

Erysipelas: Potential involvement in urogenital disease of the sow

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Summary

Although systemic infections with *Erysipelothrix rhusiopathiae* in swine are usually associated with skin lesions, vegetative endocarditis, and arthritis, infections may cause abortion or influence stillbirth rate and litter size. In a large production unit that had ceased to vaccinate against erysipelas, an increased incidence of pre- and postparturient vulval discharge, longer farrowing intervals, and a reduction in live-born litter size were reported. Anterior vaginal swabs obtained from 21 sows shortly before parturition all yielded heavy growth of *E rhusiopathiae*. When a vaccination program was re-instituted, the inci-

dence of periparturient vulval discharge decreased, the farrowing interval diminished, and live-born litter size increased. In the absence of a control group, definitive conclusions cannot be made regarding the effect of vaccination against *E rhusiopathiae* on sow fertility. However, it is not unreasonable to suggest that *E rhusiopathiae* was involved in the etiology of the reduced sow fertility in this herd and that appropriate vaccination subsequently protected the sows.

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Swine erysipelas (SE), caused by infection with *Erysipelothrix rhusiopathiae*, occurs in most parts of the world and in most areas where domestic swine are produced.¹ The primary source of infection for swine is other swine.¹ The organism is believed to be transmitted directly via oronasal and fecal secretions or indirectly via environmental contamination.² Pigs may be infected by ingestion of contaminated feed or water or contamination of skin wounds.² In indoor production systems, contamination of concrete floors with the feces and urine of infected pigs is the likely source of infection.¹ Acute SE is characterized clinically by sudden death and pyrexia associated with diamond-shaped skin lesions.¹ In pregnant sows, acute or subacute SE has also been associated with abortion.^{1,3}

According to Wood,¹ the clinical signs of SE may be classified as acute, subacute, or chronic. In addition, subclinical infections, which cause no visible signs of acute dis-

ease, may progress to chronic SE,¹ which is characterized by proliferative lesions such as arthritis or vegetative endocarditis.³ A form of the chronic disease that is not well understood is a possible effect on the reproductive performance of the sow herd.⁴ In addition to abortion, SE may cause fetal mummification and other reproductive problems in the sow herd.³

Naturally acquired active immunity is induced by previous infection with *E rhusiopathiae*. Less clearly recognized is the immunity that may be induced by organisms of low virulence, which are capable of causing subclinical infections or mild, unnoticed subacute disease.

Preventive methods include optimizing biosecurity and sanitation in order to prevent direct infections.⁴ Attenuated vaccines and bacterins against *E rhusiopathiae* are commercially available.² Vaccination failures rarely occur but have been reported when the field strain was not contained in the bacterin.⁴ A serious flaw in the effec-

tiveness of SE vaccination is its inability to prevent the chronic form of SE. Most investigators agree that vaccination has little effect on the incidence of chronic SE, although this observation is difficult to evaluate in the field. It is possible that vaccination reduces the overall prevalence of any chronic manifestation of SE. On the other hand, some believe that vaccination actually causes an increase in chronic SE lesions by initiating a state of hypersensitivity to subsequent contact with the organism.¹ In our practice, we suspect chronic SE in herds where vegetative endocarditis, arthritis, and cultural presence of *E rhusiopathiae* in vulval discharge are accompanied by poor fertility and increased prevalence of abortions, stillbirths, and small litter size.^{3,4} This report describes the effect of vaccination against *E rhusiopathiae* on subsequent fertility in a sow herd in which there had been a marked increase in occurrence of purulent periparturient vulval discharge. There have been no previous published reports concerning development of SE in the urogenital system of the sow.

Case description

The affected large commercial unit had 1500 breeding sows with a history of SE. During the previous 12 months, the prevalence of purulent vulval discharge in prepartum sows had increased from 8 to 21%, occurring during the 1 to 2 days prior to parturition. In the same period, the prevalence of purulent vulval discharge in postparturient sows rose from 15 to 34%, with a duration of >5 days postpartum and a total volume of >100 mL in older sows. Record analysis showed that during that 12-month interval, compared to earlier years of production, the farrowing rate had decreased from 81 to 74%, and litter size had decreased from a mean of 11.0 (SE 0.91) to 10.2 (SE 0.76) live pigs per litter.

