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Classical swine fever in pigs and its status in India: A review

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ABSTRACT

Classical swine fever (CSF) or hog cholera, one of the most dreaded and devastating viral disease of swine, causes serious economic losses directly due to mortality, retardation of growth, reproductive problems of affected pigs and indirectly by bringing restrictions on exports of pork and pork products. The disease is enzootic in most of the pig producing states and particularly in the North Eastern states of India. Outbreaks of CSF have not only been reported from different states of India, but the occurrence of CSF virus in the tissues of pigs slaughtered for human consumption has also been reported. The lapinized swine fever vaccine has been used in the country to protect pigs against the disease, but the vaccine is not sufficient and is facing constraints its production. The cell culture attenuated CSF virus vaccine will be the only alternative to protect pigs against the disease. Besides the vaccine, early diagnosis and understanding on the epidemiology of the disease are important not only to prevent the spread of the disease but also to evolve suitable strategy to control the disease in India.

Key words: Classical swine fever, CSF vaccine, CSF status

Classical swine fever (CSF) or hog cholera is a serious, economically damaging disease of swine which can spread in an epizootic form as well as establish enzootic infections in domestic and wild pig populations (Edwards *et al.* 2000). The presence of CSF virus (CSFV) in pig herds can have a severe economic impact on the meat production industry due to wide-spread animal deaths because of the disease as well as trade restrictions on meat exports. It is one of the listed diseases of the World organization for animal health or Office International des Epizooties (OIE). At the end of the 20th century, CSF remains widespread in many parts of the globe. Successful eradication has been achieved in many countries, including North America, Australia, and parts of Northern Europe, and many such countries have successfully maintained freedom in the absence of vaccination. The disease continue to be a problem in most of the pig producing states of India and is considered to be the major constraint for the growth of piggery in the North Eastern states of India. In this review an attempt has been made to give some information about the disease and its status in India.

Etiological agent

Classical swine fever virus (CSFV) belongs to the genus Pestivirus within the family Flaviviridae (Wengler *et al.* 1995). The virus has an envelope with glycosylated membrane proteins and icosahedral symmetry (Moennig *et al.* 2003). The virus has a non-translated region at either end (52 NTR and 32 NTR), encompassing a single open reading frame encoding a large protein that is cleaved into smaller fragments. The genes encoding the structural proteins are found towards the 52 end of the genome, and include the major envelope glycoprotein gene E2. The genes encoding non-structural proteins are located mainly in the 32 two-thirds of the genome, and include the polymerase gene NS5B (Meyers and Theil 1996).

Genetic diversity

CSFV isolates from different parts of the world have been placed into various genomic groups/subgroups (Lowings *et al.* 1996). The major genetic groups, are 1, 2 and 3 and 10 subgroups (i.e. each of the first 2 groups divided into 3 subgroups, i.e. 1.1, 1.2, 1.3, 2.1, 2.2 and 2.3 and the third group divided into 4 subgroups, viz. 3.1, 3.2, 3.3 and 3.4). Most of the CSFV isolates made all over the world during 1980 and 1990 were of group 2. CSFV subgroups 2.1 and 2.2 are more restricted in their distribution. The subgroup 2.1 viruses are sporadically reported from China and Europe. During 1997–98 the viruses of the subgroup were introduced from Germany into Netherlands and subsequently spread to Italy, Belgium and Spain (Widjojoatmodjo *et al.* 1999). The subgroup 2.2 virus was reported mostly from Central Europe, the Czech Republic, Italy, Germany, Romania and Hungary

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from 1985 onwards. CSFV of subgroup 2.3 first appeared in Germany in 1982. Subsequently these viruses were reported from Italy, France, Belgium, Austria, Switzerland, Hungary, the Czech Republic, Poland and the Slovak Republic. After phylogenetic analysis of some of the Indian isolates of CSF virus were placed in subgroup 1.1 (Singh *et al.* 2004).

Physico chemical properties and survivality

The virus can be inactivated by chloroform, ether and other lipid solvents. The virus is stable at pH 5 to pH 10, but readily inactivated at pH 3. CSFV inactivated following putrefaction, but can persist for long period in chilled or frozen meat. The virus is inactivated by heating at 66°C for 1 hr. The virus can persist for longer period in refrigerated meat and survive for 15–20 days in slurry. In environment the virus can persist for 4 weeks in winter. It is impossible to give definitive guidelines for the survival time of CSF virus in the environment. The durability of the virus is affected by many physical and chemical variables, including temperature, humidity, pH, presence of organic matter, and exposure to various chemicals. Artificially contaminated bricks or chopped hay, exposed to air but protected from direct sunlight and rain, retained infective virus at 7 days. Ultra-violet light can rapidly inactivate the virus. Infected pigs excrete virus from the respiratory, urinary and alimentary tracts, so it must be assumed that the immediate surroundings of such animals are contaminated and will remain so for a period after removal of the pigs. The virus may survive for longer periods in manure, and experimental studies suggested that inactivation occurred more rapidly in the liquid phase of slurry than in the solid phase, infectivity being lost in about 15 days. Using BVD virus as a model, it was shown that survival times of CSF virus in various types of water varied from 6–24 days at 20°C (Edwards 2000).

