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Introduction

Towards the end of the 1980s severe outbreaks of a new disease were seen in the USA. These were characterised by severe reproductive losses, respiratory disease, depressed growth rate and increased mortality. In 1990 similar outbreaks of disease were seen in Germany and then across Europe over the next four years. The same disease appeared in Japan in 1988 and Taiwan in 1991.

The aetiology of the disease was established in 1991 when Koch's postulates were fulfilled by a RNA virus. At about the same time European researchers named this new disease 'porcine reproductive and respiratory syndrome' which is now popularly referred to as PRRS.

PRRS is estimated to have cost the US pig industry \$560 million in 2005 rising to \$670 million a few years later.

The virus

The causal virus of PRRS is a close relative of the equine arteritis virus and belongs to the viral family Arteriviridae, which is closely related to Coronaviridae or the coronaviruses. There are two major lines of PRRS virus – Type 1 and 2. Type 1 predominates in Europe, while Type 2 is more common in Asia and North America. Within each line there is a lot of genetic diversity.

It is thought that Type 1 PRRS was present in the old Soviet bloc countries long before it appeared in Europe. Of the nine lines of Type 2 PRRS virus known by 2010, seven were predominantly in North America and two were exclusive to east Asia.

Epidemiology

PRRS virus can be found in most pig producing areas. Quite a few animal species have been shown not to be susceptible to PRRS virus infection. Wild pigs are susceptible to PRRS virus infection although sero-surveys of such animals show it to be relatively rare.

Infected pigs shed PRRS virus in their saliva, nasal secretions, urine, semen and, occasionally faeces. Obviously the shedding of this virus via semen is of serious concern due to the widespread use of AI.

PRRS virus produces a persistent chronic infection in pigs and this fact is very important when considering the epidemiology of this disease.

Pigs can be infected via the intranasal, intramuscular, oral, intrauterine and vaginal routes. In the field infection occurs through broken skin (ear notching, tail docking, teeth clipping, tattooing and injections) and via abrasions and bites (tail and ear biting). Contaminated fomites can also transmit this disease. The disease can be wind borne.

In viraemic gilts and sows, transplacental transmission to foetuses occurs.

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