CLASSICAL SWINE FEVER (HOG CHOLERA): REVIEW OF ASPECTS RELEVANT TO CONTROL

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ABSTRACT

Classical swine fever (CSF) has the ability to spread over large distances when human intervention, such as illegal swill feeding facilitates its movement. This was apparent during 2005 when CSF appeared in South Africa (SA) after an absence of 87 years. In this review various newly published developments in terms of the diagnosis of the disease and vaccination are described and applied to situations similar to SA. The role of wildlife such as feral pigs and European wild boar in the dissemination and maintenance of CSF virus are discussed and the dearth of knowledge on the potential of other wild pigs species prevalent on southern Africa noted. The modes of spread and control measures to prevent introduction as well as during outbreaks are discussed.

Introduction

Classical swine fever (CSF), also known as European swine fever or hog cholera, is arguably the most important disease of pigs worldwide. Its appearance in South Africa in 2005 after an absence of 87 years has been one of several unwelcome animal disease events since 2000. It is important for those concerned with animal

health to have a good understanding of CSF based on current research and in this review we present the results of recent research that are relevant to preventing and controlling CSF in South Africa and countries with similar pig farming practices.

CSF is highly contagious and manifests as a haemorrhagic fever in its acute form, but subacute, chronic, and clinically inapparent forms of the disease also occur (Moennig, 2000; Van Oirschot, 2004). The disease is caused by a *Pestivirus* (Family *Flaviviridae*) which is closely related to the viruses that cause bovine viral diarrhoea/mucosal disease and border disease, both of which are also able to infect pigs (Hoffmann et al., 2005). It is a small RNA virus, very different from the large DNA virus that causes African swine fever (ASF), although both viruses produce similar clinical signs and lesions. Like the other members of its genus, it has an immunosuppressive effect and infected animals may die as a result of secondary infections (Moennig, 2000; Van Oirschot, 2004). The way in which the disease presents depends both on the virulence of the causative virus and the immune response of the pig. Thus, although pigs of all ages are susceptible, adult pigs often develop less severe disease and have a better chance of survival.

First identified in the early 1830s in Ohio, USA, CSF was for a long time widely distributed in the Americas, Europe and Asia. It was finally eradicated from North America in 1978 (Terpstra, 1994) and has also been eradicated from Australia and most countries in Western Europe, but is currently widespread in South and Central America, the Caribbean, Asia, and Eastern Europe (Moennig, 2000). With the exception of South Africa, CSF has not been reported from the African continent according to disease information available on the World Organisation for Animal Health web site (www.oie.int), although its absence is not supported by confirmed data, and possibly more research is required. CSF was reported to occur in South Africa at the beginning of the century, but had been eradicated by 1918, and was not seen again until 2005 (Sandvik et al., 2005), when outbreaks occurred first in the Western Cape, followed shortly by the Eastern Cape, where the success of an eradication programme has been monitored by active surveillance. The only other countries in the African region where CSF has been confirmed are the islands of Madagascar, where it was introduced from Europe more than 30 years ago, and Mauritius, which reported an outbreak that lasted from 2000 to 2002. The potential of this disease to spread over long distances and cause outbreaks in areas previously

free is demonstrated not only by its arrival in South Africa but also its reappearance during the last decade in countries like The Netherlands and Britain, where it had been eradicated. Both the South African outbreaks and the outbreaks in The Netherlands and Britain were caused by viruses belonging to the subgroup 2.1, which has never become endemic in Europe and is by and large confined to Asia (Widjojoatmodjo et al., 1999; Sandvik et al., 2005).

Scientific publications on CSF during the last decade reflect the results of research focused on the development of efficacious vaccines and diagnostic tools. The 1997-1998 outbreak of CSF in The Netherlands generated a large number of publications. These include detailed descriptions of the outbreak and evaluations of the control strategy that was implemented, using modelling to compare the approach that was adopted with possible alternatives (Dijkhuizen, 1999; Elbers et al., 1999; 2001; Jalvingh et al., 1999; Meuwissen et al., 1999; Nielen et al., 1999; Terpstra and De Smit, 2000; Mangen et al., 2001). A number of papers were published on the status and control of CSF in European wild boar (*Sus scrofa*) populations (Kern et al., 1999; Floegel et al., 2000; Fritzemeier et al., 2000; Kaden et al., 2000; 2004; 2005; 2006; Laddomada, 2000; Kaden and Lange, 2001; Artois et al., 2002; Karsten and Krieter, 2005a,b; Karsten et al., 2005a,b; Klinkenberg et al., 2005; Mintiens et al., 2005; Rossi et al., 2005a,b). Aspects of monitoring and surveillance for CSF have also been described (Colijn et al., 1997; De Vos et al. 2005; Elbers et al., 2002; 2003; Engel et al., 2005; Feliziani et al., 2005).