Geographical distribution

Classical swine fever (CSF) was first recorded in 1833 in Ohio, USA, but an epizootic resembling CSF became reported in France in 1822 (Cole *et al.* 1962). The disease became widespread in Europe and America by 1866. Brich (1917) reported that spread of the virus infection may have facilitated by the development of railways during the mid 19th Century. At the end of the 20th century CSF remained widespread in many parts of the globe, but successful eradication was achieved in many countries of the world. Canada is free of CSF since 1963 (Edwards *et al.* 2000). In USA the official eradication scheme of the disease was started in 1961 and the last case being recorded in 1976 (Wise 1986). In the European Union particularly in Belgium, Germany, Italy, Spain and the Netherlands large number (611) of outbreaks were reported in 1997, but the number of outbreak came down to very low level (54) in 1998. In Austria no outbreaks of CSF was reported during 1997 and 1998. In Australia the disease was eradicated. In Central America only Belize and Panama are reported to be free of the disease (Edwards *et al.* 2000). Except Uruguay and Chile the disease is reported to be enzootic in other parts of South America. No case of CSF has been recorded in Uruguay since 1991 and the last case of CSF in Chile was reported in August, 1996. The disease is present in Cuba, Haiti and the Dominican Republic of the Islands of Caribbean. The first case of CSF in Haiti was reported in 1996 (Edwards *et al.* 2000). In Japan the first outbreak of CSF was recognized in 1888. With the development of live attenuated GP vaccine in Japan in 1969 the outbreaks of CSF have decreased markedly and the last outbreak was reported in 1992. In South East Asia particularly in Indonesia, Korea, Malaysia, Myanmar, Mongolia, Philippines, Taiwan and Vietnam the disease is prevalent. The situation in most of Africa is uncertain, but the disease is not reported as a problem there except in Madagascar (Edwards *et al.* 2000). As of 2005 Australia, Canada, Great Britain, Iceland, Ireland, New Zealand, Scandinavian Countries and USA are reported to be free from CSF.

Status of CSF in India

In India the first suspected case of CSF occurred in Aligarh in 1944 (Krishnamurthy 1964). Subsequently the disease was reported from West Bengal (Choudhury 1951), Andhra Pradesh (Rao and Satyanarayan 1961, Narayana and Rao 1963, Venkatanarayana 1963), Uttar Pradesh (Krishnamurthy and Adlakha 1962, Saxena *et al.* 1964, Singh *et al.* 1976), Maharashtra (Sapre *et al.* 1962), Rajasthan (Budh Singh 1965), Bihar (Thakur *et al.* 1998), Tamil Nadu (Damodaran *et al.* 1971, Sridhar *et al.* 1996, Govindarajan *et al.* 2003), Punjab (Sharma and Vig, 1964, Saini *et al.* 2000, Kumar *et al.* 2007), Haryana (Gupta *et al.* 1988, Jindal *et al.* 2008), Himachal Pradesh (Lal Krishna and Gupta 1992), Meghalaya (Murti and Hazarika 1982), Mizoram (Verma 1988), Nagaland (Das *et al.* 1983, Ghosh and Das 1986, Ghosh *et al.* 1988), Assam (Sarma and Sarma 1998, Barman *et al.* 2003, Rahman *et al.* 2001, Dutta *et al.* 2003, Sarma 2007, Sarma *et al.* 2007, Sarma *et al.* 2008), Tripura (Bhattacharya 2001) and Kerala (Ravishankar *et al.* 2007).

During March 2006 occurrence of an acute outbreak of CSF in a private farm in Muttil in Wayanad district of North Kerala was reported. All the 23 pigs of 4 months of age were affected and died within 3 weeks. This was reported as the first confirmed case of CSF in Kerala (Ravishankar *et al.* 2007).

In May 2000 occurrence of swine fever in Khundi Kalan village, Sangrur district of Punjab was reported (Saini *et al.* 2000). Overall 28 adults and 40 piglets fell sick with a mortality rate of 80 and 90% respectively. Death in young animals occurred between 5–10 days after the appearance of symptoms. In 2006 concurrent pasteurellosis and CSF outbreaks were reported from Punjab where overall morbidity, mortality and case fatality due to CSF in pigs below 3 months of age were reported to be 88.2, 77.5 and

87. 8% respectively (Kumar *et al.* 2007).

