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MINIREVIEW

Population Dynamics of *Salmonella enterica* Serotypes in Commercial Egg and Poultry Production▽

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Fresh and processed poultry have been frequently implicated in cases of human salmonellosis. Furthermore, increased consumption of meat and poultry has increased the potential for exposure to *Salmonella enterica*. While advances have been made in reducing the prevalence and frequency of *Salmonella* contamination in processed poultry, there is mounting pressure on commercial growers to prevent and/or eliminate these human pathogens in preharvest production facilities. Several factors contribute to *Salmonella* colonization in commercial poultry, including the serovar and the infectious dose. In the early 1900s, *Salmonella enterica* serovars Pullorum and Gallinarum caused widespread diseases in poultry, but vaccination and other voluntary programs helped eradicate pullorum disease and fowl typhoid from commercial flocks. However, the niche created by the eradication of these serovars was likely filled by *S.* Enteritidis, which proliferated in the bird populations. While this pathogen remains a significant problem in commercial egg and poultry production, its prevalence among poultry has been declining since the 1990s. Coinciding with the decrease of *S.* Enteritidis, *S.* Heidelberg and *S.* Kentucky have emerged as the predominant serovars in commercial broilers. In this review, we have highlighted bacterial genetic and host-related factors that may contribute to such shifts in *Salmonella* populations in commercial poultry and intervention strategies that could limit their colonization.

Salmonella enterica infections are a significant public health concern worldwide, with an estimated 1.028 million cases, 19,000 hospitalizations, and ~400 deaths in the United States each year (106). Human salmonellosis is typically associated with the consumption of contaminated foods, such as fresh and processed meat and poultry, eggs, and fresh produce (8, 88, 110). Meat and poultry consumption has been on the rise in the United States, with the per capita consumption of poultry products increasing 6.5-fold since 1910 (19). An increase in consumption of meat and poultry increases the potential risk for exposure to *Salmonella* through contaminated food commodities.

Commercial poultry is one of the fastest growing sectors of the animal agricultural industry (66). In 2006, commercial poultry management systems represented 95% of poultry production in the United States (85). The majority of broilers in the United States are reared in large housing operations consisting of 6,000 to 40,000 birds per housing unit (66). Broilers are generally raised cage-free in barns on litter, with the stocking density ranging from 6.5 to 8.5 lb/ft^2 (0.27 to 0.36 kg/m^2) depending on the size of the birds (90). In the United States, over 9 billion broilers are hatched, raised, and processed each year (31). In 2009, over 77 billion table eggs were produced in the United States (113). Contaminated poultry, meat, and eggs are important vehicles of *Salmonella* infections, especially when the bacterium is in the egg contents. This contamination problem was recently highlighted in a 2010 salmonellosis outbreak caused by *S. enterica* serovar Enteritidis that was traced back to contaminated eggs from Iowa (76). Several factors can affect *Salmonella* colonization in poultry, including the age and genetic susceptibility of the birds, bird stress due to overcrowding or underlying illness, level of pathogen exposure (infectious dose), competition with gut microflora for colonization sites, infecting *Salmonella* serovar, and whether the strains carry genetic factors that facilitate attachment to the birds' gastrointestinal tracts or evade host defenses (3). Young birds are more susceptible to *Salmonella* colonization of the gastrointestinal tract during the first few days by vertical transmission from infected parents or by horizontal transmission at the hatcheries during feeding, handling, and transportation (3, 77, 96).

More recently, nonconventional poultry systems, including free-range, pasture, and organic, generally use slow-growing breeds which have a longer grow-out period than the conventional breeds (41). Consumer attitudes toward "natural" and "organic" products have created high demand for these commodities (67). Several definitions of nonconventional poultry production systems exist, and various housing units are used in these production systems, including fixed houses, portable houses, and pasture pens in free-range operations (41). Consequently, the potential exists for increased bacterial contam-

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ination on these nonconventional farms due to easier access to transmitting vectors, such as birds, rodents, insects, and/or wild animals (32, 79, 89).

