Import risk analysis: Porcine reproductive and respiratory syndrome (PRRS) virus in pig meat

25 July 2006

This page is intentionally blank

Import risk analysis: Porcine reproductive and respiratory syndrome (PRRS) virus in pig meat

Biosecurity New Zealand
Ministry of Agriculture and Forestry
Wellington
New Zealand

25 July 2006

This page is intentionally blank

Ministry of Agriculture and Forestry
Te Manatu Ahuwhenua, Ngaherehere
ASB Bank House
101-103 The Terrace
P O Box 2526
Wellington
New Zealand

Telephone: +64 4 474 4100
Facsimile: +64 4 474 4133
Internet: http://www.maf.govt.nz

Biosecurity New Zealand

Import risk analysis: Porcine reproductive and respiratory syndrome (PRRS) virus in pig meat

25 July 2006

Approved for general release

Debbie Pearson
Director Preclearance
Biosecurity New Zealand

This page is intentionally blank

TABLE OF CONTENTS

CONTRIBUTORS TO THIS RISK ANALYSIS

1. Primary Authors

2001	Noel Murray	National Adviser, Risk Analysis	Animal Biosecurity MAF Biosecurity Authority Ministry of Agriculture and Forestry, Wellington, New Zealand
2005	Howard Pharo	Team Manager, Risk Analysis (Animal Kingdom)	Biosecurity New Zealand Ministry of Agriculture and Forestry, Wellington, New Zealand

2. Internal Peer Review

2.1 Release assessment (2001)

Stuart MacDiarmid	National Manager, Risk Analysis	MAF Biosecurity, Ministry of Agriculture and Forestry, Wellington, New Zealand

2.2 Full risk analysis (2005)

Leone Basher	Senior Adviser, Animal Imports	Biosecurity New Zealand Ministry of Agriculture and Forestry, Wellington, New Zealand
Stuart MacDiarmid	Principal International Adviser, Risk Analysis, International Coordination	Biosecurity New Zealand Ministry of Agriculture and Forestry, Wellington, New Zealand

3. External Scientific Review

3.1 Release assessment (2001)

Scott Dee	College of Veterinary Medicine	University of Minnesota, USA
Michael Murtaugh	Professor, Department of Veterinary Pathobiology	University of Minnesota, USA
Jeff Zimmerman	Veterinary Diagnostic Laboratory, College of Veterinary Medicine	Iowa State University, USA

3.2 Full risk analysis (2005)

Lis Alban	Veterinary Epidemiologist	Danske Slagterier Denmark
Scott Dee	Associate Professor Clinical & Population Sciences	College of Veterinary Medicine University of Minnesota USA
Spiridon Doudounakis	Head, Unit of Animal Diseases	General Veterinary Directorate Ministry of Rural Development and Food Greece
Torben Grubbe	Animal Health Adviser	Animal Health Division Danish Veterinary and Food Administration Denmark
Noel Murray	Animal Health Policy Analyst Animal Health and Production Division	Canadian Food Inspection Agency Canada
Colin Wilks	Professorial Fellow Veterinary Virology & Microbiology	Faculty of Veterinary Science University of Melbourne Australia
Jeff Zimmerman	Associate Professor Veterinary Diagnostic Laboratory	College of Veterinary Medicine Iowa State University USA

1. EXECUTIVE SUMMARY

In September 2001 the Ministry of Agriculture and Forestry (MAF) imposed sanitary measures on imported pig meat to manage the risk of introduction of porcine reproductive and respiratory syndrome (PRRS) virus. This followed the publication of a report commissioned by the Australian government demonstrating that, contrary to previous scientific opinion, PRRS virus could be present in meat of infected animals, and that transmission of the virus was possible by feeding such meat to susceptible pigs.

In late 2001 MAF completed a draft release assessment for PRRS in imported pig meat. This was subjected to international technical review in 2002, according to MAF's risk analysis process. In mid 2002 a Canadian study was initiated to test the conclusions reached in the previous study. The results from the two studies were published in the scientific literature in 2003 and 2004 respectively, opening the way for MAF to complete the risk analysis.

This document is the complete risk analysis of PRRS virus in imported pig meat. The conclusions of this analysis are as follows.

1. There is a low likelihood that chilled or frozen pig meat from a country with endemic PRRS will harbour the virus when imported into New Zealand.

2. Since cooking inactivates PRRS virus, and since pigs are the only species susceptible to this organism, effective exposure would require the feeding of uncooked pig meat to pigs in New Zealand. Although scraps may be generated from imported pig meat at several points during its preparation for human consumption, the feeding of raw meat to pigs is illegal under the 2005 garbage feeding regulations. It is concluded that an exposure pathway would exist only on pig farms that were not complying with the garbage feeding regulations.

3. If pig farms in this country did become infected with PRRS through the illegal feeding of uncooked imported pig meat, the likelihood of spread to other pig farms would be low as long as standard biosecurity practices were observed.

4. If PRRS virus were introduced into New Zealand, the consequences would be significant on affected farms, particularly in breeding units.

It is considered that the risk of PRRS in imported pig meat is non-negligible, and the following sanitary measures are recommended to manage the identified risk.

Pig meat must be:

either

- from a country free from PRRS

or

- treated prior to import or on arrival, in an officially approved facility, by approved cooking or pH change

or

- in the form of consumer-ready, high value cuts

or

- further processed on arrival, in an officially approved facility, into consumer-ready high value cuts

This page is intentionally blank

2. INTRODUCTION

2.1 BACKGROUND

Until September 2001 pig meat was imported into New Zealand without sanitary measures for porcine reproductive and respiratory syndrome (PRRS) virus, as the prevailing scientific view was that PRRS virus was unlikely to be transmitted to susceptible pigs through the ingestion of pig meat. However, a study commissioned by the Australian Government, carried out at Lelystad in 1999 (Steverink, 2000), demonstrated that it was possible to transmit the virus by this route. MAF's preliminary assessment of the Lelystad study resulted in provisional measures being adopted from September 2001. These measures required that imported pig meat be either cooked or subjected to certain pH levels before being given a Biosecurity clearance in New Zealand. Since these measures are provisional, a risk analysis was required to finalise MAF's position.

This risk analysis began with the completion in 2001 of a draft release assessment, which was then subjected to international peer review in 2002. From late 2002 until 2005 the risk analysis was on hold while MAF awaited the publication in peer reviewed scientific journals of the results of critically important trials that were known to have been carried out or were underway. The Lelystad study was published (Van der Linden et al, 2003) and these results were subsequently confirmed by a similar study carried out in Canada (Magar and Larochelle, 2004).

2.1.1 The history of pig meat imports into New Zealand

Chilled, frozen, processed and cured pig meat is imported into New Zealand in large quantities, and approximately 30-35% of the total pork consumption in this country is imported. As can be seen in Table 1, approximately 97% of imported pork is in frozen form.

Table 1. New Zealand's imports of pig meat in kg, by calendar year.

	Chilled	Frozen	Processed or cured	Total
2000	no data	13,750,159	416,602	14,166,761
2001	10,690	13,482,219	306,048	13,798,957
2002	45,362	16,814,431	405,042	17,264,835
2003	289,519	18,412,976	695,616	19,398,111
2004	66,266	18,273,315	706,120	19,045,701
2005 to May	125	9,292,398	316,332	9,608,855

Source: Pork Industry Board.

Apart from a small amount of cooked pig meat that is imported chilled, the majority of chilled imported pork comes from Australia.

Pig meat is imported from a number of countries, and the mix of countries changes over time according to market conditions. Nevertheless, Table 2 shows that more than 90% of imported pig meat currently comes from Australia and North America. Moreover, of the countries listed in Table 2, only Sweden and Australia are free from PRRS.

Table 2. Imports of pork by country of origin, from 1999-2000 to 2004-05♥.

			Imports per year (June – June)					
			1999-00	**2000-01**	**2001-02**	**2002-03**	**2003-04**	**2004-05♣**
Total imports		Tonnes	10,765	12,959	16,409	19,513	18,733	14,995
Country	**PRRS Status♠**							
Australia	✖	Tonnes	1,895.7	2,946.5	5,908.8	9,593.4	8,749.6	6,167.3
		%	18	23	36	49	47	41
Canada	✓	Tonnes	6,360.8	8,727.5	7,787.4	7,322,2	5,613.2	4,588.6
		%	59	67	47	38	30	30
China	✓	Tonnes	19.8	54.8	72.7	63.3	105.0	81.8
		%	<1	<1	<1	<1	<1	<1
Denmark	✓	Tonnes	2,178.6	793.4	512.7	3.9	115.3	181.6
		%	20	6	3	<1	1	1
Netherlands	✓	Tonnes	25.5	23.1	11.0	1.3	0.1	0.0
		%	<1	<1	<0.1	<0.1	<0.1	0
Sweden	✖	Tonnes	0	0	645.4	878.2	1,137.8	1,071.6
		%	0	0	4	5	6	7
USA	✓	Tonnes	175.4	409.9	1,440.6	1,631.7	2,952.1	2,890.6
		%	2	3	9	8	16	19
Other	✓	Tonnes	110	3.9	31.2	18.1	58.9	13.1
		%	1	<1	<1	<1	<1	<1

♥ Source: New Zealand Pork Industry Board.

♣ figures to May 2005

♠ Source: OIE Handistatus II (http://www.oie.int/hs2/report.asp?lang=en)

The countries included in the "other" category of Table 2 are:
Asia - Taiwan, Thailand, Hong Kong, Korea, Malaysia, Vietnam, Japan
Europe - Germany, Ireland, UK, Italy, France, Ukraine, Croatia
Miscellaneous - Russia, South Africa, New Caledonia
PRRS is endemic in the majority of these countries[1].

Over the past 5 years imports from Europe have declined considerably, particularly from Denmark (fallen from 20% of total imports to less than 1%), although there has been a small increase in imports from Sweden (now 6-7% of total imports). Imports from Canada declined over the same period from about 60% to about 30% of total imports, but imports from the USA have increased, from only 2% to almost 20% of total imports now. The biggest increase in imports over the last 5 years has been from Australia; this has increased from under 20% to over 40% of total imports.

It is significant to note from Table 2 that pig meat imports have increased by almost 50% since the provisional measures were put in place in September 2001, from about 13,000 tonnes annually in 2000-2001 to about 19,000 tonnes.

According to Pork Industry Board records, over the period 1993 to 2000, about 50,000 tonnes of pig meat was imported from countries where PRRS was endemic, that is, about 7,000 tonnes per year. Using the figures in Table 2, an estimate can be made of the amount of pig meat imported from countries where PRRS is endemic since 1999-2000 (assuming that all of the 'other' countries each year had PRRS). With the increase in total annual imports of pig meat since 2000 the proportion of imports from countries with PRRS has declined. Table 3 demonstrates that whereas in 1999-2000 more than 80% of imported pig meat came from

[1] OIE Handistatus, http://www.oie.int/hs2/report.asp?lang=en

countries with PRRS, imports from such countries now comprise only about 50% of the total. Nevertheless, the amount of pig meat imported from countries with PRRS has remained more or less constant at about 7,000 to 10,000 tonnes per year.

Table 3. Pig meat imports by year from countries with endemic PRRS.

	YEAR					
	1999-00	**2000-01**	**2001-02**	**2002-03**	**2003-04**	**2004-05♣**
Pig meat imported (tonnes)	8,870	10,012	9,855	9,040	8,844	7,755
% of total imports	82	77	60	46	47	52

Source: Pork Industry Board.
♣ to May 2005

2.1.2 Pig farming in New Zealand.

Pig production in New Zealand is characterised by a relatively small number of large herds, and many small herds. In 2001 the New Zealand Pork Industry Board (NZPIB) set up a voluntary farm registration system within the *AgriBase* framework[1]. NZPIB's objective was to establish an infrastructure for any emergency response, for which purpose registration of farms with 20 or more pigs was sought. As at December 2005, 384 herds with about 255,000 pigs were registered on the voluntary system. However, the NZPIB voluntary farm registration system does not capture all the farms with pigs. As at December 2005 AgriBase contained records of a further 7,132 properties with pigs, accounting for about another 82,000 pigs. Table 4 shows these records.

Table 4. Pig herds in New Zealand by herd size, as at December 2005.

Herd size	**NZPIB pork producers registered in AgriBase**	**AgriBase herds with pigs in addition to NZPIB's registered farms**
1-10	32	6,536
11-19	20	325
20 – 50	74	162
51 – 100	38	46
101 – 200	31	23
201 – 500	53	16
501 – 1000	56	10
> 1000	80	14
Total herds	**384**	**7,132**

Source: *AgriBase*
♣ Note: the New Zealand Pork Industry Board defines a pig herd as one with at least 20 pigs

NZPIB considers that the minimum size for a commercial pig unit is 80 sows, and such a herd would have a standing pig population of around 760 pigs at any time. Thus, by far the majority of pig herds in New Zealand would be considered as non-commercial units, and this would include semi-commercial herds, small backyard herds and hobby-farms. The raising of small numbers of pigs for home consumption is a feature of the New Zealand rural culture and this is a common practice on many farms where sheep, beef or dairy animals are the major source of income.

