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Intensive Swine Production and Pork Safety

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Abstract

Major structural changes in livestock production in developed countries, particularly intensive confinement production and increases in herd and flock sizes, have raised several societal concerns about the future directions and implications of livestock food production, including the safety of meat products. This review of the major parasitic and bacterial foodborne pathogens associated with pork production indicates that pork safety in the United States has improved demonstrably over recent decades. Most notably, changes in swine production methods have been associated with virtual elimination of risk of the foodborne parasites *Taenia solium*, *Trichinella spiralis*, and *Toxoplasma gondii* from pigs reared on modern intensive farms. This represents a substantial public health achievement that has gone largely unheralded. Regulatory changes have led to demonstrably lower prevalence of *Salmonella* on pork carcasses, but control of bacterial foodborne pathogens on farms remains a significant challenge. Available evidence does not support the hypothesis that intensive pork production has increased risk for the major bacterial foodborne pathogens that are common commensals of the pig (*Salmonella*, *Campylobacter*, *Listeria*, and *Yersinia enterocolitica*), or that pigs produced in alternative systems are at reduced risk of colonization with these organisms. However, pigs raised in outdoor systems inherently confront higher risks of exposure to foodborne parasites, particularly *T. gondii*.

Introduction

Livestock production in developed countries continues to undergo major structural changes, including marked reductions in numbers of farms, corresponding increases in herd and flock size, and specialization by species. The fundamental impetus for these changes has been economic, and considerable increases in efficiency (biological and managerial) have been achieved. In the United States, the inflation-adjusted price for market hogs has declined by ~50% over the last 25 years. In some countries, notably the United States, we have concurrently seen substantial concentration of ownership and vertical integration of meat industries, together with more contractual production and formation of supply chains (Kliebenstein and Lawrence, 1995). In the eyes of some of the public, this modern landscape of intensive livestock production is esthetically less appealing than that of earlier eras marked by predominance of small, mixed enterprise, farms. For others, it is wholly unacceptable. Platforms of opposition to intensive livestock production are diverse: sociological (e.g., impact on rural communities, and anticorporate sentiment); ethical (e.g., questioning the acceptability of animal housing conditions, traditional farming practices such as castration, and carnivorism itself); environmental (odor, pollution, and carbon footprint); and sanitary (zoonotic and emerging disease, and occupational health and food safety). All industries need critics and criticism, and societal pressures to change livestock production currently have considerable momentum via both legislative and commercial avenues, and in the battle to sway public opinion. Inevitably, all parties lay claim to science, yet discussions of these complex issues are mostly ideological and heavily value laden. Amalgamation and blurring of multiple issues is the norm, with food safety implications frequently invoked with scant or selective attention to facts.

Misinformation in public discourse has achieved pandemic potential with the rise in blogging and other social networking tools. For science to retain influence in public discourse, the scientific community needs to be more visibly engaged in refuting misinformation as well as presenting new information. To this end, synthesis and clarification of elemental information may be more vital for informing societal debate than communication of advances at the cutting edge. Negative implications of modern confinement swine production for food safety are commonly cited in the media and blogosphere, but to date there has been no comprehensive assessment of available data for specific foodborne hazards. Consequently, I have attempted to summarize current knowledge about major foodborne hazards in the context of pork industry evolution in the United States. My thesis is that pork safety in the United States has improved demonstrably over recent decades. For some foodborne hazards this has

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been the direct result of management practices employed in modern industry. For others, progress is attributable to changes implemented beyond the farm gate, including greater discipline imposed by government regulations and export markets. Hopefully, these perspectives will spur informed critique and help direct public discourse on pork safety toward greater factual accountability.

Parasitic Foodborne Hazards

Throughout human history, the most harmful porkborne hazards have been, and in parts of the world remain, parasites. However, contemporary commentary on pork safety in the United States rarely acknowledges the public health significance of these pathogens and the dramatic decline in risk of foodborne parasitoses that has been achieved.

Taenia solium *("Pork measles")*

At an international meeting on pork safety in Greece in 2003, Dr. Isaac Phiri, a veterinary parasitologist from Zambia and the sole African delegate, advised his audience, "You people need to come to Africa to see what a public health problem looks like" (Phiri *et al.*, 2003a). Until then, the focus of the meeting, mostly of European and North American delegates, had been on *Salmonella* and other bacterial pathogens, and the potential threat of antimicrobial resistance. Dr. Phiri's subject, *T. solium*, is a cestode tapeworm that lives only in the intestines of humans (García *et al.*, 2003; Phiri *et al.*, 2003b). Pigs are intermediate hosts for this parasite, and develop cysts in the muscles and other tissues after ingesting tapeworm eggs shed in human feces. In turn, people acquire intestinal tapeworm infections by eating undercooked pork that contains the cysts (Phiri *et al.*, 2003b). However, people can also acquire the cystic form of the disease (cysticercosis) if exposed directly or indirectly to infested human feces. The cysts can form throughout the human body, but most importantly in the brain and eyes. Neurocysticercosis (disease caused by worm cysts in the nervous system) is the main cause of seizures in many developing countries, accounting for up to 50% of cases. This dreaded infection is primarily responsible for the higher rates of epilepsy observed in developing versus developed countries. In India alone, a million people are estimated to suffer seizures because of infection with this parasite (Rajshekhar *et al.*, 2003). Besides seizures, cysticercosis causes headaches, raised intracranial pressure, psychiatric manifestations, ocular symptoms, and focal neurologic deficits (Rajshekhar *et al.*, 2006). The Food and Agricultural Organization cites estimates that some 50 million people worldwide harbor the adult tapeworm, and up to 50,000 deaths per year are attributable to cysticercosis (Aubry *et al.*, 1995; Eddi *et al.*, 2003).

