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## Review article

# The “colorful” epidemiology of PRRS

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**Abstract** – The paper describes the specifics of the epidemiology of the Porcine Reproductive and Respiratory Syndrome (PRRS), that is its “behavior” as a communicable disease in porcine populations, and compares them to the general epidemiological characteristics of communicable diseases. This analysis shows that infection with the PRRS virus “behaves” epidemiologically both as an epidemic and as an endemic disease: on the one hand it can spread like an epidemic in naïve populations, and on the other it seems to linger on infinitely in an affected population with its clinical expression varying from farm to farm like an endemic disease. The paper tries to draw “epidemiological” conclusions on the general methods for controlling and/or eradicating the disease, and to identify areas of further research.

**pig / PRRS/ epidemiology / transmission / clinical expression**

**Résumé – Particularités de l'épidémiologie du syndrome dysgénésique et respiratoire porcin.** Cet article décrit les détails de l'épidémiologie du syndrome dysgénésique et respiratoire porcin (PRRS), c'est-à-dire son comportement en tant que maladie transmissible dans la population porcine, et les compare aux caractéristiques épidémiologiques générales des maladies transmissibles. Cette analyse montre que l'infection par le virus du PRRS se comporte, épidémiologiquement parlant, à la fois comme une maladie épidémique et endémique : d'un côté elle peut se répandre comme une épidémie dans les populations naïves, et d'un autre elle semble persister indéfiniment dans une population affectée, avec une expression clinique variant de ferme à ferme, comme une maladie endémique. L'article tente de tirer des conclusions « épidémiologiques » concernant les méthodes générales pour le contrôle et/ou l'éradication de la maladie, et d'identifier des domaines pour la recherche à venir.

**porc / syndrome dysgénésique et respiratoire porcin / épidémiologie / transmission / expression clinique**

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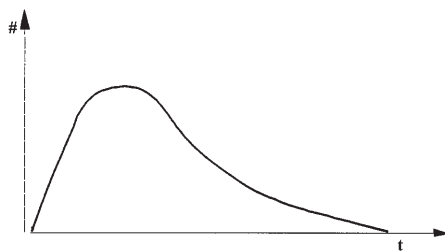
## 1. EPIDEMIOLOGICAL PATTERNS OF COMMUNICABLE DISEASES

Any communicable disease in humans and animals has its specific epidemiological pattern, i.e. its typical “behavior” (occurrence, disappearance, re-occurrence as well as distribution of outbreaks and cases in space and time). The epidemiological behavior is determined by [2, 3, 27]:

1. the biological properties of the causative agent such as pathogenicity, infectivity, contagiousness, and survivability outside the host;
2. the characteristics of the pathogen-host interactions such as host range, tissue tropism, mode of shedding and transmission, and immune response;
3. socio-economic conditions such as (in case of domestic swine) organizational structure of the swine industry, herd size, animal movement, production systems, etc.

There are two groups of communicable diseases: on the one hand the endemic diseases, the occurrence of which is unlimited in time, but limited in space, and on the other hand the epidemic diseases, the occurrence of which is unlimited in space, but limited in time.

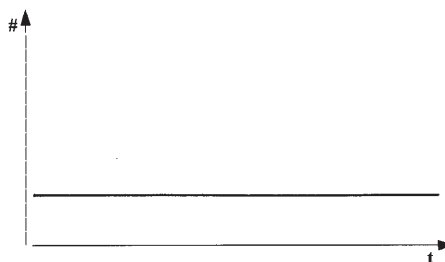
The latter are the classical epidemics, which often are even pandemic, that are caused by highly infectious and contagious pathogens with mostly a narrow host range such as the Bubonic Plague of the middle ages, the Vibronic Cholera, Polio and others in man, and the Classical Swine Fever, Food and Mouth Disease and Aujeszky's Disease in swine. These epidemic diseases have epidemiologically common features in their occurrence and distribution over time and space: in immunologically naïve populations, the outbreak of an epidemic can be described as a “high” epidemic wave (Fig. 1) with mainly acute diseases in all affected sub-populations (herds) and a fast spread.



