The spread of pathogens through trade in pig meat: overview and recent developments

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Summary

A number of animal diseases can be transmitted to pigs via meat if the animals are fed scraps of meat imported from infected countries. For this reason, garbage feeding of pigs is regulated in many countries. The major porcine diseases recognised as being significant for this transmission pathway are foot and mouth disease, African swine fever, classical swine fever and swine vesicular disease. The World Organisation for Animal Health *Terrestrial Animal Health Code* (the *Terrestrial Code*) offers risk management recommendations for meat from countries where these diseases are present. However, there is no *Terrestrial Code* chapter on porcine reproductive and respiratory syndrome (PRRS), a relatively new viral disease of pigs which, since its recognition in the 1990s, has become endemic in most pig-producing countries. This paper assesses the risk of spread of PRRS virus through trade in pig meat, and concludes that the likelihood of its transmission by this pathway is negligible.

Keywords

Import risk analysis – International trade – Pig meat – Porcine reproductive and respiratory disease – Porcine reproductive and respiratory syndrome – Pork – Swine.

Introduction

The potential animal health risks from pig meat were reviewed by Farez and Morley (13), who concluded that foot and mouth disease, African swine fever, classical swine fever and swine vesicular disease posed significant risks in uncooked pork products. There has been no significant new information since that review to alter this conclusion and the World Organisation for Animal Health (OIE) *Terrestrial Animal Health Code* (the *Terrestrial Code*) contains internationally recognised risk management recommendations applicable to meat from countries where these diseases are present. Farez and Morley (13) also concluded that porcine reproductive and respiratory syndrome (PRRS) and transmissible gastroenteritis of pigs (TGE) were not hazards in pig meat. In the case of TGE, which was well understood at the time of that review, this conclusion remains valid. However, a significant amount of new information on PRRS has arisen since 1997, some of which has informed more recent risk assessments on pig meat (10, 12, 28).

This paper will discuss PRRS in the context of pig meat and pig meat products imported for human consumption. For imported pig meat to be a vehicle for the transmission of this disease, PRRS virus (PRRSv) must be:

– present in the exporting country

– present in the herd of origin

– present in the animals at the time of slaughter at a significant prevalence and titre (animals slaughtered at the peak of viraemia)

– able to survive normal processing and storage, including withstanding the effects of pH change and decay over time at different storage temperatures involved in the supply chain

– able to survive long enough to be present in meat scraps fed to pigs

– able to initiate infection by the oral route.

Hazard identification

Porcine reproductive and respiratory syndrome virus is a small single-stranded RNA virus belonging to the genus *Arterivirus* in the family *Arteriviridae*, order *Nidovirales*. The disease syndrome was first identified in the United States in

1987 and in Europe in 1990, but it was not until 1991 that the causative virus was identified (4). Although the origin of the virus is unknown, retrospective serological evidence suggests that PRRSv emerged in North America in the late 1970s (42) and genetic studies indicate that the European and US strains had a common ancestor as recently as 1979 (15). During the 1990s PRRSv spread rapidly and has since been reported in most pig-producing countries (1).

The virus has a delicate lipid envelope which is inactivated by lipid solvents and heat (14). It persists for 1–6 days at 20–21°C, 3–24 h at 37°C and 6–20 min at 56°C. Although the virus is very stable when stored at temperatures of –70°C to –20°C, it is considerably less stable when stored at normal refrigeration temperatures; at 4°C about 90% of infectivity is lost within a week. PRRSv is stable at pH 6.5–7.5, but infectivity is rapidly lost at a pH of below 6 or above 7.5 (42).

Pigs are the only animals affected by the virus. It exhibits a very high level of genetic diversity, and there are large differences in pathogenicity between strains of the virus. The main target cells for PRRSv are macrophages, particularly those in lungs and lymph nodes. Infection has not been detected in macrophages or their precursor cells in liver, kidney, heart or bone marrow. While alveolar macrophages are the most favoured cell for replication, only about 2% of these cells become infected, even at the peak of virus replication in the lungs (12).

Transmission has been demonstrated by multiple routes of exposure: intranasal, intramuscular, oral, intrauterine and vaginal. Pigs are extremely sensitive to parenteral exposure, but considerably less so by other routes (42).

