In the late 1980’s, outbreaks of a previously unrecognized disease of swine were reported in the United States. Similar reports emerged from Europe in 1990.

Numerous causes for the outbreaks were investigated including encephalomyocarditis virus (EMC virus), classical swine fever (hog cholera), porcine parvovirus, *Leptospira interrogans serovar bratislava*, and mycotoxins.

A new RNA virus, designated Lelystad Virus (LV) in Europe and Porcine Reproductive and Respiratory Syndrome virus (PRRS) in the U.S., was identified as the cause of the outbreaks in 1991.

Evidence collected by evaluating stored blood samples showed that the virus was present in pig populations as early as 1979.

No one has conclusively determined where the source of the virus although some have speculated it may have “jumped” from another species.

It appears that the advent of larger herd sizes and increased movement of swine and swine semen during the 1980’s and 1990’s facilitated the spread of the virus within and between countries.
Historical Overview of PRRS Virus

J Zimmerman

Introduction

In the late 1980's, catastrophic clinical outbreaks of a previously unrecognized disease were reported in the United States (Keffaber, 1989; Loula, 1991). First described in herds in North Carolina, the syndrome included severe reproductive losses, extensive post-weaning pneumonia, reduction of growth performance, and increased mortality (Hill, 1990). In the absence of a recognized cause, the name "Mystery Swine Disease" (MSD) became common usage (Hill, 1990; Reotutar, 1989).

Initially, a variety of etiologies for MSD were proposed, including encephalomyocarditis virus, classical swine fever (hog cholera) virus, porcine enterovirus, porcine parvovirus, pseudorabies virus (Aujeszky's disease), *Leptospira interrogans* serovar *bratislava*, *Chlamydia psittaci*, and contamination of feed with mycotoxins (Bane et al., 1990; Daniels, 1990; Hoeffling, 1990; Joo, 1988; Joo et al., 1990; Quaife, 1989; Reotutar, 1989). In Canada, a new subtype of Influenza A virus was isolated from piglets suffering severe respiratory disease and added to the list as a possible agent of MSD (Dea et al., 1992c; Elazhary et al., 1991). Identifying the etiology was complicated by the fact that one or more of the suspected pathogens, as well as other infectious agents, were commonly isolated from cases of MSD.

In Europe, clinical outbreaks notably similar to MSD were reported in November 1990 near Münster, Germany (OIE, 1992). The disease spread rapidly and over 3,000 outbreaks were documented in Germany in May 1991. No link was found between outbreaks in Germany and MSD in the U.S. (Anon, 1991). The disease was reported in the Netherlands in January 1991 (Baron et al., 1992; OIE, 1992), followed by outbreaks in Denmark in March 1992 (Bøtner et al., 1994). The disease was confirmed to be present in Poland in 1992 (Pejsak and Markowska-Daniel, 1996), and the Czech Republic in 1995 (Valíček et al., 1997).

In Asia, outbreaks occurred in Japan in 1988 (Hirose et al., 1995) and in Taiwan in 1991 (Chang et al., 1993). Thus, the pandemic had spread to most of the major swine producing centers of the world in the space of a few years.

Until 1991, the lack of a specific etiologic agent led to a rapid proliferation of colorful and descriptive terms for the disease based on clinical signs, none of which, in the absence of a defined cause, could be considered either inappropriate or incorrect. A partial list of names can be found in Table 1.

PRRS Virus

The cause of MSD was resolved in 1991 when Koch's postulates were fulfilled with a previously unrecognized, enveloped RNA virus (Terpstra et al., 1991a; Wensvoort et al., 1991). Shortly thereafter, the virus was isolated in the U.S. (Collins, 1991; Collins et al., 1992) and Canada (Dea et al., 1992a, 1992b). The first virus isolates in the Netherlands and U.S. were designated Lelystad virus and Swine Infertility and Respiratory Syndrome (SIRS) virus (BIAH-001), respectively. Both virus isolates were shown to induce reproductive failure and respiratory signs under experimental conditions (Collins et al., 1992; Terpstra et al., 1991a). The virus is now commonly referred to as Porcine Reproductive and Respiratory Syndrome (PRRS) virus in much of the world.