[1] *AgriBase* is a national database or central index of Agricultural property ownership, location and management throughout New Zealand. It lists both farming and horticultural properties (around 100,000 in total), each with a unique identification code. See: http://www.agriquality.co.nz/page.cfm?s=178,232,368,100000275

Table 5 shows the NZPIB voluntarily registered pig farms classified by herd type, demonstrating that the majority of herds registered with the board are breeding/finishing units.

Table 5. NZPIB voluntary farm registration system, herds by herd type.

Herd type	Number of herds	Number of pigs
Breeding/finishing	140	84,464
Breeding/weaner	93	22,450
Breeding/weaner/finishing	67	62,191
Finishing	44	22,247
Total herds	**344**	**291,352**

Source: Pork Industry Board.

Although groups of pigs numbering less than 20 animals are not considered by the New Zealand Pork Industry Board to be 'herds", such units comprise more than 95% of the total pig population of New Zealand. This relatively little-understood sector of the pig industry is thought to be characterised by short term fluctuations both in the number of herds and the number of animals animals, and diets that are more likely to contain garbage from various sources. It is generally assumed that herds with less than 20 pigs are unlikely to be breeding units, and that compliance with the garbage feeding regulations is likely to be incomplete in this sector.

Since *AgriBase* does not attempt coverage in the urban areas, the backyard pig herds in Auckland and South Auckland are not included in the above figures. There are thought to be a large number of pigs in that region, particularly as a source of pig meat supply to the Polynesian community. It is estimated that up to 70,000 pigs per year may be slaughtered in this area, comprising approximately 10% of the national annual kill.

2.1.3 Regulation of feeding garbage to pigs in New Zealand.

Up to 1997 the feeding of garbage to pigs was controlled by the *Garbage (Feed for Swine or Poultry) Regulations 1980.* Under these regulations persons feeding garbage to pigs or poultry had to be licensed and obtain a permit from the Director-General of Agriculture. MAF carried out inspections to ensure that any meat in garbage was adequately cooked prior to being fed. In 1997 MAF carried out a review of these regulations and it was concluded that they were unenforceable given MAF budgets at that time. It was considered that increased border security was a more appropriate way to manage the risk of exotic diseases entering the pig population by this route. The regulations were allowed to lapse in 1998.

However, with the outbreak of foot and mouth disease in the United Kingdom in 2001, which was thought to arise from feeding pigs garbage that contained imported infected meat, the wider farming industries in New Zealand questioned whether adequate precautions were in place to prevent feeding infected meat to pigs. An analysis of international practice in this area was carried out in 2001/02 (MAF, 2001a), resulting in a recommendation that New Zealand re-impose regulation of garbage feeding to pigs. This was actioned by the development of the *Biosecurity (Meat & Food Waste for Pigs) Regulations 2005*, under which the feeding to pigs of untreated meat or untreated food waste is prohibited. Treatment (cooking) is defined in the regulations as heating throughout to 100°C for 1 hour, or an equivalent standard approved by MAF. These new regulations came into effect on 9 July 2005.

2.2 COMMODITY DEFINITION

The commodity considered in this risk analysis is chilled or frozen pig meat. Pig meat is defined broadly to include any part of the pig apart from offal and hair, which are removed during slaughter. Therefore, in addition to muscle, the commodity includes skin, bone, lymph nodes, oropharyngeal and tonsillar tissue in the case of carcasses or half carcasses, and the entire head in case of full carcasses.

2.3 RISK ANALYSIS METHODOLOGY

In developing Import Health Standards, MAF is required under Section 22 (5) of the Biosecurity Act 1993 (BSA) to consider the likelihood that the imported commodities may harbour organisms and the effect that these organisms may have on the people, the environment and the economy of New Zealand. MAF is also obliged to have regard to New Zealand's international obligations, foremost amongst which is the Sanitary and Phytosanitary (SPS) Agreement of the World Trade Organization (WTO). A key requirement under the SPS Agreement is that Members cannot impose measures on imported goods that are more restrictive than those placed on domestically-produced goods, which in effect means that measures may be considered only for exotic organisms or for endemic organisms that are under official control in this country.

MAF's risk analysis methodology follows the guidelines in Section 1.3 of the *Terrestrial Animal Health Code* of the World Organisation for Animal Health ("the OIE") (OIE 2005). The risk analysis framework is applied by MAF as described in Import Risk Analysis Animals and Animal Products (Murray 2002), which formed the basis of the OIE handbook on this subject (OIE, 2004a).

The process used in this risk analysis is shown in Figure 1.

2.3.1 Hazard identification

The first step in the risk analysis is hazard identification. This process begins with the collation of a list of organisms that might be associated with the commodity. In the case of this risk analysis, only one disease agent is considered, the virus that causes porcine reproductive and respiratory syndrome.

Next, the hazard identification discusses key aspects of epidemiology, including a consideration of the following questions:

1) whether the various commodities could potentially act as a vehicle for the introduction of the organism,

2) whether it is exotic to New Zealand but likely to be present in exporting countries,

3) if it is present in New Zealand,

a) whether it is "under official control", which could be by government departments, by national or regional pest management strategies or by a small-scale programme, or

b) whether more virulent strains are known to exist in other countries.

If the answers to questions 1 and either 2 or 3 are 'yes', the organism is classified as a potential hazard, and a risk assessment is therefore required.

2.3.2 Risk assessment

Section 22 (5) (a) of the Biosecurity Act 1993 requires consideration of the likelihood that organisms may be introduced in imported commodities. This is the focus of the release assessment. Section 22 (5) (b) of the Biosecurity Act 1993 requires consideration of the possible effects of such introduced organisms on the people, the environment and the economy of New Zealand. The exposure assessment is the first part of this consideration, comprising an assessment of the likelihood of spread and establishment of organisms introduced in specific commodities. The consequence assessment follows on from the exposure assessment in considering the impacts of such organisms, if they were to be introduced, to spread and to become established.

Thus, under the OIE methodology, the risk assessment comprises the following steps for each potential hazard:

a) Release assessment - the likelihood of the organism being imported in the commodity.

b) Exposure assessment - the likelihood of animals or humans in New Zealand being exposed to the potential hazard.

c) Consequence assessment - the consequences of entry, establishment or spread of the organism on the people, the environment and the economy of New Zealand.

d) Risk estimation - a conclusion on the risk posed by the organism based on the release, exposure and consequence assessments. If the risk estimate is non-negligible, then the organism is classified as a hazard.

Not all of the above steps may be necessary in all risk assessments. If the likelihood of release is negligible for any potential hazard, then the risk estimate is automatically negligible and the remaining steps of the risk assessment need not be carried out. The same is true if the likelihood of release is non-negligible but the likelihood of exposure is negligible, or where both release and exposure are non-negligible but the consequences of introduction are considered to be negligible.

2.3.3 Risk management

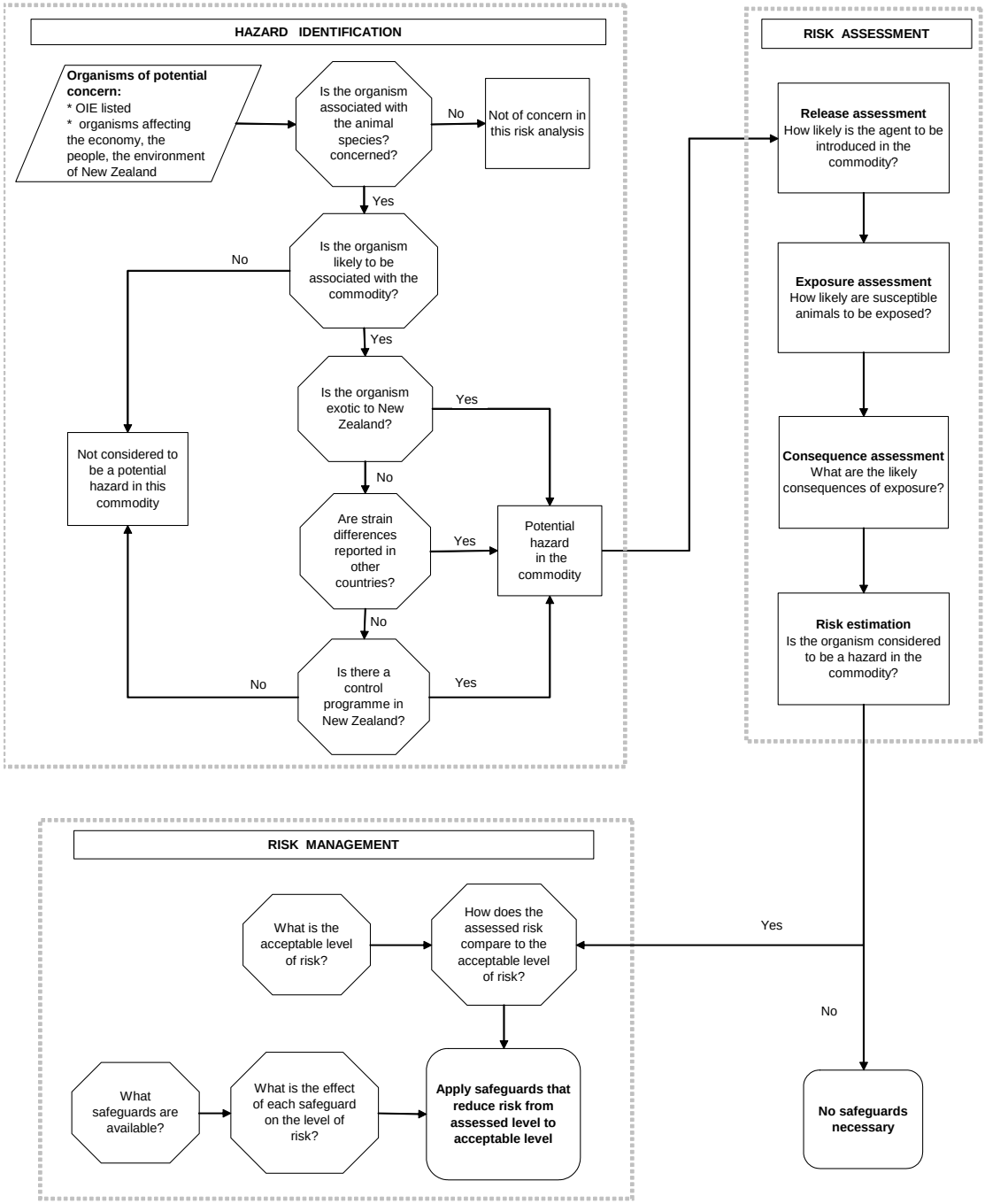
Risk management consists of the following steps:

a) Risk evaluation - to determine whether sanitary measures are necessary.

b) Option evaluation - to identify the options available for managing the risk, and consider risk reduction effects.

c) Recommended measures - the risk management option or combination of options that achieve a negligible likelihood of entry, spread or establishment, while minimising negative trade effects.

Figure 1. The risk analysis process.



3. HAZARD IDENTIFICATION

3.1 AETIOLOGIC AGENT

Porcine reproductive and respiratory syndrome (PRRS) virus, a small single-stranded RNA virus belonging to the genus *Arterivirus*, in the family *Arteriviridae*, order *Nidovirales*.

3.2 OIE STATUS

Listed as a disease notifiable to the OIE.

3.3 NEW ZEALAND'S STATUS

Exotic to New Zealand (MAF, 1996; Motha et al, 1997; Sproule and Fairley, 1998; MAF, 1999; MAF, 2001b; MAF, 2003; Stone and Kittleberger, 2004). Listed on the unwanted organisms register as a notifiable organism.

3.4 EPIDEMIOLOGY

3.4.1 History

Porcine reproductive and respiratory syndrome was first recognised as a disease in the USA in 1987 and in Europe in 1990, but it was not until 1991 that the causative virus was identified (Benfield et al, 1992). Although the origin of the virus is unknown, retrospective serological evidence suggests that PRRS virus emerged in North America in the late 1970s (Benfield et al, 1999), and recent genetic studies suggest that the European and US strains had a common ancestor as late as 1979 (Forsberg et al, 2001). Over the 1990s PRRS virus spread rapidly and it has since been reported in most pig-producing countries, affecting only pigs (Albina, 1997).

