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Inhibition of *Listeria monocytogenes* by Fatty Acids and Monoglycerides

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Fatty acids and monoglycerides were evaluated in brain heart infusion broth and in milk for antimicrobial activity against the Scott A strain of *Listeria monocytogenes*. $C_{12:0}$, $C_{18:3}$, and glyceryl monolaurate (monolaurin) had the strongest activity in brain heart infusion broth and were bactericidal at 10 to 20 μg/ml, whereas potassium (K)-conjugated linoleic acids and $C_{18:2}$ were bactericidal at 50 to 200 μg/ml. $C_{14:0}$, $C_{16:0}$, $C_{18:0}$, $C_{18:1}$, glyceryl monomyristate, and glyceryl monopalmitate were not inhibitory at 200 μg/ml. The bactericidal activity in brain heart infusion broth was higher at pH 5 than at pH 6. In whole milk and skim milk, K-conjugated linoleic acid was bacteriostatic and prolonged the lag phase especially at 4°C. Monolaurin inactivated *L. monocytogenes* in skim milk at 4°C, but was less inhibitory at 23°C. Monolaurin did not inhibit *L. monocytogenes* in whole milk because of the higher fat content. Other fatty acids tested were not effective in whole or skim milk. Our results suggest that K-conjugated linoleic acids or monolaurin could be used as an inhibitory agent against *L. monocytogenes* in dairy foods.

Listeria monocytogenes was recognized as a significant human food-borne pathogen in the 1980s and continues to be a major concern in the food industry (5, 19). The pathogen has been isolated from the food-processing environment and finished food products. Among these products, *L. monocytogenes* has been found in a variety of dairy foods (5, 19). Because *L. monocytogenes* can grow slowly at refrigeration temperature, control of the organism is of particular concern in minimally processed refrigerated foods with an extended shelf life (5, 14). For control of *L. monocytogenes* in these foods, it often becomes necessary to incorporate barriers, including preservatives (5, 14, 19).

Trace quantities of certain long-chain fatty acids have been known to inhibit microorganisms, especially gram-positive bacteria (11, 16). Susceptibility to fatty acids varies considerably among species. Certain mastitis pathogens were sensitive to trace quantities of fatty acids (9). Food-borne pathogens including *Clostridium botulinum*, *Clostridium perfringens*, and *Staphylococcus aureus* were also sensitive to certain fatty acids and monoglycerides (2, 7, 17, 20). Arachidonic acid was reported to have bactericidal activity against several gram-positive bacteria including *L. monocytogenes* (13). Recently, fractions of free fatty acids extracted from butter and lard were reported to have in vitro activity against *L. monocytogenes* (3).

Our laboratory has been investigating naturally occurring compounds in foods that have inhibitory activity against food-borne pathogens. The objectives of this study were to determine the in vitro susceptibility of *L. monocytogenes* to various fatty acids commonly present in bovine milk fat and to test the activity of the fatty compounds in milk. Our results indicate that *L. monocytogenes* is quite sensitive to certain fatty acids and that it may be possible to develop an antimicrobial system based on fatty acids for inhibition of *L. monocytogenes* in foods.

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MATERIALS AND METHODS

Sources and preparation of fatty acids. Fatty acids ($C_{12:0}$, $C_{14:0}$, $C_{16:0}$, $C_{18:0}$, $C_{18:1}$, $C_{18:2}$, and $C_{18:3}$) and monoglycerides of lauric acid, myristic acid, and palmitic acid were obtained from Sigma Chemical Co., St. Louis, Mo. They were reported by the manufacturer to be >99.9% pure. CLA (conjugated isomers of linoleic acids) and its potassium salt was a generous gift of M. W. Pariza's laboratory, University of Wisconsin. Stock solutions of fatty acids were prepared in absolute ethanol and stored at −80°C under nitrogen. Potassium (K)-CLA was prepared in 50% ethanol. The fatty acids were added to media or to foods just prior to inoculation to give final concentrations of 2, 20, and 200 μg/ml. In the screening in brain heart infusion (BHI), the ethanol concentration was 3%, which was not inhibitory to *L. monocytogenes*. In the food studies, control incubations received equal quantities of ethanol (1 to 2%), which had no effect on the growth or viability of the pathogen.