Over the last several decades, there have been significant shifts in the predominant *Salmonella* serovars associated with poultry and human infections. Some of the most commonly detected serovars in chickens over the last 25 years, such as *S. enterica* serovar Enteritidis and *S.* Heidelberg, are also among the top five serovars associated with human infections (25, 43). More recently, *S.* Kentucky has become the most commonly detected serovar in chickens, while *S.* Typhimurium remains the most common cause of human infections (25). *S.* Typhimurium is one of the more common serovars detected in poultry (43); however, it has not historically been the predominant serovar in poultry, and as such, it will not be extensively covered in this review, which highlights the potential contributing factors associated with the changing population dynamics of *Salmonella* in poultry and egg production settings.

POPULATION SHIFTS IN *SALMONELLA* ASSOCIATED WITH POULTRY

In the early 1900s, poultry diseases caused by *Salmonella* serovars Pullorum (pullorum disease) and Gallinarum (fowl typhoid) were widespread in the United States (108). To combat these diseases, the National Poultry Improvement Plan (NPIP) was established in 1935 (115), and by the mid-1960s, these diseases had been eradicated from commercial flocks (6). One potential consequence of eradicating *S.* Gallinarum in poultry was the emergence of *S.* Enteritidis. Prior to the increase in *S.* Enteritidis infections in chickens, this serovar was associated primarily with rodents (21). Several potential explanations have been theorized for the emergence of *S.* Enteritidis, including that as flock immunity to *S.* Pullorum and *S.* Gallinarum declined following eradication, *S.* Enteritidis filled their ecological niche in commercial poultry and proliferated in the bird populations (6). In addition, mathematical models have suggested that *S.* Gallinarum competitively excluded *S.* Enteritidis in poultry (101). Both *S.* Gallinarum and *S.* Enteritidis express the immunodominant O9 lipopolysaccharide antigen on their cell surfaces, which may have contributed to the exclusion of *S.* Enteritidis. This exclusion could have been due to the increased ability of *S.* Gallinarum to colonize and/or survive due to an adaptive immunity in poultry (61, 119). Additionally, changing production practices in the poultry industry over the last several decades, such as higher bird densities and increased vertical integration, may have facilitated the increased spread of *S.* Enteritidis (3, 119).

While *S.* Enteritidis has remained a significant problem in commercial poultry, the prevalence of this serovar has declined in chickens in the United States since the mid-1990s. *S.* Heidelberg supplanted *S.* Enteritidis as the predominant serovar from 1997 to 2006, and in 2007, *S.* Kentucky was the most commonly isolated serovar (43). Both *S.* Heidelberg and *S.* Enteritidis colonize the birds' reproductive tracts and enter the eggs (55, 57, 58). One of the factors that likely contributed to the decline of *S.* Enteritidis is that the NPIP has targeted these bacteria for eradication in eggs since 1989 and in meats since 1994 (115). Additionally, the decline could be associated with an increase in flock immunity to *S.* Enteritidis, either due to exposure or vaccination of birds (29). If the immune responses are specifically directed at *S.* Enteritidis surface antigens, the prevalence of *S.* Heidelberg and *S.* Kentucky could potentially increase in the absence of *S.* Enteritidis. Similarly, *S.* Heidelberg shares some common surface antigens with *S.* Enteritidis that *S.* Kentucky does not (13), which may help explain why *S.* Kentucky has increased more rapidly than *S.* Heidelberg in recent years. Another factor potentially contributing to the emergence of *S.* Kentucky is the acquisition of virulence plasmids from avian pathogenic *Escherichia coli* (APEC) (47, 70). A large percentage of *S.* Kentucky strains isolated from chickens carry these plasmids, which appear to be important to both APEC and *S.* Kentucky for the colonization of poultry (70).

