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doi: 10.2527/jas.2007-0457 originally published online September 18, 2007

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://jas.fass.org/content/86/14_suppl/E163
Gastrointestinal microbial ecology and the safety of our food supply as related to *Salmonella*¹²

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**ABSTRACT:** *Salmonella* causes an estimated 1.3 million human foodborne illnesses and more than 500 deaths each year in the United States, representing an annual estimated cost to the economy of approximately $2.4 billion. *Salmonella enterica* comprises more than 2,500 serotypes. With this genetic and environmental diversity, serotypes are adapted to live in a variety of hosts, which may or may not manifest with clinical illness. Thus, *Salmonella* presents a multifaceted threat to food production and safety. *Salmonella* have been isolated from all food animals and can cause morbidity and mortality in swine, cattle, sheep, and poultry. The link between human salmonellosis and host animals is most clear in poultry. During the early part of the 20th century, a successful campaign was waged to eliminate fowl typhoid caused by *Salmonella Gallinarum*/*Pullorum*. Microbial ecology is much like macroecology; environmental niches are filled by adapted and specialized species. Elimination of *S*. Gallinarum cleared a niche in the on-farm and intestinal microbial ecology that was quickly exploited by *Salmonella* Enteritidis and other serotypes that live in other hosts, such as rodents. In the years since, human salmonellosis cases linked to poultry have increased to the point that uncooked chicken and eggs are regarded as toxic in the zeitgeist. Salmonellosis caused by poultry products have increased significantly in the past 5 yr, leading to a USDA Food Safety and Inspection Service “Salmonella Attack Plan” that aims to reduce the incidence of *Salmonella* in chickens below the current 19%. The prevalence of *Salmonella* in swine and cattle is lower, but still poses a threat to food safety and production efficiency. Thus, approaches to reducing *Salmonella* in animals must take into consideration that the microbial ecology of the animal is a critical factor that should be accounted for when designing intervention strategies. Use of competitive exclusion, sodium chlorate, vaccination, and bacteriophage are all strategies that can reduce *Salmonella* in the live animal, but it is vital to understand how they function so that we do not invoke the law of unintended consequences.

**Key words:** ecology, intestinal, reduction, *Salmonella*

doi:10.2527/jas.2007-0457

**INTRODUCTION**

As food producers, we have a moral and ethical responsibility to produce the safest food for consumers that is possible (Rollin, 2006). The food supply of the United States is one of the safest in the world and becomes safer each year; but too many foodborne illnesses continue to occur. One of the most common and serious foodborne pathogenic bacteria in the United States is *Salmonella enterica* (Mead et al., 1999). Human salmonellosis occurs in an estimated 1.3 million people, causes >500 deaths, and is estimated to cost the US economy >$2.4 billion each year (Mead et al., 1999; USDA-ERS, 2001). Understanding the ecological niche that *Salmonella* fills in the environment and how the pathogen enters the food chain is critical because *Salmonella* are estimated to cause over 30% of all bacterial foodborne deaths in the United States (Mead et al., 1999).

Although an increasing number of human salmonellosis cases have been linked to vegetables and fruits, the most common route is through foods of animal origin (Braden, 2006). *Salmonella* are pathogens but can frequently live in animals as a transient member of the intestinal microbial population without causing disease. Thus, reliance on animals looking sick is not an

¹Proprietary or brand names are necessary to report factually on available data; however, the USDA neither guarantees nor warrants the standard of the product, and the use of the name by the USDA implies neither approval of the product, nor exclusion of others that may be suitable.

²Presented at the Food Safety symposium at the annual meeting of the American Society of Animal Science, San Antonio, TX, July 8 to 12, 2007.

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Received July 25, 2007.