**Figure 1.** The typical distribution of cases of epidemic communicable diseases in susceptible populations throughout space and time.

The longer the disease is prevalent in the affected population, however, the more the epidemic wave flattens out with decreasing incidence and more and more chronic and even sub-clinical diseases until it finally disappears, if a well-protecting population immunity has developed [3, 27].

The endemic diseases, in contrast, instead of an epidemic wave have a straight line (Fig. 2) more or less parallel to the time axis. The pathogens are not very pathogenic (often called “opportunistic”), the development of clinical disease is dependent on high infection doses (in populations it is called “infection pressure”) and on the supportive role of non-infectious factors that decrease the defense mechanisms of the host, which leads to remarkable variations in the sever-



**Figure 2.** The typical distribution of cases of endemic communicable diseases in susceptible populations throughout space and time.

ity of clinical expression from herd to herd [3, 27].

One striking difference between epidemic and endemic diseases is that the Koch’s postulates clearly “work” in cases of epidemic diseases (monocausal), where experimental infections lead to the typical clinical signs of the disease in question and there is a “yes” or “no” as to whether the investigated pathogen is the causative agent [27]. In cases of endemic diseases (multifactorial), it is mostly difficult if not impossible to reproduce the clinical signs that are observed in the field by experimental infections, since the various supportive factors that are necessary to express the disease cannot be simulated in the experiment. Therefore, Evans [13] suggested to use the following criteria (known now as “Evans’ postulates”) to determine, whether a suspected pathogen plays a causative role in an endemic disease:

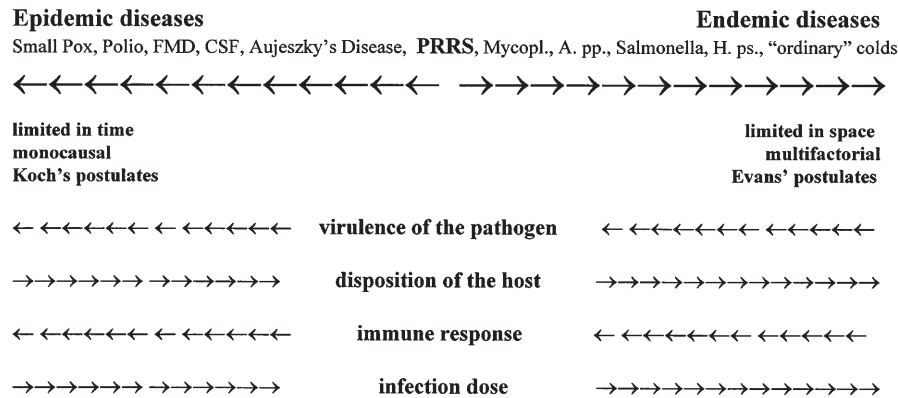
1. The proportion of individuals with the disease should be significantly higher in those exposed to the supposed cause than in those who are not.
2. Exposure to the supposed cause should be present more commonly in those with, than in those without the disease, when all other risk factors are held constant.
3. The number of new cases of disease should be significantly higher in those exposed to the supposed cause than in those not so exposed, as shown in prospective studies.
4. Temporally, the disease should follow exposure to the supposed cause with a distribution of incubation periods on a bell-shaped curve.
5. A spectrum of host responses, from mild to severe, should follow exposure to the supposed cause along a logical biological gradient.
6. A measurable host response (e.g. antibody, cancer cells) should appear regularly following exposure to the supposed cause in those lacking this response before exposure, or should increase in magnitude if present before exposure; this pattern should not occur in individuals not so exposed.
7. Experimental reproduction of the disease should occur with greater frequency in animals or man appropriately exposed to the supposed cause than in not so exposed; this exposure may be deliberate in volunteers, experimentally induced in the laboratory, or demonstrated in a controlled regulation of natural exposure.
8. Elimination (for example, removal of a specific infectious agent) or modification (for example, alteration of a deficient diet) of the supposed cause should decrease the frequency of occurrence of the disease.
9. Prevention or modification of the host’s response should decrease or eliminate the disease that normally occurs on exposure to the supposed cause.
10. All relationships and associations should be biologically and epidemiologically credible.

