Senecavirus A

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Acronyms

APHIS: Animal and Plant Health Inspection Service

cELISA: Competitive enzyme-linked immunosorbent assay

CFIA: Canadian Food Inspection Agency

CSHIN: Canadian Swine Health Intelligence Network

Ct: Cycle threshold

DDGS: Dry Distillers' Grains with Solubles

DIVA: Distinguishing infected from vaccinated animals

ELISA: Enzyme-linked immunosorbent assay

EQSP: Équipe québécoise de santé porcine (*Quebec Swine Health Team*)

ETNL: Epidemic Transient Neonatal Losses

FMDV: Foot-and-mouth disease virus

IFA: Indirect Immunofluorescence Antibody Test

ISUVDL: Iowa State University Veterinary Diagnostic Laboratory

MAPAQ: ministère de l'Agriculture, des Pêcheries et de l'Alimentation du Québec (*Quebec Ministry of Agriculture, Fisheries and Food*)

MNVDL: Minnesota Veterinary Diagnostic Laboratory

NVSL: National Veterinary Services Laboratory

OMAFA: Ontario Ministry of Agriculture, Food and Agribusiness

PBS : Phosphate-buffered saline

PCR: Polymerase Chain Reaction

PF: Processing Fluids

PFU: Plaque-forming unit

PRRS: Porcine Reproductive and Respiratory Syndrome

RT-PCR: Reverse Transcription Polymerase Chain Reaction

SHIC: Swine Health Information Center

SVA : Senecavirus A

SVDV: Swine vesicular disease virus

SVEV: Swine vesicular exanthema virus

SVV: Seneca Valley Virus

USDA: United States Department of Agriculture

VLP: Virus-like particles

VSV: Vesicular Stomatitis Virus

WOAH: World Organisation for Animal Health

1. Introduction

Six viruses of type *Picornavirus* were isolated from pigs in the United States between 1988 and 2005, which showed a variety of clinical symptoms (Knowles & Hallenbeck, 2005). Since 1999, neonatal mortality and idiopathic vesicular disease have been reported in Minnesota, USA, a virus of type *Picornavirus* was suspected (Vannucci F. , 2019).

The disease has been detected on rare occasions in Canada. In June 2007 in Manitoba, in pigs sent to the United States for slaughter, the disease was detected in a group of 187 pigs; one animal had a vesicle on its nose, 12 had coalescing reddish erosions on its snout, 25 to 30 percent had foot lesions (coronary band bleaching), and about 80 percent of them had lameness. No mortality was reported in this case. Clinical vesicular lesions were indistinguishable from vesicular disease caused by foot-and-mouth disease virus (FMDV), swine vesicular disease virus (SVDV), vesicular stomatitis virus (VSV) or swine vesicular exanthema virus (SVEV). No positive results were identified by polymerase chain reaction (PCR) tests for these viruses (Pasma, Davidson, & Shaw, 2008). However, Senecavirus A (SVA) has been identified as positive and has been proposed as a causative agent of the disease. The same situation was repeated in New Zealand, Australia, the United Kingdom and Italy (Zhang, et al., 2018).

Since 2014, a sudden increase in outbreaks of vesicular diseases in pigs has appeared in different countries, including Brazil in 2014, the United States and China in 2015, followed by Colombia and Thailand in 2016, Vietnam in 2018 and Mexico and Chile in 2022 (Preis, et al., 2022).

In November 2014, several acute epidemics have been reported in Brazil with three main features: 1) presence of vesicles and erosions on the snout and coronary band in sows; 2) mortality in piglets (30 to 70%) less than four days of age and 3) a self-limiting epidemic lasting one to two weeks (Vannucci, et al., 2015). In several cases, the first observation was significant acute mortalities in newborn piglets. These mortalities have sometimes been associated with lethargy, neurological signs or diarrhea in newborn piglets. This syndrome referred to as *Epidemic Transient Neonatal Losses* or ETNL was first described in Brazil and affects piglets between zero and seven days of age (Swine Health Information Center (SHIC), 2015). It appears suddenly, is self-limiting and causes between 40 and 80% mortality in piglets aged zero to three days and between 20 and 40% mortality in piglets aged four to seven days. Mortalities can occur within five to six hours, and no specific lesions could be identified. The mortality rate returns to normal within four to 10 days. Piglets over one week of age do not appear to be affected. In 50% of cases, piglets show diarrhoea and in 70% of cases, sows show the presence of vesicles that heal in 10 to 15 days. Cases of ETNL have also been reported in the United States, the syndrome has the same clinical signs and characteristics of the disease as those seen in Brazil. SVA was isolated from newborn piglets showing severe neonatal mortality and high morbidity,

followed by vesicular lesions on the snouts and coronary bands of sows and boars (Baker, et al., 2017).

In July 2015, four clinical cases due to SVA in U.S. pigs have been confirmed; three cases in Iowa in show pigs and one case in South Dakota in a finishing barn. Affected animals showed acute lameness, anorexia, lethargy, and transient fever with no associated mortality; they also showed coronary band hyperemia and vesicles, which sometimes progressed to skin ulcers (Singh, Corner, Clark, Sherba, & Fredrickson, 2012; Vannucci, et al., 2015). Small vesicles were also visible on the snout, in the oral cavity, or both; these vesicles variably evolved into ulcers. No specific microscopic lesions, beyond ulcerative lesions, were present in the samples submitted to the Iowa State University Veterinary Diagnostic Laboratory (ISUVDL). Samples of vesicular and blood lesions were collected from all affected animals. They all tested negative for the viruses responsible for vesicular diseases (FMDV, SVDV, SVEV, VSV). No other common swine pathogens, with the exception of SVA, have been detected at ISUVDL (Guo, et al., 2016). In the summer, SVA was associated with lameness and other clinical signs in a dozen U.S. states. SVA is also associated with some cases of neonatal mortality (Canadian Pork Council/Conseil canadien du porc, 2015).

In October 2015, the United States Department of Agriculture (USDA) notified the Canadian Food Inspection Agency (CFIA) that three Canadian-origin sows with vesicular lesions were exported to the United States for immediate slaughter. The CFIA investigated this situation and determined that two assembly yards and three farms in Ontario and Manitoba were involved. Two shipments of hogs from Canada, one from Ontario, via an Ontario assembly yard, and another from Manitoba (sows), via a Manitoba assembly yard, were going to be slaughtered in the United States. Vesicles and signs of lameness were identified at the slaughterhouse. PCR laboratory test results were positive for SVA, however, the pigs' herds of origin tested negative. Pigs at the Ontario assembly yard also tested positive for SVA.

In September 2016, in a shipment of pigs sent to the U.S. for immediate slaughter, 13 animals had vesicular lesions upon arrival at the slaughterhouse. The animals were cull sows from five Quebec farms and eight Ontario farms that had transited through the same assembly site in Ontario. The CFIA's investigation of four farms in Quebec (one farm had no hogs at the time of the investigation) and eight in Ontario confirmed that all these herds were negative for SVA. However, samples taken from pigs at the Ontario assembly yard were positive. Subsequently, over a period of approximately two weeks, 10 trailers of cull pigs were refused at the U.S. border due to the presence of pigs with lesions consistent with reportable vesicular diseases. In each case, the pig lots were quarantined and tested by the CFIA. The results indicated that nine of the 10 trailers contained SVA-infected pigs. During the investigations, it was found that the four main assembly yards for sows and cull

pigs in Ontario were contaminated with SVA or at high risk of being contaminated. This situation has led the assembly yards to take corrective measures in the management of pigs to avoid the shipment of pigs with clinical signs typical of vesicular diseases and, consequently, their refusal by the American authorities.

On June 29, 2019, the Ontario Ministry of Agriculture, Food and Agribusiness (OMAFA) was notified of a confirmed case of SVA in a sow herd in Ontario. The sow herd showed clinical signs of prolonged fasting, increased pre-weaning mortality, and vesicular lesions. The practicing veterinarian of the herd was notified and subsequently contacted the CFIA. On-farm samples collected by the CFIA were negative for foreign animal vesicular diseases but were confirmed positive for SVA.

In previous years in the United States, episodes of SVA were sporadic with two or three cases found in several regions. Today, with an average of 20 to 30 cases per month, seroprevalence studies indicate that SVA is endemic and widespread in the country and may be circulating subclinically in sows and finishing pigs (Houston, et al., 2019). In some years, the incidence increases during the warmer summer and early fall months, depending on the geographic hemisphere (Vannucci F. , 2022).

The week of July 24, 2022, USDA veterinarians rejected shipments of Canadian cull sows from two Manitoba assembly yards due to the presence of skin blisters, a lesion common to SVA and some vesicular foreign animal diseases. Pigs kept at the Brandon and New Bothwell sites were assessed as "high risk" by the USDA due to potential exposure to SVA. These assembly yards are critically important to pork producers across Western Canada, exporting millions of hogs annually to the United States. The CFIA tested samples from rejected shipments that tested negative for foreign vesicular animal disease, while there were positive results for SVA. Through close collaboration between the CFIA, USDA representatives, Manitoba Pork (the exporter) and the U.S. processor, an agreement was reached to resume exports from the assembly yards in September of that year.

In July 2025, the USDA's inspection service (APHIS) temporarily suspended all imports of pigs from certain Ontario assembly yards. This suspension followed several shipments of animals, with vesicular lesions, to American slaughterhouses. The lesions led to investigations by the American authorities and the SVA was diagnosed. In the fall of 2025, USDA APHIS revoked the export eligibility status of four Ontario cull sow assembly yards. These centres received pigs from Quebec, the Maritimes and Ontario. Quebec farms that had sent animals to these assembly yards during the period when lesions were observed had been contacted and screening tests carried out. The results indicated that none of these farms were infected with the SVA virus. Each affected assembly site had to meet USDA requirements, including emptying, cleaning, and disinfecting the site, before it could regain its export eligibility status (Ministère de l'Agriculture, des Pêcheries et de l'Alimentation

du Québec (MAPAQ), 2025; Canadian Swine Health Intelligence Network (CSHIN), 2025).

2. Etiology

2.1 Characteristics

The virus, previously known as the virus of the valley of the Senecas, has been identified in the U.S. pig population since the late 1980s, it was often diagnosed in cases of "idiopathic vesicular disease of pigs" because no cause-and-effect relationship had been proven at that time. The National Veterinary Services Laboratory (NVSL) from the United States has isolated twelve viruses of the *Picornavirus* between 1988 and 2005 in pigs with a variety of clinical signs and from several states across the country (Knowles, et al., 2006). The virus was first discovered in 2002 in a Maryland lab as a cell culture contaminant in PER.C6 cells and has been named Seneca Valley virus-001 (SVV-001). The SVV-001 virus is believed to have been introduced via bovine serum or porcine trypsin during cell culture (Hales, et al., 2008).

Sequencing highlighted the close relationship of these isolates with SVV-001 and neutralizing antibodies found in pigs, signaling pig as a natural host. Two of these isolates were used to inoculate pigs, but they did not develop any specific clinical diseases (Knowles, et al., 2006). Following this discovery, the virus was characterized and classified in the family of *Picornaviridae*. This family also contains foot-and-mouth disease virus and swine vesicular disease virus. A picornavirus is a small, single-stranded RNA virus, not enveloped with a capsid. A single serotype, *Senecavirus A*, is classified in the new genus *Senecavirus,* that is closely related to gender *Cardiovirus* of the family of *Pircornaviridae* (Hales, et al., 2008).

