Comprehensive REVIEWS in Food Science and Food Safety

Pathogens of Interest to the Pork Industry: A Review of Research on Interventions to Assure Food Safety

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Abstract: Pork is the most consumed meat in the world and is a source of foodborne diseases. To develop effective food safety interventions for pork, it is crucial to understand the nature of the important pathogens affecting the pork industry, their prevalence at different phases of pork production, and interventions against pathogens in pork. The purpose of this study was to outline the significance of *Salmonella*, *Campylobacter*, *Trichinella spiralis*, *Toxoplasma gondii*, *Listeria monocytogenes*, and methicillin-resistant *Staphylococcus aureus* to the pork industry. *Trichinella* and *Toxoplasma* are historically relevant pathogens to pork and represent the effectiveness that preharvest intervention strategies can accomplish for the control of toxoplasmosis and trichinellosis. *Salmonella* and *Campylobacter* are common inhabitants of swine intestines causing a high prevalence of these pathogens on the farm as well as potential contamination during slaughter. However, both *Salmonella* and *Campylobacter* can be reduced through on-farm strategies, hygienic slaughter practices, and processing technologies. Methicillin-resistant *S. aureus* is an emerging pathogen with increasing focus on the livestock industry and interventions pre and postharvest have been considered for reduction of this microorganism. The greatest challenge for processors is *L. monocytogenes* as contamination of the further processing environment requires adequate interventions for both pork and the environment. Novel technologies such as use of bacteriophages, feed additives, and high-pressure processing are being explored as interventions against pathogens of pork. Overall, pork does contribute to foodborne diseases and various interventions are now being used against the different pathogens found in pork.

Introduction

Foodborne pathogens are a major contributor to human illnesses, hospitalizations, and deaths each year. The Centers for Disease Control and Prevention (CDC) estimates that 47.8 million illnesses and 3000 deaths are caused by foodborne pathogens each year (CDC 2011). Moreover, foodborne illness costs the United States $152 billion dollars each year for acute medical care and long-term health-related costs (Scharff 2010). Lost productivity, product recalls, and decreased sales from damaged organizational reputation also incur significant costs (Scharff 2010). *Salmonella* spp., *Listeria monocytogenes*, *Campylobacter* spp., *Staphylococcus aureus*, and *Toxoplasma gondii* are among the top pathogens causing foodborne illness and death annually (CDC 2011). These pathogens are well-documented as being present in pigs or pork products, making pork a potential contributor to foodborne illness.

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Although pork is less associated with foodborne illness than other meat sources, it remains significant due to its large consumption in a variety of products. Pork is the most consumed meat in the world (Delgado and others 2001). In the United States, per capita consumption of pork has remained steady over the past 20 y at about 60 pounds/y (American Meat Insti. 2009), and in the European Union people eat more pork than any other meat (Devine 2003). Pork has always been a major meat source for people in China, and consumption continues to increase with economic development. From 1989 to 1993, the number of people in China consuming pork rose by 8% and daily consumption increased from 0.18 to 0.21 pounds/person (Guo and others 2000). International trade of pork is economically important to the U.S. swine industry. According to the USDA in January 2012, the United States exported over 501 million pounds of pork (ERS-USDA 2012). Because pork is so widely consumed and is an important U.S. export, ensuring a safe pork supply is crucial. Furthermore, heightened consumer awareness of food safety makes the reduction of foodborne pathogens from pork important for producers and processors. Understanding the nature of pathogens, their prevalence at different phases of pork production and processing, and current intervention methods are important in developing more efficient interventions against foodborne pathogens.

doi: 10.1111/1541-4337.12001

The purpose of this study is to outline the following pathogens and their importance in the pork industry: *Salmonella* spp., *Campylobacter* spp., *Trichinella spiralis*, *T. gondii*, *L. monocytogenes*, and *S. aureus*. These 6 pathogens were selected for their unique relationship to pork and/or their importance in causing foodborne diseases. *Salmonella* spp., *Campylobacter* spp., and *S. aureus* are among the top 5 pathogens causing foodborne illness and leading to hospitalization in the United States (CDC 2011). The recent emergence of Methicillin-resistant *S. aureus* (MRSA) and public scrutiny of this pathogen in livestock, particularly swine, make it important to understand in relation to pork production. *T. spiralis* and *T. gondii* were selected for their historic significance in the pork industry; *T. gondii* also results in 8% of the total foodborne illness hospitalizations (CDC 2011). Finally, *L. monocytogenes* was chosen for its notorious lethality and contribution of 24% of foodborne illness resulting in death (CDC 2011).

One will notice the exclusion of *Escherichia coli* in this study. The importance of *E. coli* O157:H7 to the food industry cannot be denied, but asymptomatic carriage in swine is low and pork is rarely the cause of illness from this pathogen. In a study in the United Kingdom, only 0.6% of swine were carriers of *E. coli* O157, which is significantly lower than *Salmonella* spp. and *Campylobacter* spp. with 23% and 69% of pigs positive, respectively (Milnes and others 2009). Similarly, a Canadian study also found low levels of *E. coli* O157 in pigs, none of which were identified as O157:H7 (Farzan and others 2010). *E. coli* O157:H7 is linked to cattle and plays a more important role in foodborne illnesses from beef or leafy green vegetables. Another pathogen found in swine that was excluded from this study is *Yersinia entercolitica* which, although relatively prevalent in swine intestines, is not a major cause of foodborne illness.

Salmonella

Salmonella is a Gram-negative genus belonging to the family *Enterobacteriaceae* whose other members include *Escherichia*, *Shigella*, *Yersinia* and many others. *Salmonella* spp. can grow over a wide temperature range, typically from 5 to 45 °C. The pH growth range of *Salmonella* is 4 to 9 (Doyle and Cliver 1990). The sensitivity of *Salmonella* spp. to salt depends on the temperature with many strains capable of growing in foods with 2% NaCl near their growth optimum temperature (Montville and Matthews 2008). In addition, *Salmonella* spp. are very resilient and can survive for extended periods of time in low moisture foods such as peanut butter, chocolate and infant formula (Podolak and others 2011).

Currently, there are only 2 recognized species of Salmonella: *S. enterica* and *S. bongori* (Montville and Matthews 2008). There are 6 recognized subspecies of *S. enterica* yet the vast majority of isolates associated with foodborne illness are *S. enterica* subsp. *enterica*. Strains can be further discriminated by serotyping with over 1400 serovars of *S. enterica* subsp. *enterica* that have been identified including such notable serovars as *S.* Typhimurium, *S.* Enteriditis, and *S.* Typhi. *S.* Typhi and *S.* Paratyphi are able to cause enteric fever and are considered as "typhoid" strains. Nontyphoid *Salmonella* infections result in non-bloody diarrhea and abdominal pain within 8 to 72 h of consumption (Montville and Matthews 2008). Nontyphoid *S. enterica* causes food poisoning by multiplying in the human small intestine and causing an inflammatory response (Doyle and Cliver 1990), and it is responsible for approximately 11% of all foodborne diseases in the United States (Scallan and others 2011; CDC 2011). Humans display symptoms of typical gastrointestinal illness such as diarrhea, fever, and abdominal cramps for as long as 5 to 7 d after consumption of *Salmonella*. Further infection is rare but can occur, especially if the pathogen enters the blood. Last year, an estimated 1027561 illnesses, 19336 hospitalizations, and 378 deaths occurred from nontyphoidal *Salmonella* (CDC 2011) at an estimated cost of $9146 per case of salmonellosis (Scharff 2010).

Prevalence: farm

Because *Salmonella* resides in the intestinal tract of swine, shedding of the bacteria by asymptomatic carriers on the farm is inevitable and is the major route for *Salmonella* infection in other animals. Numerous studies have sampled for *Salmonella* at various production stages and locations at the farm (Table 1). Pregnant sows had a greater prevalence of *Salmonella* than lactating or young sows (Funk and others 2001; Korsak and others 2003; Wilkins and others 2010). This increased prevalence during gestation occurred regardless of production system (all-in/all-out compared with 3-site system; Funk and others 2001). In contrast, a study from Ireland found that farrowing sows had a lower prevalence of *Salmonella* than dry sows (Rowe and others 2003). There is also disagreement about differences in *Salmonella* prevalence between breeding and market swine as well as differences among varying stages of market production. A Canadian study determined that 51% of sows were positive for *Salmonella* while 32% of nursery swine and 38% of grower-finisher swine were positive (Wilkins and others 2010). However, a Belgium study found that depending on whether fecal samples were obtained from pen floors or overshoes that pregnant sows may not have a greater prevalence than weaned and finishing swine (Korsak and others 2003). Several studies have found that as market swine progress from nursery or weaned pigs to growing and finishing swine that the prevalence of *Salmonella* is increased (Korsak and others 2003; Dorr and others 2009; Wilkins and others 2010). However, *Salmonella* was also found to decrease as swine progress through market production (Kranker and others 2003; Rowe and others 2003). These differences in findings may be a result of sampling location and method, production system, or management practices. Regardless, prevalence of *Salmonella* in swine is not uncommon in any of the production stages.

There are also differences in *Salmonella* spp. prevalence between different types of production systems and varying management practices. Most conventional production swine operations employ an all-in/all-out (AIAO) system involving the removal of all animals from a facility and disinfection before a new group of pigs is introduced (Funk and others 2001). Differences in production can exist in the source of animals entering the facility. In a dedicated pig flow system the same set of pigs at a breeding facility remain with their penmates through the nursery and finishing process. In contrast, a 3-site system will mix pigs from different breeding and nursery facilities. In a study comparing these 2 systems (both AIAO) the dedicated pig flow system tested positive in 9.4% of finisher floor samples and 10.9% of finishing pigs. The 3-site systems tested positive in 32.8% and 5.8% of floor and pig samples, respectively. This not only indicates differences between production practices, but it is also an indicator of potential transmission between animals due to contaminated floor samples. As reviewed by Dickson and others (2002), contaminated floors are a reasonable risk factor for transmission of *Salmonella* amongst pigs in the same pen.

In addition to floors being contaminated with *Salmonella*, various environmental samples have also tested positive for *Salmonella* (Rajic and others 2005; Dorr and others 2009). Workers' boots, empty pens, and drains can all harbor *Salmonella* cells. Because

Table 1–Prevalence of *Salmonella* at farm or holding.

Country	Reference	Species of pathogen	Sample	Production stage	*n*	# positive samples	% of samples positive
Belgium	Korsak and others 2003)	*S. enterica*	Pen fecal sample	Pregnant sows	135	11	8.1
				Lactating and young sows	378	11	2.9
				Weaned pigs	125	2	1.6
				Fattening stage	320	18	5.6
			Overshoes fecal sample	Pregnant sows	84	9	10.7
				Lactating and young sows	259	18	6.9
				Weaned pigs	82	16	19.5
				Fattening stage	236	25	10.6
Canada	Farzan and others (2006)		Feces	–	820	81	9.9
Canada	Rajic and others (2005)	*Salmonella* spp.	Environment-boots	Finishing	88	34	38.6
			Environment- empty pens	Finishing	155	18	11.6
			Environment- main drain	Finishing	85	27	31.8
			Total environment	Finishing	417	84	20.1
			Feces	Finishing	1344	192	14.3
Canada	Wilkins and others (2010)	*Salmonella* spp.	Fecal samples	Sows	200	102	51.0
				Nursery	255	81	31.8
				Grow-finish	295	113	38.3
				Total	1143	407	35.6
Denmark	Christensen and others (1999)	*S. enterica*	Pen fecal sample	–	15520	2993	19.3
Hungary	Biksi and others (2007)	*S. enterica*	Fecal samples	Finishing	186	40	21.5
	Rowe and others (2003)	*Salmonella* spp.	Pen fecal sample	Weaned 1 (3 to 10 wk)	202	22	10.9
				Weaned 2 (10 to 17 wk)	210	17	8.1
				Fattening	248	23	9.3
				Dry sow	118	11	9.3
				Farrowing sow	176	7	4.0
Netherlands	van der Wolf and others (1999)	*Salmonella* spp.	Fecal samples	Finishing herd	306	71	23.2
Spain	García-Feliz and others (2007)	*Salmonella* spp.	Floor fecal samples	Finishing	2320	290	12.5
Taiwan	Wang and others (2011)	*Salmonella* spp.	Swine fecal samples	–	440	58	26.4
			Housefly samples	–	220	86	19.5
U.S.	Bahnson and others (2006)	*S. enterica*	Feces	–	934	46	4.9
U.S.	Dorr and others (2009)	*Salmonella* spp.	Fecal samples	Late nursery	240	25	10.4
				Early finishing	180	28	11.6
				Late finishing	180	33	18.3
U.S.	Hurd and others (2003)	*S. enterica*	Antemortem fecal	Finishing	566	8	1.5
			Necropsy fecal	Finishing	281	2	0.7
			Cecal contents	Finishing	281	5	1.8
			Lymph nodes	Finishing	281	10	3.6
			Overall pig prevalence	Finishing	281	15	5.3
U.S.	Rostagno and others (2003)	*S. enterica*	Transportation trailer samples	–	144	63	43.8
			Holding pen samples	–	144	112	77.8
			Drinking water	–	24	8	33.3
Review	Fosse and others (2009)	*S. enterica*	Feces or rectal contents	–	–	–	6.4

Salmonella is capable of surviving outside the host, this environmental contamination can be an important source of contamination in swine. A total prevalence of 20.1% was found in all environmental samples collected, with boots and main drains having a greater incidence of *Salmonella* than empty pens (Rajic and others 2005). Trucks on the farm that are used to transport pigs from the nursery were also found positive for *Salmonella* (Dorr and others 2009).

Feed samples have also been well documented as being contaminated with *Salmonella* (Korsak and others 2003; Davies and others 2004a; Farzan and others 2006; Wilkins and others 2010). Fecal samples were collected from swine fed either liquid or dry feed (Farzan and others 2006). Fecal samples from liquid-fed pigs were positive for *Salmonella* 6% of the time, and 0.8% of fecal samples from dry-feeding were positive. Dry feeding systems also had higher rates of rodents on the farm and were comprised of smaller herd sizes than liquid feeding systems, which may have confounded the results as these factors may also increase *Salmonella* prevalence. Not only has liquid feed been determined to be a risk factor, but pelleted feed was also found to increase *Salmonella* prevalence in swine (Wilkins and others 2010). The practice of liquid-feeding is a fairly recent advent with only 20% of market swine in Canada under this production system; it is typically found in newer, bigger operations (Farzan and others 2006). This increase in *Salmonella* prevalence from liquid-feeding could become an issue if more production systems adopt this feeding practice, but its limited practice currently is likely not a major contributor of overall *Salmonella* prevalence in swine. In general, feed can be an additional contamination point during production if feed is mixed on the farm, if there is incomplete processing of the feed, or if postprocessing contamination occurs (Davies and others a).

Prevalence: transportation and holding

Although the farm is an important source of contamination, transportation and holding before slaughter significantly increase the prevalence of *Salmonella* in swine (Hurd and others 2002; Rostagno and others 2003; Duggan and others 2010; de Busser and others 2011). One study collected necropsy samples at the

farm and at the abattoir to determine that prevalence of *Salmonella was* increased at the abattoir by 24.5%, 11.9%, 5.5%, and 34.5% for fecal samples, cecal contents, lymph nodes, and total positive samples, respectively, compared with the farm samples (Hurd and others 2002). Environmental samples collected during transportation also had high *Salmonella* prevalence as 77.8% of slaughter holding pen samples, 43.8% of trailer samples, and 33.3% of water samples tested positive for *S. enterica* (Rostagno and others 2003). The stress of transportation and holding has an obvious effect on increasing the prevalence of the pathogen and is a potential source of cross-contamination between colonized and noncolonized pigs. If transportation and holding units are not properly disinfected, this can also be a source of cross-contamination for pigs (Hurd and others 2002; Rostagno and others 2003). This is indicated by *Salmonella* strains obtained from transportation trucks were found in the cecal contents and lymph nodes of swine at slaughter (Dorr and others 2009).

Prevalence: slaughter

Since 1998, the Food Safety and Inspection Service (FSIS) of the U.S. Dept. of Agriculture has been regularly testing for *Salmonella* in slaughter facilities to ensure that HACCP and pathogen reduction rulings are being effectively observed. The prevalence of *Salmonella* spp. in market swine decreased from 4.3% between 1998 and 2003 to 2.4% in 2010 (FSIS-USDA 2010). Although the prevalence is low, it is important to recognize the large number of swine that are harvested each year and their potential contribution to foodborne diseases.

There are several stages in the slaughter process that can be an avenue of carcass contamination or cross-contamination (Table 2). Commercial pork slaughter follows the general processing steps of stunning and exsanguination, scalding, dehairing and singeing, head removal, evisceration, and final wash. Because *Salmonella* are carried primarily in the intestinal tract of swine, contaminated fecal material initially on the carcass or released during the slaughter process are risks for *Salmonella* contamination. In a USDA study of 2 U.S. commercial slaughter facilities, 91% of prescald carcasses were positive for *Salmonella* (Schmidt and others 2012). Not only were the majority of carcasses contaminated with the bacteria, but 37% of carcasses contained between 1 and 3.9 log CFU/100 cm^2. Thus, there is opportunity for cross-contamination to processing equipment or other carcasses from exsanguination to evisceration, such as dehairing and polishing equipment, knives, and head removal (Dickson and others 2002).

Salmonella can become established on the dehairing and polishing machinery, especially if fecal material has remained on the carcass or been released from the rectum of the animal. However, scalding and singeing are known to decrease the prevalence of *Salmonella* on carcasses (Dickson and others 2002). Preevisceration carcasses were 19.1% positive with only 5% of samples containing enumerable levels of *Salmonella* which is greatly reduced compared to prescalded carcasses (Schmidt and others 2012). In addition to feces, various lymph nodes have also been documented as being a reservoir for *Salmonella* (Fosse and others 2009). Therefore, both evisceration and head removal provide an opportunity for contaminated intestinal tissue or lymph nodes to spread to other parts of the carcass or contaminate slaughter equipment especially if faulty procedures are used (Berends and others 1997). Overall, the slaughter process decreases the prevalence of *Salmonella* on the carcass (Saide-Albornoz and others 1995; Dickson and others 2002). After final rinse and carcass chilling, prevalence of *Salmonella* was found to be 3.7% (Schmidt and others 2012).

Up to 69% of *Salmonella* contamination on a carcass is a result of contaminated slaughter environment (Duggan and others 2010). Thus, it is important for workers to be aware of the potential reservoirs of the pathogen so that necessary decontamination of knives, saws, and other equipment occurs during the slaughter process. All of these reservoirs of *Salmonella* have the ability to contaminate the whole carcass and potentially reach consumers (Bonardi and others 2003; Korsak and others 2003; Davies and others b).