Arguably, the most lamentable and preventable public health problem linked to pork consumption globally, *T. solium*, is essentially off the radar screen of U.S. food safety dialog. However, the Centers for Disease Control and Prevention (CDC) reported 221 fatal cases in the United States between 1990 and 2002, mostly in immigrants who were exposed before entering the United States (Sorvillo *et al.*, 2007). However, 33 of the deaths were in U.S.-born patients, and both foreign travel to endemic regions and contact with tapeworm-infested individuals within the United States were implicated as sources. The same authors predicted that cysticercosis will increase in importance in the United States due to high rates of migration from endemically infected countries. This parasite thrives where sanitation is poor and traditional, free-range/scavenging pig production is practiced (García *et al.*, 2003; Phiri *et al.*, 2003b; Sikasunge *et al.*, 2007). Gonzalez *et al.* (2003) posit that elimination of *T. solium* from Europe and North America was largely a consequence of economic development that made small-scale, subsistence pig rearing uneconomic. Simple corralling of pigs in endemic countries can markedly reduce the prevalence of infection in pigs (Gonzalez *et al.*, 2003), and methods of modern confinement swine production virtually eliminate any risks of porkborne transmission of *T. solium*. The sole potential concern of inappropriate defecation in swine barns by tapeworm-infested farm workers is recognized by the U.S. swine industry and has been acknowledged in producer education efforts.

Trichinella spiralis

T. solium is not the worm in pork that most troubled our mothers and grandmothers. A completely different nematode parasite, *T. spiralis* (or trichina), also forms cysts in tissues of pigs, but has a broader host range. Natural infections with trichina have been documented in over 150 mammalian species (the genus *Trichinella* includes 11 genotypes among 8 species, but only *T. spiralis* is important in domestic livestock). In the late 19th century, consumption of trichina-infested pork caused some 8000 human illnesses, and >500 deaths in Germany alone (Campbell, 1983). The outbreaks had immense political fallout, spawning government regulation of meat inspection in Europe, bitter trade disputes between Europe and the United States (then, as now, a major pork exporter), and an enduring awareness that it is prudent to cook pork appropriately. During the 1940s, about 400 clinical human trichinosis cases (and 10–15 deaths) were recorded annually in the United States. The incidence declined to an average of 57 cases per year from 1982 to 1986, including just 3 fatal cases over the 5 years (Bailey and Shantz, 1990). This reduction was achieved through better control of trichina infections on pig farms, which has since continued. Over 10 years from 1997 to 2006, only 138 human trichinosis cases (none fatal) were recorded by the CDC (Roy *et al.*, 2003; Kennedy *et al.*, 2009). This represents more than a 95% reduction in annual cases since the 1940s, and a 76% reduction in incidence since the 1980s. Consumption of wild game (particularly bear meat) is now the most common source of infections. Only 15 cases over the 10 years were linked to domestic commercial pork products, and 9 cases were associated with consumption of noncommercial pork from "home-raised or direct-from-farm swine" (Roy *et al.*, 2003; Kennedy *et al.*, 2009). Using the upper limit of a published estimate of 500,000–750,000 pigs produced in niche pork systems in 2006 (Honeyman *et al.*, 2006), compared with ~100 million pigs per annum from commercial systems, this implies an 80-fold greater risk (per pig produced) of trichina infections resulting from eating niche market versus commercial pork products in this country. Studies in Europe similarly show that trichina infections occur only in rural areas of Western Europe in association with traditional swine-rearing practices, but not in industrialized pig farms (Pozio *et al.*, 1996), and that "modern or industrialized farming employing hygienic measures and strict rules of good production

practices combined with good veterinary practices will exclude the risk of transmitting *Trichinella*" (van Knapen, 2000).

It is inevitable that pigs with outdoor access will be at greater risk of *Trichinella* infection due to exposure to wildlife reservoirs (Burke *et al.*, 2008; Ribicich *et al.*, 2009). Even with nonconfinement production in the United States, the absolute risk of acquiring *T. spiralis* from pork is very low, and effective cooking will destroy these parasites (as well as other foodborne pathogens). However, it is irrefutable that modern swine production systems (including regulated feeding practices and improved management and rodent control) have practically eliminated the risk of infection with *Trichinella* in commercial pork in the United States and other developed countries.

Toxoplasma gondii

It is estimated that throughout history about "one third of humanity" has been infected with the protozoan parasite *T. gondii* (Dubey, 2008). Although rarely affecting healthy individuals, *Toxoplasma* infections of pregnant women can cause miscarriages and stillbirths, as well as severe brain abnormalities and mental retardation in surviving newborns. In the United States, an estimated 400–4000 children are born with congenital *T. gondii* infection each year (Lopez *et al.*, 2000), and in 1990 the societal cost of congenital toxoplasmosis alone was estimated to be up to $8.8 billion annually (Roberts and Frenkel, 1990). Together with *Salmonella* and *Listeria*, *Toxoplasma* is one of three pathogens determined to be responsible for three-quarters of fatal foodborne infections in the United States (Mead *et al.*, 1999). Life-threatening encephalitis caused by reactivation of latent infections can occur in immunocompromised patients (New and Holliman, 1994), and *Toxoplasma* infections also cause severe ocular disease, including blindness. Ocular disease is associated mainly with postnatal infections of healthy individuals, over 1.2 million Americans may be affected, and the incidence of new cases of symptomatic ocular toxoplasmosis is of the order of 100 cases per week (Jones and Holland, 2010).

The epidemiology of *Toxoplasma* is more complex than that of *Taenia* or *Trichinella*. In addition to a host-range spanning all warm-blooded creatures, there are multiple means of transmission, including foodborne, waterborne, transplacental, and environmental (Dubey, 2008). Wild and domestic felids are the only species in which the parasite reproduces sexually in the intestine, yielding large numbers of environmentally resistant oocysts in feces (Dubey, 2008). Infection of nonfelid animals with *Toxoplasma* results in cysts (bradyzoites) in multiple tissues, including muscle, and the contribution of these species to transmission of *Toxoplasma* is predominantly, if not entirely, via carnivorism.

