Mycoplasma synoviae in broiler breeders

Zeinab M. S. Amin Girh[1], Nagwa S. Rabie[1] and Mona S. Zaki[2]

[1]Department of Poultry Diseases, National Research Centre, Dokki, Giza, Egypt.
[2]Hydrobiology Department, National Research Centre, Dokki, Giza Egypt.
dr_mona_zaki@yahoo.co.uk

Abstract: Broiler breeders are one of the most important components of the poultry industry. This type of birds is susceptible to several agents that interfere with the immune system and predispose to infection. If transmission of pathogens to progeny is considered, their economic impact will be amplified in the broiler farms *Mycoplasma synoviae* (MS) is an important pathogen of poultry worldwide, causing respiratory tract infection and infectious synovitis in chickens and turkeys. A general review of the scientific literature concerning *M. synoviae* in broiler breeders is presented on their epidemiology, economic importance, pathogenesis, lesions, clinical signs, diagnosis, control, treatment and prevention.

[Zeinab M. S. Amin Girh, Nagwa S. Rabie and Mona S. Zaki. ***Mycoplasma synoviae* in broiler breeders.** *Rep Opinion* 2019;11(1):1-4]. ISSN 1553-9873 (print); ISSN 2375-7205 (online). http://www.sciencepub.net/report. 1. doi:10.7537/marsroj110119.01.

Keywords: *Mycoplasma synoviae;* broiler; breeder

Introduction

Broiler breeders stay long periods in the rearing and production sites. This means that they are susceptible to several agents that interfere with the defense system and predispose to infection. Infections are very often apparently subclinical, but still induce damage in the infected hosts and may cause immunosupression **(Feberwee *et al*., 2008)**. If transmission of these pathogens to progeny is considered, the economic impact will be amplified to the broiler farms, compromising the overall production results **(Kleven, 2003; Stipkovits *et al*., 2011).** The success of this fragile organism in infect infecting poultry flocks throughout the world indicates that evolving poultry management practices have facilitated the survival and transmission of this agent **(Ferguson- Noel and Noor mohammadi, 2013).**

Aetiology and Economic importance

Several species of the genus *Mycoplasma* are pathogens of mammals, birds, reptiles, fish and arthropods, causing a wide variety of diseases and having a predilection for the respiratory and the genital tracts as well as to joints **(Vogl *et al*., 2008)**. *Mycoplasmasynoviae* is a species of the class Mollicutes and was designated as serotype S by **Dierks *et al.* (1967)**. Ingeneral, *Mycoplasma* spp. is characterized by their small genome size and is thought to have undergone reductive evolution, losing many genes possessed by more complex bacteria in the process. They also lack many genes, including those for cell wall synthesis and for the production of all 20 amino acids, as well as genes encoding enzymes of the citric acid cycle and the majority of all other biosynthetic genes. Presumably, they can survive with a reduced genome as they have evolved in such a way as to acquire these products from their host *in vivo* **(McAuliffe *et al*.,2006)**. This accounts for the "fried egg" type of colony morphology, resistance to antibiotics that affects cell wall synthesis, and complex nutritional requirements. Mycoplasm as tend to be quite host specific, some infect *only* a single species of animal, but others may have the ability to infect several different animal species **(Ferguson-Noel and Noor mohammadi, 2013).** *Mycoplasma synoviae* is responsible for infectious synovitis and causes economic losses because of decreased egg production, growth and hatch ability rates, and downgrading of carcasses at slaughter due to air sacculitis and arthritis lesions **(Marois *et al*., 2005; Peebles *et al*., 2011)**. Fertile eggs have a reduced hatchability due to late embryonic mortality. *Mycoplasma synoviae* infection is usually subclinical in broiler breeders, but the fact that it might play an important role in an offspring complex respiratory disease has motivated breeding companies to consider eradication **(Fiorentin *et al*., 2003).**