Prevalence: retail pork

As indicated from previous research, *Salmonella* prevalence at the farm easily translates to contamination of tissues and the carcass at slaughter. This is a major concern for food safety as contaminated carcasses are strongly associated with contaminated pork products at the retail level. Numerous studies have been conducted to determine prevalence of *Salmonella* in various retail pork products (Table 3). Cuts from the shoulder of pork carcass in Denmark were reported to have a greater prevalence of *Salmonella* contamination likely due to the inverted suspension of carcasses during slaughter allowing for pooling of pathogens in the forepart of the carcass (Hansen and others 2010). Offal was also found to have a much greater prevalence of *Salmonella* when compared to retail pork (Little and others 2008).

With both whole muscle and fresh ground product there is a likelihood of *Salmonella* contamination (Duffy and others 2001). Ground products may pose a greater risk to consumers, however, due to the spread of contamination throughout the product during the grinding process. Also, re-packaging or grinding of the pork product at the retail store can be a source of *Salmonella* contamination (Duffy and others 2001). Another ground sausage product sold fresh that poses a health concern for *Salmonella* infection is chorizo. Several studies have determined that this raw product is a carrier for the pathogen and due to the dark color of the sausage can easily be undercooked by consumers (Escartin and others 1999; Hajmeer and others 2006).

The type of retail or processing facility can impact the prevalence of *Salmonella*. Butcher shops were found to have a higher prevalence of *Salmonella* compared to supermarkets (Hansen and others 2010). This is an indicator that differences in hygiene, likelihood of cross-contamination, and handling of product vary between types of retail stores and affect *Salmonella* prevalence. Moreover, fresh ground sausage from hot-boning sow and boar plants, slaughter and fresh packing plants, and further-processing plants had a *Salmonella* spp. prevalence of 10%, 7.5%, and 0%, respectively (Duffy and others 2001). Lower prevalence in further-processing plants is likely due to heat treatment and thermal inactivation of *Salmonella*.

Prevalence: ready-to-eat products

Between 2005 and 2008, FSIS sampled different ready-to-eat (RTE) meat products and determined that of all species tested, pork products accounted for 60% of the positive *Salmonella* samples, which is greater than the prevalence in chicken and beef combined (Mamber 2010). Because *Salmonella* is killed with proper cooking, the presence of *Salmonella* in RTE products is most likely caused by the following risk factors: under-processing or under-cooking of the product, contamination from raw materials, contamination from food handlers, or contamination from animals inside the facility, such as birds, rodents, and insects, or contaminated material carried into the facility by humans on shoes or clothing (Mamber 2010). Contamination from raw materials

Table 2–Prevalence of *Salmonella* at slaughter.

Country	Reference	Species of pathogen	Location	Sample	*n*	# positive samples	% of samples positive
Belgium	de Busser and others (2011)	*Salmonella* spp.	Lairage	Overshoes	61	28	45.9
			Slaughter	Oral cavity swab	278	39	14.0
			Slaughter-after polishing	Carcass swab	226	25	11.1
			Slaughter-after Splitting	Carcass swab	226	31	13.7
			Slaughter	Duodenum contents	226	26	11.5
			Slaughter	Ileum contents	226	52	23.0
			Slaughter	Rectum contents	226	30	13.3
			Slaughter	Mesenteric lymph nodes	226	40	17.7
			Slaughter-after forced chilling	Carcass swab	226	5	2.2
Belgium	Korsak and others (2003)	*S. enterica*	Slaughter	Large intestine contents	186	88	47.3
				Carcass swabs	152	17	11.2
Belgium	Nollet and others (2004)	*S. enterica*	Slaughter	Gut lymph nodes	1860	62	33.2
Canada	Lammerding and others (1988)	*Salmonella* spp.	Slaughter	Carcass	596	104	17.5
				Neck muscle	596	67	11.2
				Portal lymph nodes	278	13	4.6
				Mesenteric lymph nodes	317	43	14.2
Denmark	Christensen and others (1999)	*S. enterica*	Slaughter	Seroprevalence	9654	2730	28.3
Denmark	Sorensen and others (2004)	*S. enterica*	Slaughter	Cecal lymph nodes	1666	180	10.8
Germany	Käsbohrer and others (2000)	*Salmonella* spp.	Slaughter	Fecal swab	11930	445	3.7
				Lymph nodes	11941	391	3.3
				Carcass swab	11942	564	4.7
Germany	Nowak and others (2007)	*Salmonella* spp.	Slaughter	Jejunum/lymph nodes	383	63	16.5
				Tonsils	129	20	15.5
Germany	Steinbach and others (2002)	*Salmonella* spp.	Slaughter	Fecal sample	11960	44	3.7
				Lymph nodes	11960	396	3.3
				Seroprevalence	11896	1178	9.9
Ireland	Boughton and others (2007)	*Salmonella* spp.	Holding – beginning, short holding	Pen floor samples	60	2	3.0
			Holding- end, long holding	Pen floor samples	60	31	52.0
Ireland	Duggan and others (2010)	*Salmonella* spp.	Slaughter	Cecal contents	193	87	45.1
			Slaughter	Rectal contents	193	59	30.6
			Prechill	Carcass swab	193	29	15.0
			Postchill	Carcass swab	193	5	2.6
Ireland	McDowell and others (2007)	*Salmonella* spp.	Slaughter	Cecal contents	513	161	31.4
			Slaughter- postevisceration	Carcass swab	507	203	40.0
			Slaughter	Muscle sample	513	59	11.5
Italy	Bonardi and others (2003)	*Salmonella* spp.	Slaughter	Intestinal contents	150	55	36.7
				Tonsils	150	8	5.3
				Carcass	150	9	6.0
Portugal	Vieira-Pinto and others (2005)	*Salmonella* spp.	Slaughter	Ileum contents	101	14	13.9
				Ileocolic lymph nodes	101	19	18.8
				Mandibular lymph nodes/carcass	101	13	12.9
				Tonsils	101	10	9.9
Spain	Vico and others (2011)	*Salmonella* spp.	Slaughter	Mesenteric lymph nodes	1997	625	31.3
U.K.	Davies and others (2004)	*Salmonella* spp.	Slaughter	Cecal contents	2509	578	23.0
				Carcass swab	2509	134	5.3
				Neck muscle	2403	857	35.7
		*S.*Typhimurium	Slaughter	Cecal contents	2509	278	11.1
				Carcass swab	2509	52	2.1
U.K.	Milnes and others (2008)	*Salmonella* spp.	Slaughter	Cecal contents	529	124	23.4
U.S.	Bahnson and others (2006)	S.*enterica*	Slaughter	Cecal content	942	164	17.4
				Distal colonic content	937	37	3.9
				Ileocolic lymph nodes	941	136	14.5
U.S.	Dorr and others (2009)	*Salmonella* spp.	Slaughter	Cecal contents	180	82	45.5
				Mesenteric lymph nodes	180	103	57.2
U.S.	Hurd and others (2002)	*S. enterica*	Slaughter	Necropsy fecal	286	72	25.2
				Cecal contents	286	39	13.6
				Lymph nodes	286	26	9.1
				Pig prevalence	286	114	39.9
U.S.	Hurd and others (2005)	*S. enterica*	Slaughter – solid lairage floors	Cecal contents	209	151	72.4
				Fecal contents	200	48	23.8
				Lymph node	210	73	34.8
			Slaughter – slatted lairage floors	Cecal contents	209	132	63.3
				Fecal contents	197	49	24.7
				Lymph node	210	65	30.9
			Slaughter – slatted lairage floors and short holding time	Cecal contents	206	109	52.9
				Fecal contents	188	60	31.9
				Lymph node	210	75	35.7
U.S.	Rostagno and others (2003)	*S. enterica*	Slaughter	Cecal contents	720	220	30.6
				Ileocecal lymph nodes	720	96	13.3
U.S.	Saide-Albornoz and others (1995)	*Salmonella* spp.	Slaughter- after singeing	Ham and loin carcass surface	270	12	4.4
			Slaughter- after final rinse	Ham and loin carcass surface	270	3	1.1
			Slaughter- 24 hour chill	Ham and loin carcass surface	270	1	0.4
U.S.	Schmidt and others (2012)	*Salmonella* spp.	Slaughter- prescald	Carcass swab	1520	1386	91.2
			Slaughter-preevisceration	Carcass swab	1520	291	19.1
			Slaughter- postchill	Carcass swab	1520	56	3.7
Review	Fosse and others (2009)	*S. enterica*	Slaughter	Digestive carriage	–	–	15.5
				Intestinal contents	–	–	24.6
				Tonsils and lymph nodes	–	–	10.9
				Seroprevalence	–	–	36.7

Table 3–Prevalence of *Salmonella* spp. at retail.

Country	Reference	Location	Sample	*n*	# positive samples	% of samples positive
Denmark	Hansen and others (2010)	Butcher shop-2002	Shoulder	324	9	2.8
			Middle	410	7	1.7
			Hind	291	2	0.7
		Butcher shop- 2006	Shoulder	73	7	9.6
			Middle	94	6	6.4
			Hind	84	8	9.5
		Supermarket- 2002	Shoulder	1206	18	1.5
			Middle	1237	12	1.0
			Hind	1030	4	0.4
		Supermarket-2006	Shoulder	8	6	3.8
			Middle	160	6	2.7
			Hind	226	4	1.8
Ireland	Duffy and others (1999)	Retail	Retail pork	20	9	9.9
Korea	Hyeon and others (2011)	Retail store	Retail pork	56	5	8.9
Mexico	Escartin and others (1999)	Butcher shop	Chorizo	60	53	89.0
		Processing plant	Chorizo	40	2	5.0
		Butcher shop	Chorizo	40	31	78.0
U.K.	Little and others (2008)	Retail	Muscle tissue	1309	25	1.9
			Offal (liver, heart, kidney, tripe)	131	31	23.6
U.S.	Duffy and others (2001)	Hot-boning, sow & boar plant	Sausage	40	4	10.0
		Slaughtering and fab plant	Sausage	40	3	7.5
		Further processing	Sausage	40	0	0
		Retail store	Store-ground fresh pork/pork sausage	96	7	7.3
			Prepackaged ground pork/pork sausage	96	12	12.5
			Whole-muscle, store-packaged pork	96	8	8.3
			Whole-muscle, enhanced pork	96	10	10.4
U.S.	Zhao and others (2001)	Retail	Raw meat	209	5	2.6
		Supermarket chain	Meat products	825	25	3.0
Review	Mataragas and others (2008)	Processing plant	–	373988	6027	1.6
		Retail	Raw products	2943	249	8.5
			RTE products	4088	148	3.6

includes the spices and other ingredients used to make the final product. This problem became apparent when an outbreak of *Salmonella* Montevideo occurred in 2010. Almost 300 people were affected and 26% were hospitalized when *Salmonella*-contaminated red pepper and black pepper were used to make Italian delicatessen meats and sausages. The manufacturer of the RTE meat was forced to recall 30 products totaling approximately 1.4 million pounds of meat (CDC 2010). Although this outbreak was not a result of contaminated raw pork, it is an indicator of potential contamination of pork products from other sources and the interventions needed to control these risk factors.

Intervention: preharvest

Because *Salmonella* is carried in the live animal, interventions against the pathogen begin at the farm. In addition to good management and biosecurity practices, vaccination and antibiotics are also commonly delivered to swine to reduce *Samonella* prevalence and levels. A commercially available *Salmonella* Choleraesuis vaccine was delivered to 3 and 16 wk old pigs, which decreased the prevalence of the pathogen in the lymph nodes by 6.6% (Maes and others 2001). *Salmonella* Choleraesuis vaccination is also capable of cross-protecting the animal against other strains of the bacteria, such as *Salmonella* Typhimurium (Maes and others 2001). Regardless of whether the vaccination is given orally or in the water, *Salmonella* Typhimurium levels were reduced. Oral vaccination also resulted in higher average daily weight gains and improved overall health through reductions in diarrhea. Moreover, vaccination delivered in the water supply reduced *Salmonella* in tonsil, ileo-cecal junction, and mesenteric lymph node samples compared to controls during necropsy (Charles and others 2000).

Feeding probiotics or prebiotics can also decrease the prevalence of *Salmonella* in swine. These feed additives are thought to alter the gut microbiota of the animal causing a reduction of harmful bacteria. The feeding of Ferlac-2 and Flavomycin probiotics decreased the prevalence of *Salmonella* Typhimurium in the mesenteric lymph nodes; however, these feed additives were not effective at reducing the shedding of the pathogen (Letellier and others 2000). This same researcher also evaluated the effects of a SC54 live attenuated *S. choleraesuis* vaccine and found reductions of *Salmonella* Typhimurium in the ileum and feces. Unlike probiotics, a reduction was not observed in the mesenteric lymph nodes with the use of vaccination. Perhaps a combination of probiotics and vaccination would give the animal the greatest reduction of *Salmonella* by reducing the prevalence of shedding and the prevalence of the pathogen in the lymph nodes (Letellier and others 2000).

Phage therapy has been used in the beef industry to reduce *E. coli* on carcasses, and this technology has recently been applied to swine. Phages are viruses that only infect bacteria. Typically, the host range for phages is at the species or subspecies level. By inoculating the pig orally with an encapsulated phage that infects *S.* Typhimurium, a reduction of *S.* Typhimurium occurred in ileal, cecal, and tonsil samples (Wall and others 2010). There was also a reduction in other serovars of *Salmonella*, indicating that some cross-reactivity did occur. Unfortunately, this technology is only effective for a short period and requires delivery just prior to transportation and holding (Wall and others 2010). Administration of the bacteriophage was also been tested in the feed. This is a more economical method of delivery, and it was determined to have the same effectiveness in decreasing *Salmonella* shedding compared to oral administration (Saez and others 2011). However, both these studies involving oral and feed-based administration of phages were conducted on small samples. Thus, further research is needed to better understand whether phage therapy is an effective means of controlling *Salmonella* contamination of healthy animals during transportation and holding.

Because the slaughter holding environment can be a potential source of contamination for swine, changes in holding procedures have been considered. Reducing the amount of time that animals are in holding or the hygienic conditions of the holding environment have been considered. Using a shorter holding (15 to 45 min against 4 h) was found to reduce the prevalence of *Salmonella* in various slaughter tissues (Hurd and others 2005). However, this reduction in holding is limited by the necessary processing, inspection, and movement of pigs by plant personnel. Moreover, holding is a method to reduce stress of swine and provide desirable meat quality. This study found that meat color and water holding capacity, which are both based on muscle pH and postmortem glycolysis, was negatively affected by the shorter holding period. In contrast, using slatted flooring instead of solid flooring not only reduced levels of *Salmonella* during holding (68% against 95%) but had no negative affect on meat quality (Hurd and others 2005).

Intervention: postharvest

Interventions for *Salmonella* in raw and RTE pork have also been developed. Organic acids are commonly used and known to be effective at reducing the prevalence of the pathogen. Typically, weak acids like organic acids are used in food systems as uncharged molecules that can freely cross the bacterial membrane (Brul and Coote 1999). This undissociated state of the acid occurs at a significant concentration when the pH of the food is near or below the pKa of the organic acid. The internal pH of bacterial cells is higher, which results in the release of a proton from the uncharged acid once it has entered the cell. The release of these protons can inhibit growth of the bacteria in a variety of ways, including disruption of the membrane, metabolic reactions, or intracellular pH homeostasis (Brul and Coote 1999; Mani-López and others 2012). Organic acids can also decrease bacterial growth by lowering the pH to create an unfavorable environment in which certain metabolic functions are reduced (Mani-López and others 2012). Thus, organic acids have been used as carcass spray washes to decrease various pathogens, including *Salmonella* (Epling and others 1993; Reynolds 2003). Peroxyacetic and lactic acid carcass washes reduced *Salmonella* by 50% and 66%, respectively (Reynolds 2003). Although organic acids are effective at reducing bacteria on pork skin when used after carcass washing, this reduction was not greater than hot water washing alone (Eggenberger-Solorzano and others 2002). Organic acid treatment combined with hot water washing, however, further decreased aerobic plate count levels of bacteria when the pork carcasses were skinned.

Organic acids can also be used on cuts of pork. Acetic acid combined with lactic acid or salt was effective at decreasing the prevalence of *Enterobacteriaceae* in vacuum-packaged pork chops. However, the acid treatment caused an increased purge loss and was detrimental to meat color (Mendonca and others 1989). Other acids such as citric, propionic, succinic, tartaric, and malic have also been considered in applications to meat products (Mani-López and others 2012). Different acids have the ability to restrict the growth of bacteria at varying levels of pH. Acetic and propionic acids are known to be more restrictive, whereas citric acid may still allow the growth of *Salmonella* at pH 5. Despite the effectiveness of using organic acids, there are numerous regulatory restrictions in place that limit the level used in food products. The use of supercritical carbon dioxide in combination with lactic acid has also been found to be effective at decreasing levels of *Salmonella* in boneless pork loins (Choi and others 2009). Certain packaging strategies are also effective intervention for *Salmonella*. Storage at 2 °C for 36 d in vacuum-packaging decreased prevalence of *Salmonella* in boneless pork loins compared to carcass samples during slaughter (Saide-Albornoz and others 1995).

Campylobacter

Campylobacter, like *Salmonella*, is a Gram-negative bacterium; however, it requires low levels of oxygen for growth (Bolton and Coates 1983; Kaakoush and others 2007). This bacterium grows in a narrow temperature range between 30 and 47 °C, and minimum pH for growth is 5.8 (Doyle 1990). A unique metabolic characteristic of *Campylobacter* is the lack of the enzyme 6-phosphofructokinase, which prevents the bacteria from using sugars as an energy source. Instead, *Campylobacter* uses compounds such as fumarate, nitrate, or sulfite as an energy source. These compounds are common metabolic end products of other bacteria found in the intestines of mammals and birds. *Campylobacter* is also capable of catabolizing amino acids. The pH, microaerophilic oxygen levels, temperature, and rich energy sources found in the intestinal tract of mammals make it an ideal environment for *Campylobacter* to thrive (Anderson and others 2009). Because *Campylobacter* spp. are commonly found in meat-producing livestock, the specific growth conditions have also been studied in meat. In addition to growing at typical meat pH (5.8), certain strains of *Campylobacter* have also been reported to grow better on meat at a higher pH of 6.4. Most strains are thermally inactivated at 50 °C (Gill and Harris 1982). Although this foodborne pathogen is typically associated with poultry, it has been well documented, particularly *Campylobacter coli*, as being present as part of the normal gut microbiota of swine (Schuppers and others 2005; Fosse and others 2009; Farzan and others 2010). Similar to *Salmonella*, the common carriage of *Campylobacter* in the intestines of livestock species is generally asymptomatic and does not present a problem for the animal.