Environmental contamination with *Toxoplasma* oocysts is ubiquitous, and the annual environmental load from cat feces was estimated to be 94–4671 oocysts per square meter in an area of California (Dabritz *et al.*, 2007). Foodborne exposure can occur via consumption of oocyst-contaminated produce as well as by eating raw or undercooked meats. Attribution of foodborne illnesses to specific sources is typically problematic and often relies on expert opinion (Hoffmann *et al.*, 2007). This is particularly true for *Toxoplasma* cases because outbreaks are uncommon, people can acquire *Toxoplasma* via multiple routes, and clinical signs are usually absent or substantially delayed after exposure. One U.S. study found that *Toxoplasma* seroprevalence in a vegetarian population (24%) was approximately half that of the general population in the same area (Roghmann *et al.*, 1999). The assumption that 50% of toxoplasmosis is foodborne has been used to estimate the cost of foodborne toxoplasmosis in the United States (Roberts and Frenkel, 1990; Mead *et al.*, 1999).

Experts have long contended that of the major commercial meats, pork presents the greatest risk for *Toxoplasma* exposure in this country (Dubey, 1991; Velmurugan *et al.*, 2009). A national survey of samples from 1983/84 found that 23% of market hogs and 42% of sows were seropositive for *Toxoplasma* (Dubey, 1991). Subsequent national studies by the U.S. Department of Agriculture (USDA) found that 3% of market hogs were seropositive in 1995 (Patton *et al.*, 1996), 0.9% in 2000 (Pyburn, 2004), and 2.6% in 2006 (Hill *et al.*, 2010). Some of the variability in later studies may be due to differences in serological methods. A 1995 study of hogs reared in total confinement in North Carolina found only 1 (0.06%) seropositive pig among 1752 (Davies *et al.*, 1998). A decline in *Toxoplasma* seroprevalence has also occurred in Ontario, Canada (Poljak *et al.*, 2008), and studies in several European countries associate marked declines in *Toxoplasma* seroprevalence with intensification of pork production (Tenter *et al.*, 2000). A 2008 serological study of over 74,000 market hogs in the United States reported a seroprevalence of 0.8%, with prevalence declining with increasing farm size. The highest prevalence (2.6%) was found in pigs sourced from systems producing <1000 pigs per year, compared with 0.5% prevalence in the largest systems (McKean *et al.*, 2009).

Reliable data on the occurrence of *Toxoplasma* in pork are sparse. Small studies in the 1960s reported that 24% of samples of diaphragm and 32% of pork loins were positive (Dubey, 1986), and that 17% of samples of hearts from sows slaughtered in Iowa between 1989 and 1992 contained *Toxoplasma* (Dubey *et al.*, 1995). In a comprehensive study in 2005, only 8 of 2094 retail pork samples (0.38%) were found to contain viable bradyzoites (Dubey *et al.*, 2005). Two of the eight positive samples were labeled "naturally raised pork." The dramatic reduction seen in *Toxoplasma* prevalence in commercial swine production has been underpinned by changes in pig farming systems. Many routine practices in modern swine farms (confinement rearing, systematic rodent control, more hygienic feed handling procedures, exclusion of cats, and other biosecurity measures) combine to reduce the risk of exposure of pigs to *T. gondii*. Over the last two decades when *Toxoplasma* prevalence in commercial market hogs has declined by about 90%, there has also been a concomitant decrease in the incidence of human *Toxoplasma* infections (Jones *et al.*, 2007). At least part of this reduction in human *Toxoplasma* risk is likely the result of better control in the commercial swine industry.

As with *Trichinella*, the inevitability that pigs with outdoor access will be at elevated risk of *Toxoplasma* infection is consistently reflected in studies from various countries (Gamble *et al.*, 1999; Hove *et al.*, 2005; van der Giessen *et al.*, 2007; Gebreyes *et al.*, 2008; García-Bocanegra *et al.*, 2010). Studies of wild or feral pigs report a mean prevalence of around 20% (Dubey, 2009), and very high prevalence (>90%) of bradyzioites in pork may still occur in pigs reared in less controlled conditions (Dubey *et al.*, 2002). Similarly, elevated risk of *Toxoplasma* infection is well documented in free-range chickens

compared with confined birds. Indeed, measuring *Toxoplasma* prevalence in free-range chickens is now an accepted approach for quantifying soil contamination with *Toxoplasma* oocysts (Dubey *et al.*, 2004, 2008). The inherent trade off between parasite risk and the desired attributes of nonconfinement livestock systems is captured in the following quotation about the potential for producing *Toxoplasma*-free meat: "Modern production technologies have shown that this is feasible and have led to a marked decrease of *T. gondii* infections in meat producing animals such as pigs. Conversely, demand for animal friendly production systems may however lead to a re-emergence of *T. gondii* in pork and poultry" (Kijlstra and Jongert, 2009).

Bacterial Foodborne Hazards

Taenia, *Trichinella*, and *Toxoplasma* are parasites that evolved with carnivorous transmission as an integral or necessary component of their life cycles. Their presence in meat from farmed animals is solely determined by exposures on farms, and effective prevention can be achieved through preharvest interventions. In contrast, the bacterial pathogens of most concern to pork safety, *Salmonella*, *Campylobacter*, *Listeria*, and *Yersinia enterocolitica* (Fosse *et al.*, 2009), have their primary ecological niche in the intestinal tracts, not the muscles, of healthy birds and mammals. Their presence in meat stems from contamination events that can occur anytime during harvest and processing or up until meat is served on a plate (Davies *et al.*, 2004; Reij and Den Aantrekker, 2004). Contamination risk during processing is clearly a function of on-farm exposure; therefore, both preharvest and postharvest interventions are logical targets for control (Alban and Stärk, 2005). However, the more complex epidemiology of enteric bacteria, relative to parasites, yields them less amenable to preharvest control (Davies *et al.*, 2004; Callaway and Oliver, 2009; Oliver *et al.*, 2009). Therefore, we will first look briefly at postharvest factors that have had measurable impact on risk for these organisms in the U.S. pork supply.