From the South African perspective, there are a number of important aspects to CSF. Firstly, it is clear from the experience of The Netherlands that, even if eradication is proven beyond doubt to have been successful, continued vigilance based on an effective surveillance programme is essential. Secondly, investigation to ensure that CSF is not hiding in populations of feral domestic pigs, remnants of European wild boar populations that were introduced during the last century, or wild pigs such as warthogs (*Phacocoerus africanus*) and bush pigs (*Potamochoerus larvatus*) will be required to prove that the eradication campaign was completely successful. Thirdly, it is important to have readily available the current information on options for control in both epidemic and endemic situations, so that control strategies can be implemented that are science-based, appropriate for local conditions and as cost-effective possible. Finally, it is important to consider the different pig production

systems in South Africa. Pig farming in South Africa is diverse ranging from large commercial enterprises to small operations in urban townships to subsistence free-range producers in the rural areas. This complexity is important to consider in the event of an outbreak of CSF.

Transmission and spread of CSF virus

Pigs usually become infected by inhaling or ingesting the virus. Acutely infected pigs that are shedding large amounts of virus in their saliva, as well as lesser amounts in urine, faeces, ocular and nasal secretions, are a potent source of infection for other pigs. Pigs start to shed virus for a few days before clinical signs develop (Terpstra, 1994; Van Oirschot, 2004), and continue to do so until antibodies develop, which usually happens about 11 days after infection. Pigs in the incubation stage are particularly dangerous, as they do not show signs of disease and may be sold, sent to slaughter, or tapped for semen. Investigations carried out after an artificial insemination (AI) station became infected during the 1997-1998 Dutch outbreak demonstrated that semen collected from infected boars may contain virus (de Smit et al.; 1999; Floegel et al., 2000). Other high risk pigs are those that develop chronic disease after infection with viruses of lower virulence and periodically shed virus throughout the course of what may be a long illness with non-specific clinical signs (Van Oirschot, 2004), and piglets infected with 'late onset' CSF. Infected pregnant sows transmit virus to their foetuses via the placenta. Viruses of higher virulence generally cause the sow to abort (Van Oirschot, 2004). However, foetuses infected during the first few weeks of pregnancy, before their immune systems become functional, may develop late-onset CSF. These piglets appear normal at birth, but, in spite of persistently circulating and shedding virus, do not produce specific antibodies to CSF virus. Eventually they develop clinical CSF in response to some unknown trigger and die, usually within the first 4 – 6 months of life but occasionally as long as 11 months after birth (Moennig, 2000; Van Oirschot, 2004), during which time they are able to contaminate the environment and infect other pigs.

Virus is also transmitted to pigs by fomites contaminated with the virus (vehicles, equipment, clothing), or when they are injected with contaminated needles (Van Oirschot, 2004). Mechanical transmission over short distances by biting insects may

be possible (Van Oirschot, 2004). Aerosol transmission over distances of less than 500 metres has been demonstrated experimentally, but is not considered important except possibly under unusual circumstances (Dewulf et al., 2000). Transmission by other animals such as rodents, birds or pets contaminated by the virus has been shown to be unlikely (Dewulf et al., 2001).

The ability of the virus to persist in uncooked pork and processed pork that has not been heated to high temperatures for long periods - months when kept at cool temperatures and years if frozen - is of great importance for transmission over long distances and between continents. Edwards (2000) reviewed the survival and inactivation of CSF virus under different conditions. It survives longest in frozen pork, for more than 4 years, but can survive for several months in salted, smoked, and fresh chilled pork. To inactivate the virus it is necessary to heat the product for 30 minutes at temperatures of 65°C or higher. Feeding uncooked swill that may contain pork products to pigs is therefore an extremely dangerous practice. Smuggled meats have furthermore been identified as posing a very high risk for introduction of CSF (Wooldridge et al., 2006). The major outbreaks that spread through EU countries in 1997 – 1998 were caused by a virus that is believed to have originated in Asia (Moennig, 2000), and to have been introduced into European domestic pigs via swill illegally fed to pigs in a single area in Germany towards the end of 1996 (Elbers et al., 1999; Moennig, 2000). Vehicles used to transport pigs to that area and within the area were identified as a probable source of the 1997 CSF outbreak in The Netherlands (Elbers et al., 1999). Once an outbreak is established, particularly in areas with high pig density, the process termed “neighbourhood spread” becomes important (Elbers et al., 1999). All the components of this type of spread have not been identified or defined, but movement of people between herds is likely to be most important. However, it has been possible to develop models that can be used to predict where major outbreaks are likely to occur based on farm density and the distance between farms (Boender et al., 2007).