Outbreaks of CSF in organized piggery units in Ambala and Hisar districts of Haryana was reported recently (Jindal *et al.* 2008). The overall morbidity rate, cumulative mortality and case fatality rate during the outbreaks were 54.9, 36.6 and 66.6% respectively.

In Assam outbreak of CSF in Bogalijan village of North Lakhimpur district was first reported by Sarma and Sarma (1998). During the outbreak 8 out of the 12 affected pigs of 2–4 months died. In 2002 an outbreak of CSF in the base Pig breeding Farm, Animal Husbandry and Veterinary Department of the state was reported (Barman *et al.* 2003). A total of 141 pigs of different age groups died during the outbreak and the mortality percentages in various age groups of pigs, viz. 0–2 months, 2–4 months, 4–8 months, >8 months were 100, 60, 53 and 31 respectively. Dutta *et al.* (2003) studied pig mortality during 2002–03 in 4 organized farms of Assam and reported the death of 107 (36.51%) piglets due to swine fever in 2 different pig farms of Assam. Occurrence of CSF in vaccinated pigs in Assam, Meghalaya and Nagaland was observed during June to August 2005. Mortality of 23–50% of the affected pigs was recorded during the outbreaks (Sarma 2007). Presence of CSF virus in tissues of pigs slaughtered for human consumption in different districts of Assam was first recorded by Sarma *et al.* (2007). Recently isolation and characterization of CSF virus in 2 districts of Assam was also reported by Sarma *et al.* (2008a). Detection of CSF virus antigen in tissues of slaughtered pigs in different districts of Assam during 2005–07 was reported. CSF virus antigen was also detected from diseased pigs in different places of Assam during 2005–07 and the detection of CSF virus antigen in tissues of pigs examined during different months for 2005–07 in Assam was also reported (Sarma *et al.* 2008b).

Mode of transmission

Under natural condition transmission of CSF virus mostly occurs by the oro nasal route. The CSF virus is found in all tissues, blood, secretions, and excretions of affected animals and contaminated feed and water are the common sources of infection. Adult boars infected with CSF virus can excrete virus with semen and can, subsequently, transmit the virus to sows and their foetuses via artificial insemination (de Smit *et al.* 1999). The CSF virus may also be transmitted from sows to their offspring by intrauterine infection. The outcome of such as transplacental passage depends on the stage of gestation. Infections during the first trimester of gestation mostly lead to repeat breeding and abortion, whereas, infection during the last trimester mostly results in abortion, malformation or birth of weak or dead piglets. Only when a sow becomes infected during the second trimester, persistently infected piglets may be born. These piglets are immunotolerant and may survive for a long time, persistently shedding the virus in the environment until a late onset of disease occurs and the animals die. This phenomenon is called the carrier-sow syndrome, and is of great importance in the epidemiology of CSF since it may lead to the persistence of an outbreak as apparently healthy pigs may shed the virus without being detected in the serological screening following an outbreak (Ribbens *et al.* 2004). The role of wild boar as a reservoir of the virus and possible source of infection for domestic pigs is still unclear although epidemiological links between CSF virus infections in wild board and domestic pigs have been reported repeatedly (Kern *et al.* 1999). Swill containing products originating from CSF infected pigs and fed to susceptible pigs may cause new infections. The outbreaks of 1986 and 2000 in the United Kingdom and of 1996 in Germany are believed to be caused by swill feeding. Although swill feeding has been forbidden in the EU, experts estimate that (illegal) swill remains an important threat (Ribbens *et al.* 2004). Livestock trucks contaminated with excretions and secretion of infected pigs that are insufficiently cleaned and disinfected may be an important route of virus transmission. Transmission of CSF virus by persons is also frequently mentioned as a possible route of virus spreading. Under current management systems and hygienic precautions, the probability of between-herd spread through iatrogenic transmission is believed to be limited (Ribbens *et al.* 2004). Air-borne transmission of CSFV is possible within as well as between herds. However, in some studies air-borne transmission is found only in a radius of 250 m, whereas in others it was found in 1 km zone. Consequently, the maximum distance that the virus may spread air-borne remains unclear. Moreover, it is to be expected that air-borne transmission is largely influenced by climatological and geographical parameters, but factors such as virus strain may also influence the transmission (Ribbens *et al.* 2004).

Pathogenesis, clinical symptoms and lesion

Natural infection of pigs with CSF virus usually occurs through the oronasal route (Ribbens *et al.* 2004). Target cells of the virus are endothelial cells, lymphoreticular cells, macrophages and some specific epithelial cells. After entry the virus primarily attacks the lymphoid tissues and initial multiplication of the virus occurs in tonsil. After that viraemia occurs and the virus spreads to many other lymphoid tissues like lymphnodes, spleen, kidney and pancreas etc. The virus can cross the placental barrier and can infect the fetuses in utero. Occasionally the virus can infect the nervous tissues of brain. Depending on the site and extent of multiplication of the virus severity of the disease varies and clinical manifestation of disease can be generally seen in three different forms.

