Avian Salmonellosis: A review

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Abstract: Salmonellosis is caused by bacteria of the genus Salmonella and is a worldwide-considered major zoonosis with a risk for the public health due to the capacity of dissemination between animals and nature. Salmonellosis is the most common avian diseases that is communicable to humans. This article provides the vital information on the epidemiology, pathogenesis, diagnosis, control and public health concerns of avian salmonellosis. Several measures have been used to prevent and control Salmonella infections in poultry and vaccination is the most practical measure and effective method to control and prevent Salmonellosis. Salmonella vaccines can decrease public health risk by reducing colonization and organ invasion, including reproductive tissues, and by diminishing fecal shedding and environmental contamination. This review article will make progress in reducing and eliminating avian salmonellosis from the poultry flocks, thereby reducing potential hazards to the public health posed by these bacterial diseases.
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Introduction

Salmonella infection caused by a variety of *Salmonella* species is one of the most important bacterial diseases in poultry causing heavy economic losses through mortality and reduced production ***(Haider et al., 2004)***. Avian salmonella infection may occur in poultry either acute or chronic form by one or more member of genus *Salmonella*, under the family *Enterobacteriaceae* ***(Hofstad et al., 1992)***. Besides, motile Salmonellae (paratyphoid group) infection cause salmonellosis in chickens and have zoonotic significance.

Avian salmonellosis is the most devastating disease worldwide. The epidemiology of fowl typhoid and pullorum disease in poultry, particularly with regard to transmission from one generation to the next is known to be closely associated with infected eggs ***(Wigley et al., 2001)***. The birds that survive from clinical disease when infected at a young stage may show few signs of infection but can become carriers ***(Berchieri et al., 2001).***

Salmonella is one of the most important pathogenic genera implicated in food borne bacterial outbreaks diseases both in developed and developing countries and constitute an important public health problem ***(Erdem et al., 2005)***.

Salmonella enteritidis (*S. e nteritidis*) is the main cause of food borne salmonellosis ***(Collighan et al., 2001)*** and during the last 20y; it has been a major causative agent of foodborne gastroenteritis in humans ***(Clayton et al., 2008)***.

Epidemiology of Avian Salmonellosis

Avian *Salmonella* infections are important as both a cause of clinical disease in poultry and as a source of food-borne transmission of disease to humans. Under the family of *Enterobacteriaceae*, the genus *Salmonella* is a facultative intracellular pathogen causing localized or systemic infections; as well as a chronic asymptomatic carrier state ***(Shivaprashad, 1997)***. The etiological agent of fowl typhoid and pullorum disease is *Salmonella enterica* subsp. *enterica* serovar Gallinarum, which is divided into two distinct biovars under the serogroup D1, Gallinarum and Pullorum, which are denoted as *Salmonella. gallinarum* and *Salmonella. pullorum,* respectively ***(Shivaprashad, 1997).*** In addition to *Salmonella. gallinarum-pullorum*, other salmonellae such as *Salmonella. enteritidis, Salmonella. panama* and *Salmonella. dublin* also belong to the serogroup D1 ***(Minor et al., 1984)***. The various motile and non-host adapted highly invasive serotypes such as *Salmonella enteritidis* and *Salmonella typhimurium* are commonly referred to as paratyphoid salmonellae ***(Gast et al., 1997).*** Age wise prevalence of avian salmonellosis showed highest infection rate in adult layers (53.25%) in comparison to brooding (14.55%), growing (16.10%) and pullet (16.10%) chickens ***(Rahman et al., 2004)***. Various routes of infection have been described. Oral route of infection represents

