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Pathogenesis

The pathogenesis of Bordetellosis is somewhat complicated as it depends upon a host of factors including adhesins, toxins and other substances produced by the strain involved. This is because these interfere with or facilitate host animal functions such as adhesion, immune functions or survival properties of the bacterium.

Sometimes environmental factors, such as temperature (rising from environmental to body temperature), can trigger off one of these factors (virulence gene).

In the initial stages of infection *B. bronchiseptica* attaches to the nasal mucosa and when the bacteria are established in the respiratory tract they release toxins which contribute to the disease process. In atrophic rhinitis one of these toxins impairs bone formation.

The pathogenesis of disease can be modified by animal age and immune status. For example, non-immune young pigs usually develop a more severe disease, whether bronchopneumonia or atrophic rhinitis. Co-infection with other porcine pathogens also influences the severity of disease. These include *Pasteurella multocida*, *Streptococcus suis* and *Haemophilus parasuis*.

Co-infection with viruses can influence disease manifestation. For example, co-infection with PRRS virus makes bronchopneumonia a more likely occurrence and infection with both *B. bronchiseptica* and PRRS virus favours pulmonary infection by *P. multocida*. Co-infection with *B. bronchiseptica* and swine influenza virus or porcine respiratory corona virus leads to a more severe pneumonia.

Clinical signs

Variation in clinical signs is seen depending on age, immune status and co-infection with other porcine pathogens.

In the uncomplicated disease the signs appear 2-3 days following infection and include sneezing, nasal discharge, dry coughing and eye discharge. Signs can be more severe in young pigs. Although respiratory signs usually abate after a few weeks, the respiratory tract can remain colonised with *B. bronchiseptica* for months.

Piglets/pigs protected by maternal immunity can have their respiratory tracts colonised with *B. bronchiseptica* in the absence of clinical signs.

Co-infections often result in more severe clinical signs.

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