Bacteria, growth media, and enumeration. *L. monocytogenes* Scott A was used in this study. Stock cultures were maintained on BHI agar (Difco Laboratories, Detroit, Mich.) and stored at 4°C. Inocula were developed by inoculation of a loopful of cells in 10 ml of BHI broth in tubes (1.0 by 12.5 cm) which were incubated at 37°C overnight without shaking. This was used to inoculate BHI broth adjusted with 5 N HCl to pH 5.0 or 6.0 that contained fatty acids or monoglycerides. The initial inoculum of *L. monocytogenes* was 10^3 to 10^4 CFU/ml. *L. monocytogenes* was enumerated by plating dilutions on BHI agar each day for 6 days. The minimum listericidal concentration of fatty acids and monoglycerides was defined in this study as the lowest concentration at which no colonies grew on BHI agar after 2 days of incubation.

For inhibition tests in milk, the experimental procedure was slightly modified. Tubes containing 10 ml of raw unhomogenized whole or skim milk (obtained from the University of Wisconsin-Madison dairy plant) were autoclaved at 121°C for 12 min. Fatty acids ($C_{12:0}$, $C_{18:2}$, $C_{18:3}$, or monolaurin) dissolved in absolute ethanol were added to give final concentrations of 50, 100, or 200 μg/ml (final concentrations

of ethanol were 0.5, 1, or 2%). K-CLA was dissolved in 50% ethanol and filter sterilized before addition. The final concentration of ethanol in incubations containing CLA was 1 to 2%. In certain experiments, the media were supplemented with antioxidants and emulsifiers. The antioxidants butylated hydroxyanisole, α-tocopherol, and ascorbic acid and the emulsifier sodium citrate (all from Sigma Chemical Co.) were individually dissolved in absolute ethanol or water and filter sterilized prior to being added to tubes containing milk. The milk tubes were inoculated with 500 to 1,000 CFU of *L. monocytogenes* Scott A per ml and were incubated at 4°C, room temperature, or 30°C. *L. monocytogenes* was enumerated by plating on modified McBride agar (15) and on modified Oxford agar (4), and plates were incubated at 37°C for 48 h. The detection limit was 10 CFU/ml. The data shown in the tables and figures are the averages of two experiments done in duplicate.

TEM. For transmission electron microscopy (TEM) analysis, *L. monocytogenes* Scott A was grown at 37°C in BHI (pH 6.0) broth overnight, and the cells were centrifuged and washed with 0.67 M phosphate buffer (pH 6.0) (PB). The cells were resuspended in PB containing fatty acids or monolaurin and incubated for 24 h at 37°C. The cells were harvested by centrifugation at 12,000 × *g* for 15 min at 4°C, resuspended in PB containing 2.5% (wt/vol) glutaraldehyde, and fixed for 24 h at 4°C. The pellets were gently washed twice in PB treated with 2% (wt/vol) osmium tetroxide and then washed twice with water. The cells were successively dehydrated in graded solutions of ethanol (50/70/80/95/100%) for 10 min at each step, stained with uranyl acetate and lead citrate, passed through propylene oxide, and embedded in epoxy resin. Sections were cut with a Diatome diamond knife by using an Ultracut ultramicrotome (Reichert-Jung, Vienna, Austria). Sections were observed with a Phillips 410 electron microscope.

RESULTS

In vitro inhibition of *L. monocytogenes* by fatty acids. Fatty acids and monoglycerides, especially those commonly present in bovine milk fat, were tested singly in BHI broth at pH 5 and 6 for inactivation or inhibition of *L. monocytogenes*. Of the compounds screened, several inactivated *L. monocytogenes*, including monolaurin, $C_{12:0}$, $C_{18:2}$, K-CLA, and $C_{18:3}$ at concentrations of 10, 20, 200, and 100 μg/ml, respectively (Table 1). The shorter-chain compounds $C_{12:0}$ and monolaurin were most effective, possibly because of greater solubility. The fatty acid compounds were also slightly more effective at pH 5 than at pH 6, probably because of greater susceptibility of the organism at the lower pH. Among the unsaturated fatty acids, linolenic acid was the most inhibitory against *L. monocytogenes*. Of three monoglycerides screened, only monolaurin inactivated the pathogen (Table 1). The ethanol concentration used in the in vitro screening of fatty acids also markedly influenced the inhibitory activity of the fatty acids, probably by increasing solubility. Inhibition by fatty acids was not observed when the ethanol concentration was less than 1% in BHI broth.

