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REVIEW
Salmonella enterica serovar Enteritidis: a Mini-review of Contamination Routes and Limitations to Effective Control

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Abstract
Salmonellosis is associated with massive public health and economic losses globally. It is estimated to cost poultry farmers in the United States of America up to US$114 million annually. Attempts to develop effective vaccines and eradicate *Salmonella enterica* serovar Enteritidis (S. Enteritidis) from hen houses are undermined by serious limitations. This article reviews documented contamination routes and limitations to the rapid development of vaccines. Host-parasite interactions and clinical pathology are discussed and methods for reducing S. Enteritidis infection and transmission suggested.

Discipline: Animal health
Additional key words: chicken, farm-house environment, vaccine development

Introduction

Salmonellosis is associated with contaminated poultry products. It presents major public health and economic concerns globally. Previous reports indicate that up to 3.7 million cases of salmonellosis occur in the United States of America (USA) every year, with economic losses to poultry farmers estimated at US $64 million to US $114 million annually[13]. Attempts to develop effective vaccines and eradicate *Salmonella enterica* serovar Enteritidis from hen houses are undermined by serious limitations. To ensure safety of products and maximization of profits, there is a need to gain firm knowledge on improved breeding techniques to ensure protection against possible outbreaks of *Salmonella* food poisoning. This article reviews S. Enteritidis contamination routes and control, giving a fresh look at documented contamination routes and re-examines limitations to vaccine development. Host-parasite interactions and associated clinical pathology are discussed and methods for reducing *S.* Enteritidis infection and transmission suggested.

Salmonella enterica serovars associated with food poisoning

Salmonella enterica is one of the most common causes of food poisoning in humans[76]. It has 6 subspecies and about 2,500 serotypes[99], of which the two most frequently reported serovars are *Salmonella enterica* serovar Typhimurium (*S.* Typhimurium) and *S.* Enteritidis, a Gram-negative bacterium that negatively affects both human and animal health. Using the Colindale phage-typing scheme, 37 phages have been used to identify more than 210 phage types for *S.* Typhimurium[2,3,15,29]. On the other hand, 16 phages have been used to identify 65 phage types for *S.* Enteritidis[97]. Historically, *S.* Typhimurium was among the most common serovars isolated from poultry across many countries from the 1950s to the late 1970s, but was overtaken by *S.* Enteritidis as the most common serovar isolated from poultry in the mid 1980s to date[75]. In England and Wales, the percentage of *S.* Enteritidis isolated from poultry rose from 3.3% of all *Salmonella* serovars in 1985 to 47.8% in 1988 and 48.3% in 1989[75]. Of these, the *S.* Enteritidis phage type 4 (PT4) was the most frequently reported and accounted for 71% of the isolates in 1988[62]. A three month survey conducted in 1990

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to estimate prevalence and distribution of *S.* Enteritidis in spent laying hens in commercial egg production flocks in the USA demonstrated the presence of *S.* Enteritidis in 3% of 23,431 pooled caecal samples collected from 406 layer houses[26].

S. Enteritidis and the burden of salmonellosis in humans and poultry

Salmonella Enteritidis has been associated with pandemics in Europe and elsewhere. Up to 3.7 million cases of salmonellosis were estimated to occur annually in the United States alone[63] with economic losses to poultry farmers estimated at US$ 64 to 114 million due to salmonellosis in young chickens[13]. Persons infected with S. Enteritidis usually develop fever, abdominal cramps and diarrhea beginning 12 to 72 hours after consuming contaminated food. The illness often lasts 4 to 7 days and most persons recover without antibiotic treatment. However, in the elderly, infants and persons with impaired immune systems, diarrhea can be severe and the persons may be ill enough to require hospitalization. In such patients, infection may spread to other body organs and can cause death if prompt antibiotic treatment is not administered. The cost estimates per case of human salmonellosis range from approximately US $40 to US $4.6 million respectively for uncomplicated cases to those ending with hospitalization and death[100]. Epidemiological data in Hungary, United Kingdom, USA, and Germany have confirmed that the food most commonly associated with increased illness in humans was the egg[72,85,90] and that *S.* Enteritidis accounted for 85% of all cases of human salmonellosis in Europe[30].