A number of recent publications have described simulation models used to estimate the impact of different risk factors on the size and spread of outbreaks of CSF and predict how the disease will spread between herds, in order to determine the probable effectiveness of control measures (Karsten and Krieter 2005a; Karsten et

al., 2005a;b). The various methods used to predict spread of CSF have been reviewed by Karsten and Krieter (2005b).

Field and laboratory diagnosis of CSF

Control of outbreaks of contagious diseases depends on early recognition of the disease in the field. CSF is often difficult to recognise, because the wide range of clinical signs and lesions reported vary according to the amount and virulence of the virus and the age, breed and immune capability of the pig (Moennig, 2000; Artois et al., 2002). They can be confused with other diseases, in particular ASF, but also porcine reproductive and respiratory syndrome (PRRS) and bacterial septicaemia or pneumonia (Sandvik et al., 2005). When countries have been free of CSF and its introduction is unexpected, as was the case in South Africa, a delay in field recognition is highly likely. In The Netherlands, where CSF had not occurred since an outbreak in 1992 (Elbers et al., 1999), a study based on the 1997-98 CSF outbreaks indicated that only about 50% of infected sow herds would be identified within the first 37 days after infection, although this improved to 90% by 47 days (Engel et al., 2005). In Finland, which has been free of CSF since 1917, a simulation study suggested that 8 – 12 weeks would pass before detection of CSF in a sow breeding unit (Raulo and Lyytikäinen 2007).

Because rapid diagnosis is so important, various studies have been undertaken to evaluate how useful a variety of clinical signs and macroscopic pathological lesions are in the diagnosis of CSF (Elbers et al., 2001; 2002; 2003; Floegel-Niesmann et al., 2003). It was concluded that, although useful pointers, none of the signs and lesions, alone or in combination, were sufficiently sensitive or specific to ensure detection of an outbreak. As a consequence, the post-outbreak surveillance programme in The Netherlands included a requirement that a tonsil sample should be taken from all pigs routinely submitted to laboratories for post mortem examination to be tested for CSF viral antigen, whether or not any of the macroscopic pathological lesions were suggestive of CSF (Elbers et al., 2003).

Even when clinical signs and pathological lesions are highly suggestive of CSF, laboratory confirmation is always necessary, in particular to distinguish CSF from ASF. Samples of choice for detection of CSF virus are tonsillar tissue, lymph nodes, spleen and the distal ileum. A recent experimental study demonstrated that the

nictitating membrane (third eyelid) provides a useful source of virus in pigs which have undergone autolysis, since this organ is much less affected by autolysis than the internal organs (Teifke et al., 2005). Whole blood samples and tonsillar swabs may be taken from live pigs. Because the disease may run a chronic course, samples should be taken from several animals if possible (Van Oirschot, 2004).

The use of immunological and molecular genetic technology for laboratory diagnosis has progressed rapidly over the last decades. Using the direct immunofluorescence test to detect antigen in frozen tissue sections, results can be available in 2 hours (Van Oirschot, 2004). However, this test does not distinguish between CSF virus and other pestiviruses unless monoclonal antibodies are used (Van Oirschot, 2004). Rapid results are also obtainable using reverse transcriptase PCR (polymerase chain reaction) to detect viral genetic material, and these tests can distinguish between CSF virus and its close relatives (Vilcek and Belak, 1996; McGoldrick et al., 1998; Hoffmann et al., 2005). Recently, a study demonstrated that a PCR could detect CSF-viral RNA in formalin-fixed tissues, a considerable advantage under conditions where the submission of fresh samples is not possible (Singh et al., 2005). The availability of realtime reverse transcriptase PCR systems (Liu et al., 2007; Zhao et al., 2008; Wen et al., 2010) now makes sensitive, high throughput detection of viral genome possible. ELISA (enzyme-linked immuno-sorbent assay) technology is also used to detect viral antigen in blood or organ samples and offers a rapid way of screening large numbers of samples, but is less sensitive than the PCR (Clavijo et al., 1998; Van Oirschot, 2004). However, isolation of the virus remains an essential element of the laboratory armoury to diagnose CSF, although this is made difficult by the fact that it is a non-cytolytic virus and indirect methods are needed to detect it in cell cultures. ELISA is also used to detect antibodies in sera but only some of the tests available distinguish between antibodies produced in response to CSF virus and those elicited by related viruses (Moser et al., 1996; Müller et al., 1996; Colijn et al., 1997; Van Oirschot, 2004).

