°C, with a decrease in thermal resistance observed at the higher pH (Table 12).

In a bench-top flow-injection pasteurizer a commer-

TABLE 12. *Thermal resistance of* L. monocytogenes *in liquid egg whites*

Strain	pH	H_2O_2 (%)	*D*-values (min) at temperature (°C) of: 51.5	53.2	55	55.5	56.6/56.7	57.7	58/58.3	*z*-value (°C)
Five strains[a]	7.8	None					10.4			
Five strains[a]	8.2	None					16.5			
Five strains[a]	8.8	None					20.3			
Five strains[a]	9.3	None					20.9			
Five strains[b]	8.2	None			7.58		4.76		3.47	9.43
Five strains[b]	9.1	None			7.59		4.35		2.41	6.41
Five strains[a]	≤8.8	None				13.0	12.0	8.3		11.3
Five strains[c]	9.0	None		17.7	12.9				3.8	7.0
Five strains[a]	8.8	0.875	37.6	23.3						

[a] Palumbo et al. *(125).* Survivors were counted after growth on TSA for 48 h at 37°C.
[b] Schuman and Sheldon *(141).* Survivors were counted after growth on BHI for 48 h at 37°C.
[c] Michalski et al. *(111).* Tested in capillary tubes. Plated on TSA for 72 h at 30°C, then replica plated onto Palcam agar and counted after 24 h at 30°C.

TABLE 13. *Thermal resistance of* L. monocytogenes *in seafood*

Strain	Food	*D*-values (min) at temperature (°C) of: 51.6	54/54.4	55	57.2/58	60	62	65/66	*z*-value (°C)
Scott A[a]	Crabmeat			12.0		2.61			8.4
Five strains[b]	Lobster	97	55		8.3	2.39			5.0
O57[c]	Salmon				8.48	4.23	3.02	1.18	6.7
O57[c]	Cod				6.18	1.95		0.27	6.1
O62[c]	Salmon				10.73	4.48	2.07	0.87	5.6
O62[c]	Cod				7.28	1.98	0.87	0.28	5.7
Three strains[d]	Crawfish			10.23		1.98		0.19	5.5
Seven strains[e]	Mussels		111.1		19.84	6.3	1.69		4.25
Four strains[f]	Imitation crabmeat				9.7		2.1	0.4	5.7

[a] Harrison and Huang *(71).* Canned crabmeat was blended, inoculated, and packaged in sausage casing. Survivors were counted after growth on TSA for 48 h at 37°C.

[b] Budu-Amoako et al. *(16).* Cooked lobster was blended and inoculated with five-strain mixture. Survivors were counted after growth on TSA for 4 days at 30°C.

[c] Ben Embarek and Huss *(4).* Fish fillets were inoculated and vacuum packed in plastic bags. After heating, samples were stored at 2°C for 3 weeks simulating storage for sous vide cooked food and allowing anaerobic resuscitation of injured cells. Survivors were counted after enrichment in UVMII (Oxoid) and growth on Oxford agar.

[d] Dorsa et al. *(39).* Cooked crawfish tail meat was blended and inoculated with three-strain mixture. Survivors were counted after growth on TSAYE pyruvate for 48 h at 27°C.

[e] Bremer and Osborne *(14).* Mussels were heated, brined, inoculated, and blended. Survivors were counted after growth on TSAYE for 4 days at 35°C.

[f] Mazzotta (unpublished data). After heating and cooling, *Listeria* enrichment broth was added to pouches containing 5 g inoculated, homogenized imitation crabmeat; five replicates per time/temperature interval were used to determine most probable number survivors. Average of triplicate experiments.

cially used time-temperature regimen of 134°F for 4.1 min reduced *Salmonella* Enteritidis populations by 7.5 to 8.5 logs but caused less than a 2-log reduction in numbers of *L. monocytogenes* in egg whites *(116).* Minimal pasteurization conditions for liquid whole eggs (established for salmonellae) were also insufficient to destroy *L. monocytogenes,* and ultrapasteurization processes were recommended *(3, 57, 91).* Michalski et al. *(111)* found that although pasteurization with a plate heat exchanger provided greater lethality than did capillary tubes, liquid whole egg, egg white, and egg yolk received a process $\leq 5.4D$; the authors suggested that current processes recommended by the U.S. Department of Agriculture may not ensure an adequate margin of safety.

Another approach to the effective destruction of listeriae in eggs is the addition of other lethal factors. Nisin, a known inhibitor of *L. monocytogenes,* significantly decreased $D_{56°C}$-values from 3.4 to 2.17 min in liquid whole eggs *(91).* In the presence of 10% NaCl, there was an even greater decrease in $D_{56°C}$ values from 99.2 to 20.4 min *(91).* Therefore, this bacteriocin may be useful in egg processing.

