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Vesicular Exanthema of Swine: A Historical Curiosity for Global Pig Industry

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ABSTRACT

Vesicular exanthema of swine (VES), an acute, febrile, infectious viral disease of pigs derives its significance in veterinary medicine from its first detection in Southern California, USA in 1932 having clinically look-a-like features with three other prevalent porcine vesicular diseases caused by foot-and-mouth disease virus (FMDV), vesicular stomatitis virus (VSV), swine vesicular disease virus (SVDV). The causative agent belongs to the genus *Vesivirus* in the family *Caliciviridae.* VESV serotypes are highly infectious in swine with morbidity of up to 90%. The spread of VES occurs chiefly in three ways: the feeding of raw garbage containing infected raw pork scraps, direct contact with infected swine, and contact with mechanical carriers, including people and vehicles. Vesicular lesions in the oral cavity on the epithelium of the snout, lips, nostrils, tongue, feet and mammary glands, soles, coronary bands and interdigital areas of the feet with lameness were the hallmark of disease in all species. Vesicles alike to those of FMD, VS and SVD are observed in VES, hence all these diseases are considered for differential diagnosis of VES. Clinical materials from vesicles e.g., vesicular fluid, epithelium covering vesicle should be collected in sterile glycerol phosphate buffer solution for diagnosis using molecular techniques. VESV can be readily propagated in mammalian cell cultures of African green monkey kidney or pig kidney cells. No vaccine was developed for VES. Being eradicated, there is no current threat of VES.

HIGHLIGHTS

- Vesicular exanthema of swine is an infectious viral disease of pigs that is clinically indistinguishable from that of foot and mouth disease in swine thereby bearing a significance.
- The disease is eradicated being a historical curiosity in the global pig industry.

Keywords: Vesicular exanthema, pig, calicivirus, FMD, VS

Vesicular exanthema of swine (VES), an acute, febrile, infectious viral disease of pigs derives its significance in veterinary medicine from its first detection in Southern California, USA in 1932 having clinically look-a-like features with three other prevalent porcine vesicular diseases caused by foot-and-mouth disease virus (FMDV), vesicular stomatitis virus (VSV), swine vesicular disease virus (SVDV). VES only ever appeared among Californian pig keepers in the USA and its restricted host range was one of its differentiating features from FMD. Between the year 1932 and 1956, VES triggered huge economic loss to the US swine industry and was contained with the infected pigs being slaughtered (Madin, 1975). The US Secretary of Agriculture declared a national emergency and enforced eradication efforts with instructions for suitable treatment of garbage and fish meant for swine. In consequence, the disease got eradicated in swine in 1959 being at present a historical curiosity. Although the original source of the virus in its first appearance could never be determined, no further outbreaks were reported since its eradication, therefore is classified as exotic to the US.

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Researchers predicting the source of the virus

Until 1972, the source of the virus causing VES remained undetermined. In the year 1972, abortion enzootics and vesicular lesions among sea lions were encountered in San Miguel Island, California, and upon investigation, an essentially similar virus physico-chemically indistinguishable from VESV subsequently named as San Miguel Sea Lion Virus (SMSV) was isolated from the throat and rectal swabs. The virus produced identical vesicular lesions when inoculated into swine (Smith *et al.,* 1973). During early 1973, SMSVs were isolated from sea lions, northern elephant seals, fur seals and certain kinds of fish and continue to circulate in wild and domesticated animals, marine mammal species and fish along the California Pacific coastline of the United States. However, spontaneous outbreaks of VE in swine caused by SMSV have never been substantiated. These findings led to an assumption that the source of VESV could be from waste seafood fed or untreated garbage fed to pigs.

Flashbacks from history

On 23rd April 1932, a disease clinically indistinguishable from FMD afflicting only swine was reported on a ranch near Buena Park, Orange County, California. The disease then spread to Los Angeles and San Bernardino Counties, further diagnosed as FMD. When a suspected case is reported, animal inoculation tests with laboratory interventions are required to ascertain whether the infection is vesicular exanthema, vesicular stomatitis or FMD. But the virus from the 1932 outbreak could not succeed to produce lesions in 24 guinea pigs, 2 calves, 2 heifers,1 adult cow, and 2 horses (Traum, 1934). In March 1933, a disease again restricted to swine and clinically similar to the 1932 outbreak appeared in San Diego County, California. The virus from this outbreak was sampled and tested in a variety of animals as done at earlier instance. This time, the infection was established in all of 15 swine, in 4 of 9 horses, but all 7 cattle and 37 guinea pigs remained uninfected (Traum, 1934). Opposite/contrasting results were produced when test animals were inoculated with tissue infected with FMD virus. Because of such findings, no official diagnosis could conclusively be made, while all animals involved were slaughtered. Another disease of swine was described in 1934 by Jacob Traum of the University of California, as the cause of the 1933 outbreak. Dr. Traum reported that the agent that caused the disease was different both from that of FMD and vesicular stomatitis. Animals recovered from it were not immune to those two diseases. Dr. Traum also considered the 1932 outbreak to have been caused by this new agent, for which he suggested the name 'vesicular exanthema'.

