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**Review Article**

# Recent insights on epidemiology, diagnosis and control of Classical and African Swine Fever in pig industry

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## ABSTRACT

Classical swine fever is caused by an enveloped RNA virus in the genus Pestivirus of the family Flaviviridae, whereas African swine fever (ASF) is caused by a double-stranded DNA virus in the genus Asfivirus of the family Asfarviridae. Both diseases are devastating and cause great loss in the pig industry through mortality, growth retardation, and poor reproductive performance. The clinical symptoms of African swine fever and classical swine fever in pigs can be extremely similar; hence laboratory testing is necessary to distinguish between both diseases. Virus isolation, fluorescent antibody test (FAT), antigen capture antibody enzyme-linked immunosorbent assay (ELISA), reverse-transcription polymerase chain reaction (RT-PCR), virus neutralization test (VNT), and antibody ELISA have been developed for diagnosis of CSF. For detection of ASF, ELISA, chemiluminescent immunoassay (CLIA), PCR, luciferase immunoprecipitation assay (MB-LIPS), loop-mediated isothermal amplification (LAMP), and recombinase polymerase amplification (RPA) have been developed. For the piggery business to grow, quick diagnosis and effective preventative measures are needed to aid in the management and elimination of both diseases. Pigs have been protected against these diseases through vaccination. Preventing entry of the CSF and ASF viruses through strict quarantine measures is necessary. Early detection and knowledge of the disease's epidemiology are crucial for both preventing the disease's spread and developing an effective management strategy. This review provides insights on the etiological agent, epidemiology, transmission mode, clinical symptoms, pathogenesis, diagnosis, and control strategies of both diseases.

**Keywords:** Control, growth, mortality, pigs, virus

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## INTRODUCTION

Pigs are a primary source of household income in many countries. Pig industries are greatly affected by Classical swine fever. It is caused by an enveloped RNA virus in the genus Pestivirus of the family Flaviviridae. CSF is a severe and financially detrimental swine disease that can infect both domestic and wild pig populations enzootically and epizootically (Edwards *et al.*, 2000). Due to trade restrictions on meat exports and widespread animal deaths caused by the disease, the presence of CSF virus (CSPV) in pig herds can have a serious negative economic impact on the meat production industry. African swine fever virus (ASFV) is a member of the Asfivirus genus within the Asfarviridae family (Gaudreault *et al.*, 2020).

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The case fatality rate for the African swine fever virus (ASFV) in domestic pigs and wild boars can reach 100%. Pigs can get a variety of symptoms, from chronic and subclinical diseases to peracute (severe, short-lived, and usually rapidly lethal) diseases. Virulent virus strains can cause fever, appetite loss, and internal and skin hemorrhages, among other clinical symptoms. Pigs suffering from less virulent strains may exhibit minor symptoms including fever, depression, and a reduced appetite. The clinical symptoms of African swine fever and classical swine fever in pigs can be extremely similar; hence laboratory testing is necessary to distinguish between both diseases.

For the purpose of diagnosing CSF, several methods have been developed, including virus isolation, fluorescent antibody test (FAT), antigen capture antibody enzyme-linked immunosorbent assay (ELISA), reverse-transcription polymerase chain reaction (RT-PCR), virus neutralization test (VNT), and antibody ELISA. ELISA, PCR, loop-mediated isothermal amplification (LAMP), chemiluminescent immunoassay (CLIA), and recombinase polymerase amplification (RPA) have all been developed for the detection of ASF. ASF vaccine development can be accomplished quickly and effectively with the use of genome editing technology. A strong and effective method for developing gene-deleted ASFV that could be used as a live-attenuated vaccine is CRISPR/Cas9. Since the presence of ASF antibodies implies prior infection, serological testing has been widely used to detect ASF virus. Rapid diagnosis and efficient preventative actions are necessary to help control and eradicate such diseases. Vaccinations are developed to protect pigs from CSF and ASF. Strict quarantine regulations are required to stop the CSF and ASF virus from entering and stopping the disease's spread. Developing a successful management plan depend on early detection and understanding the epidemiology of the condition. This review paper focus on recent insights on etiological agent, epidemiology, transmission mode, clinical symptoms, pathogenesis, diagnosis and control strategies of classical swine fever and African swine fever.

**Economic loss caused by CSF and ASF**

Due to CSF, live pigs, pork products, and genetic materials from afflicted areas are subject to trade restrictions and embargoes. These limitations result in major financial losses for exporters, processors, and farmers. According to Singh *et al.* (2016), the economic losses resulting by CSF in pigs were 25.93% from morbidity and 74.07% from mortality (Singh *et al.*, 2016). Of the US$3.8 million overall cost of CSF in Colombia, 88% went toward the expense of the vaccination program. In Mizoram, the total economic losses as a result of CSF was INR 16,69,34,465. Morbidity accounted for 42.83% of the total loss, whereas death accounted for 57.17%. 30.56% of the total loss was attributable to weight loss, which included 29.13% direct loss from weight loss, 1.18% loss from an extended inter-farrowing interval, and 0.26% loss from abortions (Chaudhary *et al.*, 2019). It is expected that the global meat and animal feed markets will be greatly influenced by African swine fever. The amount of ASF varied greatly between countries, with the Philippines spending over US$58 million, North Macedonia spending US$ 33,19,666 and Lao Cai (Vietnam) spending US$8,26,911 (Casal *et al.*, 2022). In the Philippines and Vietnam, the impacted farms incurred 96–98% of the costs, whereas in North Macedonia, the movement control of the nearby and vulnerable farms incurred the highest costs (77%) (Casal *et al.*, 2022). Between 2014 and 2018, the economic cost of ASF in Africa was estimated to be $15,13,340 in Benin (Ohouko *et al.*, 2020). Due to the high mortality rate (91%) in 306 farms, an epidemic in Nigeria in 2001 cost US$9,41,492 (Babalobi *et al.*, 2007). According to Kivumbi *et al.* (2021), an epidemic in Tanzania affected 219 households, costing $41,065 USD. The

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majority of the costs due to ASF in Lao Cai Province, Vietnam, were incurred by the affected farms (96%), particularly because culling and animal destruction accounted for 89% of the total costs (Casal *et al.*, 2022).

