Up-To-Date on *Mycoplasma Hyopneumoniae* in Pigs: A Mini-Review

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Mini Review

Recent diagnostic research indicates that clinical respiratory disease in pigs is often polymicrobial, resulting from a combined infection with one or more viruses and bacteria [1]. One of the primary agents affecting the respiratory system in pigs is *Mycoplasma hyopneumoniae* (MHYO), the causative agent of enzootic pneumonia (EP) [2]. Infections with MHYO are highly prevalent worldwide and cause tremendous financial losses to the pig industry, mainly due to costs on treatment and vaccination, decreased performance, and increased mortality derived from secondary infections [3]. Its effect starts with the attachment and complete destruction of the mucociliary barrier and the hyperplasia of the epithelial cells in trachea, bronchi, and bronchioles [4]. The affected epithelium exfoliates both ciliated and non-ciliated epithelial cells, losing their intercellular connections, become rounded, exfoliate and then lysed, giving rise to a population of cellular organelles, such as mitochondria and cilia interspersed with mucus to form the exudate found within the lumen of the affected segment [5,6]. In addition, infection with MHYO is also commonly associated with immunostimulation and immunosuppression [7,8], non-specific mitogenic effects on swine lymphocytes [9], or cytokines secreted by activated macrophages [6,10], therefore, MHYO predisposes animals to concurrent infections with other respiratory pathogens [11].

The clinical disease is particularly high -but no exclusive, in animals of mid-finishing to slaughter age and the severity of clinical signs is defined by the strain of MHYO involved, infection pressure, the presence of secondary infections, and by management and environmental conditions [7]. When MHYO infection is not complicated by concomitant pathogens, the disease can take a subclinical course with mild clinical signs consisting of chronic, non-productive cough, reduced average daily weight gain, and reduced feed conversion efficiency [2].

When secondary pathogens are involved, clinical signs include labored breathing and pyrexia (morbidity ≈ 100%), and deaths may occur in around the 6% of the clinical cases [1,7].

Suggestive macroscopic lesions consist on purple-to-grey areas of lung consolidation and are mainly found bilaterally in the apical, cardiac, and antero-intermediate parts of the diaphragmatic lobes. Recovering post-mortem findings consist of interlobular scar retractions, and in case of a pure MHYO infection macroscopic lesions are resolved [12-14] weeks after infection [13,14]. Clinical signs and lesions can lead to a tentative diagnosis, but laboratory testing is necessary for a conclusive diagnosis [15]. Although culturing of the organism is described as the gold standard, it is not used for routine diagnosis, because of the isolation difficulties (i.e., specialized media, high costs associated with the technique, common overgrowth of other bacteria, and the low sensitivity of the method). The organism can be detected by immunofluorescence testing, having limited sensitivity [16]. Serology can be used to show presence of the organism at a herd-level [2] but is unsuited for diagnosis on individual animals [17]. At present, polymerase chain reaction testing is seen as the most sensitive tool to detect the infection at animal and herd-level [12,18,19].

Close contact between infected and susceptible pigs is the main route of MHYO transmission, because it is excreted from the respiratory tract of infected individuals through exhalation of microscopic droplets during coughing episodes and/or by nose-to-nose direct contact [20]. Piglets are considered free from this bacterium at birth, as *in utero* transmission has not been documented, and first exposure events occur during the lactation period, when piglets are in contact with dams shedding the microorganism [21,22]. In fact, the length of the lactation period has been suggested as one risk factor for piglet colonization with MHYO prior to weaning [23]. Dams and piglets in the breeding

herds are considered the reservoir of MHYO infections for the entire production system. Its circulation is thought to occur among existing sows and be transmitted to incoming gilts, which can maintain the pathogen within the farm and are responsible for most bacterial shedding to newborn pigs [21,24]. In this manner, the constant addition of gilts and birth of piglets provide critical susceptible populations needed to maintain pathogen transmission. On the other hand, infection with MHYO has a long duration, reaching up to 240 days [25], complicating the already slow disease transmission scenario observed in sow herds.

Due to its economic threat to the global swine industry and considering that the geographical distribution MHYO is assumed to be global [26], its consideration should follow substantial advancements in the quality, timeliness, and range of diagnostic and analytic tools available. Such approaches must be oriented to better understand which strains are present over time in different herds and production systems by the use of techniques such as molecular typing of isolates to analyze infection dynamics (establishment and monitoring of the extent of the disease), and the design of effective vaccines and vaccination strategies, without neglecting management strategies aimed at reducing herd-level load for the prevention of MHYO dissemination among susceptible animals.

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