Accepted August 30, 2007.
effective indicator of *Salmonella* colonization. A variety of animals from many environments have been found to harbor *Salmonella* (Benirschke and Adams, 1980; Kenny, 1999; Pasman et al., 2005; Bemis et al., 2007), but food animals are the primary vector for transmitting *Salmonella* to humans (Borland, 1975; Holmberg et al., 1984; Branham et al., 2005). Chickens (Zhao et al., 2001), turkeys (Berrang et al., 1998), and eggs (Bradan, 2006) can all be infected with *Salmonella*. The intestinal tracts of finishing and breeding swine (Davies et al., 1999), as well as that of beef and dairy cattle (USDA-APHIS, 2001, 2003a) can contain *Salmonella*. Further outbreaks of salmonellosis have been linked to improper pasteurization of dairy products (Hedberg et al., 1992; USDA-APHIS, 2003b) or improperly cooked ground beef (Mead et al., 1999). Other routes of exposure of humans to *Salmonella* include water runoff from farms (Thurston-Enriquez et al., 2005; Soupir et al., 2006) or swine effluent lagoons (Hill and Sobsey, 2003), and direct animal (Chapman et al., 2000) or fecal contact (Pritchard et al., 2000; Enriquez et al., 2001).

Thus, *Salmonella* are relatively widespread in the environment and within food animals (Rodriguez et al., 2006), and attempts to understand and control this pathogen must be equally broad based. Because *Salmonella* can live undetected in food animals but still pose a risk to human consumers, control strategies must be tailored to specific animal species yet be applicable to large numbers of animals. Therefore, in order to be able to target this pathogen, we must understand its role in nature and in the gut of food animals.

**What is Salmonella?**

*Salmonella* are gram-negative bacteria comprising 2 species and 6 subspecies (Coburn et al., 2007); the most important of which is *Salmonella enterica* ssp. enterica. *Salmonella enterica* infection in humans causes severe illness (e.g., nausea, intestinal cramps, diarrhea, vomiting, and possible arthritic symptoms) and can be an intracellular pathogen (FDA-CFSAN, 2006; Coburn et al., 2007). *Salmonella enterica* causes illness in humans by passing from the intestinal tract into the epithelium, where it causes inflammation and systemically releases an enterotoxin and a potent endotoxin (FDA-CFSAN, 2006). *Salmonella* exists in a typical fecal-oral life cycle, although it can be spread through the nasal cavity to the gut (Fedorka-Cray et al., 1995).

*Salmonella enterica* comprises over 2,500 known serovars, specific subtypes defined by the sugar and protein coats around the bacterium as well as by flagellar proteins that are pathogenic to humans or animals (Popoff et al., 2004). For example, a *Salmonella* serotype would commonly be known simply as *Salmonella* Typhimurium, rather than as *S. enterica enterica* Typhimurium. *Salmonella* serotypes have evolved and adapted to infect specific hosts (Kingsley and Baumler, 2000). Thus, each animal species including humans is associated with specific serotypes that cause illness in that species. However, some serotypes, such as Typhimurium, can be utilitarian and infect many species of animals, including man. Some *Salmonella* serotypes produce a clinical illness when infecting animals other than their adapted hosts, whereas other serotypes do not. The ability of serotypes to cause illness in nonhost animals is dependent upon the host adaptation to specific serogroups endemic to the host population (Kingsley and Baumler, 2000).

Adaptation allows *Salmonella* to exist as a pathogen in a suitable host environment, or as a transient member of the gastrointestinal population in a less-than-ideal host environment. What this means in a practical sense is that some serotypes can live in food animals without causing illness; however, when host animals and their carried serotypes are consumed by humans, then foodborne illness can result. However, routes other than food and water have been responsible for human illness. For instance, reptiles commonly harbor *Salmonella* (Passman et al., 2005; Bemis et al., 2007), which can be transmitted to humans and cause illness (Woodward et al., 1997; Mermin et al., 2004), but these serotypes (e.g., serotypes Nima and Pooma) do not circulate from person to person readily; therefore, the infection dies out quickly (Kingsley and Baumler, 2000).

Although *Salmonella* serotype influences the extent and outcome of human illness, elimination or treatment strategies are not different between serotypes. Therefore, focusing solely on a handful of critical serotypes is only helpful in understanding the flow of specific isolates within the food chain, with too much attention focused only on certain serotypes when making macro-scale economic, trade, public health policy, or scientific decisions. It is critical, therefore, that we understand the various serotype host preferences but continue to view *Salmonella* as the threat, rather than only watching a few serotypes. Focus on a eliminating a specific serotype can worsen a situation through the law of unintended consequences. As a society, we have inadvertently performed this experiment, resulting in the emergence and dissemination of *Salmonella* Enteritidis in poultry, as discussed subsequently in detail.