## Etiological agent

### Etiological agent of CSF

Classical swine fever is a pestivirus belonging to the Flaviviridae family (Wengler *et al.*, 1995), which also includes sheep's border disease (BD) and bovine virus diarrhea (BVD). The antigenicity and pathogenicity of CSF strains differ significantly. A single passage through pigs can boost virulence. High virulence strains are responsible for typical epidemics that have high rates of morbidity and mortality. Subacute or chronic infections are caused by strains with intermediate pathogenicity. Low virulence strains might result in reproduction failure, neonatal losses, or weak or undetectable infections. At both ends of the virus are non-translated regions (52 NTR and 32 NTR), which contain a single open reading frame that codes for a big protein that breaks down into smaller pieces. Near the 52 end of the genome are the genes that code for the structural proteins, which include the main envelope glycoprotein gene E2. The polymerase gene NS5B is one of the two-thirds of the genome that contain genes that encode non-structural proteins (Meyers & Theil, 1996). When it comes to environmental factors, the CSF virus is relatively resistant. Depending on the climate, the virus can linger in pig housing, bedding, and excrement for days or weeks. The virus can persist for months in refrigerated meats, for years in frozen pork, and for a few months in various curing techniques. Lipid solvents and 2% sodium hydroxide both render the virus inactive (Iowa State University, 2024)

### Etiological agent of ASF

The African swine fever virus (ASFV) is a large virus with double-stranded DNA. The African swine fever (ASF) is a member of the Asfivirus genus within the Asfarviridae family (Gaudreault *et al*., 2020). This virus is an average diameter of 200 nm and its genome ranging from 170 to 190 kbp, ASFV is an icosahedral, linear double-stranded DNA virus (Salas & Andrés, 2013). The two lipid membranes that ASFV has are an external one that is taken from the cellular membrane during budding and an interior one that envelops the particle's inner core and is most likely produced from the infected cell's endoplasmic reticulum. The virion is composed of a nucleoid, outer envelope, capsid, inner envelope, and core-shell virus (Salas & Andrés, 2013). The nucleotide sequence of a 478 bp variable region in the viral p72 gene's C-terminus serves as the basis for ASFV genotyping (Gaudreault *et al*., 2020; Achenbach *et al*., 2017).

## Epidemiology

### Epidemiology of CSF

Classical swine fever is extremely contagious, and it spreads quickly when susceptible pigs come into direct or indirect contact with infected pigs. Before, during, and after recovery pigs with acute infections release significant amounts of virus. Viral infections in fetuses are disseminated by the secretions and excretions of live pigs. It is commonly known that uncooked waste food containing infectious pork scraps fed to pigs can start a lot of outbreaks. Additional means of viral transmission include fomites, pets, birds, arthropods, and farm equipment (infected wagons, trucks, tractors, and machinery), as well as humans (sloppy farmers, salespeople, and veterinarians). Most likely, airborne communication is not very important (Iowa State University, 2024).

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**Epidemiology of ASF**

Through a sylvatic cycle (transmission between warthogs and soft ticks) and a domestic cycle (transmission between domestic pigs), the African swine fever virus (ASFV) spreads from a reservoir host (warthog) to domestic pigs. The virus survives by expressing a number of genes linked to virus-host interactions. The mononuclear phagocytic cells of both domestic and wild pigs are the site of ASFV replication. Soft ticks of the genus Ornithodoros serve as virus reservoirs and are the site of ASFV replication. In addition to being observed during infection on the Iberian Peninsula (*Ornithodoros erraticus*), these ticks are part of the epidemiological cycle of ASF in eastern and southern Africa (*Ornithodoros moubata*).

**Pathogenesis**

**Pathogenesis of CSF**

Following consumption, the CSF virus attacks tonsil crypt epithelial cells, travels to nearby lymph nodes, and causes viremia within 24 hours. First, the virus replicates in the tonsils. Replication can also take place in endothelial cells, bone marrow, circulating leukocytes, and lymphoid tissues (spleen, Peyer's patches, lymph nodes, and thymus: in particular). Excretions and secretions contain the virus, which spreads to many cells of the epithelial type in three to four days. Swine are more prone to other infections as a result of the virus's lymphoid depletion. Thrombocytopenia and leukopenia are caused by injury to the bone marrow. Thrombocytopenia causes petechial and ecchymotic hemorrhages at numerous locations in addition to endothelial cell destruction. Antigen-antibody complexes harm glomeruli, a swine with a prolonged CSF infection may develop glomerulonephritis. Some or all of the fetuses in pregnant gilts and sows may be infected by the virus if it crosses the placenta. Depending on the stage of pregnancy, the outcome may include abortion or the birth of stillborn piglets, mummified fetuses, or live piglets that are chronically infected. Myoclonia congenita, a disease that causes shaky piglets, is a notable example of the fetal abnormalities that can arise from in utero infections.

**Pathogenesis of ASF**

The pathogenesis of ASFV infection is multi-stage, beginning with the initial infection of lymphoid tissues, followed by replication in monocyte-macrophages and the eventual destruction of lymphoid organs and tissues. ASFV infection is characterized by the depletion of lymphocytes, which is a result of the activation of infected cells and the cytokine storm that follows. A major factor in the development and severity of the disease is the intricate interaction between the virus and the host's immune system. Only suids and soft ticks belonging to the Ornithodoros species are ASFV's hosts. ASFV infection in its wild suid hosts in Africa can lead to longer-term chronic infections and minor clinical symptoms (Jori & Bastos, 2009; Jori, *et al.*, 2013). On the other hand, the majority of ASFV isolates induce acute hemorrhagic fever in wild boar and domestic pigs, with a case fatality rate that is close to 100% (Blome *et al.*, 2013; Pietschmann *et al.*, 2015). Acute and peracute forms (Sánchez-Cordón *et al.*, 2018; Gómez-Villamandos *et al.*, 2013; Sánchez-Vizcaíno *et al.*, 2015) of the diseases seen in domestic pigs and wild boar are brought on by extremely virulent isolates and cause death 4–15 days after infection. Lower case fatality rates (30–70%) are caused by isolates that are moderately pathogenic. Isolates with low virulence cause few or no case fatalities and no vascular lesions. On the other hand, persistent disease symptoms are visible. Extremely high viral concentrations in tissues and blood (up to 109 TCID50/mL) are linked to infection. The acute symptoms of sickness in wild boars (Sus scrofa) are similar to those in domestic pigs (Sánchez-Vizcaíno *et al.*, 2015; Sánchez-Cordón *et al.*, 2018; Blome, *et al.*,

