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Infectious disease

Erysipelothrix rhusiopathiae infection in a captive white-lipped peccary (*Tayassu pecari*) in Finland

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ABSTRACT

Erysipelothrix rhusiopathiae is a zoonotic pathogen that causes infections in several animal species, including erysipelas in swine, lambs and turkeys. In October 2022, a captive, 1-year-old white-lipped peccary (*Tayassu pecari*), kept in a herd of five peccaries in a zoo in Finland, suddenly developed signs of inappetence and reluctance to move. Despite treatment, the peccary was found dead. At necropsy, the main gross finding was severe acute segmental necrotizing enteritis. Several other organs had lesions compatible with acute septicaemia, including petechiae and ecchymoses. Histopathology of the intestine revealed severe acute multifocal necrotizing enteritis with neutrophilic vasculitis, vascular fibrinoid microthrombi and myriad clusters of densely packed, rod-shaped, gram-positive bacteria on the tips of the intestinal villi. Bacterial culture was identified as *E. rhusiopathiae* by MALDI-TOF mass spectrometry. To our knowledge, this is the first report of a naturally occurring *E. rhusiopathiae* infection in a captive white-lipped peccary. Our findings suggest that regular vaccination of captive white-lipped peccaries should be taken into consideration in preventing infections due to *E. rhusiopathiae*.

The white-lipped peccary (*Tayassu pecari*) is a species of tropical and subtropical non-ruminating ungulate belonging to the order Artiodactyla, suborder Suina and family Tayassuidae [1,2]. Within the Tayassuidae family, three known species exist: white-lipped peccary, collared peccary (*Pecari tajacu*) and chacoan peccary (*Catagonus wagneri*) [1]. On the International Union for Conservation of Nature's Red List of Threatened Species (IUCN Red List), the white-lipped peccary is currently listed as vulnerable [3], whereas the collared peccary is of least concern [4] and the chacoan peccary is endangered [5]. Geographically, the habitat of wild white-lipped peccaries ranges from the southwestern USA to South America, from Mexico to Argentina [1]. Several zoos around the world have collections of this species.

Peccaries have a common ancestor with swine (Suidae) and the two families have been shown to share pathogens, although the role of these in peccaries is largely unknown [6]. It is possible that peccaries act as reservoirs for agents infecting domestic and wild swine [6]. Wild white-lipped peccaries have tested positive for several pathogens known to infect swine, including Suid alphaherpesvirus 1, porcine circovirus 2, *Mycoplasma hyopneumoniae*, *Pasteurella multocida* and *Leptospira* spp, although a lack of complete pathological examinations and epidemiological studies makes it difficult to evaluate the actual importance of these infectious agents in peccaries [6–9]. Cases of seropositivity for Aujeszky's disease have been reported [8], as well as the presence of antibodies to, or isolation of, *Toxoplasma gondii* [10,11], both currently being the most reported pathogens in wild white-lipped peccaries [12]. For other protozoan parasites, low *Trypanosoma evansi* parasitaemia was found in wild white-lipped peccaries in Brazil [13]. Interestingly, experimental studies have not proved peccaries susceptible to African swine fever virus [2], and serological tests for the virus in white-lipped peccaries have been negative [8]. In Colombia, a single white-lipped peccary tested negative for classical swine fever, whereas in collared peccaries the prevalence was 5% [9].

White-lipped peccaries have been proposed to act as reservoirs for *Erysipelothrix rhusiopathiae*. Previously, *E. rhusiopathiae* has been isolated from the tonsils of a slaughtered white-lipped peccary living in a semi-extensive pasture system in Brazil, suggesting that the

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peccary was a healthy carrier of the pathogen [14]. In addition, serology has been negative for *E. rhusiopathiae* in captive collared peccaries [15]. Thus, research regarding *E. rhusiopathiae* in white-lipped peccaries is scarce and a detailed description of *E. rhusiopathiae* infection is lacking.

Erysipelothrix rhusiopathiae is a small, slender, gram-positive, non-sporulating, non-motile, rod-shaped, facultative anaerobic bacillus that commonly causes erysipelas in swine, lambs and turkeys [16–19]. The bacterium can infect a variety of domestic and wild animals, including vertebrate and invertebrate species [16,20]. *Erysipelothrix rhusiopathiae* is a zoonotic pathogen. The most common form of infection in humans is a localized cellulitis known as erysipeloid [16].