Inhibition of *L. monocytogenes* in milk by fatty acids. Fatty acid compounds that were listericidal in BHI were evaluated for inhibitory activity in milk. Of the fatty acids and monoglycerides tested, monolaurin and K-CLA showed the strongest inhibitory action in milk. K-CLA prolonged the lag phase of *L. monocytogenes* in skim and whole milk. The length of the lag period in skim milk at 25°C was proportional to the concentration of K-CLA and ranged from 5 to 27 h at concentrations of 50 and 300 μg/ml, respectively (Fig. 1). *L. monocytogenes* was more strongly inhibited at 4°C. Addition of K-CLA at 100 to 300 μg/ml extended the lag phase in whole milk by 5 to 18 days at 4°C compared with the control (Fig. 2).

Sodium citrate is commonly used as an emulsifier and chelating agent in many food applications. Combining

TABLE 1. Inhibition of *L. monocytogenes* by fatty acids in BHI broth incubated at 37°C[a]

Fatty acid	MBC (μg/ml)	
	pH 6.0	pH 5.0
$C_{12:0}$	20	10
$C_{14:0}$	NI[b]	ND[c]
$C_{16:0}$	NI	ND
$C_{18:0}$	NI	ND
$C_{18:1}$	NI	ND
$C_{18:2}$	200	50
K-CLA	200	100
$C_{18:3}$	100	20
Glyceryl monolaurate	10	10
Glyceryl monomyristate	NI	ND
Glyceryl monopalmitate	NI	ND

[a] Growth was measured by plating cells daily for up to 6 days. When listericidal, complete killing was observed after 24 h and in subsequent platings. The cells in the control incubations containing 3% ethanol grew from 10^3 to ~10^8 CFU/ml.
[b] NI, not inhibitory at 200 μg/ml.
[c] ND, not tested since no activity was observed at pH 6.

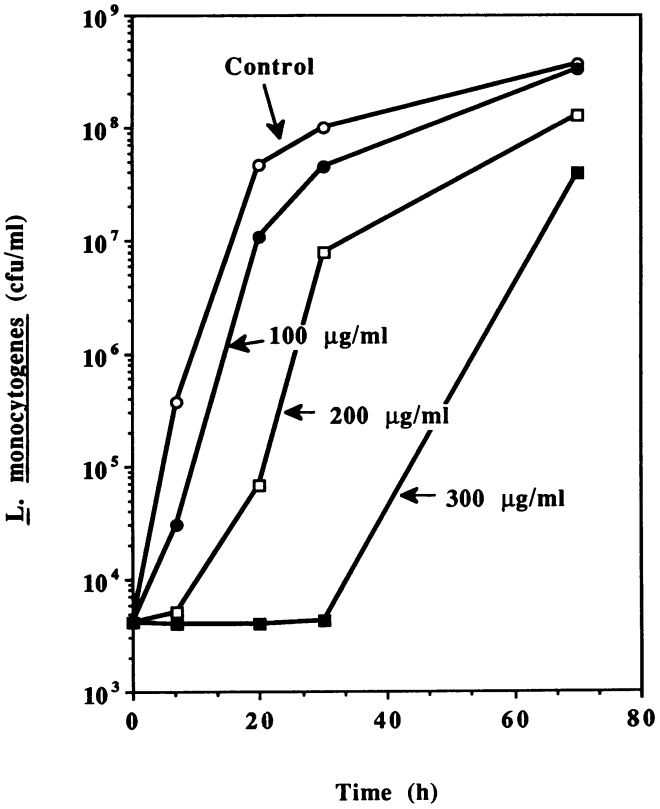

FIG. 1. Inhibition of *L. monocytogenes* by K-CLA in skim milk at 25°C.