Gao et al. processed representative SVA sequences from the GenBank database, as well as 10 newly isolated SVA strains from field samples collected in their laboratory to explore the origins, population characteristics, and modes of transmission of SVA. The SVA strains were first systematically divided into eight clades, including clade I to VII and the ancestor clade, based on phylogenetic inference of maximum likelihood (Table 3) (Gao, et al., 2022).

TABLE 3 Sequence identities (%) of different SVA clades.

	Clade ancestor	Clade I	Clade II	Clade III	Clade IV	Clade V	Clade VI	Clade VII
Clade ancestor								
Clade I	92.74							
Clade II	92.71	97.67						
Clade III	93.21	97.61	97.47					
Clade IV	93.22	97.23	97.18	97.55				
Clade V	93.88	96.05	96.05	96.46	96.66			
Clade VI	92.96	98.13	98.10	97.94	97.59	96.37		
Clade VII	93.21	98.06	98.23	98.08	97.73	96.58	98.63	

Phylogeographic and phylodynamic analysis in the Bayesian statistical framework revealed that SVA originated in the United States in the 1980s and then spread to different countries and regions. Their analysis of viral transmission routes also revealed its historical spread from the United States and the risk of global prevalence of the virus. Overall, this study provided a comprehensive assessment of the phylogenetic characteristics, origins, history, and geographic evolution of SVA globally, which provided a better understanding of the development of effective disease management strategies (Figure 2) (Gao, et al., 2022).

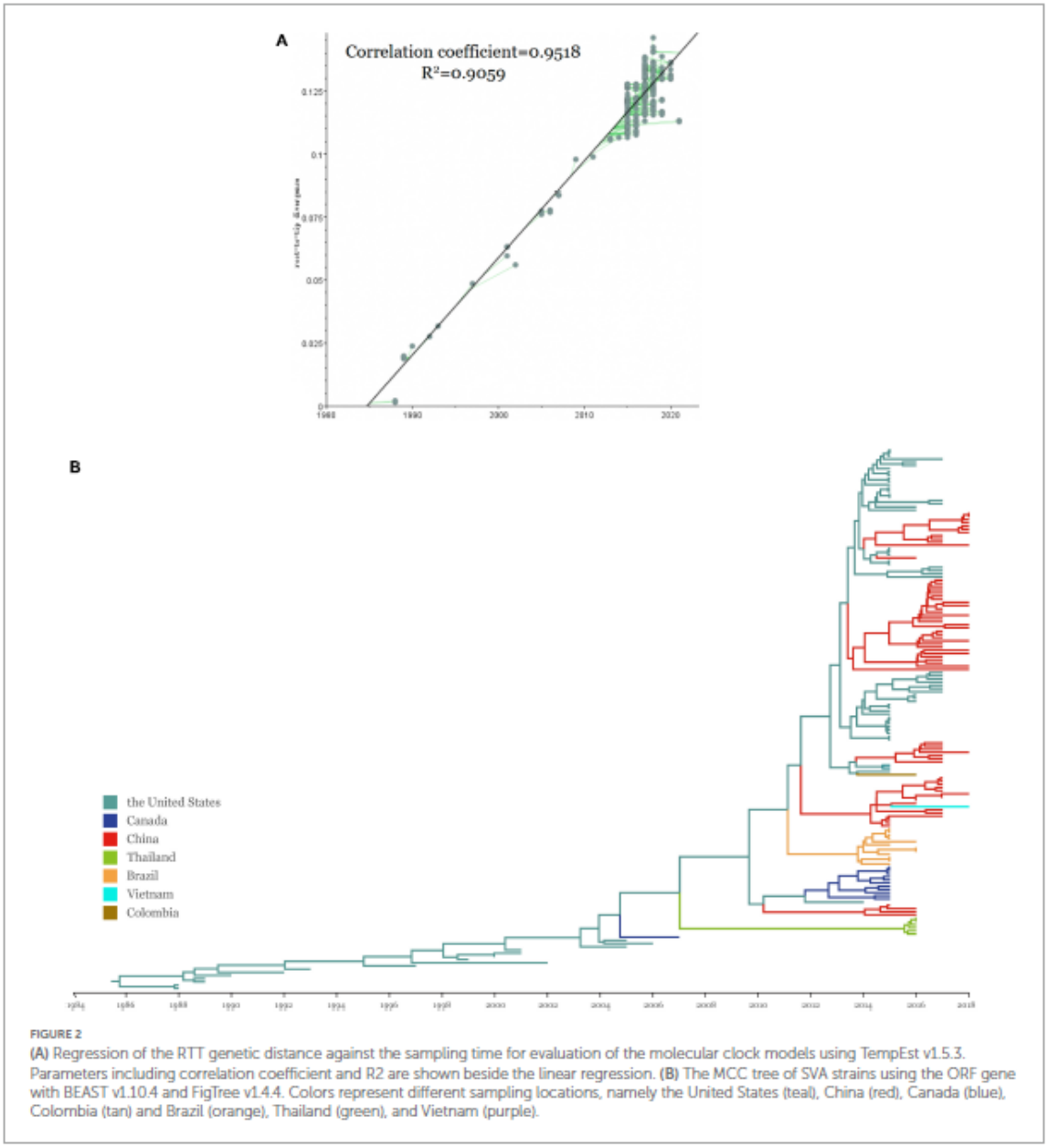


FIGURE 2
(A) Regression of the RTT genetic distance against the sampling time for evaluation of the molecular clock models using TempEst v1.5.3. Parameters including correlation coefficient and R2 are shown beside the linear regression. **(B)** The MCC tree of SVA strains using the ORF gene with BEAST v1.10.4 and FigTree v1.4.4. Colors represent different sampling locations, namely the United States (teal), China (red), Canada (blue), Colombia (tan) and Brazil (orange), Thailand (green), and Vietnam (purple).

The historical distribution of the SVA was determined and visualized via spatiotemporal dynamic maps. Consistent with the time points of dispersion patterns, the U.S. was the source of SVA, which then spread to Canada in the early 2000s. Transmission to South America, China and Southeast Asia then began in the 2010s. Since 2015, the United States and China have maintained a relatively large SVA infected population size and have also become the main SVA epidemic areas (Figure 4A). Overall, the results showed that the United States acted as the primary source of SVA and transmission from the United States to Canada, Brazil, Colombia, China, and Thailand (Figure 4B) (Gao, et al., 2022).

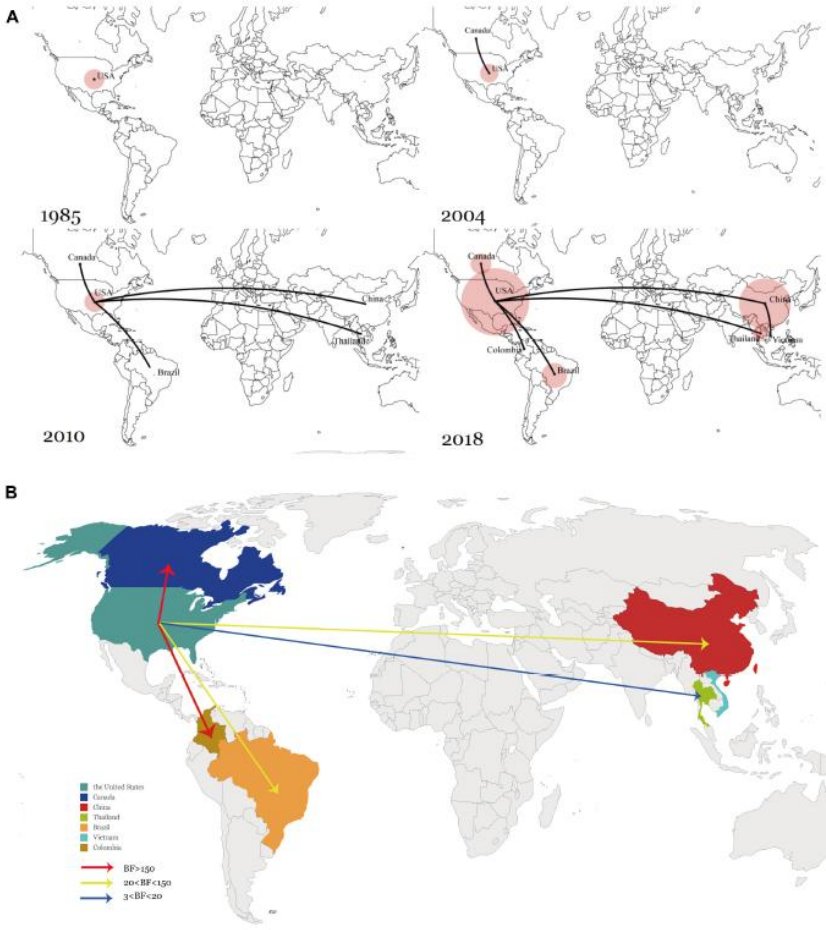


FIGURE 4
(A) Spatiotemporal dynamics of SVA among different regions. **(B)** Global transmission links of SVA tested by Bayes Factor for significant non-zero rates supported by the BSSVS. Line colors represent the relative strength by which the rates are supported: very strong (BF > 150, red) and strong (20 < BF < 150, yellow), and positive (BF > 3). Line directions indicate the direction of transmission.

2.2 Strain Variability

Phylogenetic analyses based on complete genome sequences show a phylogenetic separation between historical isolates (1988-2002) and contemporary isolates (2007-2015). Similarly, contemporary isolates from different geographic locations (United States, Brazil, and China) form separate phylogenetic groups. These data suggest a closer genetic relationship between contemporary isolates (USA, Brazil and China) compared to the links of these viruses to historical isolates. The first complete genome sequence was performed on the SVV-001 viral isolate (Hales, et al., 2008). The genome of the SVA virus appears to be more stable than those of other members of the *Pircornaviridae.* Analyses of viruses isolated by NVSL suggest the presence of a common ancestor for them in the last 30 to 40 years and a recent introduction into pig herds in the United States (Koppers-Lalic & Hoeben, 2011).

Zhang et al. analyzed the sequence of three SVA who were isolated during 2015 and compared them to each other, the SVV-001 virus sequence, the Canadian isolate and the Brazilian isolates, they found a percent homology of more than 94% between the different strains (Zhang, et al., 2015). Another study also concludes that Brazilian strains of SVA have a common ancestor with a Canadian strain and American strains encountered in the past. The sequence between the strains is more than 95% similar (Vannucci, et al., 2015). Leme et al. also indicate that the Brazilian SVA strains from the early 2015 outbreak, where piglets were affected by ETNL syndrome, are closely related to the SVA strains present in North America (Leme, et al., 2015).

As of 2015, the complete sequence of nine SVA genomes is available and the strains come from Canada, the United States, Brazil and China. The sequences of the nine genomes > 93.78% identity for nucleotides and 97.71% identity for amino acids. The lowest percentage of identity is between SVV-001 and the U.S. strains isolated in 2015 for both nucleotides and amino acids. Phylogenetic analyses in 2016 of the VP1 gene indicated that there are three clades. Clade I includes SVV-001. Clade II includes American SVA strains identified between 1988 and 1997. Clade III includes strains from Brazil, Canada, China and the United States identified between 2001 and 2015 (Segalés, Barcellos, Alfieri, Burrough, & Marthaler, 2016).