Anterior vaginal swabs obtained from 21 sows exhibiting prepartum vulval discharge were submitted for bacteriological culture.

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The results of these cultures revealed heavy growth of *E rhusiopathiae* and occasional *Escherichia coli*. Herd health records showed that vaccination against *E rhusiopathiae* had been discontinued 20 months previously.

An erysipelas vaccination program was re-instituted, with sows receiving a killed vaccine (Rhusilisat; Veterinaria, Zürich, Switzerland) at 40 and 55 days of gestation. It was necessary to administer the two doses of erysipelas vaccine in a 2-week interval because of other vaccines (for *E coli*, *Lep-tospira* serovars, and pseudorabies virus) given during gestation.

Twelve months after the re-institution of erysipelas vaccination, the monthly prevalence of both prepartum vulval discharge (mean 11.4%, SE 2.1%) and postparturient vulval discharge (mean 16.3%, SE 2.9%) had decreased compared to the 20-month period when the sows were unvaccinated ($P < .05$). Four months after vaccination, anterior vaginal swabs obtained prepartum from 17 of the 21 sows previously cultured revealed no growth of *E rhusiopathiae*.

To obtain a more objective measure of the efficacy of erysipelas vaccination, farrowing interval and litter size were examined for sows that farrowed in the pre-vaccination ($n=3511$) and post-vaccination periods ($n=3702$). Average parity of bred sows was 2.9 during the pre-vaccination period and 2.8 after vaccination had been reinstated. Data were analyzed by analysis of variance (Statistix; Analytical Software Inc, Tallahassee, Florida). The differences in production data between the 12-month pre-vaccination period and the 12-month post-vaccination period were evaluated by Student two-phase *t*-test assuming unequal variances. All management variables were evaluated with dependant variables (pre-vaccination, post-vaccination).

The farrowing interval (mean \pm SE) decreased from 159.1 ± 6.1 days in the pre-vaccination period to 146.1 ± 4.2 days in the post-vaccination period ($P=.04$). Live-born litter size increased from 10.2 ± 0.76 pre-vaccination to 11.7 ± 0.77 post-vaccination ($P=.03$). There was no difference ($P \geq .05$) in numbers of stillborn pigs (pre-vaccination, 1.3 ± 0.03 ; post-vaccination, 1.2 ± 0.03) or 21-day litter weights (pre-vaccination, $61.1 \text{ kg} \pm 2.1 \text{ kg}$; post-vaccination, $62.0 \text{ kg} \pm 1.9 \text{ kg}$).

Discussion

Infectious agents not commonly thought to be reproductive pathogens may cause failure of pregnancy either through their general systemic effects on the sow or by infecting the fetus.⁴ The possible effect of *E rhusiopathiae* in this case may have been caused by an ascending urogenital infection or by allergic response to chronic erysipelas infection. It has been suggested that *E rhusiopathiae* causes reproductive problems in the sow and may enter the uterus of the sow at mating or at parturition when the cervix is open.⁴

Vulval discharges are often associated with chronic endometritis or with swine urogenital disease.⁵ Some discharges are normal physiological events, while others – especially purulent discharges – are pathological and are particularly important, as herd fertility may be significantly affected.⁶ Postparturient discharges represent clearance of placental remnants and debris from the uterus.⁵ The volume of vulval discharge varies considerably, as does its texture, ranging from an opaque, mucoid material flecked with debris to purulent exudate.⁷ Recovery from postpartal uterine infections is influenced by many factors, including hormonal status of the sow, local immunity, and the possible influence of reduced leukocyte activity and delayed immune response of the postpartum uterus.⁴ Increased postpartum uterine immunoglobulin concentrations aid in clearing uterine infections.⁴ Approximately 80% of IgG and 60% of IgA in the uterus of the sow are serum derived,⁸ underlining the importance of vaccination status of the sow against *E rhusiopathiae* infection. There is a tendency for IgA levels to be higher in the cervix and vagina than in the oviduct or uterus.⁸ However, in response to elevated pre-farrowing serum estradiol concentrations (that rapidly decrease after parturition), there are profound increases in the total numbers of circulating leucocytes (both lymphocytes and neutrophils), the percentage of immunoglobulin-bearing mononuclear cells, and the phagocytic capacity of polymorphonuclear leucocytes.⁹ These alterations may increase the sow's response to infectious agents.