Cases of animal and human salmonellosis have been grossly underestimated due to lack of systematic methods of reporting[27]. Also, whether or not specific incidences are reported depends on intensity of surveillance[34], submission of isolates for serotyping[84], severity of illness and association with a recognized outbreak in human populations[1,88,92]. Thus, although outbreaks would easily attract media attention, 80% of all salmonellosis cases occur individually rather than as outbreaks[100].

Host–parasite interactions and clinical symptoms in chickens

The outcome of an interaction between *Salmonella* and its host is dependent upon multiple factors including the genetic background of the host[51]. Previous studies have revealed considerable differences between lines in the level of colonization of the gastrointestinal tract and response to vaccination[57,59]. Also, while salmonellosis in young chickens is characterized by severe clinical signs of systemic disease associated with diarrhea, dehydration and high mortality rates[93], adult chickens can produce *Salmonella*-contaminated eggs without evidence of discernible illness[31]. Poultry infected with other egg contaminating serotypes, such as *Salmonella enterica* serovar Pullorum and *Salmonella enterica* serovar Gallinarum, are associated with drastic weight loss, a sharp decrease in egg production, and increased mortality[80,82]. On the other hand, there are typically no clinical signs in birds infected with S. Enteritidis[44].

Contamination of eggs with S. Enteritidis

Previous reports indicate that *S.* Enteritidis is the only human pathogen that contaminates eggs routinely even though the chicken farm environment is a rich source of other *Salmonella* serotypes[12,14,83]. Also, while penetration of cracked eggshells by bacteria was seen as a frequent cause of human illness before the introduction of grading schemes in the 1970s, but it is now known that egg contamination by *S.* Enteritidis may occur by vertical transmission in the reproductive tract before deposition of the shell[37,48,64]. In one study, adult laying hens were inoculated orally with 10^8 colony-forming units (cfu) of *S.* Enteritidis and the bacterium was isolated 2 days post infection from spleen, liver, heart, gall bladder, intestinal tissues, and from various sections of the ovary and oviduct[47,49]. Additional reports indicate that eggs can be contaminated with faeces from hens excreting *Salmonella*[11,39,79,86,89,101]. In such cases, *Salmonella* in faeces are believed to penetrate egg shell pores as the egg cools and before the establishment of the proteinaceous cuticular barrier[10,31,86,101]. Fecal matter adherent to the shell may also contaminate eggs via cracks on egg shells or when eggs are broken open for preparation of food products[11]. Previous reports indicate that forming eggs are subjected to descending infections from colonized ovarian tissue, lateral infections from upper oviduct and ascending infections from colonized vaginal and cloacal tissues[49]. It has also been suggested that *S.* Enteritidis may actively gain access to the egg by penetrating marginally faulty shells that exclude most other bacteria[5].

Horizontal infection of *S.* Enteritidis in poultry

Horizontal transmissions occur during hatching of chicks, with spread of *S.* Enteritidis in aerosol contamination, litter, dust, and faecal as well as caecal droppings of littermates[71]. There is evidence that *S.* Enteritidis spreads rapidly from infected day old chicks to pen mates reared on litter[78,81]. Poultry may also get infected from feed[43,66], water[36], rodents[4,17,40], or by contact with other poults or chicks. Infection by these routes in chicks reached 100% within seven days of contact[81]. Contamination through water

results from the fact that young chicks and poults often dip their beaks, walk in, and defaecate in drinking water before drinkers are raised from the floor of the barn. In one study, laying hens experimentally inoculated with 10^5 *S.* Enteritidis phage type 4 (PT4) spread the infection via drinking water to un-inoculated hens in only 1 to 5 days[69]. Further evidence shows that short-term exposure to environmental stresses, such as introduction of young chicks to the same rearing room, and moulting (removal of feed and water from laying hens for 2 days), is associated with an increase in *S.* Enteritidis shedding by laying hens[69].