Postharvest control of bacterial hazards

The Food Safety and Inspection Service (FSIS) of the USDA issued the Pathogen Reduction; Hazard Analysis and Critical Control Point (HACCP/PRA) Systems Final Rule on July 25, 1996. This was without question the most significant regulatory event for meat safety in the United States for almost 100 years (Billy and Wachsmuth, 1997; Sofos, 2009). Specific organisms were declared to be adulterants of raw meat products, and product-specific limits on *Salmonella* were established based on national surveys, including for market hogs (USDA-FSIS, 1996). The limit of $<8.7\%$ prevalence of *Salmonella* on market hog carcasses became effective in large plants on January 26, 1998; in small establishments on January 25, 1999; and in very small establishments on January 25, 2000. Since 1998, FSIS has recorded data (now available quarterly and grouped by size of enterprise) that document progress in reducing *Salmonella* prevalence on hog carcasses over the last decade (USDA-FSIS, 2010). In 2009, the prevalence of *Salmonella* on carcasses at large packing plants (which process a large proportion of pork products in the United States) was 1%, less than one-eighth of the baseline prevalence in the mid-1990s. Although prevalence remains somewhat higher in smaller enterprises, it is also substantially below the original baseline of 8.7%. One may question the acceptability of 10–20 pig carcasses per thousand being positive for *Salmonella*, but these data clearly indicate that the risk of *Salmonella* contamination of hog carcasses in the United States was substantially lower in 2009 than 10–15 years previously.

Unlike in Europe, there has been no mandated effort to control *Salmonella* in live hog production in the United States. The observed reduction may therefore be entirely attributable to improved hygiene at packing plants instituted in response to the HACCP/PRA regulations, and particularly to interventions aimed at reducing fecal contamination. Logically, commensurate reductions in contamination should have occurred for other enteric foodborne organisms that are commensals of the swine digestive tract (e.g., *Campylobacter*, *Listeria*, and *Y. enterocolitica*). In 1996, the CDC initiated an active, population-based surveillance program (FoodNet) for confirmed human cases of several foodborne pathogens. Between 1996 and 1998 (baseline) and 2008, the incidence of human cases of *Salmonella* has remained unchanged (although serotype changes were observed), indicating that measurable reductions in *Salmonella* contamination of meat and poultry have not translated into reduced human disease (CDC, 2009). A similar scenario occurred in Australia, highlighting the difficulty of linking public health outcomes to "upstream" interventions in a complex food supply (Sumner *et al.*, 2004). This may in part be explained by the fact that in the United States $<30\%$ of *Salmonella* outbreaks from 1998 to 2006 were meatborne, with only 5% attributable to pork products (Dreyfuss, 2009). However, FoodNet data in the United States do indicate that the incidence of *Yersinia* cases decreased by $\sim 50\%$ between 1998 and 2004, and *Listeria* and *Campylobacter* incidence decreased by $\sim 30\%$ (CDC, 2009). It is likely that at least part of these reductions in incidence of clinical disease is attributable to improved meat hygiene. This is particularly likely for *Yersinia*, as pigs are the only known livestock reservoir of pathogenic strains of *Y. enterocolitica* (Bhaduri *et al.*, 2009).

Preharvest control of bacterial hazards in pig production

The allure of preharvest strategies to control foodborne disease rests on the premise that specific alterations in farming practices can reduce the risk of foodborne hazards occurring in animals used for food (Davies *et al.*, 2004; Callaway and Oliver, 2009; Oliver *et al.*, 2009). Conversely, inappropriate practices in production systems that increase the prevalence of foodborne hazards in animals would logically increase foodborne risks to consumers. As indicated above, preharvest strategies are unequivocally effective for controlling parasitic hazards, and are also mandatory to prevent physical and chemical hazards that originate on farms. In contrast, progress in preharvest control of enteric bacterial organisms has been slow, and reliably effective interventions have not yet been convincingly demonstrated.

Over the last 15 years, preharvest control of bacterial pathogens has been an active field of research in all major livestock species. However, reviews of progress typically emphasize the complexity of the epidemiology of these organisms, that some interventions show promise and that more research is necessary (Callaway and Oliver, 2009; Fosse *et al.*, 2009; Oliver *et al.*, 2009). In pigs, there has been too little

epidemiological research on *Campylobacter*, *Listeria*, or *Yersinia* to assess the influence of management factors on risk (Fosse *et al.*, 2009); therefore, further discussion on pigs will predominantly focus on *Salmonella*. Oliver *et al.* (2009) succinctly summarized the state of preharvest food safety as follows "Research and educational efforts identifying potential on-farm risk factors will better enable producers to reduce or prevent foodborne pathogen contamination of products leaving the farm. The identification of on-farm reservoirs and intervention strategies will aid in implementing farm-specific pathogen reduction programs. There is little doubt that solutions to these and many other complex issues will be delineated through science-based research that will be conducted during the next century." Salient points in this appraisal are the repeated use of the future tense and the 100-year time frame. The rather glacial pace of tangible progress in preharvest control of bacterial foodborne pathogens has done little to dent the missionary zeal for preharvest control that burgeoned among consumer groups and the scientific community during the mid-1990s. It is not difficult to find published scientific reviews or risk assessments of otherwise high caliber that foster the perception that practical and effective methods for controlling *Salmonella* on pig farms are readily available (Ojha and Kostrzynska, 2007; Delhalle *et al.*, 2009; Fosse *et al.*, 2009). Typically authors cite risk factors from epidemiological research reporting associations found in observational studies. Most of the observational studies cited have been cross-sectional and had other methodological limitations that (1) make them questionable for causal inference and (2) provide no evidence that these factors are efficacious as practical interventions. Further, with the possible exception of feed-related interventions, the consistency of identified risk factors across studies has been limited. The scarcity of randomized controlled trials (unsurprising given the cost and difficulty of conducting such studies in farm environments) greatly constrains confidence that putative interventions can be applied reliably in the field.