the normal route of infection ***(saif et al., 2003)***. Although infection in newly hatched chicks by nasal and cloacal route are also considered as the important route of transmission. Chicks may be infected early by vertical transmission either from an infected ovary, oviduct or from the infected eggs during the passage through the cloacal faeces from infected or carrier hens. The birds survive from clinical disease when infected in young stage may show few signs of infection but they become carriers ***(Berchieri et al., 2001)***. In adult carriers the reproductive organs are the predilection sites that often lead to the infection of ovarian follicles and as a result transovarian transmission of the disease occurs. The bacteria are passed out through the faeces and lateral spread takes place through the fecal contaminated feeds, water and litter ***(Agabou, 2009)*.** Although more than 2,300 serotypes of *Salmonella* have been identified, only about 10% of these have been isolated from poultry **(Hafez, 2013)**. Chickens are the natural hosts for the highly host adapted biovar *S. gallinarum* and *S. pullorum*, but natural outbreaks have also been reported in turkeys, guinea fowl, quail and pheasants **(Shivaprasad *2000*)**. Fowl typhoid is a peracute, acute or chronic form of disease affecting mostly adult chickens, whereas pullorum disease affects the very young chickens, mostly 2−3 weeks of age. In the adult it tends to be chronic **(*Shivaprashad., 1997*)**. Fowl typhoid is frequently referred to as a disease of adult birds; still, there are also reports of high morbidity and mortality in young chickens **(shivaprasad and Barrow, 2008)**. *S. gallinarum* can produce lesions in chicks, which are indistinguishable from those associated with pullorum disease ***(Shivaprashad, 1997)***. A certain percentage of chickens that survive from the initial infection become carriers with or without presence of clinical signs and pathological lesions ***(Shivaprashad et al., 2000)***. Crowding, malnutrition, and other stressful conditions as well as unsanitary surroundings can exacerbate mortality and performance losses due to salmonellosis, especially in young birds ***(Waltman et al., 2008).*** The potential risk factors responsible for *Salmonella* contamination of broiler-chicken flocks in more recent years, the use of DNA-related techniques such as plasmid analysis ribotyping ***(Usera et al., 1994)***, and SDS-PFGE ***(Olsen et al., 1994)*** have proved to be useful in discriminating isolates of *Salmonella* species. ***(Lapuz et al., 2008)*** investigated the prevalence of *Salmonella* in four layer farms in eastern Japan between 2004 and 2006 to determine the role of roof rats **(*Rattus rattus*)** in the epizootology of *Salmonella enterica* subsp. *enterica* serovar Enteritidis (*S. enteritidis*). They suggested that roof rats were carriers of *S. enteritidis* and *S. infantis* and that persistent *S. enteritidis* and *S. infantis* infections in a rat population might play an important role in the spread and maintenance of these pathogens inside the layer Pakistan ***(Jha et al., 1994)***. Fowl typhoid is common in both backyard chickens and in commercial poultry ***(Fricker et al., 1987).***

Salmonella and other food borne pathogens acquire antibiotic resistance by random chromosomal mutations, mutation of existing genes, and through specific mechanisms such as transduction, tranformation, and conjugation ***(Okolo et al., 1986)***. These mechanisms involve transfer of drug resistant genes by means of circular DNA plasmids such as R-factor, conjugative plasmid, or chromosomal elements ***(Wagner et al., 1999)***. The occurrence and proliferation of antibiotic-resistant *Salmonella* in environmental samples, poultry, and other animals and humans may be due to the use of medicated feeds ***(Gast et al., 1988)***, the practice of dipping hatching eggs in solutions containing antimicrobial agents routine inoculation of day-old poults with antibiotics **(Ekperigin *et al.*, 1983)** and treatment of other animals ***(Pacer et al 1989)*** and humans ***(Holmberg et al., 1894)*** with antibiotics. *Salmonella* strains of avian origin are also often resistant to variety of antimicrobials approved for poultry including tetracycline, sulfisoxazole ***(Parveen et al., 2007)***, oxytetracycline ***(Hoare, 2013)***, penicillin aminoglycosides ***(Poppe et al., 1995)***, fluoroquinolones ***(Herikstad et al., 1997)***. Nalidixic acid is used in poultry industry therapeutically, however when used in prophylaxis selects resistant strains, wich may be transmitted to other birds, animals and to man Layers and broilers alike represent the main reservoir of resistant salmonella **(Ribeiro *et al.*, 2007)**. On the other hand, **(Horska and Yespolov, 2013*)*** found several strains of multiple antibiotic-resistant *Salmonella* strains in chicken.

Pathogenesis and Disease Syndrome of Avian Salmonellosis

The pathogenicity of *Salmonella* depends on the invasive properties and the ability of the bacteria to survive and multiply within the cells, particularly macrophages ***(Humbert et al., 1997)***. The main site of multiplication of these bacteria is the digestive tract, which may result in widespread contamination of the environment due to bacterial excretion through feces. Following invasion through the intestinal mucosa, cecal tonsils and Peyer's patches, the organisms are engulfed by macrophages, and through the blood stream and/or lymphatic systems, they spread to organs rich in reticuloendothelial tissues (RES), such as liver and spleen, which are the main sites of multiplication **(Revolledo *et al.*, 2006)**. In case of inadequate body defense mechanism, they may lead to second invasion and be localized in other organs, particularly ovary, oviduct, myocardium, pericardium, gizzard, yolk sac and/or lungs ***(Barrow, 1993)***. In the

bird challenge, *S. typhimurium* rapidly caused inflammation of the intestinal mucosa, but *S. pullorum* preferentially targeted the bursa of Fabricius prior to eliciting intestinal inflammation **(Henderson *et al*., 1999)**. Pullorum disease manifests itself predominantly as an enteric disease of chickens, while fowl typhoid shows signs of septicemic disease ***(Shivaprashad, 1997)***. Both biovars can cause septicemic infections, which may be acute or chronic, but unlike *S. pullorum*, *S. gallinarum* is capable of producing peracute infection and hemolytic anemia in both young and adults ***(Christensen, 1996)***. *S. gallinarum* is extremely pathogenic to young broiler chicks *(Wray and Davies, 2001)*.