Campylobacter is the leading cause of human gastrointestinal illness from a zoonotic source with 2.45 million people a year suffering from *Campylobacter*-causing illness, of which 80% of the cases are foodborne (Mead and others 1999). Thus, *Campylobacter* causes 9% of all foodborne diseases in the United States (Scallan and others 2011). The economic burden of this disease in the United States is estimated at $8901 per case (Scharff 2010). *Campylobacter* infection is typically asymptomatic in the livestock, which it inhabits; however, in some cases, it can cause abortion in animals (Milnes and others 2009). In humans, the pathogen causes a typical gastrointestinal illness with symptoms of diarrhea, abdominal pain, vomiting, fever, or bloody stool (Doyle 1990). Also, in rare situations, *Campylobacter* may cause Guillain–Barré syndrome (Nachamkin and others 1998). The majority of foodborne-related campylobacteriosis is caused by *C. jejuni*, which is most commonly associated with poultry; however, low levels have been found in swine (Schuppers and others 2005). Swine are a major carrier for *Campylobacter coli*, which can also contribute to foodborne illnesses in humans.

Prevalence: farm, transportation, and holding

Pigs become colonized with *Campylobacter* less than 1 week after birth. Several researchers have determined that the sow is the main source of contamination for the piglets, but neighboring sows and piglets can be contributing risk factors (Weijtens and others 1997; Alter and others 2005). Risk factors for increased prevalence of *Campylobacter* in sows included a greater number of sows on the farm ($n > 130$), individual housing of sows, and warmer months (Denis and others 2011). Surprisingly, the following criteria were not risk factors of *Campylobacter* colonization of sows: stage of

sampling (gestation area, maternity, or service area), type of floor, management system, feed type, origin of feed, and use of antibiotic treatment.

As swine progress through the live production stages, the prevalence of *Campylobacter* generally increases (Table 4). Typically, weaned piglets have less prevalence of the bacteria than finishing swine or sows with one study demonstrating a 15% increase of infected pigs from growing stage (14 wk of age) to finishing stage (22 wk of age) (Farzan and others 2010). By the time swine reach the finishing stage, the majority of pigs are positive for *Campylobacter* (Schuppers and others 2005). Across all production stages, *C. coli* is more prevalent than *C. jejuni* in swine. More than 90% of pigs that tested positive for *Campylobacter* spp. were isolated as *C. coli* (Schuppers and others 2005; Fosse and others 2009; Farzan and others 2010).

The farm environment does not greatly contribute to the prevalence of *Campylobacter* in swine; however; the pathogen can persist in the environment (Alter and others 2005). A comprehensive German study demonstrated that a small percentage of flies and rodents were positive for *Campylobacter*. Minimal water samples also tested positive, and this is likely a result of cross-contamination from feces. Cleaning of pens and facilities was effective at decreasing the prevalence of the bacteria from 9.2% to 1.6%. Although a significant decrease was observed, cleaning is not 100% effective and poor cleaning can be a contributing point of contamination for swine (Alter and others 2005).

Transportation of pigs to slaughter facilities may also affect *Campylobacter* prevalence. Under conditions of simulated lairage, fasting for 48 h increased *Campylobacter* levels from 5 to 7.2 $\log_{10}$ CFU/g. This coincided with an increased cecal pH, which would reduce microbial competition and allow *Campylobacter* to proliferate rapidly (Harvey and others 2001). Increasing time in lairage decreased the shedding of *Campylobacter* spp. due to reduced stress, which was indicated by lower blood cortisol and lactate levels (Warriss and others 1998; Milnes and others 2009). Others demonstrated that transportation does not affect *Campylobacter* prevalence; however, these findings may be a result of intermittent shedders not detected when sampling during a short time period (Alter and others 2005).

Prevalence: slaughter

Campylobacter presence at the farm translates to prevalence of the pathogen during the slaughter process (Table 5). Similar to the prevalence of *C. coli* in live swine, this species dominates *Campylobacter* spp. found during slaughter. In a recent study of *Campylobacter* spp. in finishing swine, digestive carriage, gastric contents, and tonsils were found to have a prevalence of *Campylobacter* at 71%, 51.5%, and 24.7%, respectively (Fosse and others 2009). However, *Campylobacter* was reduced on the carcass by scalding, dehairing, singeing, and polishing (Pearce and others 2003). Evisceration provides an opportunity for carcass contamination to occur, but any contamination that occurs is likely reduced by chilling. *Campylobacter* is sensitive to drying and low temperature, which makes chilling a very effective pathogen intervention step against *Campylobacter* spp. This was observed in a study tracking the prevalence of *Campylobacter* during slaughter which found a reduction from 7% to 0% before and after chilling on pork carcasses (Pearce and others 2003).

Prevalence: retail pork

Although *Campylobacter* levels are low in retail pork products, there is still potential for contamination and ingestion by the consumer (Table 6). Much like *C. coli* is the dominant species of the bacteria in swine, the *Campylobacter* species in 90% of the positive retail pork samples from a controlled study in Ireland was identified as being *C. coli* (Whyte and others 2004). Prevalence of the bacteria was highest in ground pork and pork sausage from hot-boning, sow and boar plants with 12.5% positive samples (Duffy and others 2001). In further processing plants, *Campylobacter* spp. prevalence was 7.5%, while slaughtering and fresh packing plants did not have any positive *Campylobacter* ground pork or pork sausage samples. According to a small retail study in Italy, *C. jejuni* carriage in pigs is much lower than *C. coli* (Sammarco and others 2010). Moreover, the researchers suggest that retail pork that is contaminated with *C. jejuni* may be from cross-contamination with other meat sources or the environment. This is especially concerning as this indicates potential contamination after processing or cooking of pork products.

Intervention: preharvest

Because *Campylobacter* can be prevalent in swine throughout the production process, intervention against the pathogen begins at the farm. Feed additives with antibacterial properties can be given to swine at different production stages to decrease the prevalence of *Campylobacter*. Carbadox and copper sulfate are dietary additives which are commonly administered to nursery swine as growth-promoting agents. Nursery pigs fed a diet containing Carbadox and copper sulfate had decreased shedding of *Campylobacter* in the feces, but no reduction was seen in pigs fed larch extract, which is a nonresidual alternative to these compounds. This study also reported that the Carbadox and copper sulfate combination decreased feed efficiency and increased shedding of *Enterobacteriaceae*, such as *Salmonella*. These results bring up an important issue in feeding dietary additives to swine. It is important for researchers to examine the effects that these compounds will have on different pathogens as well as growth performance of the animal. As observed in this study, feed efficiency may suffer from the use of antimicrobial feed additives (Wells and others 2010).

A novel intervention technique is the use of deaminase inhibitors, such as thymol or diphenyliodonium chloride (DIC), which both inhibit amino acid catabolism and therefore decrease the survival of *Campylobacter*. These compounds are currently not in commercial use, but their potential intervention use has been explored. *In vitro*, DIC was found to be more effective than thymol at reducing *Campylobacter* survival. However, thymol, as an organic product, could be more appealing to producers, especially in organic production systems (Anderson and others 2009). These compounds were fed to weaned pigs and did not negatively impact growth or feed intake. Unfortunately, absorption of both DIC and thymol was a challenge *in vivo*, and it is thought that the compounds were degraded before inhibition of *Campylobacter* occurred. Potential solutions to this problem include encapsulation of the compounds or using thymol derivatives that are more resistant to degradation in the stomach. If these compounds were able to reach the intestine without being degraded, they would appear to be a viable option for reducing colonization of *Campylobacter* and cross-contamination during transportation and lairage (Anderson and others 2009).

A Gram-positive bacterium, *Brevibacillus texasporus*, has been investigated for its ability to reduce *Campylobacter* in weaned piglets. This nonpathogenic species produces BT/TAMUS 2032 (BT), which is a combination of 13 AA formed into cation amphipathic peptides. Weight gain was improved in pigs fed BT peptide, and a reduction, though not reaching significance, was achieved in *Campylobacter* counts. The researchers believe that because the

Table 4–Prevalence of *Campylobacter* at farm.

Country	Reference	Species of pathogen	Sample	Production stage	*n*	# positive samples	% of samples positive
Canada	Munroe and others (1983)	*C. coli*	Feces	Healthy pigs	144	100	69.4
				Diarrheic pigs	59	18	30.5
Germany	Alter and others (2005)	*C. coli*	Feces	Brood sows	63	32	50.8
				Newborn piglets	0	30	0
				Weaned piglets (1 wk)	586	192	32.8
				Weaned piglets (3 wk)	580	238	41.0
				Nursery unit (4 wk)	565	320	56.6
				Finishing (12 wk)	588	337	60.4
				Finishing (24 wk)	590	394	66.8
			Feedstuff	–	547	0	0
			Tap water	–	35	0	0
			Water trough	–	97	1	1.0
			Feed trough	–	91	0	0
			Rodents	–	43	1	2.3
			Cat feces	–	7	0	0
			Flies	–	436	2	0.5
			Rubber boots	–	6	0	0
			Dust	–	78	0	0
			Feces of wild birds	–	8	0	0
			Pen before cleaning	–	65	6	9.2
			Pen after cleaning	–	61	1	1.6
			Total environment	–	1417	11	0.7
			Feces	Before transport to slaughter	330	261	79.1
				After transport to slaughter	330	258	78.2
Netherlands	Weijtens and others (1999)	*Campylobacter* spp.	Feces	Finishing (10 to 25 wk)	55	48	87.3
				Finishing (10 wk)	8	7	87.5
				Finishing (13 wk)	8	8	100
				Finishing (17 wk)	7	5	62.5
				Finishing(19 wk)	8	7	87.5
				Finishing (22 wk)	8	8	100
				Finishing (25 wk)	8	5	62.5
	Weijtens and others (1997)	*Campylobacter* spp.	Feces	Sows- 1 wk before delivery	10	9	90.0
				Sows- 1 wk after delivery	10	10	100
				Sows- 4 wk after delivery	10	10	100
				Sows- 8 wk after delivery	9	9	100
				Piglets- 1 wk after delivery	60	29	48.3
				Piglets- 4 wk after delivery	60	52	86.7
				Piglets- 8 wk after delivery	60	55	91.7
Switzerland	Schuppers and others (2005)	*Campylobacter* spp.	Feces	Finishing	256	245	95.7
		C. coli	Feces	Finishing	256	236	92.2
		C. jejuni	Feces	Finishing	256	3	1.2
U.S.	Gebreyes and others (2005)	*Campylobacter* spp.	Feces	Total	292	163	55.8
Review	Fosse and others (2009)	*Campylobacter* spp.	Feces	Finishing	–	–	65.5

piglets were positive for *Campylobacter* before BT treatment began, the compound was not as effective. Perhaps giving the treatment before piglets became infected would allow BT to significantly reduce the onset of *Campylobacter* infection. Longer treatment duration could also give BT adequate time to alter the gut flora of the animal (Genovese 2010).

Short-chain nitro compounds have also been examined for their potential use to decrease *Campylobacter* through inhibition of fermentation. An *in vitro* study was conducted to compare the effectiveness of several nitro compounds against the pathogen (Horrocks and others 2007). Nitro-alcohols were most effective against *C. jejuni*, and 2-nitro-methyl-propionate was not effective against *C. coli*. Reductions in *Campylobacter* spp. were observed through the use of 2-nitro-1-proponal and nitroethane, which achieved 1.16 $\log_{10}$ and 3.92 $\log_{10}$ reductions, respectively. Because these compounds were only tested *in vitro*, their practical use in swine remains unclear. Although these compounds seem promising, it is also possible that *Campylobacter* levels were decreased because the nitro compounds either created a nonculturable state of the bacteria or the agar limited the recovery (Horrocks and others 2007).

In addition to the variety of dietary feed additives that have been explored for use as preharvest intervention, *Campylobacter*–free breeding has also been attempted. There are numerous challenges with this strategy such as strict hygienic practices and high cost of production; however, significant decreases in the prevalence of the pathogen in populations of pigs have been achieved in an extremely controlled study in Denmark (Weijtens and others 2000). Moreover, specific pathogen free breeding typically only eliminates one pathogen and does not ensure elimination of other pathogenic organisms.

Intervention: postharvest

Although typical slaughter procedures are effective at decreasing *Campylobacter* on pork skin, other methods of intervention have been investigated to ensure reduction. By adding 0.05% hydrogen peroxide every 30 min to the scalding water, mesophilic and psychrophilic bacteria were reduced on the carcass surface (de Mello and Roca 2009). Lactic acid spray was also effective at decreasing *Campylobacter* spp. on carcasses. A 2% lactic acid spray decreased the prevalence of the pathogen from 2% to 0% on the shoulder and from 6% to 1% on the ham (Epling and others 1993).

Carcass chilling is a routine aspect of pork production and can be an effective means for *Campylobacter* intervention. In an evaluation of chilling on *Campylobacter* spp., 7.4% of prechilled carcasses were positive for the bacteria. Chilling by conventional methods or by blast-chilling were equally effective at decreasing the prevalence to 0% (Cutter 2003). Blast-chilling results in less

Table 5–Prevalence of *Campylobacter* at slaughter.

Country	Reference	Species	Sample	Production/ slaughter stage	*n*	# positive samples	% of samples positive
Denmark	Nielsen and others (1997)	*Campylobacter* spp.	Fecal sample	–	316	145	46
Norway	Nesbakken and others (2003)	*Campylobacter* spp.	Tonsils	–	24	16	66.7
			Submaxillary lymph nodes	–	24	0	0
			Mesenteric lymph nodes	–	24	5	20.8
			Stomach	–	24	19	79.2
			Ileum	–	24	22	91.7
			Cecum	–	24	24	100
			Colon	–	24	24	100
			Feces	–	24	24	100
			Carcass surface site- ham	–	24	6	25.0
			Carcass surface site- pelvic duct	–	24	13	54.2
			Carcass surface site- kidney region	–	24	5	20.8
			Carcass surface site-medial neck	–	24	4	16.7
			Lymphoid tissues	–	72	23	31.9
			Intestinal tract contents	–	120	115	95.8
			Carcass surface sites	–	96	35	36.5
			Total slaughter samples	–	288	173	60.1
Sweden	Lindblad and others (2007)	Thermophilic *Campylobacter*	Carcass swab	–	541	6	1.0
U.K.	Milnes and others (2008)	Thermophilic *Campylobacter*	Cecal contents	–	528	366	69.3
U.S.	Stern (1981)	*C. jejuni*	Carcass swab	–	58	22	37.9
			Fecal sample	–	38	33	86.8
U.S.	Pearce and others (2003)	*Campylobacter* spp.	Fecal sample	–	30	30	100
			Carcass swab	Postkill	30	10	33.0
			Carcass swab	Postpolish	30	0	0
			Carcass swab	Prechill	30	2	7.0
			Carcass swab	Postchill	30	0	0
			Colon contents	–	60	48	80.0
			Slaughter equipment	–	42	2	4.8
			Fabrication equipment	–	30	1	3.0
			Total tested	–	282	93	33.0
		C. coli	Carcass swab	Postkill	15	11	73.3
			Colon contents	–	69	47	68.1
			Carcass swab	Prechill	2	2	100
			Colon contents	–	112	89	79.5
			Slaughter equipment	–	2	1	50.0
			Fabrication equipment	–	2	2	100
			Total tested	–	202	151	74.8
U.S.	Gebreyes and others (2005)	*Campylobacter* spp.	Carcass swab	Indoor and outdoor ABF[a]	254	66	26.0
Review	Fosse and others (2009)	*Campylobacter* spp.	Digestive carriage		–	–	71.0
			Gastric contents		–	–	51.5
			Intestinal contents		–	–	76.6
			Tonsils and digestive		–	–	24.7
			Serological prevalence		–	–	81.2

[a] Antibiotic free.

evaporative weight loss and less prevalence of pale soft exudative (PSE) pork, and reduced cooler space requirements (Jones and others 1987). Because blast-chilling is able to effectively reduce *Campylobacter* and has economic advantages, packers can use this method with food safety and meat quality assurance.

The most common intervention for *Campylobacter* in pork is thermal treatment. Cooking meat will kill *Campylobacter* because thermal activation occurs at 50 °C. However, antimicrobial compounds applied to retail meats are an alternative method to reduce *Campylobacter*. Total of 96 oils and 23 oil compounds were analyzed for their ability to reduce the bacteria *in vitro* (Friedman and others 2002). Several oils were very effective antimicrobials against *C. jejuni* including carrot seed, celery seed, marigold, ginger root, gardenia, orange bitter, patchouli, cedarwood, mugwort, and spikenard. The oil compounds carvacrol, cinnamaldehyde, thymol, geranyl acetate, benzaldehyde, perillaldehyde, carvone R, eugenol, citral, and estragole were the most effective against *C. jejuni*. The effects of these compounds were also studied on other pathogens, and *C. jejuni* was more sensitive to the antimicrobial agents than the other pathogens tested. This is most likely due to the narrow growth conditions of this pathogen, its unique metabolic pathway, and cellular membrane structure (Friedman and others 2002).

Packaging techniques of whole-muscle cuts can affect the survival of *Campylobacter* spp. *C. jejuni* was able to survive more effectively in oxygen-impermeable barrier bags compared to aerobic storage or vacuum-packaging with commercial barrier bags. Other nonpathogenic bacteria on the meat are able to use up oxygen, making the environment more microaerophilic and suitable for *Campylobacter* (Balamurugan and others 2011). While vacuum-packaging is effective at slowing growth of other pathogens, this is not an effective intervention against *Campylobacter* spp.

A variety of intervention strategies have been tested for *Campylobacter*; however, many of these strategies have been explored in poultry and have not been studied in swine (Horrocks and others 2009). Bacteriophages, bacteriocins, and electron irradiation are a few intervention strategies that might have potential use with live pigs or pork products.

T. gondii. In addition to bacteria, parasites are known to infect swine and cause foodborne disease in humans. One of these parasites, *T. gondii*, is a coccidian parasite which causes an intracellular infection in the host. Total of 3 infective stages exist during the

Table 6–Prevalence of *Campylobacter* in retail meat.