**Serotype Distribution**

Although the relative importance of serotype has been overstated in regard to the development of pathogen reduction strategies, serotype is still critical information to understand the spread of *Salmonella* through the food chain. Table 1 illustrates the relative frequency of different serotypes isolated from humans and animals (CDC, 2006; USDA-FSIS, 2007). Animal-associated predominant serotypes vary with geographic location as well as animal species (USDA-FSIS, 2006). The subtypes isolated most frequently from various animal sources pooled across the United States in 2005 are shown in Table 1. Although not shown in this chart, the most common serotype found in eggs in the United States is *S. Enteritidis* (Bradan, 2006). However, *Sal-
Table 1. Most common *Salmonella* serotypes isolated across the United States in 2005 (in order of prevalence)

<table>
<thead>
<tr>
<th>Order of prevalence</th>
<th>Serotypes from ground beef</th>
<th>Serotypes from market hogs</th>
<th>Serotypes from broilers in commercial plants</th>
<th>Serotypes from ground chicken in commercial plants</th>
<th>Serotypes from clinically ill humans</th>
</tr>
</thead>
<tbody>
<tr>
<td>First</td>
<td>Montevideo</td>
<td>Derby</td>
<td>Kentucky</td>
<td>Enteritidis&lt;sup&gt;3&lt;/sup&gt;</td>
<td>Typhimurium (includes Copenhagen)&lt;sup&gt;3&lt;/sup&gt;</td>
</tr>
<tr>
<td>Second</td>
<td>Anatum</td>
<td>Typhimurium (includes Copenhagen)&lt;sup&gt;3&lt;/sup&gt;</td>
<td>Heidelberg&lt;sup&gt;3&lt;/sup&gt;</td>
<td>Kentucky</td>
<td>Enteritidis&lt;sup&gt;3&lt;/sup&gt;</td>
</tr>
<tr>
<td>Third</td>
<td>Muenster</td>
<td>Infantis</td>
<td>Typhimurium (includes Copenhagen)&lt;sup&gt;3&lt;/sup&gt;</td>
<td>Heidelberg&lt;sup&gt;3&lt;/sup&gt;</td>
<td>Newport&lt;sup&gt;3&lt;/sup&gt;</td>
</tr>
<tr>
<td>Fourth</td>
<td>Newport&lt;sup&gt;3&lt;/sup&gt;</td>
<td>Anatum</td>
<td>Typhimurium (includes Copenhagen)&lt;sup&gt;3&lt;/sup&gt;</td>
<td>Enteritidis&lt;sup&gt;3&lt;/sup&gt;</td>
<td>Heidelberg&lt;sup&gt;3&lt;/sup&gt;</td>
</tr>
<tr>
<td>Fifth</td>
<td>Mbandanka</td>
<td>Saintpaul</td>
<td>I 4, 5, 12:i:-</td>
<td>I 4, 5, 12:i:-</td>
<td>Javiana&lt;sup&gt;3&lt;/sup&gt;</td>
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<td>Total <em>Salmonella</em></td>
<td></td>
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<td>accounted for by</td>
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<tr>
<td>top 5 serovars, %</td>
<td></td>
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</tbody>
</table>

<sup>1</sup>Data from the Food Safety Inspection Service, US Department of Agriculture (USDA-FSIS, 2006).

<sup>2</sup>Data from the Centers for Disease Control and Prevention (CDC, 2006).

<sup>3</sup>Serotypes in bold represent the top 5 isolates from clinically ill humans found in food animals.

*Salmonella* Enteritidis is found at a very low prevalence in shell eggs in the United States, occurring at a rate of approximately 1 in 20,000 eggs (Ebel and Schlosser, 2000). Yet this has lead to several Centers for Disease Control (CDC) recommendations for preventing *Salmonella* Enteritidis infection through shell eggs (CDC, 2003).

The 5 most common serovars (Table 1) represent from 47 to 85% of all *Salmonella* isolates from their respective sources; therefore, these are by far the most common serovars isolated from food animals in the United States. Furthermore, the top 5 serotypes account for 56% of all human isolates. As shown in Table 1, there is a high degree of correlation between the serovars found in nonclinically ill food animals (and thus more likely to enter the food chain) with those isolated from sick humans (serovars highlighted in bold). Therefore, it is critical to target the sources of these serotypes to interrupt the transmission cycle before they can cause human illnesses.