Acute classical swine fever

Piglets up to 12 weeks of age most often display the acute form. A constant finding is pyrexia, usually higher than 40°C, but in adults the temperature may not exceed 39.5°C. Initial

signs are anorexia, lethargy, conjunctivitis, enlarged and discoloured lymph nodes, respiratory signs, subcutaneous haemorrhages and constipation followed by diarrhoea. Neurological signs are frequently seen, such as a staggering gait with weakness of hind legs, incoordination of movement, and convulsions. The typical haemorrhages of the skin are usually observed on the ear, tail, abdomen and the inner side of the limbs during the second and third week after infection until death. The virus is shed from the infected animal by saliva, urine and faeces. Pathological changes visible on postmortem examination are observed most often in lymph nodes, spleen and kidneys. The lymph nodes become swollen, oedematous and haemorrhagic. Haemorrhages of the kidney may vary in size from petechiae to ecchymotic haemorrhages. Petechiae can also be observed in the urinary bladder, larynx, epiglottis and heart, and may be widespread over the serosae of the abdomen and chest. A nonpurulent encephalitis is often present. CSF virus causes severe leukopenia and immunosuppression, which often leads to secondary enteric or respiratory infections. The signs of these secondary infections can mask or overlap the most typical signs of CSF and may mislead the veterinarian. With increasing age of the infected pigs (fattening and breeding animals) the clinical signs are less specific and recovery with production of antibodies can occur. Antibodies against CSF virus become detectable 23 weeks post exposure to CSF virus (Moennig *et al.* 2003).

Chronic form

The chronic form of CSF is always fatal. It develops when pigs are not able to mount an effective immune response against the infection. Initial signs are similar to the acute infection. Later, predominantly non-specific signs are observed, e.g. intermittent fever, chronic enteritis and wasting. Animals may survive for 2–3 months before they die. CSF virus is shed from the onset of clinical signs constantly until death. Antibodies may be temporarily detected in serum samples, as the immune system starts to produce antibodies although they are not able to eliminate the virus from the host. Consequently the antibodies are neutralised by the virus and cease to be detectable. Pathological changes are less typical, especially the lack of haemorrhages on organs and serosae. In animals displaying chronic diarrhoea, necrotic and ulcerative lesions on the ileum, the ileocaecal valve and the rectum are common (Moennig *et al.* 2003).

Prenatal form

Although the course of infection in the sow is often subclinical, CSF virus is able to cross the placenta of pregnant animals, thereby, infecting fetuses during all stages of pregnancy. The outcome of transplacental infection of fetuses mainly depends on the time of gestation and viral virulence, respectively. Infection during early pregnancy may result in abortions and stillbirths, mummification and malformations. All of this will lead to a reduction in the fertility index in the holding. Infection of sows from about 50–70 days of pregnancy can lead to the birth of persistently viraemic piglets, which may be clinically normal at birth and survive for several months. After birth, they may show poor growth, wasting or occasionally congenital tremor. This course of infection is referred to as late onset CSF. These piglets constantly shed large amounts of virus and are a dangerous virus reservoir, spreading the disease and maintaining the infection within the pig population. This situation is comparable to cattle persistently infected with BVD virus. CSF must be considered in the differential diagnosis of reduced fertility due to parvovirus infection, PRRS, leptospirosis and Aujeszkys disease (Moennig *et al.* 2003).

Prognosis

The severity of clinical signs largely depends on the age of the animal and viral virulence. Usually young animals are affected more severely than older animals. In older breeding pigs the course of the infection is often mild or even subclinical. Mortality rates may reach 90% in young pigs. Death occurs 2–3 weeks after infection (acute form) or after up to 3 months (chronic form). The outcome of transplacental infection of foetuses depends largely on the time of gestation and may result in abortions, stillbirths, mummifications, malformations or the birth of weak or persistently viraemic piglets. Although persistently infected offspring may be clinically normal at birth, they invariably die from CSF. Survival periods of 11 months after birth have been observed (Moennig 2000).

