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Lack of Evidence for African Swine Fever Infection or Transmissibility in Experimentally Infected Rats

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Abstract

African Swine Fever Virus infections have significantly impacted swine production in many parts of the world. Rodents are considered vectors of several swine disease agents but their role in ASFV transmission has not been determined. This work sought to determine whether rats could be experimentally infected with ASFV.

In a pilot project prior to the experimental trial, 27 rats, mice, and shrews were trapped from 3 farms (range 7-10 per farm) with current ASFV outbreaks. These animals were euthanized, sampled, and tested for ASFV with polymerase chain reaction (PCR). None of the samples (n = 81) were PCR-positive for ASFV.

The experimental trial utilized 45 commercially obtained rats (Rattus norvegicus domestica): 9 were orally inoculated with ASFV, 9 were intraperitoneally inoculated with ASFV, 18 were non-inoculated contacts, and 9 were negative controls. On each of days 7, 14, and 21 post-inoculation, 3 orally inoculated, 3 intraperitoneally inoculated, 6 non-inoculated contacts, and 3 negative control rats were euthanized, sampled, and tested for ASFV with PCR.

Clinical signs did not develop in any of the rats. Body temperatures increased during week 2, but there was no difference between the inoculated and non-inoculated groups. None of the samples (blood, spleen, liver, lung, and ileum) were PCR positive for ASFV. Serum tested with ELISA for ASFV antibodies was negative.

Despite direct ASFV challenge, inoculated rats did not develop clinical disease symptoms or transmit ASFV to contacts. Further work is necessary to demonstrate the competence of rodents as biological vectors for ASFV transmission in swine herds.

Keywords

African Swine Fever, biosecurity, disease transmission, rodents, vectors

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Introduction

African Swine Fever (ASF) was first observed in Asia in 2018, with devastating effects on the Chinese swine population (Food and Agriculture Organization of the United Nations, 2021; Liu *et al*., 2021). In January 2019, ASF was confirmed for the first time in Vietnam, on a 20-sow farm in Hung Yen province (Le *et al.*, 2019). Only 5 months later, ASF had spread to all 62 Vietnamese provinces, resulting in the death or culling of approximately 6 million pigs, or > 20% of Vietnam's pig population. This spread occurred in spite of prevention and control efforts implemented by the government. The vast majority of outbreaks occurred in small- to medium-sized farms with poor biosecurity (Nguyen-Thi *et al.*, 2021). The rapid spread of the disease across the country exemplified the virus' ability to easily infect pigs and move between farms, and provincial and international borders.

The effects of ASF on pigs and pork-producing operations can be significant. Effects on animals and operations can vary according to the virulence of the particular viral strain present. In peracute and acute phases of the disease, fatality rates of 100% can be observed (Blome *et al.*, 2020). Clinical signs are mostly non-specific, including fever, anorexia, and respiratory and digestive abnormalities. Surviving pigs may exhibit reddening of the ears and snout, followed by more widespread reddening and bleeding from body orifices. Pregnant animals may abort. In subacute and chronic cases, emaciation, swollen joints, and respiratory difficulty can affect a high percentage of pigs, making them unmarketable (Li *et al*., 2022).

The virus causing ASF, African Swine Fever Virus (ASFV), is readily transmitted between infected and susceptible pigs. Transmission routes include direct contact with blood, saliva, feces, or urine from infected pigs (considered biological vectors because the virus multiplies within the body); fomites (such as vehicles, equipment, and feed); and ticks and blood-sucking flies, which can serve as biological (ticks) or mechanical (flies) vectors.

In addition, ASFV is stable in multiple environments, including refrigerated pork products. It can survive in affected pig carcasses for several weeks or months after death (Chenais *et al.*, 2019; Li *et al*., 2022), and can be infective for short periods of time (less than 3 days) in contaminated pig environments (Olesen *et al.*, 2018). These factors increase the chance for vectors or fomites to transmit the virus to susceptible pigs.