The strain isolated from Colombia was compared to strains from the United States, Brazil, China, Canada and SVV-001. Sequence alignment shows that the Colombian strain has a higher percentage of nucleotide identity with the recent U.S. strains (98.50% - 98.97%) and diverges the most from the SVV-001 strain (93.89%). In the phylogenetic tree, the Columbia strain shares a common ancestor with the isolated strain in North Carolina. The American cluster containing the Colombian strain shares a common ancestor with the Brazilian strains (Sun, Vannucci, Kuntson, Corzo, & Marthaler, 2017).

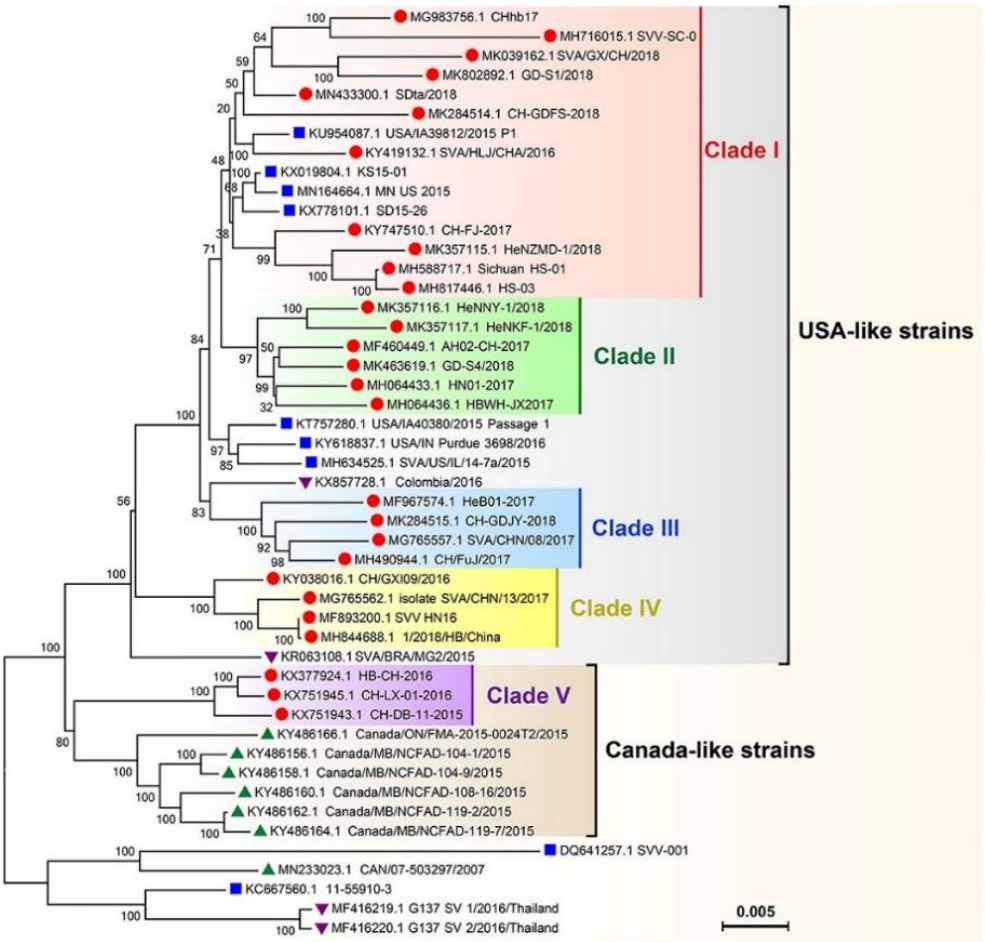
Diel et al. compared the genome of 17 SVA isolates from the United States and five isolates from Brazil to the other SVA sequences available in Genbank. Genome sequence comparisons reveal that contemporary isolates from the United States share 91-93% nucleotide identities with SVV-001 and with the isolate obtained from Canada in the 2007 case (SVA-11-55910-3). These same contemporary isolates from the United States share 98-99% nucleotide identity with other contemporary U.S. isolates, 95-97% nucleotide identity with contemporary isolates from Brazil, and 94-96% nucleotide identity with the recently identified China isolate (Diel, 2017).

One isolate from China shows a high degree of similarity to eight strains from Canada, Brazil and the United States (94.4-97.1%) (Wu, et al., 2016). A phylogenetic analysis reveals that the virus evolved into five genetic clades in China at a rapid rate. Clades I, II, III and IV belong to the USA-like cluster, and only clade V belongs to the Canada-type cluster, suggesting that USA-type strains are predominant in China. They report that the

new viruses have conferred subclinical infection of animals in some regions (Liu, Wang, Huang, Wang, & Shan, 2020).

Later, phylogenetic analysis of the global SVA genomes revealed that of the two main evolutionary groups, the USA and Canada-like strain groups and all isolates from China could be mainly grouped into five genetic branches, clades I, II, III, IV and V. They built a phylogenetic tree (Figure 1), whereby a portion of Chinese isolates were compared to exotic isolates to show their evolutionary relationship, suggesting that USA-like strains are predominant in China (Wang, et al., 2019).

Figure 1. Phylogenetic analysis on the genomes of SVA isolates.



The numbers indicated the bootstrap value (1,000 repetitions).

SVA isolates were tagged from China“ ●”, from the United States“■”, Canada “ ▲” and other countries “▼” (Wang, et al., 2019).

The closest phylogenetic relationship of the Chilean strain is with viruses collected from pigs in California in 2017. The direct cause of the introduction of SVA has not yet been identified; however, phylogenetic analyses suggest that the United States is the most likely source (Bennett, et al., 2022). Whole genome sequencing used to characterize SVA in the five cases identified in the UK between June and September 2022 shows that they form

two distinct groups, sharing a common ancestor with a strain from the United States identified as SVV/USA/TN/NADC6/2020. (Animal and Plant Health Agency (APHA), 2022)

SVA isolates from pigs with vesicular lesions in Canada from 2015 to 2023 were sequenced and phylogeographic analysis was performed from the complete genome sequences. Phylogenetic analysis demonstrated that SVA can be classified into two main groups, clade I (pre-2007 strains) and clade II (post-2007 strains), although further attribution to clade II is possible based on the genetic divergence of lineages forming distinct monophyletic groups. It is important to note that the selective force analysis showed that clade II evolved under natural selection compared to clade I (Wu, et al., 2022). The genetic structure of the SVA phylogeny is strongly influenced and directed by propagation events that lead to the formation and diversification of distinct phylogenetic clades. Existing sequence data suggest that SVA originated in Iowa in the Midwestern United States. Clade 1 viruses are likely extinct as they are no longer detected in the field. Clade II first inferred that the spread occurred from the United States to Canada in 2007. The cross-border spread of SVA between the U.S. and Canada has since occurred several times due to the highly integrated hog industries in North America. Phylogeographic data suggest that SVA spread to different regions, including from the United States to Brazil, Colombia, Chile, and China on 10 separate occasions between 2008 and 2019, from China to Vietnam, but also from Canada to Thailand, India, and Mexico (Hole, et al., 2025).

As the only member of the genus Senecavirus within the Picornaviridae family, the SVA has posed a huge challenge to the global swine industry. In a previous study, a strain of SVA was isolated from a buffalo with mouth ulcers. To systematically assess its pathogenicity, this study compared the results of piglets and buffaloes artificially infected with different viral doses of the original SVA strain of buffalo (SVA/GD/China/2018). These results indicated that vesicular diseases can occur in infected piglets and buffaloes. Severe clinical symptoms were observed in piglets and buffaloes with an inoculation of $10^{5,0}$ at 50% of the infectious dose in tissue culture (TCID 50/mL). SVA antigen was also detected in lung tissue, chin blister lesion tissue, nasolabial piglet tissue, and blistered upper lip tissue of buffaloes. This study demonstrated that the original SVA strain of buffalo was pathogenic to piglets and buffaloes, revealing the possibility of inter-species transmission of SVA between pigs and buffaloes (Zhou, et al., 2025).

3. Virus Characteristics

3.1 Survival

Environmental survival for SVA is not yet known. Previously reported outbreaks of vesicular disease caused by SVA appear to have a seasonal pattern as the majority of cases

occurred between spring and fall (Pasma, Davidson, & Shaw, 2008). In 2015, several cases occurred during the warm season in the United States and several cases occurred in Brazil where warm temperatures are present. In 2020, the virus was isolated from sow slaughterhouses in the United States, with positive samples for SVA most frequently found in summer (78.3% June-September, compared to 59.4% October-December), with a peak of 85% in August (Hoffman, et al., 2022).

SVA can also be detected in the feces of infected mice after about two weeks (Joshi, et al., 2016) and it can also be detected in the feces of uninfected mice after exposure to infected mice (Houston, Temeeyasen, & Piñeyro, 2020).

A study aimed to model the survival of foreign animal disease-causing viruses in feed ingredients for pigs travelling from Asia to the United States. Several pathogenic viruses have been identified as having a significant risk to the U.S. pork industry. Surrogate viruses were used to experiment because it was impossible to work with exotic viruses without controlled conditions. The foot-and-mouth disease virus was substituted by the SVA in this study. The experimental design is the same as the one used to assess the risk of contamination of pig feed ingredients with porcine epidemic diarrhea virus during cross-border crossings (Dee, et al., 2017).

The next study was designed to evaluate the survival of the same important viral pathogens in feed ingredients imported daily into the United States (conventional soybeans, soybean meal, distillers dried grains with solubles (DDGS), lysine, choline, vitamin D, dry and wet pet food, feed) and in pork sausage casings, under simulated cross-border conditions. The results showed a survival of the SVA during the 37 days of shipment, from China to the United States and that the highest stability was observed for the SVA, as the viable virus was recovered from 10 of the 11 ingredients tested. The positive control, from the SVA alone without the presence of a food matrix, did not survive (Dee, et al., 2018).

Caserta et al. determined the rate of decomposition of SVA in swine feed ingredients. At the three temperatures tested (4, 15 and 30°C) for a period of 91 days, DDGS and soybean meal provided the most stable supports for SVA, resulting in half-lives of 25.6 and 9.8 days, respectively. At 30°C, SVA was completely inactivated in all ingredients and in the control sample, which did not contain food. Although the ineffectiveness of the virus was lost, the viral RNA remained stable and at constant levels throughout the experimental period. In addition, they evaluated the ability of the SVA to infect pigs via feed ingestion in weaned pigs three weeks of age. The results demonstrate that the food can prolong the survival of SVA, protecting the virus from decomposition and that eating contaminated food can lead to SVA infection (Caserta, et al., 2022).

3.2 Disinfection

Since vesicular diseases are clinically indistinguishable, the SHIC recommends following disinfection protocols for FMD, even if SVA is suspected. This includes the use of sodium hydroxide, sodium carbonate, 0.2% citric acid, aldehydes, and oxidizing disinfectants, including sodium hypochlorite (Swine Health Information Center (SHIC), 2017).

