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88 – PRRS III

Pathogenesis of PRRS infection

Following exposure to the PRRS virus, viral replication is mainly in local permissive macrophages and then the virus rapidly spreads to mainly the lymphoid organs and lungs. Viral loads peak in the blood and lungs 7-14 days post infection and the highest numbers of virus are seen in the lungs. Clinical disease and lesions correspond to the time and organs with the highest PRRS virus titers.

After peaking, PRRS virus titers in serum decline rapidly and most pigs are no longer viraemic after the fourth week post infection. Viraemia can persist longer in congenitally infected pigs.

Following viraemia, congenitally and postnatally infected pigs are persistently infected with virus in their tonsils and/or lymph nodes.

Clinical signs

The clinical manifestation of PRRS varies from being asymptomatic to a disaster! The outcome is governed by the variant of PRRS virus involved, the host's immune status, host susceptibility, exposure to lipopolysaccharides, concurrent infections and management factors.

The clinical picture seen is a consequence of viraemia and reproductive failure in females.

Clinical epidemics occur when PRRS virus enters an immunologically naïve unit and all ages are affected at the same time. Endemic PRRS occurs in herds that have homologous immunity to the infecting PRRS virus. In endemic PRRS the disease is encountered in susceptible subpopulations, such as nursery/grower pigs with declining immunity, or gilts and sows, which have no immunity, and their congenitally infected progeny.

As antigenic variation within PRRS can be great, the entry of a new, relatively unrelated virus can cause an epidemic in an endemically infected herd. This was seen in China about 10 years ago when a highly virulent new strain of the Type 2 virus rapidly spread through the east of the country.

Epidemic PRRS

In the first phase anorexia and lethargy are seen in 5-7% of pigs of all ages. The second phase, which can commence before the completion of the first phase, usually lasts for up to four months and is characterised by reproductive failure, especially in those sows which are in their third trimester, and high pre-weaning mortality,

When reproduction and pre-weaning problems have returned to normal, endemic infection normally persists.

Factors affecting the severity of PRRS

PRRS viruses differ genetically in terms of the severity and lesions produced in respiratory disease. PRRS appear to differ between pig breeds.

Bacterial lipopolysaccharide is a major component of bacterial cell walls and found in high levels in the dust in poorly ventilated houses. If pigs inhale such dust, a more severe clinical respiratory disease picture is seen.

Infection with PRRS virus makes pigs more susceptible to various bacterial and viral diseases. These include infections caused by Streptococcus suis, Bordetella bronchiseptica, Salmonella cholera suis and PCV-2.