Detailed reviews by epidemiologists with substantial experience as researchers in swine production have highlighted the paucity of proven management interventions for implementation on farms (Funk and Gebreyes, 2004; Mounchili *et al.*, 2006; O'Connor *et al.*, 2008; Fosse *et al.*, 2009). A systematic review of interventions in feeding practices, which have been often identified in observational studies and are frequent recommendations for preharvest control of *Salmonella* in pigs (including feed withdrawal before slaughter, feed acidification, heat treatment of feed, pelletized feed vs. mash, coarse vs. fine grind, and wet vs. dry feeds), concluded that there is a lack of strong evidence of any association between the potential interventions and *Salmonella* prevalence. Even for the best supported factor (feeding of nonpelleted feed), the authors stated that "there should be a low level of comfort among qualified scientists that the claimed association between non-pelleted feed and reduced *Salmonella* prevalence is scientifically valid" (O'Connor *et al.*, 2008).

Finally, perhaps the most convincing body of information questioning the wisdom of investing resources in preharvest control of *Salmonella* in swine production lies in wealth of data and experience generated from the Danish national *Salmonella* control and surveillance program. Initiated in 1993, founded largely on the desire to control *Salmonella* at farm level (Mousing *et al.*, 1997), several analyses of outcomes of this program indicate that resources are better directed at improving hygiene in slaughter processing (Goldbach and Alban, 2006; Hurd *et al.*, 2008; Baptista *et al.*, 2010a, 2010b). Of four strategies for *Salmonella* control used by the Danish pork industry (hot-water decontamination of carcasses; sanitary slaughter for farms with high *Salmonella* prevalence; use of home-mixed feeds; and use of acidified feed for slaughter pigs), only hot-water decontamination of carcasses was determined to be "socio-economically profitable" (Goldbach and Alban, 2006). Further, the most recent analyses from this program concluded that large reductions in the numbers of *Salmonella* seropositive pigs delivered to slaughter led to only small reductions in the risk of *Salmonella*-positive carcass tests, and that future strategies should focus on abattoir interventions (Baptista *et al.*, 2010a). This conclusion is coherent with a large body of research identifying exposure of pigs to *Salmonella* after leaving the farm, and particularly the abattoir environment, as a major source of contamination (Galton *et al.*, 1954b; Hurd *et al.*, 2001; Rostagno *et al.*, 2003).

The least equivocal outcome from the last 15 years of preharvest research in swine production is that elimination of organisms that are normal flora or commensals of the swine intestinal tract will not be achieved by facile interventions in farm management. Indeed, rather than employing the rather blunt instrument of observational research into farm management practices, preharvest research may be better directed at understanding the biological phenomena that underpin prolonged colonization of pigs by bacterial foodborne pathogens, and with specific measures targeting destruction of pathogens immediately before marketing (Boyen *et al.*, 2008; Callaway *et al.*, 2008; Stevens *et al.*, 2009). From a farm to table perspective, it remains uncertain whether preharvest interventions will ultimately prove cost effective relative to interventions applied later in the food supply chain.

Intensive Swine Production and Salmonella *Risk*

The paucity of validated preharvest interventions for *Salmonella* in swine does not mean that farm facilities and management do not influence *Salmonella* risk, but rather that the complexity of *Salmonella* epidemiology does not lend itself to simplistic interventions for control (Davies *et al.*, 2004). Most commentary claiming adverse effects of modern swine production systems on pork safety rests on assumptions that factors such as confinement rearing, stocking density, and herd size increase transmission of foodborne pathogens among animals and therefore increase risk to consumers. Although theoretically plausible, it is important to examine the evidentiary support for these assumptions, including the magnitude of the putative effects. Unfortunately, there is sparse research directly comparing confinement (or intensive) and nonconfinement (or less intensive) production of pigs, so I will approach the question from a broader perspective. Relevant comparisons include *Salmonella* prevalence in (1) domestic pigs versus wild or feral pigs; (2) historic versus contemporary domestic pig populations and pork products in developed countries; (3) domestic pigs in developed versus less developed countries; and (4) domestic pigs raised in different production systems in developed countries. A major caveat in making these comparisons is that methodological differences in sampling and testing greatly influence apparent prevalence (Davies *et al.*, 2000; Funk *et al.*, 2000), and any

Table 1. *Salmonella* Prevalence in Pigs Before 1960

Year	*Location*	*Sample*	*Prevalence (%)*	*Reference*
1936	Uruguay	Ab—MLN	50	Hormaeche and Salsamende (1936)
1942	United States	Ab—MLN	40	Rubin *et al.* (1942)
1951	England	Ab—fecal	0.67	Smith and Buxton (1951)
1954–1956	England	Ab—various	25	McDonagh and Smith (1958)
1954	United States	Farm—rectal swabs	27	Galton *et al.* (1954b)
		Ab—rectal swabs	78	
1959	Northern Ireland	Farm—rectal swabs	9	Newell *et al.* (1959)
1959	England	Ab—MLN	12	Smith (1959)

Ab indicates abattoir sampling; Farm indicates sampling on farm.
MLN, mesenteric lymph nodes.

inferences drawn from studies using different methods are necessarily qualitative rather than quantitative, and must be made cautiously.