The lesions of vesicular exanthema are vesicles (blisters) that develop on the feet and snouts of infected pigs and rupture quite rapidly. The name assigned to the disease tends to describe the typical lesions e.g., 'vesicular' means vesicles, or blisters, while 'exanthema' refers to their eruption. The source of the infection could be traced to swine near Cheyenne, Wyoming, Nebraska which had been fed uncooked garbage from transcontinental trains originating from California. Thus, VE appeared for the first-time outside California at a plant manufacturing biological in Grand Island, Nebraska in June 1952. Few literatures also mention that the only case outside the USA was in slaughtered pigs on ship from the USA bound for Hawaii in 1947 and in pigs fed uncooked raw garbage containing pork scraps from an American military base in Iceland in 1955. After the report of VES in Nebraska in June 1952, subsequently by September 1953 the disease spread to neighboring herds in 42 states. Enactment of federal and state garbage-cooking laws coupled with slaughter policy with a ban on feeding of uncooked garbage to pigs resulted in a rapid decline in the disease incidence. The last case of VES was reported in 1956 in New Jersey. Finally, the Secretary of Agriculture declared the successful eradication of VES and the disease was declared to be exotic/foreign animal disease in the USA in 1959 (Madin, 1975; Smith and Akers, 1976).

Etiological agent

VESV is a calicivirus. The name takes its origin from the characteristic appearance of the virus particles on electron micrographs consisting of a scalloped border with cup-like indentations on its surface, from which the Latin name 'chalice' or 'calyx' (means cup) is derived. VESV is believed to have originated from SMSV that is biophysically indistinguishable from VESV and is capable of producing vesicular disease in swine. There are 13 serotypes of VESV and the virus is closely related to at least 14 other serotypes of caliciviruses found in SMSV group. VESV consists of a capsid with 32 cup-shaped

depressions that are non-enveloped, round with icosahedral symmetry. The isometric capsid has a diameter of 35-39 nm. Capsids appear round to hexagonal in outline. The genome is 7900 nucleotides long, non-segmented with a single molecule of linear positive-sense, single-stranded RNA and the virus belongs to the genus *Vesivirus* in the family *Caliciviridae.* The 5'-end of the genome has a viral protein genome-linked (VPg) and the 3'-terminus has a poly (A) tract. The genomic nucleic acid itself is infectious being positive sense. The virus measures 0.000002 inch of diameter.

Strain variations

Feline calicivirus (FCV) and VESV are two species within the *Vesivirus* genus. Several additional viruses e.g., canine calicivirus and mink calicivirus may be classified as vesiviruses but have not been recognized as a species (Knowles and Reuter, 2012). There are 40 serotypes within VESV phylogenetically referred to as marine vesiviruses that are genetically similar and non-host-specific. These include SMSV that is morphologically similar to VESV. Thirteen serotypes are known as VESV (i.e., VESV-A48, VESV-B34), 17 as SMSV (i.e., SMSV-1, SMSV-2), and rest others are named as per the hosts they were recovered from e.g., bovine calicivirus Bos-1 (BCV Bos-1) and Stellar sea lion virus V810 (SSLV-V810).

Five virus types have been identified as A-48, B-51, G-52, D-53, and E-54. The letter indicates the order of isolation and the numbers indicate the year when they were found. Out of these five types, none immunized against the other. Only B-51 has been identified as the causative agent of outbreaks outside California. This 'type' classification was initiated by Dr. R.A. Bankowski. Dr. A.B. Crawford in 1937 isolated four strains of the virus, identified as A, B, C and D, of which all affected swine, but only B and D were infectious to the horse.