Swine are believed to be the most important reservoirs for *E. rhusiopathiae* [19]. Clinically healthy carriers can harbour the bacteria in their tissues and excrete them with their secretions [2,16,19]. *Erysipelothrix rhusiopathiae* is ubiquitous and persistent in the environment [20]. Therefore, the most common route of infection is ingestion of contaminated material with subsequent entry of the pathogen through mucous membranes or tonsils. Infection can also occur via cutaneous wounds [16,18]. Infection in swine causes porcine erysipelas, which most commonly affects young pigs (2–12 months of age) and pregnant sows [18]. The disease can be either acute, subacute or chronic [16,18,21]. In domestic swine, vaccination with either attenuated live or inactivated bacterin vaccines is used to control the disease [16,19,22]. Vaccines protect from the acute and subacute forms of the disease, while protection against the chronic form is less effective [19,23]. Vaccine failures have been reported with the use of vaccines containing killed bacterins [24]. Attenuated live vaccines have caused chronic disease, characterized by arthritis as a side effect, in some swine [25].

In farmed wild boars, *E. rhusiopathiae* has caused acute septicaemia with lesions resembling porcine erysipelas [26,27]. The reported cutaneous lesions consisted of marked ventral irregular erythematous or cyanotic areas [26]. Several internal organs were congested and showing petechiae or ecchymoses compatible with acute septicaemia. Histopathological findings in the skin, kidneys and urinary bladder included fibrinous microthrombi and vasculitis with fibrinoid necrosis. Necrotizing vasculitis was also found in the eye, along with bilateral acute panuveitis [26].

In October 2022, a captive 1-year-old castrated male white-lipped peccary was found dead in its enclosure in Helsinki Zoo. The peccary was housed in a herd with four other white-lipped peccaries. Two days earlier the peccary had been losing appetite and was reluctant to move due to a suspected trauma. It was treated with a peroral dose of non-steroidal anti-inflammatory drug (meloxicam, 0.3 mg/kg; Metacam 15 mg/ml; Boehringer Ingelheim Vetmedica, www.boehringer-ingelheim.com). These clinical signs persisted on the following day with additional diarrhoea and polydipsia. Anaesthesia was induced to perform further clinical examination with no abnormal findings detected except for weak peripheral blood flow. The peccary was treated with intravenous fluids (Ringer-acetat Fresenius Kabi solution for infusion; Fresenius Kabi, www.fresenius-kabi.com) and antibiotics (trimethoprim-sulfadiazine, 15 mg/kg; Borgal vet. 240 mg/ml; Ceva Santé Animale, www.ceva.com). The peccary recovered from anaesthesia but was found dead the next morning in the enclosure. Another 1-year-old peccary from the same enclosure developed similar clinical signs 11 days after the death of the first peccary. This animal was treated with antibiotics (penicillin, 15 mg/kg; Ethacilin vet. 300,000 IU/ml; Intervet International, www.msd-animal-health.com) and trimethoprim-sulfadiazine (15 mg/kg; Borgal vet., Ceva Santé Animale) and recovered completely. The older animals in the herd remained asymptomatic.

The dead peccary was submitted to the Pathology Unit at the Department of Veterinary Biosciences, Faculty of Veterinary Medicine, University of Helsinki, Finland, where it underwent a full post-mortem examination with fresh tissue samples obtained from the heart, lungs, spleen, liver, kidneys and small intestine and stored in −18°C. Tissue samples from the brain, pituitary, thyroid, adrenal and prostate glands, lungs, heart, spleen, liver, kidneys, pancreas, stomach, small and large intestines and urinary bladder were collected and fixed in neutral buffered 10% formalin and processed routinely. Paraffin-embedded sections (4 μm) were stained with haematoxylin and eosin (HE) for light microscopy. Selected sections from the heart and intestines were stained with Gram stain to visualize the bacteria.

The main gross finding was severe acute segmental necrotizing enteritis. The contents of the distal duodenum and proximal jejunum were watery and dark reddish. The small intestinal wall was thin, smooth and dull. The glandular stomach had moderate acute focal hyperaemia with mild acute multifocal ulcerations. Other findings included multifocal acute petechiae and ecchymoses in the oral mucosa, moderate acute diffuse splenomegaly, severe acute diffuse pulmonary oedema and moderate acute congestion at the renal corticomedullary junction, which were interpreted as evidence of shock. No parasite eggs were detected in faeces in passive test tube flotation in saturated $MgSO_4$.

