



Effect of Salmonella on Hatchability and Fertility

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Abstract: *Salmonella* infection is not only infecting poultry but also emerging as a pandemic in public health. *Salmonella* affecting poultry are *Salmonella* Pullorum (*S. Pullorum*), *Salmonella* Gallinarum, (*S. Gallinarum*) *Salmonella*, Typhimurium (*S. Typhimurium*), which are collectively categorized under *S. Enterica* (*S. Enterica*). It causes a systemic infection in poultry birds primarily gastroenteritis with colonization of bacteria in liver, spleen, intestines, ovary, oviduct and vagina. Poultry eggs are more importantly contaminated with salmonella infection that originate from transovarian route (vertical route) or from contaminated hen house (horizontal route). In severe cases *Salmonella* infection causes a decrease in egg production, reduction in fertility and low hatchability of infected eggs. Albumin, yolk and other shell contents get contaminated with bacteria. They serve as source of nutrition for microorganisms. Hatcheries and egg storing places also present risk of *Salmonella* contamination. They possess bacterial micro flora that may contaminate eggs before they are processed or set in the incubator. More simply the decrease in hatchability of eggs occurs due to persistence of *Salmonella* infection in hen reproductive tract.

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Introduction

Salmonella is a cause of many serious infections in poultry as well as other avian species involved in heavy economic losses to the industry causing decrease in production by poultry industry through illness and mortality. *Salmonella* infections of poultry are categorized mainly as three types; *Salmonella* Pullorum, *Salmonella* Gallinarum and *Salmonella* Typhimurium. *Salmonella* is transmitted by horizontal as well as vertical route. Bacterial transmission is reported may be due to transmission through ovule (Saif et al., 2008) and later by contamination of egg just after ovulation from *Salmonella* excreting hens, as egg once laid is wet, warm and prone to microbial transferring into shell (Williams et al., 1968) *Salmonella* when passed in the egg shell and egg shell membranes it is rather difficult to restrict further entry of *Salmonella* into the contents or developing embryo. It may be ingested by the embryo (Cason et al., 1994) or may grow and spread in the cabinet (Cason et al., 1993). This leads to production of a bird that spreads the bacterial contamination to the body organs like intestines, feathers etc of other birds (Cox et al., 1990).

Simply a *Salmonella* contaminated egg is responsible for vertical transmission of infection to the chick (Methner et al., 1995) and hatching of that contaminated egg causes horizontal transmission of

infection to the mates (Cason et al., 1994, Bailey et al., 1994). However, ovary infection with motile *Salmonella* after systemic infection is rarely seen (Kim et al., 1989). *Salmonella* is concerned contaminant in broiler breeder eggs however the outcomes may be affected by some characteristics like breed or line (Beaumont et al., 1994, Kinde, et al., 2000). *Salmonella* infection signs may be shell-less, infertile eggs with early embryonic mortality (Coufal et al., 2003, Welish et al., 1997). Persistent fecal shedding of *Salmonella* is also seen in past after inoculation (Brownell et al., 1969, Gast and Beard, 1990b). Infection susceptibility of chicks is age dependent, similarly day-old chicks are most susceptible due to their frail immune system (Barrow et al., 1987, Smith and Tucker, 1980). The presence of cracked shelled eggs in those hatcheries brings problem of penetration of *Salmonella* into the eggs. In this way, single eggs causes contamination of whole batch at the time of hatching or whenever it is broken during incubation period. *Salmonella* starts multiplication when it is inside an egg and keeps multiplying as long as there is integrity of vitelline membrane and it may die during the storage (Himathongkham et al., 1999, Baker, 1990). However, it may survive if it is provided with high relative humidity (Baker, 1990) accompanied with low

temperature (**Baker, 1990**) A mortality of 10-93% is observed in chicks while morbidity may be much more than the mortality (**Saif et al., 2008**). There is a need of *Salmonella* monitoring of eggs using a system by which layer birds are to be tested for any infection. Also hatcheries play a role as the check point in avoiding infection in production cycle (**Cox et al., 2001**). Leaving the floor eggs and strict hygiene of the nests at the farm is necessary to reduce the bacterial load which is a hazard for personal and equipment safety (**Bruce et al., 1990, Wineland, 1990**). Secondly hatcheries should show compliance with rules as described in appendix 3.1.4 of the *OIE* (or equivalent) so that hazards from *Salmonella* can be minimized that will assure the health of flock (**OIE, 2005**)