Early Evidence of PRRS Virus

The earliest direct evidence of PRRS virus infection in domestic swine comes from a retrospective serologic study of herds in Ontario, Canada. Carman et al. (1995) found that none of 50 herds sampled in
1978 were serologically positive for antibodies against PRRS virus by enzyme linked immunosorbent assay (ELISA) or indirect fluorescent antibody (IFA), but antibodies were detected in serum samples from 2 of 51 (3.9%) herds collected in 1979 and 8 of 51 (15.7%) herds sampled in 1980.

In the U.S., a retrospective survey found no evidence of infection in 1,425 serum samples collected from 118 Iowa swine herds in 1980 (Zimmerman et al., 1997). One of 26 herds (3.8%) sampled in 1985 was PRRS virus-infected and each successive year showed an increase in prevalence. The data from samples collected in 1985 suggests that the virus entered Iowa during or shortly prior to 1985. By 1988, 17 of 27 herds (63.0%) and 313 of 658 (47.6%) animals were seropositive. Similar to the Iowa data, the earliest evidence of infection in the state of Minnesota was found in banked serum samples originally collected in 1986 (Yoon et al., 1992).

Most of Europe followed the pattern seen in Canada and the U.S., prevalence increasing rapidly in 1988 and 1989. About 48 percent of 1,480 serum samples collected in eastern Germany prior to the outbreaks in northwestern Germany in 1990 were positive for PRRS virus antibodies.

In Asia, antibodies against PRRS virus were retrospectively documented in serum from pigs imported into the Republic of Korea (South Korea) in October 1985 (Shin et al., 1993), in serum samples collected in 1987 in Taiwan (see Section 6.17), and in samples collected in June 1988 in Japan (Hirose et al., 1995). Again, well before the recognition of clinical disease.

Molecular studies of PRRS virus isolates provide indirect evidence of the early presence of the virus. Based on a study by Forsberg et al. (2001), PRRS virus isolates from Denmark, Italy, the United Kingdom, and the Lelystad virus were linked to a common ancestor that existed about 1979, i.e., more than 10 years prior to the outbreaks in Europe.

Thus, the serological and virological evidence indicate that PRRS virus was circulating in the domestic swine population by 1979 in North American and perhaps Europe, as well, i.e., several years prior to the actual recognition of clinical disease: This raises several questions, but two in particular: Where was PRRS virus before 1979? and Why were clinical signs not reported prior to 1987?

Regarding the first question, Forsberg et al. (2000) suggest two possibilities:

1. The virus came from another species.
2. The virus circulated in an isolated pig population for a "long time" prior to the pandemic, during which time it acquired a high degree of genetic diversity.

There is no direct evidence to fully support or repudiate either of these hypotheses, but further discussion is warranted because of the consequences PRRS virus has had on swine health and because there is no reason to believe something similar could not happen in the future.

Forsberg et al. (2000) reasoned that, if PRRS had been the result of a single interspecies transmission event followed by explosive transmission in the swine population, i.e., a "point-source epidemic," then current virus isolates should be genetically

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### Table 1: Historical names for PRRS