However, communicable diseases are not strictly either epidemic or endemic; there is rather a wide range from clearly epidemic to clearly endemic with most diseases being somewhere in between. Figures 3 and 4 show some selected determinants for and consequences of the epidemiological pattern of any communicable disease.

## 2. THE EPIDEMIOLOGICAL PATTERN OF PRRS

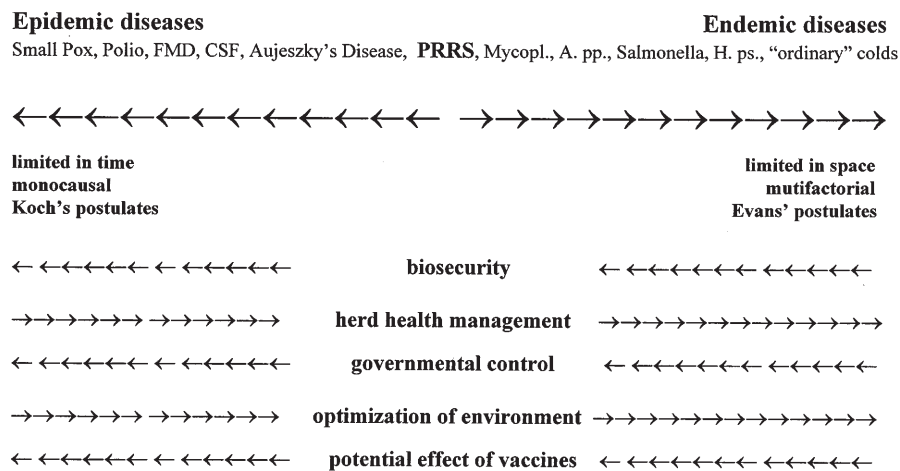
Whereas most diseases can be positioned as an entity somewhere on the scale of epidemiological pattern in Figures 3 and 4, the Porcine Reproductive and Respiratory Syndrome (PRRS) does not fit into the scale as an entity - only individual criteria such as virulence of the pathogen, immune response, etc. can be positioned at certain points on

## Epidemiological Patterns of Communicable Diseases (Determinants)



**Figure 3.** The factors that determine the position of a communicable disease on a scale of epidemiologic patterns between clearly epidemic and clearly endemic diseases (← or → = direction of increasing importance).

## Epidemiological Patterns of Communicable Diseases (Consequences)



**Figure 4.** The consequences of the position of a communicable disease on a scale of epidemiologic patterns between clearly epidemic and clearly endemic diseases (← or → = direction of increasing importance).

the scale, with every criterion having a different position. In other words, PRRS has characteristics that are clearly epidemic, others that are clearly endemic and most of its characteristics somewhere in between. Even within one and the same host, the different diseases at the two different sites of expression (the reproductive tract and the lung), the infection with the PRRS virus shows a different epidemiologic pattern: the reproductive disease seems to have more characteristics of an epidemic developing a quite good protective immunity (herds with an outbreak of the reproductive form mostly return to normal performance within some months [10]), whereas the respiratory disease seems to have more of the characteristics of an endemic disease with a weak immune response and a greatly varying severity of clinical symptoms [12].

Although the PRRS virus is highly contagious, and has only the pig as a reservoir and epidemiologically important host species [16, 28, 30], the epidemiological features of PRRS seem to differ from the expected patterns of a classical epidemic infectious disease in three ways:

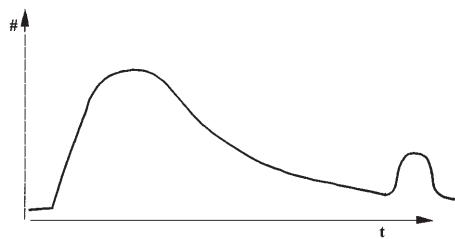
1. There is evidence that the PRRS virus has entered the domestic swine population obviously several years before the clinical disease became visible and economically devastating. Several swine sera collected and stored in the mid-1980s from Canada, Korea, Japan and East Germany, investigated for PRRS antibodies in the 1990s, tested positive [9, 10, 15, 22, 26].