San Miguel Sea Lion Virus (SMSV)

Although sometimes listed as a separate virus, it is now clear that this virus is the same as VESV and was indeed the source of the disease in swine. It was recovered in 1972 from samples collected from California sea lions inhabiting San Miguel Island exhibiting several signs including abortion and vesicular lesions of the flippers. This virus produced lesions when inoculated into swine. In California, carcasses of seals and sea lions were frequently fed to swine thereby opening the window of infection. Retrospective evidence suggested that the multiple antigenic types of VESV were generated in the natural hosts of the virus, sea lions, rather than in swine. Seven antigenic types have been identified from the small number of SMSV isolates that have been made since 1972.

Morbidity and mortality

VESV serotypes are highly infectious in swine with morbidity of up to 90% (Smith and Akers, 1976). Animals generally recover 1-2 weeks after the onset of clinical signs. Clinical disease seldom leads to mortality with VESV-induced infections (Gelberg and Lewis, 1982) or the mortality is less than 5%. There has been no report of long-term carriers of VESV. However, infected pigs harbouring the virus are threats for other susceptible animals. Despite eradication of VES from swine, SMSV and other serotypes are still circulating along the North American Pacific coastline. The presence of marine vesiviruses in wild and marine mammals indicates that VESV is still a threat to the US swine industry. Further research is needed to definitively determine the reservoir hosts for VESV.

Geographic distribution and susceptible hosts

California in USA was the origin of VES in 1932 and the virus has not been reported in pigs in any other regions of the world. Although VESV has been eliminated from domestic swine, other marine vesiviruses likely continue to circulate or remain prevalent in natural reservoirs and several marine and terrestrial mammals along the California coastal zone or Pacific coast of the United States (Smith and Akers, 1976). Collectively, VESV, SMSV, and others within the VESV species are considered marine vesiviruses. Low levels of serum neutralizing anti-SMSV, anti-VESV and antibodies to several other closely related caliciviruses have been detected in both marine and terrestrial animals e.g., feral swine, donkeys, cattle, foxes, buffalo, California sea lions, California gray whales, sperm whales, and see whales near the California coastal zone (Smith and Latham, 1978). Natural VESV infections have occurred in a variety of marine and terrestrial animals including pigs, cattle, horses, skunk,

primates (including humans), pinnipeds (seals), cetaceans, reptiles and fish (Prato *et al.,* 1974; Smith *et al.,* 1998; Knowles and Reuter, 2012). Pigs of all age groups and all breeds appear to be susceptible. Other indistinguishable viruses from SMSV have been reported in reptiles (Smith *et al.,* 1986), cattle (Smith *et al.,* 1983), primates (Smith *et al.,* 1985) and skunks (Seal *et al.,* 1995). SMSV occurs in sea lions, fur seals, elephant seals, opal-eye fish and several other marine animals of the western coast of the United States. Over half of the marine vesiviruses have experimentally been capable of producing vesicles in swine including bovine serotypes (Smith *et al.,* 1980; Gelberg and Lewis, 1982; Knowles and Reuter, 2012). It is speculated that oceanic fishes are the suspected natural reservoirs for VESV (Smith *et al.,* 1980).

Zoonotic potential

Though human infections have occasionally been documented, marine vesiviruses are not considered a serious public health threat. VESV infection in humans is inferred, but it went unproven. There was only ever a solitary report of VES incidence in a human acquired during laboratory work, not from an infected animal. A 32-year-old male researcher working closely with purified calicivirus isolates developed flu-like illness followed by blisters on his hands and feet that subsided and blisters healed within a couple of weeks. Finally, SMSV serotype 5 was isolated.

The second partially documented human case report was a handler of Stellar's sea lions that developed deep, painful blisters on the mouth and facial area that was originally diagnosed as herpesvirus infection, but one month later, a calicivirus closely related to SMSV was isolated from the washings of the patient's throat (Smith *et al.,* 1998). Records of human infection, neutralizing antibody to SMSV and the non-specific host range of marine caliciviruses suggest that VESV infection could extend to humans (Smith *et al.,* 1998).