Hen-house management and environmental considerations

The ubiquitous presence of *Salmonella* within the poultry shed environment provides opportunity for multiple modes of transmission[18]. However, management styles or the manner in which chickens are kept in poultry houses can influence the rate of infection. In one study, a lower prevalence of *S.* Enteritidis PT4 was reported among 176,000 caged laying hens compared to free-range hens (1.7% vs. 50%) and the prevalence in culled hens kept in dirt-floor houses ranged from 14% to 42%[52]. Exposure to airborne *S.* Enteritidis PT4 can lead to generalized infection, even at low exposure doses[7]. Also, *S.* Enteritidis PT4 can survive in aerosols up to 2 hours with a negligible reduction in numbers[61]. A report by Davies and Wray[19] indicated that *S.* Enteritidis can survive for at least one year in empty poultry sheds, where naturally infected flocks have been housed, but will decline rapidly in litter. Elsewhere, *S.* Enteritidis from human sewage effluent discharged upstream of a chicken farm infected chickens in a commercial farm in southern California[19,20,53]. Isolates from hens were of the same phage type (PT4) as those found in eggs, sewage effluent and mice trapped in hen houses, and from cats and skunks on the premises[19,20,95]. Another report revealed that *S.* Enteritidis and fifty other different Salmonella serovars were isolated from hen house environment samples (litter and water) in Canadian flocks[74].

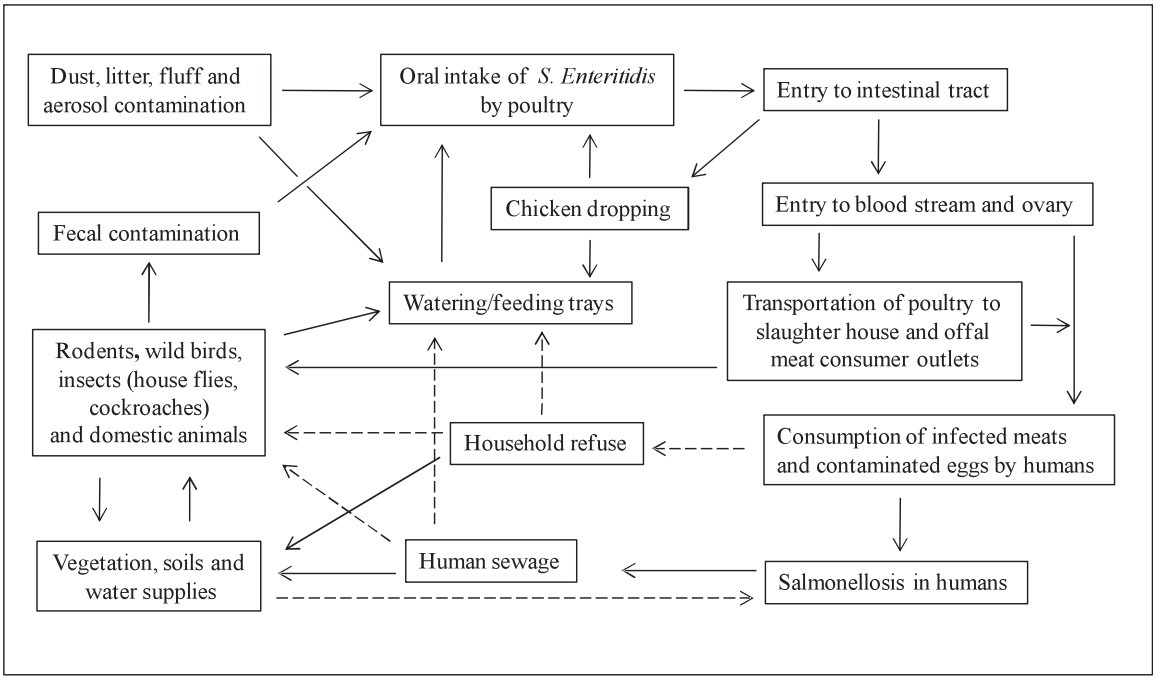
Infected domestic animals can become healthy carriers, latently infected or less frequently clinically ill, but may excrete *Salmonella* in their feces and form a large reservoir and source of environmental contamination for other animals and humans. Cross-contamination between the environment and domestic animals then progresses through mass transportation and slaughter[68]. Subsequently, wild birds including pigeons and sparrows, rodents, cats, dogs, and insects may be contaminated by contact or ingestion of spilled meat meal, feather meal and other animal by-products outside rendering departments at slaughtering plants, poultry houses and open trucks. These may lead to contamination of effluents, surface waters, creeks, lakes, rivers, pastures, and soils to colonization of birds, cattle, pigs, sheep, and horses as well as to contamination of animal feeds, or direct re-colonization of farm animals[11,24,54,67,87]. Reportedly however, contamination of the environment by *S.* Enteritidis from meat waste of domestic sources going into the refuse system may be small compared to animal waste and human sewage[68]. Figure 1 presents contamination routes of *S.* Enteritidis in the hen house environment.