3.4.2 Virus properties

Arteriviruses have a high mutation rate due to their mechanism of RNA replication. Thus, PRRS virus is genetically highly unstable, and isolates vary considerably in both nucleic acid sequence and pathogenicity. North American and European isolates of the virus have shown marked genotypic and phenotypic differences, such that they are generally considered to belong to two different genotypes (Yoon, 2003). There is also considerable strain variation within each of these genotypes (Benfield et al, 1999). Moreover, the high divergence observed in key amino acids among British isolates suggests that the virus is continuing to change rapidly (Drew et al, 1997).

The physical stability of Arteriviruses is dictated primarily by the characteristics of their lipid envelope. They are relatively stable at a pH of 6.0 to 7.5, but are inactivated at higher or lower pH. They are inactivated by lipid solvents and heat, and the half life decreases progressively with increasing temperature (Fauquet et al, 2005). PRRS virus persists for 1-6 days at 20-21°C, 3-24 hours at 37°C and 6-20 minutes at 56°C (Benfield et al, 1999). PRRS virus is very stable when stored at temperatures of –70° to –20°C, but less so at normal refrigeration. At 4°C, about 90% of infectivity is lost within 1 week, but low titres of virus can be detected for over 30 days. The fragility of PRRS virus means that it is relatively quickly inactivated in the environment (Benfield et al, 1999).

3.4.3 Virus persistence

In infected pigs PRRS virus can co-exist with antibody for quite some time. Animals will usually become seropositive within 7-10 days post infection (dpi), and it is not uncommon for viraemia to continue up to 20 dpi. The virus replicates almost exclusively in cells of the monocyte/macrophage lineage, although the sensitivity of these cells to virus depends on cell age and organ system; macrophages in the lungs, lymph nodes, tonsil and spleen are most likely to be infected. Antibody apparently assists virus entry into cells by a phenomenon known as antibody dependent enhancement (Yoon et al, 1997; Tirado and Yoon, 2003). Thus, antibody to PRRS virus does not necessarily clear infection or protect against new infections with the virus, and this may explain how different strains of PRRS can infect a herd simultaneously (Dee et al, 2001).

In experimental infections, PRRS virus has been isolated from muscle (Bloemraad et al, 1994; Mengeling et al, 1995; Done et al, 1996; Van der Linden et al, 2003), bone (Mengeling et al 1995; Done et al, 1996) and regional lymph nodes associated with the musculature (Bloemraad et al, 1994; Magar et al, 1995; Mengeling et al 1995). It has also been isolated from samples of fresh pork from commercially killed animals (Frey, 1995a; Magar and Larochelle, 2004).

The virus survives for at least 4 weeks in frozen meat and bone derived from experimentally infected market weight pigs (Frey, 1995b). Viraemia is prolonged and the virus has been shown by virus isolation techniques to persist for 35 – 40 days in young pigs (Yoon, et al, 1993) and up to 16 weeks by PCR in piglets that are viraemic at birth as a result of in-utero infection (Benfield et al, 1996). Persistence of PRRS virus in lymphoid tissues has been demonstrated for up to 135 days in gilts (Batista et al, 2004).

3.4.4 Clinical signs

Clinical signs of PRRS are extremely variable, influenced by strain of the virus, the immune status of the herd, and management factors. An outbreak of PRRS in a naïve herd may involve an acute onset of reproductive failure in the breeding herd with sows aborting or farrowing pre-term, the birth of stillborn and mummified piglets, sow deaths and pre-weaning mortality amongst piglets. Respiratory disease and mortality occurs amongst weaners and fattening pigs. However, in many herds even the epidemic period does not have dramatic consequences. For example, in one study, only 20% of seropositive herds actually experienced obvious clinical signs (Pejsak and Markowskadaniel, 1997).

Therefore, the short term economic consequences of PRRS are extremely variable, ranging from very serious, where 55-80% of farm income may be lost (Brouwer et al, 1994; Polson et al, 1990), to no obvious consequences (Pejsak et al, 1997).

The epidemic period is usually short-lived, and production figures returned to values close to what was considered normal after 4 months in the USA (Polson et al, 1990) and 11 weeks in the UK (Hopper et al, 1992). Low levels of chronic production loss in weaners and fattening pigs may be experienced in herds where the infection is endemic (Stevenson et al, 1993; Brouwer et al, 1994; Dee and Joo, 1994; Pejsak and Markowskadaniel, 1997; Pejsak et al, 1997; Goldberg et al, 2000a; Nodelijk et al, 2000;), and the virus may persist in the herd for a long time in large herds, although it may disappear completely from small herds (about 100

sows) after several years, presumably due to a lack of susceptible animals to maintain it (Nodelijk et al, 2000).

Thus, in epidemic situations PRRS infection is typically characterised by an acute systemic illness resulting in abortions in sows, pre-weaning mortality of piglets and ill thrift in weaned and grower pigs. However, in endemically infected breeding herds, clinical signs are generally limited to immunologically naïve individual animals and reproductive problems are therefore considerably less dramatic.

3.4.5 Transmission between herds

In countries where PRRS virus is endemic, its presence in a previously free herd is usually not recognised quickly enough to allow the origin of infection to be accurately determined, so the precise mechanisms by which the virus is able to spread in any particular area have been the subject of some speculation (Benfield et al, 1999). For example, while Le Potier et al (1997) attributed 56% of new herd infections in one area of France to movement of infected pigs, and 20% to infected semen, he also considered that 21% of introductions were due to "fomites" and 3% to "unidentified other sources".

3.4.5.1 Movement of infected animals

It is widely agreed that the movement of infected pigs, particularly the purchase of weaners and replacement breeding stock, is likely to be the major route of spread between herds in most countries (Benfield et al, 1999). Infection of susceptible pigs probably takes place by nose to nose contact or by breaks in the skin of susceptible animals being contaminated with urine or faeces of infected animals (Hermann et al, 2005).

3.4.5.2 Semen

Semen is agreed to be the second most important route of between-herd transmission of PRRS virus. If infection is introduced into artificial insemination (AI) centres, as occurred in Denmark in 1996 (Mortensen, 2002), there is potential for rapid and widespread dissemination. By contrast, Albina (1997) considered that so long as AI centres do not become infected, then control of PRRS can be achieved by standard biosecurity measures.

3.4.5.3 Fomites

Although Pirtle and Beran (1996) considered that under most conditions PRRS virus is likely to be rapidly inactivated in urine, saliva, faecal slurry and non-porous fomites, a range of fomites (boots, clothing, hands, equipment, vehicles) have been suggested to be involved in "area spread" of PRRS virus (Benfield et al, 1999). Transmission has been demonstrated via contaminated boots and clothing and via hypodermic needles (Otake et al, 2002c; 2002d). However, there is limited information from the field that can be used to estimate the importance of fomites relative to other routes of transmission. While mechanical transmission via fomites can occur even during warm weather (Dee et al, 2002b), it is more likely to happen in winter conditions when the virus might be expected to survive for longer periods in the environment (Dee et al, 2002a).

In North America, most producers employ strict biosecurity protocols to reduce the risk of introduction of infectious diseases into pig herds (Benfield et al, 1999), and the potential

importance of fomites is recognised in specific measures such as 24-48 hour downtime for visitors, shower-in for employees and professional pest control (Otake et al, 2002d; Moore, 1992). Although biosecurity measures can substantially reduce the risk of introduction or re-introduction of PRRS virus into free herds (Zimmerman, 2003) breakdowns are common, and systems are considered essential to prevent transmission by contaminated fomites (Dee et al, 2002a; 2002b).

3.4.5.4 Vectors

The short distance mechanical transmission of PRRS virus by mosquitoes has been demonstrated under experimental conditions (Otake et al, 2002a). Other experiments showed that mallard ducks were susceptible to infection via drinking water and although these birds showed no signs of infection they shed the virus in faeces for up to 3 weeks (Zimmerman et al, 1997). However, these results have been difficult to reproduce (Trincado et al, 2004) and the significance of these observations for spread under natural conditions is not known.

3.4.5.5 Aerosols

Airborne transmission by aerosols was assumed to be responsible for much of the so-called "area spread" up to 3 km that occurred in the early years of PRRS introduction into several countries, especially England (Edwards et al, 1992; Robertson, 1992) and Denmark (Mortensen and Madsen, 1992). Among the circumstantial evidence used in support of claims for airborne spread was the finding of virtually identical viruses (96-100% nucleotide homology of ORF5 gene) on farms up to 20 miles apart (Lager and Mengeling, 2000), and since at that time the existence of subclinical infections was not recognised, airborne spread was assumed to be responsible and the distance between outbreaks was taken to indicate the extent of that airborne spread (Benfield et al, 1999).

As understanding of the epidemiology of PRRS improved, such speculation gradually abated, but in the middle 1990s it was still thought that airborne spread was possible up to about 2 – 3 km (Blaha and Büker, 1995), and field data tended to be interpreted accordingly. For example, in a case-control study of risk factors for spread in 1071 Danish sow herds from June 1996 to October 1997 (Mortensen et al, 2002) the relative risk for a farm to become infected was strongly influenced by distance from known infected farms, a variable that was taken as a proxy for airborne spread. The relative risk of infection for a farm 1km away from an infected farm was 1.5, while for farms 500m and 300m away the relative risk was 4.0 and 45.0 respectively. This led the authors to conclude that that spread of PRRS virus from infected neighbouring herds by aerosols was a frequent mode of transmission.

It was not until the late 1990s that experiments began to be conducted to test the hypothesis of airborne spread. Torremorell et al (1997) could not isolate PRRS virus from air samples from nurseries housing experimentally infected pigs, and also could not demonstrate any airborne spread when using a field strain of PRRS virus. However, they did demonstrate short distance (1 metre) airborne transmission when using a short passage reference strain of PRRS virus (VR-2332). Airborne spread over approximately 1 metre was also shown to occur in several other trials (Brockmeier and Lager, 2002; Kristensen et al, 2004). However, others found no airborne spread between isolation chambers placed only 50 cm apart (Lager and Mengeling, 2000).

Wills et al (1997c) carried out a series of trials on short distance aerosol spread, which they apparently expected would occur quite readily based on the early 1990s reports from the UK and Denmark (Robertson et al, 1992; Edwards et al, 1992; Mortensen and Madsen, 1992). Therefore it came as a surprise that PRRS could not be transmitted across short distances in a single room containing five or more acutely infected pigs (Wills et al, 1997c). In further trials, which involved pens of pigs 45-100 cm apart and separated by a sheet of aluminium, spread between pens occurred in only 2 out of 5 trials. Moreover, since the aluminium sheets were found to be unable to prevent the transfer of feed, faeces and urine between pens, it was not possible to determine whether the spread was due to aerosols or contaminated materials. Thus, Wills et al (1997c) concluded that airborne transmission was less likely to occur than had previously been believed, and they considered that alternative explanations for "area spread" needed to be investigated.

Several trials have been carried out to investigate longer distance airborne spread under simulated field conditions. Dee et al (2003) demonstrated the movement of PRRS aerosol over a distance of 150 m in a rigid PVC 10 cm diameter pipe. However, this was achieved using mechanically generated virus, and the authors noted that the virus titre in the air may have been higher than would be expected under natural conditions. Fano et al (2005) reported that PRRS virus was not transmitted from an infected barn to sentinel pigs in either of two trailers that were exposed to the exhaust air from the barn. One trailer was 1 m from exhaust fans, and another trailer was 6 m from exhaust fans but connected to the exhaust fan by a PVC pipe to maximise the delivery of exhaust air (after Dee et al, 2003). This result was similar those previously reported by others (Otake et al, 2002b; Trincado et al, 2003) who failed to detect PRRS virus in air samples and sentinel pigs up to 30 metres from an infected barn under controlled field conditions. It was concluded (Fano et al, 2005) that airborne transmission of PRRS may be a rare event under field conditions.

Finally, Dee et al (2005) reported further on the trials involving the transport of virus in aerosols over 150 m through PVC pipe. Although virus remained viable over this distance, the log concentration of PRRS virus decreased by 50% in 33 m, and virus survival was probably limited by the warming and drying of the air the further it was moved. The conditions of this experiment (large volumes of air containing high concentrations of virus transported at high speeds and prevented from dispersal) were highly artificial, making extrapolation to the field impossible. Even under the optimal conditions of this experiment, only three out of six pigs became infected, despite being exposed to large quantities of air that had been inoculated with large quantities of PRRS virus. The authors concluded that the transmission of PRRS virus by aerosols is probably a rare event in the field, if it occurs at all, and that investigators of unexplained outbreaks of PRRS should first examine all the known routes of PRRS virus transmission and consider the possibility of a breakdown in farm biosecurity before considering aerosol transmission as the route of virus introduction. Recent work has indicated that the pathogenicity of the PRRS virus isolate may play a role in shedding by aerosols (Cho et al, accepted for publication).