Country	Reference	Location	Species	Sample	*n*	# positive samples	% of samples positive
Ireland	Whyte and others (2004)	Retail	*Campylobacter* spp.	–	197	10	5.1
Italy	Sammarco and others (2010)	Retail	*C. jejuni*	Pork steaks	106	3	2.8
			C. coli	Pork steaks	106	3	2.8
			Campylobacter spp.	Pork steaks	106	6	5.7
U.K.	Little and others (2008)	Retail	*Campylobacter* spp.	Muscle tissue	1309	66	5.0
				Offal (liver, heart, kidney, tripe)	131	24	18.3
U.S.	Duffy and others (2001)	Retail	*C. jejuni* and *C. coli*	Whole muscle, store-packaged pork	96	1	1.0
				Whole muscle, enhanced pork	96	1	1.0
				Store-ground fresh pork/pork sausage	96	0	0
				Prepackaged ground pork/pork sausage	96	3	3.1
				Total samples	384	5	1.3
		Processing plant	*C. jejuni* and *C. coli*	Ground pork/ pork sausage	120	8	6.7
U.S.	Zhao and others (2001)	Retail	*Campylobacter* spp.	Raw meat	181	3	1.7
		Supermarket chain	*Campylobacter* spp.	Meat products	719	164	22.8
		Retail	*C. jejuni*	Raw meat	387	2	0.5
			C. coli	Raw meat	298	9	3.0
			Campylobacter spp.	Raw meat	722	11	1.5
New Zealand	Wong and others (2007)	Retail outlets	*C. jejuni* and *C. coli*	Raw pork	230	21	9.1
			C. jejuni	Raw pork	230	18	7.8
			C. coli	Raw pork	230	1	1.1

life cycle of the parasite: sporozoites, tachyzoites, and bradyzoites. Felids are major carriers of the *T. gondii* and the only animal that fecally shed sporozoites as oocysts, which are capable of reproducing (Cliver 1990). These cysts can remain in the environment and can be transmitted to wildlife, livestock, and humans through ingestion of contaminated tissue, soil, or water. Tachyzoites and bradyzoites, which are non-reproducing stages of *T. gondii*, are found in the tissues of infected animals, especially in muscle, brain, and heart tissues. *T. gondii* is a concern for humans, and a large percentage of people have antibodies for the parasite indicating that these people ingested the pathogen at some point during their lifetime. The immunocompromised, especially AIDS patients, are known to be particularly susceptible to toxoplasmosis. Pregnant women are also at high risk for *T. gondii* infection because it can cause abortion and stillbirths (Cliver 1990). If the fetus survives, the parasite can be passed to the infant and result in numerous health problems from congenital toxoplasmosis (Carvalheiro and others 2005). Of the 225000 cases of toxoplasmosis a year in the United States, 50% are food-related (Mead and others 1999). Moreover, *T. gondii* is responsible for 8% of hospitalizations and 24% of deaths from foodborne illness (Scallan and others 2011). The cost of toxoplasmosis is $29429 per case with a total of $45 million spent on medical costs for patients with congenital toxoplasmosis (Roberts and others 1994; Scharff 2010). Based on annual pork consumption and prevalence of *T. gondii* in pork, it was calculated that people in the northeastern United States have a 78% chance of purchasing retail pork contaminated with *T. gondii* in a 10-y time period (Dubey and others 2005). All of these factors make *T. gondii* a pathogen with as much burden on society as *Salmonella* or *Campylobacter* (Kijlstra and Jongert 2008).

Prevalence: farm

The prevalence of *T. gondii* in swine in various countries has been well documented (Table 7). A study on the seroprevalence of *T. gondii* antibodies in naturally infected livestock has determined that, in 38 countries, *T. gondii* prevalence in domestic swine ranged from 0% to 97% between 1952 and 1985 (Dubey 1986). However, the prevalence has decreased significantly since that study due to improved production practices. In a national seroprevalence survey in the United States in 1991, it was determined that 24% of swine were positive for the parasite (Dubey and others 1991). The most recent Natl. Animal Health Monitoring Survey (NAHMS) was conducted in 2006 for swine and revealed a 2.6% seroprevalence of *T. gondii* in pigs from 16 of the leading swine production states in the United States (Hill and others 2010). *T. gondii* prevalence at the farm is highly dependent on production stage and management practices, with a higher prevalence in breeding swine than market swine (Dubey and others 1991; Wang and others 2002). Prevalence in breeding swine can be decreased with management practice. Lower sow prevalence of *T. gondii* has been associated with larger herd size and indoor confinement (Wang and others 2002). Sow prevalence in the United States decreased from 20% in 1990 to 6.5% in 2000, likely due to the switch to indoor confinement and increased biosecurity measures (Hill and others 2010). Despite the reductions in the sow herd, levels of the pathogen in growing and finishing pigs were maintained from 1990 to 2000 at approximately 2% (Hill and others 2010).

In addition to production stage, the prevalence of *T. gondii* is quite dependent on type of management (Venturini and others 2004). Intensive production systems have the lowest prevalence of the pathogen. In contrast, organic and free-range production systems have a higher prevalence of *T. gondii* (van der Giessen and others 2007; Hill and others 2010). The risk for *T. gondii* -positive swine is increased when intensive production systems are not used; moreover, free-range production is a greater risk for infection compared to organic production (van der Giessen and others 2007). This increased risk is likely due to increased exposure to wildlife or cats that carry the parasite (Smith and others 1992; Dubey and others 2002; Lehmann and others 2003; García-Bocanegra and others 2010; Hill and others 2010; Jiang and others 2012). Other risk factors on the farm related to management include: improper disposal of pig carcasses, contaminated feed or water source, and antibiotic-free production systems (Gebreyes and others 2008; Hill and others 2010).

Wildlife in proximity to the farm is also a risk factor for *T. gondii* infection in swine. Raccoons, skunks, opossums, rats,

Table 7–Seroprevalence of *T. gondii* at farm.

Country	Reference	Production stage	*n*	# positive samples	% of samples positive
Argentina	Venturini and others (2004)	Gilts	37	2	5.4
		Gilts	26	1	3.8
		Sows	25	1	4.0
		Sows	23	23	100.0
		Nursery pigs	30	12	40.0
		Growers	29	4	13.8
		Finishers	30	6	20.0
Canada	Gajadhar and others (1998)	Finishing pigs	2800	240	8.6
Ghana	Arko-Mensah and others (2000)	Coastal savannah	214	94	43.9
		Forest belt	128	39	30.5
		Guinea savannah	299	127	42.5
		Total swine	641	260	40.6
		1 to 5 mo old	114	13	11.0
		6 to 12 mo old	184	67	36.4
		> 12 mo old	135	65	48.1
Netherlands	van der Giessen and others (2007)	Organic	402	11	2.7
		Free-range	178	10	5.6
		Intensive	265	1	0.4
		Total	845	22	2.6
Spain	Garcia-Bocanegra and others (2010)	3 wk old	230	24	10.4
		7 wk old	210	30	14.3
		11 wk old	150	46	30.7
		15 wk old	150	53	35.4
		20 wk old	140	25	17.9
		Sows	322	50	15.5
		Total	1202	228	19.0
U.S.	Dubey and others (1995)	Sows	2617	395	15.1
		Finishing pigs	4252	97	2.3
	Dubey and others (1991)	Market pigs	11229	2584	23.0
		Breeding pigs	613	254	41.4
		Total	11842	2838	24.0
	Davies and others (1998)	Free-range	63	12	19.0
		Total confinement	1752	1	0.01
		Total	2238	13	0.6
	Wang and others 2002	Sows: confined	1884	218	11.6
		Sows: not confined	1149	232	20.2
		Market hog: confined	2096	48	2.3
		Market hog: not confined	1334	59	4.4
	Patton and others (2002)	Sows	8086	487	6.0
		Finishing pigs	5720	49	0.9
		Total pigs	13835	536	3.9
	Gamble and others (1999b)	Pigs	1897	900	47.4
		Herd	85	77	90.6
	Gebreyes and others (2008)	Total pigs	616	25	4.1
	Smith and others (1992)	Sows	273	39	14.3
Zimbabwe	Hove and others (2005)	Fattening pigs	238	47	19.8
		Cull pigs (+ 4 y old)	55	17	30.9
		Free-range	70	25	35.7

white-footed mice, and house mice were found to have a *T. gondii* prevalence of 67%, 38.9%, 22.7%, 6.3%, 4.9%, and 2.1%, respectively (Dubey and others 1995). Although raccoons have a high prevalence of *T. gondii*, they are not a real risk factor for infection of pigs as they do not shed oocysts, and racoons are not typically consumed by swine. However, this study may be an underestimation of the true prevalence of the parasite in rodents. A small sample size was used due to inability to catch a large number of rats or mice. Moreover, antibodies may have not formed yet in the mice that were collected, or positive mice which are known to be more prone to predation may have been captured by cats or died from a *T. gondii* infection (Dubey and others 1995). In addition to mammals being carriers of the pathogen, birds, especially robins, had a high prevalence of the parasite (Lehmann and others 2003).

Cats on the farm put swine at the highest risk for *T. gondii* because felines are the only known shedders of the cysts (Wang and others 2002; García-Bocanegra and others 2010; Hill and others 2010). Cats become infected by consumption of contaminated rodents, wildlife, meat, soil, or water. Cats have the ability to shed up to 20 million oocyst units/d during a primary infection and 1 million oocyst units/d during a secondary infection (Jiang and others 2012). Thus, outdoor cats on farms which scavenge other animals are a major concern for spreading *T. gondii* to swine. Sows were 14.3% more likely to be infected with the parasite when cats were present (Wang and others 2002).

Prevalence: slaughter and retail pork

Jacobs and others (1960) were the first to report *T. gondii* in pork diaphragm tissue and the risk that infected pork poses for consumers. Since that time, several studies have been conducted to determine the prevalence of the pathogen at slaughter (Table 8) and in retail meat (Table 9). Because *T. gondii* can remain infective in the tissues of swine for at least 171 d, any presence of *T. gondii* is a concern for humans (Dubey and others 1984). In one study, pork was the only retail meat positive for the parasite in the United States (Dubey and others 2005).

Intervention: preharvest

Intervention against *T. gondii* is especially critical at the farm because parasites require a living host to multiply (Patton and others

Table 8–Prevalence of *Toxoplasma* and *Trichinella* at slaughter.

Country	Reference	Species	Sample	Production stage	*n*	# positive samples	% of samples positive
U.S.	Jacobs and others (1960)	*T. gondii*	Diaphragm muscle	–	50	12	24.0
Switzerland	Berger-Schoch and others (2011)	*T. gondii*	Diaphragm tissue	Finishing pigs	50	1	2.0
				Sows	120	3	2.5
				Free-range	100	2	2.0
				Total	270	6	2.2
Argentina	Ribicich and others (2009)	*Trichinella*	Seroprevalence	–	3224	67	2.1
U.S.	Schad and others (1985a)	*T. spiralis*	Diaphragm muscle	Total slaughter	33482	196	0.6
				Commercial slaughter	28948	202	0.7
				Custom slaughter	4534	5	0.1
U.S.	Schad and others (1985b)	*T. spiralis*	Diaphragm muscle	–	5315	39	0.7

Table 9–Prevalence of *Toxoplasma* or *Trichinella* in retail meat.

Country	Reference	Location	Species	Sample	*n*	# positive samples	% of samples positive
U.K.	Aspinall and others (2002)	Retail	*Toxoplasma*	Raw pork	57	19	33.3
U.S.	Dubey and others (2005)	Retail	*T. gondii*	Raw pork	2094	8	0.4
Brazil	Dias and others (2005)	Processing	*T. gondii*	Fresh sausage	149	13	8.7
U.S.	Zimmerman and others (1961)	Retail	*T. spiralis*	Fresh sausage- bulk	8402	88	1.0
				Fresh sausage-link	1432	34	2.4
				Processed sausage-link	861	2	0.2

2002). Both live and DNA vaccinations have been considered for use in swine production. There are many concerns with using a live vaccine, including challenges with refrigeration and shelf-life, mortality or morbidity from toxoplasmosis infection, and risk of activation of the parasites when consumed by humans (Jenkins 2001; Garcia and others 2005). Mutant strains or strains not found in pigs are candidates for live vaccine use in pigs, but may only provide partial protection (Pinckney and others 1994; Dubey and others 2005). Additional challenges with using a vaccination against *T. gondii* arise due to the many stages in the life cycle of the parasite and its ability to adjust quickly to host immunity. Subunit vaccines with antigens against each life cycle stage of the parasite are most effective (Jenkins 2001). DNA vaccines seem promising alternatives to live vaccines as they elicit a humoral and cellular immune response through the development of antibodies against *T. gondii* with less possibility of a toxoplasmosis infection (Jongert and others 2008). If the obstacles to vaccines can be overcome, they are promising intervention techniques.

As an alternative to vaccinating swine, vaccines against *T. gondii* in cats can significantly reduce the prevalence of the parasite in cats and mice on swine farms, leading to a subsequent reduction in pigs (Araujo 1994; Mateus-Pinilla and others 1999; Kijlstra and Jongert 2008). Over a 3-y period, vaccination of cats decreased *T. gondii* by 21% in sows and up to 14.4% in finishing swine (Mateus-Pinilla and others 1999). Because other environmental factors contribute to prevalence in swine, complete eradication over a short period of time is not possible. Furthermore, stray or neighboring cats that did not receive the vaccination could also contribute to infection in pigs.

Despite the advances in vaccination use in cats, management practices for controlling cross-contamination from cats or other animals to swine is probably more realistic and economical. Various researchers determined that farms where swine were totally confined and cats were not present had a significantly lower prevalence of *T. gondii* (Wang and others 2002; García-Bocanegra and others 2010). Additional farm management techniques, including heating of feed to destroy the pathogen and ensuring clean drinking water is provided, also reduce prevalence of the parasite (Mateus-Pinilla and others 1999).

Intervention: postharvest

Cooking is the most common method of postharvest intervention against *T. gondii* (Cliver 1990; Dubey and others 1990; Lundén and Uggla 1992; Mateus-Pinilla and others 1999). Destruction of the parasite is time- and temperature-dependent, and if not performed to the proper extent could still allow survival of the parasite. In general, a minimum of 67 °C is required to ensure that inactivation occurs regardless of the duration the pork is heated (Dubey and others 1990). Linear regression equations have been developed for determining the time required to inactivate *T. gondii* at different temperatures (Dubey and others 1990). Freezing is also an effective means of intervention with a temperature of less than or equal to –12.4 °C being the minimum temperature to ensure destruction of the parasite in a short time (Lundén and Uggla 1992; Kotula and others 1994; Dubey and others 1998;). However, temperatures of –8, –6, and –1 °C will inactivate *T. gondii* when frozen for 2.8, 17, or 34 d, respectively. *T. gondii* is inactivated by freezing and heating more easily than *Trichinella* (Dubey and others 1990). Undercooking or improper cooking of contaminated meat is a major risk factor. Microwave heating is often uneven and not rapid enough to destroy the parasite (Lundén and Uggla 1992). Thorough, even heating is important to inactivate *T. gondii*.

Further processing steps such as curing, enhancement, or smoking are also effective at destroying *T. gondii* (Lundén and Uggla 1992; Warnekulasuriya and others 1998). The high concentration of salt in curing or enhancement solutions prevents survival of the parasite. Because approximately one-half of pork retail cuts are enhanced, ingredients that will reduce the risk of the pathogen are important considerations for processors (Hill and others 2004). Which ingredients are in the enhancement solution play a critical role in inactivation of *T. gondii*. Lactate-based solutions or salt solutions inactivated the parasite, while sodium phosphate had no destructive effect on *T. gondii* (Dubey and others 2005). Sodium chloride at greater than 1%, sodium lactate, and potassium lactate were effective in decreasing the viability of cysts (Hill and others 2004). Sodium tripolyphosphate and sodium diacetate, however, were not effective at inactivating *T. gondii* when used alone or in combination. The ability of enhancement or curing to reduce

Table 10–Seroprevalence of *Trichinella* at farm.

Country	Reference	Species	Production stage	*n*	# positive samples	% of samples positive
U.S.	Cowen and others (1990)	*T. spiralis*	Cull swine	10765	49	0.5
			Breeding swine	30162	150	0.5
			Total	40927	154	0.4
U.S.	Gamble and Bush (1999)	*T. spiralis*	NAHMS 1990	3048	5	0.2
			NAHMS 1995- sows and finishing	7987	1	0.01
U.S.	Gamble and others (1999a)	*T. spiralis*	Pig	4078	15	0.4
			Herd	156	10	6.4
U.S.	Gebreyes and others (2008)	*Trichinella* spp.	Antibiotic free	324	2	0.3
			Conventional	292	0	0

T. gondii was previously suggested by Warnekulasuriya and others (1998) who found very low levels of the pathogen in cured, RTE pork.

It is unknown how fermentation, drying, spices with antimicrobial properties, organic acids, or nitrites affect *T. gondii*. Because the effectiveness of these processes has not been determined, these processing techniques cannot be considered intervention steps for the pathogen. This uncertainty is of particular concern in certain RTE pork products which may not be thermally treated or frozen, thus may require a higher salt concentration or a longer maturation step of the product to ensure inactivation of *T. gondii* (Mie and others 2008). Because the majority of meat from sows goes to further processing, the higher prevalence of *T. gondii* in sows at the farm is not a large risk for consumers. However, low levels of the parasite in market swine which are not processed thermally or frozen can be a real risk for consumers.

Alternative nonthermal processing technologies such as high-pressure processing (HPP) and irradiation are effective at inactivating *T. gondii*. Use of HPP has been established as an antimicrobial processing technology (Shigehisa and others 1991; Hayman and others 2004). In ground pork, HPP at 400 MPa was more effective at destroying the parasite at all 3 time intervals (30, 60, and 90 s) compared to 200 and 100 MPa (Lindsay and others 2006). Irradiation with dosages between 0.5 and 0.7 kGy have been determined to be effective against *T. gondii* and seems to be an effective means of controlling the pathogen, especially in raw retail pork (Song and others 1993; Dubey and Thayer 1994; Dubey and others 1998). However, there are some concerns for consumer acceptability and lipid oxidation acceleration when using irradiation.

Trichinella. *Trichinella*, another parasite found in the tissues of animals, can be a concern for consumers. There are difference species of the pathogen, and *T. spiralis* is the most common species found in domestic swine. Moreover, *T. spiralis* is the species that most significantly contributes to foodborne disease in humans (Mead and others 1999). People who eat contaminated meat contract trichinellosis with symptoms of fever, abdominal pain, periorbital edema, and eosinophilia. Cysts that invade the small intestine during the primary infection are capable of releasing larvae, which can migrate and invade striated muscle. This can lead to inefficiency of muscle contraction, myalgia, and joint pain (Cliver 1990; Kennedy and others 2009).

Although trichinellosis has decreased significantly since the 1980s, the pathogen is still prevalent at low levels. Approximately 150 cases of illness are caused by *T. spiralis* each year in the United States (Scallan and others 2011); 100% of those cases are foodborne and approximately 20% are caused by pork in the United States (Kennedy and others 2009). The prevalence of *T. spiralis* also extends beyond the United States. A survey of 198 countries with domestic animals revealed that 21.9% of the countries had detectable *Trichinella* spp. in swine herds (Pozio 2007). A CDC summary of trichinellosis from 2002 to 2007 revealed that 19% of the cases in the United States were caused by consumption of domestic pork, while 50% were from consumption of wild game (Kennedy and others 2009). Most of the pork that caused an infection was purchased from commercial retail stores and consumed either raw or undercooked. For example, 40 people in Wisconsin ate infected pork sausage without proper cooking (Moorhead and others 1999). Salami made in Serbia, likely from a *Trichinella*-endemic area, caused trichinellosis in 8 individuals in the United Kingdom (Milnes and others 2001). Moreover, political and economic changes in southeastern Europe may cause decreased resources for commercial production operations and slaughtering facilities, which could lead to more backyard farmers and potential *Trichinella* infection in swine (Cuperlovic and others 2005).