Each year the prevalence of *Salmonella*-positive samples detected by USDA Food Safety and Inspection Service (FSIS) from nontargeted, or ‘A’ sets in processing facilities varies (Figure 1). Over the past 5 yr, the number of *Salmonella*-positive samples in ground beef has decreased and the percentage of positive samples from broilers has increased. This increase in broilers has led to the 2006 implementation of a “Salmonella attack plan” by FSIS that focuses on increased sampling frequency in “dirty” plants. The relative frequency of *Salmonella* serotypes represented among human illnesses also varies from year to year, as can be seen in data from the past 5 yr (Figure 2). In the 1980s and 1990s, *S*. Enteritidis increased in humans to the point that it was the leading cause of salmonellosis in humans, but in recent years has maintained a steady ranking as the second-most-common cause of salmonellosis. In recent years, the proportion of isolates represented by *S*. Enteritidis had declined, until 2005 when there was a dramatic increase (Figure 2). It remains to be seen...
whether this was a transient increase or a long-term increase in *S.* Enteritidis illnesses.

**Seasonality of Salmonella**

Seasonality of fecal shedding is critical to understanding the flow of *Salmonella* through the food chain. There is a correlation between shedding in animals and human outbreaks. Shedding by food animals can approach zero during the winter months and reaches its peak in summer and early fall (McEvoy et al., 2003; Fossler et al., 2005), especially in cattle and swine, and human outbreaks also peak during this period. The crucial National Animal Health Monitoring System (NAHMS) animal surveys were conducted in February through July and March through September, which is when fecal shedding appears to be at its highest (USDA-APHIS, 2001; Huston et al., 2002). Interestingly, it has been shown recently that the human *Salmonella* incidence peaks 13 d after the peak in ambient temperature (Naumova et al., 2007). However, other researchers have found that as herd size increased, fecal shedding of *Salmonella* increased (Warnick et al., 2003). Researchers have shown that herd size did not play a role in *Salmonella* shedding (Fossler et al., 2005).

Cattle can carry many different serotypes of *Salmonella*. The top 5 serotypes isolated from ground beef (Table 1) account for 47% of the reported serotypes (USDA-FSIS, 2007). However, 25 different *Salmonella* serotypes were isolated from lactating dairy cows on farm, and another 24 serotypes were isolated from dairy cows being culled from the herd (Wells et al., 2001). In more recent field studies that examined the prevalence of *Salmonella* in healthy lactating dairy cows in New Mexico, a wide diversity of serotypes was also found (Edrington et al., 2004a,b). Other cattle surveys have isolated *Salmonella* spp. from dairy bulk milk tanks (Jayarao and Henning, 2001; USDA-APHIS, 2003b), with the most common bulk tank milk serotypes across the United States being Montevideo, Newport, Muen-

![Figure 2. *Salmonella* serotypes isolated from humans from 2000 through 2005; data excerpted from the Centers for Disease Control and Prevention (CDC, 2006). Serotypes: ○ = *Salmonella* Typhimurium, ◆ = *Salmonella* Enteritidis, △ = *Salmonella* Newport, ▲ = *Salmonella* Heidelberg, and □ = *Salmonella* Javiana.](image-url)
ster, Meleagridis, and Cerro. The presence of these serotypes in raw milk contributes to concerns about consumption of raw milk and cheeses made from unpasteurized milk (Cody et al., 1999; Villar et al., 1999).

**Salmonella in Swine**

Swine can be asymptomatic reservoirs of foodborne pathogenic bacteria that are transmissible to humans via consumption of contaminated pork products or through the environment (Davies et al., 1999; Rostagno et al., 2003). Foodborne pathogenic bacteria such as *Salmonella* can persist in the environment or within a herd at subclinical levels for years (Sandvang et al., 2000). It has been estimated that between 25 and 48% of the US swine herd may be colonized with *Salmonella* species on the farm (Davies et al., 1997; Funk et al., 2001); however, the percentage of marketed swine that test positive for *Salmonella* remains under 10% (USDA-FSIS, 2007). *Salmonella* Choleraeaus is a swine-adapted pathogen that has a serious impact on infected humans (usually by direct animal contact), but it does not spread through the human population. The most common *Salmonella* serotypes isolated from swine include Derby, Typhimurium, and Infantis (USDA-FSIS, 2006, 2007).