Transmission:

Transmission and spread of *Salmonella* occur by vertical and/or horizontal routes. The true-vertical transmission occurs by ovarian transmission, by passage through the oviduct or by contact with infected peritoneum or air sacs (**Saif et al, 2003**) Also, Pseudo-vertical transmission by fecal contamination of the egg shell from cloaca and/or contaminated nests, floor or incubators, the organism penetration into the eggs (**Saif et al, 2003**). Transmission of *Salmonella* spp. to breeder flocks by use of contaminated commercial turkey semen (**Iaffaldano et al. 2010**). Horizontal spread of *Salmonella* occurring during hatching was shown when contaminated and *Salmonella*-free eggs were incubated together (**Cason et al., 1994**). Hatched birds may become infected by aerosols containing *Salmonella* (**Agabou, 2009**) or through environment and tools.

Routes of egg contamination by *Salmonella*

Many studies indicate that ovary is more often colonized by *S. Enterica* than other parts of reproductive system e.g. oviduct (**Gast Guraya et al., 2007**). *S. Pullorum* and *S. Gallinarum* also colonize in the ovary (**Pomeroy, 1984, Snoeyenbos, 1984**). Additionally *Salmonella* can escape from the immune system of the hen by perhaps colonizing inside the cells of tract (**Gast and Holt, 2000**) and follicles as well (**Okamura et al., 2001a**). *Salmonella* is found to colonize the oviduct tissue (**Okamura et al., 2001a, Gantois et al., 2008b**). It is indicated that this is the area most frequently contaminated by *Salmonella*, while talking about oviduct colonization (**Keller et al., 1995**). Vagina when experimentally inoculated by *Salmonella* caused high level of bacterial contamination because *Salmonella* has a great affinity for attachment to the vaginal epithelium (**Miyamoto et al., 1997**). *Salmonella* attachment to isthmus and magnum also showed problems of contamination by invading tissue cultures of these organ epitheliums (**De Buck et al., 2004**). It has special affinity for magnum

as well, which reinforce the hypothesis that contamination of egg is by contamination of egg contents [albumin] (**Schoeni et al., 1995**). However, experiments have showed that high levels of colonization do not give rise to high level of contamination (**Methner et al., 1995**). It demonstrates that *Salmonella* when infects egg before it is laid, infection is transferred to the hatchling (**Methner et al., 1995**). Some researchers claim that horizontal transmission to be most important (**Barrow and Lovell, 1991, Bichler et al., 1996**), meanwhile some call vertical transmission to be important (**Gast and Beard, 1990a, Miyamoto et al., 1997, Guard-Petter, 2001**). Some researchers also suggested that *Salmonella* transmission to farms also took place by vertical transmission (**Lister, 1988, Rodrigue et al., 1990**)

Effect of *Salmonella* on hatchability

In freshly laid contaminated eggs there is small number of bacteria (**Humphrey et al., 1989a, Gast and Beard, 1992**). In contaminated eggs the most frequent site of contamination is outside of vitelline membrane (**Humphrey, 1994**). When *Salmonella* infected eggs are incubated, there is an increase in proliferation of microorganism (**Hammack et al., 1993**) but there is no change in color, consistency and smell of the infected egg when kept at room temperature (**Humphrey and Whitehead, 1993**). *Salmonella* infection signs may be shell-less, infertile eggs with early embryonic mortality (**Welish et al., 1997**). It has been observed that fowl typhoid or salmonellosis is associated with mortality, morbidity, decreased growth rate and poor hatchability and fertility in birds (**Haider et al., 2004, Mamta et al., 2010**). Another study showed that a high mortality observed in young birds was associated with *Salmonella* (**Kumari et al., 2013**). If *Salmonella* is transmitted during incubation there may be unpipped or pipped with dead chick eggs (**Hafez, 2007**). Horizontal transmission of *Salmonella enteritidis* occurs during hatching, it cause infection from dust, litter, fecal of chicks (**Oosterom, 1991**). Bruce and Johnson reported that infection increases as the flock hatching age increases (Bruce J and Johnson, 1978). But susceptibility of chicks to *Salmonella* colonization decreases post 1 week of hatching (**Gast and Beard, 1989**). **Kim et al (1989)** reported that *Salmonella* Enteritidis was recovered from yolk of contaminated eggs and ovary of breeder hens. But small number of micro-organisms does not start multiplication inside the albumin (**Benson and Eckroade, 1988**). The newly hatched chicks are gnotobiotic and are vulnerable for *Salmonella* infection Research has shown contamination of ovaries, dead embryos, hatched chicks with high mortality can be due to other serovars of *Salmonella* other than *Salmonella*