3.4.5.6 Oral transmission

Several experiments have demonstrated that PRRS can be transmitted to naïve pigs by the feeding of meat harbouring PRRS virus (Van der Linden et al, 2003; Magar & Larochelle, 2004).

3.4.6 Control

Control programmes are primarily based on the fact that the major sources of infection for new herds are infected pigs and semen (Dee, 2003; Albina, 2003). For all herds, semen should be sourced from known free sources, and for free herds, this policy also must apply to gilts.

In France, PRRS has been under control in the Pays de la Loire region since 1993. This region has 2,200 pig farms with a total pig population of 750,000 pigs and it borders on Brittany, which has the highest pig density in the country. Implementation of movement control, test and removal, and AI centre freedom has kept the herd prevalence below 2% in this region (Albina et al, 2003).

In the USA, control efforts on infected farms typically aim to produce pigs that are free from PRRS virus at time of weaning (Dee, 2003; FitzSimmons and Daniels, 2003). To achieve this, one approach is to "stabilise" the breeding herd (Dee, 2003). A stable breeding herd is defined as a herd of adult pigs and their offspring where there is no detectable evidence of sow-to-sow or sow-to-pig transmission. Infected farms have been classified as:

- Stable/inactive – infected adult population that is not actively shedding virus and therefore not infecting piglets prior to weaning
- Stable/active – as above, but there is evidence of transmission to piglets in the late nursery or finishing periods, and of clinical disease post-weaning
- Unstable – either chronically infected or recently acutely infected

Management of gilts in stabilised herds is of utmost importance, to ensure that replacement gilts are exposed to local strains of the virus well before they join the breeding herd as replacements (Allison, 2003). Introduction of gilts is often done by a three-stage process, each being on an all-in/all-out basis; isolation, acclimatisation and recovery. Partial depopulation may be appropriate to consider in some herds, particularly where the virus may be circulating in only a specific stage of the population e.g. in nursery or finisher, but is absent from breeding herd. An all-in/all-out pig flow in the weaned animals is often practised to control concurrent bacterial infection in the weaners and growers (Dee, 2003; McCaw, 2003).

Vaccines do not prevent infection with PRRS, but they have been used in an attempt to minimise the impact of the virus. Modified live vaccines (MLVs) and killed vaccines are used in North America and Europe (Gillespie, 2003; Thacker et al, 2003, OIE, 2004b). Although these vaccines, which are based on single field strains, may protect against clinical disease, they do not prevent infection with genetically diverse field strains (Meng, 2000). The duration and titre of viraemia may be reduced by MLVs but inactivated vaccines have no impact on the onset, duration or level of viraemia (Christopher-Hennings et al, 1997; Neilsen et al, 1997). While MLVs are registered for use as a single intra-muscular injection at 3 to18 weeks of age they are sometimes used "off-label" to vaccinate breeding and gestating animals and as an intranasal vaccine prior to weaning (Dee et al, 1996a; Sornsen et al, 1998a; Dewey et al, 1999; USDA, 1997). MLVs are considered to be better than killed vaccines in terms of the protection they provide, and although shedding of the vaccine strain has been observed when naïve pigs are vaccinated, transmission has not been detected following revaccination (Dee, 2003).

Field studies have demonstrated that MLV virus can actively cycle amongst naïve pigs (Sorensen et al, 1998b) and problems associated with their use have arisen. For example, following registration in Denmark in 1996, MLVs were only used in seropositive herds. Many

of these herds had problems with growers and finishers but no clinical signs in the breeding herd. However, reproductive failure soon arose in the breeding herd as a result of the spread of vaccine virus to unvaccinated gilts and sows. In addition a number of herds, not previously infected with field strains and not practising vaccination were found to be infected with the vaccine strain and some of these herds experienced acute PRRS like symptoms (Botner et al, 1997). Abortion storms and high sow mortality in herds using vaccine were observed in the USA in 1996 (Bush et al, 1999) and a field study in Canada found that MLVs administered to sows at different stages of gestation in seronegative herds significantly decreased reproductive performance (Dewey et al, 1999). As a result of these problems the usage of MLVs has apparently been curtailed, at least in the USA, with most practitioners using them sparingly (Wilson, 2000). However, in 2003 there were two MLV vaccines registered for use in the USA (Gillespie, 2003), and protocols for mass vaccination in combination with herd closure and other management tools have been developed.

There are a number of factors that may provide an explanation for the problems experienced with MLVs. They may interact with field strains, through quasispecies evolution and RNA recombination, and lead to the emergence of vaccine related strains that have been shown to produce more pronounced pathological changes than the vaccine virus alone. Certainly, outbreaks of atypical or acute PRRS in the USA in 1996 may provide a warning of the potential for new strains to emerge (Meng, 2000). In addition, reversion to virulence is possible (Meng, 2000). This is more likely to occur if a perpetual cycle of transmission is established following vaccination in which naïve pigs continue to be exposed to MLVs, for example when pigs are periodically weaned into the nursery. Each in-vivo passage of an attenuated strain provides an opportunity for mutation and reversion to virulence (Mengeling et al, 1999). Genomic sequencing of viruses can play an important part in monitoring the success of a vaccination program (Roberts, 2003).

3.4.7 Eradication

Despite persistent infections of pigs with PRRS, eradication of the virus from herds is possible, predominantly using management techniques that are based on a detailed understanding of the epidemiology of the disease in pig-dense areas (Dee and Molitor, 1998; Dee et al, 2000; Dee et al, 2001; Allison, 2003; McCaw, 2003; Dee et al, 2004). In the USA, techniques include whole herd depopulation and repopulation with virus-free replacement stock, segregated early weaning, test and removal, mass vaccination with unidirectional pig flow, and herd closure (Dee, 2003; Torremorell et al, 2003).

Herd level eradication programmes in Denmark have been based on partial and total depopulation combined with disinfection of nurseries and grower-finisher units, serological monitoring of sows to ensure that the population was ‘immunologically stable’, and ensuring that gilts entering the herd were immune to the same type of virus that was present locally (Bötner, 2003).

3.5 HAZARD IDENTIFICATION CONCLUSION

PRRS virus is exotic to New Zealand and is listed on the unwanted organisms register as a notifiable organism.

Considering that:

i) PRRS virus has been isolated from pig meat derived from both experimentally infected and commercially slaughtered animals,

ii) PRRS virus survives in frozen meat for at least 4 weeks,

iii) PRRS virus can initiate infection when fed to naïve pigs,

iv) modified live virus vaccine strains can actively cycle amongst pigs and have been associated with reproductive failure and sow mortality,

PRRS virus, whether it is a field strain or modified live virus strain, is classified as a potential hazard in the commodity.

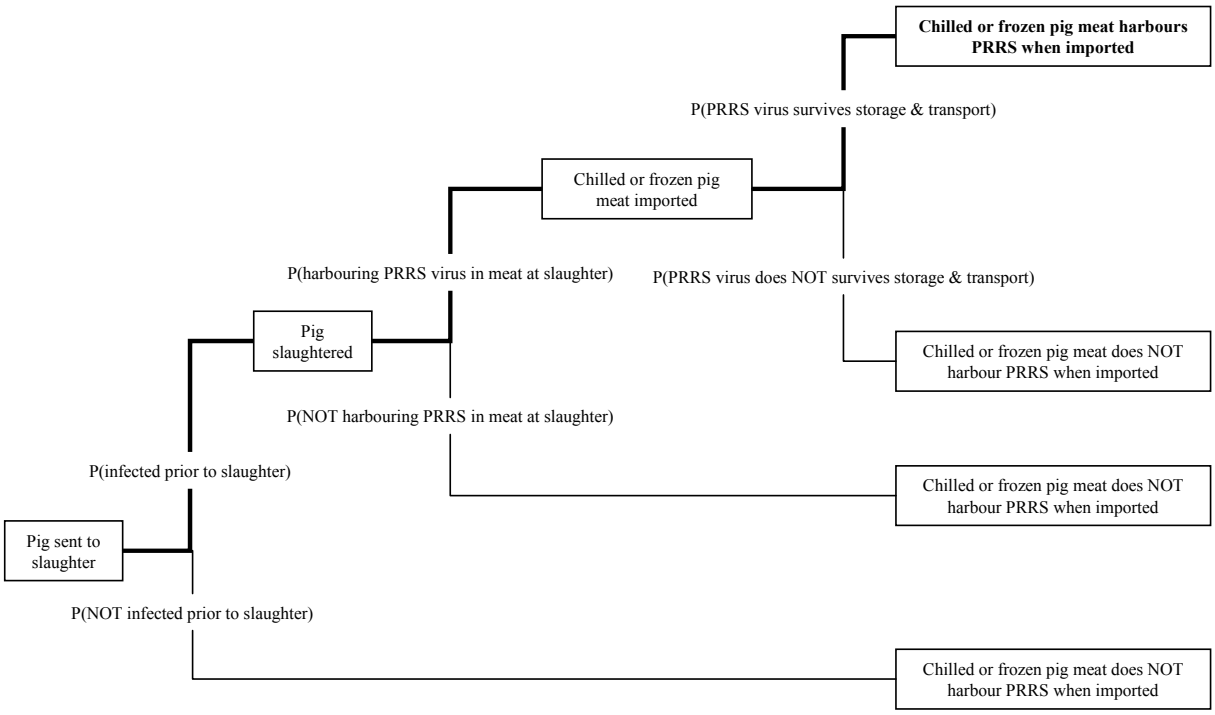
4. RISK ASSESSMENT

4.1 RELEASE ASSESSMENT

The release assessment considers the likelihood of PRRS virus being present in the commodity at the time of importation.

For pig meat imported into New Zealand to harbour PRRS virus the meat would have to be derived from a pig that came from an infected herd, the virus would have to be present in the meat of that pig at the time of slaughter and the virus would have to be able to survive subsequent storage and transportation. The biological pathways involved in the introduction of PRRS virus into New Zealand in this way are shown in Figure 1.

Figure 1. The biological pathways necessary for pig meat to harbour PRRS virus when it is imported into New Zealand.



The likelihood of pig meat harbouring the virus at the time of slaughter could be established most accurately by carrying out random sample surveys of pigs at slaughterhouses, and testing samples for evidence of PRRS virus presence. However, the literature contains very little information from such surveys, and an alternative approach is to model the likelihood based on what is reported in the literature on the prevalence of infection on farms, the likely time of infection, the duration of viraemia, and the likely age of pigs at slaughter. This risk analysis uses both approaches.

4.1.1 The likelihood of a pig being infected prior to slaughter

In the USA approximately 60% of unvaccinated finishing herds were found to be seropositive for PRRS in a study carried out in 1995 (USDA, 1997). Approximately 10% of finishing herds practice vaccination with either modified live or killed vaccines to control PRRS (USDA, 1997; OIE, 2004b). Since neither of these vaccines prevent infection (OIE, 2004b)

and live vaccine virus can be disseminated from vaccinated to naïve pigs (Benfield et al, 1999), it is reasonable to assume that the combined results from the NAHMS study for samples derived from both vaccinated and unvaccinated herds are indicative of the likelihood of a randomly selected pig from the general finishing population being exposed to either a field or vaccine strain of PRRS prior to slaughter. Overall 57.5% of 4,756 pigs from 284 finishing herds were seropositive. In Canada, 85% of slaughter pigs sampled in Quebec in 1993 were antibody positive (Magar et al, 1995), while the average seropositivity rate for 1039 sera collected at random from pigs arriving at two abattoirs (one in Quebec, one in Manitoba) was 74%, indicating that Quebec had slightly higher seropositivity rates than Manitoba (Magar and Larochelle, 2004). The seropositivity rate for market pigs in Taiwan has been reported as 85% (Wang, 1999).

In Europe it is believed that more than 50% of farms are infected, although there are regional differences. For example, some areas in France and Great Britain, where there are low densities of pigs, have a much lower prevalence (Albina, 1997). Since the seroprevalence within affected farms can reach 85-95% within 2-3 months after the introduction of PRRS followed by prolonged persistence of infection (Albina, 1997), the likelihood of a pig having been infected with PRRS prior to slaughter in most of Europe can be expected to be similar to that observed in the USA.

Considering that:

i) approximately 50-60% of farms are likely to be infected with PRRS in endemic regions,

ii) the seroprevalence in infected herds may be as high as 85-95%, and

iii) the seroprevalence amongst randomly selected finishing pigs has been found to be as high as 75%,

it is considered that there is a moderate to high likelihood of a pig being infected with PRRS virus prior to slaughter.

4.1.2 The likelihood of PRRS virus being present in pig meat at the time of slaughter

The likelihood of infectious PRRS virus being present in pig meat at the time of slaughter is dependent on the age at which a pig becomes infected, the presence and duration of viral infection/contamination in pig meat and the age at which it is slaughtered.