Dr. Goyal's team at the University of Minnesota evaluated the effectiveness of three commercial disinfectants against SVA on five different surfaces (cement, rubber, plastic, stainless steel and aluminum) at ~25°C and 4°C (Singh, et al., 2017). The disinfectants tested in this study are household bleach (chlorine), Tek-Trol® (phenol) and Synergize® (quaternary ammonium and aldhehyde). At 25°C (77°F), bleach (5.25%, 1:20 dilution) is highly effective against SVA on aluminum, rubber, plastic, stainless steel, and cement after a contact time of 10 to 15 minutes. At 4°C (39°F), bleach inactivates the SVA in five to 15 minutes on all surfaces; disinfection is a little less effective for rubber but still exceeds 99.9%. Tek-Trol® (1:250) was the disinfectant with the least efficacy and Synergize® (1:256) showed moderate results (Table 1) (Singh, et al., 2017).

Table 1: Efficacy of three commercial disinfectants against *Senecavirus A* (Singh, et al., 2017).

Disinfectant	Temperature	Surface	Contact Time	% reduction
Bleach	25°C	Aluminium	10 to 15 minutes	≥99.99 %
	25°C	Plastic	10 to 15 minutes	≥99.99 %
	25°C	Rubber	15 minutes	≥99.99 %
	25°C	Stainless steel	15 minutes	99.97 %
	25°C	Cement	15 minutes	99.98 %
	4°C	Stainless steel	5 to 15 minutes	≥99.99 %
	4°C	Plastic	10 to 15 minutes	≥99.99 %
	4°C		15 minutes	≥99.99 %
	4°C	Cement	15 minutes	≥99.99 %
	4°C	Rubber	15 minutes	99.91 %
Synergize®	4°C and 25°C	All surfaces	60 minutes	93.54 to 99.81%
Tek-Troll®	4°C	Stainless steel	60 minutes	82.41 %

A study by Hole et al. evaluated a disinfectant based on accelerated hydrogen peroxide® (AHP®) (Prevail® concentrate; Virox® Technologies Inc.) against foreign animal vesicular disease pathogens such as FMDV, SVDV and SVA. AHP® disinfectant is effective against SVA (wet film) at a 1:20 dilution and a contact time of 10 minutes resulting in a > 4-log decrease in SVA virus titer. The reconstituted accelerated hydrogen peroxide® disinfectant is stable for at least six weeks at room temperature when stored in a sealed bottle. The Virkon® disinfectant used as a positive control at a concentration of

1% with a contact time of 10 minutes is effective against SVA (wet and dry film) (Hole, et al., 2016).

More research needs to be done on disinfection protocols for SVA to identify disinfectants that are effective against this virus. The EQSP document "*Useful information on disinfectants registered in Canada and known to be effective against porcine epidemic diarrhea (PED), porcine Deltacoronavirus (PVC) and Senecavirus A (SVA) viruses in pigs*" lists the disinfectants that can be considered effective against SVA and foot-and-mouth disease (Équipe québécoise de santé porcine (EQSP), 2022).

4. Epidemiology

4.1 Species Affected

A serological study demonstrated the presence of antibodies against SVA in pigs, cattle and wild mice, but rarely in humans (1/110 samples (0.9%)). A similar study was also performed in four primate species, and none had the presence of antibodies against SVA (Knowles, et al., 2006). Another study, in mice, demonstrated no horizontal transmission between infected and naïve mice over a period of 30 days (Koppers-Lalic & Hoeben, 2011). No studies have reported clinical signs in cattle, mice or humans.

4.2 Zoonotic Potential

Although serological investigations have revealed the presence of SVA-specific antibodies in pigs, cattle, and mice (Pasma, Davidson, & Shaw, 2008) (Koppers-Lalic & Hoeben, 2011), the virus does not affect humans and is not pathogenic to normal human cells. It is not a zoonotic disease and does not pose a food safety risk.

4.3 Geographic Distribution

SVA has been reported in North America, Australia, New Zealand, Italy, Brazil, Colombia, Chile and recently the United Kingdom. The virus is believed to be spreading across the United States (Hales, et al., 2008). According to a retrospective study, seven SVA isolates have already been reported from California, Illinois, Iowa, Louisiana, Minnesota, New Jersey, and North Carolina in pigs with various clinical signs between 1988 and 2001 (Knowles, et al., 2006). Outbreaks of idiopathic vesicular diseases, caused by SVA, have also been reported in Florida, Iowa and Indiana.

In a first case of SVA reported in China in the summer of 2015 (Wu, et al., 2022), the virus was detected in sows with clinical signs of vesicular disease in combination with neonatal mortality. In February 2016, a 300-sow farrowing unit in Colombia reported the appearance of vesicles on the snout and coronary bands with lameness in 4 sows located in the farrowing barn, while the piglets showed no clinical signs of disease. Samples of epithelial tissue, blood, and vesicular fluid tested negative for FMD, but positive for SVA. This was

the first case of SVA infection reported in Colombia (Sun, Vannucci, Kuntson, Corzo, & Marthaler, 2017).

Chile was historically considered free of swine vesicular diseases, including SVA. In April 2022, a veterinarian reported sows with suspicious signs of vesicular disease. SVA was confirmed and other vesicular diseases were excluded. An epidemiological investigation and phylogenetic analyses were carried out to identify the origin and extent of the epidemic. They sampled 44 pig farms with feces, oral fluids, processing fluids (PFs), fresh semen, environmental samples, and lesion tissues for real-time RT-PCR detection. In June 2022, SVA was detected in 16 of the 44 farms, all epidemiologically linked to the initial farm (Bennett, et al., 2022).

In January 2023, the UK's Chief Veterinary Officer confirmed that all five cases of vesicular disease in pigs identified on farms in England between June and September 2022 were SVA. This is the first case reported in Europe. Even though SVA is not a reportable disease in the UK, nor is it a disease listed by the World Organisation for Animal Health (WOAH), it is considered of high importance due to the clinical signs that resemble reportable vesicular diseases, particularly foot-and-mouth disease (UK Government - Department for Environment, Food & Rural Affairs, 2023).

5. Transmission

The route of transmission of the virus is not yet well understood. Many have speculated that this virus could be contracted during transport. Some researchers indicate that the means of transmission of the virus are similar to those of FMD: direct contact with infected animals, by mechanical vectors or by aerosol.

Montiel et al. reported vesicular disease in nine-week-old pigs under experimental conditions using a strain of SVA from a commercial pig farm in South Dakota, USA. Pigs, which received a dose of $5X10^7$ Plaque-forming Unit (PFU)/animal of SVA intranasally, developed vesicular and skin lesions on the legs, as well as on the snout, and lameness was observed in some animals. In addition, viral RNA was detected in the serum of all pigs three days post-infection and in vesicular lesions five days post-infection (Montiel, et al., 2016).

Cases reported in the United States from previous studies with the SVA prototype (SVV-001) failed to induce vesicular disease in pigs (Fernandes, et al., 2018). However, experimental studies with SVA isolates from 2015 resulted in vesicular disease in pigs, indicating that a more virulent phenotype has emerged (Joshi, et al., 2016). Isolates of SVA 2016, obtained from the tonsil of a pig naturally infected in Ontario, were experimentally

inoculated into nine-week-old pigs and all pigs developed clinical signs (Hole, Ambagala, & Nfon, 2019).

An investigative study was conducted in herds with and without vesicular disease and neonatal mortality in the United States and Brazil. SVA was detected by PCR and isolated in cell culture from clinical samples (oral swabs, vesicular fluids, coronary hoof band in sows, tonsils, intestinal homogenate and coronary hoof band in piglets) and environmental samples including, dust from outlet fans, outdoor soil, the tractor shovel to transport mortalities, interior corridors, mouse bait boxes outside the building, mouse feces and the small mouse intestine. The RNA of the virus was also detected by PCR in a homogenate of whole flies on infected farms in the United States and Brazil and on an uninfected farm in the United States 300 metres from the infected farm. The detection of SVA in mouse and housefly samples and the recovery of viable viruses from mouse feces and small intestine suggest that these pests may play a role in the epidemiology of SVA (Joshi, et al., 2016).

Epidemiological investigations conducted on livestock farms affected by SVA suggested that workers from contaminated farms, trailers or deadstock handling equipment were likely routes for the introduction of the virus (Baker, et al., 2017).

Persistently infected carrier animals also appear to play an important role in the epidemiology of *Picornaviruses*, serving as a source of infection for susceptible animals. Results on sows that have been inoculated orally with SVA show that transport stress leads to a slight increase in disease severity after infection.

Maggioli et al. demonstrated that animals with persistent infection, transport, immunosuppression, and parturition stressors did not result in clinical disease, but they detected intermittent viremia and virus shedding up to day 60 after infection. Real-time PCR and in situ hybridization (ISH) tests confirmed that the amygdala hosts the SVA RNA during the persistent phase of infection. Double-stranded RNA (dsRNA)-specific Indirect Immunofluorescence Antibody (IFA) test demonstrated the presence of double-stranded viral RNA in tonsillar cells. Infectious SVA was isolated from the amygdala of two animals on day 60 post-infection, confirming the presence of carrier animals after SVA infection. These results were supported by the fact that piglets born to persistently infected sows were infected with SVA (11/44), demonstrating successful transmission of the virus from carrier sows to piglets. These results confirm the establishment of persistent SVA infection and suggest that persistently infected pigs may function as reservoirs for SVA (Maggioli, et al., 2019).

A scientific report describes the prolonged and constant excretion of SVA in the semen of two boars and the persistence of SVA in the tonsils and testes of three boars. Two boars were infected on a Minnesota sow farm in 2017 and they excreted SVA RNA in semen for

more than three months after the outbreak began on the farm. The third boar infected with SVA was identified on an Indiana sow farm in 2020. They detected SVA in the testes and tonsils by PCR, with lower Cycle Threshold (Ct) values obtained for the testes than for the tonsils (Sturos, et al., 2022).

Studies have shown that animals infected with SVA appear to develop short-term viremia for up to 10 days after infection and to excrete the virus for up to 28 days after infection in oronasal secretions and feces. Thereafter, viremia lasts about 10 to 14 days and infected individuals present clinical symptoms for two to 10 days after experimental oral-nasal inoculation (Joshi, et al., 2016). One study demonstrated that viremia can last more than 14 days when pigs are exposed to high doses of the virus (Zhang, et al., 2021), but viremia is not developed in all pigs (Buckley & Lager, 2021).

The detection of the virus in the tissues of one- and two-day-old piglets suggests that there is vertical transmission from the sow. Sows transmit to farrowed piglets more than 45 days after resolution of clinical signs (Buckley & Lager, 2021).