Recent advances in the search for effective vaccines to *S.* Enteritidis

Following the dramatic increase in the last two decades in prevalence of *S.* Enteritidis worldwide, it is becoming a leading cause of food borne illnesses and serious attempts have been made to examine potency of many candidate vaccines to *S.* Enteritidis. There is evidence that administration of live *Salmonella* bacterium orally to newly hatched chicks results in extensive gut colonization and a strong adaptive immunity often leading to rapid protective effects within 24 hours[94]. In this process, establishment and colonization by other bacteria is inhibited by competitive exclusion[96] probably due to competition for binding sites. The presence of a large number of bacteria originating from a live *Salmonella* vaccine in the intestine can also induce infiltration of polymorphonuclear cells into intestinal walls and confer resistance to invasion and systemic spread by virulent *Salmonella* strains.

A recent report suggested the role of SEF14 fimbrial protein in adhesion of *S.* Enteritidis to the host[60]. *SefA* gene which encodes the subunit of the SEF14 fimbrial protein was cloned, ligated into a temperature sensitive expression vector and transformed into an avirulent strain of *Escherichia coli*. The recombinant strain was used as a vaccine to elicit specific immune responses against the SefA protein of *S.* Enteritidis in one-day old chickens and was re-isolated from the intestines of treated birds for up to 21 days thereafter. Using ELISA, IgA against SefA protein was detected in intestinal secretions from treated birds at 7 days and in bile samples from 14 to 21 days post treatment. Untreated birds did not show evidence of intestinal colonization by the recombinant strain or anti-SefA IgA response in their bile or intestinal secretions.

Additional studies evaluated the effect of oral *S.* Enteritidis and *S.* Typhimurium vaccine (metabolic drift mutants produced by chemical mutagenesis) on colonization of reproductive tract and internal egg contamination among laying hens[35]. Three groups of 30 laying hens were vaccinated orally at day 1, 6 weeks and 16 weeks of age with one or a combination of both vaccine strains and a fourth group (control) was not vaccinated. Birds were intravenously



Broken arrows denote probable but infrequently reported routes of contamination.
Full arrows indicate frequently reported routes.

Fig. 1. *Salmonella* Enteritidis: contamination routes in the hen-house environment

challenged at 24 weeks of age with 0.5 mL of inoculum containing 5×10^7 cfu of *S.* Enteritidis PT4 S1400/94. At three weeks post challenge, the number of oviducts from which *Salmonella* was isolated was significantly lower in the vaccinated than in the non-vaccinated hens. Twelve of 105 (11.4%) batches of eggs were contaminated in birds vaccinated with either vaccine compared to 28 of 105 (26.7%) batches in the unvaccinated group. Contamination was much lower at only 1 of 105 (1.0%) in hens vaccinated with both vaccine strains. These data support the thesis that live vaccines could be valuable in controlling intestinal colonization and internal egg contamination with *S.* Enteritidis. There is compelling evidence that an iron-restricted S. Enteritidis PT4 strain 109 vaccine is effective in laying chicken following intramuscular injections and intravascular challenge[102]. Since *S.* Enteritidis spreads through a flock primarily via faecal contamination, vaccines would be considered effective if it increased specific antibody levels in the digestive tract and reduced the amounts of *S.* Enteritidis shed in feces while preventing egg contamination and overall disease transmission by limiting invasion of the bird's internal organs such as the ovaries where eggs are infected internally.

While killed vaccines stimulate only humoral immunity, live *Salmonella* vaccines have the capacity to stimulate both cell-mediated and humoral immunities. Table 1 presents a list of recent attempts by different laboratories to evaluate efficacy of *S.* Enteritidis vaccine candidates.