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2013). While certain reduced-virulence isolates have been isolated from infected wild pigs in the Baltic States, the majority of isolates circulating in Europe, the Russian Federation, and Asia cause the acute form of the disease (Gallardo *et al.*, 2018; Nurmoja *et al.*, 2017). Even after recovering from an illness, animals may still have the infection for several months (de Carvalho Ferreira *et al.*, 2013).

**Geographical distribution**

**Geographical distribution of CSF**

Brich (1917) reported that the development of railways during the mid-19$^{th}$ century may have facilitated the spread of the virus infection. Classical swine fever (CSF) was first documented in Ohio, USA, in 1833, but an epizootic resembling CSF was reported in France in 1822 (Cole *et al.*, 1962). By the end of the 20th century, CSF was still widespread in many parts of the world, but it was successfully eradicated in many countries. For example, Canada has been free of CSF since 1963. The disease's official eradication plan in the United States began in 1961, and the final case was reported in 1976 (Wise, 1986). In 1997, there were a lot of outbreaks in the European Union, especially in Belgium, Germany, Italy, Spain, and the Netherlands (611). However, in 1998, the number of outbreaks significantly decreased to 54. In 1997 and 1998, there were no outbreaks reported in Austria. There was no longer any disease in Australia. According to reports, the disease is only absent in Belize and Panama of Central America (Edwards *et al.*, 2000). The disease is enzootic in various countries of South America, with the exception of Uruguay and Chile. Since 1991, Uruguay has not reported any CSF cases, and Chile last reported a case in August 1996. Cuba, Haiti, and the Dominican Republic of the Caribbean Islands are all affected by the disease. According to Edwards *et al.* (2000), the first recorded case of CSF in Haiti occurred in 1996. In 1888, the first CSF outbreak was identified in Japan. Outbreaks of CSF have significantly diminished since the live attenuated GP vaccine was developed in Japan in 1969; the last recent incidence was documented in 1992. The disease is common throughout South East Asia, especially in Indonesia, Korea, Malaysia, Myanmar, Mongolia, the Philippines, Taiwan, and Vietnam except for Madagascar. The disease is not reported to be an issue across most of Africa, however the status there is unclear (Edwards *et al.*, 2000).

**Geographical distribution of ASF**

In 1910, ASF was first identified as an independent disease entity in Kenya (Montgomery, 1921). ASF was found to be in distribution in a number of African states following its initial detection. The disease was later found to have existed in wild hosts in eastern and southern Kenya for a very long time. After that, it was found in Central and West Africa, but it was only seen in sub-Saharan African nations until 1957, when it was first discovered outside of Africa in Lisbon, Portugal, from where it had fled West Africa.

In the 1950s, the disease first appeared in the Iberian Peninsula of Europe. In the late 1970s and early 1980s, it was discovered in Cuba, Brazil, Haiti, and the Dominican Republic. By the middle of the 20th century, Spain and Portugal had eradicated this disease. It then moved to Russia and Ukraine in 2007 after first showing up in Georgia and the Caucasus region. The disease's potential to spread to north-west Europe, which includes Germany, Denmark, the Netherlands, and northern France, as well as the major pig-producing regions of west and north Poland, is still quite real. ASF has occurred in Asia but this disease has never been reported in the United States, Canada, Australia, or New Zealand.

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**Mode of transmission**

**Transmission of CSF**

In a natural condition, the oro-nasal route is the primary means of CSF viral transmission. Infected animals' blood, secretions, and excretions contain the CSF virus. Contaminated feed and water are also common causes of infection. CSF virus-infected adult boars can excrete the virus with their semen, which can then be used to artificially inseminate sows and their fetuses (de Smit *et al.,* 1999). Another way that the CSF virus can spread from sows to their young is through intrauterine infection. The result of such a transplacental transit is contingent upon the gestational stage. While infections in the final trimester of pregnancy typically end in abortion, deformity, or the birth of weak or dead piglets, infections in the early trimester of pregnancy typically cause repeated breeding and abortion. Persistently infected piglets can only be born if the sow contracts the infection during the second trimester. These immune tolerant piglets may live for a very long time, continuously releasing the virus into the environment until the sickness strikes late and the animals die. In the epidemiology of CSF, this phenomenon, known as the carrier-sow syndrome, is crucial because it can cause an outbreak to persist because seemingly healthy pigs may shed the virus without being picked up by serological screening after an outbreak (Ribbens *et al.,* 2004).

Despite several reports of epidemiological connections between CSF virus infections in domestic pigs and wild boar, the significance of wild boar as a virus reservoir and potential source of infection for domestic pigs remains uncertain (Kern *et al.,* 1999). When vulnerable pigs were given swill containing products from CSF-infected pigs. It resulted in new infections. Swill feeding is thought to be the source of the epidemics in 1996 in Germany and in 1986 and 2000 in the United Kingdom. Despite the ED's ban on swill feeding, experts believe that (illegal) swill is still a significant concern (Ribbens *et al.,* 2004). Inadequately cleaned and disinfected livestock trucks that are tainted with the excretions and secretions of diseased pigs could be a major source of viral transmission. Human-to-human transmission of the CSF virus is also commonly cited as a potential pathway for virus transmission. The likelihood of iatrogenic transfer between herds is thought to be low under present management practices and sanitary measures (Ribbens *et al.*, 2004). It is possible for CSFV to spread through the air both within and across herds. However, in some investigations, airborne transmission was only detected within 250 sick people, whereas in other studies, it was discovered within a 1 kilometer radius. Thus, the maximal airborne transmission distance of the virus is still unknown. Furthermore, climatological and geographic factors are likely to have a significant impact on airborne transmission; however, other factors, such as viral strain, may also have an impact (Ribbens *et al.,* 2004).