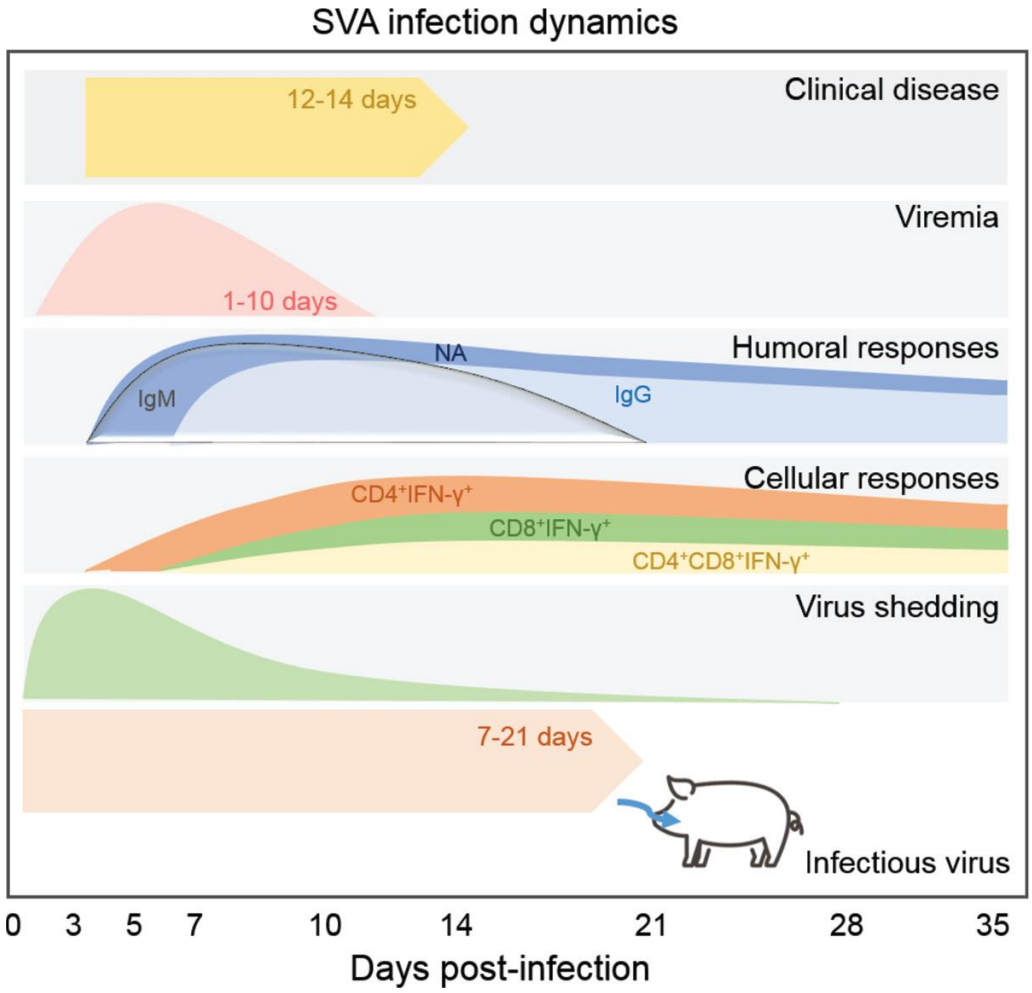
Environmental sampling to determine the level of SVA contamination is most often tested by PCR. Buckley and Lager found that the lowest infectious dose of a 2011 SVA isolate after intranasal inoculation in finishing pigs and oral inoculation in newborns had a Ct value of 25.6 and 29.7, respectively. Inoculum Ct values of 32.6 for finishing pigs and 33.2 for newborns were not able to infect pigs. Environmental samples with Ct values of approximately 32.0 or higher may not pose a high risk of infecting pigs and spreading SVA (Buckley & Lager, 2022).

6. Infection in pigs

6.1 Pathogenesis

Magglioli et al. studied the dynamics of SVA infection in pigs. This figure presents highlights from this and previous studies on the infection and pathogenesis of SVA. After a short incubation period (three to five days), animals infected with SVA show clinical signs characterized by lethargy and lameness. Vesicular lesions develop on the snout or feet around days 3 to 5 post-infection (p.i.) and usually last 10 to 12 days (14 days p.i.). Short-lived viremia follows SVA infection, with viral RNA detected in serum between days 1 and 10 p.i. A robust neutralizing antibody response is detected around day 4 or 5 p.i., with high neutralizing antibody titers detected up to 35 to 38 p.i. days. Notably, the neutralizing antibody response is parallel and correlated with decreased viremia levels. SVA-specific T cell responses are characterized by an increased frequency of αß T cells, especially CD4 T cells^{+}, producing y-NFI. These lymphocytes are detected for the first time around day 7 p.i. and their frequency increases until day 14 p.i. The frequency of CD8 T cells^{+} and doubly positive CD8+ and CD4+ (effector/memory T cells) expressing IFN-y

or proliferating in response to SVA increases after day 10 p.i. Recall stimulation of T cells after disease resolution at day 35 p.i. showed an increase in CD4 T cell frequency^{+}, SVA-specific CD8+ and CD8+ CD8+ were doubly positive at the time, suggesting the effective induction of memory T cell populations. Shedding of the virus in feces and nasal and oral secretions is detected up to 21 to 28 days p.i. The infectious virus was isolated from nasal secretions, feces, and oral secretions up to days 7, 14, and 21 days p.i., respectively (Maggioli, et al., 2018; Joshi, et al., 2016; Montiel, et al., 2016; Yang, van Bruggen, & Xu, 2012).



Pathogenesis in pigs is not yet fully elucidated, but it may be related to the virulence of the virus and the age of the pigs. Morbidity and mortality of the disease vary according to the category of pigs. On a naïve farm, morbidity can vary between 4 and 70%, depending on the symptoms and the age of the pigs. In weaned piglets, morbidity can be between 0.5 and 5% and between 5 and 30% in fattening pigs and breeding pigs, but it can vary depending on the geographical location and origin of the animals (Leme, Alfieri, & Alfieri, 2017). Morbidity of 70-90% has been reported in sows (Baker, et al., 2017). However, mortality

in these categories is close to 0.2% and, usually, outbreaks are self-limiting and have a duration of 10 to 15 days (Leme, Alfieri, & Alfieri, 2017).

In newborn pigs, morbidity and mortality rates are considerably higher, especially in piglets one to four days old, with morbidity rates of up to 70%, but mortality rates range from 15 to 30% (Leme, Alfieri, & Alfieri, 2017).

6.1.2 *In piglets*

- SVA has been detected in the urinary epithelium, suggesting that urine may be a source of contamination on farms infected with this virus (Leme, Oliveira, Alfieri, Headley, & Alfieri, 2016);
- In piglets from sows with vesicular lesions, 30% did not develop viremia. Viremia was detected in 21% of weaned piglets, 7% at six weeks and 3% at nine weeks, with a mean Ct of 35.7 to 38.0 (Tousignant, et al., 2017);
- The virus was present in tonsil and faecal swabs up to six weeks post-weaning, 2% of piglets did not shed or only tested positive (Tousignant, et al., 2017).

6.1.3 In growing pigs

In eight 15-week-old pigs that were inoculated via the oral-nasal route, Joshi et al., described that:

- The virus was detected in serum between three and 10 days post-infection (a.i.), but mainly at three days;
- No viral RNA was detected in serum at 10 days p.i.;
- Inoculated pigs showed lameness and lethargy for four days p.i. and these signs persisted for two to 10 days;
- Vesicular lesions were first observed on the legs or snout for four days p.i. (six pigs out of eight);
- Erythema of the skin that has progressed and developed into vesicles 0.5 to three cm in diameter;
- The vesicles ruptured five to six days p.i. and left erosion on the skin and, finally, a crust eight to nine days p.i.;
- The lesions are completely healed 12 to 16 days p.i.;
- Infected pigs developed a short viremia and the virus was excreted in the oronasal secretions for 28 days p.i.;
- Excretion in the faeces occurred between three and 14 days p.i., but only a few animals shed the virus up to 28 days p.i. (Joshi, et al., 2016).

Rademacher et al., found viral RNA at a low level (20-40% positive) in tonsils, rectal swabs, and serum up to three weeks post-infection. SVA was isolated from less than 10% of piglets in the first two weeks, but all piglets were negative by the third week

(Rademacher, 2016). These results suggest that SVA is a short-term risk for other herds and the risk of SVA transmission is lower after 30 days.

Zhang et al. inoculated five- and six-week-old pigs via the nose:

- they detected the virus in the blood between three and 14 days after inoculation;
- qRT-PCR analysis showed that SVA was detected in the heart, liver, spleen, lungs, kidneys, submandibular and inguinal lymph nodes, intestine, tongue, tonsils, and hooves with blisters. The highest viral loads were in the hooves with blisters (at approximately 105 copies/µl) and in the submandibular lymph node, inguinal lymph node and tonsils (> 104 copies/µl) (Zhang, et al., 2021).

Transported pigs manifest lesions more rapidly, about 24 h before, than pigs that were not transported, but the shedding dynamics and neutralizing antibody response is similar for both groups (Buckley & Lager, 2021).

6.1.4 In sows

In a longitudinal study of SVA shedding in sows and piglets (Tousignant, et al., 2017), they found that:

- in the first week post-infection, 97% (n=33) 34 of the sows tested had a positive result for laryngeal specimens with Ct ranging from 20 to 27 (mean=24.8);
- 91% of rectal swabs collected from sows were positive in the first week post-infection, but none in six to nine weeks post-infection;
- only 20% (n=7) of sows showed viremia one week post-infection (Ct between 17 and 36, mean=33.1), none after two weeks post-infection, a few sows individually had suspicious and positive results at three and nine weeks post-infection and 32% (n=11) did not have viremia;
- Most lesions were healed by two weeks post-infection and lesions were no longer present by three weeks post-infection.

Fresh vesicular lesions on sows showed the highest amounts of SVA; however, the lesions were only present for a short period of about two weeks. The Ct obtained by aspiration of fluid from the vesicular lesions ranged from 12 to 19 (mean = 16) and the Ct obtained from laryngeal samples one to two weeks post-infection were of similar values.

Rademacher et al. found small amounts of viral RNA in tonsils and rectal swabs up to six weeks post-infection; viral RNA could not be detected in serum until three weeks post-infection.

In some cases, only five to 10% of the herd is infected and recovery is seen in less than 10 days. In other cases, up to 80% of the herd shows a positive result and the animals need about 21 days to recover (Fernandez, 2016) (USDA-APHIS, 2015).

A study was conducted to document the pathogenesis of SVA infection in sows and their offspring. Ten sows were inoculated intranasally late in gestation (n = 5) or within fourteen days of birth (n = 5). Each sow replicated the SVA after intranasal inoculation, but only one in ten sows developed a vesicular lesion on the snout. Signs of transplacental infection were observed in two litters and two additional litters were infected after farrowing in five litters of inoculated sows in late gestation. No clinical signs were observed in infected newborns. Similarly, no clinical signs were observed in the other five litters inoculated after farrowing, although each piglet reproduced the inoculated virus. In this study, experimental SVA inoculation did not result in neonatal mortality contrary to field observations; however, it has shed light on the pathogenesis of the virus, the transmission of SVA between sows and their offspring, as well as the host immune response that can help shape control measures in the field (Kim, et al., 2024).

6.2 Clinical Signs

Not all animals with lesions test positive for SVA. According to epidemiological investigations carried out by a team of American researchers (Holtkamp, 2016) (Baker, et al., 2017) in a series of cases of SVA in sow herds, there does not seem to be a typical clinical presentation (Holtkamp, 2016).

The clinical signs observed in pigs infected with SVA cannot be clinically distinguished from those caused by FMDV or other vesicular disease. The researchers observed that clinical signs and vesicular lesions are transient, i.e. their diagnostic value decreases rapidly over time (Gimenez-Lirola, et al., 2016).