Salmonella *in wild pig populations*

Recent studies of wild pig populations reported *Salmonella* seroprevalences of 19% in Italy (Montagnaro *et al.*, 2010) and 47% in Slovenia (Vengust *et al.*, 2006) using the IDEXX HerdCheck ELISA. In contrast, using the same test (but on meat juice rather than serum samples), a large study of commercial market pigs in the U.S. midwest ($n = 27{,}964$ pigs) reported a prevalence of 7.4% (O'Connor *et al.*, 2006). A similar seroprevalence (median within herd prevalence of 8%) was reported in a study of 369 commercial pig herds across five European countries (Lo Fo Wong *et al.*, 2004). A polymerase chain reaction-based study of wild boar in Switzerland (Wacheck *et al.*, 2010) found 12.6% of pigs to be positive for *Salmonella*, and also high prevalences of *Y. enterocolitica* (35%), *Yersinia pseudotuberculosis* (20%), and *Listeria monocytogenes* (17%). All pigs tested negative for *Campylobacter*, which is surprising given that *Campylobacter coli* is widely considered to be normal flora of pigs. However, Wahlström *et al.* (2003) cultured *Campylobacter* from 12% of wild boars in Sweden, and further studies are needed to document the prevalence of *Campylobacter* in wild pig populations. However, these overall findings support the general paradigm that major bacterial foodborne pathogens are common commensals of wild pigs in their natural environments, and their presence in domestic herds is not an artifact of farming practices.

Historic studies of Salmonella *in pigs and pork*

Intensification of swine production has increased greatly in most developed countries since the 1960s, and reduction in numbers of herds, with concomitant increases in herd sizes, has continued over the last 50 years. I have been unable to identify published surveys of *Salmonella* prevalence of national scope before the era of widespread confinement pig production. However, individual reports using convenience sampling indicate that *Salmonella* was a highly prevalent organism in pigs before intensive production methods were developed (Table 1). This includes prevalences from 27% to 78% in early U.S. studies. More comprehensive surveillance data have been published in Europe, United States, and Japan in recent years. An extensive survey including 19,071 pigs from 24 European countries (EFSA, 2008) found an overall bacteriological prevalence of 10.3% in the mesenteric lymph nodes of slaughtered pigs, although prevalence varied widely among participating countries (Table 2). In the United States, the USDA National Animal Health Monitoring System has published data from large surveys of fecal samples on swine farms. The observed bacteriological prevalence changed little between 1995 (6.2%) and 2006 (7.2%) (USDA-APHIS, 1997, 2008). In Asia, a 2008 national survey of pigs on 218 farms in Japan found a bacteriological prevalence of 3.1% in fecal samples (Kishima *et al.*, 2008). Although the different samples used in these prevalence surveys prohibit quantitative comparison of the European and U.S. data, and between eras, the key point is that the results from all these recent studies compare favorably with the data published on mesenteric lymph nodes or fecal samples respectively in studies conducted before 1960 (Table 1).

Table 2. Prevalence of *Salmonella* in Mesenteric Lymph Nodes of Pigs Slaughtered in Countries of the European Union and Norway (EFSA, 2008)

Country	*Prevalence (%)*
Austria	2.1
Belgium	13
Bulgaria	19.9
Cyprus	13.1
Czech Republic	5.8
Denmark	8
Estonia	6.4
Finland	0
France	18.5
Germany	12.7
Greece	21.2
Hungary	11.6
Ireland	15.4
Italy	16.4
Latvia	5.4
Lithuania	1.7
Luxembourg	16
Poland	6.4
Portugal	23.7
Slovakia	7.8
Slovenia	6.3
Spain	30.7
Sweden	1.5
The Netherlands	8.5
United Kingdom	21.8
Norway	0.2

Table 3. Reported *Salmonella* Prevalence in Pork Products in the United States

Year	*Sample*	*Prevalence (%)*	*Reference*
1943	Hog carcasses	6	Cherry *et al.* (1943)
	Pork chop	9.5	
	Sausages	2.3[a]	
1954	Hog carcasses	51	Galton *et al.* (1954a)
	Sausages—national distributors	7.5	
	Sausages—local distributors	57.5	
1969	Pork carcasses	56	Weissman and Carpenter (1969)
	Pork sausage (fresh)	38	
	Pork sausage (smoked)	9	
1984	Pork Sausage	27	Silas *et al.* (1984)
2001	Various[b]	9.6	Duffy *et al.* (2001)

[a]Silas *et al.* (1984) considered the values of Cherry *et al*. (1943) to be underestimates due to inferior isolation methods available for *Salmonella* at that time.
[b]Both whole muscle pork and sausage products (prevalence range 7.3% to 12.5% across product types).

From a public health perspective, it is also pertinent to look at temporal comparisons of *Salmonella* prevalence in carcasses or pork products rather than pigs. Again sampling and methodological differences among studies constrain inference. The most representative current data on *Salmonella* in retail meats in the United States come from the USDA-NARMS survey (Zhao *et al.*, 2006). In that study, 6% of retail meat samples (all major species) were contaminated with *Salmonella*, with the highest prevalence in ground turkey (52%) or chicken breast (39%), but prevalences were <2% in ground beef and pork chop samples over 5 years. However, Duffy *et al.* (2001) reported higher prevalences (7.3% to 12.5%) in a range of retail pork products in the United States. Table 3 lists earlier studies reporting *Salmonella* prevalence on hog carcasses or pork products in the United States. Apart from the 1943 study by Cherry *et al*. [considered unreliable by Silas *et al.* (1984) due to the inferior culture methods available in 1943], prevalences found in studies during the 1950s and 1960s greatly exceed those reported in recent surveys in the United States. Recognizing the limitations due to sampling, changing diagnostic methods over time (although culture sensitivity is unlikely to have declined), and possibly publication bias, a conservative inference is that there is no evidence to support claims that *Salmonella* infection of pigs, or *Salmonella* contamination of hog carcasses or retail pork products, is more prevalent in today's industry than in the era before widespread intensification of the pork industry.