**Transmission of ASF**

ASFV is spread via soft ticks, wild suids, and domestic pigs. It is thought that the most significant reservoir host in Africa and the first vertebrate host of ASFV were common warthogs (Phacochoerus africanus) (Costard *et al.*, 2013). Tampan (Ornithodoros spp.) bites, ingestion of contaminated meat, direct contact with an infected animal, and indirect contact with contaminated things are the ways in which the virus is spread (Penrith & Vosloo, 2009). One of the main causes of the virus's proliferation and transmission is the transportation of contaminated goods and diseased animals (Sánchez-Vizcaíno *et al.*, 2015). Another potential pathway is short-distance airborne transfer (Penrith & Vosloo, 2009). Up to 48 hours after swallowing the virus, the stable fly (Stomoxys calcitrans) can also serve as a mechanical vector (Mellor *et al.*, 1987). Although sexual transmission of ASFV is unknown, it can be eliminated by vaginal secretions (Penrith & Vosloo, 2009).

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Direct contact between sick animals and consumption of infected pork or other items can spread ASFV (Boklund *et al.*, 2018; Chenais *et al.*, 2019; More *et al.*, 2018). Items that can spread infection include clothing, transport trucks, and feed supplies. In warthog burrows, Ornithodoros spp. is mostly spread by soft tick vectors. In areas where they are found, they may potentially contribute to transmission on pig farms. It has been shown that continuously infected animals can spread the infection to uninfected ones (de Carvalho Ferreira *et al.*, 2013; Gallardo *et al.*, 2015). Nevertheless, there is insufficient data to support the idea that long-term carrier status plays a part in ASF transmission in the field (Petrov *et al.*, 2018). In the current epidemic in Europe, outbreaks in domestic pig herds have peaked over the summer, leading to increased conjecture regarding the possible contribution of mechanical arthropod vectors such stable flies (Stomoxys calcitrans) or tabanids (Olesen *et al.*, 2018; Boklund *et al.*, 2018). However, there is currently insufficient proof to conclude that these contribute significantly to the disease's spread.

**Clinical symptoms**

**Clinical symptoms caused by CSF**

According to Ribbens *et al.* (2004), the oronasal route is often how the CSF virus infects pigs naturally. The disease typically only affects a small number of animals during the first 10 days of an outbreak, but beyond that, morbidity could affect up to 100% of the herd. A susceptible herd may also have mortality rates as high as 100%. After two to four days of incubation, the classical form of CSF typically manifests as anorexia, depression, and hyperthermia, followed by vomiting, diarrhea, constipation, and pneumonia brought on by opportunistic infections. There may also be neurological symptoms like tremors, paralysis, circling, and occasionally convulsions. The symptoms of classical swine fever are given in Figure 1.

**Acute form of CSF**

Most frequently, the acute form is seen in piglets up to 12 weeks of age. Pyrexia is frequently observed in adults, the temperature may not rise above 39.5°C, but it is typically greater than 40°C. Anorexia, fatigue, conjunctivitis, swollen and discolored lymph nodes, respiratory symptoms, subcutaneous hemorrhages, and constipation followed by diarrhea are the first symptoms. Neurological symptoms include seizures, incoordination of movement, and a staggering stride with paralysis in the rear legs.

During the second and third weeks following infection till death, the ear, tail, abdomen, and inner side of the limbs typically exhibit the typical skin hemorrhages. The infected animal excretes the virus through its feces, urine, and saliva. On postmortem inspection, pathological alterations are most frequently seen in the kidneys, spleen, and lymph nodes. The lymph nodes become enlarged, bloated, and bleeds. From petechiae to ecchymotic hemorrhages, kidney hemorrhages can range in size. The larynx, heart, epiglottis, and bladder can all have petechiae, which can also be found throughout the abdominal and chest serosae. Often, there occurs non-purulent encephalitis. The CSF virus induces immunosuppression and severe leukopenia, which frequently results in subsequent respiratory or gastrointestinal infections. The veterinarian may be misled by the symptoms of these secondary diseases, which might overlap or conceal the most common CSF symptoms. The clinical symptoms become less specific as the infected pigs (fattening and breeding animals) age, and recovery with antibody production may take place. Twenty-three weeks after CSF viral exposure, antibodies against the virus can be found (Moennig *et al.*, 2003).