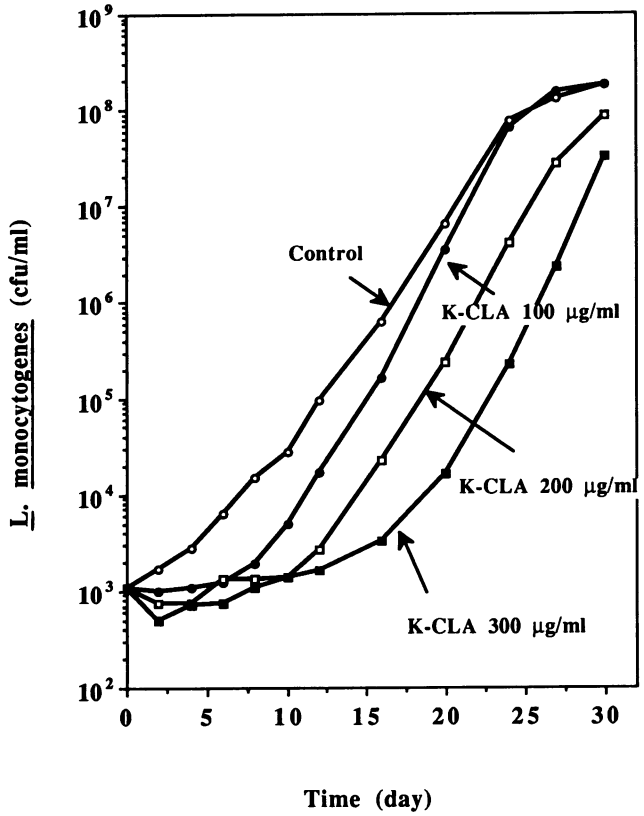

FIG. 2. Inhibitory activity of K-CLA against *L. monocytogenes* in whole milk at 4°C.

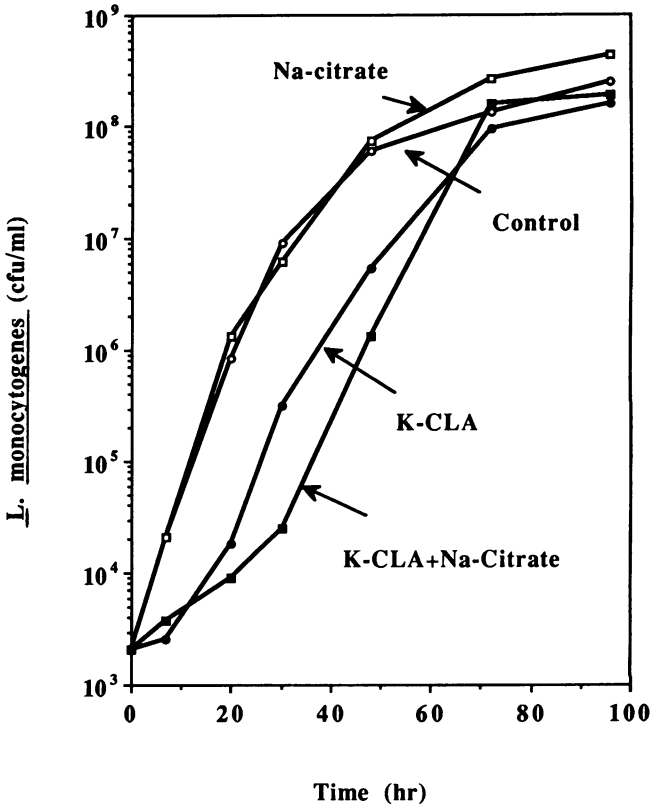

FIG. 3. Potentiation of K-CLA inhibitory activity by sodium citrate in whole milk at 25°C.

K-CLA (200 μg/ml) with sodium citrate (2,000 μg/ml) increased the effectiveness of K-CLA in whole milk (Fig. 3), probably by increasing the solubility of CLA. The influence of certain antioxidants on the inhibitory activity of K-CLA was evaluated. Butylated hydroxyanisole (100 to 200 μg/ml) potentiated the inhibitory activity of K-CLA in whole and skim milk (Table 2), whereas butylated hydroxyanisole alone was not inhibitory. Ascorbate (vitamin C) and α-tocopherol (vitamin E) at 100 to 200 μg/ml also enhanced the inhibitory activity of K-CLA in skim and whole milk (Table 2).

