Changes in Porcine Reproductive and Respiratory Syndrome (PRRS) virus incidence

Steve Tousignant, Andres Perez, Bob Morrison

Between 2009 and 2012, the frequency of PRRS virus breaks in sow herds in the United States had remained extremely stable, showing no significant differences in the cumulative incidence between these 4 years (p > 0.05). Additionally, the onset of the annual epidemic predictably occurred in the middle week of October. Depending on the year, herds that reported a PRRS outbreak in a previous year were 1.9 – 2.5 times as likely to report a PRRS outbreak in the current year (p < 0.05).

In the fall of 2013, the annual epidemic was signaled approximately 3 weeks later than the previous 4 years and the overall cumulative incidence was significantly lower than the previous four year average (p > 0.05). The epidemic for the current year appear to be similar to last year, with cumulative incidence about the same (PRRS report, chart 1) and the epidemic signal delayed again by about 3 weeks (PRRS report, chart 5). For the first time since 2009 when we started tracking these data, herds that reported a PRRS outbreak in the previous year (2012/13) did not have significantly higher odds of reporting an outbreak in 2013/14 (p > 0.05).

There are many possible explanations for these observations. Perhaps most prominently was the introduction of Porcine Epidemic Diarrhea (PED) virus into the US swine herd. Since then, many herds increased biosecurity in an effort to prevent entry of this new virus. Our data show that herds reporting a PRRS virus outbreak were 3.31 times as likely to report a PED virus outbreak (95% CI 1.94 – 5.94, p > 0.05). Some of the biosecurity measures implemented to prevent a PED virus outbreak in a herd may also have prevented PRRS outbreaks on some farms.

Awareness of the annual PRRS virus epidemic may also have changed biosecurity practices on some farms, leading to better preparedness before the epidemic season. Additionally, many farms are implementing the use of bio-aerosol filtration, which may not only reduce the frequency of airborne transmission of PRRS virus into herds, but also elevates and reinforces other biosecurity practices, training, and auditing. The combined effect of all of this may have played a role in the differences in the annual epidemics.

The number of herds opting to remain as ‘stable exposed’ (status 2v, PRRS chart 2) has increased significantly (p > 0.05) during the 2013/14 epidemic season. While it is currently not known if the herds in this category are using commercial vaccines or field virus inoculation to maintain immunity, it could be speculated there is more immunity in the US sow herd and that may have mitigated the severity of the epidemic.

Weather, especially the duration and intensity of cold temperatures, has also been hypothesized to have changed the epidemic patterns. While difficult to study, the dramatic and predictable seasonal pattern makes this seem plausible. These patterns also could be the result of a longer secular cycle over many years where there are 3 or 4 ‘bad years’ and then several ‘good years.’ Changes in PRRS virus diagnostics during PED virus outbreaks could have explained some of this, but this seems less plausible as PED virus stabilizes and the PRRS epidemics remain quiet.

It is important to understand that the participants of this project are voluntary, and our findings do not necessarily reflect all areas of swine production in the US. It is also important to understand, that no strictly formalized ‘case definition’ was used, and there may be some differences among systems. That said, the repeatability of these data among the participating systems is striking.