In addition to the NPIP, the FDA issued a final rule, entitled "Guidance for industry: prevention of *Salmonella* Enteritidis in shell eggs during production, transportation, and storage" in July 2009 (45). As *S.* Enteritidis is further targeted, there is potential concern as to what will fill the potential niche left after the elimination of *S.* Enteritidis from commercial poultry and egg production. With further steps to eliminate this pathogen from the human food supply, opportunities will likely exist for other serovars to proliferate and potentially cause diseases in humans.

POULTRY-ASSOCIATED SEROVARS THAT CAUSE HUMAN INFECTIONS

Fresh and processed poultry account for ~29% of all *Salmonella* infections in humans (12). The most commonly identified serovars associated with human infections in the United States are *Salmonella enterica* serovars Typhimurium, Enteritidis, Newport, Heidelberg, and I 4,[5],12:i:− (25). The number of *S.* Enteritidis infections in the United States increased dramatically starting in the 1980s, to the point at which *S.* Enteritidis became the predominant *Salmonella* serovar from human sources in 1994 (99). Among the nearly 41,000 *Salmonella* isolates from human sources reported to the Centers for Disease Control and Prevention (CDC) in 2006, *S.* Enteritidis was the second most commonly identified cause of infection, representing 16.6% of the cases (25). More recently, the CDC's Foodborne Diseases Active Surveillance Network (FoodNet) reported that *S*. Enteritidis caused 19.2% of all *Salmonella* infections in 2009 (24). When the data from 2009 were compared with 1996 to 1998 baseline numbers, *S.* Enteritidis infections increased by 32%, even though their rates declined from 1999 to 2003 (24). Studies have shown that contaminated shell eggs and egg products are the most important sources of *S*. Enteritidis (12, 23, 109, 111).

S. Heidelberg is also among the most commonly detected serovars from poultry and is among the top five serotypes associated with human salmonellosis (25, 46). Compared to other gastroenteritis-causing *Salmonella* serovars, which usually cause mild to moderate and self-limiting illness, *S.* Heidelberg tends to cause invasive infections. The FoodNet data from 1996 to 1999 indicate that *S*. Heidelberg was responsible for ~84,000 human infections per year in the United States, including 11% of all invasive cases of salmonellosis and 7% of the *Salmonella*-related deaths in the United States (second highest after *S*. Typhimurium) (73, 120). The incidence of human infections by *S*. Heidelberg increased by 20% from

1996 to 2005, even though the overall number of cases of salmonellosis decreased by 9% (22). However, since 2005, the incidence of *S.* Heidelberg infections has decreased such that in 2009, the overall incidence had decreased by 33% compared to 1996 baseline data (24). *S*. Heidelberg infections are likely caused by the consumption of contaminated meat, poultry, eggs, or egg-containing products (17, 28, 64). The FoodNet data indicated that the principal risk factor for *S.* Heidelberg infections is the consumption of eggs prepared outside the home (64).

S. Kentucky was commonly isolated from chicken carcasses during processing and from retail chicken breasts sampled as part of the National Antimicrobial Resistance Monitoring System (NARMS) program in 2007, representing 45% and 25% of all *Salmonella* isolates originating from these sources, respectively (46). However, in contrast to *S.* Enteritidis and *S.* Heidelberg, *S.* Kentucky is less commonly identified as a source of human salmonellosis, averaging about 62 cases per year from 1996 to 2004 before climbing to 123 cases in 2006 (25). The prevalence of *S.* Kentucky in chickens has increased from 25% in 1997 to ~50% in 2007 (46, 47). It is unclear as to why this serotype has become a prolific colonizer in chicken ceca but has not posed as significant a threat to humans as *S.* Typhimurium, *S.* Enteritidis, or *S.* Heidelberg (68). Although *S.* Kentucky is not among common *Salmonella* serovars causing human diseases, the prevalence of multidrug resistance (MDR) in *S.* Kentucky isolates from poultry is significant. According to the 2007 NARMS executive report, 50% of isolates from chickens that were resistant to ≥5 antimicrobials were *S.* Kentucky (46). When the overall resistance in *S.* Kentucky isolates from poultry-related sources was examined, over 8% demonstrated MDR, second only to *S.* Typhimurium (46).