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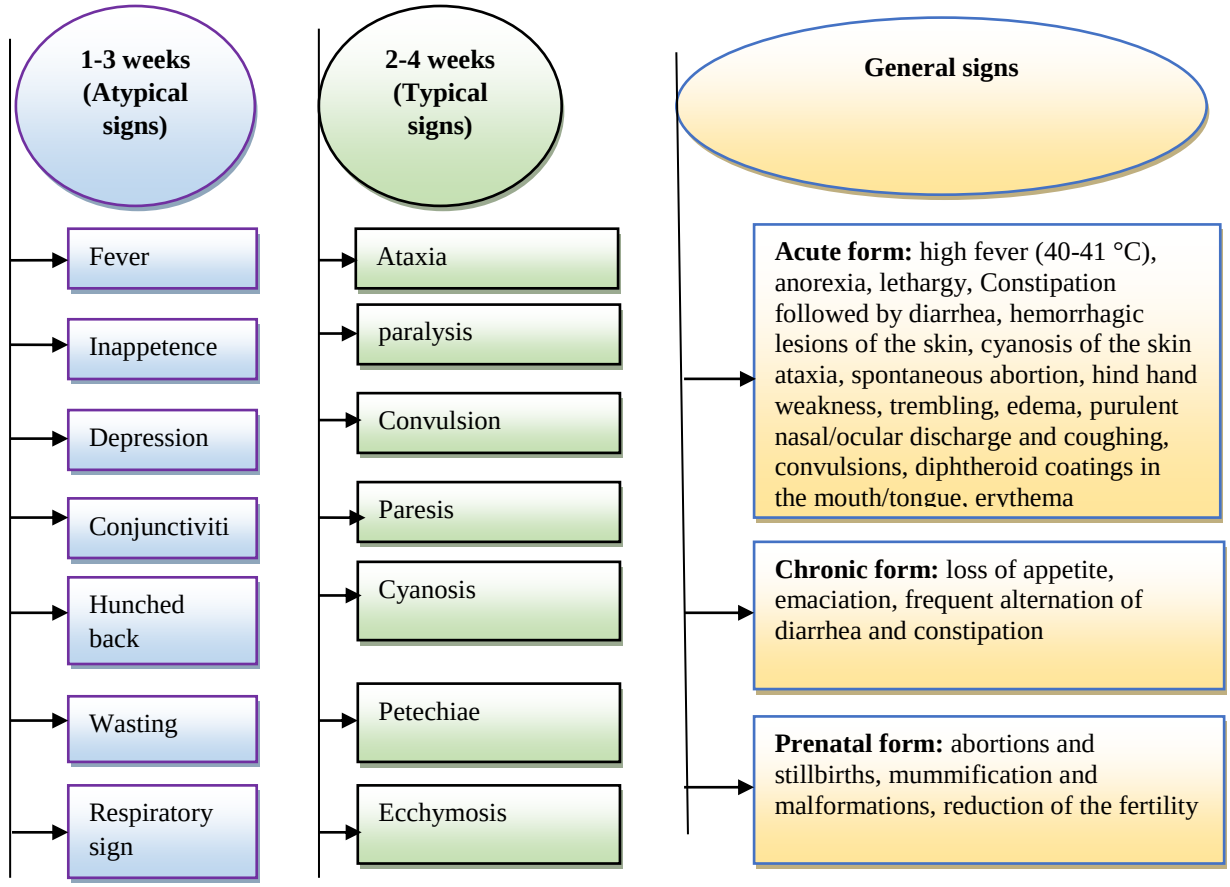


**Figure 1: Symptoms of classical swine fever (CSF)**

**Chronic form of CSF**

Chronic form usually follows the acute form, followed by an appearance of brief recovery, with death in 1–3 months. Chronic CSF is invariably lethal. It appears when pigs are unable to fight off the infection with a strong immunological response. Initial symptoms are comparable to those of an acute infection. Later, there are mainly non-specific symptoms, such as wasting, chronic enteritis, and intermittent fever. Before dying, animals may live for two to three months. The CSF virus is continuously shed from the moment clinical symptoms appear until death. Serum samples may momentarily contain antibodies since the immune system begins to create them even while they are unable to eradicate the virus from the host. The virus neutralizes the antibodies as a result, making them undetectable. Pathological alterations are less common, particularly the absence of organ and serous hemorrhages. The rectum, ileocecal valve, and ileum frequently have necrotic and ulcerative lesions in animals with chronic diarrhea (Moennig *et al.*, 2003).

**Prenatal form of CSF**

The CSF virus can spread through pregnant animals' placenta and infect fetuses at any point during pregnancy, even though the sow's infection course is frequently asymptomatic. The duration of gestation and the aggressiveness of the virus respectively, determine the outcome of a transplacental infection. An infection in the early stages of pregnancy can cause deformities, mummification, abortions, and stillbirths. A decrease in the holding's fertility index will result from all of this. Piglets that are persistently viraemic may be born to sows infected between 50 and 70 days of pregnancy. These piglets may be clinically normal at

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birth and live for several months. Following birth, they could have congenital tremor, wasting, or poor growth. The term "late onset CSF" describes this infectious history, because they are a deadly virus reservoir and continuously shed significant amounts of virus, these piglets propagate the disease and keep the pig herd infected. Similar circumstances apply to cattle that have a prolonged BVD viral infection. According to Moennig *et al.* (2003), CSF must be taken into account when making a differential diagnosis of decreased fertility brought on by parvovirus infection, PRRS, leptospirosis, and Aujeszky's disease.

**Clinical symptoms caused by ASF**

ASF takes 2–7 days to incubate, and the length of time varies depending on the infection method (Mebus, 1988). It is possible to categorize the disease as acute, subacute, chronic, or peracute (Wardley *et al.*, 1983). Although hemorrhages will be seen at post mortem, the majority of animals in the peracute form pass away with few gross lesions and no clinical symptoms (Wardley *et al.*, 1983). While 100% morbidity and mortality may occur, certain animals may show clinical symptoms including temperature (41–42 °C), fast breathing, or skin hyperemia (Mebus, 1988). Both acute and subacute forms are frequently seen and are brought on by strains that are extremely and moderately virulent, respectively (Mebus, 1988). Abortion, vomiting, epistaxis, cyanosis in the legs, ears, and tail, anorexia, lethargy, inactivity, raised body temperature (40–42 °C), and death with 90–100% mortality within 7–10 days are the symptoms linked to the acute type (Wardley *et al.*, 1983; Sánchez-Vizcaíno *et al.*, 2015; Sánchez-Vizcaíno *et al.*, 2019 ). Acute diarrhea following secondary bacterial infection is linked to this condition (Mebus, 1988). The acute form is characterized by widespread necrosis and bleeding of lymphoid tissues, splenomegaly, and early leucopenia (Sánchez-Vizcaíno *et al.*, 2019). Clinical indications of the subacute disease are comparable to those of acute instances, but they are less severe ((Wardley *et al.*, 1983). For 10–12 days, there is a fever and decreased feed intake (Mebus, 1988). The surviving animals can recover in two to three weeks, whereas the mortality rate can range from 30 to 70% (Sánchez-Vizcaíno *et al.*, 2019). Lesions can be seen in bacterial coinfection; however a chronic illness caused by infection with a low virulent strain does not exhibit any particular clinical symptoms or vascular lesions (Sánchez-Vizcaíno *et al.*, 2015). Clinical symptoms include pneumonia, stunting, emaciation, arthritis, and skin ulcers may be observed, and it may last longer. Hemorrhages may also occur during post-mortem, coupled with fibrinous pericarditis and pleuritis (Wardley *et al.*, 1983). The chronic variety is also linked to skin lesions, soft, painless joint swelling, poor growth, and fluctuating fevers (Mebus, 1988).