Following infection, there is a rapid onset of viraemia with replication in a number of organs. Viraemic titres peak within four days of infection. While most animals will not be viraemic after 28 days, extended periods of viraemia of up to nine weeks have been reported (12).

Since viral replication has not been demonstrated in muscle or endothelial cells, the presence of virus in meat is assumed to reflect viraemia. However, lymphoid tissue can be expected to contribute to viral titres in some cuts of meat (12).

Epidemiology

Very few pig-producing countries are considered free from PRRS. In Europe, Norway, Finland and Switzerland have remained free. New Caledonia, New Zealand, Australia and several countries in South America are also considered free (42). Chile was found to have a low prevalence of PRRS infection in 2000 (32) but has been successful in

eradicating the virus (33), with the last reported occurrence being in 2007 (40).

An incursion of PRRS on a farm in Sweden was detected by routine serosurveillance in July 2007 and further infected properties were identified by serological sampling of pigs at slaughter. Control measures included movement controls, depopulation of pigs weighing less than 75 kg, and supervised slaughter of pigs weighing more than 75 kg, with careful cleaning and disinfection of the slaughter plant. No restrictions were placed on the meat from slaughtered animals (8). Scenario tree modelling supports Sweden's claim to have eradicated the virus by December 2007 (17).

In endemic areas, most herds become infected by movement of infected pigs or by contaminated semen. Spread from neighbouring farms can also occur via a range of fomites (42), and in high-pig-density areas the virus is probably transmitted through aerosols as well (31). However, genetic studies in the United States support the conclusion that long-distance spread, most likely by infected animals and semen, is the major route, even when local spread is suspected by insects or aerosols (18).

In countries where PRRS is present, the rate of herd infection varies. In the United States 60% of unvaccinated herds were reported to be infected (34). Within-herd prevalence can be very high in some herds. In Canada 85% of slaughter pigs sampled in Quebec in 1993 were antibody-positive (26), and the seropositivity rate in 1,039 pigs arriving at two slaughterhouses (one in Quebec, one in Manitoba) was 74% (25). In Chinese Taipei 85% of market pigs were reported to be seropositive (37). The seroprevalence in infected farms can reach 95% within two to three months of introduction (1).

Presence of porcine reproductive and respiratory syndrome virus in meat

Determining the presence of PRRSv in meat requires the application of one or more of the following diagnostic tests:

– virus isolation

– reverse-transcription polymerase chain reaction (RT-PCR)

– feeding studies.

Virus isolation has limited sensitivity in detecting low titres; the limit of detection in meat was reported by Bloemraad *et al.* (5) to be about $10^{2.8}$ median tissue cell

Rev. sci. tech. Off. int. Epiz., **30** (1)

141

infective dose ($TCID_{50}$) per g while later van der Linden *et al.* (35) considered it to be somewhat lower at $10^{1.8}$ $TCID_{50}$ per g. Reverse-transcription polymerase chain reaction is considerably more sensitive, and its use has led to higher estimates of infectivity in meat (25, 35). However, since RT-PCR detects viral RNA rather than infectious virus, its use alone appears to result in overestimation of the likelihood of infectivity in meat (2, 19, 21). While feeding trials appear at first sight to be the most objective test of infectivity in meat, they must be carefully designed to avoid horizontal transmission between recipient pigs (25, 35). Moreover, the high cost of feeding trials prevents their wider use in more fully exploring the issue of the infectious dose of PRRSv.

As discussed by Farez and Morley (13), several studies carried out in the 1990s reported the isolation of PRRSv from meat and associated regional lymph nodes of small numbers of pigs. Bloemraad *et al.* (5) took meat samples from four artificially infected pigs, two of which were slaughtered at 5 days post inoculation (PI), and two at 10 days PI. Virus was present in leg muscle of one pig at 5 days PI; the titre at 0 h post mortem was $10^{3.7}$ $TCID_{50}$ and by 24 h post mortem (stored at 4°C) the titre was $10^{2.9}$ $TCID_{50}$. In another pig slaughtered at 10 days PI the virus was found in diaphragm muscle 24 h post mortem at a titre of $10^{2.8}$ $TCID_{50}$; this titre was considered to be the limit of detection by tissue culture. However, by 48 h post mortem, no virus was detectable in any of the muscle specimens from any of the four pigs held at 4°C. Mengeling *et al.* (27) isolated PRRSv from meat of only one of six experimentally infected pigs, while Magar *et al.* (26) were able to isolate virus from muscle and lymph nodes of two pigs at 7 days PI but not at 14 days PI.