4.1.2.1 The age at which a pig is likely to become infected

The transmission dynamics within a herd can be complex and are influenced by herd size and opportunities for contact between subpopulations of susceptible, infectious and immune animals within and between the breeding, nursery and fattening units.

Attempts have been made to disrupt the transmission of PRRS virus amongst weaners, growers and fatteners by stabilising immunity in the breeding herd, either through vaccination or natural exposure prior to mating or by serological profiling. The aim was to prevent transplacental infection so that the nursery could be restocked with uninfected pigs following depopulating and disinfection (Dee and Joo, 1996; Dee et al, 1996a; Dee et al, 1996b; Dee et al, 1996c; Dee, 1997; Dee and Joo, 1997; Dee et al, 1997a; Dee et al, 1997b; Sornsen et al,

1998a; Dee and Phillips, 1999; Rajic et al, 2001). While results in small herds (250-700 sows) have been promising (Dee and Joo, 1997; Dee and Phillips, 1999; Rajic et al, 2001) mixed results have been obtained in larger herds (>1000 sows) where clinical disease but not PRRS virus has been eliminated. It is likely that sub-populations of susceptible and infectious breeding animals are still present in these larger herds enabling the virus to cycle continuously. The existence of these sub-populations is likely to be the result of, amongst other things, a rapid turnover of breeding animals, commingling from several sow herds and less stringent biosecurity measures. (Dee and Joo, 1996; Dee et al, 1996b; Dee et al, 1997b; Rajic et al, 2001). These results confirm other observations that once a large herd is infected PRRS virus tends to circulate indefinitely (Benfield et al, 1999).

Pigs may become infected either in utero as a result of transplacental infection or during the period from birth to slaughter.

Both live vaccine and field strains of PRRS virus can lead to transplacental infection. Its occurrence is influenced by the stage of gestation at which sows or gilts are exposed and their immune status. Naïve sows or gilts exposed to PRRS virus during mid to late gestation are likely to produce piglets that are viraemic at birth. This is a result of transplacental infection, which occurs at a higher incidence during late gestation (100% at day 90) (Lager, 1997a) than early to mid gestation (25% at day 30) (Lager, 1997b), (0% at day 45-50) (Mengeling et al, 1996a). Only 4% of near term piglets from gilts infected at 30 days gestation were viraemic while 35% (Lager, 1997a), 68% (Lager, 1997b) and 80% (Benfield et al, 1996) of near term piglets and 27% (Mengeling et al, 1995), 55% (Mengeling et al, 1998) and 76% (Mengeling et al, 1994) of live born piglets from gilts infected at about 90 days were viraemic. These animals are likely to be infectious for a prolonged period as viraemia has been shown using cell culture techniques to persist for at least 8 but not 9 weeks (Mengeling et al, 1995) and up to 16 weeks post farrowing using PCR (Benfield et al, 1996). In the latter study, sentinel pigs became infected at 64, 84 and 98 days post farrowing.

Sows or gilts previously infected with PRRS virus may develop a protective immunity that persists for the productive life of an animal. This immunity has been shown to prevent infection in sows challenged during the last trimester (day 90) with an homologous strain of PRRS virus 8-20 months following primary exposure, as there was no evidence of transplacental infection (Lager, 1997a). Although immunity develops it may not afford cross protection against challenge with heterologous strains. This is certainly the case with vaccines based on a single strain as they are not effective in protecting against infection with genetically diverse field strains (Meng, 2000).

Piglets infected in utero with either a vaccine or field strain of PRRS virus provide a source of infection for littermates and other pigs in the nursery and grower/fattening units. Uninfected littermates may become rapidly infected. For example, 80% of the uninfected littermates were infected within the first week of birth (Mengeling, 1998 et al).

Maternal immunity may provide short-term, partial protection against infection in neonatal pigs (Benfield et al, 1999). It is reported to reach its lowest levels when piglets are around 6 to 8 weeks of age (Wen-Bin Chung et al, 1997). The protection afforded by maternal immunity is likely to depend on several factors including the strain(s) of PRRS virus the sow or gilt has been previously exposed to, the ingestion of sufficient colostrum, the rate of decay of maternal antibodies and the strain(s) of PRRS virus to which piglets are exposed.

A number of studies have been undertaken in commercial piggeries in an attempt to estimate the most likely time of exposure to PRRS virus. A field study (Wen-Bin Chung et al, 1997) was undertaken in pigs from seven farrow-to-finish continuous-flow pig herds (2000 to 2500 sows per herd), which had endemic PRRS. About 70% of the sows in these herds were reported to be seropositive. The pigs were monitored fortnightly from 2 to 22 weeks of age. They were weaned at 4 weeks of age, moved to growing houses at 8 to 9 weeks of age and then to finishing houses at 18-19 weeks of age. PRRS virus was isolated from 1 to 16 week old pigs with the highest isolation rates observed (70 to 100%) when pigs were 6 to 9 weeks of age which coincided with the lowest levels of maternally acquired immunity at around 6 to 8 weeks of age. Virus was not isolated from pigs at 22 weeks of age.

These results indicate that virus was cycling in the breeding herd leading to transplacental infection in some animals and the birth of viraemic piglets which provided a source of virus for littermates and other pigs in the nursery and growing sheds. Figure 2a, which depicts the results from each farm, indicates that most animals are viraemic between 6 to 9 weeks of age. Although these results are based on random samples from only 10 pigs each fortnight, it is assumed that the percentage of viraemic pigs on each sampling occasion is indicative of the prevalence of infection in each age class of pigs for each farm.

Figure 2a. The combined results of the number of viraemic animals per age class from 7 farms with endemic PRRS (Wen-Bin Chung et al, 1997).

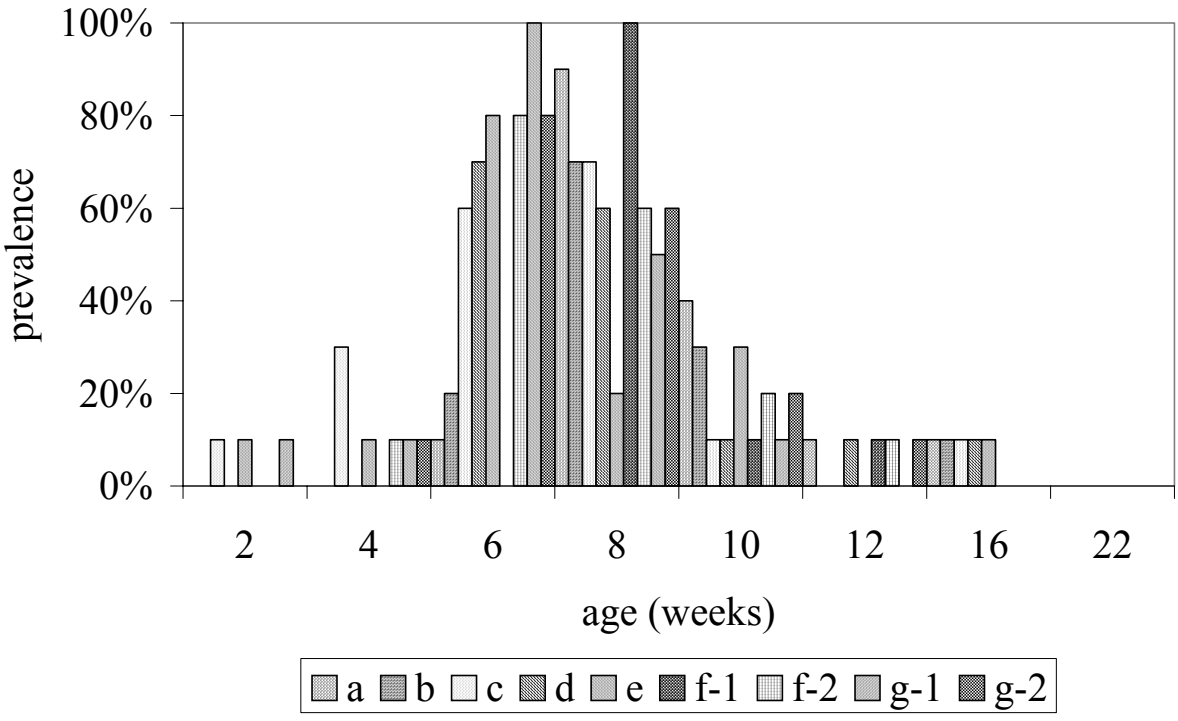
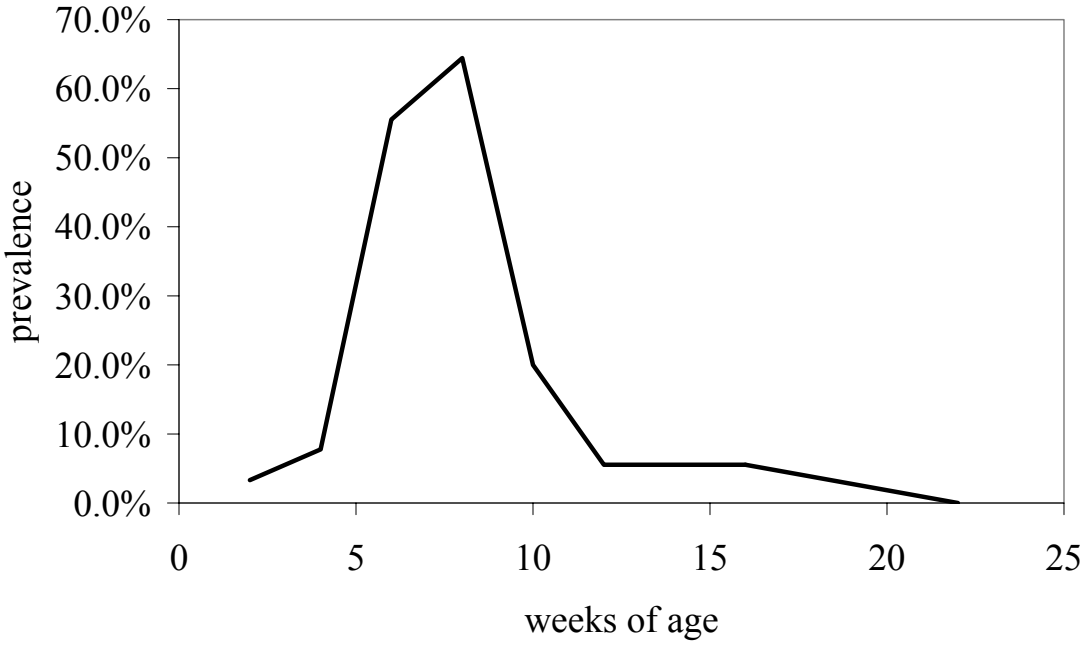


Figure 2b. a summary distribution of the prevalence of viraemia by age (Wen-Bin Chung et al, 1997).



Since there appears to be a similar pattern of infection on each farm, the estimates of prevalence per fortnight per farm is averaged across all farms to obtain a summary estimate of the prevalence of viraemic pigs per fortnight (Table 6 and Figure 2b).

Table 6: Prevalence estimates of viraemia for pigs from 2 to 22 weeks of age from a field study on 7 farrow to finish continuous flow pig herds (2000 to 2500 sows per herd) with endemic PRRS. The results for farms "f" and "g" are for two consecutive years (Wen-Bin Chung et al, 1997).

Age (weeks)	a	b	C	d	e	f-1	f-2	g-1	g-2	Average prevalence
2	0	0	0.1	0	0.1	0	0	0.1	0	3.3%
4	0	0	0.3	0	0.1	0	0.1	0.1	0.1	7.8%
6	0.1	0.2	0.6	0.7	0.8	0	0.8	1	0.8	55.6%
8	0.9	0.7	0.7	0.6	0.2	1	0.6	0.5	0.6	64.4%
10	0.4	0.3	0.1	0.1	0.3	0.1	0.2	0.1	0.2	20.0%
12	0.1	0	0	0.1	0	0.1	0.1	0	0.1	5.6%
16	0.1	0.1	0.1	0.1	0.1	0	0	0	0	5.6%
22	0	0	0	0	0	0	0	0	0	0.0%

Other field studies support these observations. If virus is actively cycling in the breeding herd some piglets are likely to be infected before weaning as a result of transplacental infection and spread it among uninfected littermates (Dee 1996; Dee and Joo, 1997; Dee, 1997; Dee and Molitor, 1998; Dee et al, 1998; Dee and Phillips, 1999, Rajic et al, 2001;). Serological profiling indicates that most animals are likely to be infected by 6 to 8 weeks of age, for example, a seropositive rate of 90-100% by 9-10 weeks of age (Sornsen et al, 1998b), 80-100% by 8-9 weeks of age (Dee et al, 1994), 40-100% by 8-10 weeks of age (Dee and Joo, 1997b) and a high seroprevalence (percentage not provided) amongst 8 week old pigs (Dee and Joo, 1996).