Limitations to complete eradication of *S.* Enteritidis from the hen house

Although identification of eggs as the main source of *S.* Enteritidis food poisoning outbreaks suggested that effective control could be achieved by intensive sampling of birds and diversion of suspect eggs from the market, farmers, public health officials and retailers have been confounded by the intractable nature of the problem of *S.* Enteritidis[12,14,83]. The *S.* Enteritidis pandemic has been most frustrating because it involves the interaction of the pathogen with multiple environments including the hen house (presence of rodents, insects, wild birds, poor ventilation, and accumulation of dust in the hen house), poultry and eggs, as well as the human host. Thus while many laboratories have put in place elaborate efforts to control *Salmonella* infections through vaccinations, creation of a *S.* Enteritidis free hen house remains evasive. This is confounded by the fact that (i) infected adult chickens may remain asymptomatic while producing *Salmonella* contaminated eggs, (ii) infections can be transmitted through cracks on egg shells (iii) horizontal infection is possible among birds in a flock, and (iv) infections are associated with environmental factors such

Table 1. Recent attempts at evaluation of Salmonella Enteritidis vaccine candidates

Source	Vaccine type	Methods	Observation
De Buck et al. 2005[22]	Purified type 1 fimbriae from *S.* Enteritidis.	Chickens immunized intra-peritoneally at 18 and 21 weeks of age; Three weeks later, immunized and non-immunized birds (n = 18) were challenged intravenously with 2×10^7 live *S.* Enteritidis.	IgG and IgA were found in eggs and sera of immunized birds. Higher infections in oviducts and *Salmonella* contamination in eggs of non immunized birds
Betancor et al. 2005[9]	AroC derivative (LVR02) of Uruguay strain *S.* Enteritidis.	Oral administration to newly hatched chicks and second dose at 15 days post-hatching.	Protective immunity to oral challenge with *S.* Enteritidis. Systemic and intestinal infection prevented. Shedding of challenge strain in birds faeces significantly reduced.
De Buck et al. 2004[21]	Parent strain *S.* Enteritidis Or *fimD* mutant (without type 1 fimbriae).	Chickens inoculated intravenously with fimD mutant or its parent strain.	*fimD* mutant present in blood 3 weeks after infection but wild-type parent strain was cleared in the first 3 days. Eggs of birds infected with *fimD* were less frequently contaminated with *Salmonella*, but shells of eggs were more frequently contaminated with the wild-type strain than with the mutant.
Babu et al. 2004[6]	Live and killed *S.* Enteritidis.	Chickens 1st immunized at 2 weeks of age followed by a booster dose at 4 weeks, then challenged with *S.* Enteritidis 2 weeks later (6 weeks of age) and tested for CMI and antibody responses *to S.* Enteritidis 1 week post-challenge (7 weeks of age).	*S.* Enteritidis flagellae and con A induced higher splenic cell proliferation. *S.* Enteritidis shedding was lower in live vaccine group. Splenic CD3 significantly lower and B cells were higher in control group compared to *S.* Enteritidis challenged group. Serum antibodies to *S.* Enteritidis flagella and envelope were significantly higher in killed vaccine group.
Khan et al. 2003[50]	*S.* Enteritidis outer membrane proteins (OMPs) : 75.6 and 82.3kDa proteins	Twelve 9-wk-old specific-pathogen-free chickens were put in 3 groups of four (I&II immunized subcutaneously with 10 µg of 82.3 kDa, 75.6 kDa, respectively and III; not immunized). Immunized chickens were boosted twice with the same amount of proteins at two week intervals and challenged one week after last boost with 1 ml of 8×10^8 cfu *S.* Enteritidis culture. All chickens were sacrificed 48 h after the challenge.	Immunization with OMPs 75.6 and 82.3 kDa proteins led to reduced colonization of *S.* Enteritidis in intestinal mucosa of chicken. Sera from immunized birds reacted with the 76.5 and 82.3 kDa proteins and not unimmunized sera
Fukutome et al. 2001[32]	Liposome-associated ultrasonicated whole cell extracts of *S.* Enteritidis PT4 strain 582	Eight-week-old chickens were immunized with liposome-associated or liposome-free *S.* Enteritidis cell lysates either intraocularly or intranasally (200 µg or 4 mg protein/100 µL into both eyes or nasal cavities or the same doses in 4 mL orally into the gizzard using a vinyl catheter). Immunization was repeated twice or thrice at 2-week intervals and peripheral blood and intestinal mucosa were collected 1 or 2 weeks after the last immunization.	Liposome-associated antigen induced Serum IgA, IgG, and IgM, but immunization with antigen alone induced only IgG in the intestines. Higher responses were obtained with intra-ocular immunization followed by intra-nasal then oral immunization.
Cooper et al. 1994[16]	Wild-type *S.* Enteritidis, LA5 and a genetically defined *S.* Enteritidis *aroA* vaccine candidate CVL30 (attenuated in BALB/c mice).	Newly hatched chicks were orally dosed with 10^9 cfu of bacteria.	Wild-type *S.* Enteritidis LA5 caused death of 1 in 25 chicks, and gross pathologic symptoms, including pericarditis and perihepatitis in 6 of the 24 survivors after an oral dose of 10^9 cfu. *S.* Enteritidis *aroA* attenuated in BALB/c mice was not virulent.