Epidemiology and transmission

The spread of VES occurs chiefly in three ways: the feeding of raw garbage containing infected raw pork scraps, direct contact with infected swine, and contact with mechanical carriers, including people and vehicles. If we look back to the pages of history, it will be evident that the access of pigs to untreated/infected/uncooked pork scraps/garbage/fish scraps has been the key factor behind origin of novel outbreaks as exemplified in 1932 in California. In California, the probability of VE in garbage-fed swine was a thousand times more in comparison to that in grain-fed swine. Rapid transmission was reported in the original outbreak through direct contact of infected pigs and fomites owing to the more contagious nature of the disease in pigs. Oral infection with VESV requires 100-1000 times the amount of virus needed to produce lesions by intradermal inoculation into the snout. Direct contact with vesicular fluid, oronasal and lachrymal secretions and vesicle coverings (Knowles and Reuter, 2012), urine, feces, insemination, blood transfusion, feeding of raw or improperly cooked meat can spread the virus. Large amount of virus is liberated during rupture of blisters with the fluid and epithelium that constitute the cover. Once established within a herd, further pig to pig transmission is facilitated by direct contact. Neither long-term carriers nor aerosol mode of spread has been demonstrated for VES.

The opal-eye fish (*Girella nigricans*) is believed to be the primary host of the calicivirus that may further be passed to pinnipeds and swine. The method of spread of the virus among sea lions and other sea mammals is speculative that could be through ingestion of infected fish or marine creatures, by coastal contamination, or direct contact. Human beings generally do not contract VE, but can carry the virus to swine on their clothing especially their shoes particularly when they have moved through the infected premises or have been in contact with infected/diseased swine.

Pathogenesis/disease progression

The incubation period of VES in both spontaneous/natural and experimental situation usually varies from 24 to 72 hours (ranging from 12 hours to 12 days) (Madin and Traum, 1953). Experimentally, it requires much more virus by oral exposure to produce the disease than is required by intradermal injection suggesting that the virus can be spread more efficiently through skin abrasions. In pig, after the ingestion of virus through uncooked garbage, VESV enters the epithelium via abrasive lesions and multiplies in the basal layer (stratum basale) of the epidermis. Intracellular and intercellular edema and coalescence of

disintegrating cells leads to vesicle formation. During the vesiculation period, infected pigs become febrile (up to 104-107°F / 42°C) coincident with vesicle formation on the snout, lips and on the feet persisting for 24 to 36 hours that begins 1 day post infection (dpi) through 5 dpi. Then they recede rapidly, but a second rise in temperature may occur 24 to 72 hours later. Those rises in temperature coincide with two stages of the disease. The first may occur when a primary lesion forms on the snout. The virus then enters the hematogenous tract, and secondary lesions appear on other parts of the body. The spread of virus to all parts of the body is termed generalization. It is at this time that the second rise in temperature occurs, and it is the point at which most livestock owners first start appreciating the disease. Foot lesions cause the animal to limp or reluctant to move.

Once most vesicles have ruptured, around 5 dpi, the fever begins to drop and returns to normal around 11 dpi (Gelberg and Lewis, 1982). Formation of vesicles is limited to non-haired portions of the integument and tongue 24 hours post infection (Gelberg and Lewis, 1982). Sites of vesicles are on the snout, oral mucosa, soles of the feet, coronary bands and between the toes and may also occur on teats (Knowles and Reuter, 2012). As lesions develop and progress, the virus spreads gradually from cell to cell. A low-grade viremia ensues leading to secondary lesions at other sites with severe lymphocyte destruction in regional lymph nodes. Because of survival of major chunk of the basal layer of epidermis, epithelial regeneration in swine usually observed within 1-2 weeks.

During the initial phase, vesicles are thick-walled withless than 2 cm in diameter containing a meager amount of fluid. After 2 dpi the vesicles become larger and thin-walled due to pressure of accumulating large amounts of fluid. Generally, vesicles form within 24 hours post-infection and rupture 3-4 dpi or 24-48 hours after vesicle formation (Gelberg and Lewis, 1982; Knowles and Reuter, 2012). Direct spread of virus to the tonsillar epithelium occurs upon rupture of lingual vesicles. Secondary vesicle formation occasionally takes place. Epithelial cells are more prone to infection when a breach in the skin allows virus to get an access to susceptible cells. Fluid released by the rupture of larger primary vesicles prepares the ground for secondary vesicle formation. Vesicles lead to ulceration in 4-7 dpi and healing begins approximately 10 dpi and is well advanced by 15 dpi (Gelberg and Lewis, 1982).