GENETIC FACTORS CONTRIBUTING TO THE EMERGENCE OF *SALMONELLA* HEIDELBERG AND *SALMONELLA* KENTUCKY IN POULTRY

Infection and colonization of poultry. Of the more than 2,500 *Salmonella* serotypes, only a small proportion (~10%) is associated with the commercial egg and poultry industry (54, 82). In recent years, *S.* Heidelberg and *S.* Kentucky were the top serovars isolated from turkey and chicken samples, respectively (25). These two serovars have been frequently isolated from pre- and postharvest poultry sources (47, 80, 92). *Salmonella* can colonize the birds through fecal-oral transmission (42, 82); however, in newly hatched chicks, colonization can also take place via the nose or cloaca (14). Vertical transmission of *Salmonella* has been reported in infected ovaries, oviducts, or infected eggs; these infections may be asymptomatic in adult birds (82). Several risk factors, such as inadequate hygiene, contamination in the previously placed flock, contaminated day-old chicks, farm structure/management challenges, and contaminated production facility environments (feed, water, insects, air, litter, etc.) have been attributed to *Salmonella* contamination of broiler flocks (54, 80, 91).

Salmonella can multiply in the gastrointestinal tract of birds and contaminate the environment due to excretion of the bacteria through feces. These bacteria can also invade the intestinal mucosa, cecal tonsils, and Peyer's patches, survive and multiply in macrophages, spread to the liver and spleen via the bloodstream or lymphatic system, and eventually infect other organ systems (ovary, oviduct, gizzard, yolk sac, or lungs) (27, 38, 82). *S.* Heidelberg colonizes the reproductive tracts of layers and enters eggs through mechanisms similar to those of *S.* Enteritidis (55–57). *S.* Heidelberg can colonize the ovary and oviduct and penetrate and thrive inside the hen's egg (51, 58, 95). *S.* Typhimurium, *S.* Enteritidis, or *S.* Pullorum preferentially colonizes the reproductive organs (ovary and preovulatory follicles) of mature laying hens and causes higher mortality in chicks than *S.* Heidelberg or *S*. Kentucky (94, 105).

The invasion, colonization, and proliferation mechanisms involve several genetic changes in the bacteria (20, 52, 53). *Salmonella* can express acid shock proteins (RpoS σ-factor, PhoPQ, and Fur) for survival at a low gastrointestinal pH and exposure to short-chain fatty acids in the poultry gut (7, 38) as well as fimbria-associated proteins (Fim, Lpf, and Pef) to facilitate adhesion of the bacteria to the host intestinal cell surfaces (2, 33, 37). *Salmonella* can also express a type III secretion system (T3SS), which facilitates endothelial uptake and invasion within the host cells (50). The T3SS is associated with *Salmonella* pathogenicity island 1 (SPI-1), which comprises regulatory and effector virulence factors, such as *prgHIJK*, *spaMNOPQRS*, and *invABCEFGH* for adhesion, invasion, and toxin formation (62, 81, 86) and the SopB protein required for activation of secretory pathways and attraction of neutrophils to the sites of infection and causing diarrhea (93, 121). Other SPI-1 T3SS proteins, such as SipA, SopA, SopD, and SopE2, may also play an important role in *Salmonella*-associated gastroenteritis (121, 123). *S.* Kentucky exhibited greater invasive capabilities in *in vitro* assays involving chicken embryo hepatocytes than *Salmonella enterica* serovars Enteritidis, Typhimurium, Hadar, Mbandaka, or Senftenberg (68). Additionally, invasive infections caused by *Salmonella* spp. have also been associated with SPI-2 T3SS (42, 68). The genes from this system are exclusively expressed in the host cell *Salmonella*-containing vacuole (SCV) (75), and they encode the apparatus (*ssaGHIJKLMNOPQRSTU*), effector (*sseABCDEF*), chaperones (*sscAB*), and regulator (*ssrAB*) required for a functional T3SS (42, 65) and the *Salmonella*-induced filaments (SIF) motor proteins that may play a role in intracellular replication of *Salmonella* (1, 65, 75). The SPI-1 and SPI-2 T3SS genes have been detected in *S*. Heidelberg isolated from poultry-associated sources (125).