**Diagnosis**

**Diagnosis of CSF**

Due to the modest virulence of the prevalent strains of the CSF virus, clinical diagnosis is challenging, particularly in older animals. Laboratory confirmation of the disease is typically necessary, even for secondary cases during big outbreaks because the clinical indications of CSF are not pathognomonic. Before becoming clinically noticeable, CSF frequently requires multiple cycles of amplification and has an incubation period of a few weeks depending on the herd. Therefore, the control of disease would greatly benefit from pre-clinical detection. Since fever is a fairly noticeable symptom in CSF, it would be very helpful if pigs could be microchipped or screened in large numbers (for instance, using infra-red devices) to identify the ones with the highest temperatures for further analysis and sample. Immunofluorescence or immunoperoxidase staining and antigen-capture ELISA allow rapid detection of viral antigens in tissues and RT-PCR assays facilitate very rapid identification of viral nucleic acids. To differentiate the swine fever virus from other pestiviruses, monoclonal antibodies

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might be used. The ELISA is a quick and easy way to screen ill or pyrexic pigs, and it has the benefit of being able to analyze a lot of blood samples. In addition to being quicker and more costly, RT-PCR is also more sensitive, making it suitable for preclinical diagnosis and pooled samples. It could be used to verify that pigs at an abattoir were not infected with viruses at the time of slaughter if it were automated enough to analyze a lot of blood samples. Since the amount of virus in meat is probably quite tiny and the amount of testing that would be necessary is very large, there is currently no practicable way to screen imported pig meat in bulk. As has been done for other disorders, testing meat juices for CSF antibodies would be a potential substitute strategy. The CSF virus suppresses the immune system, and antibodies specific to the virus develop slowly. The Dutch tested 2.1 million blood samples for antibodies in 1997/1998; however, the tests are not completely CSF specific and can detect antibodies induced by other pestiviruses (bovine viral diarrhoea virus and border disease virus), which occasionally infect pigs. Therefore, large-scale serology is possible with commercially available ELISA kits. Consequently, it could be necessary to use the time-consuming and slow comparative neutralization tests to validate positive ELISA results. A comparative ELISA system could be a solution to this issue (Paton and Greiser-Wilke 2003). The polyclonal serum generated against cell culture-adapted CSF viral antigen, formed the basis for a sandwich ELISA test that was effective in detecting the virus antigen in the tissues of disease-symptomatic pigs as well as from pigs that had been killed for human consumption (Sarma *et al.*, 2007). Various detection methods for classical swine fever are given in Figure 2.

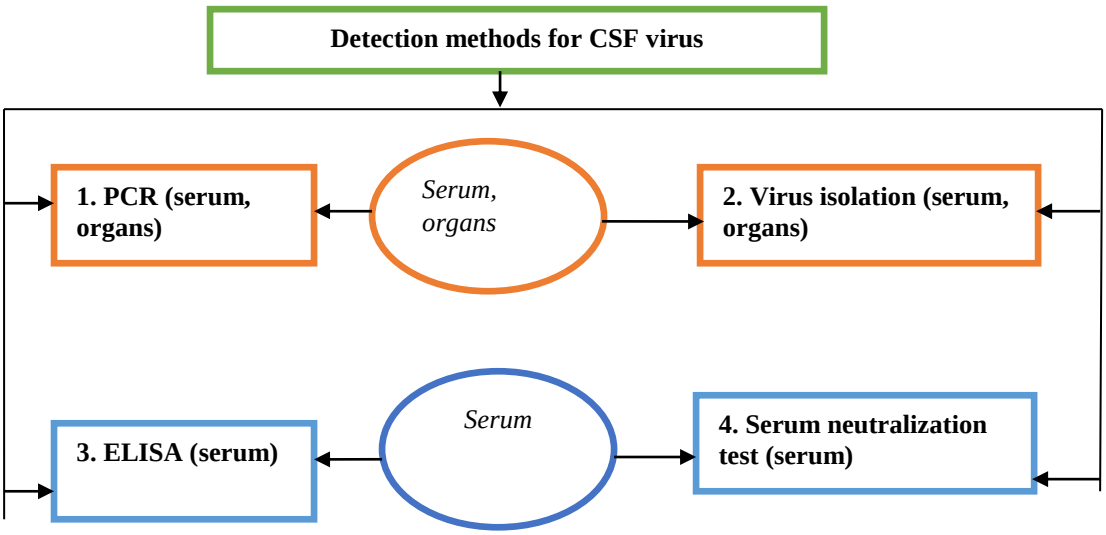


**Figure 2: Various detection methods for classical swine fever**

A reverse transcriptase PCR assay for the classical swine fever virus (CSFV) using fluorogenic probe hydrolysis (TaqMan) was created and tested on experimentally infected pigs. Viral RNA from related pestiviruses was not amplified by the assay, but it did detect CSFV, which represents distinct phylogenetic groupings (Risatti *et al.*, 2003). When making a differential diagnosis for erysipelas, porcine reproductive and respiratory syndrome (PRRS), purpura haemorragica, cumarin poisoning, post-weaning multisystemic wasting syndrome (PWMS), porcine dennatitis and nephropathy syndrome (PDNS), Sabnonella or Pasteurella infections, or any enteric or respiratory syndrome with fever that does not improve with antibiotic treatment, CSF must also be taken into account.

