PRRS virus area spread and aerobiology

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Introduction

PRRS continues to impose significant costs on producers, for prevention and control regimens, and in reduced productivity. As reported by participants in the USDA Swine 2000 survey, PRRS was the second most frequent disease problem in both breeding herds and nursery-age pigs (USDA, 2002).

Elimination of PRRS virus improves health and productivity, but can only be considered a temporary solution because, despite ever-tighter biosecurity measures, re-introduction of PRRS virus is a near certainty in swine-dense areas.

Until we fully understand the mechanism(s) of transmission and are able to provide the means to protect PRRS virus-negative herds, re-infection will continue to be the major roadblock to the elimination of PRRS virus on a broad scale.

Defining the problem

For a variety of reasons, most of which have to do with funding priorities, research in PRRS virus transmission has lagged behind research in other areas. Field data has been used to compensate for funding deficiencies, but it has inherent deficiencies:

- Retrospective field data cannot prove a cause and effect relationship—although the attempt is often made. Classically, clinical outbreaks occur too long after the introduction of the virus into a herd to establish the route of entry with any certainty. Under pressure to explain events, hypotheses are offered that promptly metamorphose into fact.
- A lack of rigor in the collection and analysis of field data has sometimes led to dubious conclusions. For example, early in the pandemic, the distance between outbreaks was measured as the distance the virus traveled and subclinically infected herds located between outbreaks were ignored. As a result, early investigators postulated that PRRS virus was “capable of spread in air over at least 3km from initial foci and perhaps, on occasion, as far as 20km” (Anon, 1992).

Recently, following the latest in a long series of unexplained introductions of PRRS virus, attention has returned to area spread. A number of potential means of transmission are currently under investigation (alternate hosts, arthropod, aerosols). The purpose of this paper is to review current knowledge regarding aerosol transmission and discuss experimental approaches to the problem.

Aerobiology

Aerobiology is the study of airborne particles, including infectious microorganisms. Aerosol transmission is frequently cited as the route of spread of PRRS virus and a variety of other pathogens, but such statements invariably lack the benefit of validation with hard data. The fundamental premise of this discussion is that aerosol transmission is a biomechanical process that can be understood through the process of experimentation. As in any problem-solving exercise, the correct approach is to dismantle the problem into its smallest parts, then solve each part independently. At a minimum, the parts we need to understand in relation to aerosol transmission are the following:

1. (Number of organisms aerosolized by an infected pig) (Unit of time) over the period of shedding.
2. The rate of inactivation of airborne organisms under specific environmental conditions: temperature, light, relative humidity, dust, etc.
3. The rate of dispersion of infectious particulates and direction of air flow. Dispersion is a function of gravity and atmospheric stability. Higher ground-level concentrations occur in stable atmospheres because less mixing with surrounding air occurs. Wind speed, temperature, relative humidity, and solar insolation are factors in atmospheric stability.
4. The number of viable organisms required to infect the next susceptible pig (minimum infectious dose).

All of the above are quantifiable variables for which estimates can be obtained either in the field or in a research setting. For PRRS virus, it should be recognized that we are missing all of parts 1 and 2 above. In addition, for
part 1 we have no information as to whether concurrent respiratory tract infections affect the quantity of aerosolized PRRS virus. Part 3 is a function of weather and particle size. Parameters affecting part 3 will vary between localities and over time, but estimates of the concentration of virions at any point \((x, y)\) on a grid can be made using the Gaussian plume model. Yoon et al. (1999) have provided acceptable estimates for the minimum infectious dose requirement in part 4.

This information is powerful because it becomes possible to make predictions. For example, in March 1981, the French reported outbreaks of foot-and-mouth disease (FMD) on 13 swine farms in Brittany and one in Normandy (Donaldson et al., 1981). Based on the meteorological conditions (relative humidity, windspeed, temperature, cloudy conditions, etc.) and equations developed to model the behavior of FMD virus, it was correctly predicted that the virus would be carried 300 kilometers to the south coast of England. Forewarned is forearmed, and FMDV infections in cattle in Jersey and on the Isle of Wight were detected and disaster averted. Unfortunately, FMD virus is one of the few agents for which we have good aerobiological data.

The parts of the process

Many people view infectious microorganisms in aerosols as simple and static. A pig sneezes and a cloud of infectious particles wafts over the countryside. This is far from the truth. An overview of the process may provide an appreciation of the dynamic complexity of the process.