Prevalence: farm

Prevalence of *T. spiralis* (Table 10) has decreased to very low levels in the United States across all production stages (Davies and others 1998; Gamble and Bush 1999; van der Giessen and others 2007). There are several risk factors, however, that can increase the prevalence of *T. spiralis* in swine. In breeding swine, males were found to have a higher prevalence of the parasite than females (Cowen and others 1990). Wildlife, wildlife carcasses, and swine carcasses near swine farms can contribute to *T. spiralis* in pigs (Gamble and others a). Consumption of these contaminated sources by swine provides an opportunity for swine to be infected with *Trichinella*. Recent increases in organic and free-range swine production practices also put pigs at a greater risk of *T. spirali* infection (Murrell and Pozio 2000; van der Giessen and others 2007). In finishing pigs, 0.24% of organic farms were found positive for *T. spiralis* compared to 0% in intensive production (van der Giessen and others 2007). Similarly, increased prevalence of *Trichinella* was detected among organic producers compared with intensive producers in Europe which has led to a re-emergence of trichinellosis in Europe (Dupouy-Camet 2006). Another significant risk factor of *T. spiralis* infection in swine is the feeding of garbage (Cliver 1990; Ribicich and others 2009). This food source can become contaminated with the parasite, which is then easily passed to swine through consumption.

Prevalence: slaughter

Because farm interventions against *Trichinella* are very effective, little information is available regarding prevalence of *T. spiralis* at slaughter (Table 8). A 1985 investigation of 5,315 diaphragm tissues at slaughter revealed a *T. spiralis* prevalence of 0.73% (Schad and others a). These low levels of the pathogen at slaughter have also been observed in other countries. A study in Argentina determined the seroprevalence of *T. spiralis* to be 2.1% (Ribicich and others 2009). A study of *T. spiralis* in China determined the prevalence of slaughter tissues to be between 0.0001% and 23% (Cui

and others 2006). This large range is likely due to differences in sampling area and whether swine were raised in rural or industrial regions.

Prevalence: retail pork

Because the prevalence of *T. spiralis* is no longer a concern in live production, limited research, especially in the United States and Europe, has been conducted at the retail level. As early as 1953, there was a marked decrease in the prevalence of *T. spiralis* (Table 9) in retail pork products (Zimmermann and others 1961). In 1961, fresh bulk, fresh link, and processed link pork sausages were sampled and had a *T. spiralis* prevalence of 1%, 2.4%, and 0.2%, respectively (Zimmermann and others 1961). However, a study of *T. spiralis* in China between 1999 and 2004 revealed a prevalence of 1.57% to 3.66% in retail meat (Cui and others 2006). This indicates that, although *T. spiralis* is not a concern in the United States, it is still a concern in other countries.

Intervention: preharvest

As an on-farm intervention against *T. spiralis*, the Federal Swine Health Protection Act was passed by the U.S. Congress in 1980 that prohibited the feeding of potentially contaminated food waste to swine (Kennedy and others 2009). Several other farm control strategies against *Trichinella* were also suggested: rodent and wildlife control, indoor housing, and a safe feed supply (Gamble and others 2000; van Knapen 2000). Since the 2008 Farm Bill, the U.S. Natl. Trichinae Certification Program has also been in place (Kennedy and others 2009). The Certification Program is an alternative to individual carcass testing at slaughter, which is known to be costly. The program audits farms based on their management and intervention practices and, if qualified, farms receive certification of their farm as *Trichinella*-free (Pyburn and others 2005).

The use of vaccines has also been explored as intervention against *Trichinella* in pigs. A live vaccine using newborn larval antigens was used in pigs (Marti and others 1987). Both whole larvae and the insoluble fraction of the larvae were found to be equally effective at providing 85% immunity in pigs. However, the soluble fraction of the larvae was not effective at generating immunity (Marti and others 1987). Other vaccines although tested in mice and not yet in pigs seem promising for future use in swine. For example, a nasal immunization with a peptide antigen was found to provide intestinal immunity (McGuire and others 2002). Moreover, a DNA vaccine provided partial protection against *T. spiralis* infection (Wang and others 2006). This vaccine induced a cellular and humoral immune response leading to a 37% reduction in muscle larvae. Delivery by both intramuscular injection or gene-gun were found equally effective (Wang and others 2006).

Intervention: postharvest

Thorough cooking to an adequate temperature is the easiest method to inactivate *T. spiralis* in a meat product (Kotula and others 1983; Cliver 1990; Gajadhar and others 2009). This intervention is time and temperature dependent. Cooking to 55 °C and holding that temperature for 4 min allowed for some infectivity of *T. spiralis* but this same temperature for 2 additional min rendered the parasite completely inactivated (Kotula and others 1983). Cooking regulations to inactivate *T. spiralis* in commercial RTE products is 58 °C, while home cooking recommendation is 71 °C (Gajadhar and others 2009). Microwave cooking, especially of pork roasts, is not recommended as an intervention for *Trichinella* (Zimmermann 1983).

Regulations on freezing as an intervention against *T. spiralis* have also been established (Shaver and Mizelle 1955; Smith 1975; Cliver 1990; Gajadhar and others 2009). This intervention is also time and temperature dependent (Gajadhar and others 2009). Freezing to –30 °C ensures that no viable *T. spiralis* remain (Smith 1975). Using liquid nitrogen or liquid carbon dioxide was also found to be effective at destruction of the parasite when –29 °C was achieved (Rust and Zimmermann 1972). Recently there has been concern of freeze-resistant strains of *Trichinella*; however, these strains are not found in domestic pigs (Pozio and others 2006; Hill and others 2009). The Intl. Commission on Trichinellosis does not recommend freezing as the sole intervention method because time and temperature are highly variable (Gamble and others 2000). Instead, thorough cooking remains the best option to inactivate *Trichinella*.

Other further processing technologies have been evaluated as interventions for the parasite. The Intl. Commission on Trichinellosis does not recommend curing, smoking, drying, or microwave-cooking as intervention steps (Gamble and others 2000). However, aging hams or shoulders for 4 wk after curing and smoking was found to be effective at removing all *T. spiralis* in muscle (Gammon and others 1968). Fermentation and drying of Genoa salami also reduced *T. spiralis* below detection or by 2.3 log larvae/g of batter (Porto-Fett and others 2010). Dry-curing was also found to be effective at inactivating *T. spiralis* in prosciutto, proscuittini, and Genoa salami due to the dehydrating effects of the salt (Smith and others 1989). These researchers also found that inclusion of sodium nitrate decreased the effectiveness of the salt. Because there is great variability in opinions on what interventions are appropriate for *T. spiralis*, it is important for processors to validate that their specific manufacturing process is capable of inactivating the parasite.

High-pressure processing was found to be effective at higher levels of pressure (483 and 600 MPa at 1 min or 30 s), and it reduced *T. spiralis* below the levels of detection (Porto-Fett and others 2010). Irradiation also has the ability to inactivate *T. spiralis* (Brake and others 1985; Cliver 1990). In ground pork, 20 krad significantly reduced the reproductive capacity of the parasite and 30 krad was sufficient irradiation to completely inactivate *T. spiralis* (Brake and others 1985).

L. monocytogenes. *Listeria* is a Gram-positive, facultative anaerobic bacteria that have the ability to grow at low temperatures (Bahk and Marth 1990). Among the different species of *Listeria*, *L. monocytogenes* is the only pathogenic species in humans. All strains of *L. monocytogenes* are pathogenic, but other species of *Listeria*, such as *L. innocua*, *L. grayi*, *and L. ivanovii*, are nonpathogenic (Szabo and Desmarchelier 1990; Ryser and Marth 2007). Headache, fever, back pain, and chills are common symptoms of listeriosis, with mortality from meningitis, encephalitis, or sepsis occurring in 20% to 30% of cases, mainly in immunocompromised individuals (Mead and others 1999; Schlech 2000). Pregnant women are at high risk of abortion or fetal infection from listeriosis. There are approximately 1500 cases of listeriosis a year in the United States with 99% of the cases being foodborne (Scallan and others 2011). *Listeria monocytogenes* has one of the highest hospitalization and fatality rates among foodborne pathogens (Mead and others 1999; Scallan and others 2011). The high morbidity and mortality causes the cost per case of listeriosis to be over $1.6 million and *L. monocytogenes* to be one of the most costly foodborne pathogens in the United States (Scharff 2010).

L. monocytogenes is comprised of 13 uniquely identified serotypes (Ryser and Marth 2007). *L. monocytogenes* serotypes found in

Table 11–Prevalence of *Listeria* at farm.

Country	Reference	Species of Pathogen	Sample	Production stage	*n*	# positive samples	% of samples positive
Finland	Hellstrom and others (2010)	*L. monocytogenes*	Rectal swab	Organic	121	4	3.3
			Feed/liter	Organic	15	3	20.0
			Rectal swab	Conventional	243	0	0
			Feed/liter	Conventional	23	1	4.3
			Rectal swab	All	364	4	1.1
			Fee/liter	All	38	4	10.5
France	Beloeil and others (2003)	*Listeria* spp.	Finishing pens	Wet feed pens	27	25	92.6
				Dry feed pens	20	10	50.0
				All pens	47	35	74.5
			Feedstuffs	Wet feed pens	25	21	84.0
				Dry feed pens	20	1	5.0
				All pens	45	22	48.9
Spain	Esteban and others (2009)	*L. monocytogenes*	Feces	–	17	0	0
U.K.	Fenlon and others (1996)	*L. monocytogenes*	Feces	Sows	7	1	14.3
				Piglets	30	0	0
U.S.	Kanuganti and others (2002)	*L. monocytogenes*	Tonsil scrapings	Market	297	1	0.3

retail pork are 4b, 1/2a, and 1/2c and represent 28%, 24%, and 22% of positive samples, respectively (Farber and Peterkin 1991). This agrees with a more recent investigation of *L. monocytogenes* serotypes in pork slaughter tissues and ground pork where serotypes 1/2a and 1/2b comprised 50% of the isolates while 4b was found in 25% of positive isolates (Kanuganti and others 2002). These 3 serotypes of *L. monocytogenes* account for the most cases of human listeriosis (Farber and Peterkin 1991).

Prevalence: farm

L. monocytogenes is ubiquitous and persists in the environment (Ryser and Marth 2007), which allows for infection of swine at the farm (Table 11). Unlike *Salmonella* and *Campylobacter*, which exist at high levels in the feces of pigs, *L. monocytogenes* does not flourish in the intestines of swine perhaps due to the competitive microflora (Bunčić 1991). In a study of finishing swine, only 1.7% of conventionally raised swine were positive for shedding *L. monocytogenes*, which is much lower than *Salmonella* and *Campylobacter* prevalence (Fosse and others 2009). However, production stage and management practices can significantly affect the prevalence of *L. monocytogenes* in swine. In feces, sows have a higher prevalence of *L. monocytogenes* than piglets (Fenlon and others 1996). However, cull sows had a lower prevalence of *L. monocytogenes* than market pigs when tested during slaughter, which could be attributed to lower pig densities of sows at the farm compared to market hogs (Wesley and others 2008). Moreover, fewer animals being processed at sow harvesting facilities allows for less cross-contamination to occur and reduces water use, contributing to a drier slaughter environment and reduced likelihood of contamination with *Listeria* spp. (Wesley and others 2008).

In addition to production stage, production practices affect *Listeria* spp. prevalence in swine. Heat-treated, manufactured diets have a lower prevalence of *L. monocytogenes*, while wet feed, which is not heat treated, can leave residues in feeding pipes and act as a reservoir for survival and growth of *Listeria* spp. (Bunčić 1991; Fenlon and others 1996; Beloeil and others 2003; Fosse and others 2009). Coarse feed can also increase the prevalence of swine that shed *Listeria* (Hellstrom and others 2010). This is likely due to the reduced processing and higher inclusion of silage, which has also been indicated to increase *Listeria* prevalence in swine (Fenlon 1985; Bunčić 1991; Fenlon and others 1996).

L. monocytogenes can also persist in other environmental locations on the farm. Litter or bedding, pens, and boots are all potential sources of *Listeria* contamination. Risks of *Listeria* contamination of pigs from the environment can be reduced if proper hygienic measures are taken, such as boot cleaning, change rooms for workers at the entrance of the facilities, and one or more days for swine pens to be disinfected and left empty without pigs (Beloeil and others 2003). Like other pathogens, weak biosecurity on the farm, including wild birds carrying *L. monocytogenes*, is a risk factor for swine contamination (Fenlon 1985). Because the environment is an important route of *Listeria* contamination in swine, less stringent biosecurity and hygienic management, often observed in organic production, can lead to increased *L. monocytogenes* prevalence (Hellstrom and others 2010). Organic production led to increased prevalence of *L. monocytogenes* in tonsil (47%) and pluck (13%) samples compared to conventional production (12% and 1%, respectively) (Hellstrom and others 2010).

Prevalence: slaughter

Contaminated pig carcasses at slaughter can occur in 2 ways: previously infected live animal or cross-contamination of the carcass from the slaughter environment (Table 12). Both of these routes of contamination have been verified through serotype tracings of *L. monocytogenes* during the slaughter process (Hellstrom and others 2010). Although there are generally low levels of *L. monocytogenes* in swine feces, the pathogen is present in higher levels in the tonsils with a prevalence of 12% to 45% (Bunčić 1991; Autio and others 2000; Hellstrom and others 2010). In a nationwide pork microbiological baseline survey of market hogs, 7.4% of carcass swabs (composite of belly, ham, and jowl samples) were positive for *L. monocytogenes* after chilling (FSIS 1996). Levels of *L. monocytogenes* were generally very low with approximately 78% of positive carcass swab samples containing <0.03 MPN/cm^2 (FSIS 1996). However, higher levels are not uncommon as 6.8% of positive carcass samples had greater than 3.1 MPN/cm^2 (FSIS 1996). Sows were found to have a lower level of positive tonsil samples than finishing swine, which may be a result of increased *Listeria* resistance with age or better management practices on the farm (Autio and others 2004).

Prevalence: retail pork

Consumers typically fully-cook fresh pork products and, therefore, these products are not a large risk factor for listeriosis. However, a majority of pork is consumed as further processed products, many of which are fully cooked products ready for consumption by consumers upon purchase (National Pork Board 2009). Because *L. monocytogenes* is capable of growing at refrigeration temperatures, proliferation of the bacteria to harmful levels can easily occur during retail and home storage of meat products with long shelf-lives

Table 12–Prevalence of *Listeria* at slaughter.

Country	Reference	Species	Sample	Production/ slaughter stage	*n*	# positive samples	% of samples positive
Belgium	Van Renterghem and others (1991)	*L. monocytogenes*	Feces		25	4	16.0
Canada	Gill and Jones (1995)	Total *Listeria*	Skinned pork loin		48	45	93.8
		L. monocytogenes	Skinned pork loin		48	2	4.2
Finland	Autio and others (2000)	*Listeria* spp.	Tongues		50	8	16.0
			Tonsils		50	6	12.0
			Hearts		50	3	6.0
			Livers		50	3	6.0
			Kidneys		50	4	8.0
			Carcass swab		50	6	12.0
			Environment-drains		10	4	40.0
			Environment-aprons		10	1	10.0
			Environment-tables		10	1	10.0
			Environment-knives		10	0	0
			Environment-doors		10	1	10.0
			Environment-saws		10	3	30.0
			Environment-other		13	1	10.0
			Pluck and environment		373	41	11.0
		L. monocytogenes	Tongues		50	7	14.0
			Tonsils		50	6	12.0
			Hearts		50	3	6.0
			Livers		50	3	6.0
			Kidneys		50	3	6.0
			Carcass swab		50	6	12.0
			Environment-drains		10	1	10.0
			Environment-aprons		10	0	0
			Environment-tables		10	1	10.0
			Environment-knives		10	0	0
			Environment-doors		10	1	10.0
			Environment-saws		10	2	20.0
			Environment-other		13	0	0
			Pluck and environment		373	33	8.8
Finland	Autio and others (2004)	*L. monocytogenes*	Tonsils	Finishing	132	29	22.0
				Sows	139	9	6.5
				Total	271	38	14.0
		L. innocua	Tonsil	Finishing	132	5	3.8
				Sows	139	7	5.0
				Total	271	12	4.4
Finland	Hellstrom and others (2010)	*L. monocytogenes*	Intestinal contents	Organic	119	4	3.4
				Conventional	239	1	0.4
				Total	358	5	1.4
			Tonsil	Organic	119	56	47.1
				Conventional	231	27	11.7
				Total	350	83	23.7
			Pluck set	Organic	120	15	12.5
				Conventional	234	2	0.9
				Total	354	17	4.8
			Carcass	Organic	120	2	1.7
				Conventional	239	0	0
				Total	359	2	0.6
Italy	Bonardi and others (2003)	*L. monocytogenes*	Cecal contents		150	2	1.3
			Feces		150	1	0.7
Japan	Iida and others (1998)	*L. monocytogenes*	Cecal contents		5975	46	0.8
			Carcass swab		4330	321	36.4
Netherlands	van den Elzen and Snijders (1993)	*L. monocytogenes*	Carcasses- outside		45	3	6.6
			Carcasses-inside		45	1	2.2
			Cutting room- hams		44	12	27.3
			Cutting-rooms- bellies		44	5	11.4
			Cutting room- shoulders		44	16	36.4
			Cutting room- necks		44	16	36.4
			Environment- slaughter		232	6	2.6
			Environment- cutting room		29	25	86.2
			Distribution room- loins		20	1	5.0
			Distribution room- bellies		20	19	95.0
			Distribution room-shoulders		20	9	45.0
			Distribution room- loin chops		20	9	45.0
			Distribution room- bacon		20	6	30.0
			Environment-distribution room		20	4	20.0
Sweden	Lindblad and others (2007)	*L. monocytogenes*	Carcass swab		251	6	2.0
U.S.	Saide-Albornoz and others (1995)	*L. monocytogenes*	Ham and loin surfaces	After singeing and polishing	270	4	1.5
				After final rinse	270	5	1.9
				After 24-hour chill	270	5	1.9

(*Continued*)

Table 12–(*Continued*)

Country	Reference	Species	Sample	Production/ slaughter stage	*n*	# positive samples	% of samples positive
U.S.	Kanuganti and others (2002)	*L. monocytogenes*	Tonsils		252	18	7.1
			Rectal contents		255	0	0
			Ileocecal lymph nodes		257	0	0
			Thoracic lymph nodes		259	9	3.5
			Superficial inguinal lymph nodes		262	5	1.9
			Carcass swabs		267	11	4.1
U.S.	Wesley and others (2008)	*L. monocytogenes*	Slaughter tissues	Cull sows	2727	2	<0.001
			Muscle tissue		213	3	1.4
			Total		2775	5	0.2
Yugoslavia	Bunčić (1991)	*L. innocua*	Tonsil		103	49	47.6
		L. monocytogenes	Tonsil		103	46	44.7
		L. innocua	Feces		97	45	46.4
		L. monocytogenes	Feces		97	3	3.1

(Sim and others 2002). Thus, RTE pork has been linked to numerous outbreaks of listeriosis including pork rillettes (a popular French meat spread) and frankfurters. The most recent listeriosis outbreak related to RTE pork in the United States was in 1998 with 101 cases and 21% mortality from eating contaminated hot dogs (Schlech 2000). In France, there was a nationwide outbreak in October 1999 involving rillettes and again in February 2000 from jellied pork tongue (de Valk and others 2011). Therefore, extensive sampling of retail and RTE pork products in various countries (Table 13) has been conducted (Schlech 2000). In a risk assessment of foods causing listeriosis, deli meats and frankfurters which were not reheated were classified as very high risk while fermented sausages, reheated frankfurters, and meat spreads were moderate risk products (Mataragas and others 2008). Fermented RTE meats, such as salami and semi-dry sausages, were not the greatest risk but their potential to harbor the bacteria are still important (FAO 2004).