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**Diagnosis for ASF**

ASFV can currently be detected using a number of early diagnostic methods, such as PCR, enzyme-linked immunosorbent assay (ELISA), fluorescence quantitative PCR, colloidal gold fast strip, hemadsorption, loop-mediated isothermal amplification, and other serological assays (Ward *et al.*, 2021). Serological testing has been utilized extensively in projects to eradicate and control the disease since the presence of ASF antibodies indicates prior infection. Serology is therefore appropriate for identifying possible animals that are infected with viruses during epidemics. ELISA can be used extensively for screening since it is the most widely used serological test (Mao *et al.*, 2019). Purified pB602L was used as a diagnostic antigen in an indirect ELISA method for the detection of anti-ASFV, setting the stage for the development of ASFV early diagnosis kits and functional research of the pB602L protein. For detection of ASF, chemiluminescent immunoassay (CLIA), luciferase immunoprecipitation assay (MB-LIPS), loop-mediated isothermal amplification (LAMP), recombinase polymerase amplification (RPA) are also used. The various methods of detection of ASF are given in Table 1.

**Table 1: Various methods of detection of ASF**

| Methodology | Detection time (minute) | References |
|---|---|---|
| Enzyme-linked immunosorbent assay (ELISA) | 120-240 | Tabatabaei and Ahmed, 2022. |
| Ploymerase chain reaction (PCR) | 60-180 | Hwan *et al*., 2023 |
| loop-mediated isothermal amplification (LAMP), | 30-90 | Tanner and Evans, 2014; Notomi *et al*., 2000 |
| recombinase polymerase amplification (RPA) | <30 | Jailani and Paret, 2023; Ahamed *et al*., 2024 |
| chemiluminescent immunoassay (CLIA), | 60-120 | Zhao *et al*. 2024; Fu *et al*., 2024 |
| luciferase immunoprecipitation assay (MB-LIPS) | 120-180 | Ding *et al*., 2022 |

(Table adopted from Gu *et al*., 2024)

## Vaccines and vaccination

### Vaccination against CSF

One of the most important tools in CSF control measures is vaccination. A variety of vaccinations, such as marker vaccines, and live attenuated vaccines, were developed (Greiser-Wilke & Moennig, 2004; Moennig, 2004). Commercial CSF live, attenuated vaccines (LAVs) provide protection within the first week of vaccination and are both safe and effective. With an exceptional safety record, these LAVs—originally the C strain—have been in use for more than 50 years globally. In the European Union, a live, attenuated vaccine administered via baits has been successfully used to vaccinate wild boars orally. When parenteral vaccine distribution is not practical, oral immunization has proven to be an essential method of controlling CSF.

Around the world, lapinized vaccinations are still being used to prevent the disease in domestic pigs. The CSF attenuated live vaccine (GP vaccine) was created in 1969 by passing the virus through guinea pigs. On an experimental basis, the C strain of the virus was also utilized for oral vaccination to reduce the illness (Kaden *et al.*, 2000). Lapinized vaccinations are safe and produce a high level of neutralizing antibodies. By exposing the inoculated animals to a virulent virus as soon as five days after immunization, the effectiveness of the vaccine can be shown. However, the vaccination only provides immunity for roughly six months, and there is the issue of making large quantities of the vaccine at once. Additionally,

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it is challenging to standardize the viral concentration in every vaccine batch. Limited quantities of the lapinized vaccine are still being produced by a few Indian state institutes of veterinary biology. Not even 1% of the nation's pig population can be immunized with the lapinized vaccine currently on the market. Thus, it is imperative that enough CSF vaccine doses be produced in cell culture systems for our country's pig population. By passaging in cell lines, especially the PK 15 cell line, the CSF virus can be readily attenuated. The lapinized vaccine and the cell culture attenuated vaccine both induce a high level of protection and are safe. The primary benefit of the cell culture vaccination is its large-scale production and the ease with which the viral concentration in the cell culture system can be measured.

**Marker vaccine for CSF**

The use of live attenuated vaccines to prevent the disease has a significant drawback in light of current international trade regulation because the antibody pattern produced by the vaccine virus is similar to that of animals recovering from an infection. So, it is impossible to discriminate between vaccinated and infected animals. In order to solve this issue, so called marker vaccines that contain the single viral surface proteins have been developed and put into use (Van Rijn *et al.,* 1996).

Two subunit vaccines that contain the CSF virus's glycoprotein E2 have been created. Despite being safe, marker vaccinations are said to provide a lower level of protective immunity than live vaccines. There have been reports of further advancements in CSF marker vaccines through the creation of viral vector vaccines, DNA vaccines, and infectious cDNA clones of the CSF virus that have undergone molecular changes. Researchers are now able to create DNA copies of the whole RNA genome of CSFV thanks to recent advancements in molecular technology. Pigs and rabbits have been used to describe two recombinant CSFVs (Flc2. Flc3) that were transcribed from a DNA copy of the C-strain's genome in vivo. The findings showed that the two recombinant viruses in pigs and rabbits had maintained the beneficial immunogenic and biological characteristics of the parent C-strain. After vaccination, both chimeric viruses offered good clinical protection against a challenge with virulent CSFV one or two weeks later (de Smit, 2000). The envelope glycoprotein E2 has also been used to create a subunit vaccination against CSFV. Therefore, this subunit vaccine has the potential to be a marker vaccination since antibodies against Ems and/or NS3 can be used to distinguish between vaccinated and infected pigs (BollIna *et al.,* 1999).

**Vaccination against ASF**

The most effective technique to prevent and control anti-ASFV is vaccination. It has been found that inactivated vaccinations are ineffective against ASF. However, On June 1, 2022, Vietnam produced the first vaccine to prevent African swine fever. The vaccine strain was created by removing the I177L gene from the extremely virulent ASFV isolate Georgia 2007/1 (ASFV-G), and it is a live attenuated vaccine (LAV) (Qi *et al.,* 2023). In Vietnam, AVAC ASF LIVE, which is based on the ASFV-G-Δ-MGF strain, is the other commercially available ASF vaccine (Juszkiewicz *et al.,* 2023).