Epidemiology and pathogenesis

Mycoplasma synoviae can be found in eggs laid by infected breeders. Vertical (i.e. transovarial) transmission is not very efficient, as peak egg transmission from an infected breeder flock is low. If complicating factors are present, such as immune suppression, there may be a higher shed of the organism **(Behbahan *et al*., 2005)**. Vertical transmission plays a major role in spreading of *M. synoviae*. When commercial breeder flocks become infected during egg production, egg-transmission appears to be higher in the first 6 weeks after infection. After the chicks are hatched, *M. synoviae* organisms are spread horizontally. Transmission

occurs among birds by the aerosol route and by contamination of the feed and water. The entire flock may be infected at 3weeks of age **(Kleven, 2003).** Horizontal transmission readily occurs by direct contact. In general, *M. synoviae* appears to spread more rapidly than *M. gallisepticum*. The former can be present in the respiratory tract of infected chickens for 4 weeks and during that time the spread between houses occurs **(Ferguson-Noel and Noor mohammadi, 2013).** Natural infection can be observed from the first week of age, but acute infection is more often seen when chickens are adult. This fact suggests that the incubation period can be relatively short, but it generally lasts for 11-21 days. Chronic infection may or may not follow the acute phase at any age and persist for the entire life of the flock **(Kleven, 2003)**. Therefore, *M. synoviae* contaminated environment is a potential hazard to birds. *Mycoplasma synoviae* is also well known for its interactions with other infectious agents and environmental factor salike in producing clinical disease. Control of clinical manifestations is simplified when concurrent infections are minimized and optimal environmental conditions are provided. Respiratory infections are considerably affected by environmental factors and disease severityis increased during the winter months. Temperature, ventilation, humidity, atmospheric ammonia and dust all have important interactions with infectious agents in producing respiratory disease **(Landman, 2014).** Atmospheric dust significantly increased the severity of air sac lesions, and chickens maintained at environmental temperatures of 7-10ºC were more susceptible to airsacculitis caused by *M. synoviae* than chickens maintained at 24-29ºC **(Kleven, 2003)**. Mycoplasm as also infect other domestic and wild avian species, so it is important to ensure they are not in contact with commercial chickens. Some data provide strong evidence that in direct transmission of *Mycoplasma* spp. via contaminated feeders occurs **(Feberwee *et al.*, 2005).** Although *M.synoviae* can be transmitted via fomites, birds infected this way can quickly overcome mild disease and may on recovery be protected against more virulent infections acquired by direct bird-to-bird contact **(Behbahan *et al.*, 2005)**. Such indirect transmission is rather unexpected for wall-less bacteria, which are supposed to be sensitive to osmotic shock, heating or chemical treatments. However, *M. synoviae* may persist on feathers up to 2or 3 days at room temperature and its high dissemination capacity has been demonstrated **(Marois *et al.*,2005).** Mycoplasmas are more likely to spread among farms by the mechanical route, which includes spread via contaminated equipment, shoes and other fomites **(Kleven, 2003)**. *Mycoplasma synoviae* most frequently occurs as a subclinical upper respiratory infection. It may cause air sac lesions when combined with other respiratory agents such as Newcastle disease virus (NDV), infectious bronchitis virus (IBV), or both **(Landman, 2014)**. Other times, *M. synoviae* becomes systemic and results in infectious synovitis, an acute to chronic infectious disease of chickens and turkeys, primarily involving joint synovial membranes and tendon sheaths, and producing exudative synovitis, tenovaginitis, or bursitis **(Ferguson-Noel and Noor mohammadi, 2013)**. Infectious sinusitis grossly distends infraorbital sinuses, with fibrin, heterophils, epithelial cell hyperplasia, and hypertrophy of mucous glands. Later, there is lymphocytic infiltrates in the lamina propria or nodular formation, and tracheitis and airsacculitis can occur **(Kleven,2003).** The pathogen city of *M. synoviae* generally involves attachment and colonization of the respiratory tract, and other additional factors like imunosupression can produce systemic invasion and clinical signs.

Clinical signs and lesions

Common disease signs like pale comb, lameness and retarded growth are the first noticeable manifestations. Disease progression debilitates the bird that became ruffled, and swellings usually occur around joints, especially the hock and foot pads joints **(Ferguson-Noel and Noor mohammadi, 2013)**. Air sacullites may occur in chickens infected via respiratory tract at any age. In recent years, the occurrence of arthropathic and amyloidogenic strains of *M. synoviae*, as well as strains that induce eggshell apex abnormalities and egg production losses, has increased **(Feberwee *et al.*,2008)**. The progeny of *M. synoviae* infected breeders may have increased condemnation, poor conversion rates and poor weight gain. Morbidity varies from2 to 75%, usually reaching 5 to 15%, and mortality ranges between 1 and 10%. As *M. synoviae* infection progresses, caseous exudates involve tendon sheats and joints that became thinned over time and may evolve in to the muscle and air sacs. In the respiratory form, airsacculitis may be seen **(Kleven, 2003)**.