Pigs may become colonized with *Salmonella* by ingesting contaminated feces; however, esophagotomized swine can become colonized with *Salmonella* following intranasal inoculation and through snout-to-snout contact (Fedorka-Cray et al., 1995; Lo Fo Wong et al., 2004). Placing swine in *Salmonella*-contaminated pens for a lairage period before slaughter can also result in the colonization of pigs immediately before entry into the food chain (Hurd et al., 2001; Rostagno et al., 2003). *Salmonella* can be commonly found in the environment of pig farms (Letellier et al., 1999; Funk et al., 2001). As many as 66% of swine farms in Alberta had at least 1 positive *Salmonella* sample, and 20% of the on-farm environmental samples were positive for *Salmonella* (Rajic et al., 2005). Additionally, feed samples have been found to be *Salmonella* positive (Davies et al., 1999; Jones and Richardson, 2004), posing a threat to human food safety (Crump et al., 2002). Studies examining the incidence of *Salmonella* in free-range pigs found a diverse group of serotypes in the environment of these outdoor-reared swine (Callaway et al., 2005).

**Salmonella in Poultry**

*Salmonella* is found commonly in chickens and turkeys, and it spreads easily from bird to bird through a fecal-oral route within poultry houses (Rodriguez et al., 2006). *Salmonella* also can be spread via other reservoirs (e.g., insects, rodents, farm animals, and humans); thus, the need for stringent biosecurity and pest control plans on most poultry farms. Broiler flocks in the United States are currently positive for *Salmonella* at an average of 19% (USDA-FSIS, 2006). According to the CDC, the most common poultry-associated *Salmonella* serovars account for 33.3% of the total human foodborne illnesses in the United States (CDC, 2006). From this data, we can extrapolate that each year, poultry-related *Salmonella* alone costs the US economy approximately $966 million in direct and indirect costs. *Salmonella* Typhimurium and Enteritidis are the human illness-causing serovars most commonly associated with poultry meat and eggs, respectively, in the United States (Braden, 2006; CDC, 2006). Both can cause illness in poultry and are isolated from clinically ill birds, but are frequently present as an asymptomatic infection, allowing them to enter the food chain without triggering a simple detection tripline.

*Salmonella* is a serious threat to broiler and egg production, both as a direct food safety threat in poultry meat and eggs and via vertical transmission to a new generation of infected broilers or layers. Because *Salmonella* can survive in the gut of birds or invade host tissues, it can be transmitted to consumers through various routes. For example, *S.* Enteritidis can invade the ovaries and be directly encapsulated in eggs, or it can live in the intestinal tract and enter eggs through cracks in the shell as the egg intersects the intestinal tract (Braden, 2006) in addition to being transmitted through poultry meat (Kimura et al., 2004). The 2 former routes can result in the production of contaminated eggs that are consumed by humans. Additionally, fertilized eggs can be infected with *Salmonella* via semen (Reiber et al., 1995). Thus, when an infected egg is hatched, the chick can already contain *Salmonella*, which can then be spread quickly to “clean” birds through contact, as well as through the common fecal-oral route. Even when only 5% of chickens are *Salmonella* (Typhimurium) positive upon entering the growing house, 72 to 95% of birds may be *Salmonella* positive within 3 wk of entry (Byrd et al., 1998). Thus, no matter what intervention strategies are implemented to reduce *Salmonella* in chicks or hatching eggs, further interventions designed to reduce horizontal spread from *Salmonella*-infected birds are necessary.

**Other Factors Affecting Salmonella Populations**

There has been a great deal of research aimed at understanding what effect stresses have on populations of *Salmonella*, especially dietary and transportation stresses. Ruminal populations of *Salmonella* declined more rapidly in cattle fed hay compared with cattle starved for 2 d (Brownlie and Grau, 1967). In other studies, the longer the time between leaving the farm of origin and the time of slaughter increased the incidence of *Salmonella* in the rumen and feces of cattle (Grau et al., 1968). Taking pigs for a “joy ride” in an open truck for several hours to simulate transport to market has been shown to increase fecal shedding of *Salmonella* significantly (Williams and Newell, 1971), although more recent research has shown an opposite effect (Marg et al., 2001). Effects of transportation on
Salmonella levels on cattle hides have also been variable, but it has been shown that more Salmonella were found on the hides at slaughter compared with levels at the feedlot (Reicks et al., 2007).