Histopathological findings in the small intestine, lungs, liver, heart and kidneys were compatible with bacteraemia and acute septic shock. In the small intestine, the surface epithelium was multifocally necrotic with karyorrhectic nuclei, cellular debris and toxic neutrophils, including myriad clusters of densely packed rod-shaped bacteria on the tips of the villi (Figs. 1 and 2). The lamina propria was diffusely oedematous with multifocal superficial infiltrations of toxic neutrophils. There was severe acute multifocal oedema, congestion and haemorrhages together with multifocal acute vascular fibrinoid microthrombi, intravascular bacteria and toxic neutrophils in the submucosa. The liver, lungs, heart and kidneys had mild to moderate acute neutrophilic inflammation with variable degrees of haemorrhage. Intralesional rod-shaped bacteria were identified within the tissue parenchyma and blood vessels. The liver and heart had evidence of acute neutrophilic vasculitis and fibrinoid thrombosis, respectively. Gram staining of heart and small intestine samples revealed short, slender, gram-positive, rod-shaped bacteria admixed with moderate numbers of larger and longer, unevenly stained gram-positive rods and fewer gram-negative short rods (Fig. 2).

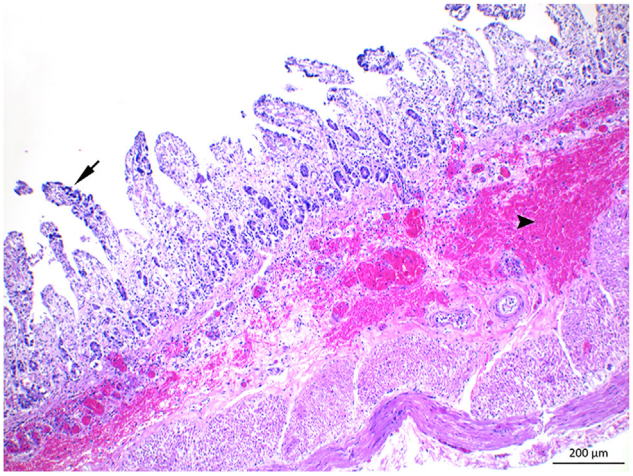

Fig. 1. Erysipelas, small intestine, white-lipped peccary. Necrosis of surface epithelium with karyorrhectic nuclei, cellular debris, toxic neutrophils and myriad clusters of densely packed rod-shaped bacteria on villous tips (arrow). Haemorrhage in submucosa (arrowhead). HE.

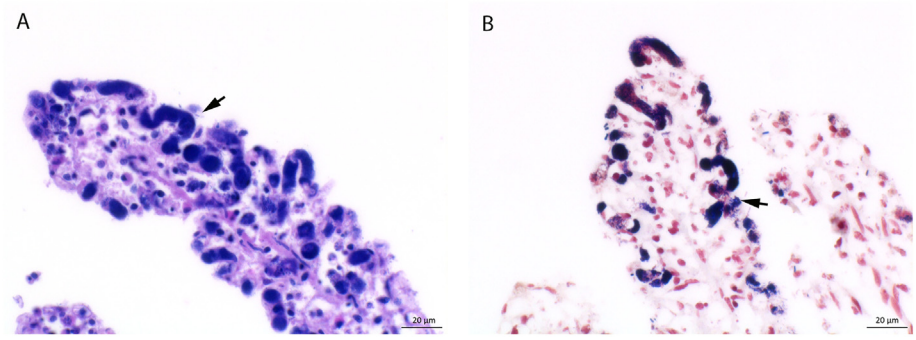

Fig. 2. Erysipelas, small intestine, white-lipped peccary. Short, slender, gram-positive rod-shaped bacteria morphologically compatible with *E. rhusiopathiae* on tips of small intestinal villi (arrows). (**A**) HE. (**B**) Gram.

Tests were conducted for bacterial and viral diseases on organ suspensions of fresh frozen tissue samples from the lungs, liver, kidneys, spleen, heart and small intestine at the Animal Health Diagnostic Unit, Finnish Food Authority, Helsinki, Finland. Several serious swine viruses were ruled out by polymerase chain reaction (PCR), including African swine fever, classical swine fever, Aujeszky's disease, transmissible gastroenteritis virus and porcine reproductive and respiratory disease virus. For identification of bacterial infections, the samples were cultured on blood agar incubated aerobically and on fastidious anaerobe agar (Neogen, www.neogen.com) with 5% horse blood incubated anaerobically. Plates were incubated overnight at 37°C. The growth on the plates was identified as *E. rhusiopathiae* using MALDI-TOF mass spectrometry analysis (Bruker Daltonics, www.bruker.com) and the commercial Bruker MBT Compass reference library (version 2020).