Salmonella *in pigs in developing countries*

Table 4 lists results from prevalence studies of *Salmonella* in pigs in developing countries in Africa and Asia published in the last 5 years. Studies from Japan, South Korea, and Thailand were not considered, as these countries have relatively well-developed, modern pig industries. Similarly, due to the rapid expansion of intensive pig production in many Latin American countries, studies from these countries were not included. Recognizing that all studies cited have been abattoir based (and therefore reflect both on farm and abattoir influences), the results again show relatively high prevalence of *Salmonella* in the developing countries compared to the United States, Europe, and Japan, and further indicate that *Salmonella* is very common in pig populations of countries with generally less intensive swine industries.

Salmonella *in pigs produced in alternative versus intensive production systems*

For this discussion, I use the word "alternative" as a generic term for any categories of farms that have been designated by authors to differ from standard commercial production (variably described as niche, outdoor, animal friendly, organic, extensive, etc). There are relatively few studies that have directly compared the prevalence of bacterial foodborne pathogens in pigs reared under intensive conditions with pigs reared under alternative conditions. The enormous diversity of facilities and management practices that exists among

Table 4. Reported *Salmonella* Prevalence in Pigs in Recent Studies in Countries of Africa and Asia

Location	*Sample*	*n*[a]	*Prevalence (%)*	*Reference*
Kenya	Ab—fecal	58	8	Kikuvi *et al.* (2010)
	Ab—carcass	58	19	
Cameroon	Ab—GI tract[b]	30 samples	40% of samples	Akoachere *et al.* (2009)
Lao People's Republic	Ab—cecum	49	76	Boonmar *et al.* (2008)
Ethiopia	Ab—various	278	43% of pigs	Aragaw *et al.* (2007)
Vietnam	Ab—cecum	117	52.1	Le Bas *et al.* (2006)
	carcass	46	95.1	

Ab indicates abattoir sampling.
[a]Number of samples.
[b]Gastrointestinal tract—rectal, ileum, and gall bladder.

farms, particularly among alternative systems but also among intensive systems, makes the external validity (i.e., the ability to extrapolate the findings to populations beyond the study populations) of such studies dubious, and therefore any inferences again should be conservative. However, broad reviews of food safety implications of industrial versus alternative livestock systems did not identify any body of evidence indicating that bacterial foodborne pathogens are generally more prevalent in intensive swine farms than in pigs reared in alternative systems (Kijlstra *et al*., 2009; Young *et al*., 2009).

In a study comparing *Salmonella* prevalence in finishing pigs reared under different conditions on 29 North Carolina farms (all-in/all-out indoor barns [14 farms]; continuous flow indoor barns [7]; continuous flow barns with outdoor access, including concrete pens [4], dirt lots [3] and pasture [1]), more variability in prevalence was found within than between farm systems (Davies *et al*., 1997). However, the highest prevalence was found in the three herds raising pigs on dirt lots. Other studies in the United States have also reported higher bacteriological prevalence (Gebreyes *et al*., 2006) and seroprevalence (Gebreyes *et al*., 2008) of *Salmonella* in alternative systems compared with intensive operations. Similarly, van der Wolf *et al.* (2001) and Wingstrand *et al.* (1999) reported significantly higher *Salmonella* seroprevalence in pigs finished in alternative systems than intensive systems in Holland and Denmark. Other studies have reported no difference between alternative and intensive systems (Ledergerber *et al*., 2003; Zheng *et al*., 2007). In contrast, Meyer *et al.* (2005) reported mixed results, with seroprevalence highest in sows in organic herds, and in fattening pigs in indoor herds. However, the overall body of evidence does not support the contention that *Salmonella* risk is higher in modern intensive pig production than in the alternative production systems studied. This is not surprising given the widespread distribution of *Salmonella* in wild and domestic vertebrates, and its ability to persist in outdoor environments, including wallows (Callaway *et al*., 2005; Jensen *et al*., 2006b). Similarly, several studies have reported either higher prevalence of *Campylobacter* in alternative systems, or no difference between intensive and alternative systems (Gebreyes *et al*., 2005; Thakur and Gebreyes, 2005; Rollo *et al*., 2010). Interestingly, for both *Salmonella* (Jensen *et al*., 2004) and *Campylobacter* (Jensen *et al*., 2006a), there are reports indicating greater diversity of these bacterial pathogens in alternative systems than in confinement production, suggesting different reservoirs of infection and possibly increased food safety risk. Specifically, Jensen *et al.* (2006a) suggested that exposure of outdoor pigs to *Campylobacter jejuni* from the environment may cause a shift from a normal dominance of *C. coli* in pigs and be of concern for food safety. Although there is general consensus that control of *Listeria* should focus on food-processing establishments rather than on farms (Thévenot *et al*., 2006), a recent study from Sweden found *Listeria* prevalence was significantly higher in organic than in conventional pig production at both the farm and slaughterhouse level (Hellström *et al*., 2010).

Herd size and risk of foodborne pathogens

The importance of herd size as a determinant of disease frequency and disease emergence is among the most discussed yet least researched questions in swine production. Much of the discussion is based on "first principles" along the lines that "as a general principle, the concentration of humans or animals in proximity enhances the potential for transmission of microorganisms among members of the group" (Gilchrist *et al*., 2007). Although "herd size" effects are commonly assumed, and in some cases have been demonstrated, the biological basis for such effects is typically poorly expounded in published studies (Gardner *et al*., 2002). Despite the theoretical challenge, herd sizes in the United States have continued to increase, as have productivity levels. This apparent defiance of gravity may in part be attributed to the fact that increasing herd size has occurred in concert with (and likely depends upon) implementation of management practices that have positive benefits for herd health (e.g., all-in/all-out management, age segregation, improved biosecurity and hygiene, uniform sourcing of pigs). The confounding of herd size with such management factors complicates assessment of the impact of herd size on pathogen prevalence and disease. Herd size may be more important for transmission of respiratory than enteric pathogens (Gardner *et al*., 2002).