Fowl typhoid is indistinguishable from pullorum disease unless the etiological agent is isolated and identified **(Lowry *et al.*, 1999)**. Clinical signs in chicks and poults include anorexia, diarrhea, dehydration, weakness and high mortality ***(*Shivaprasad and Barrow, 2008*).*** In mature fowls, fowl typhoid and pullorum disease are manifested by anorexia, drop in egg production, increased mortality, reduced fertility and hatchability. *S. pullorum* infected adult birds may or may not exhibit any clinical signs, or they cannot be detected by their physical appearance ***(Shivaprashad, 2000)***. Furthermore, the exact mechanisms of getting these poultry diseases are still remained to be obscured.

Diagnosis of avian salmonellosis: should be confirmed by isolation, identification, and serotyping of *Salmonella* strains. Infections in mature birds can be identified by serologic tests, followed by necropsy evaluation complemented by microbiologic culture and typing for confirmation. A serological ELISA test for the diagnosis of avian salmonellosis either with *S. typhimurium* or *S. enteritidis* has been established **(Kles *et al*., 1993). (Szmolka *et al*., 2006)** established a diagnostic and a real-time PCR system for rapid and reliable genus- and serovar- (*S. enteritidis* and *S. typhimurium*) specific detection of *Salmonella* for monitoring purposes in the poultry food chain. Molecular techniques for accurate, rapid, economical diagnosis of multiple serotypes of Salmonellae molecular techniques have become available as ribotyping, differentiation by genes, PCR, multiplex PCR ***(Singh et al., 2012).***

Preventive Measures for Controlling *Avian Salmonellosis*

Although fowl typhoid and pullorum disease are widely distributed in most parts of the world, the diseases have been eradicated from commercial poultry in developed countries such as the United States of America, Canada and most countries of Western Europe ***(Shivaprashad, 1997)***. Successful control programs can be achieved by developing good hygiene and management together with routine serological tests and slaughter policy ***(Barrow et al., 1993)***. The principal management procedures should include chicks free from infections, and the chicks should be placed in a cleaned, sanitized and *S. gallinarum* and *S. pullorum* free environment with strict biosecurity measures **(Pomery *et al*., 1991).** The feed and water should be free from *Salmonella* contamination. The dead birds need to be well disposed. Adequate precautions are needed to prevent infections from mechanical carriers like footwear, human clothing, hatchery disciplines, equipments, litters, crates, trucks and processing plants **(Christensen *et al*., 1994)**. Wray *et al., 1996* described that the birds need to be tested at the age of 16 weeks due to immunologic maturity, at the point of lay due to stress and two consecutive times one month apart to provide the acceptable evidence that the flock is free from fowl typhoid **(Barrow *et al*., 1994)**. Kabir, 2009 demonstrated the potential role of probiotics for the controlling of *Salmonella* strains of poultry via the mechanisms of competitive exclusion. Vaccines may be used to control the disease, and antibiotics can be used for the treatment of fowl typhoid and pullorum disease.

Live vaccines consist of live organism and can be given at a younger age, can be administered by drinking water, eye drop or inhalation. Killed vaccines must be injected. In case of a disease outbreak diagnosis should be confirmed. Laying hens should be vaccinated with live and killed vaccines to stimulate mucosal and systemic immunity and reduce the prevalence of *S. Enteritidis* contaminated eggs ***(Davies and Breslin, 2004)***

Immunization significantly reduce number of hens infected and the rate of egg transmission, but does not completely eliminate *S. Enteritidis* ***(Gast, 2007)***. Maternal antibodies in the yolk can resulted in slow the growth of Salmonella in internally contaminated eggs. Vaccination of commercial laying flocks against *S. Enteritidis* has been associated with a dramatic reduction of human infections in the UK ***(O'Brien, 2013).***

Vaccination of fattening turkey flock with live commercial *S. Enteritidis* at day 1 of age via spray and boostered at 6 and 11 weeks of age via drinking water did not reduce shedding or colonization of internal organs when birds were challenged with *S. Enteritidis* PT4 ***(Krüger et al., 2008).***

Public Health Concerns

Salmonellosis is of public health concern because most of the strains of *Salmonella* are potentially pathogenic to humans and animals. Avian salmonellosis can pose a health risk to people if exposed. Symptoms appear similar to food poisoning, such as diarrhea and acute gastroenteritis. However, it appears that birds mainly acquire the disease from the

environment and that infected birds play a relatively small role in the transmission of disease to domestic animals and humans. Public health concerns and the potential for food borne zoonotic transmission have made *Salmonella* the subject of numerous international, national, and local surveillance programs ***(Yan et al., 2003).***

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