Clinical symptoms

Though highly infectious VESV induced disease is rarely fatal with an exception in suckling piglets as similarly observed in FMD. Lesions in sea lions closely resemble to those in swine. Epithelial lesions are identical to that in other vesicular diseases, while no typical systemic lesions are usually observed. The course of the disease is usually about 1-2 weeks. Vesicular lesions in the oral cavity on the epithelium of the snout, lips, nostrils, tongue, feet and mammary glands, soles, coronary bands and interdigital areas of the feet with lameness were the hallmark of disease in all species. Both primary and secondary vesicle formation is possible (Gelberg and Lewis, 1982). Vesicles may also form on the lips and tongue and other parts of mouth cavity. The oral lesions cause excessive salivation, tongue protrusion, dysphagia and anorexia due to reduced food intake. Lesions have also been reported on the teats of nursing sows. As the vesicles begin to form, there is whitening of the affected area, which then forms a blister filled with fluid containing huge amounts of virus. Slight friction or pressure may tear away the vesicle epithelium and leave a raw, eroded surface. Nasal lesions may cause airway obstruction, while pedal lesions at the coronary band cause lameness. Lesions in VES seem to be deeper and granulation tissue commonly forms especially on the feet. VESV has also been associated with reproductive failure in swine (Neill *et al.,* 1995). Abortion also occurs in infected female sea lions with vesicles on their flippers.

Gross and microscopic pathology

Vesicles alike to those of FMD, VS and SVD are observed in VES. The primary lesions may cover the entire surface of the snout and lips. Foot lesions commonly found after generalization may be so severe that the entire hoof/claw may slough off. Formation of new hoof may take 3 to 6 months and the junction of the new and old hoof is often marked by a black line.

Primary viral replication occurs in the basal layer of stratum germinativum of the snout, lips, gums, tongue, and coronary band as discussed earlier. Histologically, the vesicle consists of a circular area, ‘eaten’ out of the stratum malpighii. The center of the area usually contains cellular debris and serous fluid. Following hydropic degeneration and edema, keratinocytes in affected areas take spherical shape (ballooning degeneration) floating

into the vesicular fluid. Histopathology of lesions reveals squamous epithelial swelling, pyknosis and karryorhexis. The malpighian layer of the skin where the virus primarily replicates become infiltrated with neutrophils and weakened and edematous, causing the upward displacement of the epidermis above it and thus the characteristic vesicle. Epithelial cells left intact usually exhibit evidence of degeneration and intracellular edema. Following cell death, the virus spreads to neighbouring cells. In contrast to other vesicular diseases, the stratum basale may be disrupted.

Local lymph nodes may be involved, characterized by congestion and edema followed by lymphocyte depletion. Edema and focal necrosis can also be observed in draining lymph nodes (Gelberg and Lewis, 1982). Viral replication was reported to have occurred here with evidence of virus isolation from these sites. Histology and fluorescent antibody test show intense fluorescence on the snout, tongue, coronary band and tonsillar epithelium. Extracellular fluid accumulation results in separation of individual epithelial cells. Hemorrhage and edema have also been seen in the subcutaneous tissues. Concentrated around vessels there is abundance of inflammatory cells in the dermis. Extra-epithelial lesions like small, multi-focal, lymphocytic perivascular cuffs accompanied by mild gliosis were observed in the medulla oblongata (Gelberg and Lewis, 1982).

Diagnosis

Diagnosis of VES depends upon the following aspects:

Clinical history

Vesicles in mouth and extremities of febrile swine are suggestive of VES (Knowlesand Reuter, 2012). As symptomatology is indistinguishable from those in other vesicular diseases, laboratory intervention is a must for concrete diagnosis.

Detection ofvirus, nucleic acids or antigens

Virus isolation

As considered a gold standard test in diagnostic virology, virus isolation should be performed to confirm the diagnosis. VESV can be readily propagated in mammalian cell cultures (commonly African green monkey kidney or pig kidney cells). Replication results in rapid and drastic cytopathic effects (Knowles and Reuter, 2012). Experimental inoculation of pig kidney cells with VESV serotypes A48 and H54 indicate peak viral titres in 8 hours post-infection and virus replication occurs in the cytoplasm of infected cells (Zee *et al.,* 1967).

Electron microscopy

Electron microscopy can be applied on epithelial tissue suspensions or after the passage of virus in pig tissue cultures. Vesicular fluid samples can also be tested by electron microscopy to detect virus particles having cup-shaped morphology.

Polymerase chain reaction

Reverse transcription-polymerase chain reaction (RT-PCR) and real-time RT-PCR have been developed to detect VESV nucleic acid (Reid *et al.,* 1999; Reid *et al.,* 2007; McClenahan *et al.,* 2009). Further, detection of antigen in infected tissues can be achieved using specific antisera against particular gene segment of the virus.