Several studies on meat at the point of slaughter were also carried out in the 1990s. Larochelle and Magar (24) collected meat samples from packages of frozen meat ready for export from four Canadian processing plants in an area where PRRS was endemic. No virus could be isolated from 2,190 individual carcass samples pooled in groups of five prior to testing. Frey *et al.* (16) sampled fresh pork derived from commercially slaughtered pigs in the United States. Virus was isolated from six sample pools out of a total of 1,049 sample pools taken from 178 lots of fresh pork (40,000 lb per lot). Most positives were obtained only after multiple cell culture passages, and virus titres were so low that confirmation by re-isolation was not always successful and had to be done by RT-PCR. In Chinese Taipei 85% of pigs tested at three abattoirs were seropositive for PRRSv, but none of 472 carcass samples of market pigs at slaughter was positive by RT-PCR (37).

These studies collectively demonstrated that the likelihood of isolating virus from meat of pigs at slaughter was low and, as a result, it was generally considered in the 1990s that meat was unlikely to be a vehicle for transmission of PRRS.

Feeding trials

Motivated by concerns about the limitations to the sensitivity of virus isolation, several feeding trials were carried out in the 2000s.

Van der Linden *et al.* (35) took meat samples at slaughter from 24 pigs that had been artificially infected with PRRSv 11 days earlier. At this point 12 of the 24 samples were positive for PRRS by virus isolation. After freezing for 10 days at −23°C, samples were tested by virus isolation and RT-PCR. Although only two out of the 24 samples were positive by virus isolation at this point, all but one sample were RT-PCR positive. After 14 days' storage at −23°C, two 500-g samples of raw muscle meat from each donor pig were thawed, cut into pieces about 7 cm³ and fed over two days (250 g per day) to two recipient pigs. Thus, each of the 48 recipient pigs consumed 500 g of raw meat over two days. Recipient pigs had been deprived of food for two days, and uptake by these animals was classified as good or moderate, and recipient pigs were observed to chew the meat samples. Three days after feeding, 50% of the recipient pigs (24 of 48) were viraemic. Although six days after feeding all 48 recipient pigs were viraemic, the authors were unable to determine whether they had become infected by eating meat or by horizontal transmission from the other recipient pigs. Nevertheless, four of the recipient pigs that became viraemic by day three had been fed meat from which virus could not be detected, either before or after freezing, suggesting that there was sufficient infectivity in 500 g of raw muscle meat to infect recipient pigs even when the titre was below the detection limit of virus isolation. Although the question of infectious dose was not examined in detail, van der Linden *et al.* (35) also demonstrated oral transmission of PRRS by feeding 500 g meat samples spiked with PRRSv at a titre of $10^{2.8-3.3}$ $TCID_{50}$ per g.

Magar and Larochelle (25) found that 19 of 1,027 meat samples (1.85%) randomly collected at two Canadian slaughterhouses were positive for PRRSv by RT-PCR, even though only one sample was positive by virus isolation. When meat from 11 of the RT-PCR-positive carcasses was fed to pairs of recipient pigs, in quantities from 1.05 kg to 1.8 kg over two days, seven of the 11 pairs (63%) became infected. From this study it may be concluded that approximately 1.2% of pigs *at slaughter* can be expected to have infectious virus in meat, at least under North American conditions, but the titre of virus will be below the threshold of detection by virus isolation.

Both of the above feeding trials exhibited design deficiencies. The large amounts of meat fed to each of the

recipient pigs (500 g over two days in the case of van der Linden *et al.* [35], and a variable amount, from 1.05 kg to 1.8 kg over two days, in the case of Magar and Larochelle [25]) leave ample room for speculation as to how this result should be interpreted with respect to the degree of risk posed by scraps of meat that may be incorporated into pig swill. Both of these studies reported that pigs were reluctant to eat the pork pieces, even though in the case of the van der Linden *et al.* study (35) the meat had been cut into pieces just under 2 cm^3. Apparently in view of the meat's low palatability, the recipient pigs in both trials were starved for 24 h prior to the feeding event in order to encourage consumption.