<table>
<thead>
<tr>
<th>Name</th>
<th>Description</th>
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<tbody>
<tr>
<td>Abortus blauw</td>
<td>Porcine reproductive and respiratory syndrome (PRRS)</td>
</tr>
<tr>
<td>Blue ear disease</td>
<td>Blue-eared pig disease</td>
</tr>
<tr>
<td>Enfermedad misteriosa del cerdo</td>
<td>Rätselhafte schweinekrankheit</td>
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<tr>
<td>Epidemisch spätabort der sauen</td>
<td>Seuchenhafter spätabort der schweine</td>
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<tr>
<td>Lan er bing (Chinese)</td>
<td>Sindrome disgenésico y respiratorio del cerdo</td>
</tr>
<tr>
<td>Heko-Heko disease (Shimizu et al., 1994)</td>
<td>Sindrome misterioso del cerdo</td>
</tr>
<tr>
<td>Maladie blue du porc</td>
<td>Syndrom reproductif et respiratoire du porc</td>
</tr>
<tr>
<td>Maladie mystérieuse du porc</td>
<td>Swine infertility and respiratory syndrome (SIRS)</td>
</tr>
<tr>
<td>Mystery pig disease</td>
<td>Swine plague</td>
</tr>
<tr>
<td>Mystery swine disease (MSD)</td>
<td>Swine reproductive and respiratory syndrome</td>
</tr>
<tr>
<td>New pig disease</td>
<td>Syndrome dysgénésique et respiratoire du porc</td>
</tr>
<tr>
<td>Plague of 1988 - 1989</td>
<td>Syndrome HAAT (Hyperthermie-Anorexie-Avortement de la Truie)</td>
</tr>
<tr>
<td>Porcine epidemic abortion and respiratory syndrome (PEARS)</td>
<td>Wabash syndrome</td>
</tr>
</tbody>
</table>
linked to a common viral ancestor that emerged around the time of the outbreaks - which they are not. Instead, extensive genetic diversity already existed within Europe, within North America, and between Europe and North America at the time PRRS was first recognized.

In support of the hypothesis that PRRS virus circulated undetected in isolated swine populations is the observation that smaller herds tend to have fewer episodes of clinical PRRS. For example, the USDA (2002) reported that 15 percent of "smaller breeding herds," defined as sites fewer than 250 sows and gilts, reported clinical PRRS, versus 40 percent of "medium herds" (250 to 499 females) and 58 percent of "large herds" (500 or more females). These data are important because they link to the past. From the perspective of current production systems, the average breeding herd size for most of the 20th century was extremely small. According to the U.S. Census of Agriculture, the average total inventory among U.S. swine operations in 1959 was 37 animals (USDA, 1997). This increased to a mean of 81 per operation in 1969, 130 in 1978, 215 in 1987, and 301 in 1992 (USDA, 1997). Thus, if PRRS virus produces few clinical signs in small herds, then the virus could conceivably have circulated in the small herds that were the standard in the past without attracting excessive attention.

Of course, this still leaves the problem of identifying the original source of introduction. At present, we simply have insufficient data with which to resolve the question.

**Changes in Swine Production**

As suggested by Nelsen et al. (1999) and elsewhere, the dramatic changes in swine production and management that occurred in the latter half of the 20th century may have created an environment well-suited to the dissemination and perpetuation of the virus in the domestic swine population. Changes favorable to the virus included extensive horizontal integration resulting in fewer but larger herds, reliance on fewer and larger companies for replacement breeding stock, increased transport of live animals both within and between countries, and greater use of artificial insemination in breeding programs. It should be recognized that the changes that occurred in swine production and management in the last two decades have imposed an entirely new epidemiology on the infectious diseases of swine. In the case of PRRS virus, larger herds and increased movement of pigs and semen facilitated the spread of the virus within and between countries (Dewey et al., 2000; Millán Suazo et al., 1994; Plana Duran et al., 1992; OIE, 1994; Shin et al., 1993).

**Summary**

In the late 1980's, catastrophic clinical outbreaks of a previously unrecognized disease were reported in the United States, followed by outbreaks in Europe and Asia in the early 1990's. In 1991, European workers reported the cause to be a previously unrecognized Arterivirus and introduced the term "porcine reproductive and respiratory syndrome."

The earliest direct evidence of PRRS virus infection in domestic swine is the presence of anti-PRRS virus antibodies in serum samples collected in 1979 in Canada. Retrospective studies also found antibodies in samples collected in the U.S. in 1985, the Republic of Korea in 1985, in Japan in 1988, and in the former East Germany in 1987. Molecular studies of PRRS virus isolates suggest that the virus may have been present in Europe as early as 1979. The original source of the virus is unknown, but once introduced into domestic swine, the larger herds and increased movement of pigs and semen that became increasingly common in the 1980's and 1990's facilitated the spread of the virus both within and between countries.

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