6.2.1 Clinical signs in neonates (< 7 days) or " Epidemic Transient Neonatal Losses" (ETNL)

SVA is also associated with sudden mortality, severe and sometimes fatal diarrhea, dehydration and lethargy in newborn piglets born to healthy sows showing clinical signs of SVA or recovering from SVA infection. Mortality in piglets five to seven days of age is high, but lower in this age group (Segalés, Barcellos, Alfieri, Burrough, & Marthaler, 2016).

The main signs are:

- infection occurs shortly after birth (five to six hours of age);
- diarrhea may be present (differential diagnoses: DEP, GET, DCVP, PRRS, *E. coli*, *Rotavirus* and *Clostridium spp.*);
- nonspecific clinical signs, including weakness, lethargy, growth retardation, salivation, rash, diarrhea, and sudden death;
- neurological signs may present alone in 24% of piglets (Oliveira, et al., 2017);
- sudden increase in pre-weaning mortality rate (30 to 70%), piglets less than three days old have higher mortality (40 to 80%), piglets four to seven days old have

lower mortality (0 to 30%) and return to normal in two to three weeks (Leme, Alfieri, & Alfieri, 2017; Segalés, Barcellos, Alfieri, Burrough, & Marthaler, 2016);
- these clinical signs persist for three to 10 days before disappearing in surviving piglets;
- piglets without clinical signs may be carriers of the virus (Linhares, 2015);
- an increase weaning of serum-positive piglets for SVA nearly three weeks after the virus has been eliminated from the serum of most piglets (Buckley & Lager, 2021).

6.2.2 Clinical signs in growing pigs

- development of vesicular lesions in the coronary bands between three and six days, but may be within the first 48 hours, and their disappearance within seven to 14 days, followed by ulcerations and erosions on the snout, nostrils, oral mucous membranes, tongue, skin and limbs (most often in the forelimbs) (Buckley & Lager, 2021);
- erythema and swelling near the hooves;
- appearance of a white band at the level of the coronary band (junction of the hoof and the foot);
- 80 to 90% of cases have lesions on the hooves and less than 25% have lesions on the snout and therefore the mouth (Fernandez, 2016);
- acute lameness in a group of pigs, ranging from discomfort to refusal to move, is often the first sign that is observed and can affect more than 50% of pigs, euthanasia may be considered when severe lameness is present;
- ulcerated wall of the feet and deep bleeding at the base of the hooves;
- flaking of the hooves;
- submandibular edema;
- anorexia, lethargy;
- fever (body temperature does not exceed 40° C).

Figure 1. Vesicular lesion observed in pigs infected with SVA. Lesions were observed on the snout (A) and legs (B), hemorrhages under the hooves (C). Source: (Zhang, et al., 2021)

6.2.3 *Clinical signs in sows*

- onset of clinical signs in farrowing and then towards gestation;
- decreased appetite to the point of anorexia;
- lethargy or fever (up to 40.5°C at the beginning of the disease);
- onset of vesicular signs on the snout and in the oral cavity (10% to 70% of sows) 10 days after the onset of the first signs;
- presence of vesicles on the udders of recently parting sows;
- erosions and ulcers in the coronary bands leading to lameness, only one case reported severe lameness in 90% of sows;
- normal breeding data in most affected herds (Segalés, Barcellos, Alfieri, Burrough, & Marthaler, 2016).

Some authors suggest that transport stress may increase the clinical severity of the disease and this may be relevant in sows shipped to slaughterhouses (Maggioli, et al., 2019).

Apparently, there is a constant evolution of the SVA towards a more virulent phenotype (Zhang, et al., 2018), which could predispose farms to different clinical presentations. Farms that report clinical outbreaks of SVA do not necessarily present ETNL, in any case, clinical presentations are self-limiting and last only one or two weeks. It has also been shown that not all infected animals necessarily develop vesicular lesions during clinical outbreaks; some animals may remain subclinical in clinically affected herds. It has been reported that clinical signs may persist for months in pig farms with a continuous flow of pigs into the growth site or farrow-to-finish sites.

A farrowing unit exposed to a natural infection of the SVA, lasting nine weeks from the onset to the end of clinical signs, demonstrated the presence of vesicular lesions for approximately two weeks (Buckley & Lager, 2021).

6.3 Post-Mortem Lesions

6.3.1 *Gross lesions*

Newborn piglets often have milk or colostrum in their stomachs at necropsy and 50% of piglets with diarrhoea have mesocular edema.

The gross lesions reported in twelve piglets aged one to five days were rib impressions on the pleural surface of the lungs (n=9), diphtheria glossitis (n=6) and ulcerative lesions in the coronary band (n=5) (Leme, Oliveira, Alfieri, Headley, & Alfieri, 2016). Histopathological lesions included interstitial pneumonia (n=12), myocarditis (n=6), diphtheria glossitis (n=3), encephalitis (n=3) and intestinal villi atrophy with the presence of vacuoles on superficial epithelial cells (n=6) (Leme, Oliveira, Alfieri, Headley, & Alfieri, 2016).

Oliveira et al. also reported lesions in 43 piglets who died from ETNL who were two to five days old. All piglets had a milk-filled stomach and intestines with watery stools (91%), renal petechial hemorrhages (79%), weak rib impressions on the pleural surface of the lungs (77%), edema and pulmonary congestion (60%), and cachexia (9%). In addition, there were concomitant vesicles on the snout with ulcerative lesions to the coronary band (21%) of the piglets studied (Oliveira, et al., 2017).

In pigs, gross lesions include round multifocal erosions or ulcerations in the hind limbs, specifically in the coronary bands and hoof peeling, fluid-filled vesicles, and chronic superficial or deep multifocal ulcers in the oral mucosa, snout, and nostrils. Peritonitis and serofibrinous pericarditis, hemorrhagic jejunitis, and focal gastric ulcer have been reported in a six-month-old pig infected with SVA (Singh, Corner, Clark, Sherba, & Fredrickson, 2012).

6.3.2 Microscopic lesions

In a first study, microscopic lesions observed in the limbs, oral cavity and snout included orthokeratotic and parakeratotic hyperkeratosis, and epidermal hyperplasia. There may also be ulceration and regional neutrophil infiltration with fibrin, edema, acute haemorrhage and karyorhectic nuclear debris (Singh, Corner, Clark, Sherba, & Fredrickson, 2012).

In another study, histopathological lesions were reported to include interstitial pneumonia (100%), myocarditis (50%), diphtheria glossitis (25%), encephalitis (25%), and intestinal villi atrophy with the presence of vacuoles on superficial epithelial cells (25%) (Leme, Oliveira, Alfieri, Headley, & Alfieri, 2016).

And in a final study, the main histopathological alterations were balloon degeneration of the transitional epithelium of the bladder (100%) and the epithelium of the renal pelvis (95%), villous atrophy of the small intestine (93%) and interstitial pneumonia (84%). Viral inclusion bodies of SVA have been observed in areas of balloon degeneration of the bladder and in neurons in areas of non-suppurative meningoencephalitis. Evidence of brain disease was observed in 46% of piglets with clinical manifestations consistent with ETNL (Oliveira, et al., 2017).

7. Diagnostic

The recognized diagnostic tools for this disease are PCR for virus detection and ELISA for antibody response to infection and exposure over time.

7.1 Samples

Specimens to be collected include, at a minimum, any lesion, regional lymph nodes, spleen, liver, lungs, kidneys, heart, tonsils, small and large intestine, brain, and spinal cord (Snelson, 2015) (Segalés, Barcellos, Alfieri, Burrough, & Marthaler, 2016).

Once the suspicion of a reportable disease has been ruled out by the authorities, the following samples should be collected:

- The best sample to take when fresh vesicles are present is the tissue and vesicular fluid, aspirated with a needle and syringe and transferred to a Falcon tube, often the Ct obtained are low. Another choice is to scrape the ruptured vesicles with a dacron/polyester swab (not cotton) and place it in one to two ml of saline or phosphate-buffered saline (PBS);

- Oral fluids are an excellent choice for detecting SVA in pig populations because they can detect viral RNA over a long period of time. The virus can be detected in oral fluids for up to 30 days in growing pigs and up to 20 days in piglets (Rademacher, 2016; Bjustrom-Kraft, et al., 2018).

- In a preliminary study, SVA was detected in processing fluids (PFs), including castration or tail dock tissues, collected from a breeding herd before and after the detection of an episode of the disease. As a result, a study was conducted to estimate the mean number of weeks that PFs remained positive for SVA after an outbreak. Ten producers with farrow-to-wean breeding herds volunteered to participate in this study by collecting PF samples after an outbreak of SVA was detected and submitting them for RT-PCR testing. PF samples from the 10 farms were positive for SVA for an average of 11.8 weeks after the outbreak. This study demonstrated that PF analysis could be an effective method to detect SVA and help stop its spread in endemic areas (Preis, et al., 2024).

- Another choice is to collect nasal swabs, feces, semen or serum.

7. 2 Diagnostic Tests – Antigens

Viral isolation is most effective in the first week post-infection, from scraping of vesicular lesions and oropharyngeal fluids, although not always successful, these samples have obtained a positive result with PCR to identify SVA in pigs (Pasma, Davidson, & Shaw, 2008) (Singh, et al., 2017). Sequencing of two regions of the virus, VP1 and 2C, is also possible to assess epidemiological links between strains (Hales, et al., 2008).

Branch et al. used RT-qPCR to assess the presence of SVA RNA in 85 tissue samples collected over a 10-year period from 50 pigs showing clinical signs of vesicular disease. All samples tested negative for significant vesicular diseases, including foot-and-mouth disease, vesicular stomatitis, vesicular exanthema and swine vesicular disease. A total of 67 tissue samples (79%), representing 44 cases (88%) with clinical signs, tested positive on RT-qPCR with Ct ranging from 11 to 38. Positive results from an RT-qPCR test are found in tissue samples from lymph nodes, spleen, esophageal (probang) samples,

epithelium, tonsils, lungs, blood, nasal swabs, and a pool of vesicular samples (vesicular fluids, tissues, or swabs from lesions) (Bracht, O'Hearn, Fabian, Barrette, & Sayed, 2016).

In a longitudinal study of a farrowing unit with clinical signs in Iowa, USA, tonsil and rectal swabs were collected from sows (with and without signs) and their piglets one week after the onset of clinical signs for six weeks. SVA RNA or antibodies were detected in 100% of the animals, regardless of their clinical status during the study (Gimenez-Lirola, et al., 2016).

In sows, the detection of SVA by RT-PCR in tonsils and rectal swabs was greater than 90% at the initial week after infection and remained as high as 50% until the fifth week. These types of specimens should be collected and submitted, in addition to fresh vesicular lesion swabs and fluid (if present), for vesicular disease investigations and for the detection of SVA (Tousignant, et al., 2017).

The live virus has been isolated from oral/nasal secretions and feces, which makes oral-fecal transmission important, in addition to direct contact. Oral, nasal, and fecal specimens are usually PCR-positive from one to 21 days post-infection, but oral and nasal specimens are sporadically positive for up to more than 28 days (Buckley & Lager, 2021).

The virus has been found in the intestines of sows and piglets without the presence of clinical signs. In neonatal piglets with diarrhoea, positive PCR results were reported in multiple tissues, serum, faeces and tonsils. In the testes and tonsils, the virus has been found for more than 92 days (Vannucci F. , 2022).