4.1.2.2 The presence and duration of PRRS viral infection/contamination in pig tissues

Following infection, the primary site of replication of PRRS virus is in alveolar and other regional macrophages with distribution via peripheral blood mononuclear cells to regional lymph nodes and other tissues (Halbur et al, 1995; Rossow et al, 1995; Benfield, 1996; Rossow et al, 1996; Christopher-Hennings et al, 1998) followed by viraemia within 12 hours (Rossow et al, 1995; Rossow et al, 1996). Viral replication in pulmonary macrophages may continue for up to 7-8 weeks post infection (Shibata et al, 1997). Because macrophages are found in all tissues, there is apparently no limitation to the types of tissue that can harbour PRRS virus, although the productivity of virus replication does vary between tissues (Benfield et al, 1999). Pulmonary macrophages are the most productive location for virus replication (Rossow et al, 1996) but several populations of the macrophage/monocyte lineage do not permit complete replication, for example blood monocytes and progenitor cells in the bone marrow (Duan et al, 1997). As immunity develops the virus localises in tissues such as oropharynx and tonsils where it has been found to persist for prolonged periods of between 15 and 22½ weeks post infection (Wills et al, 1997a; Allende et al, 2000; Horter et al, 2000).

i) *Oropharyngeal and tonsil tissues*

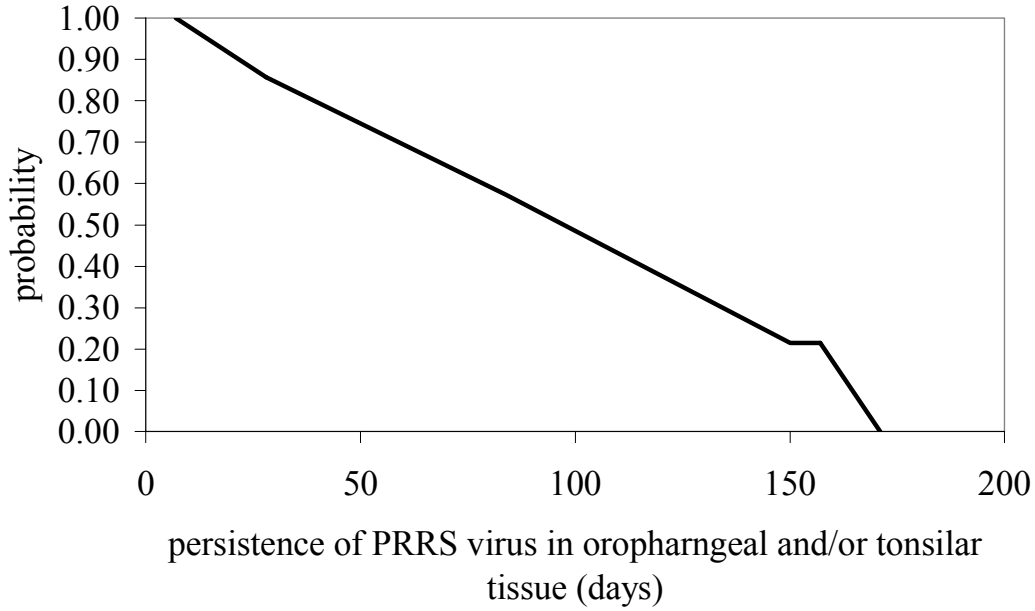
The results of two studies (Wills et al, 1997a, Allende et al, 2000) to investigate the persistence of infection are presented in Table 7 and Figure 3 respectively. In summarising the results in this way it is assumed that the pigs used in these experiments represent a random sample from the general pig population, survival times are independent, the probability of survival is constant throughout an interval and censoring is random with censored pigs being at risk for half the interval. While not all pigs are similarly affected, it is apparent that a significant proportion harbour infectious virus in oropharyngeal tissues for many weeks.

Table 7: Data for an actuarial (life table) adapted from experimental results from two trials investigating persistence of infection with PRRS virus. Virus was isolated from oropharyngeal samples in cell culture (Wills et al, 1997a) or in tonsil tissue using PCR and a tonsil, lung, bronchial lymph node pool by bioassay (Allende et al, 2000).

Days post infection (dpi)	Pigs harbouring virus		Pigs harbouring virus at the beginning of the interval	No longer harbour virus during the interval	Withdrawals	Survival (cumulative probability)
	4 x 4-week old pigs Wills et al, 1997a	10 x 4-8 week old pigs Allende et al, 2000				
0-7	4	10	14	0	0	1.00
7-28	4	8	14	2	0	0.86
28-84	3	5	12	4	0	0.57
84-150	1	2	8	5	0	0.21
150-157	1	-	3	0	2	0.21
157-171	0	-	1	1	0	0.00

In contrast, Batista et al (2002) found no evidence of PRRS virus in tonsil and lymph node from 120 pigs that were experimentally infected at 4 months of age and slaughtered in groups of 40 at 8 months, 9 months and 10 months. Combining the results from the 120 pigs used in this trial gives a Beta(0+1, 120-0+1) distribution with an expected value of 0.008 which is of the same order of magnitude as Frey's results for pig meat as shown later in Figure 5 (Frey, 1995a). This notwithstanding, in a subsequent study Batista et al (2004) confirmed persistence of PRRS virus by PCR in a range of lymphoid tissues between day 30 post-infection (the end of viraemia) and day 135.

Figure 3. An actuarial (empirical survival) plot of persistence of PRRS virus in tonsil or oropharyngeal tissue.



ii) Muscle, bone, and regional lymph nodes

PRRS virus has been isolated from muscle (Bloemraad et al, 1994; Mengeling et al, 1995; Done et al, 1996; Van der Linden et al, 2003;), bone marrow (Mengeling, 1995; Done et al, 1996) and regional lymph nodes associated with the musculature (Bloemraad et al, 1994; Mengeling, 1995; Magar et al 1995; Christopher-Hennings et al, 1998) in experimentally infected pigs sampled immediately after euthanasia. The virus has been isolated from:

- muscle in 1 of 3 pigs slaughtered 7 days post infection but not from any of the remaining 15 pigs slaughtered at weekly intervals from 14 days post infection (Rossow et al, 1995),
- muscle in 12 of 24 pigs slaughtered 11 days post infection (Van der Linden et al, 2003),
- bone marrow in 1 of 18 pigs 7 days post infection (Mengeling et al, 1995),
- iliac lymph nodes at the end of a trial in 4 of 7 pigs 21 days post infection (Christopher-Hennings et al, 1998).

In addition, PCR studies have demonstrated the presence of PRRS viral antigen at the end of a trial in bone marrow in 4 of 7 pigs up to 21 days post infection (Christopher-Hennings et al, 1998).

It appears that the PRRS virus in muscle and bone most likely originates from blood plasma in capillary vessels as a result of viraemia. In addition immuno-histochemistry (IHC) staining of muscle samples indicates that the cells associated with the virus may originate from blood monocytes (Magar et al, 1995). Since the presence of PRRS virus in muscle is likely to be

associated with an existing viraemia, it is important to note that the duration of viraemia is prolonged, despite the appearance of antibodies early in the course of infection (Done et al, 1996; Wills et al, 1997a). Antibodies have been detected as early as 6 days post infection, usually within 14 days, peaking at 5-6 weeks and persisting for variable periods of time with some pigs becoming seronegative after 4-6 months (Done et al, 1996).

iii) Duration of viraemia in experimentally challenged naïve animals

Using various cell culture techniques to isolate virus, viraemia has been demonstrated to persist for at least 35 but not 42 days in one of 16 pregnant gilts (Mengeling et al, 1996a), and for at least 35 but not 40 days in two of four piglets infected at 3 weeks of age (Yoon et al, 1993). Viraemia in piglets infected in-utero has been found to persist for up to 8 weeks after birth in one of three affected animals (Mengeling et al, 1995). PCR techniques indicate that viraemia persists for at least 31 but not 35 days in adult boars (Christopher-Hennings et al, 1995a) and 16 weeks in piglets that are viraemic at birth (Benfield et al, 1996).

The results from a number of experimental studies where the duration of viraemia has been determined by cell culture for various age classes of naïve pigs are presented in Tables 8 to 12. These results are divided into three groups.

The first group (Table 8) is for pigs between the ages of 1 to 14 weeks as it has been reported that pigs between 1 to 10 weeks of age exhibit a similar response to infection based on virus isolation rates, clinical signs, white blood cell counts, microscopic changes and seroconversion (Rossow et al, 1994).

The second group (Table 9) consists of adult pigs and includes boars, sows and pregnant gilts.

The third group consists of three studies where the duration of viraemia associated with vaccine virus was investigated (Table 9).

The combined results for each of these groups are presented in Tables 10 to 12, which are set out as life tables to enable the survival of PRRS virus to be calculated and provide an estimate of the duration of viraemia. It is assumed that the pigs used in these experiments represent a random sample from the general pig population, survival times are independent, the probability of survival is constant throughout an interval and censoring is random with censored pigs being at risk for half the interval. Figure 4 compares the results for these 3 groups in an empirical survival plot.

Table 8. Experimental results on the duration of viraemia, as determined by cell culture, in young pigs (1-14 weeks of age).

days post infection	Hirose et al, 1995 5-13 day old pigs	Wesley et al, 1998 2 week old pigs	Park et al, 1995 3 week old pigs	Wills et al, 1997b 3 week old pigs	Yoon et al, 1993 3 week old pigs	Shibata et al, 1997 4 week old pigs	Wills et al, 1997a 4 week old pigs	Allende et al, 2000 1-2 month old pigs	Mengeling at al, 1995 6 week old pigs	Mengeling et al, 1995 6 week old pigs	Shibata et al, 1998 6 week old pigs	Rossow et al, 1994 1-10 week old pigs	Mengeling et al, 1996b 14 week old pigs
Period: 2-7 days													
Viraemic at the beginning	8	18	4	6	4	6	4	10	8	16	6	15	3
No longer viraemic	0	0	0	0	0	0	0	0	0	0	0	0	0
Withdrawals	0	0	0	0	0	0	0	0	0	0	0	0	0
Period: 8-14 days													
Viraemic at the beginning	8	18	4	6	4	6	4	10	8	16	6	15	3
no longer viraemic	0	0	0	0	0	0	1	0	0	5	0	8	1
Withdrawals	0	0	0	0	0	0	0	10	2	0	0	0	0
period: 15-21 days													
viraemic at the beginning	8	18	4	6	4	6	3		6	11	6	7	2
no longer viraemic	0	2	0	5	0	3	1		1	8	0	2	1
Withdrawals	0	0	0	0	0	0	0		2	0	0	0	0
period: 22-28 days													
viraemic at the beginning		16	4	1	4	3	2		3	3	6	2	1
no longer viraemic	0	1	4	1	0	3	2		1	2	3	0	0
Withdrawals	0	0	0	0	0	0	0		1	0	0	0	0
period: 29-35 days													
viraemic at the beginning	8	15			4				1	1	3	2	1
no longer viraemic	0	0			2				1	1	3	0	1
Withdrawals	8	15			0				0	0	0	2	0
period: 36-42 days													
viraemic at the beginning					2								
no longer viraemic					2								
Withdrawals					0								

Table 9. Experimental results of the duration of viraemia, as determined by cell culture, in adult pigs.

days post infection	Mengeling et al, 1994 pregnant gilts	Mengeling et al, 1996a pregnant gilts	Lager et al, 1997b pregnant gilts	Park et al, 1995 sows	Christianson et al, 1993 pregnant sows	Prieto et al, 1996 10 month old boars	Swenson et al, 1994 1-1.5 year old boars	Christopher-Hennings et al, 1995a boars	Neilsen et al, 1997 1-1.5 year old boars	Teuffert et al, 1998 boars	**Vaccine virus**		
											Molitor et al, 1995 boars	Neilsen et al, 1997 1-1.5 year old boars	Christopher-Hennings et al, 1997 boars
period: 2-7 days													
viraemic at the beginning	8	16	8	6	6	9	4	4	10	2	3	5	4
no longer viraemic	0	0	0	0	0	0	0	1	0	0	1	0	0
withdrawals	0	0	0	0	0	0	0	0	0	0	0	0	0
period: 8-14 days													
viraemic at the beginning	8	16	8	6	6	9	4	3	10	2	2	5	4
no longer viraemic	6	8	0	5	4	0	0	2	3	0	1	4	2
withdrawals	0	0	0	0	2	0	0	0	0	0	0	0	0
period: 15-21 days						7							
viraemic at the beginning	2	8	8	1		9	1	1	7	2	1	1	2
no longer viraemic	2	5	0	1		2	1	0	5	0	0	0	1
withdrawals	0	0	0	0		0	0	1	0	0	1	0	0
period: 22-28 days													
viraemic at the beginning		5	8			7			2	2		1	1
no longer viraemic		4	8			7			2	0		0	0
withdrawals		0	0			0			0	0		1	0
period: 29-35 days													
viraemic at the beginning		1								2			1
no longer viraemic		0								1			1
withdrawals		0								0			0
period: 36-42 days													
viraemic at the beginning		1								1			
no longer viraemic		1								0			
withdrawals		0								0			