Diagnosis

Prevailing strains of CSF virus are of only moderate virulence, making clinical diagnosis difficult especially in older animals. This increases the danger of delayed detection of primary cases as occurred in England in 2000. The recent emergence of porcine dermatitis and nephropathy syndrome also complicates the diagnosis, since it can have a similar clinical appearance to CSF. Because the clinical signs of CSF are not pathognomonic, laboratory confirmation of disease is normally required, even for secondary cases during large outbreaks. CSF often has an incubation period of some weeks, on a herd basis, requiring several cycles of amplification before it becomes clinically apparent. Pre-clinical detection would therefore be of enormous benefit to disease control. Fever is a very prominent sign in CSF and it would be extremely useful if pigs could be microchipped or screened en masse (for example using infra red devices) to select those with the highest temperatures for closer examination and sampling. There are no descriptions of pen-side tests for CSF virus in the literature and tests are currently laboratory based, although portable RT-PCR methods are under investigation. The traditional laboratory diagnostic methods are virus

isolation and the demonstration of viral antigens in sections of frozen organs. These have been augmented by the use of antigen detection ELISA and RT-PCR. The ELISA is a simple and rapid method for screening sick or pyrexic pigs and has the advantage that it can be used on large numbers of blood samples. RT-PCR is more complicated and expensive but is also rapid and due to its greater sensitivity can be used on pooled samples and for preclinical diagnosis. If sufficiently automated to enable large numbers of blood samples to be examined, it might be usable as a means of certifying that pigs at an abattoir were non-viraemic at slaughter. There is currently no practical means of wholesale screening of pig-meat imports, as the level of virus in meat is likely to be very low and the scale of required testing would be very large. Tests for CSF antibodies in meat juices would be a possible alternative approach as has been done for other diseases. Infection with CSF virus is immunosuppressive and virus-specific antibodies are very slow to appear. A Dutch study confirmed that routine serosurveillance is not an effective method for early detection of newly introduced CSF. Large-scale serology is possible using commercially available ELISA kits. In 1997/1998 the Dutch tested 2.1 million blood samples for antibody. Unfortunately, the tests are not absolutely CSF specific and can detect antibodies induced by other pestiviruses (bovine viral diarrhoea virus and border disease virus), which occasionally infect pigs. Therefore, positive ELISA results may need to be confirmed by comparative neutralisation tests, which are laborious and slow. A system of comparative ELISA might be a way round this problem (Paton and Greiser-Wilke 2003). A sandwich ELISA test based on the polyclonal serum raised against cell culture adapted CSF virus antigen was useful for detection of the virus antigen in tissues of the pigs showing symptoms of the disease and also from slaughtered pigs for human consumption (Sarma *et al.* 2007). A fluorogenic-probe hydrolysis (TaqMan)-reverse transcriptase PCR assay for classical swine fever virus (CSFV) was developed and evaluated in experimentally infected swine. The assay detected CSFV, representing different phylogenetic groupings, but did not amplify viral RNA from related pestiviruses (Risatti *et al.* 2003). CSF must also be considered in the differential diagnosis of erysipelas, porcine reproductive and respiratory syndrome (PRRS), cumarin poisoning, purpura haemorragica, post-weaning multisystemic wasting syndrome (PWMS), porcine dermatitis and nephropathy syndrome (PDNS), *Salmonella* or *Pasteurella* infections or any enteric or respiratory syndrome with fever not responding to antibiotic treatment.

Vaccines and vaccination

From the beginning of the 20th Century attempts have been made to develop vaccines against the disease. In the 1940, first experiments were made to attenuate the virus by adapting it to rabbits (Baker 1946, Koprowski *et al.* 1946). The most attenuated vaccines against the disease were based on lapinized (rabbit adapted) CSF virus using the Chinese strain (C-strain). Lapinized vaccines are still being used world wide for control of the disease in domestic pigs. In 1969 a CSF attenuated live vaccine (GP vaccine) by passaging the virus in guinea pig was developed. The C-strain of the virus was also used for oral immunization to control the disease on experimental basis (Kaden *et al.* 2000). Lapinized vaccines are safe and induce good neutralizing antibody titre. Efficacy of the vaccine can be demonstrated by challenging the vaccinated animals with virulent virus as early as 5 days after vaccination. But the duration of immunity produced by the vaccine is only for about 6 months, and there is the problem of producing bulk doses of the vaccine at a time. Besides, there is difficulty in standardizing the virus concentration in each batch of the vaccine. Some of the state Institute of Veterinary Biologicals of India, is still producing limited doses of the lapinized vaccine. The presently available lapinized vaccine in the country is not sufficient to immunize even 1% of the total pig population of the country. Therefore there is an urgent need to produce sufficient doses of CSF vaccine in cell culture system for the pig population of our country. CSF virus can be easily attenuated by passaging in cell lines particularly in PK 15 cell line. Cell culture attenuated vaccine is also safe and produce good level of immunity similar to the lapinized vaccine. The biggest advantage with the cell culture vaccine is that the vaccine can be produced in large doses and it is easy to determine the virus concentration in the cell culture system.