In a recent study, 20% of fresh and frozen pork samples were determined to be positive for *L. monocytogenes*, and 16% of all processed meats, not just pork, were contaminated with *Listeria* (Jay 1996). This is in contrast to an earlier study of *L. monocytogenes* in pork which noted a range of 1.5% to 80% pork samples positive for *Listeria* including ham, minced pork, and a variety of sausage products (Farber and Peterkin 1991). While the majority (50%) of these positive samples had low *Listeria* counts (<20 CFU/g), higher levels were common with 33% of positive samples containing greater than 10^2 CFU/g (Farber and Peterkin 1991). This agrees with a survey of RTE of 2 states in the United States, which determined 88% of positive luncheon meat samples less than or equal to 10 CFU/g, 11% had less than 10^4 CFU/g, and no samples were found to have greater than 10^4 CFU/g (Gombas and others 2003). A more recent study found *L. monocytogenes* contamination in raw and RTE pork products to be 9.9% and 3.2%, respectively (Mataragas and others 2008). Although raw products have a higher prevalence, the cooking process will destroy *L. monocytogenes.* The prevalence of *Listeria* in RTE meats has decreased considerably due to intervention technologies and governmental regulation (Wong and others 2005).

Prevalence: environment

Because *L. monocytogenes* is ubiquitous in the natural environment, contamination can also occur from soil, water, and transmission by humans on clothes or shoes. Regardless of the source of contamination, *L. monocytogenes* can remain active in the environment for more than a year despite cleaning and disinfection (Gill and Jones 1995; Giovannacci and others 1999). Others have determined that *L. monocytogenes* can persist for as long as 8 y and cause listeriosis outbreaks (Warriner 2011). For example, *L. monocytogenes* from pork tonsils or tongue, spread to a backbone saw, was a source of further *L. monocytogenes* contamination in the slaughtering facility (Autio and others 2000). In addition to saws being contaminated, floor drains, doors, and tables are also reservoirs for *L. monocytogenes* in slaughter facilities (Autio and others 2000).

Retail fresh and RTE meats have a higher prevalence of *L. monocytogenes* than slaughter samples, which is also an indicator of environmental contamination (Iida and others 1998; Lunden and others 2003; Mena and others 2004). Although the slaughter environment is often contaminated with *L. monocytogenes*, the processing environment, especially postprocessing, has been found to have a greater prevalence of *L. monocytogenes* (van den Elzen and Snijders 1993). Contaminated processing equipment such as slicers, tumblers, transportation belts, metal tables, knives, and meat containers are the most important risk factors for *L. monocytogenes* contamination of retail meats (Salvat and others 1995; Samelis and Metaxopoulos 1999; Uyttendaele and others 1999; Tompkin 2002; Wilks and others 2006). Regular cleaning and proper sanitation can drastically reduce the levels of *L. monocytogenes* on equipment (Lunden and others 2003; Peccio and others 2003; Thévenot and others 2006). However, areas that are not typically cleaned, such as floors and drains, have a much higher prevalence of *Listeria* spp. (Buege and Ingham 2003). In a *Listeria* spp. audit of small plants in Wisconsin, 3.5% of food contact surfaces were found positive, while 13.1% and 27.8% of non-food contact surfaces and floors or drains were positive for *Listeria* spp., respectively. Although there is an extremely low risk of product contamination from floors and drains, it is important to improve sanitation and decrease the persistence of *L. monocytogenes* as much as possible in processing facilities (Buege and Ingham 2003). *L. monocytogenes* is capable of forming biofilms on a variety of materials typically found in the processing environment, such as stainless steel, aluminum, conveyor materials, Buna N rubber, silicone, polypropylene, polyurethane, and brick (Beresford and others 2001; Somers and Wong 2004). These biofilms grow better at 4 °C compared to 10 °C and exhibit increased resistance to sanitation when meat residues are present (Somers and Wong 2004). As reviewed by Tompkin (2002), a wide range of processing equipment and locations within the processing area can become a niche with contaminated *L. monocytogenes.* These sites include hollow rollers on conveyors, walls, rubber seals on doors, nozzles on spray brine units, frankfurter peels, and safety covers on machinery.

Because processing environments have the ability to be contaminated with *Listeria*, additional handling of RTE products after

Table 13–Prevalence of *Liseria* in retail meat.

Country	Reference	Location	Species	Sample	*n*	# positive samples	% of samples positive
Australia	Ibrahim and Mac Rae (1991)	Retail store	*Listeria* spp.	Fresh pork	50	15	30.0
			L. monocytogenes	Fresh pork	50	5	10.0
Belgium	Uyttendaele and others (1999)	Retail market	*L. monocytogenes*	Cooked ham, before slicing	1069	15	1.4
				Cooked ham, after slicing	879	54	6.1
				Raw, cured ham	169	20	11.8
				Cooked loin, before slicing	87	3	3.5
				Cooked loin, after slicing	127	13	10.2
				Cured, prepackaged loin	121	24	19.8
		Supermarkets	*L. monocytogenes*	Prepackaged, blood sausage	137	12	8.8
				Not prepackaged, blood sausage	18	2	11.1
				Ham salad	159	33	20.8
Brazil	de Fatima Borges (1999)	Retail markets	*L. monocytogenes*	Salami	81	7	8.6
Chile	Cordano and Rocourt (2001)	Industries, markets, restaurants, hospitals	*L. monocytogenes*	Ham	31	1	3.2
				Sausages	443	20	4.5
				Meat pates	160	2	1.3
				Total processed meat	634	23	3.6
Finland	Hellstrom and others (2010)		*L. monocytogenes*	Cut pork- conventional production pigs	80	3	3.8
				Cut pork- organic production pigs	60	2	3.3
				Total cut pork	140	5	3.6
France	Chasseignaux and others (2001)	Processing	*L. monocytogenes*	Raw pork meat	24	8	33.3
				Retail pork products	6	1	16.7
				Shelf-life pork products	8	4	50.0
France	Thévenot and others (2005b)	Processing plant	*L. monocytogenes*	Raw pork	121	41	33.9
				Dried sausage	30	3	10.0
				Equipment- before operation	383	58	15.1
				Equipment-after operation	318	162	50.9
Greece	Samelis and Metaxopoulos (1999)	Processing plant	*L. monocytogenes*	Ham	6	1	16.7
				Pork shoulder	6	0	0
				Ham-like product	4	1	25.0
				Bacon	4	0	0
				Pariza	2	0	0
				Mortadella	4	0	0
				Frankfurter-type sausage	8	0	0
				Country-style sausage	10	1	10.0
				Dry fermented sausage (salami)	4	0	0
				Raw pork- hind leg	7	1	14.3
				Raw pork- shoulder	6	0	0
				Raw pork- trimmings	10	6	60.0
				Raw pork- loins	5	1	20.0
				Raw pork- mechanically deboned	6	5	83.3
				Pork back fat	7	3	42.9
Ireland	Sheridan and others (1994)	Retail outlets	*Listeria* spp.	Pork	20	9	45.0
			L. monocytogenes	Pork	20	3	15.0
			Listeria spp.	Sausages	20	13	65.0
			L. monocytogenes	Sausages	20	9	45.0
			Listeria spp.	Cooked ham- open package	20	12	60.0
			L. monocytogenes	Cooked ham- open package	20	2	10.0
			Listeria spp.	Roast pork-open	20	5	25.0
			L. monocytogenes	Roast pork-open	20	0	0.0
Ireland	Wilson (1995)	Retail displays	*Listeria* spp.	Bacon	20	0	0
				Ham	1141	74	6.0
				Pork	794	35	4.0
				Fermented sausage	53	1	2.0
				Pate	222	2	1.0
Japan	Iida and others (1998)	Retail	*L. monocytogenes*	Sliced pork	209	76	36.4
Japan	Inoue and others (2000)	Retail stores	*L. monocytogenes*	Minced pork	34	5	12.2
Japan	Ryu and others (1992)	Supermarket, department stores		Whole pieces pork	5	2	40.0
				Sliced pork	13	5	38.5
				Minced pork	6	4	66.7
				Raw pork ham	3	0	0
Latvia and Lithuania	Bērziņš and others (2007)	Supermarket	*L. monocytogenes*	Cold-smoked pork, sliced vacuum-packaged	212	120	38.0
New Zealand	Hudson and others (1992)	Retail outlets	*L. monocytogenes*	RTE pork	34	1	2.9
New Zealand	Wong and others (2005)	Retail outlets	*L. monocytogenes*	Prepackaged ham	104	1	1.0
Portugal	Mena and others (2004)	Retailers, producers	*L. monocytogenes*	Cooked ham	4	1	25.0
				Dry cured ham	44	1	2.3
				Smoked sausage	48	0	0
				Blood sausage	9	1	11.1
				Spanish-style sausage	27	1	3.7
Serbia	Dimíc and others (2010)	Retail markets	*Listeria* spp.	Pork	10	9	90.0
Taiwan	Wong and others (1990)	Retail market	*L. monocytogenes*	Domestic pork	34	20	58.8
Trinidad	Adesiyun 1993	Retail store	*Listeria* spp.	Fresh pork	71	1	1.4

(*Continued*)

Table 13–(*Continued*)

Country	Reference	Location	Species	Sample	*n*	# positive samples	% of samples positive
Turkey	Colak and others (2007)	Retail markets	*Listeria* spp.	Turkish-style fermented sausage (sucuk)	300	6	21.0
			L. monocytogenes	Turkish-style fermented sausage (sucuk)	300	35	11.6
U.K.	MacGowan and others (1994)	Retail store	*Listeria* spp.	Raw pork	15	9	60.0
				Ground pork	3	2	66.7
				Bacon gammon	13	8	61.5
				Pork sausage	19	17	89.5
U.S.	Saide-Albornoz and others (1995)	Plant	*L. monocytogenes*	Boneless loins- before packaging	135	0	0
				Boneless loins- 36 d of storage	45	2	4.4
		Total	*L. monocytogenes*	ground pork and/or pork sausage	120	32	26.7
U.S.	Duffy and others (2001)	Hot-boning, sow and board plant	*Listeria* spp.	Ground pork and/or pork sausage	40	16	40.0
		Slaughtering and fabrication plant	*Listeria* spp.	Ground pork and/or pork sausage	40	23	57.5
		Further processing plant	*Listeria* spp.	Ground pork and/or pork sausage	40	18	45.0
		Total	*Listeria* spp.	Ground pork and/or pork sausage	120	57	47.5
		Hot-boning, sow and boar plant	*L. monocytogenes*	Ground pork and/or pork sausage	40	5	12.5
		Slaughtering and fabrication plant	*L. monocytogenes*	Ground pork and/or pork sausage	40	13	32.5
		Further processing plant	*L. monocytogenes*	Ground pork and/or pork sausage	40	14	35.0
		Retail stores	*Listeria* spp.	Whole muscle, store-packaged pork	96	27	28.1
				Whole muscle, enhanced	96	24	25.0
				Store-ground fresh pork and/or pork sausage	96	59	61.5
				Prepackaged ground pork and/or pork sausage	96	51	53.1
				Total	384	161	41.9
			L. monocytogenes	Whole muscle, store-packaged pork	96	14	14.6
				Store-ground fresh pork and/or pork sausage	96	22	22.9
				Prepackaged ground pork and/or pork sausage	96	26	27.1
				Total	384	76	19.8
U.S.	Gombas and others (2003)	Retail stores	*L. monocytogenes*	Luncheon meats	9199	82	0.89
U.S.	Kanuganti and others (2002)	Packing plant	*L. monocytogenes*	Ground pork	300	134	44.7
		Retail grocery	*L. monocytogenes*	Ground pork	40	37	92.0
		Packing plant and retail grocery	*L. monocytogenes*	Ground pork	340	171	50.2
		Packing plant	*L. monocytogenes*	Raw chittlerlings	300	28	9.3
Yugoslavia	Bunčić (1991)	Supermarkets, butcher shops	*L. monocytogenes*	Fermented sausages	21	4	19.0
				Vacuum packaged, hot-smoked sausages	14	3	21.0
				Hot-smoked sausages	15	0	0
Review	Mataragas and others (2008)	Processing	*L. monocytogenes*	Pork	513	63	12.3
		Retail raw products	*L. monocytogenes*	Pork	3031	301	9.9
		Retail RTE products	*L. monocytogenes*	Pork	90667	2869	3.2

processing increases the risk of product contamination (Hudson and others 1992; Samelis and Metaxopoulos 1999). Thus, RTE meats purchased at a delicatessen counter have a greater risk of *L. monocytogenes* contamination than prepackaged products (Hudson and others 1992; Endrikat and others 2010). This is evident from a survey of RTE products which found only 0.4% of manufacture packaged luncheon meat samples positive for *L. monocytogenes* whereas 2.7% of luncheon meat packaged in the store were contaminated with the bacteria (Gombas and others 2003). This increased prevalence in store-packaged delicatessen meats are calculated to be 5 times more likely to cause listeriosis each year (Endrikat and others 2010). Although cross-contamination can occur between uncontaminated and contaminated products, contamination of food contact surfaces and equipment is more likely (Pradhan and others 2011). Frequency of cross-contamination at 2.3% was estimated to increase the probability of death from *L. monocytogenes* by approximately 6-fold. Moreover, the rate of cross-contamination was found to be a more important risk factor for listeriosis than the initial contamination prevalence and initial contamination level (Pradhan and others 2011). Even raw products have an increased prevalence of *L. monocytogenes* due to further processing as raw sausage products had a greater prevalence of *L. monocytogenes* than whole-muscle pork cuts (Duffy and others 2001). Other risk factors of *L. monocytogenes*, according to a study in Latvia and Lithuania, are brine injection and hours of cold smoking (Bērziņš and others 2007).

Intervention: postharvest

Slaughter. Because live animals and raw pork are not the greatest risks for *Listeria* contamination of meat, interventions at the farm are limited. Small steps towards intervention have been investigated during the slaughter process. Enclosure of the rectum before evisceration reduced *Listeria innocua* on the carcass from 33% to 10% (Nesbakken and others 1994). More recently, novel intervention strategies to assist small slaughter facilities such as household steam cleaners have been studied. Steam treatment

caused 5.75 and 7.61 $\log_{10}$ CFU/cm^2 reductions of *L. monocytogenes* when pork skin was inoculated with 10^5 or 10^7 CFU/cm^2, respectively. Reductions in generalized microbe levels were also realized when household steam cleaners were used on carcasses, with maximum reduction on the jowl, belly, and ham of the carcasses (Chen 2005).

Processing. In the United States, there is a zero tolerance regulation of *L. monocytogenes* in RTE meat products (FSIS 2003). Thus, the vast majority of *L. monocytogenes* intervention has been studied in RTE meat products. Thermal treatment is the most common intervention strategy against *Listeria* (Hudson and others 1992; Thévenot and others 2006). Although *L. monocytogenes* is more thermotolerant than other pathogens, it is inactivated when heated above 70 °C (Thévenot and others 2006). However, despite the thermal inactivation tendencies of *L. monocytogenes*, postprocessing contamination is still a concern.

The organism may also be eliminated or reduced through fermentation, smoking, and drying of pork products (Foegeding and others 1992; Hudson and others 1992; Ingham and others 2004; Thévenot and others a). However, these reductions are sometimes minimal. A reduction in pH and water activity (Aw), as well as increasing salt concentration through the drying and maturation process, was found more effective at *L. monocytogenes* inactivation than fermentation alone (Thévenot and others a). Interestingly, this study involving experimentally contaminated French sausages suggested that the safest fermented sausages may be those closest to the expiration date when sausages are the most mature and at their lowest water activity (Thévenot and others a). Storage conditions of uncooked, fermented products can also decrease *L. monocytogenes.* In addition to A_w below 0.90, increased days of ripening, higher storage temperature, and air flow during storage decreased *L. monocytogenes* in chorizo sausages (Encinas and others 1999; Hajmeer and others 2005; Hew and others 2005).

A unique characteristic of *L. monocytogenes* is its adaptive ability to respond to sublethal stress with a greater resistance to lethal stresses and greater virulent capacity (O'Driscoll and others 1996; Lou and Yousef 1997). For example, sublethal pH or heat shock allowed *L. monocytogenes* cultures to become acid tolerant as well as more resistant to salt, ethanol, hydrogen peroxide, crystal violet, nisin, and cleaning agents such as ammonium compounds, ammonium chloride compounds, and persulfate (O'Driscoll and others 1996; Lou and Yousef 1997; Lunden and others 2003; Bonnet and Montville 2005). Reduced pH caused by the lactic acid production in the manufacturing of fermented foods allowed *L. monocytogenes* to adapt and withstand pH conditions as low as 3.5, (O'Driscoll and others 1996; Koutsoumanis and others 2003; Bonnet and Montville 2005). This characteristic of *L. monocytogenes* is an important risk factor in contributing to foodborne illness and crucial to keep in mind when developing interventions.