While few studies have examined the colonization factors of *S*. Heidelberg and *S*. Kentucky, several studies have identified genomic regions of other *Salmonella* serovars implicated in colonization of the avian gastrointestinal tract. In *S*. Typhimurium, genes implicated in colonization include the type III secretion system genes of SPI-1 and SPI-2 (71), *lpf* and *pef* (78), and lipopolysaccharide biosynthesis genes (112). In *S*. Enteritidis, genes implicated in colonization include the type VI secretion system genes of SPI-19 (9) and the type III secretion system genes of SPI-2 (122), *hilA* (10), and the genes encoding fimbrial types SEF17 and SEF21 (34). However, given that the colonization mechanisms differ between hosts, between serovars, and even within an individual serovar, there is still much to understand related to colonization mechanisms employed by *S*. Heidelberg and *S*. Kentucky within the avian host.

Role of plasmid-borne and chromosomal genes. Plasmids likely play a key role in the dissemination of antimicrobial resistance among *S*. Heidelberg, *S*. Kentucky, and other serovars (72, 83). In a recent study (63), *S*. Heidelberg isolates from chickens, turkeys, and humans were examined for pulsed-field gel electrophoresis (PFGE) profile, resistance phenotype, plasmid types, and the presence of genetic resistance determinants. Plasmids were detected in 88% of the isolates examined, with those belonging to the incompatibility (Inc) groups IncHI2 and IncI1 being the predominant types identified. *S*. Heidelberg has shown resistance to tetracycline, ampicillin, gentamicin, streptomycin, and sulfonamides (72, 84, 98, 124). The genes associated with these resistances included *tetB*, bla_{CMY}, bla_{TEM}, *aacC*, *aph*, *strA*, *sul1*, and *sul2* (63). Many of these traits have been localized to class 1 and class 2 integrons and identified among *S*. Heidelberg and *S*. Kentucky strains (74, 89, 98, 126). However, the prevalence of these resistance elements and their conferred phenotypes in *S*. Heidelberg appears to depend on the antimicrobial agents used at the production facility (40). In addition to their detection among *S.* Heidelberg strains, extended-spectrum β-lactamase-encoding genes, such as bla_{CMY}, bla_{TEM}, and bla_{CTX}, have also been recently detected in plasmids from *S*. Kentucky isolates from avian sources (44, 47, 59). In addition to the increasing identification of MDR-encoding avian *S.* Heidelberg clones among poultry and humans, these clones have also been identified on retail poultry meat, indicating a source for their zoonotic transfer to humans (124).

While the virulence and colonization mechanisms of *S*. Typhimurium and *S*. Enteritidis have been well studied, surprisingly little work has been done with *S*. Heidelberg and *S*. Kentucky. Genome sequencing has greatly increased our knowledge of *S. enterica*'s repertoire of core and accessory genomic components, including the plasmid complements. The accessory genomes of *S*. Heidelberg and *S*. Kentucky have been used to shed light on their capacity to colonize the avian host. Bronowski and Winstanley (15) used suppression subtractive hybridization (SSH) to identify genes specific to *S*. Heidelberg and not *S*. Typhimurium strain LT2, and they identified only two uncharacterized genes specific to *S*. Heidelberg. Furthermore, fimbrial operons, such as *tcf* and *stk*, were present in multiple serovars, including most of the *S*. Heidelberg isolates. This study also indicated that SSH genes are differentially expressed among the *S*. Heidelberg strains examined. Thus, while the presence or absence of certain genetic regions seems important for the ability of *S*. Heidelberg and *S*. Kentucky to colonize and invade the host (11), gene expression almost certainly plays an important role in these activities.