Marker vaccine

With respect to today's global trade policy there is a serious disadvantage of using live attenuated vaccines against the disease. Because, vaccinated and infected animals cannot be distinguished as the antibody pattern induced by the vaccine virus resembles that of reconvalescent animals. To eliminate this problem development and use of so called marker vaccines containing the single viral surface proteins have been initiated (Van Rijn *et al.* 1996). Two subunit vaccines containing the glycoprotein E2 of the CSF virus have been developed. Marker vaccines are safe but so far their protective immunity is concerned they are reported to be inferior to live vaccines. Further improvement of the CSF marker vaccines by developing viral vector vaccines, DNA vaccines and molecularly altered infectious cDNA clones of CSF virus has been reported. Recent developments in molecular technology have enabled researchers to construct DNA copies of the complete RNA genome of CSFV. Two recombinant CSFVs (Flc2, Flc3) transcribed from a DNA copy of the genome of the C-strain have been characterized *in vivo* in rabbits and pigs. The results demonstrated that the 2 recombinant viruses had retained the advantageous biological and immunogenic properties of the parent C-strain in rabbis and pigs. Both chimeric viruses provided good

clinical protection against a challenge with virulent CSFV at 1 or 2 weeks after vaccination (de Smit 2000). A subunit vaccine against CSFV has also been developed based on the envelope glycoprotein E2. This subunit vaccine is thus a potential marker vaccine, as discrimination between vaccinated and infected pigs can be based on the detection of antibodies against Erns and/or NS3 (Bouma *et al.* 1999).

Control and prevention

In countries where CSF is endemic vaccination is the main choice to control the disease. The preventive measures adopted by the European Union for trade with third countries stipulate that live pigs and fresh pig meat can only be imported from regions or countries where no CSF has been reported for 12 months and no vaccination is applied during the period. Stamping out of infected pig herds, movement restrictions for live pigs and pig meat, which can transmit CSF virus within zones surrounding the infected farms are important for control. There is a general consensus that number of measures must be introduced to reduce the vulnerability of regions at risk e.g. structural changes in the pig industry including trade. However implementations of appropriate programs might be difficult. Control of CSF in wild boar is still an unresolved problem. Comprehensive information about the disease situation in wild boar population is essential and new strategies have to be devised. CSFV was a difficult virus to work with, and major progress only became possible with the development and the availability of sophisticated virological methods especially during the last 15 years (Moennig 2000).

Future thrust areas

One of the major challenges faced by our country today is the food as well as nutritional security for the ever increasing human population. Foods of animal origin have been considered as the most nutritious and major dietery source to the people worldwide. Pork is nutritious with high fat and low water content and has got better energy value than other meats. In 2002 Indian share in pig meat production to the total world meat product was only 0.63%. Out of the 939.35 million pig in the world only 1.9% (17.5 millions) is from India. There is also good export potentiality for pig meat and other processed products. Therefore there is not only a need to popularize scientific pig production amongst the unemployed educated youth to earn livelihood, but also to protect the animal species from the dreaded disease like classical swine fever.

REFERENCES

Baker J A. 1946. Serial passage of hog cholera virus in rabbits. *Proceedings of the Society of Experimental Biology and Medicine* **63**: 183–87.

Barman N N, Nath A J, Hazarika M P, Barman B and Thakuria D. 2003. Outbreak of classical swine fever in regularly vaccinated pig herd. *Indian Journal Comparative Microbiology Immunology and Infecious Disease* **24**: 89–90.

Birch R R. 1917. Hog cholera transmission through infected pork. *American Veterinary Journal* **51**: 303.

Bhattacharya A. 2001. An outbreak of swine fever in a quarantine pig farm in Tripura. *Indian Journal of Animal Health* **40**: 85–86.

Bouma A, de Smit A J, de Kluijver E P, Terpstra C, Moormann R J. 1999. Efficacy and stability of a subunit vaccine based on glycoprotein E2 of classical swine fever virus. *Veterinary Microbiology* **66**: 101–14.

Budh Singh. 1964. Paper presented at the 11th Conference on Animal Diseases. Madras. *(fide* Damodaran *et al.* 1967.)

Choudhury B. 1951. Annual Report Disease investigation officer, West Bengal. *(fide* Thakur *et al.* 1998.)

Cole C G, Henley R R, Dale C N, Mott L O, Torrey J P and Zinober M R. 1962. History of hog cholera research in the US Department of Agriculture 1884–1960. Agriculture Information Bulletin No. 241, USDA, Washington DC.

Damodaran S, Ramakrishnan R and Rahmatulla K. 1967. Swine fever in Madras. *Indian Veterinary Journal* **48**: 1203–07.

Das P, Ghosh S S, Dutta M, Sonowal D K, Mehta S N and Borthakur D N. 1983. Resources Inventory, Kohima District (Nagaland). ICAR Research Complex for NEH Region.

de Smit A J, Bouma A, Terpstra C, van Oirschot JT. 1999. Transmission of classical swine fever virus by artificial insemination. *Veterinary Microbiology* **67**: 239–49.