Molecular genetic studies of the CSF virus provide information about the relationships of viruses isolated from different outbreaks (Hofmann et al., 1994; Lowings et al., 1996; Vilcek et al., 1996; Vilcek and Paton, 1998). This offers pointers to the possible origin of outbreaks, and demonstrates whether in a particular area all the outbreaks of CSF are likely to be due to a single introduction (Pereda et al., 2005). It

also allows monitoring of the evolution of CSF viruses over time (Diaz de Arce et al., 2005). Using this technology, it was established that the virus that caused the South African outbreak in 2005 is of Asian origin (Sandvik et al., 2005). The same virus has caused several recent outbreaks in Europe, including the 1997-1998 Dutch outbreak (Widjojoatmodjo et al., 1999; Sandvik et al., 2005), and was presumed to have been introduced into Europe from Asia, as it has never been associated with CSF in wild boar.

Detection, maintenance and control of CSF in wild pig populations

The European wild boar, which is ancestral to domestic pigs, is widely distributed from western Europe to India in the East and North Africa in the south. Sporadic outbreaks of CSF that occur in domestic pigs in Europe, including European Union (EU) member countries (Germany, Italy) otherwise free of CSF are generally linked to either indirect or direct contact of domestic pigs with wild boar (Laddomada, 2000). It was estimated that 59% of primary outbreaks of CSF in Germany over a decade were caused by contact between wild boar and domestic pigs (Fritzemeier et al., 2000; Mintiens et al., 2005). Wild boars usually become infected through swill feeding, either intentionally by hunters or by accidental contact with garbage containing infected material (Moennig, 2000; Artois et al., 2002). The outbreaks are not necessarily self-limiting (Moennig, 2000). Studies of wild boar associated with outbreaks over extended periods in Germany indicated that young animals less than a year old were frequently infected with CSF virus, while older animals were only rarely infected (Kern et al., 1999; Kaden et al., 2005). The key factor for maintenance of the virus in wild boar is believed to be the existence of dense populations of wild boar with large numbers of young animals susceptible to infection (Kaden et al., 2005; Rossi et al., 2005a). Investigations have indicated that persistently infected wild boars, either due to transplacental or post-natal infection, are unlikely to play a role in maintaining the virus (Kaden et al., 2005), although this was suggested in the earlier study (Kern et al., 1999). Wild boar gruntlings that are persistently infected and immunotolerant have not been demonstrated to survive longer than 39 days (Artois et al., 2002). Spread from wild boars to domestic pigs occurs when free-ranging domestic pigs come into direct contact with infected boars or scavenge the remains of infected boars that have died or been killed by hunters (Fritzemeier et al., 2000; Laddomada, 2000; Mintiens et al., 2005).

Various methods have been devised to detect infection or demonstrate freedom from CSF infection in wild boar populations. Random sampling is difficult in wild populations where animals are not individually identified. Serological monitoring has been used in preference to virus detection for long-term monitoring of wild boar populations in which infection is known to occur (Rossi et al., 2005b). Mintiens et al. (2005) described the method that they used to confirm with a high degree of probability that wild boar in Belgium were free of CSF infection. This method can be used when the infection rate among wild pigs is unknown but likely to be extremely low. Blood and lymph node samples from 789 hunted boar and boar found dead over a period of three years (1998 – 2001) were submitted to three laboratory tests to detect antibodies, virus and viral RNA. Statistical methods were used to determine that the negative results obtained were applicable to the entire wild boar population in the target area.