Protective bacterial cultures. Although the fermentation process alone can cause a reduction of *L. monocytogenes*, the reductions can be minimal and *L. monocytogenes* survival is possible (Porto-Fett and others 2010). Lactic acid bacteria (LAB), which are responsible for the fermentation of meat products, may provide additional antilisterial capacity through the production of antimicrobial compounds such as bacteriocins, hydrogen peroxide, and organic acids (Foegeding and others 1992; Bredholt and others 1999; Benkerroum and others 2005). Improved reductions of *L. monocytogenes* were observed in sausages when *Pediococcus pentosaceus* or *P. acidilactici* were used as starter cultures (Foegeding and others 1992; Farber and others 1993). In an *in vitro* study evaluating different LAB, *P. acidilactici*, *Lactobacillus casei*, and *L. paracasei* were found to be the most effective at inhibiting *L. monocytogenes* (Brashears and Amézquita 2001). These species were then evaluated in cooked ham and frankfurters and found to be effective at reducing *L. monocytogenes* through 23 d of storage. Additional advantages of LAB include the lack of pathogenicity, unaffected sensory properties of meat, and *in situ* production of bacteriocin (Brashears and Amézquita 2001; Benkerroum and others 2005). In a comparison of 2 starter culture mixtures containing either *Staphylococcus xylosus* with *Pediococcus acidilactici* and *Lactobacillus bavaricus* or *S. carnosus* with *L. curvatus* in fermented sausages, the culture with *P. acidilactici* was able to inactivate *L. monocytogenes* at higher levels than the other starter culture (Lahti and others 2001). Thus, a combination of starter cultures has the potential to be more protective against *L. monocytogenes*, especially when one of the cultures produces pediocin, which is an antimicrobial peptide (bacteriocin) that is produced by *P. acidilactici*. The lactic acid produced by starter cultures is also able to inhibit *L. monocytogenes* (Bedie and others 2001). In addition to these mechanisms for inactivating *L. monocytogenes*, LAB typically have faster growth rates allowing them to deplete nutrients and overwhelm *L. monocytogenes* (Brashears and Amézquita 2001).

Protective cultures have also been studied in nonfermented pork products. Pediocin from *Lactobacillus pentosus* reduced *L. monocytogenes* in chilled, tray-packaged pork (Zhang and others 2010). *Lactobacillus sakei* was also found effective at reducing *L. monocytogenes* in pork products, not just as starter cultures for fermentation in sausage (de Martinis and Franco 1998; Vermeiren and others 2006). Different strains of *L. sakei* have been compared for use as antimicrobials in cooked ham. *L. sakei* 10a, a nonbacteriocin-producing strain, was more effective than *L. sakei* 148, a lactocin S-producing strain, as *L. sakei* 148 was not able to reduce inoculated levels of *L. monocytogenes.* Combining *L. sakei* with modified atmosphere packaging (50% CO_2) was also capable of inactivating *L. monocytogenes* (Vermeiren and others 2006).

Other species of bacteria have been investigated *in vitro* for their potential intervention against *Listeria*. *Enterococcus faecalis*, which produces enterocin, was antlisterial with no improvements in antlisterial ability observed when the bacteria were combined with sodium benzoate, sodium chloride, sodium acetate, or sodium trypolyphosphate (García and others 2004). However, combining *E. faecalis* with potassium nitrate or sodium nitrite improved the effectiveness against *L. monocytogenes* (García and others 2004). *L. innocua*, a non-pathogenic species of *Listeria*, was found to produce a trypsin-sensitive bacteriocin-like substance that had an inhibitory effect against *L. monocytogenes* when studied *in vitro* (Yokoyama and others 1998).

Cure ingredients. There are conflicting results on the effectiveness of curing solutions as an intervention against *Listeria*. *In vitro*, nitrite alone or with polyphosphate was able to reduce *L. monocytogenes* growth while polyphosphate alone was not effective (Bunčić and others 1995). However, others have found that *Listeria* was capable of growing in cured and smoked ham (Seman and others 2002). Salt is not the most useful intervention as high concentrations are required, and *L. monocytogenes* was not reduced when using typical salt levels (0.80% and 3.5%) present in processed meat products (Foegeding and others 1992; Seman and others 2002; Ryser and Marth 2007).

Antimicrobials. Although further processing reduces the survival of *L. monocytogenes*, concerns of postprocessing contamination from the environment have prompted a variety of additional

interventions against *L. monocytogenes*. Antimicrobial compounds are a simple strategy to include in a product formulation or apply to finished products, and their use in a variety of pork products has been an effective *Listeria* intervention. Plant oil aromatics including eugenol, carvacrol, and cinnamaldehyde were found to have bactericidal potential against *L. monocytogenes* when studied *in vitro* (Gill and Holley 2006; Pérez-Conesa and others 2006). These compounds have the ability to disrupt the membrane of *Listeria* cells through inhibition of membrane-bound ATPase activity. Use in meat products may be limited due to impact on sensory traits, but their use against *L. monocytogenes* biofilms in the environment seems promising (Gill and Holley 2006; Pérez-Conesa and others 2006). Many essential oils contain compounds, which are antilisterial, thus the use of oils in pork products meets the consumer demand for natural products while effectively inhibiting *L. monocytogenes* (Oussalah and others 2006).

Organic acids. Organic acids have been established as generally recognized as safe (GRAS) compounds and are commonly used as an intervention against *L. monocytogenes* in a variety of pork products, from sausages to cured ham, as long as levels do not exceed FSIS regulations (Seman and others 2002; Barmaplia and others 2005). The maximum allowed levels of sodium lactate by FSIS is 3%, which has been found effective at suppressing *L. monocytogenes* in frankfurters though 90 d of storage (Bedie and others 2001). Maintaining a reduction of *L. monocytogenes* for a longer period can be achieved with higher concentrations of sodium lactate; however, these increased levels are not approved (Bedie and others 2001). In addition, increased effectiveness of sodium lactate, sometimes with lower concentrations, is often achieved through combination with other organic acids (Samelis and others 2002). Sodium lactate combined with one or more pH-reducing compounds, such as glucono-delta-lactone (GDL), sodium acetate, or sodium diacetate, further inhibited *L. monocytogenes* growth (Qvist and others 1994; Samelis and others 2002; Barmaplia and others 2005). Low levels of sodium acetate (0.25%) with 2.5% sodium lactate in both cervelat sausage and cooked ham reduced the growth of *L. monocytogenes* without negatively impacting sensory traits (Blom and others 1997). However, despite effective inhibition of *L. monocytogenes* through combination of sodium lactate with 0.2% sodium diacetate in cooked ham, a sensory panel detected an unpleasant odor and taste in these products (Stekelenburg and Kant-Muermans 2001). Thus, using higher levels of these acidifiers may be limited due to the potential negative impacts on sensory characteristics of the product (Qvist and others 1994; Samelis and others 2002; Barmaplia and others 2005). Overall, use of organic acid growth inhibitors in delicatessen meats can considerably reduce the risk of *L. monocytogenes* in these products (Pradhan and others 2009).

In addition to sodium lactate being effective against *L. monocytogenes*, the use of other organic acids has been investigated. Potassium sorbate at very low levels (0.3%) was bacteriostatic, but *L. monocytogenes* reductions were not improved when potassium sorbate was combined with sodium lactate (Bunčić and others 1995). Although potassium lactate can be used alone or in combination with sodium diacetate, it was not effective at controlling *L. monocytogenes* growth in temperature-abused products (Lianou and others 2007). Sodium citrate was unable to reduce *L. monocytogenes* in cooked ham and, in fact, when used at 1%, actually increased the growth of *L. monocytogenes* (Stekelenburg and Kant-Muermans 2001).

Nisin. Nisin, a bacteriocin, is used as an antimicrobial in various foods, including meat, and has the potential to enhance other interventions against *Listeria* (Mikel and Newman 2002). Although some reduction of *L. monocytogenes* was observed when using acetic acid, lactic acid, or potassium benzoate alone, inactivation of *Listeria* was greatly improved when used in combination with nisin (Geornaras and others 2006). Despite the initial bactericidal effects of nisin *in vitro*, after 14 d of storage, growth of *L. monocytogenes* was observed (Bunčić and others 1995). However, combination of nisin with organic acids, such as sodium lactate, and cure ingredients, such as nitrate and polyphosphate, reduced *L. monocytogenes* for longer periods (Bunčić and others 1995). Combination of nisin with sorbate resulted in maximum reductions of *L. monocytogenes* and was the most promising antimicrobial combination for low- pH cured meats in this study (Bunčić and others 1995). A synergistic inhibitory effect against *L. monocytogenes* was also observed *in vitro* with acidic calcium sulfate, octanoic acid, and nisin and is promising for use in RTE meats (Taylor 2009). Nisin with steam treatment also produced a synergistic antilisterial effect in country-cured ham slices (Mikel and Newman 2002).

High-pressure processing. High-pressure processing (HPP) is currently used to inactivate *L. monocytogenes* in meat products. The effectiveness of HPP against *L. monocytogenes* is dependent on the amount of pressure and length of application. High-pressure processing treatment at 450 MPa for 10 min or 600 MPa for 3 min was effective at reducing *L. monocytogenes* in cured ham and sausages (Hayman and others 2004; Morales and others 2006). Treating fermented Genoa salami with HPP (483 or 600 MPa) decreased *L. monocytogenes* significantly more than fermentation and drying alone (Porto-Fett and others 2010). However, treatment for 10 min at 300 MPa was not effective at inactivating *L. monocytogenes* in fermented sausages, and a slight increase of *Listeria* was actually observed (Marcos and others 2005). This is likely due to insufficient pressure treatment for *L. monocytogenes* inactivation but sufficient levels for LAB destruction, which prevented the reduction of *L. monocytogenes* through normal fermentation (Marcos and others 2005).

Another concern with HPP is its influence on sensory traits. Although no negative sensory characteristics were detected when HPP was used on ham, discoloration occurred when sausages were treated with high pressure (Marcos and others 2005; Morales and others 2006). Resuscitation capacity of *L. monocytogenes* from sublethal stress is also a concern for HPP (Ritz and others 2006). In culture broth, growth of *L. monocytogenes* was observed after HPP (600 MPa) treatment and storage at room temperature, while no *L. monocytogenes* was detected when stored at 4 °C. Resuscitation capacity was also reduced by decreasing the pH of the broth solution (Ritz and others 2006).

Multi-hurdle approach. Recently, multiple processing technologies have been combined to maximize the intervention effectiveness against *L. monocytogenes.* High-pressure processing combined with antimicrobials provide an extremely effective means of first inactivating *L. monocytogenes*, and then suppressing growth if any bacteria survive the HPP treatment. For examples, HPP combined with tert-butylhydroquinone (TBHQ), a phenolic antimicrobial additive, or HPP with TBHQ and nisin in sausages was more effective than HPP (400 MPa) alone at reducing *L. monocytogenes* (Chung and others 2005). In another study, the most effective intervention against *L. monocytogenes* was observed when HPP, nisin, and potassium lactate were combined in sliced, cooked ham compared with any of these intervention strategies when used

alone (Jofré and others 2008). Although lactate salts are effective against *L. monocytogenes* alone, their effectiveness is often limited to refrigeration temperatures. By combining sodium lactate with HPP, *L. monocytogenes* was reduced even when sliced, cooked ham was held at a higher temperature (6 °C against 1 °C) (Aymerich and others 2005).

The antilisterial effects of antimicrobials were also enhanced when combined with heat treatment in frankfurters or ham (Samelis and others 2002; Thippareddi and others 2002). Frankfurters treated with organic acid, vacuum-packaged, and then treated with hot water for 60 s reduced *L. monocytogenes* more than organic acids or thermal pasteurization alone (Samelis and others 2002). Thermal pasteurization likely causes structural damage to *Listeria* cells making those that survive more susceptible to antimicrobial destruction (Samelis and others 2002). Increased reductions were observed with increased pasteurization temperature, and individually packaged frankfurters revealed greater *L. monocytogenes* reduction than 4 frankfurters packaged together (Thippareddi and others 2002). A concern with this intervention technology is the effect on color and texture as hotdogs were found to be harder after lactic acid and postprocessing pasteurization (Thippareddi and others 2002).

Bacteriophages. Bacteriophages were first used as part of a typing scheme for *Listeria* due to strain specificity that most phages display (Loessner and Busse 1990; Hagens and Loessner 2007). However, some *L. monocytogenes* phages have a broader host range, thus making them useful as an intervention against *L. monocytogenes* (Loessner and Busse 1990). There are over 400 bacteriophages that have been isolated for use against *Listeria*; however, many of these phages are specific to other nonpathogenic species of *Listeria* and not effective against *L. monocytogenes* (Hagens and Loessner 2007). Two strains of bacteriophages, P100 and A511, have been effective against *L. monocytogenes*, especially both the 1/2 and 4 serovars. Both A511 and P100 reduced *L. monocytogenes* in frankfurters with higher levels of the phages (3×10^8 against 3×10^6 PFU/g) required to suppress *L. monocytogenes* in solid food (Guenther and others 2009). Higher concentrations of the phages also allowed for greater *L. monocytogenes* reductions for a longer time in liquid and solid food systems (Guenther and others 2009). P100 bacteriophage has also been combined with the protective culture *Lactobacillus sakei* in cooked ham to reduce *L. monocytogenes* growth through 28 d of storage (Holck and Berg 2009). Although high levels of *L. sakei* are required (10^6 CFU/g), this species of *Lactobacillus* is effective at low temperatures and in a vacuum package with no impact on sensory characteristics of ham (Holck and Berg 2009).

Packaging. Pork products packaged with modified atmosphere packaging (MAP) were less often contaminated with *L. monocytogenes* than non-MAP products (Sheridan and others 1994). However, more complex packaging systems have been investigated as interventions against *L. monocytogenes.* Packaging films with nisin and organic acids incorporated into the film inhibited *L. monocytogenes* growth *in vitro*, and the films seem promising for use in packaging meat products (Grower and others 2004). Protective cultures, such as *Lactobacillus plantarum*, *Enterococcus casselifavus*, and *L. sakei*, were incorporated into packaging biofilm and effective at reducing *L. monocytogenes* (Guerrieri and others 2009; Gialamas and others 2010).

Environmental interventions. *L. monocytogenes* biofilm cells attach to environmental surfaces in a cluster of cells and exhibit unique resistance to stresses due to protective coatings. Because the slaughter and processing environments pose a great risk for *L. monocytogenes* biofilm formation with subsequent contamination of pork products, sanitation of these areas is incredibly important. For sanitizers and detergents to be considered effective against biofilms, a 3-log reduction of *L. monocytogenes* must be obtained (Somers and Wong 2004). Planktonic cells, which are single cells with no attachment to surfaces, are more sensitive to sanitation but biofilm cells may require as much as 100 times greater concentration of sodium hypochlorite, a common cleaning agent, to be inactivated (Norwood and Gilmour 2000). Chlorine is also a common cleaning intervention which is effective at reducing *L. monocytogenes* biofilm on processing equipment (Taormina and Beuchat 2001). Adjusting the pH of the chlorine solution to 6.5 improved *L. monocytogenes* reductions on stainless steel and conveyor belt material, and the adjusted solution was more effective than using higher concentrations of nonadjusted chlorine (Bremer and others 2002). Increased exposure time of the biofilms to the chlorine solution also increased *L. monocytogenes* reductions from 92% to 99.8% (Bremer and others 2002). Additionally, in a study using planktonic cells, chlorine treatment resulted in *L. monocytogenes* that was more sensitive to heat treatment (Taormina and Beuchat 2001). In contrast, using sanitizers based on alkaline pH induced cross-protection of *L. monocytogenes* cells against heat treatment (Taormina and Beuchat 2001). Peroxides and quaternary ammonium compounds (QAC) are also often used in the food industry as sanitizers (Pan and others 2006). Approximately 100 strains of *L. monocytogenes* were tested against QAC sanitizer; the majority of the strains became resistant and required a very high minimum inhibitory concentration (MIC) of QAC to become inactivated (Mereghetti and others 2000). Resistance to peroxide can also occur, which cross-protected *L. monocytogenes* against other sanitizers (Pan and others 2006). *L. monocytogenes* sanitizer resistance is surface-dependent as stainless steel showed less resistance development than Teflon and PVC (Pan and others 2006). In a comparison of 2 commercial cleaning combinations, a combination including chlorinated alkaline solution, low-phosphate detergent, and dual peracid sanitizer was less effective than the combination of solvated alkaline solution with hypochlorite sanitizer (Somers and Wong 2004). Although both reduced biofilm levels significantly, a 3 log reduction was achieved by the chlorinated solution 86% of the time while the solvated alkaline solution only achieved such reductions 50% of the time (Somers and Wong 2004). Regardless of which sanitizer was used, *L. monocytogenes* biofilm on conveyor materials (TURE-2 and Buna N) were more resistant to sanitation than stainless steel or silicone (Somers and Wong 2004). In a comparison of 9 different compounds commonly used in sanitizers, all compounds were found effective against *L. monocytogenes* when tested on clean surfaces (Aarnisalo and others 2000). However, only isopropanol-based compounds achieved a 3 log reduction of *L. monocytogenes* on surfaces with residual pork tissue while tertiary alkylamine and dimethylamine betaine were the least effective in soiled conditions. Compounds that reached a 3 log reduction of *L. monocytogenes*, in clean surface conditions, included hydrogen peroxide, peracetic acid, acetic acid, hypochlorite, QAC, potassium persulfate-based sanitizer, and an isopropanol-based compound (Aarnisalo and others 2000). The ability of biofilms to become resistant to sanitizers over time does occur in clean surface conditions, as 5-day biofilm *L. monocytogenes* cells were more resistant than 2-day cells (Somers and Wong 2004). Regardless of age of biofilm in soiled-surface conditions, resistance was not significantly different, which is likely an indicator that these cells develop resistance very quickly when meat residues are present (Somers and Wong 2004). Thus, the importance of thorough cleaning

to reduce soiled-surface conditions and potential *L. monocytogenes* biofilm development and sanitizer resistance is evident.

Methicillin-Resistant *S. aureus*

Staphylococci bacteria are Gram-positive, facultative anaerobes whose main habitat is the skin and upper respiratory tract of animals, birds, and humans (Bergdoll 1990; Ray 2001; Kluytmans 2010). Among the various staphylococci species, *S. aureus* is responsible for foodborne illness in humans causing approximately 241000 cases a year in the United States (Scallan and others 2011). Symptoms develop in 1 to 6 h after ingestion and are generally mild, such as nausea, diarrhea, vomiting, and abdominal cramping (Bergdoll 1990). More severe symptoms are rare, and death is very uncommon. *S. aureus* has the ability to release enterotoxins, which are the source of illness in humans and responsible for a hospitalization rate of 14% (Noskin and others 2007). However, for sufficient enterotoxin levels to cause illness at least 10^5 cells/g of *S. aureus* must be present (ICMSF 2005; Bahtia and Zahoor 2007). Because of the low severity of foodborne illness from *S. aureus* the cost per case is only $818 (Scharff 2010). Moreover, *S. aureus* is not a strong competitor against other bacteria in food systems (Argudín and others 2010), which limits growth of the bacteria and subsequent enterotoxin production. Thus, *S. aureus* contamination is typically associated with handling of meat products after processing when competition has been eliminated. This transmission route was confirmed through typing of the bacteria which determined that *S. aureus* strains, which caused foodborne illness were from human origin and not livestock or raw meat (ICMSF 2005).