Table 10. Data for an actuarial (life table) of the duration of viraemia in young pigs (1-14 weeks of age) from the combined experimental results presented in Table 8.

days post infection	pigs harbouring virus at the beginning of the interval	no longer harbouring virus during the interval	Withdrawals	survival (cumulative probability)
2-7	108	0	0	1
8-14	108	15	12	0.85
15-21	81	23	2	0.61
22-28	45	17	1	0.38
29-35	35	8	25	0.24
36-40	2	2	0	0.00

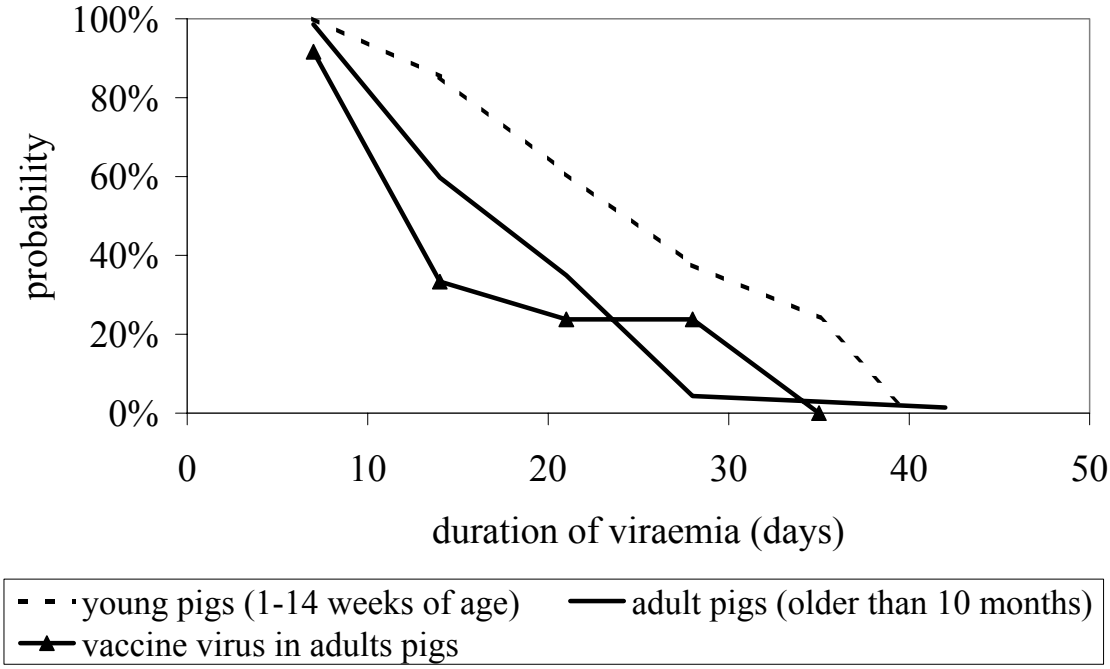
Table 11. Data for an actuarial (life table) of the duration of viraemia in adult pigs from the combined experimental results presented in Table 9.

days post infection	pigs harbouring virus at the beginning of the interval	no longer harbouring virus during the interval	Withdrawals	Survival (cumulative probability)
2-7	73	1	0	0.99
8-14	72	28	2	0.60
15-21	39	16	1	0.35
22-28	24	21	0	0.04
29-35	3	1	0	0.03
36-40	2	1	0	0.01

Table 12: Data for an actuarial (life table) of the duration of viraemia in adult pigs challenged with a vaccine virus from the combined experimental results presented in Table 9.

days post infection	pigs harbouring virus at the beginning of the interval	no longer harbouring virus during the interval	Withdrawals	Survival (cumulative probability)
2-7	12	1	0	0.92
8-14	11	7	0	0.33
15-21	4	1	1	0.24
22-28	2	0	1	0.24
29-35	1	1	0	0.00
36-40				

Figure 4: Actuarial (empirical survival) plot of the duration of viraemia of PRRS virus.



It appears that a greater proportion of younger pigs are viraemic at any given point in time compared to older animals and that the maximum duration of viraemia is very similar in both groups. It also appears likely that the survival curves associated with either the field or vaccine strains in adult animals are similar.

It is worth noting that these results are for naïve animals that were experimentally inoculated with PRRS virus. There are a number of factors that may have an important influence on the duration of viraemia in pigs that are infected with PRRS virus in the field. These factors include vaccination, natural exposure and antibody dependent enhancement.

iv) Duration of viraemia in experimentally challenged vaccinated animals

Animals previously vaccinated with a MLV vaccine have been shown to be more resistant to infection with PRRS virus. For example, only one animal in each of two trials of five boars, became infected when challenged with a field strain. Each infected boar had a lower level and shorter duration of viraemia compared to the control animals (Christopher-Hennings et al, 1997; Neilsen et al, 1997). In another trial involving vaccinated boars PRRS virus was detected by PCR but not by cell culture up to 15 days post infection (Molitor and Shin, 1995), indicating that the titres of virus were very low.

v) Duration of viraemia in pigs naturally exposed to field strains of PRRS virus

The duration of viraemia may also be less in pigs that are naturally exposed to PRRS virus compared to experimentally infected pigs. Naïve sentinels placed in contact with four experimentally infected pigs, that were all viraemic at 3, 10 and 24 days post infection, had a shorter viraemia, particularly those sentinels placed in contact with the infected pigs later in the course of experiment (Table 13) (Yoon et al, 1993). This could indicate that they were exposed to less virus. However, since the number of animals involved was small there is considerable uncertainty.

Table 13. Duration of viraemia for sentinel pigs placed in contact at various times following the experimental inoculation of three-week old piglets (Yoon, 1993).

Pigs	Number of pigs	Exposure to PRRS virus	Number of pigs with viraemia	Average duration of viraemia
Experimentally infected pigs	4	intra-nasal inoculation	4	28 days
Sentinel group 1	4	3 days post infection	4	19 days
Sentinel group 2	4	10 days post infection	4	16.7 days
Sentinel group 2	4	24 days post infection	2	3.5 days

vi) Duration of viraemia in pigs with low antibody titres

PRRS virus is susceptible to antibody dependent enhancement (ADE). Both the rates of infection of, and replication in, porcine alveolar macrophages are significantly enhanced by sub-neutralising levels of PRRS antibody. These low titres of antibody also lead to a greater mean titre and duration of viraemia in infected pigs. As a result ADE has the potential to enhance the severity of disease and susceptibility to infection in pigs with declining titres of antibody of maternal origin or induced by exposure to field or vaccine strains of PRRS virus. (Yoon et al, 1996; Yoon et al , 1997). Field isolates of PRRS virus have been observed to vary widely with respect to their susceptibility to ADE (Yoon et al, 1997).

In a study involving 17-day old pigs with naturally acquired maternal antibody (Shibata et al, 1998) it was concluded that the duration of viraemia in the presence of low titres of maternal antibody was prolonged. Viraemia lasted for at least 28 but not 35 days post infection in seronegative control pigs compared to at least 49 but not 56 days post infection in pigs with maternal antibody. While all six control pigs were viraemic by day 7 post infection only two of the 10 pigs with maternal immunity were viraemic. The remaining pigs in this group developed viraemia sporadically over the next 6 weeks. Their antibody titres initially decreased to undetectable levels by day 14 post infection, when they were 31 days of age, and then increased at varying times. The authors' conclusion that viraemia is prolonged is based on an assumption that all the pigs became infected on the day of intra-nasal inoculation with PRRS virus. However, given the sporadic pattern of viraemia in these animals it may be likely that only a few of these pigs became infected initially and that infection subsequently spread horizontally to others in the group. If this occurred then a conclusion about prolonged viraemia is not tenable. Alternatively, if infection were established in each pig the circulating antibodies may have interfered with attempts to isolate the virus. However, the pattern of rising antibody titres in response to an active infection in these pigs indicates that not all pigs were initially infected. As a result there is considerable uncertainty with the conclusion that viraemia is prolonged in the presence of low levels of maternal immunity.

vii) Presence of PRRS virus in pig meat at slaughter

A number of trials have been undertaken to attempt to isolate PRRS virus from carcasses of pigs slaughtered either in commercial abattoirs or in conditions that mimicked commercial abattoirs. However, a number of limitations are apparent in each of these trials ranging from a small number of animals to uncertainty about the disease status of the pigs from which samples were derived:

a) PRRS virus was isolated sporadically at 24 but not 48 hours post-slaughter from muscle of four 6-month old artificially infected pigs, two of which were killed on day 5 post infection and two on day 10. Their carcasses were stored hanging in a cold room at 4^{o}C, simulating the common procedure to cool and harden the meat in a slaughterhouse. Virus was not recovered from bone of any of these animals (Bloemraad et al 1994).

b) Muscle and lymph node samples were collected from 44 4-8 month old pigs from 18 seropositive farms in Canada (approximately two pigs per farm). Some of the pigs were seropositive, and some were not. All the muscle samples collected 20 to 24 hours following slaughter were negative by both virus isolation and immuno-histochemistry. Virus was isolated from one lymph node and evidence of virus was detected by immuno-histochemistry in three lymph nodes (Magar et al, 1995). The small number of pigs from each farm and the uncertainty regarding their disease status make these results difficult to interpret.

c) Meat samples were collected from packages of frozen pig meat ready for export from four Canadian processing plants in an area where PRRS is endemic. A total of 2,190 individual carcass samples were pooled in groups of five prior to testing[1]. All samples were negative by virus isolation, and one sample was also tested by RT-PCR, also with negative results (Larochelle and Magar, 1997). As neither the age of pigs slaughtered nor the PRRS status of the herds of origin was specified, the likelihood that the animals came from infected farms or whether they were infected is unknown. In addition, the effect of pooling on tests applied is unknown. Nevertheless, if it is assumed that each package of meat represents an independent pig, that if PRRS virus were present it would be distributed uniformly within a pool, and test sensitivity of 100%, then these results indicate that PRRS virus is unlikely to be found in pig meat from an endemic area. Under such assumptions these results for the probability of pig meat harbouring PRRS virus may be specified as Beta(0+1,2190+1). The expected value for this distribution is 0.00046, meaning that less than five pigs per 10,000 would be expected to harbour the virus based on these results.

d) Meat samples were collected from fresh pork derived from commercially slaughtered pigs in the USA and were tested by virus isolation (Frey, 1995a). Six sample pools were positive 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 levels were so low that confirmation by re-isolation was not always successful and had to be done by PCR. These results demonstrate that PRRS may be present in meat of market weight pigs slaughtered in commercial abattoirs. Assuming that each sample pool represents an independent pig, that if the virus were present it would be distributed uniformly within a pool, and a test sensitivity of 100%, these results can be used in a Beta(6+1,1049-6+1) distribution for the probability of a randomly chosen pig harbouring infectious PRRS virus in its muscle tissue at slaughter. The expected value of this distribution is 0.0067, meaning that less than seven pigs per 1,000 would be expected to harbour the virus based on these results.

e) PRRS virus could not be isolated at the time of slaughter from any of 403 out of 472 pigs that were seropositive to PRRS and were therefore known to have been previously infected with PRRS virus (Wang, 1999). These results can be used in a Beta(0+1,403-

[1] 73 lots tested where each lot comprised six pools each made up from five samples, where each sample was obtained from a different package of meat, that is 73 x 6 x 5 = 2,190

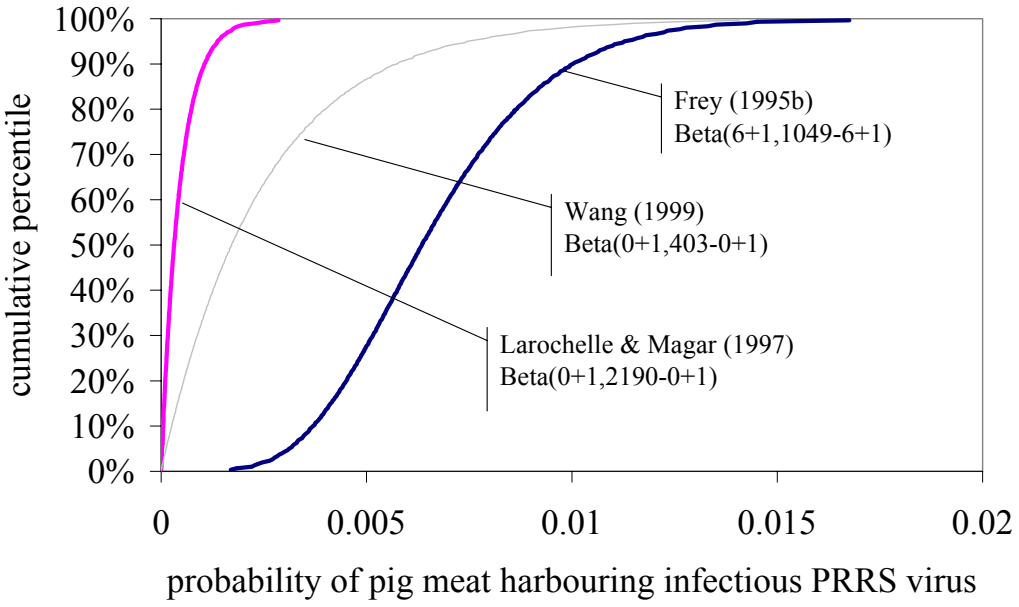
0+1) distribution, which has an expected value of 0.0025. These results suggest that less than 3 pigs per thousand may be expected to harbour the virus at slaughter.