Detection of antibody

Serum samples from suspected animal (preference to paired serology) should be tested for anti-VESV neutralizing antibodies against VESV using complement fixation, virus neutralization (VN), and enzyme linked immunosorbent assay (ELISA) (Ferris and Oxtoby, 1994; Knowles and Reuter, 2012).

Differential Diagnosis

A differential diagnosis should be made while giving a diagnosis for any known vesicular disease. The lameness and blisters are recognizable indicators of vesicular disease. But to ascertain the particular vesicular disease, differential diagnosis should be attempted by inoculation of horse, cattle and pig as followed for FMD. Differential diagnosis of VES from other infectious diseases should include FMD, VS and SVD. The range of affected species may help in the diagnosis of the vesicular disease. If only pigs are affected other differentials must include swine pox and pseudorabies.

Samples required for laboratory analysis

The virus persists for at least a week in tissues of the snout, tongue, coronary band, tonsil, and lymph nodes. VESV is concentrated in the fluid in the blisters/vesicles formed during the febrile stage as well as in the tissue flap covering the blister. Materials from vesicles e.g., vesicular fluid, epithelium covering vesicle in sterile glycerol phosphate buffer solution may be collected. Old necrotic or fibrinous material difficult to remove should not be collected due to its high probability of contamination with bacteria.

The virus is present in the blood during the pyrectic stage and viremia ends about 5 days after the onset of disease. Heparinized blood,serum, representative tissue specimens in 10% neutral buffered formalin can be collected. Virustitres are highest in gross epithelial lesions and oral fluids. Virus has reportedly persisted for 3 days in oral swabs and 5 days in nasal swabs. Higher virus titers were generally observed in nasal swabs (Gelberg and Lewis, 1982).

Vaccines and infection-induced antibody

No vaccine was developed for VES. Being eradicated, there is no current threat of VES.As multiple VESV serotypes result in vesicular disease, developing a multivalent VES vaccine against all strains may pose a challenge (Knowles and Reuter, 2012).Research on cross-protection between serotypes is unavailable.

Neutralizing anti-VESV antibody increases dramatically 3 dpi and peaks 7-10 dpi. Virus can only be detected 6-7 dpi; therefore, it is presumed that neutralizing antibody formation is protective (Gelberg and Lewis, 1982). Anti-VESV antibody can be detected for 6 months post-infection (Knowles and Reuter, 2012).

Effective disinfectants against VESV

VESV is sensitive to common disinfectants like 2% sodium hydroxide and inactivated in pH 3-5. Vesiviruses are labile below pH of 4.5-5.0 (Knowles and Reuter, 2012). VESV serotypes are more resistant to disinfectants than SMSV serotypes (Blackwell, 1978). Caliciviruses are generally stable in the environment and resistant to inactivation by heat and some chemicals (ether, chloroform, and mild detergents). Sodium hypochlorite (0.1%), 2%sodium silicate, 2%citric acid, 5%acetic acid, 5%phenol,1%formalin, 4% anhydrous or 10% crystallinesodium carbonate, 2% lye solution (caustic soda), cresol, ionic and non-ionic detergents, strong iodophores (1%) in phosphoric acid, lipid solvents such as chloroform inactivate VESV. The virus can remain infective for 2.5 years at 45°F in unground vesicle coverings stored in 50% glycerin phosphate buffer.

Prevention and control

The control of VES could be possible because of effective quarantine, efficient disposal of infected and in-contact animals, cleaning and disinfection, proper inspection and enforcement/ prohibition of feeding raw garbage. Control measures received a momentum by avoiding feeding of VESV infected meat or cadavers of marine mammals or garbage / waste seafood feeding to pigs. VESV infections became scarce and VESV was eradicated consequent upon regulations on cooking garbage before feeding to swine (Smith and Akers, 1976). The ban on feeding raw pork garbage to pigs and quarantine measures were the keys behind successful VES eradication campaign in the USA. The most important practice towards control is cooking all garbage and fish fed to swine at 100°C for 30 minutes (Knowles and Reuter, 2012). Tracking of the animals with an effective identification systemis immensely helpful. Serological serosurveillance targeting breeding sows becomes very useful. Standard biosecurity practices should also be in place as appropriate for any other infectious disease. As a precautionary measures and alertness, monitoring of SMSV in wild populations may prove useful.

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