Dutta B, Rahman T and Barman N N. 2003. Swine fever in piglets: A pathological study. *Indian Journal of Veterinary Pathology* **27**: 87–89.

Edwards S. 2000. Survival and inactivation of classical swine fever virus. *Veterinary Microbiology* **73**: 175–81.

Edwards S, Fukusho A, Lefevre P C, Lipowski A, Pejsak Z, Roehe P and Westergaard J. 2000. Classical swine fever: the global situation. *Veterinary Microbiology* **73**: 103–19.

Ghosh S S and Das A B. 1986. Retrospective survey on the diseases of livestock and poultry in Nagaland. *Indian Veterinary Medical Journal* **10**: 24–28.

Ghosh S S, Murti A and Dutta B M. 1988. An outbreak of swine fever in an organized pig farm in Nagaland. *Indian Journal of Animal Health* **27**: 45–47.

Govindarajan R, Vengadabady N, Albert A and Purushothaman V. 2003. Detection of hog cholera in desi pigs. *Cherion* **32**: 47–48.

Gupta S C, Singh J, Kulshreshtra R C, Kaushik R K and Arora A K. 1986. Prevalence of swine fever in pigs in Haryana. *Indian Journal of Virology* **2**: 102–03.

Jindal N, Sharma P, Mittal D, Tiwari A K, Narang G and Shukla C L. 2008. Occurrence of swine fever in vaccinated piggery units in Haryana: Detection by Rt-PCR. *Indian Journal of Virology* **19**: 44–46.

Kaden V, Lange E, Fischer U and Strebelow G. 2000. Oral immunization of wild boar against classical swine fever: evaluation of the first field study in Germany. *Veterinary Microbiology* **73**: 239–52.

Kern B, Depner K R, Letz W, Rott M, Thalheim S, Nitschke B, Plagemann R and Liess B. 1999. Incidence of classical swine fever (CSF) in wild boar in a densely populated area indicating CSF virus persistence as a mechanism for virus perpetuation. *Zentralblat Veterinarmedizine B* **46**: 63–67.

Koprowski H, James T R and Cox H R. 1946. Propagation of hog cholera virus in rabbits. *Proceedings of the Society of Experimental Biology and Medicine* **63**: 178–83.

Krishnamurty D. 1964. Paper presented at the 11th conference on animal diseases, Madras. (*fide* Damodaran *et al.* 1967.)

Krishnamurthy D and Adlakha S C. 1962. *Indian Veterinary Journal* **39**: 406 (*fide* Damodaran *et al.* 1967.)

Kumar H, Mahajan V, Sharma S, Alka Singh, R., Arora A K, Banga, H S, Verma S, Kaur K, Kaur P, Meenakshi and Sandhu K S. 2007. Concurrent pasteurellosis and classical swine fever in Indian pigs. *Journal of swine health and Production* **15**: 279–83.

Lal Krishna and Gupta V K. 1992. Swine fever in Himachal Pradesh. *Indian Journal of Veterinary Pathology* **16**: 110–11.

Meyers G and Thiel H J. 1996. Molecular characterisation of pestiviruses. *Advances in Virus Research* **47**: pp. 53–118.

Moennig V. 2000. Introduction to classical swine fever: virus, disease and control policy. *Veterinary Microbiology* **73**: 93–102.

Moennig V, Floegel-Niesmann G and Greiser-Wilke I. 2003. Clinical signs and epidemiology of classical swine fever: a review of new knowledge. *Veterinary Journal* **165**: 11–20.

Murti P S R C and Hazarika G C. 1982. Retrospective epidemiological studies on common diseases of livestock and poultry in Meghalaya. *Journal of Research Assam Agricultural University* **3**: 56–59.

Murty D K and Adlakha E C. 1962. *Indian Veterinary Journal* **39**: 405–19 (*fide* Saxena *et al.* 1964.)

Paton D J, Greiser-Wilke I. 2003. Classical swine fever—an update. *Research in Veterinary Science* **75**: 169–78.

Rahman T, Sarma D K, Baruah G K, Chakraborty A, Pathak D C, Goswami S and Tamuli S M. 2001. Pathology of atypical form of swine fever. *Indian Journal of Veterinary Pathology* **25**: 5–7.

Rao P O and Satyanarayana K. 1961. Annual Technical Report of Disease Investigation Officer, Andhra Pradesh (*fide* Thakur *et al.* 1998).

Ravishankar C, Priya P M, Mini M, Rameshkumar P, Selvan S, Jayesh V, Sunil K S, Sharmadha M K, Sreekumaran T and Jayaprakasan V. 2007. First confirmed occurrence of CSF in Kerala. *Journal of Swine Health and Production* **15**: 156–59.