cfu: colony-forming units; MI: cell-mediated immunity.

as presence of wild birds, insects, rodents in or around the hen house, and farm material. These factors are further confounded by globalization of food supplies, inadvertent introduction of pathogens into new geographic areas by shipping eggs or chickens, movement of travelers, refugees and immigrants, and changing lifestyles with more people eating out at restaurants, canteens, fast food outlets, and street vendors. Insects such as cockroaches and house-flies, as well as chicken droppings can easily get into drinking and feeding trays. Also, wild birds using the hen house as shelter, contamination with human sewage and other wastes, poor ventilation, and high dust levels are believed to aid dissemination of bacteria among chickens by colonization of mucosal surfaces i.e. nares, and conjuctiva[46]. Of the rodents, the house mouse, *Mus musculus* is a rich source of *S.* Enteritidis[20,38,40,42] and the possibility that it may enrich both chicken and human pathogenic strains in the hen house is real.

Although bacteriological screening of hen houses can help to identify flocks at risk[19,42,44], it is equally difficult to isolate *S. Enteritidis* from hen houses even if the flock is producing contaminated eggs[14,19,33,41,77]. Previous reports have suggested that *S.* Enteritidis infection can be decreased in poultry using competitive exclusion by inoculating hatching chicks with beneficial bacterial cultures[65,70]. Also, moulting, a temporary cessation in egg production induced in older birds by withholding feed and water[23] has been suggested as a strategy that can alter commensal gut flora of poultry. Although this practice increases productivity and extends useful life of the flock, it leads to increased shedding of *Salmonella* in feces[45,46] and risk analysis has suggested that it may double the incidence of egg contamination[91]. According to Kogut et al. (1999)[55], moulting is stressful to birds.

Limitations to faster development of viable vaccines

Despite the elaborate efforts by many laboratories to produce viable vaccines to *Salmonella* infection (Table 1), serious limitations remain to be addressed. The fact that chicks are essentially gnotobiotic when hatched and have gastrointestinal tracts filled with meconium makes them particularly susceptible to colonization of the gastro-intestinal tract by *Salmonellae* and other microflora[73] and may affect attempts to control colonization by live oral vaccines[16].

Although unrelated studies have suggested that inactivated Salmonella vaccines can protect experimental animals against salmonellosis[28,56,58], there are contradictions in the literature relating to their efficiency. These contradictions partly arise from variations in formulations, adjuvants, methods of bacterial inactivation, and animal models used thereby making it difficult to compare results[8]. That inactivated vaccines would lead to serum antibody responses but fail to elicit cell mediated immune responses which are considered important for long term protection and secretory IgA which is needed for protection of mucosal surfaces raises more questions about their efficacy. Use of live vaccines on the other hand raises fears that residual virulence could enter the human food chain or that the public may not readily accept use of genetically manipulated organisms in the food producing animals. Also, given the relatively longer time required to develop and register new live vaccines before use and the threat of rapid emergence and decline of different serovars as seen in *S.* Enteritidis PT4, there is a possibility that the *Salmonella* problem for which a vaccine is developed may no longer exist by the time the vaccine is licensed for use in the field[8]. The risk that residual virulence may also result in vertical transmission and affect productivity cannot be overstated. On the contrary, use of inactivated or subunit vaccines overcome these problems in that they are relatively quick to produce, are stable, and do not contaminate the environment with genetically modified micro-organisms. While fully defined genetic deletions are preferable in live vaccines for purposes of quality control, attenuated pathogens though attractive lack the virulence of live pathogens[25]. Also, differences in types of antigens used, dosing, routes of administration, timing, as well as age and genotypes of chicken used[6,32,35,60] (Table 1) have slowed consensus on the way forward. In addition, while it is not clear whether data obtained from environmentally naive day-old or young chicks can be applicable to chickens of all ages, data obtained from *in vitro* experiments in complete exclusion of *in vivo* regulatory mechanisms may arguably not be entirely representative of the *in vivo* situation.