**Control and prevention**

**Control and prevention of CSF**

Control of CSF can be achieved through eradication, vaccination or exposure prevention. In most countries, efforts are made to prevent exposure by limiting or outlawing the importation of live pigs, fresh pork, underheated pork products, and other potential virus sources

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(imported swine embryos and semen, biologics). Additionally, it is illegal to feed uncooked waste food and to dump trash from ships while they are in port. The primary method of disease control is immunization. Live pigs and fresh pig meat can only be imported from areas or countries where no CSF has been detected for a full year and no vaccinations have been administered during that time. According to the European Union's precautionary measures for commerce with third countries. In order to control the spread of the CSF virus in the areas surrounding the affected farms, it is crucial to stamp out sick pig herds and restrict the movement of live pigs and pig meat. There is widespread agreement that a number of steps such as structural adjustments to the pig industry, including trade, must be taken to lessen the vulnerability of areas that are at danger. Implementing suitable programs, however, may prove challenging. The management of CSF in wild boar remains an open issue. It is crucial to have thorough knowledge of the illness state in the wild boar population, and new tactics must be developed. Working with CSFV proved challenging, and significant advancements were only made possible when advanced virological techniques were developed and made available, particularly in the previous 15 years (Moennig, 2000). Various control methods for classical swine fever is given in Figure 2.

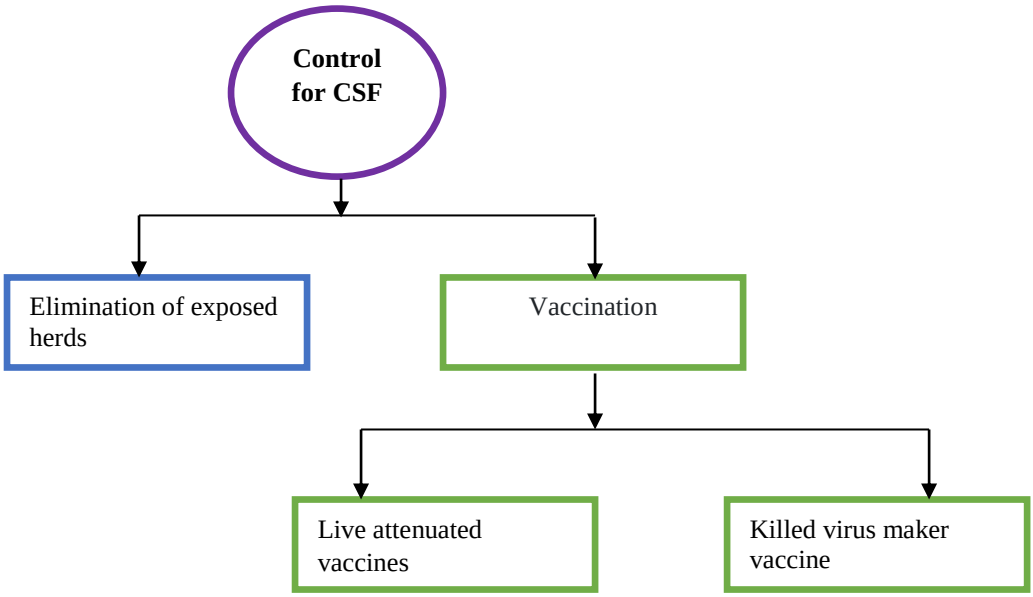


**Figure 3: Control methods for classical swine fever**

**Control and prevention of ASF**

To effectively control the ASFV infection, there is currently no vaccination (Sánchez-Vizcaíno *et al.*, 2015). ASFV may persist in meat and meat products, feeds, and a variety of reservoirs, such as ticks and wild boars, making it very difficult to eliminate once the disease has taken hold in a given area (Wardley *et al.*, 1983). One successful measure to stop the spread of the infection is culling a lot of pigs. Nevertheless, it has financial, ethical, and environmental issues (Penrith, 2009). Strict biosecurity protocols continue to be the best way to prevent ASF. There must be restrictions on the movement of pigs from affected areas. There should be no interaction between domesticated pigs and wild boars. Instead of feeding pigs contaminated trash from international airports and dock, which are a major source of the virus, it should be burned (Sánchez-Vizcaíno *et al.*, 2015). Other successful methods to ASF preventive efforts include creating user-friendly practical biosecurity courses, guaranteeing good communication among all concerned stakeholders, raising awareness among all parties,

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and offering incentives like compensation and insurance fees (Gavier-Widén *et al.*, 2015). Given the geographic location, economic conditions, epidemiological state, and ASF status in nearby nations, contingency plans ought to be created and prepared for implementation when needed (Sánchez-Vizcaíno *et al.*, 2015).

## CONCLUSION

African swine fever (ASF) and classical swine fever (CSF) are highly contagious viral diseases of pigs, causing a persistent threat to the pig industry worldwide. The diseases cause pig mortality, growth retardation and disruption of reproduction as well as lower pork quality. Vaccination is used where the disease is endemic to prevent virus expansion. Numerous techniques, such as virus isolation, fluorescent antibody test (FAT), antigen capture antibody enzyme-linked immunosorbent assay (ELISA), reverse-transcription polymerase chain reaction (RT-PCR), virus neutralization test (VNT), and antibody ELISA, have been developed for the purpose of diagnosing CSF. For the detection of ASF, several techniques have been developed including PCR, ELISA, chemiluminescent immunoassay (CLIA), luciferase immunoprecipitation assay (MB-LIPS), loop-mediated isothermal amplification (LAMP), and recombinase polymerase amplification (RPA). Early diagnosis, prompt removal of infected sources, and disruption of transmission pathways are current strategies for controlling and preventing both diseases.

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### Author's Contribution

Sajana Rai wrote the manuscript.

### Conflict of Interest

The author declares no conflict of interest.

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