The role of blood-feeding arthropods as mechanical vectors for ASFV transmission has been documented (Mellor *et al.*, 1987, Vergne *et al.*, 2021). Factors such as the need for these insects to feed on several hosts to complete a blood meal, the regularity with which they complete meals, and the ability to regurgitate blood during feeding promote the ability of these arthropods to serve as mechanical vectors for ASFV. Rodent-pig interactions are different from insect-pig interactions, but nonetheless important. Their frequent entry into pens in search of food creates the potential for mechanical ASFV spread from contaminated environments to susceptible pigs (Akande, 2008).

Despite the extensive history of ASF infections globally, the role of rodents as biological or mechanical vectors has not been well-defined. Rodents have long been known to serve as important vectors of diseases such as leptospirosis (Makovska, 2023, Ospina-Pinto *et al.*, 2017), yet few studies have characterized their potential to transmit ASFV. Guinat *et al.* (2016) reported that blood samples from rodents on affected farms in Lithuania and Russia tested negative for ASFV. Fasina *et al.* (2012) noted that Nigerian pig farmers who implemented rodent control measures had higher risks of ASF infection in their pigs, presumably because controls were more likely imposed on farms with existing heavy rodent infestations. Experimental evidence that rodents can or do not serve as vectors for ASFV is lacking.

The objective of this work, therefore, was -- following a pilot project that sought to detect ASFV in and on captured rodents in swine facilities with recent ASF outbreaks – to determine if experimental rodents are susceptible to infection with ASFV and

whether those rodents could transmit the virus to uninfected contacts.

Materials and Methods

Experimental processes were reviewed and approved by leadership of the Key Laboratory of Veterinary Biotechnology at the Vietnam National University of Agriculture (VNUA).

Three swine farms that recently experienced ASF outbreaks were identified by investigators based on diagnostic submissions to VNUA (**Table 1**). The farms were located in 3 different provinces and ranged in size from 26 to 374 pigs. Also, the farms had recently experienced clinical ASF outbreaks, confirmed via real-time PCR testing of serum, blood or tissues from pigs present on the farm in July and August 2020.

Live traps were placed in each farm location within 5-14 days of positive ASFV results in affected pigs. On each farm, one trap was placed outside a barn (near drains, which served as entry and exit points for rodents), and one inside a barn (near feeders or feed storage, water sources, and access holes in walls). A variety of baits were used in each trap, including fried chicken, longan, sweet potato, and grilled crab. Traps were placed at approximately 5 PM each evening and collected at approximately 6 AM the following morning.

Trapped rodents were removed from live traps, placed in a holding cage on the farm and transported to VNUA every 2-3 days. Feces from the trapped rats were also collected from the cage, and placed in sterile conical tubes containing PBS, before being refrigerated. Trapping continued until 10 rats were obtained from each farm, or until several days had passed with no rodents captured.

Rodents were individually identified by farm source, euthanized by cervical dislocation, weighed and measured for length. Spleen, right front paw, and fecal samples were obtained from each rodent and submitted to the VNUA laboratory for real-time PCR testing.

The real-time PCR testing for ASFV in samples utilized primers and TaqMan® probe located within the VP72 genome region described previously (King *et al.*, 2003). DNA samples were extracted from blood and tissue samples using a commercial viral DNA extraction kit (Thermo-Fisher, MA, USA) with the King Duo Prime Automatic Extraction system (Thermo-Fisher, MA, USA). Real-time PCR test procedures were performed using the Invitrogen Real-time PCR kit (Invitrogen, MA, USA). Briefly, the real-time PCR master mix was prepared in a volume of 20 microliters containing 0.4 microliters each of sense and antisense primer and 0.25 microliter of probe primer. African Swine Fever Virus DNA amplification was accomplished with the following incubation program: 3 minutes at 95°C, 45 cycles at 95°C for 10 seconds and 58°C for 30 seconds, with fluorescence reading in the FAM channel at the end of each cycle.