In contrast to the bacteriostatic activity observed with CLA, monolaurin was bactericidal at ≥200 μg/ml in skim milk at 4°C (Fig. 4). Growth of *L. monocytogenes* was not detected for up to 30 days in skim milk containing 200 μg of monolaurin per ml, whereas at 100 μg/ml growth started after 12 to 14 days. The antimicrobial activity of monolaurin in skim milk was temperature dependent. Monolaurin was listericidal at 4°C but was not inhibitory at 30°C (Fig. 5). Monolaurin alone did not inhibit *L. monocytogenes* in whole milk. Its inactivity in whole milk compared with skim milk could be due to its sequestration by fat globules or lipophilic proteins. This was supported by experiments showing that inhibitory activity varied inversely with the fat content in milk (Table 3). Although monolaurin alone was not inhibitory against *L. monocytogenes* in whole milk, it increased the activity of K-CLA in both skim and whole milk (Table 2).

We also observed that monolaurin more strongly inhibited *L. monocytogenes* in 2% chocolate milk than in 2% milk at 4°C (Table 2). This may be due to the presence of cocoa, which has been reported to inhibit *L. monocytogenes* in broth media and is neutralized by casein (18). Hence, it seems likely that monolaurin at 200 μg/ml may overcome the neutralization by casein in milk.

TABLE 2. Effect of monolaurin and various antioxidants on the inhibitory activity of K-CLA against *L. monocytogenes* in skim and whole milk at room temperature[a]

Addition to milk	No. of *L. monocytogenes* (log CFU/ml) after given incubation time			
	Skim milk[b]		Whole milk[c]	
	24 h	48 h	24 h	48 h
Control	5.21	7.08	5.32	7.91
K-CLA	2.45	7.20	2.54	3.78
Monolaurin	4.04	5.70	5.59	7.30
K-CLA + monolaurin	2.82	5.51	2.36	3.70
BHA[d]	5.26	6.04	5.32	7.34
K-CLA + BHA	2.50	5.14	2.21	3.36
Ascorbate	6.11	7.32	5.65	7.54
K-CLA + ascorbate	2.45	5.65	2.48	4.59
α-Tocopherol	5.63	7.45	5.56	7.28
K-CLA + α-tocopherol	2.88	5.70	5.54	4.38

[a] The initial inoculum was 2.65 log CFU/ml of milk. The control and all treatments grew to 7 to 8 log CFU/ml after 96 h of incubation at room temperature.

[b] The concentrations of K-CLA, monolaurin, and antioxidants in skim milk were all 100 μg/ml.

[c] The concentrations of K-CLA, monolaurin, and antioxidants in whole milk were all 200 μg/ml.

[d] BHA, butylated hydroxyanisole.

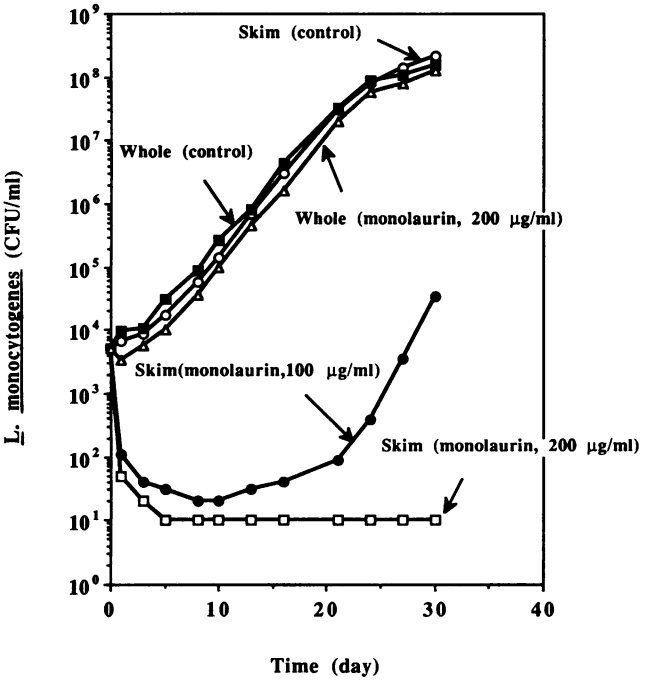

FIG. 4. Inhibition of *L. monocytogenes* by monolaurin in skim and whole milk at 4°C.