Despite the relative low-risk nature of *S. aureus*, emergence of methicillin-resistant *S. aureus* (MRSA) has become a human health concern. Typically associated with health-care facilities, MRSA is endemic to hospitals and has recently been found in various pets and livestock, including swine (Leonard and Markey 2008; Kluytmans 2010). Through sequence typing, the strain of MRSA in livestock has been identified as ST398 and is genetically different from hospital-associated MRSA (Kluytmans 2010). Methicillin-resistant *S. aureus* ST398 is not considered as virulent as other strains of MRSA because it generally does not possess the genes that encode for enterotoxin production and is less easily transmitted to humans (Wulf and Voss 2008; Kluytmans 2010; Köck and others 2010). *S. aureus* can be carried asymptomatically in the nasal passage of humans and livestock. Methicillin-resistant *S. aureus* nasal carriage has been increasing among humans despite reductions in *S. aureus* infection (Gorwitz and others 2008). Livestock-associated MRSA (ST398) has been isolated in humans, especially those in close contact with swine. Given the press attention to MRSA, the emergence of a livestock-specific strain harbored in pigs and its isolation in humans is alarming to the public. However, it is still unclear how prevalence of MRSA in pigs relates to human illness, especially foodborne illness (Lewis and others 2008; Kluytmans 2010; Weese and others 2010a).

Prevalence: farm

Methicillin-resistant *S. aureus* has been identified in numerous countries across various swine production stages (Table 14). Nursing pigs have a high prevalence of MRSA with 20% to 49% containing positive nasal samples (Khanna and others 2008; Gómez-Sanz and others 2010; Weese and others b). In a study of MRSA infection in piglets, those that came from MRSA-positive sows were all positive at some point during their life, while only 84% of piglets from negative sows were positive during their life (Weese and others b). Thus, the status of sows is a risk factor for piglet infection but is not the only factor for MRSA prevalence. There is also evidence that the percentage of positive MRSA pigs postwean (85%) is greater than prewean (35%) (Weese and others b). While there is a high prevalence of MRSA in nursery and weaned pigs, the prevalence of MRSA in finishing swine either decreases (Smith and others 2009; Gómez-Sanz and others 2010) or stays the same (Khanna and others 2008). Levels of MRSA in finishing swine were as low as 1.3% in Malaysia (Neela and others 2009) and were recorded at 4.6% and 26% in Canada (Khanna and others 2008; Weese and others a). There is disagreement whether antimicrobial use in swine is a risk factor for MRSA contamination in swine. Although some identify it as a potential risk, the MRSA isolated in pigs was not resistant to the antimicrobial drugs typically given to swine (van Duijkeren and others 2008). Others have found that antibiotic-free farms can still be positive for MRSA, suggesting infection of swine is not solely caused by antimicrobial use in pigs (Weese 2010; Weese and others 2011b).

Infection with MRSA in finishing swine is influenced by infected nursery or finishing pigs sourced from other farms, contact with human carrying MRSA, and persistence in the environment (van Duijkeren and others 2008). Although pigs from other farms can be a source of contamination, closed farms that do not bring in swine from other farms were also found MRSA-positive (van Duijkeren and others 2008). Others have found a strong correlation between MRSA infection in pigs and MRSA infection in swine workers (Khanna and others 2008). Workers, veterinarians, and other personnel have been found positive for MRSA (van Duijkeren and others 2008). Worker prevalence was found to be 20% and 50% in Canada and the United States, respectively (Khanna and others 2008; Smith and others 2009). Much lower prevalence was found among swine farm workers in China and Malaysia (Cui and others 2009; Neela and others 2009). By serotyping MRSA from positive pigs, both livestock-associated and human-associated types have been isolated in swine (Lim and others 2012).

Prevalence: slaughter

Much like the variability of MRSA prevalence in finishing swine, the prevalence at slaughter varies by country and sampling location (Table 15). The greatest prevalence of MRSA is found in nasal samples compared to fecal or carcass swab samples, and as high as 65% of nasal samples were positive in a study in Germany (Beneke and others 2011). However, this poses a low risk to carcass contamination as hygienic removal of the head eliminates the pathogen (Beneke and others 2011). Fecal samples are not positive for MRSA likely due to the established gut microbiota in market swine and competition with *Salmonella* and *Campylobacter* (Baba and others 2010; Weese and others 2011a). However, there is still concern for cross-contamination from the head to the carcass as carcass swabs have been found positive for MRSA (Beneke and others 2011). Carcass swab samples from the shoulder had higher prevalence of MRSA than from the back or belly likely due to the inverted suspension of the carcass during slaughter and proximity to the head (Beneke and others 2011). While the carcass is frequently contaminated with *S. aureus*, it is less often contaminated with MRSA (Schraft and others 1992; Lin and others 2009; Lim and others 2010).

Transportation to slaughter and lairage are risk factors for MRSA contamination in swine at slaughter (de Neeling and others 2007; Broens and others 2011). Swine that were negative for MRSA before loading onto transportation trucks became

Table 14–Prevalence of MRSA at farm.

Country	Reference	Species of pathogen	Sample	Production stage	*n*	# positive samples	% of samples positive
Canada	Khanna and others (2008)	MRSA	Nasal swab	Nursing pigs	85	17	20.0
				Weaned pigs	95	27	28.4
				Finishing pigs	105	27	25.7
				Total pigs	285	17	24.9
Canada	Weese and others (2010a)	MRSA	Nasal swab	Piglet- d1	100	1	1.0
				Piglet- d3	97	3	6.2
				Piglet- d7	94	8	8.5
				Piglet- d14	91	4	4.4
				Piglet- d21	91	18	19.8
				Overall prewean	–	–	34.5
				d28	91	31	34.1
				d42	88	57	65.0
				d56	88	44	50.0
				d70	87	36	41.4
				Overall postwean	–	–	85.0
Canada	Weese and others (2011a)	MRSA	Nasal swab	Finishing pigs	460	21	4.6
China	Cui and others (2009)	MRSA	Nasal swab		509	58	11.4
Korea	Lim and others (2012)	MRSA	Nasal swab		657	21	3.2
Malaysia	Neela and others (2009)	MRSA	Nasal swab	Total pigs	360	5	1.4
				Weaned pigs	75	4	5.3
				Finishing pigs	75	1	1.3
Spain	Gómez-Sanz and others (2010)	MRSA	Nasal swab	Nursing pigs	53	26	49.1
				Finishing pigs	53	11	20.7
U.S.	Smith and others (2009)	MRSA	Nasal swab	Total	209	147	70.3

Table 15–Prevalence of MRSA at slaughter.

Country	Reference	Species	Sample	Production/ slaughter stage	*n*	# positive samples	% of samples positive
Germany	Beneke and others (2011)	MRSA	Nasal swab	Stunning	133	86	64.7
			Carcass surface- shoulder		50	6	12.0
			Carcass surface- belly		50	1	2.0
			Carcass surface-back		50	2	4.0
			Total carcass surface		150	9	6.0
			Total environment		50	6	12.0
			Platforms		25	5	20.0
			Saws		25	1	4.0
Germany	Tenhagen and others (2009)	MRSA	Nasal swab		1026	596	58.1
Japan	Baba and others (2010)	MRSA	Nasal swab		115	1	0.9
			Fecal sample		115	0	0
Korea	Lim and others (2010)	MRSA	Carcass swab		999	0	0
Netherlands	de Neeling and others (2007)	MRSA	Nasal swab		540	209	38.7
Switzerland	Schraft and others (1992)	*S. aureus*	Chilled hindquarter swab		223	75	33.6
Taiwan	Lin and others (2009)	MRSA	Carcass swab		1410	128	9.1
U.S.A.	Saide-Albornoz and others (1995)	*S. aureus*	Carcass swab-ham and loin	After singeing and polishing	270	12	4.4
				After final rinse	270	20	7.4
				After 24-hour chill	270	34	12.6

positive (21.1%) due to contaminated trucks or contact with contaminated pigs from other farms. Moreover, lairage only further increased the prevalence of MRSA to 60%. Surprisingly, this increase was not found to be related to length of time in the holding pens (Broens and others 2011).

The slaughter environment can become contaminated with MRSA through the processing of pigs. For example, at the beginning of the day, MRSA was only found in holding pens, but by the end of processing had spread to other parts of plant (van Cleef and others 2010). In a separate study, 20% of platforms and 4% of saws became contaminated with MRSA during the slaughter process (Beneke and others 2011). Environmental contamination of MRSA is a concern as it provides a route for cross-contamination to carcasses.

Prevalence: retail pork

Because MRSA is a recent concern in retail meat, several sampling studies have been conducted and the focus has been primarily on raw pork (Table 16). *S. aureus* has been identified in approximately one-half of retail raw pork samples in The Netherlands, United States, and Germany while MRSA prevalence was considerably less in these countries (Atanassova and others 2001; van Loo and others 2007; Pu and others 2009; Beneke and others 2011; Hanson and others 2011). Between 3.1% and 10.7% of raw pork, including ground pork and pork chops, can be considered to be contaminated with MRSA (van Loo and others 2007; de Boer and others 2009). Although no differences between MRSA prevalence have been found between whole muscle or ground pork products (Weese and others 2010b), a separate study by the same researchers determined ground pork contamination (6.3%) to be less than MRSA prevalence in pork chops (13.6%)(Weese and others 2010a). These differences could be a result of differences in enrichment and detection methodology. The packaging condition of collected samples was not reported, but vacuum-stuffing and oxygen-impermeable-packaging of ground pork could also reduce the survival of MRSA in ground product. Pork is more often contaminated with MRSA than other retail meat, including beef, chicken, or turkey (Weese and others 2010b). In a study of retail meat in Iowa, MRSA was only detected in pork samples (Hanson and others 2011).

Table 16–Prevalence of MRSA in retail meat.

Country	Reference	Location	Species	Sample	*n*	# positive samples	% of samples positive
Canada	Weese and others (2010b)	Retail outlets	MRSA	Total raw pork	402	31	7.7
				Pork chops	296	23	7.8
				Ground pork	94	7	7.4
				Pork shoulder	12	1	8.3
Canada	Weese (2010)	Retail outlets	MRSA	Total pork	230	22	9.6
				Ground pork	127	8	6.3
				Pork chops	103	14	13.6
Germany	Atanassova and others (2001)	Processing	*S. aureus*	Raw pork	135	84	62.2
				Salted pork	135	75	55.6
				Uncooked, smoked ham	135	48	35.6
Germany	Beneke and others (2011)	Processing	MRSA	Shoulder	48	1	2.1
				Belly	48	2	4.2
				Back	48	3	6.3
				Total meat	144	6	4.2
				Final products	71	2	2.8
				Environment	44	0	0
Korea	Lim and others (2010)	Retail	MRSA	Raw pork	56	4	7.1
Netherlands	de Boer and others (2009)	Retail trade	MRSA	Raw pork	309	33	10.7
Netherlands	van Loo and others (2007)	Supermarkets and butcher shops	*S. aureus*	Raw pork	64	29	45.3
			MRSA	Raw pork	64	2	3.1
U.S.	Hanson and others (2011)	Retail stores	*S. aureus*	Pork chops and ground pork	55	10	18.2
			MRSA	Pork chops and ground pork	55	2	3.6
U.S.	Pu and others (2009)	Grocery stores	*S. aureus*	Pork chops	90	41	45.6
			MRSA	Pork chops	90	5	5.6
U.S.	Saide-Albornoz and others (1995)	Processing plant	*S. aureus*	Boneless loins before packaging	135	4	2.6
				Boneless loins vacuum-packaged, 36 d	45	2	4.4
U.S.	Waters and others (2011)	Grocery stores	*S. aureus*	Pork chops and ground pork	26	11	42.3

Carryover of MRSA from the live animal to retail can occur but is rare (Beneke and others 2011). Environmental contamination of MRSA in processing facilities is less than in slaughter facilities due to lower room temperature (Beneke and others 2011). Many researchers have found the MRSA strains isolated from retail meat are not livestock-associated and, instead, are typically carried by humans, which is a strong indicator of poor handling of products and cross-contamination from humans (de Boer and others 2009; Pu and others 2009; Weese and others 2010b). Increases in prevalence from slaughter to retail also indicate MRSA contamination in pork is of human or environmental sources and not from the live animal (Simeoni and others 2008; Lim and others 2010). Moreover, strains of *S. aureus* which are enterotoxin producing are typically associated with humans instead of livestock (ICMSF 2005). All of these factors allowed the International Commission of Microbiological Specifications in Foods to determine the major risk for foodborne *S. aureus* is from further processed products which are improperly processed or handled product (ICMSF 2005). For example, in 2005 an outbreak of *S. aureus* in southeast Kansas occurred from a catered event (Huang and others 2006). Smoked sausage was implicated as the source of infection and was likely a result of contaminated equipment or humans in combination with improper cooling, reheating, or holding of product.

Intervention: postharvest

Thermal processing is the most common intervention against *S. aureus* and MRSA, and inactivation occurs at 71.1 °C (Palumbo and others 1977). In a recent study on heat treatment, it was found that chilled storage does not increase the heat resistance of *S. aureus*, but inactivation temperatures were found to be higher than expected at 75 °C (Kennedy and others 2005). *S. aureus*, and subsequently MRSA, is typically a low-risk pathogen in retail pork but becomes a high risk in RTE products due to postprocessing contamination from equipment or humans (Bergdoll 1990; Mataragas and others 2008). Thus, although heat treatment is a sufficient intervention against MRSA, concerns of contaminated RTE products have prompted the investigation of various antimicrobials for use against *S. aureus* and MRSA.

In an evaluation of plant essential oils *in vitro*, tea tree, thyme, peppermint, lavender, and juniper were found to be effective against *S. aureus* (Nelson 1997). The minimum inhibitory concentration (MIC) and minimum lethal concentration (MLC) of tea tree oil were found to be 1% and 2%, respectively (Low and others 2011). A 3-log reduction of *S. aureus* was achieved at the MLC of tea tree oil, and no additional reduction was seen when tea tree oil was combined with silver ions (Low and others 2011). Lavender oils were also found equally effective against MRSA compared to Methicillin-susceptible *S. aureus* (MSSA) (Roller and others 2009). Direct contact of lavender oil was more effective than exposure to the vapor phase, and improved reductions of MRSA were seen when different species of lavender were combined (Roller and others 2009). In a comparison of the effects of 21 different plant essential oils *in vitro* against *S. aureus*, bay, spearmint, thyme, clove, eucalyptus, basil, and sage were found to be the most effective (Smith-Palmer and others 1998). Bay was bactericidal at 0.075%, while clove, cinnamon, and thyme were bactericidal against *S. aureus* at ≤0.04% (Smith-Palmer and others 1998).

Oregano essential oil is a common antioxidant and flavoring component in food, and it has shown antimicrobial potential against *S. aureus* (Lambert and others 2001; Nostro and others 2004). The phenolic compounds thymol and carvacrol in oregano oil are responsible for membrane destruction of bacterial cells (Lambert and others 2001). *In vitro*, oregano oil was found to be equally effective against MRSA and MSSA (Nostro and others 2004). Because thymol and carvacrol are small components of oregano oil, a higher concentration of oregano essential oil (0.06% to 0.125%) compared to thymol alone (0.03% to 0.06%)

or carvacrol alone (0.015% to 0.03%) is needed for the same MIC against MRSA (Nostro and others 2004). Oregano essential oil was also effective against *S. aureus* biofilm cells, but it required 2- to 4-fold greater concentrations than levels used against planktonic *S. aureus* cells (Nostro and others 2007). A concern with using oregano oil or its components against *S. aureus* biofilm cells is the potential for sublethal levels to actually increase *S. aureus* biofilm growth (Nostro and others 2007). A common way to preserve the antioxidant property of oils is through encapsulation. Through encapsulation of oregano essential oils, antimicrobial activity against *S. aureus* was improved in addition to reduced oxidation of the oil (Arana-Sánchez and others 2010). Along with greater biological activity, encapsulation resulted in a greater solubility of the oil and improved ease-of-working with the oil (Arana-Sánchez and others 2010). Liquid-phase exposure of *S. aureus* biofilms to 1% carvacrol was more effective than exposure to the vapor phase (Nostro and others 2009). Oregano essential oils, thymol, and carvacrol are all promising antimicrobials in product formulations or as surface disinfectants in slaughter or processing environments where biofilms commonly form (Nostro and others 2009).

More novel approaches against *S. aureus* have been explored due to consumer desire for natural products. *Eleutherine americana* is an herbal plant commonly used in Thai cuisine which has been shown to be promising *in vitro* against *S. aureus* but was not as effective in a cooked pork model (Ifesan and Voravuthikunchai 2009; Ifesan and others 2009). Perilla oil, a natural medicine and culinary herb from East Asia, was inhibitory against MRSA and MSSA (Qiu and others 2011). Chinese green tea extract, especially the components epicatechin gallate (ECG) and epigallocatechin gallate (EGCG), are also inhibitory against MRSA and MSSA (Si and others 2006).

Nisin, a common bacteriocin used as an antimicrobial, at concentrations greater than 150 μg/g in fermented sausage, was able to maintain *S. aureus* reductions through 35 d of storage (Hampikyan 2009). Lower concentrations of nisin allowed growth of *S. aureus*, and no advantage in reduction was seen when higher levels were used (Hampikyan 2009). Combining nisin with sodium lactate in cooked ham showed improved reductions of *S. aureus* (Jofré and others 2008). However, these reductions showed no advantage over refrigeration alone. Interestingly, high-pressure processing at 600 MPa, a common pressure treatment for the destruction of other pathogens, was not effective in reducing *S. aureus* (Jofré and others 2008).

Conclusions

Because pork and pork products are widely consumed in the United States and the world, it is crucial for producers, packers, and processors to be aware of the risk factors for contamination of products with foodborne pathogens. This study has outlined the common interventions that are currently being conducted at each step in pork production as well as more novel technologies that show promise for greater use. *T. spiralis* and *T. gondii* are excellent examples of the effectiveness that preharvest intervention strategies, especially farm management and biosecurity practices, can be in controlling pathogens and decreasing the prevalence of foodborne illness. *Salmonella* spp. and *Campylobacter* spp. will require not only on-farm interventions but hygienic slaughter practices and processing technologies to ensure a safe fresh and processed pork supply to consumers. Methicillin-resistant *S. aureus* is an emerging problem which will likely require both on-farm and processing technologies to control. The greatest challenge for processors is *Listeria monocytogenes* with its psychrotrophic and resistant abilities, but research and intervention technologies have been devoted to controlling it. There are still gaps in the control of many foodborne pathogens which will require a combined effort across the production chain and increased public awareness if foodborne diseases from pork are going to be eliminated.

Acknowledgments

We would like to thank Dr. Edward McGruder of Elanco Animal Health and Mr. Doug Roth of Elanco Animal Health for supporting this study financially.

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