The sequenced plasmid complements of MDR strains *S*. Kentucky CVM29188 and *S*. Heidelberg SL476 revealed that they harbor numerous plasmids (47). Plasmid pCVM29188_146, isolated from *S.* Kentucky (chicken breast sample), shared a highly conserved (>90% nucleotide similarity) genetic backbone with virulence plasmids pAPEC-O1-ColBM and pAPEC-O2-ColV from APEC strains, suggesting that an *S.* Kentucky isolate from chicken may have potentially acquired virulence genes from APEC (47). The genes that are common to the *S*. Kentucky and APEC plasmids include *iutA*, *iucABCD*, *sitABCD*, *etsABC*, *iss*, and *iroBCDEN* (47). In *S.* Kentucky CVM29188, three plasmids were found to belong to the IncFIB/FIIA, IncI1, and IncFII types (47). The IncFIB/FIIA plasmid was a ColV plasmid harboring the *strAB* and *tetAR* resistance genes. The IncI1 plasmid carried the bla_{CMY-2} resistance gene. *S*. Heidelberg strain SL476 contained a small, cryptic plasmid and a 91-kb IncI1 plasmid. These and other reports (47, 63, 70) collectively suggest that *S*. Heidelberg and *S*. Kentucky have the propensity to acquire and disseminate multiple large plasmids encoding MDR and virulence.

Little work has been done to understand the biology of *S*. Kentucky in the avian host. In a comprehensive study, *S*. Kentucky was compared to other serovars for the presence of known virulence genes, invasiveness toward chicken embryo hepatocytes, growth in laboratory media, biofilm formation, stress response, and pH response (68). Of the traits examined, only the acid response phenotypes were found to differ between *S*. Kentucky and other serovars, such that *S*. Kentucky grew better than other serovars at pH 5.5 and worse than other serovars at pH 2.5. These results suggest that *S*. Kentucky might have a slight fitness advantage in locations where moderately acidic conditions exist, such as the chicken cecum (68). Other advantages appear to be conferred to *S*. Kentucky through its recent acquisition of an *E. coli* ColV virulence plasmid (69, 70). Fricke et al. (47) determined, through genome sequencing and gene prevalence studies, that most avian-source *S*. Kentucky isolates harbor this plasmid. Further analysis indicated that a single apparent clone of *S*. Kentucky exists among poultry isolates containing the ColV plasmid, which enhances the ability of these bacteria to colonize the chicken cecum and to persist in the avian extraintestinal environment (70). These studies provide clues to some of the mechanisms by which *S*. Kentucky colonizes and persists in the avian host, but more work is needed to elucidate the precise mechanisms by which these advantages are conferred.

INTERVENTIONS THAT MAY AFFECT THE PREVALENCE OF *SALMONELLA* IN POULTRY

Because of concerns with *Salmonella* in poultry, there have been a number of efforts to limit disease through different rearing/management practices, pre-/probiotic use, antimicrobial therapy, and/or vaccination of birds against *Salmonella* and other pathogens. When data from nonconventional (organic, free-range, etc.) and conventional farms are compared, *Salmonella* prevalence is dependent on the individual farm and not the farming system (4, 32, 118). Regardless of contamination levels, *Salmonella* serovars dominating conventional production systems also dominate nonconventional systems (79, 89, 118). Thus, management practices may influence the prevalence but not the serovar of *Salmonella* detected.

The use of vaccines in commercial poultry is increasing (54, 116). Due to public health problems associated with *S.* Enteritidis and *S.* Typhimurium, these serovars are the targets of most *Salmonella* vaccines. Both inactivated (killed) and attenuated (live) vaccines are available; however, neither type of vaccine provides complete protection or cross-protection against all serogroups (26, 54). Given that vaccines target *S.* Typhimurium (serogroup B) and *S.* Enteritidis (serogroup D), selection pressure for other prevalent serovars, including *S.* Kentucky (serogroup C), may intensify. Because the efficacy of a vaccine against antigenically dissimilar serovars is reduced

or absent, this strategy could enhance new problems caused by emerging serotypes by providing a vacant niche for other serovars to proliferate (54).

