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► **To cite this version:**

Kashif Aziz Khan, Shakil Akhtar Khan, Asim Aslam, Masood Rabbani, Muhammad Yasin Tipu. FACTORS CONTRIBUTING TO YOLK RETENTION IN POULTRY: A REVIEW. Pakistan Veterinary Journal, 2004, 24 (1), pp.46-51. <hal-00179202v2>

HAL Id: hal-00179202

<https://hal.science/hal-00179202v2>

Submitted on 29 Jul 2008

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FACTORS CONTRIBUTING TO YOLK RETENTION IN POULTRY: A REVIEW

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ABSTRACT

Yolk retention and yolk sac infection is considered as an important cause of death in chicken as well as in guinea fowl, duck, turkey, quail and goose. The factors which slow down the rate of yolk absorption and may in turn, lead to yolk retention are discussed. Yolk sac infection of bacterial origin is most important among these factors. Other factors which may contribute include posthatch starvation, type of initial feed, brooding temperature, prolonged exposure to hatcher environment and size of birds.

Key words: Yolk retention, poultry, yolk sac infection.

INTRODUCTION

During incubation, extraembryonic membranes encircle the yolk substance and constitute the yolk sac, which is attached to gut of the chick by yolk stalk. Just before hatching, the yolk sac is pulled from the egg cavity to abdomen of chick as an extension of intestine. Residual yolk comprises 20-25% of body weight at hatch but within the first week of life it becomes negligible in size (Ramnoff, 1960).

There are certain factors that affect yolk absorption and in turn may lead to its retention. Once bacteria get entry to yolk, other factors favour rapid bacterial growth and these include the fact that the yolk contains a lot of fat and water, favoured nutrients for bacteria. In addition, the yolk sac is maintained at the temperature of the hatcher and then at the chick's body temperature, which are the ideal temperatures for multiplication of certain bacteria (Anonymous, 2000). So yolk retention due to any cause may lead to yolk sac infection.

YOLK RETENTION

a) Incidence

Incidence of yolk retention and yolk sac infection is widely reported in literature. Anjum (1997) stated that it was the commonest cause of early chick mortality in Pakistan. Jordan (1990) and Singh *et al.* (1993) also reported it as the most frequent cause of death in chicks. Incidence of yolk retention and yolk sac infection was reported as 7.5, 10.5, 9.9, 5.1 and 8.9% in chickens by Schonhofen and Garcia (1981), Shrivastava (1982), Rathore *et al.* (1985), Suresh *et al.* (1988) and Bhattacharjee *et al.* (1996), respectively. Incidence was reported to be 15.20 and 20.71% in two

different strains of White Leg Horn by Viswanath *et al.* (1985). Ghodasara *et al.* (1992) found 31.45% mortality in chicks due to yolk sac infection.

Yolk retention is not only the cause of death in chicken but also in other species of poultry including guinea fowl, duck, turkey, quail and goose. It was reported as most frequent cause of death in indigenous guinea fowl by Rudy (1991). Sharma and Kaushik (1986b) and Roy and Misra (1989) found it as an important disease of ducks. Sharma and Kaushik (1986a) found that incidence of yolk retention was 20% in turkey while Thyagarajan *et al.* (1987) found it as 20.8%. Unabsorbed yolk was observed as principal lesion in quails died upto one week of age by Suneja *et al.* (1983) and incidence was reported as 16% by Sharma and Kaushik (1986c). Boado and Rojas (1990) found 7.3% incidence of omphalitis in goose.

Suneja *et al.* (1983) observed that incidence of omphalitis was most frequent in January. Similarly, Sainsbury (1992) reported it's higher incidence towards the end of winter or in the early spring.

b) Effects on host

Putrefactive and offensive odour was observed as characteristic clinical sign of yolk sac infection by Sainsbury (1992) and Anjum (1997). Abdomen of chick felt soft and distended with thickened, inflamed and moist umbilicus. Unabsorbed yolk sac was present in the abdomen and therefore it was named yolk retention. Yolk sac contents changed from viscid yellow green to watery yellow brown due to denaturation of yolk by bacteria (Jordan, 1990; Sainsbury, 1992; Anjum, 1997). Deeming (1995) reported that infected yolk sacs were, in general, larger in mass than uninfected sacs from poult of same age. Yolk sac and subcutaneous blood

vessels were dilated and engorged with blood (Jordan, 1990; Anjum, 1997). Chicks surviving more than four days might have pericarditis as well as infected yolk indicating systemic spread (Barnes and Gross, 1997). Haemorrhagic serous peritonitis was also observed by Jordan (1990) and Anjum (1997). Maximum deaths occurred upto 3 days of age (Sainsbury, 1992; Anjum 1997). In some cases, there might be no mortality with retained infected yolk as only manifestation (Barnes and Gross, 1997).