Cano *et al.* (7) reported that juice from thawed meat of pigs slaughtered at the peak of viraemia may contain sufficient virus to initiate infection by the oral route within 30 min of meat thawing. The relevance of this to field situations is unclear.

However, Molina *et al.* (29) further investigated the transmissibility of PRRS by ingestion of meat from infected animals, and reported that, while 13 of 89 muscle samples (14.6%) were positive for PRRS by RT-PCR at various intervals post infection, in none of these 13 cases did the feeding of 100 g to 200 g of meat to individually housed recipient pigs result in infection.

None of the feeding trials described to date has used meat samples that have been subject to normal commercial processing and handling conditions. As discussed later in this article, post-slaughter bleeding, maturation, refrigeration, and other processes that increase the time delay between slaughter and the product's arrival at the point of retail can be expected to have a profound effect on the titre of PRRSv in pig meat.

Infectious dose

In general, the infectious dose of viruses is known to vary with route of exposure. In the case of the lactic dehydrogenase-elevating virus (LDV) of mice (also an arterivirus), transmission by oral exposure requires much higher doses than other routes. A single LDV particle may be able to transmit infection by intraperitoneal or tail cartilage injection (6), whereas only 18% to 50% of mice became infected after drinking 20 ml to 50 ml water containing a median infectious dose (ID$_{50}$) of $10^{8.1}$ to $10^{8.8}$ per ml, and no mice became infected as a result of consuming a similar amount of water that contained only $10^{6.4}$ ID$_{50}$ per ml (30).

The literature contains limited information on the infectious dose for PRRSv. Yoon *et al.* (41) reported that 2 ml of an inoculum containing 10 fluorescent units of

PRRSv per ml were sufficient to infect young pigs by the intranasal or intramuscular routes. Benfield *et al.* (3), in a study on the titre of PRRSv in semen required to infect pigs by artificial insemination, found that only one in five gilts seroconverted after exposure to $10^{3.3}$ TCID$_{50}$, and none of five seroconverted after exposure to $10^{2.3}$ TCID$_{50}$.

Hermann *et al.* (20) estimated the ID$_{50}$ for PRRSv to be $10^{5.3}$ TCID$_{50}$ (95% confidence interval [CI] $10^{4.6}$ – $10^{5.9}$) by the oral route. The infectious dose by the intranasal route, by comparison, was estimated as $10^{4.0}$ TCID$_{50}$ (95% CI $10^{3.0}$ – $10^{5.0}$) while intramuscular exposure of animals to $10^{2.2}$ TCID$_{50}$ (positive controls) resulted in infection in all animals, indicating that pigs were most susceptible to infection via parenteral exposure. However, these studies were conducted in healthy young animals, and it is known that infectious dose may also be affected by other factors such as virus strain, concurrent infections and host factors, including sex and age (22).

In estimating a dose-response curve for oral PRRSv, Hermann *et al.* (20) essentially rejected the notion of a minimum infectious dose. The limit of detection of PRRSv in meat is $10^{1.8}$ TCID$_{50}$ per g of meat, so 500 g of such meat would contain about $10^{4.5}$ TCID$_{50}$ of virus which, from the findings of Hermann *et al.* (20), would be considered likely to infect 25% to 30% of pigs receiving this dose by the oral route. The finding that meat containing virus below detectable levels can result in infection when 500 g to 900 g of meat is fed to pigs (25, 35) is consistent with the dose-response relationship estimated by Hermann *et al.* (20).

Quantifying the risk of porcine reproductive and respiratory syndrome virus from imported meat

Based on the approach first described by the European Food Safety Authority (EFSA) (12), sufficient data are now available to allow the construction of a simple model to estimate the likelihood that pork imported from a country where PRRS is endemic will contain an infectious dose of virus if fed directly to a susceptible recipient (Fig. 1).

