Introduction

With a world-wide distribution, *Mycoplasma gallisepticum* (MG) is the greatest economically significant mycoplasma pathogen of avian species. As a highly transmissible avian pathogen, it is the causative agent of chronic respiratory disease (CRD) in chickens and infectious sinusitis in turkeys. The infection is globally distributed and is highly transmissible between birds of all ages. Birds infected remain carriers throughout their lifetimes posing threats to other bird populations. This article provides a brief description of *M. gallisepticum* infection, particularly in poultry.

**Keywords**: *Mycoplasma gallisepticum*; Avian; Respiratory diseases

Abstract

*Mycoplasma gallisepticum* (MG) is the primary etiologic agent of chronic respiratory disease (CRD) in chickens and infectious sinusitis in turkeys. The infection is globally distributed and is highly transmissible between birds of all ages. Birds infected remain carriers throughout their lifetimes posing threats to other bird populations. This article provides a brief description of *M. gallisepticum* infection, particularly in poultry.

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Introduction

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*Mycoplasma gallisepticum* is a pathogenic species belonging to the Class Mollicutes within the genus *Mycoplasma* of the family Mycoplasmataceae [6]. *M. gallisepticum* are the smallest self-replicating eu-bacteria that lack cell walls. *M. gallisepticum* are prokaryotes with reduced genome [7] minimal genetic information, many metabolic pathways and the capability of growing on artificial cell-free media [2,6,7]. These attributes are mirrored with a high degree of interdependence between *M. gallisepticum* and the host species as well as in the fastidious nature of the organism in vitro including its difficulty to culture, selective antibiotic sensitivity, inhibition of phagocytosis, and its intimate association with host cells. MG constitutes two copies of a 23S-5S rRNA gene cluster, both not found near the single 16S rRNA gene [8]. Differentiating from other avian mycoplasmas, *M. gallisepticum* was first classified by serotyping [9,10] and was entitled as serotype A [11]. Molecular techniques aided in distinguishing mycoplasmas with phenotypic and antigenic similarities to *M. gallisepticum* by 1993 and M. imitans was found to be closely similar [12].

*M. gallisepticum* convoluted with a respiratory virus infection such as infectious bronchitis (IB), Newcastle disease virus (NDV), Avian Influenza virus (AIV) and very often along with a secondary infection of *Escherichia coli* (E. coli) [13,14]; Haemophilus, or avian rhinotracheitis virus causes severe air-sacculitis, also known as “Air sac disease”, leading to aggravated clinical CRD, high morbidity, mortality and/or increased condemnations at processing [15-17].

*Mycoplasma gallisepticum* features a small genomic size comprised of only 996,442 bp nucleotides for R₃ strain [18] with limited biosynthetic capabilities. With the lack of cell wall, MG membrane proteins are crucial in establishing MG morphology, nutrient transport and colonization of the host [19,20]. As an opportunistic pathogen, MG depends on its parasitic lifestyle and despite several limitations, their degenerative evolution allow them to travel over inert surfaces, such as glass, plastic and eukaryotic cells, absentee of locomotory appendages such as flagella or pili [21]. Antigenic variation, phase variation, superantigens are a few
of the mechanisms adopted by MG to evade the host’s lymphatic system.

In chickens, *M. gallisepticum* is outlined by severe inflammation of the trachea, air sacs and lungs; conjunctivitis; rales; nasal and mucosal discharge. *M. gallisepticum* cytadheres to the tracheal epithelial cells mediating infiltration of macrophages, heterophils and lymphocytes to the tracheal submucosa. Yet, the molecular infectious mechanisms associated with the severe inflammatory response of MG is elusive [22,23]. Of an infinitesimal size and minimal genetic information, MG lack bacterial cell wall; and hence it is unaffected by β-lactam antimicrobials which target cell wall synthesis. *Mycoplasma gallisepticum* have the ability to penetrate cells; possess a trilaminar membrane and they are highly polimorphic. MG are facultative anaerobes with optimal temperature of 35-37°C and require an enriched media of 10-20% animal serum and yeast extract [13,24].

MG causes significant economic losses despite the absence of clinical signs. Condemnations of carcasses, reduced feed efficiency and egg production, reduced hatchability and growth, aggravating and co-existing with other disease agents and increase in medication or vaccinations, control programs prepare MG as the costliest disease facing poultry production globally [2,12,15,25].

MG outbreak persists in many countries around the globe and various measures have been enforced with outcomes demonstrated far from satisfactory. Extensive biosecurity and surveillance is a well control MG-program. In the United States, an extensive National Poultry Improvement Plan has been adopted by hatcheries and poultry breeders with success in increasing MG-free flock [26].

**References**