The experimental challenge study took place in a dedicated laboratory room within the VNUA veterinary hospital. The room was equipped with negative pressure ventilation, filtration of entry and exit air, and maintained a controlled temperature of 21-23^{o}C.

Forty-five rats (Rattus norvegicus domestica) were obtained from a commercial supplier and divided into 7 cages. Six cages contained 6 rats each, with an additional cage holding 9 to serve as non-inoculated/non-contact controls. Individual weights were recorded for each rat.

ASFV challenge material was prepared by inoculating primary porcine alveolar macrophages (PAMs) with supernatant of a homogenate of ASFV-infected pig spleen. The PAMs were collected from 10–20-day-old specific-pathogen-free pigs obtained from the Pig Research Centre of the National Institute of Animal Science, Vietnam. The PAM cells were maintained in RPMI 1640 medium supplemented with 10% FBS (Thermo-Scientific, MA, USA) at 37°C with 5% CO2. Supernatants from the cell culture were collected 4 days post-inoculation, passaged 4 times, and then assayed for infectious virus particles with haemadsorption. A titer of 10^5 50% haemadsorption (HAD50) units per inoculum was used to infect each rat.

On Day 0, 3 rats from each challenge cage were inoculated either intraperitoneally or orally with the ASFV preparation, leaving 3 in each cage as non-inoculated contacts.

Body temperature for each rat was recorded once daily each morning, using Acroson type soft tip thermometers. Three temperature readings were taken each morning and averaged for a daily reading. Rats were fed daily, with fresh water provided every 2 days, and fresh bedding provided (rice husks) every 3 days.

On each of days 7, 14, and 21 post-inoculation, rats from one intraperitoneally inoculated cage (n = 6, 3 inoculated and 3 contact) and from one orally inoculated cage (n = 6, 3 inoculated and 3 contact) were euthanized, weighed, necropsied, sampled, and tested. Three were chosen at random from the non-inoculated/non-contact cage for similar testing at each time point as well.

Rats were anesthetized with ether and euthanized by exsanguination. Blood samples were taken during exsanguination. Necropsy examinations were performed on all rats, and tissue samples (spleen, liver, lung, ileum) were taken for PCR analysis. PCR analysis was conducted as described above. Serum was also tested for ASFV-specific antibodies via indirect ELISA testing.

Indirect ELISA testing of rat serum samples for ASFV-specific antibodies followed procedures outlined in the OIE Manual of Diagnostic Tests and Vaccines (OIE Manual, Chapter 3.9.1, Paragraph 2.1.2). This test is based on the use of whole antigens obtained from cell cultures infected with ASFV. The ASFV was propagated in PAM cells and inactivated with 0.05% β-propiolactone (BPL; Millipore Sigma, WI, USA) prior to use as antigen for the indirect ELISA (using goat anti-rat horseradish peroxidase (HRP)). This whole antigen (10^7 HAD50 units per well) was added to wells in a 96-well plate. Plates were incubated for 10 hours at 4°C, then washed twice with PBS Tween-20. Blocking buffer (200µL) was added, and plates were incubated for 2 hours at 67°C before being washed three times with PBS Tween-20. Serum samples were serially diluted, and 100µL of each dilution was added to the wells in the plate, which was incubated at 37°C for 2 hours and then washed 4 times with PBS Tween-20. Goat anti-mouse HRP solution was prepared by diluting at 1:4000 in blocking buffer; 100µL were added to each well. Plates were then incubated for 1.25 hours at 37°C and washed 4 times with PBS Tween-20. TMB substrate (100µL) was added and the plate was incubated for 15 minutes at room temperature. Stop solution (H_2SO_4 0.18M) was then added and the plate was shaken well. Optical density (OD) was measured in a plate reader set to 450nm.

All of the sampling and testing described above were repeated on Day 14 and Day 21 post-infection.