TABLE 3. Effect of fat content on inhibition of *L. monocytogenes* by monolaurin in milk[a] at 4°C

Milk sample	*L. monocytogenes* (log CFU/ml)	
	3 days	7 days
Whole milk (control)	3.23	3.78
+ Monolaurin		
100 μg/ml	3.30	3.81
200 μg/ml	3.10	3.42
2% milk (control)	3.28	3.60
+ Monolaurin		
100 μg/ml	3.02	3.32
200 μg/ml	2.60	1.60
2% chocolate milk (control)	3.20	3.45
+ Monolaurin		
100 μg/ml	3.17	2.92
200 μg/ml	<1[b]	<1
1% milk (control)	3.32	3.78
+ Monolaurin		
100 μg/ml	2.84	2.58
200 μg/ml	<1	<1
Skim milk (control)	3.34	3.75
+ Monolaurin		
100 μg/ml	1.39	<1
200 μg/ml	<1	<1

[a] The initial inoculum was 2.80 to 3.1 (average, 2.95) log CFU per ml of milk.
[b] The detection limit was 10 cells per ml.

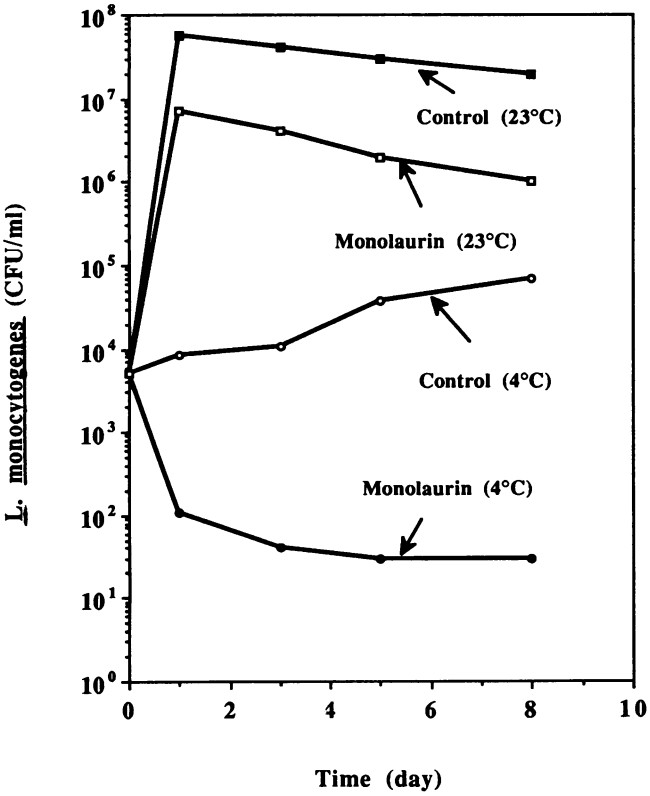

FIG. 5. Effect of temperature on monolaurin activity in skim milk.

TEM analysis of *L. monocytogenes* after treatment with monolaurin and fatty acids. *L. monocytogenes* was treated for 24 h with monolaurin, $C_{12:0}$, $C_{18:2}$, K-CLA, and $C_{18:3}$ at 50, 50, 200, 200, and 100 μg/ml, respectively, and morphological changes were examined by electron microscopy. The cell surface and cytoplasm in control cells and in cells treated with ethanol alone appeared uniform and homogeneous (Fig. 6a and b). Cells treated with 50 μg of monolaurin per ml had marked morphological changes (Fig. 6c). In several cells, the cytoplasmic contents appeared to separate from the cell envelope but the cell surface appeared to remain intact. In other cells, however, breakage of the cell envelope and leakage of cytoplasmic contents were observed. The cytoplasm in cells treated with $C_{18:3}$ appeared more filamentous than in control cells and the cell surface appeared slightly irregular, but the cells were not lysed (Fig. 6d). Similar changes were observed with $C_{12:0}$, $C_{18:2}$, and K-CLA. The filamentous appearance in cells treated with fatty acids indicated that cell envelope permeability was increased.