Home cooking of eggs by frying sunnyside up decreased populations of *L. monocytogenes* by only 0.4 log. Cooking scrambled eggs was much more effective, with all or nearly all listeriae destroyed unless there were populations of 10^8 CFU/ml or several eggs were cooked together *(9).*

Fish and shellfish. Several factors were found to significantly affect thermal destruction of *L. monocytogenes* in shellfish and fish products. Among these were the presence of salt, smoke, or liquid smoke and the presence or absence of a cover during cooking.

Catfish fillets, inoculated with *L. monocytogenes* and covered with polyvinylidene wrap or left uncovered, were microwaved to internal temperatures corresponding to well cooked (70°C), minimally cooked (60°C), or slightly undercooked (55°C). Only the well-cooked fish that were covered during cooking had no detectable *L. monocytogenes* cells. At each temperature, fish that had been covered had about half the number of surviving *L. monocytogenes* as detected in the uncovered fish *(77).*

Thermal destruction studies with vacuum-packaged salmon and cod revealed that *D*-values at 58 to 65°C were approximately 1.5 to 4.4 times higher for *L. monocytogenes* in salmon than in cod (Table 13) *(4).* The significantly higher fat content of salmon (10.6 to 17.2%) as compared to cod (0.5 to 0.8%) appeared to protect *L. monocytogenes* from thermal destruction.

The smoking process for brined salmon affected temperatures required for inactivation of *L. monocytogenes (132).* Required minimum temperatures for complete inactivation of *L. monocytogenes* in salmon exposed to smoke during all, half, or none of the heating process were 153°F (67.2°C), 176°F (80°C), and 181°F (82.8°C), respectively. Thermal processing required for contaminated salmon treated with liquid smoke also varied inversely with the concentration of the liquid smoke product used.

Thermal tolerance of *L. monocytogenes* in cooked crab *(71),* crawfish *(39),* and lobster *(16)* were somewhat similar, with $D_{60°C}$-values of 2.61, 1.98, and 2.39 min, respectively.

TABLE 14. *Thermal resistance of* L. monocytogenes *in vegetables*

Strain	Food	*D*-values (min) at temperature (°C) of: 50	54	55	56	60	62	64	68	*z*-value (°C)
Scott A[a]	Cabbage juice pH 4.6	25.0	6.71		3.64					7.01
Scott A[a]	Cabbage juice pH 5.6	>60	8.35		6.8					5.63
LCDC 81-861[a]	Cabbage juice pH 4.6	13.3	4.88		2.04					7.54
LCDC 81-861[a]	Cabbage juice pH 5.6	>60	8.93		4.74					6.45
Scott A[b]	Carrot homogenate					5.02		1.47	0.3	7.04
NCTC 11994[b]	Carrot homogenate					7.76		2.53	0.44	6.70
NCTC 11994[d]	Potato slices	30.6		3.3		0.5				5.59[c]
Four strains[e]	Onions				0.8	0.23	0.10			6.7
Four strains[e]	Broccoli				2.3	0.62	0.39			7.8
Four strains[e]	Green peppers				3.9	0.92	0.31			5.5
Four strains[e]	Mushrooms				5.0	0.69	0.30			4.9
Four strains[e]	Peas				5.2	1.04	0.41			5.5

[a] Beuchat et al. *(6).* Survivors counted after growth on TSA for 24 h at 30°C.
[b] Gaze et al. *(59).* Survivors counted after growth on TSAYE for 48 h, 30°C.
[c] Calculated from data in table.
[d] Doherty et al. *(35).* Survivors counted after growth on Palcam agar for 48 h at 30°C.
[e] Mazzotta *(102).* After heating and cooling, *Listeria* enrichment broth was added to pouches containing 3 g inoculated, homogenized vegetable; five replicates per time/temperature interval were used to determine most probable number survivors. Average of duplicate experiments.

Data obtained from experiments with crabmeat indicate that current processing conditions for canned crabmeat (30 min at 85°C) are more than adequate to destroy *L. monocytogenes (71).* Mazzotta (unpublished data, Table 13) studied the heat resistance of *L. monocytogenes* in imitation crabmeat made from surimi. *D*-values at 58, 62, and 66°C were 9.7, 2.1, and 0.4 min, respectively, for stationary-phase cells.

Addition of nisin (25 mg/kg) to lobster meat before heating reduced the time and temperature required for a 4-log kill of *L. monocytogenes (15).* When used alone, nisin or heating at 65°C for 2 min decreased numbers of *L. monocytogenes* about 2 logs. When nisin and heat were used in combination, *L. monocytogenes* decreased 4 logs.

In a different experiment, 10^5 to 10^6 (total number of cells) *L. monocytogenes* were injected into raw shrimp. After boiling for 5 min, viable listeriae were detected in one of eight samples. However, all boiled samples were positive following 3 days of incubation at 4°C *(104). L. monocytogenes* may be destroyed more easily on naturally contaminated shrimp because the listeriae would most likely be on the surface of the shrimp.

In blended green shell mussels, a mixture of seven *L. monocytogenes* strains exhibited a $D_{60°C}$-value of 6.3 min (Table 13) *(14).* These shellfish had been soaked in a brine solution in preparation for hot smoking, and it is likely that the salt, sugar, and acetic acid enhanced the thermotolerance of *L. monocytogenes* in these mussels.

