species have different geographic distributions (*1*). Adult *Armillifer* spp. and *P. crotali* parasites infect the respiratory tract of snakes (the definitive hosts) and lay eggs that are released into the environment through bronchial secretions or feces. Mammals are intermediate hosts and become infected with larvae and then nymphs after ingesting the parasite eggs. Nymphs encyst within the tissues of intermediate hosts and transform into adults after ingestion by a snake. Humans are incidental hosts and are typically infected through ingestion of raw or undercooked snake meat or ingestion of parasite egg–contaminated food or water.

In humans, larvae migrate and encyst mostly in the peritoneum, liver parenchyma, or abdominal lymph nodes. Because humans are not normally consumed by definitive hosts, the parasitic life cycle is not completed. Encysted nymphs can remain in tissues for several years before degenerating and eventually becoming calcified, which produces the characteristic C-shaped calcifications visible during imaging (*2*). Subcutaneous localization is less frequently reported and might manifest as a painful abdominal wall mass, potentially mimicking a surgical emergency. Similar manifestations involving inguinal hernias have previously been described (*1*,*3*). Most infections are asymptomatic. Symptomatic cases are uncommon and might be caused by inflammatory reactions to degenerating larvae. A diagnosis is usually made according to imaging or histopathology, but species identification is confirmed by using molecular methods (*4*).

In conclusion, this case illustrates an unusual clinical manifestation of *A. armillatus* infection that mimicked a surgical emergency in an *A. armillatus*–nonendemic setting. The long interval between exposure and symptom onset suggests a prolonged asymptomatic infection had occurred. The mechanisms underlying delayed clinical manifestations remain unclear. With increasing travel and human migration, clinicians in regions that are not endemic for *A. armillatus* infections should be aware of this parasitic infection, particularly in patients with relevant exposure history. Molecular tools are essential for *A. armillatus* identification but morphologically based diagnostic orientation and procedures are also required.

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Emerging Protoparvovirus in Pigs and Wildlife, Denmark

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DOI: https://doi.org/10.3201/eid3212.261184

We detected a protoparvovirus recently associated with an outbreak in pigs in the Netherlands in pigs with exophthalmos and in fox feces and spleens in Denmark. Pig viruses were phylogenetically closer to each other than to viruses found among wildlife of their respective countries, suggesting a link between the farm cases.

In December 2024, a parvovirus closely related to one discovered in red fox (*Vulpes vulpes*) feces in the Netherlands (fox parvovirus, *Protoparvovirus carnivoran4*) (Appendix Figure 1, https://wwwnc.cdc.gov/EID/article/32/12/26-1184-App1.pdf) (*1*) caused a large multifarm outbreak involving >80 pig herds in the Netherlands (*2*). Pigs infected with fox parvovirus had exophthalmos, erythema, and other nonspecific clinical signs. In June 2026, similar signs, including exophthalmos, strabismus (Appendix Figure 2), weakness, erythema, and alopecia, were noted in 1.5%–2% of piglets and recently weaned pigs from a herd of 920 sows in Denmark. Follow-up inspections revealed exophthalmos in 20% of pigs 2 weeks of age, 40% of recently weaned pigs, 60%–80% of pigs 2 weeks after weaning, and 0.5% of pigs 6 weeks after weaning. Among pigs 2 weeks after weaning with exophthalmos, ≈50%–60% also had erythema and low body condition scores. The herd tested free from porcine pathogens that are routinely surveyed annually (Appendix). Antimicrobial drugs were administered to recently weaned pigs but were ineffective (Appendix).

The farmer selected 6 weak piglets (1–2 weeks of age) with exophthalmos for euthanasia and subsequent necropsy. Examination revealed mucus in the nasal cavities of all 6 piglets and mild exophthalmos, eyelid edema, and eyelashes adhered by remnants of ocular discharge in 4 piglets. We observed consolidation of the lobus medius (≈7% of lung tissue) and serous fluid in 1 bulla tympanica in 1 piglet. Two piglets had minimal stomach content; the others had moderate milk amounts. Two piglets had increased synovial fluid in several joints, and we cultured streptococci from 1 elbow joint. We also noted swelling of the liver, spleen, and kidneys, possibly from euthanasia with pentobarbital. We collected liver and kidney samples from all 6 pigs and tested them for fox parvovirus by seminested PCR (Appendix). All samples were PCR-positive. The animals were coccidia-negative, and no other specific diagnostic tests were performed.