Likelihood of pork being infectious at slaughter

The proportion of pork imported from a country where PRRS is endemic that would be expected to contain an infectious dose of virus at slaughter can be derived from the findings of Magar and Larochelle (25), which suggest

Rev. sci. tech. Off. int. Epiz., **30** (1)

143
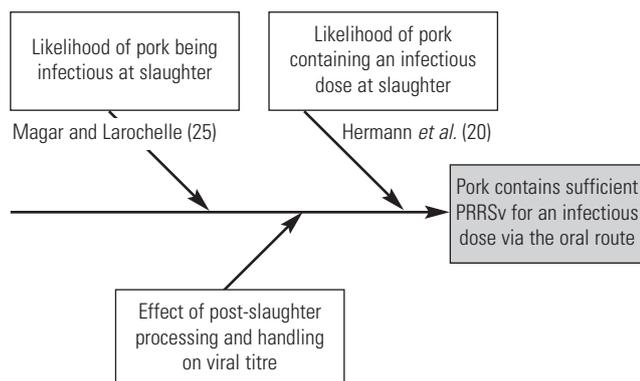
Fig. 1
Ishikawa diagram of a simple model to estimate the likelihood of imported pork containing an infectious dose of porcine reproductive and respiratory syndrome virus

that 1.2% of meat samples taken from pigs at slaughter are likely to contain an infectious dose of PRRSv.

Effect of post-slaughter processing and handling on viral titre

Meat containing an infectious dose of PRRSv at slaughter is likely to come from those pigs which are at the peak of viraemia. Meta-analysis of published studies on pigs over 13 weeks old (5, 9, 23, 36, 39) suggests that the mean concentration of PRRSv in the blood of pigs at the peak of viraemia at the time of slaughter would be $10^{3.18}$ TCID$_{50}$/ml blood.

The residual blood content of lean meat is 2 ml/kg to 9 ml/kg muscle (38), so the viral titre of meat after bleeding out will be no more than 0.9% of the peak viraemic titre.

Maturation takes a minimum of 30 h and the use of fast or intensive cooling results in a quick reduction of the carcass temperature to 7°C. On the basis of this, EFSA (12) estimated that the minimum effect of maturation would be a 1 log (90%) reduction of the amount of viable virus. However, a more recent publication (21) suggests that (as a worst-case scenario) storage at 7°C for 30 h may result in as little as a 14% reduction in viable virus.

The delay between maturation and the arrival of the product at the point of retail will affect the viral titre in meat. As with maturation, this aspect was considered by EFSA (12), which concluded that thawing after freezing would result in a 1 log reduction in the titre of virus present in pig meat. The findings of Bloemraad et al. suggest that for any meat imported chilled (not frozen) there would be a 1 log reduction in virus after 30 h at 4°C (5). However, as noted above, Jacobs et al. (21) suggest the reduction may not be as much, and for a shipping time of four days, they predict that storage at 4°C may result in as little as a 32% reduction in viable virus.

Likelihood of pork containing an infectious dose of PRRSv at point of retail

Hermann et al. (20) estimated the probability of infection by the oral route for different doses of PRRSv. The logistic regression model derived by Hermann et al. (20) predicts the following relationship between oral dose (y) and probability of infection (p):

$$p = (10^{((y-5.2692)/1.7929)}) / (1+10^{((y-5.2692)/1.7929)})$$

As the likely concentration of virus in imported pork at the point of retail (taking into account the effect of bleeding out, maturation and refrigeration/freezing) can be estimated as described above, the formula derived from the findings of Hermann et al. (20) allows us to predict the probability that a known weight of pork taken from a viraemic individual will contain an infectious dose of virus if fed to a susceptible recipient (Table I). Furthermore, as the available evidence suggests that only 1.2% of meat samples taken from pigs at slaughter are likely to harbour an infectious dose of PRRSv, we can also estimate the probability that a known weight of pork imported from a country where PRRSv is present will contain an infectious dose of the virus (Table II).

It should be noted that the logistic regression model predicts that very small doses of PRRSv (conceptually less than one virus particle) will have a non-zero probability of being infectious when administered by the oral route. However, the Hermann et al. studies administered doses only as low as $10^{2.2}$ TCID$_{50}$ and no infection was recorded when these lower doses were given by either the oral or the intranasal route (20). Alternative mechanistic modelling approaches using the data from Hermann et al. (20) may therefore be more appropriate when considering the probability of PRRS infection using small doses of virus (less than $10^{2.2}$ TCID$_{50}$).