The EU recommends a multi-faceted approach to control and eradication of CSF in wild boar populations that focuses on reducing population density but also includes intensive diagnosis and good hygiene practice during hunting (European Commission, Scientific Opinion XXIV/B3/R09/1999). Very dense wild boar populations exist in some forested parts of Germany. Because it is impossible to achieve sufficient population reduction in a reasonable time, the control measures have for more than a decade been supplemented by oral vaccination using the modified live C-strain virus (Kaden et al., 2000; Kaden and Lange, 2001). This has significantly contributed to the control of CSF in wild boar populations in Germany (Kaden et al., 2000; 2004). However, oral vaccination must be combined with the other measures, in particular hunting efforts focused on reducing the number of young wild boars less than one year old, as these animals showed relatively poor uptake of bait and are important in the spread of virus (Kaden et al., 2000; Laddomada, 2000).

Experimental infection has demonstrated that the warthog (*Phacochoerus aethiopicus*) and the bush pig (*Potamochoerus larvatus*) are susceptible to infection and can spread disease to in-contact animals of the same species (Gers et al., 2008). More research is needed to establish whether these species might become

important in the epidemiology of CSF in southern Africa if they become infected in the wild.

The use of vaccines in the control of CSF

Since effective vaccines against CSF exist, their inclusion in measures to control CSF can reduce costs and limit spread (Van Oirschot, 2003). The efficacy of vaccines depends on their ability to induce a strong immune response. Ideally, a vaccine should protect an animal not only against the effects of a disease but also against infection with the agent that causes the disease. A modified live vaccine that has been widely used both to control CSF in countries where it is endemic and in eradication programmes aimed at achieving freedom without vaccination fulfils these criteria. Studies based on serial transmission of the live attenuated CSF vaccine in pigs have demonstrated that reversion to virulence does not occur (Van Oirschot, 2003). Numerous studies using the modified live vaccine based on the C strain in both domestic pigs and wild boar have failed to demonstrate any basis for the concern that vaccinated, subsequently infected pigs may become carriers capable of shedding virus and infecting susceptible pigs (Van Oirschot 2003; Kaden et al., 2006). When pigs are correctly vaccinated with the live attenuated C strain vaccine, challenge with virulent virus did not result in infection of the vaccinated pigs or shedding of the virus (i.e. the pigs were fully protected against both infection and disease). Occasional breakdown under field conditions was attributed to incorrect vaccination procedures (Van Oirschot, 2003). A further advantage is that pigs vaccinated with modified live vaccine may be immune to infection before neutralising antibodies are detected about 11 days post infection (Suradhat et al., 2001; Van Oirschot, 2003; 2004) owing to a cellular response that precedes the humoral response. The production of CSF virus-specific gamma interferon has been demonstrated to occur as early as 6 days after vaccination or infection and is thought to be a key component of the cellular immune response (Suradhat et al., 2001).

The main stumbling block to using vaccination as a control measure is the potential impact on trade. Until recently it has not been possible to distinguish between vaccinated and naturally infected serologically positive animals. This problem has discouraged countries with an exotic outbreak of CSF from using vaccination as

control option (Terpstra and de Smit, 2000), even though it is a less drastic measure than culling. To overcome this problem, research efforts have concentrated on developing technology that will distinguish between infected and vaccinated animals (DIVA). Subunit marker (DIVA) vaccines against CSF with a companion diagnostic test to trace residual infections by detecting antibodies not induced by the vaccine have been developed (Moormann et al., 2000; Beer et al., 2007). The main disadvantage of the E2 marker vaccine was that protection was only complete 3 weeks after vaccination, which would diminish its usefulness as an emergency vaccine. Studies in different laboratories suggest that the vaccine behaves differently under different conditions. Although the initial study showed that pigs were fully protected after 3 weeks, another study showed that pigs vaccinated with the subunit E-2 marker vaccine and subsequently infected with CSF virus developed antibodies to the infecting virus, and blood samples were positive for viral antigen on RT-PCR, although virus could not be isolated (Dewulf et al., 2002). Experiments conducted to determine whether these pigs were capable of infecting other pigs indicated that horizontal transmission, either via injected serum or close contact, did not occur, but vertical transmission by pregnant sows was possible (Dewulf et al., 2005). Possible explanations included continuous exposure of foetuses to virus, less virus needed to accomplish vertical transmission, and transmission of the virus in white blood cells. However, it was concluded that persistently infected piglets from vaccinated and subsequently infected dams might play a role in maintaining infection in marker-vaccinated herds. The initial study showed that vertical transmission could occur as a rare event after a single vaccination with E2 marker vaccine but twice-vaccinated sows did not transmit the virus transplacentally (Moormann et al., 2000). Marker vaccines are commercially available, but efforts to develop more efficacious DIVA technology are ongoing, because the immune response is delayed and less protective than that to the live attenuated vaccines (Beer et al., 2007). Research is also directed towards improving the tests used to distinguish naturally infected and vaccinated animals (Beer et al., 2007, Zhao et al., 2008).