Suggested methods for reducing S. Enteritidis infection and transmission

Although the data so far generated are of good scientific merit, the threat of *S.* Enteritidis remains and consensus on data from different laboratories using different approaches (Table 1) is hard to achieve. That *S.* Enteritidis was found in sheds after cleaning and disinfection[21] suggests that total elimination from poultry houses would be a daunting task. However, infection rates can be reduced substantially if multi-sectional approaches are embraced by researchers, environmental law enforcement agencies, public health departments, breeders, food handlers including transporters, those working at slaughter houses, retailers, restaurant workers, and consumers. The following approaches could be considered.

(i) Establishment of inter-laboratory research groups

each addressing specific questions. These studies should be consistent with regard to genotypes of chickens used, study environments, age, sex, feed composition, vaccine types and quantities administered per unit weight of chicks, and routes and timing of vaccine delivery, as well as the types of data collected. Regular peer review of research findings at appropriate symposia would facilitate consensus on management strategies. This approach would be effective especially in areas where chickens or chicks are traded.

(ii) Since vaccinations do not always provide a 100% guarantee that a disease would be prevented from causing losses and widespread use of antibiotics may lead to resistance, breeders should be trained on cleaning and disinfection, use of drag swabs to monitor *Salmonella* contamination before re-stocking poultry houses and hatcheries, regular cleaning of ventilation inlets and fans, occupational health inspections and cool storage to limit growth of Salmonella in eggs, and observance of environmental hygiene and prompt vaccination. Testing of feeds especially those of animal origin, and testing to identify positive flocks should be conducted regularly.

(iii) Strict enforcement of environmental safety laws governing transportation of livestock to slaughter houses, hygienic handling at processing plants, safe transportation of offal meats to consumer outlets, prompt sterilization of sewage, dirt removal, and safe disposal of wastes should be observed.

(iv) Because S. Enteritidis passes through the food chain from primary production to food service establishments, institutions, and house-holds, there is a need to educate food handlers including those working at slaughter houses, processing factories, transporters, and retailers on good manufacturing practices. Home-based consumers should be cautioned to cook poultry products adequately before consumption.

(v) Constructing a prompt reporting system of new *S.* Enteritidis isolates for researchers would counter emerging threats and facilitate epidemiological analysis. Actually, PulseNet of the Centers for Disease Control and Prevention in the USA (http://www.cdc.gov/pulsenet/) and PulseNet Europe (http://www.pulsenet-europe.org/) provide useful information on *S.* Enteritidis. A similar approach is under consideration in Japan[98]. These databases provide standardized molecular subtyping (or "fingerprinting") of food borne disease-causing bacteria by pulsed-field gel electrophoresis.

Conclusion

Because poultry products are regularly consumed by humans, the risks of *S.* Enteritidis-derived salmonellosis with the attendant public health and economic problems are real. Proper management practices can lead to significant reduction in the degree of transmission and infections. Inter-laboratory approaches to vaccine development and training of breeders on environmental management at and around hen houses can lead to significant reductions in transmission and overall infections. Good manufacturing practices, hygienic handling of poultry products at processing plants and retail outlets as well as strict adherence to environmental protection laws governing safe transportation of livestock to slaughter houses and offal meat to consumer outlets should be observed. Prompt and efficient sewage treatment and safe waste disposal, as well as hygienic handling and adequate cooking of poultry products before consumption can also limit spread of infections.

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