Rat body temperatures were analyzed in a univariate repeated models ANOVA procedure with a statistical software package (JMP Pro Version 16.0, NC, USA). Daily average temperature was used as the dependent variable, with day and infection group as the independent variables. Rat weight gain among groups was analyzed using ANOVA with a statistical software package. *P*-values of < 0.05 were used to determine statistical significance.

Results and Discussion

In the pilot project, 27 live rodents and shrews were trapped from the 3 farms (**Table 1**). The number of rodents trapped per farm ranged from 7 (farm 3) to 10 each (farms 1 and 2). Rodents varied in weight from 85 grams to 405 grams. The total number of samples taken per farm ranged from 21 (farm 3) to 30 (farms 1 and 2). Each sample taken from each trapped rodent or shrew (81 samples total) was PCR-negative for ASFV.

This pilot attempted to detect active infection in rodents and shrews trapped from farms experiencing ASFV infections in their pigs. No evidence was found among any of the trapped rodents or shrews to indicate they were infected with ASFV and able to serve as biological vectors of the disease. Furthermore, there was no evidence of these animals' ability to serve as mechanical vectors, as PCR testing of their paws was negative as well. However, it

Table 1. Characteristics of ASF-affected farms and rodent trapping, Vietnam, 2020

Farm	1	2	3
Location (province)	Hung Yen	Ninh Binh	Phu Tho
Pig population at the time of rodent trapping	64	26	374
ASF outbreak timing	July 2020	June 2020	May 2020
Pig test date(s)	July 20, 2020 July 30, 2020 August 6, 2020	July 25, 2020	July 23, 2020 July 30, 2020
Pig test procedure(s)	Serum PCR, tissue PCR (piglet spleen, kidney; sow spleen)	Whole blood PCR	Whole blood PCR
Pig test results	5/6 sows positive	3/9 sows positive	17/17 fattening pigs positive
Trapping date range	July 30-Aug. 20, 2020	Aug. 2-Sept. 8, 2020	Aug. 4-Sept. 4, 2020
No. and species of rodents captured	9 rats 1 mouse	5 shrews 4 rats 1 mouse	5 rats 2 mice
No. rodent samples tested (spleen, paw, feces)	30	30	21
No. ASF-positive rodent samples	0	0	0

could not be determined whether any trapped rodents had direct contact with pigs or environments directly contaminated by infected pigs. Although rodent trapping occurred soon after each farm's ASF outbreak, culling of pigs and disinfection of premises could have limited the amount of ASFV to which rodents were exposed; environmental sampling to detect ASFV in premises was not carried out. Rodents that were sickly or that had died at the farms were not captured or examined. It was possible that only healthy, robust animals were captured, as these were more likely to be hungry and prone to being trapped.

In the experimental inoculation, none of the inoculated rats, or those in contact with them, showed clinical signs of illness. Two control (non-inoculated/non-contact) rats died of unknown causes.

The average daily body temperature within the experimental groups is presented in **Figure 1**. Body temperatures of all rats varied significantly by day (P <0.0001), but not by group (P = 0.0929). There was a significant group by day interaction (P = 0.0030).

Mean rat weight gains among experimental groups ranged from 14.3 grams (orally infected) to 19.1 grams (controls; data not shown). No significant differences in rat weight gain were observed among experimental groups.

Sampling, PCR, and ELISA results from each observation group are presented in **Table 2**. Of the 225 samples taken from 65 rats over the three observation groups, none were PCR-positive for ASFV. Evidence of ASFV antibody production was not found in any of those examined, as determined by ELISA testing.

Despite intraperitoneal and oral challenges with ASFV, there was no evidence that the experimentally-infected rodents displayed an ASFV infection, or that ASFV transmission to uninfected contacts occurred, even after PCR testing multiple body organs. At no time during the experiment did any of inoculated or contact rats show signs of illness.