South Africa is a net importer of pork, but a small amount of pork is exported, mainly to countries in the southern African region. Although since 2000 this amount has almost invariably been less than 1 million kg per annum (Anon, 2010 and statistics available from the web site of the South African Department of Trade and

Industry, www.dti.gov.za/) this helped to influence the decision not to vaccinate as part of the control strategy.

Evaluation of control strategies

To achieve eradication of CSF after an outbreak, stamping out of infected and in-contact pig herds with destruction of the carcasses is traditionally considered to be the best option (Elbers et al., 1999; Garner et al., 2001). There are plausible arguments against this option in terms of feasibility, costs, and public acceptance (Mangen et al., 2001; Van Oirschot, 2003). Unless market-related compensation for pigs slaughtered is paid promptly, farmers are encouraged to evade the measures and, since movement control is seldom invincible, this can result in rapid dissemination of the virus (Penrith and Thomson, 2004). The South African experience with CSF in 2005 confirms the importance of prompt payments. Payments to subsistence farmers in the former Transkei were initially efficient and prompt. This resulted in subsistence producers bringing their pigs to culling teams to ensure that they would receive compensation for culled animals. When the payment system began to unravel due to the scale and complexity of the outbreak, subsistence farmers began evading culling teams. Problems with compensation may have led to the persistence of the disease in the Transkei for longer than might have otherwise been the case.

The cost and effectiveness of eradication is also related to the speed with which CSF can be diagnosed, since any delay in diagnosis will inevitably result in spread of the virus by translocation of infected pigs to other premises or abattoirs as occurred in the 1997-1998 outbreak in The Netherlands (Elbers et al., 1999). The cost of controlling that outbreak exceeded 1.3 billion US dollars, while the total loss due to the outbreak was estimated to be 2.3 billion US dollars (Meuwissen et al., 1999). Eleven million pigs were slaughtered, of which only 0.7 million were confirmed as infected; 1.1 million were pre-emptively slaughtered, and 9.2 million were slaughtered for welfare reasons (Dijkhuizen, 1999). The ban on commercial slaughter and sale of pigs meant that all of these pigs had to be destroyed in such a way that they did not enter the food chain, creating a massive problem of disposal. The experience generated various publications aimed at evaluating the control measures applied and providing models for similar and alternative interventions

(Jalvingh et al., 1999; Nielen et al., 1999; Mangen et al., 2001). Nielen et al. (1999) concluded that, while the control strategy applied was effective and the costs were justified if compared to that of control failure, they could have been greatly reduced if good biosecurity had been implemented during the outbreak.

Emergency vaccination around the outbreak focus can limit disease spread and thus reduce the number of animals killed. This strategy has proven successful for various diseases, for example the 2000 outbreak of foot and mouth disease in Kwa-Zulu Natal, South Africa (Brückner et al., 2002). When the outbreak has been controlled, the vaccinated animals are usually slaughtered or destroyed to regain previous status of freedom without vaccination. Modelling studies carried out using data from the 1997 – 1998 CSF epidemic in The Netherlands suggested that emergency vaccination in addition to eradication could have lowered the costs provided that the vaccinated animals could be sold for commercial slaughter and not destroyed at the end of the operation (Mangen et al., 2001). The modified live vaccine for CSF would be the vaccine of choice, since immunity develops rapidly (Suradhat et al., 2001; Van Oirschot, 2003). Although preferable, the use of a marker vaccine is not essential for emergency vaccination, provided vaccinated animals are clearly identified by some other means such as ear tags or tattoos.

After an outbreak has been eradicated, it is necessary to put in place a monitoring and surveillance system to demonstrate, at an acceptable level of confidence, that the eradication was successful and the area or country is free of CSF infection. Such surveillance systems need to be maintained for a sufficient period to be sure that eradication has been accomplished, and ongoing systems designed to ensure early detection of CSF. Guidelines for surveillance are provided in the *Terrestrial Animal Health Code* of the World Organisation for Animal Health (Office International des Épizooties (www.oie.int). Periodic evaluation of the adequacy of the surveillance programme is necessary (Feliziani et al., 2005; Klinkenberg et al., 2005).