The reproductive and urinary tract are predisposed to infection after farrowing because of the periparturient increase in the numbers of both nonpathogenic and facultative pathogens in the caudal vagina.⁵

Farrowing is accompanied by contamination of the cranial vagina in nearly all sows and contamination of the cervix and uterus in the majority of sows.⁶ While most bacteria are eliminated within 12 to 30 hours after delivery, facultative pathogens may overgrow nonpathogenic microflora and establish mildly invasive endometritis.⁶ Numerous agents have been isolated from vulval discharges (and from uteri) of sows, but it is still unclear which are pathogens and which are opportunistic invaders. *Erysipelothrix rhusiopathiae* may be an opportunistic invader of the urogenital tract of the sow, and may be able to overgrow other opportunistic organisms in the urogenital tract of the periparturient sow. It is also possible that soon after parturition, the urogenital organs are simultaneously colonized by multiple organisms from the cranial vagina, which may include indigenous microbes, bacterial contaminants, and pathogens (in this case, *E rhusiopathiae*). Perhaps immunological naivete of the endometrium, especially in young parity females or in non-vaccinated sows, facilitates colonization by *E rhusiopathiae*.

In acute SE, areas of congestion may be found in the urinary bladder mucosa.³ Chronic urogenital infections caused by *E rhusiopathiae* often result in proliferative bladder mucosal changes, ie, coagulopathy and fibrinous exudates.⁴ Mucosal hyperemia and congestion, mucosal ulceration, and accumulation of fibrinopurulent exudate over affected areas of the urinary bladder mucosa has been reported in chronic SE.⁵ Similar changes may occur in the early postpartum endometrium as well. Because tissue decomposition provides a medium conducive to the growth of a variety of opportunistic microorganisms, retention of remnants of the placenta may predispose sows to mild metritis and endometritis.⁴ In this case, we suspect that the sows developed mild chronic postparturient endometritis caused by *E rhusiopathiae*, and that this infection continued throughout lactation. It is possible that in the infected sows, the cervix closed normally within a few days of farrowing, preventing drainage of the uterine content. In the absence of cyclic changes in ovarian activity during lactation, there is inadequate endocrine modulation of mucosal immune responses. Consequently, the ability of affected sows to resolve the infection was further compromised.

Other large herds owned by the same breeding organization in the same geographic area shared some managers and technicians, who moved frequently between the herds. Within a 30-km radius of the investigated production units, the following infectious agents were identified in vulval discharges of sows: *Clostridium* spp, *Actinobaculum suis* (formerly *Eubacterium suis*), *Klebsiella* spp, *Proteus* spp, and *E coli*. However, *E rhusiopathiae* was identified only in the case herd. In our opinion, there is strong circumstantial evidence that *E rhusiopathiae* played a role in the fertility problems observed in this herd.

Under field conditions, in large breeding units, erysipelas vaccination is not always entirely effective in preventing disease. Despite vaccination, some sows may still be affected by the disease, but vaccination provides a worthwhile means of control for the whole herd. In this case, we noted a marked reduction in periparturient vulval discharge after erysipelas vaccination. Four months after vaccination, *E rhusiopathiae* was not cultured from anterior vaginal swabs of 17 of the 21 sows that had been culture-positive prepartum. We have no explanation for this phenomenon other than that vaccination against SE was protective in these sows.

Implications

- In herds experiencing an increase in periparturient vulval discharges, a culture of the discharge material is indicated to rule out less obvious causes, such as *E rhusiopathiae*.
- Because of the ubiquity of *E rhusiopathiae* and its poorly understood ability to exist in nature, the possibility of eradication of the organism seems remote; therefore, vaccination should be used in conjunction with good management practices to control swine erysipelas.

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