African Swine Fever Virus (or its nucleic acid) was not found in any of the sampled organs following incubation periods of 7, 14, and 21 days. Body temperatures of infected rats did not vary among groups and were not different from controls. Weight gains during the experimental

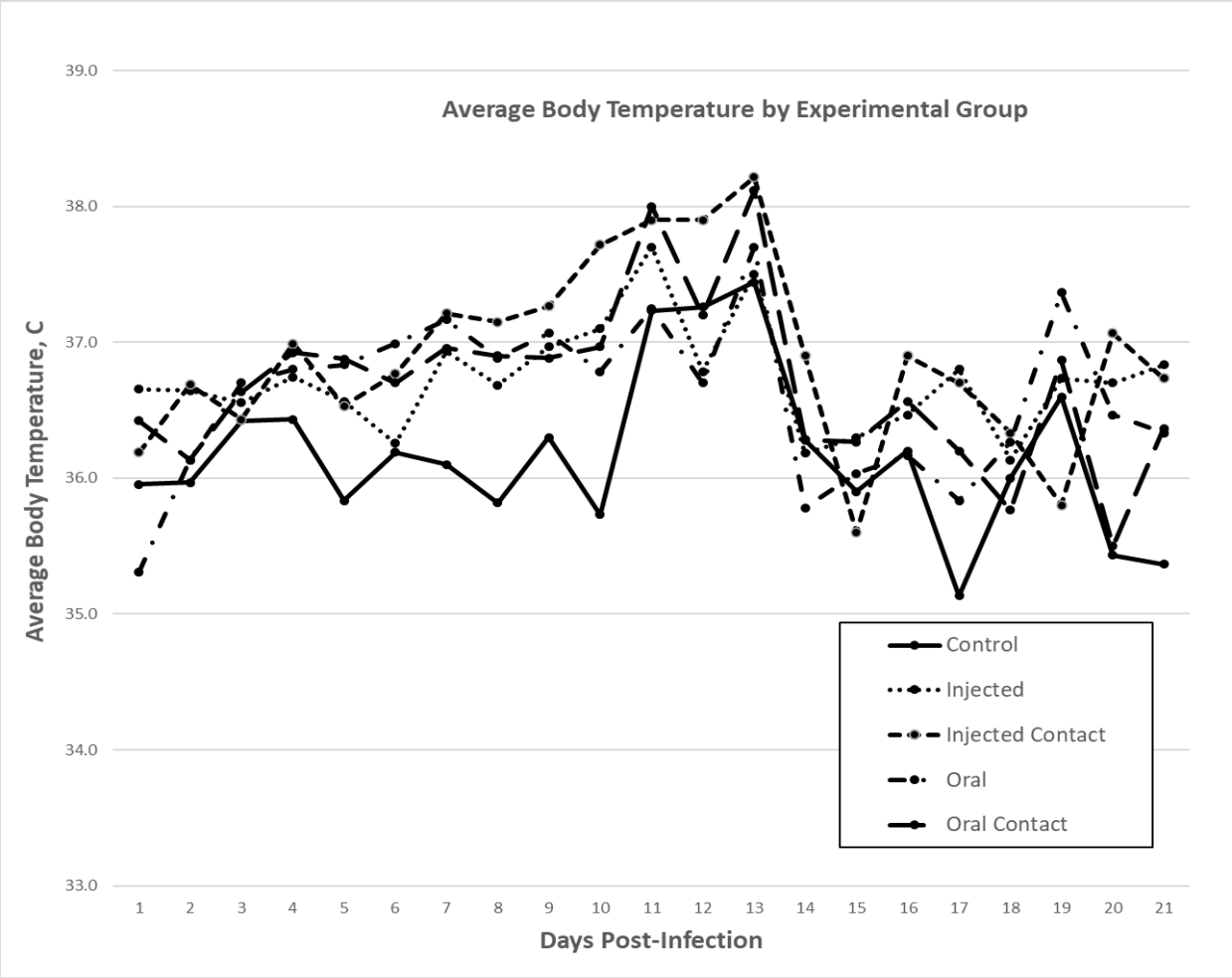

Figure 1. Average daily body temperature of ASFV-inoculated rats

Table 2. Sampling results from ASFV-inoculated rats

Day post-infection	Day 7	Day 14	Day 21
No. directly infected rats sampled*	3 intraperitoneally injected 3 orally dosed	3 intraperitoneally injected 3 orally dosed	3 intraperitoneally injected 3 orally dosed
PCR testing results	30 samples negative 0 samples positive	30 samples negative 0 samples positive	30 samples negative 0 samples positive
ELISA testing results	6 samples negative	6 samples negative	6 samples negative
No. contact rats sampled*	3 intraperitoneal contacts 3 oral contacts	3 intraperitoneal contacts 3 oral contacts	3 intraperitoneal contacts 3 oral contacts
PCR testing results	30 samples negative 0 samples positive	30 samples negative 0 samples positive	30 samples negative 0 samples positive
ELISA testing results	6 samples negative	6 samples negative	6 samples negative
No. control rats sampled	3	3	3
PCR testing results	15 samples negative 0 samples positive	15 samples negative 0 samples positive	15 samples negative 0 samples positive
ELISA testing results	3 samples negative	3 samples negative	3 samples negative

*Note: *Samples taken = blood, spleen, liver, lung, and ileum from each animal.*

period did not vary among groups and were not different from those of controls.

No evidence of antibody production as a result of ASFV infection, as measured with an indirect ELISA procedure, was found in any of the experimentally infected rats, indicating that, despite robust exposure, the virus did not sufficiently stimulate the rat's humoral immune system.

Because little was previously known about infectious doses of ASFV in rodents, and potential clinical signs, some potential limitations of this study include uncertainty surrounding the dose needed for experimental infection, and the length of observation necessary to detect clinical signs. Based on experimental intramuscular infections of pigs in which 0.1 HAD50 units were sufficient to infect healthy 8-week-old pigs (Yamada *et al.