Porcine Reproductive and Respiratory Syndrome (PRRS)

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PATHOGENESIS

The target cells in which PRRSV replicates are subsets of macrophages that have specific glycoprotein receptors for the virus. These include pulmonary alveolar and interstitial macrophages and macrophages in lymphoid tissues. In order for replication to take place maturity and/or activation are required. The pathogenic effects of PRRSV consist mainly of pyrexia and inflammation, the latter particularly in the lung, and immune suppression, which predisposes to secondary infections.

The effects of infection with PRRSV are the result of various processes induced by the virus. There is apoptosis of infected cells and also of non-infected cells, mostly if not exclusively macrophages, in proximity to infected cells (also known as bystander or indirect apoptosis). Inflammatory cytokines are released by activated macrophages that cause increased vascular permeability and leucocytosis. Effects in the lung include oedema and inflammation that can lead to constriction of the bronchioles, while systemic effects include pyrexia that results in anorexia, reduced activity and flushing to purple discolouration of the extremities. Piglets die as a result of severe pneumonia or high fever and dehydration. Transplacental infection of foetuses can lead to foetal death and resorption or abortion.

The mechanisms that cause immune suppression have not been fully elucidated. Response to viral infection is reduced by the down-regulation of interferon (IF-α), which has been demonstrated to occur with PRRSV infection. The induction of an interleukin, IL-10, by PRRSV is considered to be of importance as it interacts with a wide range of cell types including monocyte-macrophages and lymphocytes to produce immunosuppressive effects. Replication of PRRSV in macrophages in lymphoid organs has
been demonstrated to activate polyclonal B cell activity, resulting in lymphoid hyperplasia and often the circulation and deposition of immune complexes. Non-neutralising antibodies produced by these cells may enhance viral infectivity (antibody-dependent enhancement). Destruction of the macrophages in which the virus replicates reduces the defences against bacterial infection as these cells are responsible for the phagocytosis and killing of bacteria.