The *Minnesota Veterinary Diagnostic Laboratory* (MNVDL) identified SVA by RT-PCR on fluid from vesicles and blisters, skin of snouts and hooves, lymph nodes, and serum from sows affected and tested simultaneously for exotic vesicular diseases, including FMDV, VSV, SVDV, and SVEV. In addition, the virus has been detected in several tissues, including the brain, liver, spleen, lungs, intestines, and heart, of acutely dead piglets that were born to clinically infected sows (University of Minnesota - Veterinary Diagnostic Laboratory, 2023). Immunohistochemistry is also used to identify the presence of the virus in vesicular lesions (Yang, van Bruggen, & Xu, 2012). In a case report of 54 piglets with clinical manifestations of ETNL, 80% of them had, in at least one of the tissues evaluated by immunostaining, a positive for SVA by immunohistochemistry (IHC) where the SVA RNA was identified by RT-PCR (Oliveira, et al., 2017).

A diagnostic method has been described following the introduction of SVA in Brazil. The diagnosis was made by RT-PCR, using primers that determine the amplification of an internal region of the 3D gene. In addition, samples were inoculated into BHK-21 cell culture for viral isolation. During the first culture run, a cytopathogenic effect compatible with SVA replication was observed. Viral identity was confirmed using two additional

assays: IFA and nucleotide sequencing. Both tests confirmed that the infection was caused by SVA (Muller, Faria, Machado, & Martins, 2020).

7.3 Diagnostic Tests – Serology

Seroconversion of infected animals is around five to seven days post-infection and is persistent for 180 days or more (Vannucci F. , 2022).

An SVA-specific monoclonal antibody was developed for serodiagnosis and a competitive enzyme-linked immunosorbent assay (cELISA) was established using this antibody (Yang, van Bruggen, & Xu, 2012). This method was validated in 2017 by detecting anti-SVA antibodies in serial bloodletting during SVA epidemics. The results indicate a high agreement of the test results with a specificity and sensitivity of 98.2% (97.2–98.9%) and 96.9% (94.5–98.4%) for cELISA, and 99.6% (99.0–99.9%) and 98.2% (95.8–99.4%) for viral neutralization assay, respectively, suggesting that these tests are suitable for the serological detection of SVA in pigs (Goolia, et al., 2017).

Antibody (IgG) levels were detected by the ELISA test in the sows one week after the onset of clinical signs, which showed a significant increase during the first three weeks after the onset of vesicular lesions and then reached a plateau. Antibody levels were detected in all sows showing clinical signs four to six weeks after the onset of infection. In sows without clinical signs, the detection rate is 72.7% after three and four weeks of the onset of infection and 81.8% after five and six weeks (Gimenez-Lirola, et al., 2016).

In piglets, antibody levels were detected one week after the onset of infection. This rate decreases as early as the second week after infection and there is no detection of antibodies at five and six weeks after the onset of infection in piglets from sows with or without clinical signs (Gimenez-Lirola, et al., 2016).

A seroprevalence study of SVA was conducted in the United States on 219 commercial hog farms in the top 18 swine-producing states. Samples taken from pigs and sows were analyzed by rVP1, ELISA, and IFA (Houston, et al., 2019).

Validation work of the ELISA assay for the VP2 viral protein of the SVA is ongoing by private companies, public organizations and universities.

An ELISA assay based on a non-structural viral protein has been developed, capable of distinguishing infected animals from vaccinated animals (DIVA). Different expression systems (eukaryotes and prokaryotes) have been used to express recombinant proteins. The developed ELISA DIVA SVA, based on recombinant SVA proteins, expressed in both the baculovirus and E. coli systems, demonstrated high sensitivity and specificity for detecting antibodies against SVA. These tests offer great potential as alternative diagnostic tools for large-scale screening for SVA antibodies. Their simplicity, reliability and usefulness make them suitable for serological diagnosis, epidemiological investigations and disease control

in pig herds. In addition, the ELISA DIVA SVA developed in this study could serve as immunity tests, providing valuable support for the approval and implementation of vaccination programs in pig herds (Watcharavongtip, Jermsutjarit, Tantituvanont, & Nilubol, 2025).

8. Prevalence

Outbreaks have occurred on farms of all sizes with different levels of biosecurity and originating from high-density and non-high-density swine areas.

A retrospective study by the SHIC on oral fluid samples was conducted at the end of August 2015 on 2033 samples (441 cases) from 25 U.S. states, Canada and Mexico to assess the prevalence of SVA in pig herds showing no clinical signs associated with vesicular disease. Oral fluid samples were from diagnostic laboratories at the University of Iowa and the University of Minnesota. A positive PCR test result was obtained in five different samples from five different states (prevalence of 1.2%). One of these cases had clinical signs of vesicular disease in the following days (Main, et al., 2015).

A study was carried out in the southeastern United States to identify viruses present in market pigs using metagenomic sequencing. Nasal and rectal samples were collected from market pigs from different producers. A second sampling took place a month later. In the first sampling, SVA was identified in less than 10% of the pooled nasal and rectal specimens and in the second sampling, SVA was identified in 28% of the pooled nasal and rectal specimens (Myers, Duff, Smith, Nemechek, & Hause, 2016).

Another study was done on 1517 samples to assess the presence of SVA by real-time PCR at the University of South Dakota. Samples came from 13 states and were submitted for routine diagnostic testing. All samples were negative, suggesting a low prevalence of SVA in pigs in the United States (Baker, et al., 2017).

The SVA seroprevalence study, on 219 commercial pig farms in the top 18 U.S. swine producing states, demonstrated the presence of anti-SVA IgG antibodies in pigs from clinically healthy grower-finisher herds (42.7%) and sows (75.8%). These results suggest that SVA is circulating subclinically in sow farms and grower-finisher pig farms in major swine-producing states in the United States (Houston, et al., 2019).

A study conducted in the United States estimated that seroprevalence at the farm level was 17.3% and 7.4% in breeding and growing pig farms, respectively. Farrowing sites were 2.64 times more likely to have SVA seropositivity than growing pig sites (Preis, et al., 2022).

A seroprevalence study conducted in Brazil indicated that SVA was not circulating in that country until 2014 (2007-2013); nevertheless, SVA was circulating between 2014 and

2016 with an overall seroprevalence of 36.4%, including clinically and non-clinically affected animals (Saporiti, et al., 2017).

Ran et al. conducted a global meta-analysis and systematic review to determine the status of SVA infection in pigs. Research data spanned from 2014 to 2020, a total of 34 articles were included in this analysis. The pooled prevalence of SVA was estimated in pigs using the random-effects model. A study risk of bias assessment and subgroup analysis were undertaken to explain heterogeneity. The prevalence of SVA was estimated to be 15.90% (1,564/9,839; 95% confidence interval [CI], 44.75-65.89) globally. Prevalence decreased to 11.06% (945/8,542; 95% CI 28.25-50.64) after 2016. The highest prevalence of SVA with the VP1-based RT-PCR and immunohistochemistry assay was 58.52% (594/1,015; 95% CI 59.90-83.96) and 85.54% (71/83; 95% CI 76.68-100.00), respectively. In addition, the prevalence of SVA in piglet herds was highest at 71.69% (119/166; 95% CI 68.61-98.43) ($p < 0.05$). In addition, their analysis confirmed that subgroups, including country, year of sampling, sampling position, gene detected, method of detection, season, age, and climate, may be the heterogeneous factors associated with the prevalence of SVA. The results showed that SVA currently exists widely in various countries and that the prevalence of SVA in North and South America was higher than in Asia. For seasonal subgroups, SVA infections are more common in the spring and fall, but the rate of infection was significantly higher in the fall than in other seasons ($p<0.05$). At the same time, the combination of climatic factors revealed that areas with a temperature of 15°C to 20°C and annual rainfall of 50 to 80 mm have the highest prevalence of SVA. Therefore, the SVA may prefer to survive in a warm, low-humidity environment (Ran, et al., 2023).

Li et al. conducted a study to determine the prevalence of SVA in swine herds in China from 2018 to 2021. A total of 4,901 samples of pig tissue were collected from 18 provinces, autonomous regions and municipalities for the purposes of epidemiological investigation, virus isolation and genetic analysis. In 2021, the individual positivity rates (IPRs) from the perspective of spatial distribution in eastern, southern, central, northern, southwestern, northwestern, and northeastern China were 0, respectively; 0; 1,69; 0,94; 11,70; 3.31 and 2.21%. The positive herd rates (PHRs) were 0; 0; 9,52; 9,09; 50,00; 7.69% and 23.08%. From a temporal distribution perspective, PHR showed a general downward trend from 2018 to 2021, with only a slight increase in 2020, and PHR decreased from 36.63 to 10.07%. In terms of population distribution in 2021, the IPR (2.62%) and PHR (12.00%) in apparently healthy pig herds (abattoirs) were higher than those in unhealthy pig herds (2.10% and 5.13%, respectively), which is consistent with the 2019 results. Regarding the characterization of the prevalent strains, 10 SVA strains isolated from positive samples in 2019 were grouped into clades I and VII. In conclusion, until 2021, the prevalence of SVA in pig herds in China was still relatively high, the contaminated area was still large, and there were a number of hidden infections (Li, et al., 2024).

A study analyzed the incidence and geographic distribution of SVA outbreaks in U.S. breeding flocks from January 2015 to December 2024. Diagnostic data from veterinary laboratories and reports from practitioners were combined to assess the incidence of SVA, identifying temporal fluctuations and regional patterns. The results indicate that the cumulative incidence of SVA in U.S. breeding flocks is low overall (<2.5% per year), with a peak incidence observed in the third and fourth quarters of the calendar year. Outbreaks are most frequently reported in the Midwest. The results suggest potential seasonality or regional factors influencing the transmission of the disease. The median interval between SVA outbreaks for sites with more than one outbreak is 402 days (Kikuti, Yue, Melini, Vadnais, & Corzo, 2025).

9. Treatment

There is no treatment at the moment. It is a self-limiting disease.

10. Immunity

10.1 Vaccination

There is currently no vaccine available for this virus.

Researchers in China produced the virus with BHK-21 cells in rolling vials and inactivated it. The results showed that animals receiving one dose of the inactivated vaccine with an oily adjuvant developed high neutralizing antibody titers and showed no clinical signs after viral infection compared to unvaccinated animals, indicating good vaccine protection (Yang, et al., 2018).

In the same vein, in the United States, researchers at the University of Iowa have developed a recombinant SVA strain (rSVAm SacII) and have evaluated its protective immunogenicity. To assess the immunogenicity and protective efficacy of the strain, four-week-old piglets were immunized with formulations based on the inactivated or live strain, and then the vaccines were inoculated with a heterologous contemporary SVA strain. A single immunization with live virus via the intramuscular (IM) and intranasal routes resulted in strong antibody responses, detected between three and seven days after infection and no piglets showed clinical signs. Neutralizing antibody responses in animals immunized with the IM-inactivated virus were delayed and were only detected after a booster on day 21 post-infection (Sharma, et al., 2019).