FACTORS CONTRIBUTING TOWARDS YOLK RETENTION

A. YOLK SAC INFECTION

A 1: Natural infection

a) Causative organisms

Different types of bacterial agents are attributed for causation of yolk sac infection/omphalitis. *Escherichia coli* was frequently the main one involved. Its isolation from yolk sac infection was reported by Zahdeh *et al.* (1984), Sarma *et al.* (1985), Linzitto *et al.* (1988), Jordan (1990), Utomo *et al.* (1990), Sainsbury (1992), Ali (1993), Choudhury *et al.* (1993), Deeming (1995), Rehman *et al.* (1996), Anjum (1997), Sharada *et al.* (1999) and Anonymous (2000). Next frequently found bacteria was genus *Salmonella* which was reported by Zahdeh *et al.* (1984), O'Brien (1988), Mutalib and Hanson (1989), Ali (1993), Choudhury *et al.* (1993), Rehman *et al.* (1996), Anjum (1997), Anonymous (2000) and Shivaprasad (2000).

Staphylococcus species were found to be involved by Zahdeh *et al.* (1984), Utomo *et al.* (1990), Choudhury *et al.* (1993), Deeming (1995), Rehman *et al.* (1996), Anjum (1997) and Anonymous (2000) and streptococcus species by Utomo *et al.* (1990), Ali (1993) and Anonymous (2000). Involvement of proteus species was observed by Zahdeh *et al.* (1984), Sarma *et al.* (1985), Utomo *et al.* (1990), Choudhury *et al.* (1993), Anjum (1997) and Anonymous (2000) and of bacillus species by Utomo *et al.* (1990), Ali (1993), Choudhury *et al.* (1993), Deeming (1995), Anjum (1997) and Anonymous (2000).

Other bacterial genera found to be involved include *Pseudomonas* (Zahdeh *et al.*, 1984; Sarma *et al.* 1985; Utomo *et al.*, 1990; Choudhury *et al.*, 1993; Anjum, 1997; Anonymous, 2000), *Klebsiella* (Zahdeh *et al.*, 1984; Choudhury *et al.*, 1993; Anonymous, 2000), *Clostridium* (Anjum, 1997; Anonymous, 2000), *Micrococcus* (Utomo *et al.*, 1990), *Yersinia* (Ali, 1993), *Enterobacter* (Utomo *et al.*, 1990; Ali, 1993)

Aerobacter (Bhatia *et al.*, 1971), *Citrobacter* (Utomo *et al.*, 1990), *Achromobacter* (Deeming, 1995), *Enterococci* (Zahdeh *et al.*, 1984; Anjum, 1997) and *Alcaligenes* (Utomo *et al.*, 1990). Involvement of *Aspergillus fumigatus* in yolk sac infection was also reported by Schonhofen and Garcia (1991).

b) Source of infection

Farm faecal contamination of shell was reported as source of infection by Anjum (1997). Poor hatchery hygiene condition was considered as another important source (Sainsbury, 1992; Anjum, 1997). William (1975) reported that the source of infection was the waste in the hatchery or contaminated poult boxes or poult box pads. Other source of infection include breeder, feed, environment, feathers, human skin, floor and dirty equipment (Anonymous, 2000).

c) Route of infection

Transmission of bacteria through unhealed naval was revealed by Jordan (1990) and Anjum (1997). Infection through blood stream and contamination of yolk before it is internalized into the chick were reported as other routes of infection by Anonymous (2000).

A 2: Experimental infection

Fuller and Jayne-Williams (1968) demonstrated sub-clinical yolk sac infection after oral administration of pure cultures of bacteria. It was concluded that the infection arose through translocation of bacteria across the gut wall. Seigo *et al.* (1970) inoculated *Bacillus cereus* through intrayolk, intraperitoneal, subcutaneous and oral routes. Omphalitis was reproduced only when inoculated into the yolk sac. Singh *et al.* (1997) studied the pathogenicity of *Escherichia coli* by intraperitoneal injection into 2-day-old chicks. Unabsorbed yolk sac was among the main lesions. Sander *et al.* (1998) observed retained yolk in chicks which received *Enterococcus faecalis* broth inoculation into yolk sac. Omphalitis was also observed as gross lesion in experimental infection with *Salmonella harder* and *Salmonella enteritidis* by Desmidt *et al.* (1998) and Dhillon *et al.* (2001), respectively. Khan *et al.* (2002) inoculated *E. coli* broth into yolk sac of day-old chicks and observed high yolk sac weight in these chicks as compared with the chicks inoculated with sterile broth. Seigo *et al.*, (1970) produced the disease by inoculating *Bacillus cereus* inside the egg shell of pipped eggs. Zahdeh (1987) observed severe oedematous swelling around navel orifice, severe omphalitis and incomplete withdrawal of yolk sac in chicks hatched from embryonated eggs that were dipped in 24-hours bacterial broth culture on 18th day.