We screened for fox parvovirus DNA previously isolated from wild animals sampled throughout Denmark during 2018–2024 (*3*) (Appendix Figure 3). We detected fox parvovirus DNA in 1 of 2 fecal samples from red foxes, supporting previous results by Bodewes et al. and Lojkić et al., who found this virus in fox stool samples in the Netherlands and Croatia (*1*,*4*). In addition, we detected fox parvovirus DNA in 2 (2.5%) of 80 spleen samples from red foxes (2022–2023) but none in 27 spleen samples from other wild carnivorans (Appendix). The 2 foxes positive for fox parvovirus had no pathologic signs of disease (Appendix), but 1 fox was also amdoparvovirus-positive (*3*).

We obtained the full viral genome sequence from the PCR-positive fox fecal sample (XFM39) by using metagenomic sequencing (18,876 reads with an average coverage of 558×, range 29×–782×), as previously described (*5*). In addition, we performed Sanger-based complete genome sequencing of fox



Figure 1. Phylogenetic analysis of fox parvoviruses from study of emerging protoparvovirus in pigs and wildlife, Denmark. Midpoint-rooted maximum-likelihood phylogenetic tree (left) was built with a concatenated alignment of nonstructural protein 1 and virus protein capsid 2 gene fragments (1,152-nt) of members of *Protoparvovirus carnivoran4*. Animal icons indicate host species; colors indicate country of origin. Map demonstrates country of detection and was created with MapChart (https://www.mapchart.net). Sequence labels indicate GenBank accession number, strain name, and year of detection. We used concatenated fragments because only partial sequence data was available for virus 55589, but analyses of complete genomes and individual genes produced consistent results. Tree constructed in Stranded (VerCan ApS, https://www.vercan.dev) by using IQ-TREE 3 (https://iqtree.github.io) and a transition probability matrix 2 with unequal base frequencies plus empirical base frequencies model, identified as the best fitting for genetic distance estimation via the modelfinder function in IQ-TREE. Values at nodes indicate the outcomes of the Shimodaira–Hasegawa approximate likelihood-ratio test and ultrafast bootstrapping on the basis of 1,000 replicates. Final figure image created in BioRender (BioRender.com). Scale bar indicates nucleotide substitutions per site.

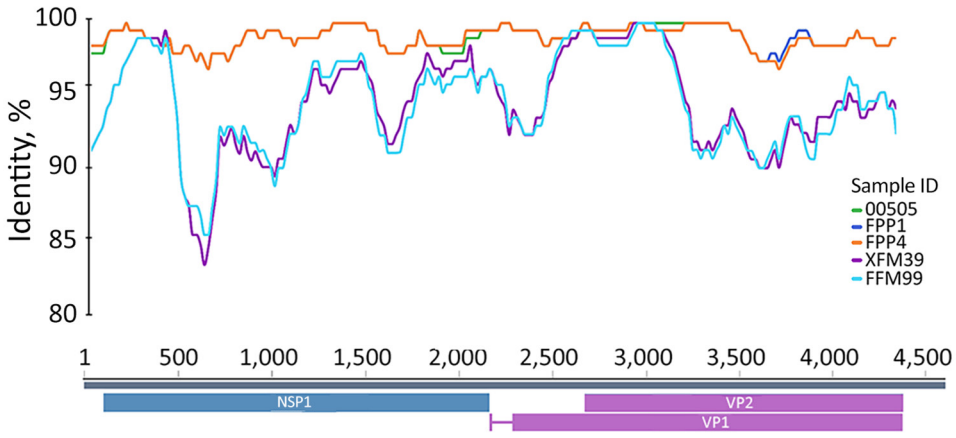


Figure 2. Complete genome comparisons of fox parvoviruses from study of emerging protoparvovirus in pigs and wildlife, Denmark. Sequence identities across complete genomes of fox parvoviruses from pigs and foxes were compared with Fox 6 as the reference sequence. The y-axis depicts pairwise identity across the genome, whose positions are reported on the x-axis, illustrated at the bottom. Colored rectangles indicate predicted open reading frames after splicing. Analyses performed with Stranded (VerCan ApS, https://www.vercan.dev). ID, identification; NSP1, nonstructural protein 1; VP1, virus capsid protein 1; VP2, virus capsid protein 2.

parvoviruses from 1 PCR-positive fox spleen sample (FFM99) and 2 PCR-positive pig liver samples (FPP1 and FPP4) (Appendix). We obtained partial fox parvovirus sequences from all other PCR-positive samples: the sequence from the second positive fox spleen sample was identical to XFM39, and sequences from the other 6 pig samples were identical to FPP1 and FPP4.