**Multidrug-Resistant Salmonella: An Increasing Threat**

In recent years, concerns have grown about antimicrobial resistance in general, but specifically resistance within the food supply (Corpet, 1998; Molbak et al., 1999; Threlfall et al., 2000a,b). The incidence and severity of antimicrobial resistance among Salmonella spp. has grown, at least in perception, in recent years. Most alarmingly, the recognition of multidrug-resistant (MDR) Salmonella has prompted significant concerns about the safety of the food supply directly as a source of MDR Salmonella, and indirectly as a reservoir of antimicrobial genetic elements that can be exchanged between intestinal bacteria (Angulo et al., 2004; Salyers et al., 2004; Salyers and Shoemaker, 2006). Death or serious illness will occur more frequently as outcomes of infection as the human exposure to MDR Salmonella via the food supply increases (Helms et al., 2002; Angulo et al., 2004).

Multidrug-resistant Salmonella have been isolated from retail meats (White et al., 2001, 2004; Zhao et al., 2006a), imported foods (Zhao et al., 2006b), and from food animals and their facilities (Besser et al., 2000; Wright et al., 2005; Poppe et al., 2006). Multidrug-resistant Salmonella have been isolated from poultry, swine, and cattle and represent a growing concern to public health. Recently, Salmonella Newport-MDRampC has emerged as an agent of significant concern to the meat industry. This MDR S. Newport infection is most often acquired through the US food supply, most likely from bovine or poultry sources, particularly among persons already taking antimicrobial agents (Varma et al., 2006).

**Reduction Strategies**

A test-and-slaughter flock (depopulation of farms positive for Salmonella) approach may be effective for eliminating S. Enteritidis from parent and grandparent breeder flocks and in layer flocks because S. Enteritidis can be transmitted through eggs. However, the origin and transmission of other Salmonella serotypes in poultry, swine, and cattle are unclear.

Vaccination using inactivated S. Gallinarum prevents colonization of poultry by S. Enteritidis; however, if using blood tests to determine Salmonella populations, vaccinated birds are indistinguishable from those infected by Salmonella. Thus, special care must be given when reporting blood results from vaccinated flocks. Antibiotics used in human or veterinary medicine have been suggested as potential methods to reduce specific pathogens, such as Salmonella. However, because of fears of antibiotic resistance, especially among Salmonella spp., the use of antibiotics for this purpose is actively discouraged.

Other reduction strategies that are likely to be successful and acceptable include probiotics, prebiotics, and competitive exclusion (CE) cultures. All of these techniques attempt to utilize the normal microbial ecosystem to control pathogens (Callaway et al., 2002; Doyle and Erickson, 2006). The best described system is CE, in which day-of-hatch chicks are treated with a pathogen-free mixture of normal intestinal bacteria to jump-start their intestinal population and exclude pathogens from colonizing the gut through competition for nutrients (Nisbet, 2002; Zhang et al., 2007b,a). The use of sodium chlorate is a method to reduce Salmonella in poultry, and it is currently under regulatory review for use as a feed additive. Chlorate is toxic to some bacteria because of an intracellular enzyme they possess (i.e., nitrate reductase), but it does not kill all bacteria (Anderson et al., 2000, 2001). Salmonella spp. possess nitrate reductase and, therefore, are killed by chlorate treatment. If parental flocks, newly hatched chicks, sows, calves, piglets are reared and(or) maintained on these products, then it is likely that Salmonella serovars can be reduced in the food supply. However, simply cleaning them up at the time of birth or hatch may actually worsen the situation by providing a clear ecological field for another pathogen to exploit, as has been demonstrated by the expansion of S. Enteritidis in poultry during the 20th century.