However, no dramatic outbreaks and no epidemic waves were known before the catastrophic clinical outbreaks of the “mystery disease” in the late 1980s across the USA [18, 20], although the populations, which were hit in the early 1980s by the virus, must also have been immunologically naïve prior to their encounter with the virus. The European appearance of the disease with clinical outbreaks compatible with the mystery

disease of North America fulfilled much more the expectations of a first occurrence of an epidemic disease in naïve populations: the first outbreak of the disease in November 1990 in the Northwest of Germany triggered an abortion “storm” throughout Germany and soon after, through the Netherlands to Belgium, France and Spain [1, 4, 6, 11, 14, 24, 28].

On its way through Europe, however, the virus seemed to have lost its high virulence, since the disease was often mild, if not in-apparent in European areas (Austria, Czech Republic, East Germany and Denmark) that were affected one or two years after the dramatic onset in Western Europe [6, 7, 19, 23], although previously their swine populations must have been naïve as well.

2. In contrast to the described PRRS phenomenon of latent infection or at least mild disease in naïve populations prior to epidemic waves, there is the opposite PRRS phenomenon of sudden onsets of highly acute disease in more or less immune populations. The severe outbreaks of reproductive failure with sow and boar mortality greater than 5% and abortion rates greater than 10% in the USA in late 1996 and 1997 occurred exclusively in endemically infected herds [17, 25, 31]. This fact led naturally to the assumption of a new causative agent. Intensive investigation by the USDA Emergency Response Team did not, however, reveal any evidence of a new infectious agent. The findings of a USDA case-control study [31] to evaluate the importance of various factors for the development of the disease suggest factors associated with the affected herds rather than the emergence of a highly “dangerous” variant of the PRRS virus, since the relatively few acute PRRS outbreaks occurred in rather diffuse geographical locations without obvious swine relations. Figure 5 shows the somewhat unique epidemic curve of PRRS



**Figure 5.** The epidemiological curve of PRRS.

with a low and flat part before the epidemic wave (clinically in-apparent occurrence of the virus) and a peak at the already flattened part after the epidemic wave (reoccurrence of severe clinical cases).

3. Whereas in well-known "classical" epidemics, the degree of the clinical expression of the infection is comparable in all herds that get affected at the same time (first acute then gradually milder and milder), the PRRS disease pattern can vary greatly between herds that are infected at the same time: both mild clinical expressions on certain farms during acute outbreaks and severe disease on certain farms in endemically infected areas have been reported [5, 10, 11, 21]. This phenomenon seems to apply to vaccinated and non-vaccinated herds. The conclusion is that farm and management associated factors play a very important role for how severe the disease will be following the introduction of the virus into a herd, which supports the above mentioned reasoning about acute PRRS [8, 29, 31].

There are still inconsistencies (better: gaps of knowledge) in the PRRS epidemiology that need further research and exchange of experiences. The most striking features are:

1. The observations of many a practitioner about the aggravation of bacterial respiratory disease in growing pigs due to the

infection with the PRRS virus, theoretically supported by the affinity of the virus to the lung macrophage, has not yet been efficiently proven by experimental infections.

2. The assumption of an easy airborne transmission of the virus, mostly concluded from the storm-like spread of the disease through Europe immediately after the first European outbreak, has also not yet been proven by experiments. Furthermore, the easy airborne transmission is not supported by the fact that in chronically infected sow herds, sows with and without antibodies and active virus are often neighbors over certain periods of time without an infection of the negative animals.
3. The importance of organizational and management factors (pig flow, consistency of all-in/all-out, age-group segregation, external and internal biosecurity, etc.) is generally recognized, but there is no list of best management practices that, if implemented at the farm level, could guarantee the adverse effects of an infection with the PRRS virus.

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