It is logical that preventing the occurrence of a highly contagious disease is likely to be more cost-effective than controlling it. Nevertheless, preventive measures will not be without cost. A study was undertaken in The Netherlands to determine the most cost-effective way to prevent entry of CSF, using a model based on a scenario tree for introduction of CSF (De Vos et al., 2005). It would be useful for South Africa and

other countries in the region to undertake a similar exercise, which could also be applied to other diseases that might be introduced by the same route. The details of the Dutch study are not relevant to CSF in South Africa, since the greatest risk in The Netherlands is posed by the extensive transport of live pigs between EU member states, while the use of swill emanating from infected countries is the most likely route of entry for countries that do not share land borders with infected countries. However, such a study would necessitate an in-depth examination of what exactly would be necessary to block this route of entry, which would be much more helpful than vague recommendations about better policing of harbour and airport swill and prevention of swill feeding, without an exact evaluation of what measures would be required and the feasibility and cost of implementing them.

In relation to prevention of contagious diseases like CSF, the importance of on-farm biosecurity cannot be overestimated. A review of the most important sources of disease on pig farms and thus the key biosecurity measures that should be implemented was published by Pritchard et al. (2005).

Conclusions

Recent experiences emphasise the threat that CSF poses to pig industries worldwide. The 1997-1998 experience in The Netherlands demonstrated that eradication cannot be accepted as a permanent state. The South African experience demonstrated that a lack of infected neighbouring countries will not necessarily protect against the introduction of CSF, which is a successful long-distance traveller.

Studies on the transmission of the virus have emphasised the importance of movement of live pigs, feeding of swill that can contain insufficiently cooked pork, and the central role that humans play in spreading CSF. They have sounded a note of caution with the discovery that CSF virus can be transmitted in semen, while some means of transmission previously considered possible (rodents and pets) have been discounted.

Rapid diagnosis has been emphasised as crucial for the control of outbreaks. Diagnostic tools developed over the last three decades have improved the ability of laboratories to provide rapid and accurate results, but they can only do so if they

receive the correct samples in good condition. Field diagnosis remains difficult because of the wide variety and lack of consistency in clinical signs and pathological lesions, and it is recommended that surveillance programmes include routine laboratory testing for CSF in any pigs that are brought to veterinary diagnostic facilities to determine cause of death, or display signs or lesions of disease at abattoirs; the fact that secondary infections are common in pigs with CSF means that another diagnosis may conceal the presence of CSF.

Considerable research and surveillance have been directed at detecting and controlling CSF in European wild boar populations. In South Africa, further investigation is required to determine whether CSF virus is likely to become established in wild pig (warthog and bush pig) populations. It is important, however, to remember that these species are unlikely to play the same role in CSF that at least warthogs play in the epidemiology of ASF, because of their complete resistance to disease caused by that virus and the fact that they can only transmit the virus naturally to domestic pigs via the bites of tampans (*Ornithodoros* spp.). This has resulted from lengthy co-existence and co-evolution with ASF virus. This differs from maintenance in wild boar populations, which simply circulate the virus in large populations that offer a continuous supply of susceptible young pigs, much as may occur in domestic pig populations.

A study of recent information on vaccines against CSF indicates that vaccination is a viable tool both for controlling CSF in areas where it is endemic and as an emergency measure in support of outbreak control. However, the ideal situation, namely a marker vaccine that induces immunity sufficiently rapidly to be used as an emergency tool and offers full protection from both disease and infection while allowing differentiation between vaccinated and infected animals, has not yet been attained.

Finally, an evaluation of control measures, based largely on the measures implemented during the 1997 – 1998 epidemic in The Netherlands, has demonstrated that, while these measures were effective, a less drastic approach may need to be considered in future. The application of excellent biosecurity measures both to prevent disease on pig farms and to prevent spread during outbreaks is strongly emphasised. Surveillance also contributes strongly to prevention, and must

include a strategy to detect antigen as well as antibodies. Since costs may preclude the extensive laboratory testing recommended in The Netherlands, the importance of the farmers themselves in monitoring their animals for CSF and other diseases cannot be over-emphasised. Ensuring a proper level of information in all the pig farming sectors should be a combined effort on the part of the veterinary authorities, private practitioners, and producer and welfare organisations.

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