Gravity

As small as they are, droplets are still subject to gravity. In fact, this is one of the difficulties in studying aerosolized clouds of microorganisms in the research setting—the droplets fall to the ground.

In humans, exhalation droplets range in size from 15 to 100 microns in diameter—so most sediment quickly and do not travel far. A sneeze or cough creates a population of mixed droplet sizes. In humans, most sneeze-generated particles are one micron or less in diameter. Particles 100 microns in diameter fall at approximately 0.9 feet per second; 20 micron particles at about 2.25 feet per minute; 10 micron diameter particles at about 0.5 feet per minute; 1 to 3 micron particles will remain suspended nearly indefinitely.

Suspension medium

Pathogens generated from the respiratory tract are hurled into the air suspended in a liquid globe composed of a complex medium (sputum). Under the effect of relative humidity and temperature, water is quickly removed and the droplet becomes a droplet nuclei. Human sputum is composed of:

<table>
<thead>
<tr>
<th>Component</th>
<th>Concentration</th>
</tr>
</thead>
<tbody>
<tr>
<td>mucin</td>
<td>2.0–3.0g per 100ml</td>
</tr>
<tr>
<td>protein</td>
<td>0.1–0.5g per 100ml</td>
</tr>
<tr>
<td>fats</td>
<td>0.3–0.5g per 100ml</td>
</tr>
<tr>
<td>sodium</td>
<td>123–207g per 100ml</td>
</tr>
<tr>
<td>potassium</td>
<td>7.8–18.6g per 100ml</td>
</tr>
<tr>
<td>chloride</td>
<td>102–222g per 100ml</td>
</tr>
<tr>
<td>calcium</td>
<td>2.1–4.1g per 100ml</td>
</tr>
<tr>
<td>phosphorus</td>
<td>25.4–28.6g per 100ml</td>
</tr>
</tbody>
</table>

Droplet composition is important because proteins and mucin are highly protective of aerosolized viruses. For that reason, aerosols generated experimentally need to attempt to duplicate the natural suspension solution.

Relative humidity (RH) and temperature

RH and temperature heavily impact the survival of infectious aerosolized pathogens. A common misperception is that enveloped viruses remain infectious longer at higher relative humidities and unenveloped viruses persist better in lower humidities. This is not the case. To the contrary, enveloped viruses tend to survive better at mid-range relative humidity and unenveloped viruses at high relative humidity, although inactivation curves for viruses in aerosols tend to be specific for each individual virus species (Donaldson and Ferris, 1976).

For example, aerosolized FMD virus (a non-enveloped virus) survived best at RH above 60%. In contrast, PRV survived better at 55% humidity than 85%. Schoenbaum et al. (1990) found that PRV decayed logarithmically with mean half-lives of 17.4 minutes (85% relative humidity, 22°C), 36.1 minutes (55% relative humidity, 22°C), 27.3 minutes (85% relative humidity, 4°C), and 43.6 (55% relative humidity, 4°C) minutes.

The survival of PRRS virus aerosols under various conditions of RH and temperature has not been described. However, Donaldson and Ferris (1976) showed that equine arteritis virus, an arterivirus like PRRS virus, survived best at 30% RH or less when tested at 18 to 23°C.

Discussion

In the PRRS virus control program in France, the only well controlled, large-scale, field study to date, Le Potier et al. (1997) reported that 56% of herds acquired the infection through introduction of infected pigs, 20% through contaminated semen, 21% through fomites, and 3% through unidentified sources. Le Potier et al. (1997) found that 45% of herds suspected of becoming infected through area spread were located within 500 meters (0.3 miles) of the postulated source herd and only 2% were located at a distance of 1 kilometer.

In the research setting, the question of PRRS virus spread through aerosols remains unresolved. Using “whole animal” transmission protocols, investigators have not been
able to transmit PRRS virus beyond a distance of a couple of meters (Kristensen et al., 2002; Lager and Mengeling, 2000; Otake et al., 2002; Torremorell et al., 1997; Wills et al., 1997). Based on these experiments, some have concluded that aerosol transmission occurs and is important, whereas others, looking at similar data, have formed the opposite judgment. It would appear that a resolution of the question will require more precise research focus.

References