A large Danish study from several years of surveillance data found that *Salmonella* seroprevalence was positively associated with herd size, but the magnitude of the effect was biologically unimportant (Carstensen and Christensen, 1998). Similarly, the prevalence of *Salmonella* in pigs was comparable for small producers (7.3%) and large producers (8.1%) of pigs slaughtered in Iowa (O'Connor *et al*., 2006). In Holland, smaller herd size (<800 pigs per year) was associated with higher *Salmonella* seroprevalence than found in larger herds, whereas the opposite was seen in Spain (van der Wolf *et al*., 2001; Garcia-Feliz *et al*., 2009). A recent case–control study of high and low seroprevalence herds in the United Kingdom found that the odds of being a high seroprevalence herd were three to six times higher for farms with <1000 pigs inventory than in larger herds (Twomey *et al*., 2010). However, herd size was not retained in the final multivariate model, in which several biosecurity factors were associated with prevalence category. The significant association of these biosecurity factors, but not herd size, with *Salmonella* prevalence in this analysis suggests that better biosecurity practices on larger herds could explain the lower seroprevalence observed. Overall, conclusions about the impact of herd size have been mixed, and positive effects have been of relatively small magnitude and therefore questionable biological significance. There is currently no convincing evidence that herd size is an important risk factor for *Salmonella* in swine herds.

Antimicrobial Resistance

I have deliberately not addressed antimicrobial resistance in this review. Genes encoding for antimicrobial resistance are not foodborne pathogens, and the issues of food safety and antimicrobial resistance in the pork industry should not be conflated. People have always been exposed via food to bacteria carrying genes coding for antimicrobial resistance, and the advent of the antibiotic era logically has introduced some incremental risk to health. Likewise, foodborne transmission unquestionably has some role in the overall epidemiology of antimicrobial resistance. However, the magnitudes of these risks pale alongside the public health burdens of (1) direct foodborne illnesses and (2) clinical treatment failures due to antimicrobial resistance unrelated to food.

The enshrined estimate of the annual burden of foodborne illness in the United States is 76 million cases per year, 325,000 hospitalizations, and 5000 deaths, based on the 1997 population (Mead *et al.*, 1999). These estimates translate to approximately one case of foodborne illness per 4200 meals; one hospitalization for every 1 million meals; and one death for every 65 million meals, indicating that the U.S. food supply is indeed relatively safe. However, the aggregate burden of disease remains troubling because the low risk is borne by >300 million people. A frequently cited modeling study estimated that antimicrobial resistance in *Salmonella* and *Campylobacter* in the United States results in an additional 12 deaths (estimated 2% increase) from *Salmonella* infections and 95 hospitalizations (estimated 0.7% increase) with *C. jejuni* infections annually (Barza and Travers, 2002). These estimates may well be inflated as the authors assumed that 80% of human infections for both agents were from food animals. However, their cited source (Swartz, 2002) stated only that 80% of *Campylobacter* infections were foodborne (not just food of animal origin), and provided no estimate for *Salmonella.* Using outbreak data, Dreyfuss (2009) calculated that only 30% of *Salmonella* outbreaks involved meat. All illnesses and death are regrettable, but the estimates of Barza and Travers (2002) reflect a minor increment in health risk and position the health consequences of antibiotic resistance in *Salmonella* and *Campylobacter* substantially below lightning strike or dog bites as public health concerns in the United States.

Antibiotic resistance is a pressing problem in human medicine, but there is a marked dichotomy of emphasis in the medical literature on the subject. Analyses by infectious disease experts who confront antimicrobial resistance in clinical settings seldom refer to foodborne pathogens such as *Salmonella* and *Campylobacter* or the importance of animals as sources of resistant organisms (Cosgrove, 2006; Maragakis *et al.*, 2008). In response to a survey, senior medical microbiologists in the United Kingdom ranked *Salmonella* and *Campylobacter* 15th and 18th, respectively (out of 20 organisms) in clinical importance for antimicrobial resistance (Bywater and Casewell, 2000). In contrast, authors addressing antimicrobial use in agriculture predominantly focus on these two organisms, typically without meaningful context of their relative contribution to treatment failures due to resistance in human clinical medicine (Angulo *et al.*, 2004). Relative to the respective public health impacts of foodborne illnesses and antimicrobial resistant infections, their intersection is arguably trivial.

Summary

Eating, be it of animal- or plant-based products, will never be entirely risk free. Globalization and food industry evolution have provided consumers in developed countries with unprecedented choices of foods available year-round. As methods of producing, sourcing, processing, distributing, and preparing food have become more complex, it is inevitable that the profile of foodborne hazards and patterns of foodborne diseases will be altered. Substantial changes in pork production systems in developed countries have resulted in drastic reductions in the incidence of the foodborne parasitoses of *T. solium*, *T. spiralis*, and *T. gondii*. Control of bacterial foodborne pathogens in pork production remains a significant challenge in the preharvest sector. Currently available evidence does not support the hypothesis that intensive pork production has increased risk of *Salmonella*, *Campylobacter*, *Listeria*, and *Y. enterocolitica*. Reported prevalences of *Salmonella* in modern pork industries in developed countries compare favorably with those reported in studies of (1) wild pig populations, (2) developed countries before intensification of production, and (3) recent studies from developing countries with largely traditional industries. Currently available data do not indicate that pigs produced in alternative systems present reduced risk of foodborne hazards.

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