Ribbens S, Dewulf J, Koenen F, Laevens H and de Kruif A. 2004. Transmission of classical swine fever. A review. *Veterinary Quartarly* **26**: 146–55.

Sapre S N, Moghe R G, Bhagmat S V, Chaudhry P G and Purohit B L. 1962. *Indian Veterinary Journal* **39**: 527. (*fide* Thakur *et al.* 1998)

Saini S S, Dhand N K, Sharma D R and Sood N K. 2000. An outbreak of swine fever in Punjab. *Indian Journal Veterinary Pathology* **24**: 135–36.

Sarma D K. 2007. Occurrence of classical swine fever in vaccinated pigs. *The Vets Communications* **1**: 32–33.

Sarma D K and Sarma P C. 1995. ELISA for detection of hog cholera virus antigen. *Indian Journal Animal Sciences* **65**: 650–51.

Sarma P C and Sarma D K. 1996. Localization of hog cholera virus antigen in tissues of infected piglets and rabbits by ELISA. *Indian Journal Virology* **12**: 105–08.

Sarma P C and Sarma D K. 1998. Isolation and characterization of virulent strain of hog cholera virus from field outbreaks. *Indian Journal of Virology* **14**: 41–42.

Sarma D K, Sarma S, Tiwari A K, Rajkhowa S, Meshram D J, Singh N K and Baruah K K. 2007. The occurrence of classical swine fever virus in tissues of slaughtered pigs in different districts of Assam. *Indian Journal of Virology* **18**: 61–64.

Sarma D K and Bostami B. 2008. Isolation and growth characteristics of classical swine fever in PK–15 cell line. *Journal of Applied Bioscience and Biotechnology* **3**: 29–32.

Sarma D K, Mishra N, Rajukumar K, Sarma S and Singh N K. 2008a. Isolation and characterization of classical swine fever virus from pigs in Assam. *Indian Journal of Animal Sciences* **78**: 37–39.

Sarma D K, Kayastha Rajeev B, Saikia Bina and Neog Bhrigu K. 2008b. Classical swine fever virus infection in domestic pigs of Assam during 2005–07. *Proceedings of 53rd Annual Technical Session of Assam Science Society* **9**: 124–31.

Saxena R P, Asthana V S and Bisht K S. 1964. Control of swine fever outbreak at the central dairy farm, Aligarh, UP. *Indian Veterinary Journal* **41**: 185–92.

Sharma R M and Vig R P. 1964. Paper presented at the 11th Conference on Animal Diseases, Madras.

Singh V P, Misra R P, Srivastava N C and Sinha K C. 1976. Diagnosis of swine fever by indirect haemagglutination test. *Indian Journal of Animal Health* **37**: 454–8.

Singh V K, Saikumar G, Bandyopadhyay S K and Paliwal O P. 2004. Phylogenetic analysis of classical swine fever virus (CSFV) by cloning and sequencing of partial 5′nontranslated genomic region. *Indian Journal of Animal Sciences* **74**: 1093–7.

Sridhar R, Hemalata S, Manohar B M and Sundaraj A. 1996. Swine fever in a pig-a case report. *Cherion* **25**: 50–52.

Thakur D K, Singh S K and Kumar V. 1998 . Effect of an outbreak of swine fever on different economic traits in pig. *Indian Veterinary Journal* **75**: 185–86.

Van Rijn P A, Bossers A, Wensvoort G and Moormann R J. Classical swine fever virus (CSFV) envelope glycoprotein E2 containing one structural antigenic unit protects pigs from lethal CSFV challenge. *Journal of General Virology* **77** (1996), pp. 2737–45.

Venkatanarayana J. 1962. *Annual Technical Report of Disease Investigation Officer*. Andhra Pradesh (*fide* Thakur *et al.* 1998).

Verma N D. 1988. An outbreak of swine fever in Mizoram State. *Indian Journal of Animal Sciences* **58**: 774–76.

Wengler G, Brandley DW, Collet MS, Heinz FX, Schlesinger RW, Strauss J H 1995. Flaviviridae. *Virus Taxonomy—6th Report of the International Committee on Taxonomy of Viruses*. (Eds) Murphy F A, Fauquet C M, Bishop D H L, Ghabrial S A, Jarvis A W, Martelli G P, Mayo M A and Summers M D. New York, Springer-Verlag.

Widjojoatmodjo M N, van Gennip H G P, de Smit A J and Moormann R J M. 1999. Comparative sequence analysis of classical swine fever virus isolates from the epizootic in the Netherlands in 1997–1998. *Veterinary Microbioogy* **66**: 291–99.

Wise G H. 1986. Eradication of hog cholera from the United States. *Practices in Veterinary Public Health and Preventive Medicine in the United States*. (Ed.) Woods G T. Iowa State University Press. pp. 199–223.