Furthermore, PRRSv in meat may be bound to cells or neutralising antibodies which will reduce virus bioavailability. The Hermann et al. (20) study used cell culture supernatant where infectious virus particles are not bound to a matrix, whereas the infectivity of a piece of meat is likely to be much lower than that of a cell culture supernatant containing the same dose of virus.

It should be recognised that using the Hermann et al. study to predict the infectivity of pieces of meat, especially small pieces that are likely to contain doses of virus below $10^{2.2}$ TCID$_{50}$, is to take a conservative approach that is likely to overestimate the risk of infection.

These calculations assume that imported pork does not contain lymph nodes. Duan et al. (11) found that the titre of PRRSv in porcine lymph nodes up to 14 days after experimental inoculation was likely to be between

Table I
The probability that a given weight of pig meat taken from a viraemic individual will contain an infectious dose of porcine reproductive and respiratory syndrome virus after different stages of post-slaughter handling and processing

Weight of meat (g)	Probability that meat derived from an infected individual at the peak of viraemia will contain an infectious dose of PRRSv			
	At slaughter	After bleeding out	After maturation	After shipping
20	0.267	0.026	0.024	0.019
100	0.471	0.061	0.056	0.046
250	0.598	0.097	0.090	0.074
1,000	0.763	0.189	0.176	0.147
5,000	0.888	0.364	0.344	0.298
10,000	0.921	0.457	0.436	0.384

Table II
The probability that a given weight of pig meat from a country where porcine reproductive and respiratory syndrome virus is present will contain an infectious dose of virus after different stages of post-slaughter handling and processing

Weight of meat (g)	Probability that meat imported from a country where PRRSv is present will contain an infectious dose of PRRSv			
	At slaughter	After bleeding out	After maturation	After shipping
20	3.2×10^{-3}	3.1×10^{-4}	2.8×10^{-4}	2.3×10^{-4}
100	5.7×10^{-3}	7.3×10^{-4}	6.7×10^{-4}	5.5×10^{-4}
250	7.2×10^{-3}	1.2×10^{-3}	1.1×10^{-3}	8.9×10^{-4}
1,000	9.2×10^{-3}	2.3×10^{-3}	2.1×10^{-3}	1.8×10^{-3}
5,000	1.1×10^{-2}	4.4×10^{-3}	4.1×10^{-3}	3.6×10^{-3}
10,000	1.1×10^{-2}	5.5×10^{-3}	5.2×10^{-3}	4.6×10^{-3}

$10^{1.7}$ and $10^{4.9}$ $TCID_{50}$/g and bootstrap analysis of these data suggests that the mean titre of virus in the lymph nodes of viraemic pigs would be $10^{3.2}$ $TCID_{50}$/g. If it is assumed that bleeding out will have no effect on this viral titre then, using the simple model described above, the probability that a 20 g lymph node taken from an imported joint would transmit infection to a susceptible recipient is estimated to be 0.0026 (0.26%). However, it would be reasonable to suggest that any lymphoid tissue buried within a joint of pork would be unlikely to be discarded before cooking, which would effectively inactivate any virus present.

A number of local factors are not considered in the simple model described here, such as:

– domestic legislation to prevent waste feeding of pigs

– the national prevalence of backyard pig-keeping

– the amount of waste likely to be generated by different cuts of meat

– the volume of imports from countries where PRRSv is present

– the cuts of pork likely to be imported

– whether imports are more likely to be fresh or frozen

– the likelihood that a backyard pig-keeper would purchase imported pork rather than rely on his or her own supply

– the effect of delays between pork arriving on a supermarket shelf and being purchased

– any delay that may occur between scrap generation in a domestic kitchen and the feeding of such scraps to a backyard pig.

Furthermore, it is unlikely that significant quantities of fresh raw meat waste would be generated from the forms of pig meat likely to be traded internationally. Nevertheless, the results of this exercise illustrate that international trade in pig meat is unlikely to result in the dissemination of PRRSv.