Vegetables. Although *L. monocytogenes* has been found on vegetables, including those subjected to minimal thermal processing, limited heat resistance studies have been conducted. In two studies that included thermal inactivation of *L monocytogenes* in beef as well as vegetables under the same testing conditions similar *D*-values were obtained for minced meat and potato slices *(35)* and for homogenates of meat and carrots *(59).* Strain NCTC 11994 was approximately 15 times more heat resistant in carrots than in potatoes (Table 14), but this may be a result of test conditions. In the former experiment, listeriae were mixed with homogenized carrot, and survivors were enumerated on a nonselective medium; in the latter experiment potato slices were inoculated by dipping, and survivors were counted on selective medium.

L. monocytogenes was more sensitive to heat in cabbage juice at pH 4.6 than at pH 5.6, and strain LCDC 81-861 was more sensitive than strain Scott A in these experiments (Table 14) *(6).* The *z*-values for strain Scott A were similar in cabbage juice, pH 4.6, and in carrot homogenate (7.01 and 7.04 °C, respectively).

Mazzotta (Table 14) *(102)* studied the heat resistance of a composite of four strains of *L. monocytogenes* in onions, broccoli, peppers, mushrooms, and peas. Heat resistance was highest for mushrooms and peas, with $D_{56°C}$-values of 6.7 and 5.8 min, respectively; *z*-values were 6.7°C for mushrooms and 5.1°C for peas.

Other products. *L. monocytogenes* (10^6 CFU/g) inoculated into meat, cheese, and egg ravioli survived and grew slowly at 5°C for 14 days. However, boiling for 3 to 7 min, similar to a home-cooking process, destroyed all *Listeria* cells *(5).*

In chicken gravy *L. monocytogenes* populations of 100 CFU/ml can be destroyed by heating to 65°C for 1.3 min. However, *Listeria* populations in gravy increased 4 logs during cooling and during 1 week of storage at 7°C *(76).* Such high cell concentrations would require more intense heating for destruction.

Heating of pudding and cream sauce to 60°C in a microwave oven reduced *L. monocytogenes* populations by

about 2.4 and 1.6 logs, respectively *(73)*. Addition of 1% NaCl to the cream sauce slightly increased the effects of heating.

Mazzotta *(103)* studied the heat resistance of *L. monocytogenes* in apple, orange, and white grape juices. Heat resistance was higher after acid adaptation. At 56 to 62°C *L. monocytogenes* was more heat resistant than *Salmonella* and less heat resistant than *E. coli* O157:H7. Depending on growth conditions and the juice tested, *z*-values ranged from 5.6 to 6.6°C. The study found that normal processing conditions calculated for hot-filled, shelf-stable juices achieve a lethality in excess of 50,000*D* for *L. monocytogenes*. Unpublished data on the influence of sorbate and benzoate on the heat resistance of *L. monocytogenes* in fruit juices indicate that both preservatives decreased *D*-values by as much as 100-fold at temperatures below 60°C. However, because *z*-values were consistently lower when the preservatives were added, heat sensitization did not appear to be significant at higher heating temperatures.

RESEARCH NEEDS

Most heat resistance research on *L. monocytogenes* has been done in dairy, meat and poultry, and egg products. Limited data exist for seafood, fruits, and vegetables. With increasing concerns for the safety of minimally processed vegetables, more heat resistance studies need to be conducted on these types of products.

More information is also needed on the variability of heat resistance among different *L. monocytogenes* strains. Food processors need to know whether strains used in the published literature are representative of all pathogenic listeriae. Systematic and comparative heat resistance studies should be used to identify and select the most resistant strains for future thermal resistance studies in order to determine conservative criteria for safe processing.

Because food components influence heat resistance of *L. monocytogenes,* further research with different formulations of certain foods such as frozen desserts and processed meats should determine their relative effects on survival and heat resistance of listeriae. In particular, more data are needed on the heat resistance of *L. monocytogenes* in foods with reduced water activity and in foods containing novel preservatives such as bacteriocins.

As new processing technologies become available, research on the combined effects of the technology and heat may be needed to provide the most effective processing approach. Other food processing conditions, such as heating rates and the use of moist or dry heat, should be investigated further to determine their effect on heat resistance and death of listeriae. Although advantageous for some products, slow heating rates can increase the heat resistance of *L. monocytogenes*.

It has been demonstrated that stressed *L. monocytogenes* cells are much more resistant to heat than unstressed cells. Additional data are needed on *D*- and *z*-values using stressed *L. monocytogenes* in specific food commodities. Such studies may provide more realistic conservative heat resistance data for use by food processors.

We are aware of no regulations that specify that a specific log reduction of *L. monocytogenes* be achieved in a food. Thus, while not strictly a research need, it is important that the scientific community have open and transparent discussions on the appropriate level of inactivation needed to achieve a tolerable level of risk and to define the food safety objectives *(155)* to be achieved with respect to *L. monocytogenes* in foods.

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