B. POSTHATCH STARVATION

Slow absorption of yolk due to fasting has been reported by many workers. Moran and Reinhart (1980) observed that fasting led to a reduced uptake of yolk as compared to fully nourished birds. It was reported further that fasting favoured removal of moisture and lipid to a greater extent than protein while the converse was true if access to feed and water was permitted. Observations of Noy *et al.* (1996) also showed that yolk utilization was more rapid in fed than in fasted chicks, suggesting that the transport of yolk through the intestine could be increased by the greater intestinal activity found in fed chicks. Similar findings were observed by Santos and Silversides (1996) that starving chicks were unable to use the yolk sac nutrients, suggesting that yolk sac utilization seems to be correlated with activation of the digestive system.

In contrast, Murakami *et al.* (1992) found that posthatch starvation in chicks decreased carcass lipid content but did not modify the disappearance rate of yolk in the abdomen. Similarly, Chamblee *et al.* (1992) observed that availability of feed and water did not affect body weight or yolk sac absorption during first 24 hours. Al-Rawashdeh *et al.* (1995) observed non-significant difference in yolk weight of chicks after subjecting one-day-old chicks to five days starvation, with drinking water available freely. Baiao *et al.* (1998) delayed the housing of chicks for 24, 48 and 72 hours after hatching and observed that yolk sac absorption was not affected by the period of feed withdrawal between hatching and housing.

Efficient absorption of yolk due to fasting was reported by Pisarsaki *et al.* (1998b). The author delayed first feeding and watering of chicks for 24, 48 and 72 hours in two subsequent experiments and observed that yolk absorption/body weight ratio was higher in chicks fed 24-72 hours later than the control ones.

C. TYPE OF INITIAL FEED

Pisarsaki *et al.* (1998a) substituted commercial starter ration with ground corn in the first 12, 24 and 36 hours of life and concluded that corn feeding in early life led to slow absorption of yolk sac contents. The levels of lysine and methionine also have some effect on absorption of yolk sac in chicks (Wang *et al.*, 1994).

D. BROODING TEMPERATURE

Leeson *et al.* (1978) reported that incidence of unabsorbed yolk was increased by the fluctuating environmental temperature, with little difference for the cold and hot environments, compared with a control situation. Thaxton *et al.* (1974) found that yolk sac weight was not changed by lowered brooding temperature, while Yousaf (1985) observed that yolk

sac weight was decreased by cold temperature brooding but not until 72 hours.

E. MISCELLANEOUS FACTORS

Martin (1996) studied the effect of prolonged exposure to hatcher environment on yolk sac size and observed enlarged yolk sac in broiler chicks. In contrast, Chamblee *et al.* (1992) reported that chicks kept in hatcher for 24 hours and chicks kept in the hatcher for 12 hours and then on litter for 12 hours exhibited non-significant differences in body weight and yolk weight. Knizetova *et al.* (1989) observed rapid resorption of yolk sac contents in large duckling and goslings than in small ones.

CONCLUSION

It can be inferred from the above given findings that yolk retention and yolk sac infection is widely recorded in different species of birds and assumed as an important factor for early chick mortality. Some workers have related its incidence with winter.

The cases of yolk retention and yolk sac infection were recorded upto 10 days of age but high rate of mortality was observed upto 3 days of age. Putrefactive odour from the birds can be assumed as first signal for the farmers. The flabbiness and distention of abdomen, moist umbilicus and change in size, consistency and appearance of yolk can be thought as indicative of yolk sac infection. The affected birds also exhibit systemic manifestations such as pericarditis and peritonitis.

Diverse species of bacteria and *Aspergillus fumigatus* cause natural cases of yolk sac infection. Out of more than a dozen genera of bacteria, mainly the members of family Enterobacteriaceae are predominant. It is difficult to establish, which species of bacteria acted as primary factor and which one acted as secondary opportunist. On the basis of information relating to bacterial origin of yolk sac infection, it is tempting to speculate that *Escherichia coli* and *Salmonella* spp, recorded in natural cases, are the primary pathogens. The main route of infection is through unhealed navel but in some instances transmission is possible by blood stream and by contamination of yolk before it is inverted into the chick. Main sources of infection are faecal contamination of hatching eggs, contaminated hatchery equipments, poor hatcher environment and unhygienic poult boxes.

Experimental yolk sac infection can be produced in chicks by different routes i.e. intrayolk, intraperitoneal,

navel swabbing, oral and subcutaneous, but intrayolk and intraperitoneal are the best. Infection can also be produced by inoculation inside the egg shell of piped eggs or by exposing embryonating eggs to bacterial growth in broth. Different types of bacteria were used by different workers.

Reports regarding posthatch starvation are controversial. According to many workers yolk absorption is delayed due to posthatch starvation while other reported that it did not affect yolk absorption. Even efficient absorption of yolk due to fasting is also reported.

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