Conclusion

Feeding trials have demonstrated that it is possible to transmit PRRSv to susceptible recipients through the consumption of infected meat. However, as described above, these studies have had a number of shortcomings and, most significantly, none of these studies has attempted to transmit PRRS using meat that has been subject to normal commercial handling practices.

There are now sufficient data available in peer-reviewed literature to allow the construction of a simple model which illustrates that the international trade of commercially produced pig meat is unlikely to result in the dissemination of PRRS. Although lymph nodes which may be present in certain cuts of pig meat may represent a higher risk of disease transmission than muscle tissue, if these tissues are likely to be subject to cooking before disposal, then the removal of lymph nodes from traded pig meat to manage the perceived risk of PRRS may be unwarranted.

In New Zealand, there was a three-and-a-half year period (the beginning of 1998 to mid-2001) during which pig meat was imported from PRRS-infected countries without any sanitary measures against PRRS being imposed on imports. Despite this, New Zealand remained free from PRRSv. About 30,000 tonnes of pig meat may have been imported from PRRS-infected countries over that three-and-a-half year period without any controls on garbage

Rev. sci. tech. Off. int. Epiz., **30** (1)

145

feeding being in place in the country. Similarly, both Sweden and Chile have recently eradicated PRRS and, in both these cases, meat from infected pigs was released without restriction for domestic consumption.

When all the above evidence is considered together, it is difficult to avoid the conclusion that the likelihood of transmitting PRRS through the international trade in pig meat must be considered negligible. ■

La dissémination d'agents pathogènes lors des échanges internationaux de viande porcine : état de la situation et évolutions récentes

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Résumé
La viande utilisée dans l'alimentation des porcs est une source de transmission potentielle de maladies lorsqu'il s'agit de restes de viande importée de pays infectés. En conséquence, de nombreux pays ont réglementé l'utilisation des déchets pour l'alimentation porcine. Les principales maladies porcines concernées par cette voie de transmission sont la fièvre aphteuse, la peste porcine africaine, la peste porcine classique et la maladie vésiculeuse du porc. Le *Code sanitaire pour les animaux terrestres* (le *Code terrestre*) de l'Organisation mondiale de la santé animale (OIE) contient des recommandations permettant de gérer les risques associés à la viande importée de pays non indemnes de ces maladies. En revanche, le *Code terrestre* n'a pas de chapitre consacré au syndrome dysgénésique et respiratoire du porc (SDRP), maladie virale relativement récente, décrite pour la première fois dans les années 90 et devenue endémique dans la plupart des pays producteurs de porcs. Après avoir évalué le risque de dissémination du virus du SDRP lors des échanges internationaux de viande porcine, les auteurs concluent que la probabilité de transmission par cette voie est négligeable.

Mots-clés
Analyse du risque à l'importation – Commerce international – Porc – Porcin – Syndrome dysgénésique et respiratoire du porc – Viande porcine.
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Propagación de agentes patógenos por el comercio en carne porcina: panorámica general y evolución reciente

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Resumen
Varias enfermedades animales pueden transmitirse por la carne de cerdo cuando los animales se han alimentado con sobras de carne importada de países infectados. De ahí que en muchos países la alimentación de los cerdos con basura esté sujeta a reglamentación. Las principales enfermedades

porcinas que según se ha comprobado se transmiten por esta vía son la fiebre aftosa, la peste porcina africana, la peste porcina clásica y la enfermedad vesicular porcina. En el *Código Sanitario para los Animales Terrestres* (el *Código Terrestre*) de la Organización Mundial de Sanidad Animal se ofrecen una serie de recomendaciones para gestionar los riesgos ligados a la carne procedente de países en los que están presentes dichas enfermedades. Sin embargo, en ningún capítulo del *Código Terrestre* se habla del síndrome disgenésico y respiratorio porcino, enfermedad vírica de los cerdos relativamente nueva, que, desde que fue descrita en los años noventa, se ha convertido en endémica en la mayoría de los países con una gran producción porcina. Los autores, tras evaluar el riesgo de diseminación del virus de ese síndrome por el comercio de carne de cerdo, concluyen que las probabilidades de transmisión por esta vía son ínfimas.

Palabras clave

Análisis del riesgo de importación – Cerdo – Comercio internacional – Carne porcina – Síndrome disgenésico y respiratorio porcino.

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147

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