DISCUSSION

Medium- and long-chain fatty acids have long been recognized as inhibitors of spores and vegetative cells of gram-positive bacteria (11, 16). Antimicrobial activities of fatty acid compounds, particularly monolaurin, have also been demonstrated in some foods (2, 7, 17). In this study, we have found that certain free fatty acids and monolaurin are bactericidal to *L. monocytogenes* in BHI broth. The medium-chain lauric acid ($C_{12:0}$) had stronger activity against *L. monocytogenes* than longer-chain fatty acids that were tested. Decreasing effectiveness with longer chain length

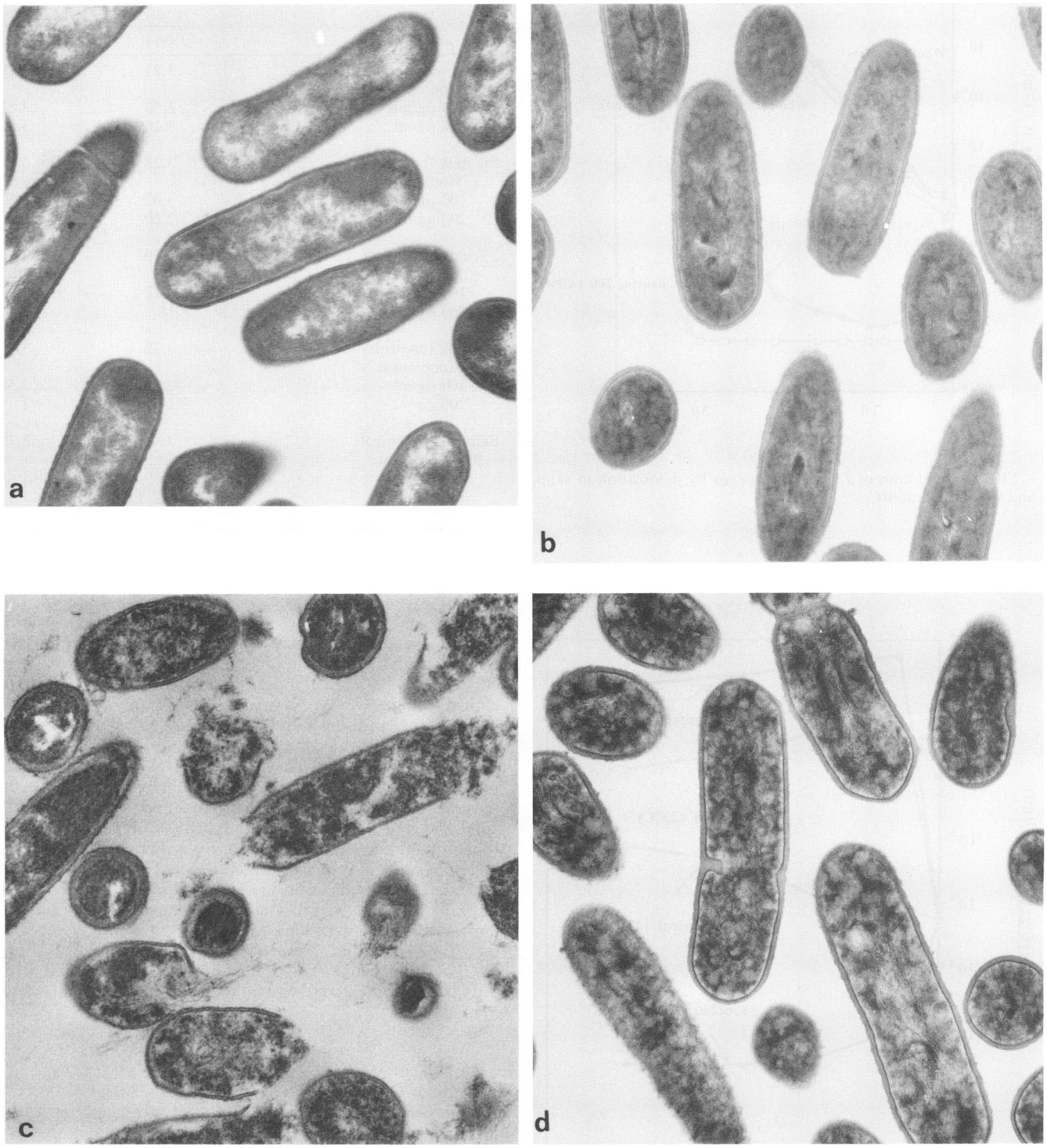

FIG. 6. TEM images (magnification, ×44,080) of *L. monocytogenes* Scott A treated with monolaurin or $C_{18:3}$. (a) Treated in sodium phosphate (pH 6) for 24 h at 37°C; (b) treated with 3% ethanol alone in sodium phosphate (pH 6) for 24 h at 37°C; (c) treated with 50 μg of monolaurin per ml and 3% ethanol; (d) treated with 200 μg of $C_{18:3}$ per ml and 3% ethanol.

may be related to increased hydrophobicity and decreased solubility. Inhibitory fatty acids must be sufficiently water soluble to reach an effective concentration in the aqueous solution and yet sufficiently hydrophobic to interact with hydrophobic proteins or lipids on the bacterial cell surface. Inhibitory activity of fatty acids and monolaurin was also increased at pH 5 versus pH 6, probably due to increased susceptibility of *L. monocytogenes* at the lower pH. This was supported by the observation that monolaurin, which is nonionic and equally soluble at both pHs, also had stronger activity at pH 5. Monolaurin had bactericidal activity against *L. monocytogenes* in milk. The pathogen was more susceptible to monolaurin in skim milk at low temperature, and monolaurin was bactericidal at 4°C but was not inhibitory in skim milk at 30°C or in whole milk at 4 or 30°C. The effectiveness of monolaurin was influenced by the fat content of milk and by potentiating agents, including antioxidants. Of the several free fatty acids that were inhibitory in vitro, including linolenic acid, only K-CLA showed activity in milk. The potassium salt may have had higher solubility, or the slightly different three-dimensional structure may have preferentially reacted with cells rather than with food components.