Gallinarum Pullorum (**Lindgren, 1985**). Similarly a study on *Salmonella* serotypes showed that *Salmonella harder* and *Salmonella* Kentucky which are adapted in the intestinal environment are also found in hatcheries. They contaminate the hatcheries and horizontal transmission is followed by their contamination. This study also reveals that an infection from these serotypes is found in day-old chicks when they hatched from contaminated eggs (**Muhammad et al., 2009**) it can initiate during hatching (**Cason et al., 1994**). Bacteria can be isolated from hatchery fluffs and meconium of day old chicks (**Bhathia and McNabb, 1980**). **Erbeck et al (1993)** suggested *Salmonella* infection as systemic disease with signs and symptoms and drop in egg production, decreased fertility and reduced hatchability of eggs in pullorum disease [PD] and Fowl typhoid (FT) all depending on severity. Usually the infection originating in hatcheries is not coming from egg shells but contamination of hatchery environment after the hatching of infected egg is thought to be most acceptable. As the fan driven air in hatchery causes movement of *Salmonella* from infected eggs to the noninfected eggs (**Cason et al., 1994, Berrang et al., 1995**). When these infected chicks become pullet, they also produce infected eggs (**Hopper and Mawer, 1988**).

Effect of *Salmonella* infection on fertility

Salmonella enteritis is not always associated with effecting badly on fertility in hens rather infection of *S. Enteritidis* may support its vertical transmission (**Lister 1988 and O'Brien, 1988**). *Salmonella* is recovered from the ovaries and oviduct in the layer flock (**Hopper and Mawer, 1988**). A study to understand the causes of early embryo and chick mortality in Nigeria was conducted, which concluded Omphalitis is associated with infection, probably *Salmonella* or *E.coli* (**Muhammad et al., 2009**). According to study conducted by Seneviratna [1969] and Okoame [1983] concluded that *S. pullorum* infection is a cause of unfertile eggs (**Onasanya and Ikeobi, 2013**). The birds infected with *Salmonella* serotypes such as *S. Enterica* serovar Pullorum and *S. Enterica* serovar Gallinarum are characterized by weight loss, decrease in egg production and high mortality (**Shivaprasad, 2000, Snoeyenbos, 1991**). The production of egg by infected bird is said to descend infection from ovarian tissue. Experimental infection of poultry birds showed that if egg production was depressed, then the eggs produced were also contaminated (**Gast and Beard, 1990b**). **Wigley et al (2001)** showed by experimental inoculation that *Salmonella* Pullorum contaminated eggs when reached maturity, they produced *Salmonella* contaminated eggs. Some studies regarding susceptibility of *Salmonella* infection showed brown shelled egg layer hens are more

susceptible than white egg shell producing hens (**Keller et al., 1995, Kinde et al., 2000**). **Erbeck et al (1993)** suggested *Salmonella* infection in hens as drop in egg production, decreased fertility of eggs in pullorum disease and Fowl typhoid all depending on severity.

Control of *Salmonella* infections in poultry farms

Control of *Salmonella* infections in poultry farms needs to begin with good farming practices and appropriate management associated with strict sanitary measures. Preventive and curative strategies have been widely applied for reducing the incidence of *Salmonella* colonization in chickens at the farm level (**Vandeplas et al., 2010**). Various prophylactic measures have been employed to prevent and control *Salmonella* infection in poultry production, and vaccination is one of them. *Salmonella* vaccination aims to mimic the development of naturally acquired immunity by inoculation of non-pathogenic but still immunogenic components of the pathogen, reducing or eliminating the risk for the consumer. Killed and live attenuated products have been used for controlling *Salmonella* in poultry production, and vaccination with live attenuated products has proved to be more effective (**Cerquetti and Gherardi, 2000**).

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