Following confirmation of the diagnosis, all remaining white-lipped peccaries in the zoo were vaccinated against *E. rhusiopathiae* intramuscularly by darting (Porcilis Ery vet.; Intervet). Based on the vaccine manufacturer's instructions, a vaccination programme was commenced with regular future vaccinations. Peccaries were trained to voluntarily accept manual vaccination to reduce the amount of stress related to vaccination.

To our knowledge, this is the first report of a naturally occurring *E. rhusiopathiae* infection in a captive white-lipped peccary with clinical signs resembling *E. rhusiopathiae* infection in domestic swine and wild boars [21,26,27]. The pathological findings suggested an acute course of disease with no evidence of chronicity in the heart or joints. No typical rhomboid skin lesions were detected. However, peccary skin is dark and hairy and can therefore mask subtle lesions associated with vasculitis. Palpation of the skin or visual examination of areas with raised hairs may aid in diagnosing skin lesions in dark-skinned animals [19].

The origin of the infection could not be confirmed, but environmental contamination was considered as a potential source, as *E. rhusiopathiae* is known to be ubiquitous in the environment with the most common route of infection being ingestion of contaminated material. The white-lipped peccary was housed in an enclosure with indoor and outdoor compartments. Since *E. rhusiopathiae* has been isolated from the tonsils of a healthy white-lipped peccary [14], it is also possible that the pathogen was persisting in the tonsils or other tissues of an asymptomatic animal shedding the pathogen into the environment via its excretions [16].

The affected white-lipped peccary was only 1 year old, corresponding to the typical age of infection in swine [18]. In swine, very young animals may be protected by maternal antibodies, whereas older animals are likely to have developed immunity against the pathogen [16]. Other factors related to the susceptibility of an individual animal could be the virulence of the specific *E. rhusiopathiae* strain [16]. In our case, the outbreak occurred simultaneously with a temperature drop, which may have increased host susceptibility to infection. Stress, such as temperature change, is another important risk factor described in swine infections [16]. In domestic swine, *E. rhusiopathiae* infection can be prevented by vaccination, which is widely used [16,19]. The white-lipped peccaries in Helsinki Zoo had not been previously vaccinated because their susceptibility to *E. rhusiopathiae* infection was unknown and vaccination by darting is stressful. Furthermore, the zoo had housed non-vaccinated wild boars for many years without any *E. rhusiopathiae* infections.

Peccaries are herd animals [1]. Consequently, a fast and accurate diagnosis is essential for protecting other animals in the affected herd. Therefore, efficient cooperation between clinicians, pathologists and microbiologists is crucial. In addition, collaboration is important from a One Health aspect, since *E. rhusiopathiae* is a zoonotic pathogen [16]. The infected white-lipped peccary was thus posing a zoonotic risk for the employees of the zoo. However, no human infections related to the case were reported.

Our findings suggest that *E. rhusiopathiae* infection in captive white-lipped peccaries can have a rapid clinical course leading to the death of the animal. Besides being apparent carriers of *E. rhusiopathiae* [14], white-lipped peccaries seem to be susceptible to the acute disease, with pathological findings resembling acute erysipelas in swine and wild boars. However, further studies, especially on virulence factors of the *E. rhusiopathiae* strains, are required to better assess risk factors for *E. rhusiopathiae* infection in white-lipped peccaries.

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Hanna-Maaria Javela: Writing – original draft/review and editing; Histopathological analysis. **Niina Airas:** Supervision; Histopathological analysis; Writing – reviewing and editing. **Taru Lienemann:** Bacteriology; Writing – reviewing and editing. **Heli Nordgren:** Histopathological analysis; Writing – reviewing and editing. **Sanna Malkamäki:** Gross findings; Writing – reviewing and editing. **Sanna Sainmaa:** Clinical investigation; Writing – reviewing and editing. **Jonna Kyyrö:** Virology; Writing – reviewing and editing.

Declaration of competing interests

The authors declared no conflicts of interest in relation to the research, authorship or publication of this manuscript.

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