Maximum-likelihood phylogenies (*6*) showed that the virus from the outbreak in the Netherlands was the closest relative to the ones from the Denmark pig farm. The parvoviruses from the pig farm outbreaks in Denmark and the Netherlands shared 99.9% genomic identity, including 2 nonsynonymous substitutions in the nonstructural protein gene (Figures 1, 2; Appendix Figure 4). Those viruses clustered in a clade that also included fox viruses from Croatia and the Netherlands (Figure 1). Viruses from foxes in Denmark shared <95% identity with all other viruses and formed a distinct clade (Figures 1, 2; Appendix Figure 4).

Our study supports the hypothesis that this parvovirus is a porcine pathogen and reports its detection in Denmark. Clinical signs resembled those observed in herds in the Netherlands. Controlled and epidemiological studies are needed to establish viral association with disease and viral prevalence and diversity in pig farms across countries.

Detecting fox parvovirus in fox organs suggests that this virus is not simply carried in feces but can infect and replicate in carnivorans, similar to other protoparvoviruses (*7*). However, its pathogenic role in wildlife remains unknown. Viruses from pig farms in the Netherlands and Denmark were more closely related to each other than to wildlife viruses from their respective countries, suggesting an epidemiologic link between farm outbreaks and possible multicountry spread after a single introduction into pigs. Although fox parvovirus detection in foxes preceded the recognition of the pig syndrome, definitively concluding whether foxes and pigs were infected by the same source or the virus originated from wild animals and was introduced into farms, or vice versa, was not possible because previous farm outbreaks might have gone undetected. Given the environmental stability of parvoviruses (*7*), we hypothesize an environmental-related transmission between wild and farmed animals (e.g., through contaminated material introduced into farms or exposure to contaminated farm-originating biological byproducts).

Our study was limited to cases from a single farm, lacked histopathologic evaluation, and included few recently collected wildlife samples. Larger-scale studies are needed to determine virus origin and spread among pig farms and wildlife globally and to clarify pathogenicity and ecology, including identifying maintenance and spillover hosts, host range, and transmission routes.

In summary, the close genetic relationship between viruses from pig outbreaks in Denmark and the Netherlands suggest a farm-related international epidemiological link. Those findings support additional surveillance of fox parvovirus among domestic pigs and wildlife.

Acknowledgments

We thank Joost T.P. Verhoeven for implementing the metagenomic pipeline V2D (https://github.com/jtpverhoeven/v2d) and Nina Dam Grønnegaard, Hue Thi Thanh Tran, Anna Cecilie Boldt Eiersted, and Sally Xin Hildebrandt for technical assistance.

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Heartland Virus in Lone Star Ticks, South Carolina, USA, 2021–2024

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DOI: http://doi.org/10.3201/eid3212.260320

Tickborne arboviruses are a serious health threat, but surveillance gaps remain. We tested lone star nymphs collected in South Carolina, USA, for Heartland and Bourbon viruses. Six pools were positive for Heartland virus; none were positive for Bourbon virus. Longitudinal surveillance provides insights into the emergence and distribution of tickborne pathogens.

Heartland virus (HRTV; family *Phenuiviridae*, genus *Bandavirus*) and Bourbon virus (BRBV; family *Orthomyxoviridae*, genus *Thogotovirus*) are emerging tickborne pathogens. Both viruses were detected in the contiguous United States within the past 15 years (*1*,*2*). Symptoms of identified infections included acute febrile illness characterized by thrombocytopenia and leukopenia, with progression to multiorgan failure and death in severe cases (*3*). Wildlife serosurveillance suggests both pathogens are more widely spread than reported cases indicate (Appendix, https://wwwnc.cdc.gov/EID/article/32/12/26-0320-App1.pdf).

The lone star tick (*Amblyomma americanum*) is the primary vector for both Heartland and Bourbon viruses (*4*,*5*). Lone star tick range is well established across the eastern United States and continues to expand northward and westward, driven by host movement, land use changes, and climate change (*6*). White-tailed deer (*Odocoileus virginianus*) are a keystone host for lone star ticks; a retrospective wildlife serosurvey conducted in South Carolina reported HRTV antibodies in 20% (1/5) of white-tailed deer samples collected in 2001 and in 40% (2/5) of samples collected in 2008 (*7*). Similar studies have reported evidence of BRBV in neighboring states (*3*). Although