The composition of a food could affect the inhibitory activity of fatty acids and monoglycerides. Certain nutrients in relatively high concentrations, such as lipophilic proteins (e.g., albumin), fat globules, starch, or others, could interact with fatty acids and decrease their availability (11). In some foods, the use of fatty acids as inhibitors could be undesirable because they can affect the organoleptic and functional properties (1, 12).

An intriguing and unresolved question concerns the mechanism by which fatty acids are bactericidal to cells. Cellular membranes have long been regarded as a primary target for antimicrobial fatty acids (6). Fatty acids have been demonstrated to affect cell permeability and transport of nutrients (8). Recent studies have indicated that micromolar concentrations of fatty acids can affect the activity of enzymes in the cell membrane (21, 22). Polyunsaturated fatty acids have also been reported to inhibit organisms through autoxidation and formation of peroxides and radicals (10). Knapp and Melly (13) reported that the bactericidal effects of polyunsaturated fatty acids were mediated by a peroxidative process involving hydrogen peroxide and bacterial iron. Our results showed that linolenic acid ($C_{18:3}$) was more inhibitory than linoleic acid ($C_{18:2}$) and oleic acid ($C_{18:1}$) in BHI, which would be consistent with a peroxidative mechanism. However, lauric acid, which is saturated and does not readily autoxidize, was the most inhibitory of the fatty acids tested. Furthermore, *L. monocytogenes* survives in macrophages and probably has considerable natural resistance to peroxides and organic radicals. Further work is being carried out to understand the mechanism of inhibition of *L. monocytogenes*.

In conclusion, we have found that certain fatty acids are inhibitory to *L. monocytogenes* in media and in milk, particularly at refrigeration temperatures. The activity we observed at low temperature suggests that fatty acids could be added or generated and could serve as barriers to prevent growth of *L. monocytogenes* in refrigerated foods when processing or intrinsic protection is inadequate.

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