**A Lesson for the Future: Unintended Consequences and the Ecology of Salmonella enteritidis Expansion into a New Host**

Any actions that we take to reduce S. enterica colonization in animals must be tempered with the knowledge that our actions can yield unintended consequences. A case in point is the emergence of S. Enteritidis as a human pathogen associated with poultry eggs (Kingsley and Baumler, 2000; Rabsch et al., 2001). During the latter part of the 19th and early 20th century, Salmonella Gallinarum/Pullorum were common in the United States and Europe (as fowl typhoid or pullorum disease), where it caused morbidity and mortality amongst poultry flocks; S. Enteritidis was virtually unknown as a human pathogen. The development of the first voluntary National Poultry Improvement Program (NPIP) was conceived in 1935, at least in part, to reduce economic losses caused by fowl typhoid. In this effort, the NPIP was highly successful; because Gallinarum and Pullorum have but a sole animal reservoir (domestic and aquatic fowl), S. Gallinarum was virtually eliminated from the US and United Kingdom flocks by the early 1970s by following conventional test-and-slaughter methods.

However, as the S. Gallinarum and Pullorum incidence in the national flock decreased, the incidence of S. Enteritidis-caused human illnesses increased. Salmonella Enteritidis did exist naturally in poultry at low
incidence but has another reservoir, rodents. Human cases of S. Enteritidis rapidly increased through the 1980s and 1990s, reaching a peak as the most frequently reported serovar isolated from human illnesses. As of 2005, S. Enteritidis is the second most commonly isolated serovar, responsible for 15% of the reported human salmonellosis cases in the United States. How did this change occur? Bacterial ecology is much like that of macroecology; niches within an environment are filled by a succession of species best adapted to each niche. Salmonella Gallinarum and Pullorum filled a niche in the microbial ecology of the gut of chickens, and when Gallinarum/Pullorum was eliminated, that vacuum was filled by a similar bacterium, S. Enteritidis. While the reservoir for Gallinarum was depleted from infected flocks S. Enteritidis was able to jump from its natural rodent reservoir into poultry. The interaction between these 2 serovars is further demonstrated by the fact that effective S. Enteritidis vaccines for laying hens are commonly made from inactivated (attenuated) strains of S. Gallinarum.

Although by no means a complete answer to the question of why S. Enteritidis populations increased in poultry, the niche-filling explanation poses a concern for Salmonella-reduction strategies. This should not preclude the development of strategies in the live animal, but should be considered when developing potential strategies. The removal of Salmonella from the intestinal population will create a vacuum that will be rapidly filled with bacteria from the intestinal tract or environment that are most fit to occupy that niche; frequently, the most-fit competitor would be another Salmonella species. Thus, a strategy that eliminates Salmonella in the live animal should be coupled with a complementary strategy that provides an alternative bacterial species or provides nutrients that select for an already existing intestinal population that can occupy Salmonella’s niche.

In conclusion, Salmonella is a diverse, widespread bacterium in the farm and food animal environment that can cause significant problems in animal and human health. One of the most serious foodborne pathogens, Salmonella is a member of the family Enterobacteriaceae and thus, is at home within the gut of food animals. Although much is known about the physiology and genetics of this bacterium, the microbial ecology of this organism is complex and rather poorly understood, especially within the gastrointestinal tract.

Salmonella occupy a variety of niches in the intestinal microbial and on-farm environment ecologies and can colonize reptiles, poultry, and mammals (including humans). Effects of Salmonella colonization on hosts can range from an asymptomatic carrier state to severe disease and even death. The extent and severity of illness in humans and animals is determined by which serotype(s) are present; however, the importance of the individual serotype should not be overstated, because nearly all Salmonella can be pathogenic to humans, not just a few important serotypes.

Salmonella in the food animal meta-intestinal tract is a very dynamic situation. Serotypes that most commonly affect humans change over time, as do serotypes isolated from food animals. Changes that we make in an effort to eliminate Salmonella can have unintended consequences, evidenced by the increase in S. Enteritidis in poultry during the 20th century following fowl typhoid elimination. Simply cleaning up animals may temporarily reduce the incidence of Salmonella, but intestinal populations of other Salmonella serotypes will increase to fill the vacuum. Therefore, it is critical to include other strategies (e.g., CE, probiotics, or prebiotics) that provide other intestinal bacteria a selective advantage to occupy the niche vacated by Salmonella spp., thereby preventing the entry of a new (or old) pathogen into animal populations and the food supply.

LITERATURE CITED