Of all the reported SVA vaccines, only the inactivated SVA vaccine has been successfully developed. However, to ensure the elimination of this pathogen, safer and more effective vaccines are needed. A virus-like particle (VLP) vaccine is probably the best alternative to the inactivated vaccine. In this study, the SVA CH-HB-2017 strain was used to infect pigs,

determine routes of administration and infective dose. The experimental pigs were then immunized with the VLP-based vaccine emulsified in an ISA 201 adjuvant. The results showed that the VLP-based vaccine induced neutralizing and specific antibodies at levels similar to those of an inactivated SVA vaccine after immunization. These results also indicated that the VLP-based vaccine could simultaneously elicit cellular and humoral immune responses. It is important to note that after the investigational infection, the VLP-based vaccine offered similar levels of protection as the inactivated vaccine. In this study, we successfully obtained novel VLP-based SVA vaccines and confirmed their high immunogenicity, providing a superior vaccine candidate to inactivated vaccine for the protection and elimination of SVA (Mu, et al., 2020).

Buckley et al. developed an inactivated vaccine against SVA mixed with an oil and water adjuvant and an inactivated whole-virus SVA vaccine against epidemics in nursery pigs and mature sows to assess the protection of passive maternal immunity generated by immunized mothers. This study presented promising inactivated vaccine candidates for the protection of nursery piglets, sows and their offspring (Buckley & Lager, 2022). Knowing that immune effects are influenced by different viral strains, preparation processes, adjuvants, immune doses, enhanced immunity, and other factors, Zhang et al. compared two adjuvants in a subsequent study. Experiments on mice with the inactivated vaccine SVA CH-GX-01-2019 showed that the immune response was superior when the vaccine was mixed with the Montanide ISA 201® adjuvant, compared to the imject® Alum adjuvant. As a result, Montanide ISA 201® adjuvant was a better choice than Imject® Alum in the preparation of the inactivated vaccine SVA CH-GX-01-2019, which offered effective protection to pigs (Zhang, et al., 2024).

In this study, a strain of SVA isolated from China, named SVV LNSY01-2017 (MH064435), was used as the reference virus for the preparation of an inactivated SVV vaccine. The SVV culture was directly inactivated using binary ethyleneimine (BEI) and ß-propiolactone (BPL). GLP showed a better effect as an SVV inactivator based on the results of pH variation, inactivation kinetics and detection of VP1 viral protein content. The GLP-inactivated SVV was then emulsified using different adjuvants, including the adjuvants MONTANIDETM ISA 201 VG (ISA 201) ® and MONTANIDETM IMG 1313 VG N (IMS 1313)®. The immunoreactivity and protective efficacy of inactivated vaccines were subsequently evaluated in feeder pigs. The SVV-GLP-1313 vaccine showed a better humoral response after immunization and further challenge tests showed that the SVV-GLP-201 and SVV-GLP-1313 combinations could withstand exposure to a virulent strain of SVV. The inactivated vaccine candidate SVV LNSY01-2017, developed in this study, has shown to be a promising alternative for the protection and prevention of SVV infection in pigs (Liu, et al., 2022).

10.2 Cross Protection

Knowles et al. showed that sequences between SVV-001 virus (laboratory strain) and seven viruses isolated from pig farms have a high degree of viral sequence similarity. In addition, these viruses could be serologically related to each other, as well as to the SVV-001 laboratory strain (Knowles, et al., 2006).

Immunization with FMD vaccine shows no cross-protection against SVA infection (Zhu, et al., 2017).

Later, Fernandes et al. demonstrated that the historical SVA SVV 001 strain has low virulence in pigs compared to the contemporary SVA SD15-26 strain. Also, that immunological assays indicate that SVA SVV 001 and SD15-26 are antigenically related and share conserved antigenic determinants (Fernandes, et al., 2018).

11. Prevention and control

11.1 Prevention

Between July and October 2015, epidemiological studies were conducted in a series of cases in sow herds in the United States, diagnosed with SVA. The studies identified the most common biosecurity gaps. A questionnaire was used to obtain information on cases and risk factors that may have occurred in the four weeks prior to the outbreak. Risk factors vary greatly between herds, depending on the size of the herd and the type of herd (weekly versus batch farrowing). The risk factors considered high for the introduction of SVA identified by the survey teams are: employee entry onto the farm, disposal of deadstock, movement of sows to another building for slaughter, and entry of replacement gilts. Three of these farms had low biosecurity. One farm had high biosecurity, but there was a high frequency of risk factors given the size of the farm. Finally, two farms with average biosecurity demonstrated epidemiological links with other positive farms or breaks in biosecurity protocols that caused the outbreaks. Outbreaks have occurred on farms of all sizes and from high and non-high density swine areas (Baker, et al., 2017; Holtkamp, 2016).

Canning et al. also assessed the highest risk of introducing SVA in a case of a farrow-to-finish operation. The authors found that the movement of employees or visitors and access to pets and rodents in the pigs' vicinity were important risk factors (Canning, et al., 2016).

A study conducted in the United States showed that livestock farms that reported sending dead animals for rendering were more likely to be SVA seropositive, while farms reporting five or six different biosecurity measures were less likely to be SVA seropositive. The biosecurity measures included in the survey were: 1) registration of visitors required to enter the farm, 2) procedures for entering and exiting the shower, 3) installation of the

Danish entry system, 4) use of the farm-specific boots, 5) use of farm-specific clothing, and 6) a stop time of visits before entering the farm (Preis, et al., 2022).

Other risk factors to consider are pigs from multiple sites, foodborne transmission and semen as potential vectors of the virus.

11.2 Control methods

Pending further information on the origin, transmission and pathogenesis of the virus, suggested control methods for other viruses in the *Picornaviridae* can be used. Humans are always an important vector in the transmission of the virus from an infected animal or a contaminated surface to susceptible animals, so it is important to have adequate biosecurity measures in place to prevent this mode of transmission. Additional biosecurity measures, such as limiting vehicle and visitor traffic on the farm, washing and disinfecting vehicles and equipment, and not sharing equipment between farms or buildings unless it has been washed and disinfected, are also required (Kansas Department of Agriculture Division of Animal Health, 2015).

Biocontainment is the best way to control the spread of the disease outside the premises. The PCR test can be used for surveillance, and the introduction of new subjects can start again when there is no longer any virus circulating in the herd. After gilts are introduced into a herd, it is necessary to ensure that there are no antibodies in the weeks that follow. Control of SVA infection depends on improving the management of animal movements, inputs (e.g., feed and semen) and the environment of pig farms. Expanded surveillance of the incidence of SVA infection and disinfection of facilities and the environment are essential to reduce the risk of occurrence.

It is known that an outbreak of SVA is self-limiting and that the virus can persist in the tonsils for more than nine weeks in a sow herd after the outbreak (Tousignant, et al., 2017). A farrow-to-finish farm of 1800 sows was confirmed to have been previously exposed to SVA by IFA test with no evidence of clinical signs. The farm suspended the introduction of gilts for 51 weeks as part of a PRRS elimination program. The objective of the study was to monitor the persistence of SVA in the sows' tonsils during the PRRS elimination protocol in the closed sow herd. Herd closure without mass inoculation was effective for the elimination of SVA in a naturally infected population; it took 47 weeks to stop the detection of SVA in the tonsils. Therefore, targeting the elimination of SVA could be complementary to PRRS elimination protocols (da Silva de Pita & al., 2024).

12. Economic Impact

Although it is a virus with direct consequences for production, the most important problem is that the symptoms are similar to those of other exotic vesicular diseases that may be present in pigs such as foot-and-mouth disease, vesicular stomatitis, vesicular exanthema

and swine vesicular disease, and this has a disruptive effect on trade, until the diagnosis is confirmed. It is important to note that SVA is not a reportable disease, but the other vesicular diseases listed above are (except vesicular exanthema). Since it is impossible to distinguish the disease of concern solely by observation of clinical signs, laboratory tests must be done to confirm the pathogen responsible.

Regarding the impact of the disease on the affected animals and farms, a variable number of pigs do not feed at first due to a short, transient fever and later because of vesicles in the oral cavity that can rupture. Coronary band vesicles cause pain and lameness that reduce motility and access to feeders. The number of pigs affected during acute episodes in naïve farms ranges from 10 to 90%. Although many of them may be affected, the disease is not associated with increased mortality in adults, but the impact is much more evident in piglets with a mortality rate of around 80%. Some affected farms may show a lower rate of parturition and a high return of sows to heat after an acute episode. Current epidemiological data confirm that occasional outbreaks still occur and cause a multifactorial economic burden to the hog industry.

13. Discussion

SVA is not considered a disease limiting swine production, but the clinical signs resemble those of foot-and-mouth disease. It is therefore important to monitor the presence of injuries on the nose, mouth or feet and to make a diagnosis quickly. An investigation must be done to avoid ending up with a more clinically and economically important disease.

As SVA is not a reportable disease in Canada, its presence does not affect international trade, but it can disrupt the pace of production on the farm, in assembly yards and in slaughterhouses for the duration of confirmatory diagnostic testing of the disease.

Differential diagnoses include foot-and-mouth disease, swine vesicular disease, swine vesicular exanthema, and vesicular stomatitis.

14. Recommendations

The EQSP invites all farmers and stakeholders in the Quebec pork sector in contact with pigs to be particularly vigilant regarding this disease. Farmers should monitor their animals regularly in order to quickly report any suspicions. If SVA or any other type of foreign animal vesicular disease is suspected, it is important to report it immediately to your veterinarian, to the CFIA (Contact the CFIA by phone - inspection.canada.ca), to MAPAQ (1-844-ANIMAUX (264-6289) and to contact the EQSP emergency line at 1-866-218-3042. It is also essential not to move pigs until the situation has been clarified, knowing that any detection at a slaughterhouse will likely have disruptions to slaughter and trade while the results of the diagnosis are obtained.

Wet culled sows shipped outside of Quebec must be dried off in Ontario assembly yards for a period of five to seven days before being shipped to a slaughterhouse, which is more than enough time to become infected with the SVA. Since these centres may be infected with the SVA virus, the EQSP recommends that farmers, as far as space and their production system allows, dry off sows before they leave the herd in order to minimize the risk of spreading the disease to pig farms through transport.

15. CFIA Response Protocol

15.1 On the Farm

1- Voluntary cessation of the movement of animals is required from the suspicion of compatible clinical signs until the CFIA inspector arrives. The veterinarian must not leave the site. The veterinarian must call the CFIA regional office (Contact the CFIA by phone - inspection.canada.ca) and make a report. The case must be documented (% mortality, % morbidity, types of lesions, etc.). He must also call the MAPAQ at 1-844-ANIMAUX.

2- The CFIA will be the chief operating officer until a diagnosis excludes the notifiable vesicle diseases.

3- The CFIA will make a decision on whether or not to quarantine the site after the site visit. The CFIA veterinarian will examine the animals and take samples. Sampling by the CFIA veterinarian and testing at the CFIA's National Centre for Foreign Animal Disease (NCFAD) laboratory in Winnipeg is mandatory to confirm the diagnosis of a reportable vesicular disease. The processing time, from the time the NCFAD laboratory receives the samples, is within 24 hours. Pigs with vesicles cannot be transported into Canada.

4- Epidemiological data collection: duration between 1h30 and 2h00.

5- Risk Determination.

6- Sample Submissions and Notifications.

7- Declaration of the infected place (quarantine).

15.2 At the slaughterhouse

The CFIA may request a temporary shutdown of the abattoir if samples are submitted from that location. Slaughterhouses must have an action plan to stop their activities for 48 hours.

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