Diagnosis, control, treatment and prevention

Diagnosis is based on epidemiological data, clinical signs, and analysis of macroscopic lesions, specific serology, isolation and molecular characterization of *M.synoviae*. Monitoring must be part of control programs performed in breeder flocks and is mostly feasible by routine serology and PCR **(Kleven, 2003)**. Serologic procedures are useful for flock monitoring in *M. synoviae* control programs and to aid in diagnosis when infection is suspected. A positive serologic test, together with history and signs typical of the disease, allows a presumptive diagnosis pending isolation and identification of the organisms **(Fergunson-Noel *et al.*, 2011)**. The tube agglutination

test was a common procedure, especially during the *M. gallisepticum* control program for turkeys in the 1960s and 70s but is now rarely used. Serum plate agglutination (SPA) antigen for the detection of antibodies to *M. synoviae* is commercially available. Because the SPA test is quick, relatively inexpensive and sensitive, it has been widely used as an initial screening test for flock monitoring and sero-diagnosis. However, nonspecific reactors occur in some flocks infected with *M. synoviae* due to cross-reactive antigens, or those recently vaccinated with oil-emulsion vaccines and/or vaccines of tissue-culture origin against various agents. The SPA test is highly efficient in detecting IgM antibodies, which are the first class of immunoglobulins produced in response to infection **(Kleven,2003)**. The hemagglutination inhibition (HI) test has been commonly used to confirm reactors detected by SPA or, more recently, enzyme-linked immunosorbent assays (ELISA). However, the HI test is time consuming, the reagents are not commercially available and the test may lack adequate sensitivity. ELISA assays were developed to increase testing efficiency and improve sensitivity and specificity of results compared to the SPA and HI tests. Commercial ELISA test kits are now commonly used for serodiagnosis and flock monitoring. In general, ELISA tests are slightly less sensitive but more specific than SPA tests; and less specific but more sensitive than HI tests **(Kleven, 2003; Ferguson-Noel and Noor mohammadi, 2013)**. **Ewing *et al.* (1998)** reported that the SPA test missed infected commercial layer and breeder flocks that were detected by ELISA. Further confirmation of serologic results may be made by isolation and identification of *M. synoviae* from the upper respiratory tract or by PCR) **(Carli and Eyigor, 2002; Ramírez *et al.*, 2006)**. However, few laboratories are equipped for culturing this organism, as specific culture media are required. Techniques for the detection and analysis of DNA through PCR arise as a very interesting alternative diagnostic method, because they offer sensitivity, specificity, capability of performing exams on a large scale and economic viability nowadays **(Hammond *et al.*, 2009).** The sensitivity observed in PCR is important for detection of pathogenic agents in clinical samples taken from subclinically infected animals or those undergoing antibiotics treatment. Furthermore, it is possible to detect a pathogenic agent even before the host's immunologic response, or in hosts with immune depression, which points out advantages over the serologic tests **(Buim *et al.*, 2009; Kempf, 1998)**. The antibiotic treatment of breeders is not effective for the elimination of *M. synoviae*, although egg transmission level is reduced **(Kleven, 2003)**. Macrolides like tylosin and tilmicosin and fluoroquinolones likeenrofloxacin and difloxacin are among the antibiotic families most widely used in poultry in many countries **(Gerchman *et al.*, 2011)**, but *M. synoviae* is susceptible *in vitro* to several other antibiotics including chlortetracycline, lincomycine, oxytetracicline, spectinomycine, tetracycline and tiamulin **(Ferguson-Noel and Noormohammadi, 2013)**. In the past, mycoplasmaseradication programs were based on antibiotic or heat treatment of fertile eggs, but more recently the intensive poultry industry relies heavily upon the application of vaccines for disease control **(Ferguson-Noel *et al.*,2012)**. Vaccination programs are presently being used to control outbreaks of the more virulent strains of *M.*synoviae **(Ferguson-Noel and Noormohammadi, 2013)**. Regarding the presence of mycoplasmas in breeder farms, their concentration in some regions and the inexistence of adequate sanitary barriers that may enable the isolation of farms are predisposing factors for the disease dissemination. Other contributing factors are related to the resistance to antimicrobial treatments and to the immunologic system escape mechanisms that these pathogens make use of. The high *M. synoviae* occurrence in layer and breeder birds is probably due to the fact that vaccines are still not oftenly used **(Kleven,1998; McAuliffe *et al.*, 2006).** The primary objective for any poultry farm *M. synoviae* control is to prevent the introduction of the organism into a clean flock by use of a comprehensive biosafety program.

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1/12/2019