Progression of Mycoplasma hyosynoviae infection in three pig herds. Development of tonsillar carrier state, arthritis and antibodies in serum and synovial fluid in pigs from birth to slaughter - DTU Orbit (06/12/2018)

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In this investigation, natural infection with Mycoplasma hyosynoviae was followed in groups of individual pigs in three different herds with regard to occurrence of tonsillar carrier state, clinical arthritis and development of antibodies in serum and in synovial fluid. Antibodies were detected by a polyclonal enzyme-linked immunosorbent assay (ELISA) developed for experimental use. The infection with M hyosynoviae progressed very differently in the three herds investigated. In one herd, the infection was apparently limited to adult pigs. In a second herd, all pigs became tonsillar carriers of M. hyosynoviae, but no mycoplasma-related arthritis nor any serological response was demonstrated within the growing-finishing period. In the third herd investigated, tonsillar infection was detected in all pigs, clinical cases of M. hyosynoviae arthritis followed and a moderate serological response was observed in some, but not all, pigs. In all three herds, M. hyosynoviae infection was carried in the tonsils of the adult pigs, but it was only occasionally transmitted from sows to piglets. Maternal antibodies were transferred to the piglets and persisted for approximately 8-12 weeks. After weaning, some pigs became infected before 20 weeks of age, while others did not. In the majority of cases, the tonsillar infection was established from 11 weeks of age or older. A latent tonsillar infection was present for a period of several weeks within the group of investigated pigs before cases of generalized infection and arthritis were seen. In some cases, generalization of M. hyosynoviae infection in the blood and in joints was observed in spite of the detection of an active serological response a few weeks earlier. The present work suggests that generalization of the infection and development of arthritis may depend on age, immunity virulence factors and/or infection pressure; in some herds maybe combined with certain triggering mechanisms such as stress and lowered general resistance.