Vaccines, including genetically modified organisms, may be used in some organic (nonconventional) systems (114). However, for these systems, most growers prefer probiotics and prebiotics to control *Salmonella* (41). Probiotics are live cultures of beneficial microorganisms given to the birds in either the feed or water, and prebiotics are indigestible substrates that select for a specific population of beneficial bacteria (48, 60). These treatments attempt to manipulate the microbiota within the gut, which can provide protection against colonization of *Salmonella* in several ways: (i) production of antimicrobial substances (volatile fatty acids, bacteriocins, or hydrogen peroxide), (ii) reduction of the availability of niches for colonization, (iii) competition for limited nutrients, and (iv) stimulation of the immune system (35, 48, 60). In addition to limiting *Salmonella*, probiotics and prebiotics can also provide protection against other pathogens, including *E. coli*, *Yersinia enterocolitica*, and *Campylobacter jejuni* (107).

Probiotics and prebiotics have had limited success for various reasons (104). When feed is withdrawn prior to slaughter (broilers) and during molting (layers), birds are most susceptible to *Salmonella* colonization due to large shifts in gut and crop microbial populations (30, 39, 102). It is during these time periods that probiotics and prebiotics administered in feed either do not protect or offer only limited protection of the birds (36, 117). Probiotics applied in the water during this time reduce *Salmonella* titers but typically do not eliminate the bacteria. A reduction in probiotic effectiveness occurs partially because of the die-off of anaerobic bacteria as a result of their sensitivity to oxygen (5, 87, 97, 107). An additional problem encountered with probiotic and prebiotic use is the ability of some *Salmonella* serovars to become invasive (18, 103). In the initial stages of intestinal infection, *Salmonella* may cross the intestinal barrier into macrophages (16, 49). Once inside the macrophages, *Salmonella* has the ability to evade lysis, potentially leading to systemic *Salmonella* infection in birds (10, 71, 100).

The shift in the dominant serovars of *Salmonella* due to intervention is well described for conventional systems (6). However, there is limited information describing any shift in nonconventional systems partially because there is little microbiological survey information available for these systems. The choice of intervention measures may impact the selection pressures on *Salmonella* serovars.

CONCLUDING REMARKS

Understanding the dynamics of *Salmonella* contamination in poultry and eggs is very important due to the increasing consumption of poultry and egg-containing products in the United States. There have been a number of significant shifts in *Salmonella* populations associated with poultry-associated sources over the last century. The predominant serovars in the first half of the last century, *Salmonella* Pullorum and Gallinarum, were successfully eradicated from commercial poultry through programs like the NPIP. Subsequently, *S.* Enteritidis became the predominant serovar in poultry and eggs. *S.* Enteritidis not only colonized birds but also developed into a leading cause of salmonellosis in humans. Because of these problems, *S.* Enteritidis has been targeted by a number of control programs over the past few decades and was recently replaced by *S.* Heidelberg and *S.* Kentucky as the leading serovars isolated from poultry and poultry-associated products. *S.* Heidelberg is also one of the top serovars associated with human disease, including invasive infections, which is a significant concern because a number of these strains display MDR (63). *S.* Kentucky, while historically not a significant human pathogen, often displays MDR (46), which could be a significant problem if it develops into a more prominent human pathogen. These observed shifts in *Salmonella* serovars in commercial poultry-associated environments appear to be driven by a combination of bacterial genetic factors, host-related factors, and management practices. Therefore, an improved understanding of the historical factors that likely contributed to population shifts will provide insights for developing strategies to control current *Salmonella* problems and also limit the emergence of additional serovars that are an increased threat to public health.

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