*, 2020), and 10 TCID50 units were sufficient to orally infect larger pigs (Niederwerder *et al.*, 2019), investigators postulated that a dose of 10^5 HAD50 units would be sufficient to infect much smaller rodents. Moreover, the work by Yamada *et al.* (2020) showed that experimentally infected pigs showed fevers by 4 days post infection, and clinical signs by 6 days post infection. As a result, investigators postulated that 21 days was sufficient time to allow the development of clinical signs in experimentally infected rodents. Results of this work were also subject to the limitation of the lack of positive susceptible controls (pigs) infected simultaneously as the rodents.

While the lack of infection in wild rodents trapped on ASF-affected farms could potentially be explained by lack of contact with infected pigs, or by the varying infection status on affected farms, attempts to experimentally infect rodents with ASFV were unsuccessful. Inoculated rodents did not show clinical signs of infection, and efforts to detect virus in the organs of inoculated rodents were unsuccessful as well. No evidence of an immune response was present in any of the inoculated rodents either. In total, these results lend evidence that rodents do not serve as competent biological vectors in the transmission of ASFV between pigs and farms. While it is plausible that these animals could serve as mechanical vectors of the virus, this was not demonstrated by the current work either.

Swine producers should still take steps to reduce rodent burdens on their farms because of their well characterized role in the spread of other swine diseases such as leptospirosis and salmonellosis. While rodent control is important overall, it does not appear to play a special role in the ecology of ASFV and its control. Further work to elucidate the role of rodents (and other animals) as mechanical vectors for ASFV may be useful, however.

Conclusions

In this study, experimental inoculation of ASFV into healthy rats did not result in evidence of illness, infection, or immune response. Further work is necessary to demonstrate the competence of rodents as biological vectors for ASFV transmission in swine herds.

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