The distributions fitted to the likelihood of virus being present in meat of slaughter age pigs for the results reported by Larochelle and Magar (1997), Frey (1995a) and Wang (1999) are shown in Figure 5. The expected values for the three distributions are approximately of the same order of magnitude.

However, even if virus cannot be isolated by tissue culture on samples from infected pigs it does not necessarily indicate that the samples are free from infectious virus, since there are limits to the sensitivity of tissue culture. A feeding trial is the ultimate test of whether meat of slaughter age pigs contains enough virus to infect other pigs when eaten. There are only two reports of such feeding trials.

Van der Linden et al (2003) took meat samples at slaughter from 24 pigs that had been artificially infected with PRRS virus 11 days earlier. After freezing for 10 days at -23°C, the meat samples were tested for the presence of PRRS virus, by virus isolation and PCR. Although only two out of 24 samples were virus positive, all but one was PCR positive, suggesting that the titre of virus was below the limits of detection by virus isolation, which the authors state is $10^{1.8}$ $TCID_{50}$ per g of meat.

Figure 5. the probability of pig meat harbouring infectious PRRS virus assuming the sensitivity of both virus isolation by cell culture and detection of viral antigen by PCR is 100%. Data from Larochelle and Magar (1997), Wang (1999) and Frey (1995b).



Further, Van der Linden et al (2003) reported that after freezing at – 23°C for 14 days, portions of meat were thawed and fed to 48 recipient pigs (2 recipients for each of the 24 samples) in quantities of 250g per recipient pig over 2 days. Half of the recipient pigs were viraemic 3 days and all 48 were viraemic 6 days after feeding, confirming that even though

the concentration of virus in the meat was below the limits of detection by virus isolation, there was enough virus present to infect pigs by the oral route[1].

In a study designed to test the conclusions of Van der Linden et al (2003), Magar and Larochelle (2004) took samples of meat at random from 1,027 pigs arriving at two Canadian abattoirs. Of 1,027 meat samples, 19 (1.85%) were PCR positive to PRRS virus, and although the virus could be isolated from only one of the 19, when meat from 11 of the PCR-positive carcasses was fed to pairs of recipient pigs, seven of the 11 pairs (63%) became infected. From this study it can be concluded that approximately 1.2%[2] of pigs at slaughter can be expected to have infectious virus in meat, at least under North American conditions, despite the titre of virus being below the threshold of detection by virus isolation.

4.1.2.3 The age at which a pig is slaughtered

The age at which pigs are slaughtered in the USA (USDA, 1997) is presented in Table 14. A similar distribution is assumed for other countries.

Table 14: The age at which pigs are slaughtered in the USA (USDA, 1997)

age at slaughter (days)	percent of pigs
< 160	12.40%
160 - 165	8.90%
166 - 180	37.00%
181 - 209	37.10%
>210	4.60%

4.1.2.4 Modeling the likelihood of pig meat harboring infectious PRRS virus at slaughter

When conducting a preliminary release assessment for this commodity in 2001, the relative lack of data on surveys of pigs at slaughter was noted, and it was considered appropriate to develop a computer simulation model in order to estimate the likelihood of pig meat harbouring infectious PRRS virus at the time of slaughter. The model is presented in Appendix 1.

In summary, the model inputs were as follows:

P1 : probability of a pig being infected prior to slaughter
P2 : probability of infectious PRRS virus being present at the time of slaughter
P2a : probability of infectious PRRS virus being present in oropharyngeal and tonsil tissue at the time of slaughter
P2b : probability of infectious PRRS virus being present in meat (muscle, bone and regional lymph nodes) at the time of slaughter
V1 : age at slaughter
V2 : age when infected
V3 : duration of persistence in oropharyngeal and tonsil tissue
V4 : duration of viraemia

[1] However it is possible that the recipient pigs that became viraemic at 6 days post feeding were infected horizontally from littermates rather than by consuming infected meat.
[2] 7/11 of 19/1027 = 0.6364 x 0.0185 = 0.0118 or approximately 1.2% of sampled pigs

The model results are reported in detail in Appendix 1. It was estimated that there was a moderate to high likelihood (0.26 or about 1 in 4) of infectious PRRS virus being present in oropharyngeal and tonsillar tissue at the time of slaughter, and there was a low likelihood (0.003 or about 1 in 300) of it being present in meat (muscle, bone and regional lymph nodes).

4.1.2.5 Comparison of model predictions with survey results

Although Larochelle and Magar (1997), Frey (1995a) and Wang (1999) concluded that there was a low likelihood of virus being present in meat of slaughter-age pigs in Canada, USA and Taiwan, the surveys carried out in these studies were not really random, and the test that was used to determine virus presence was virus isolation, which has been recently demonstrated in two studies to be a relatively insensitive method of determining whether meat of pigs at slaughter contains sufficient virus to infect other pigs (Van der Linden et al, 2003; Magar & Larochelle, 2004). Notwithstanding the higher sensitivity of feeding trials, the 0.3% likelihood predicted by the model is of the same order of magnitude as the 1.2% likelihood estimated from the random survey carried out by Magar & Larochelle (2004).

Considering that:

i) in endemic situations the majority of animals are viraemic from about 6 to 9 weeks of age;

ii) viraemia is typically prolonged, despite the appearance of antibodies early in the course of infection , with the duration of viraemia in natural infections being as long as 4 weeks;

iii) virus is present of meat of pigs during viraemia;

iv) virus persists in lymphoid tissues for 15-22 weeks;

v) most pigs are slaughtered at around 24-30 weeks of age;

vi) virus can be demonstrated in 1.2% of meat of pigs randomly sampled at slaughter;

it is concluded that there is a low likelihood of PRRS virus being present in pig meat at the time of slaughter.

4.1.3 The likelihood of PRRS virus surviving storage and transportation

Although PRRS virus survives relatively poorly in the external environment (Done et al, 1996) it survives freezing quite well. Although 90% of infectivity is lost within 1 week at 4°C (Benfield et al, 1999), infectivity is retained after incubation for one month at 4°C, for 4 months at -70°C (Benfield et al, 1992) or for 10 weeks at both -20°C and -70°C (Bloemraad et al, 1994). In studies involving survival of PRRS virus in muscle derived from infected pigs, virus was successfully isolated in samples collected from pigs on day 7 post infection and stored for 1 month at –20°C (Magar et al, 1995) and from samples collected from pigs on day 11 post infection and stored for 10 days at –23°C (Van der Linden et al, 2003). Although there was a 75% fall in the proportion of samples that were virus positive after freezing and

storage at –23°C, infectious virus was still present in sufficient amounts to initiate infection when this meat was fed to naïve pigs (Van der linden et al, 2003).

Market weight pigs challenged with PRRS can harbour infectious virus in tissues at 7 days post infection which will survive in meat/bone for at least 4 weeks at -20°C and between 3-4 weeks at 4°C (Frey et al, 1995b).

The optimal pH range for PRRS virus was found to be between 5.5 and 6.5 in titration studies of virus suspensions in alveolar macrophage cell cultures. It was noted that the pH of post-mortem muscle tissue kept at 4°C varies between 5.4 and 6. At this temperature the half-life of the virus in muscle tissue depends on the pH reached during the cooling and hardening period. For example, the half-life at pH 6.25 is approximately 50 hours while at pH 5 it is approximately 19 hours (Bloemraad et al, 1994).

Considering that:

i) the titre of PRRS virus present in meat falls by 90% after a week at 4°C and by 75% in a single freeze/thaw cycle;

ii) the interaction of pH and temperature in pig meat post slaughter on the stability of PRRS virus is not known with sufficient certainty;

iii) despite the fall in titre of PRRS virus in pig meat during chilling and freeze/thawing, meat retains its infectivity and is able to initiate infection when fed to naïve pigs;

it is concluded that it is likely that the infectivity will persist in chilled and frozen pig meat during storage and transport to New Zealand.

4.1.4 Release assessment conclusion

Considering that:

i) the model results indicate that there is a moderate to high likelihood (0.26 or about 1 in 4) of infectious PRRS virus being present in oropharyngeal and tonsillar tissue at the time of slaughter;

ii) there is a low likelihood (0.0118 or about 1.2%) of infectious PRRS virus being present in meat at the time of slaughter;

iii) it is likely that significant levels of PRRS virus infectivity will survive the chilling and freezing temperatures for the length of time that pig meat is held at during storage and transport to New Zealand;

it is considered that there is a non-negligible likelihood that chilled or frozen pig meat from a country with endemic PRRS will harbour infectious PRRS virus when imported into New Zealand.

4.2 EXPOSURE ASSESSMENT

The exposure assessment examines the likelihood that any PRRS virus present in imported meat will come into contact with, and result in infection in, susceptible species in New Zealand. Since PRRS virus only infects pigs, this is the only species considered in this assessment.

Throughout the 1990s it was assumed, based on the difficulty of isolating the virus from muscle, that meat was an unlikely vehicle for the dissemination of PRRS (Bloemraad et al, 1994; Magar et al, 1995; Frey 1995b; Larochelle and Magar, 1997; Wang, 1999). However, two recent studies have examined this question through feeding trials.

(i) Van der Linden et al (2003) collected meat samples from pigs that were slaughtered 11 days after being infected with PRRS by the intranasal route. These samples were frozen at -23°C and stored, mimicking normal production and processing. After 10 days of storage at this temperature, thawed meat was tested by virus isolation, and only two of 24 samples (12.5%, $CL_{95\%}$ 1-27%) were virus positive, although all but one muscle sample was PCR positive at this point, suggesting that either the titre of virus in the meat was below the level of detection or that the freezing and thawing cycle had inactivated the virus. After 14 days storage at -23°C, two 500g samples of raw muscle meat from each donor pig was cut into pieces about $7cm^3$ (approx 2 cm cubes) and fed over 2 days (250g per day) to two recipient pigs. That is, each of the 48 recipient pigs consumed 500g of raw meat over 2 days. Recipient pigs had been deprived of food for 2 days, and uptake by these animals was classified as good or moderate. The recipient pigs were observed to chew the meat samples. Three days after feeding, 50% of the recipient pigs (24 of 48) were viraemic, and 6 days after feeding all 48 recipient pigs were viraemic (some of these might have been the result of horizontal transmission between the recipient pigs). Four of the recipient pigs that became viraemic by day 3 had been fed meat from which virus could not be detected either before or after freezing, suggesting that there was sufficient infectivity in 500g of raw muscle meat to infect recipient pigs even when the titre was below the detection limit of virus isolation, which is accepted as $10^{1.8}$ $TCID_{50}$ per g of meat.

   Van der Linden et al (2003) suggested that the time from feeding meat to the development of viraemia probably depends on the amount of virus consumed, as recipient pigs that were fed meat that was positive for virus by virus isolation either before or after freezing became viraemic more quickly than recipients that were fed meat that was negative. Although the question of infectious dose was not explored in detail, Van der Linden et al (2003) demonstrated the transmission of PRRS by spiked meat samples (eight pigs fed four samples) which showed that transmission could occur by feeding 500g of meat containing virus at a titre of $10^{2.8\text{-}3.3}$ $TCID_{50}$ per g.

(ii) Magar and Larochelle (2004) reported similar results. They found that 19 of 1027 meat samples (1.85%) randomly collected at two Canadian slaughterhouses were positive to PRRS virus by PCR, even though only one sample was positive by virus isolation. When thawed meat from 11 of the PCR-positive carcasses was fed to recipient pigs (SPF pig pairs, 9 weeks old), in quantities from 1.05 kg to 1.8 kg over 2 days, seven of the 11 recipient pig pairs became infected, again confirming that infectivity is present in meat from which the virus cannot be isolated.

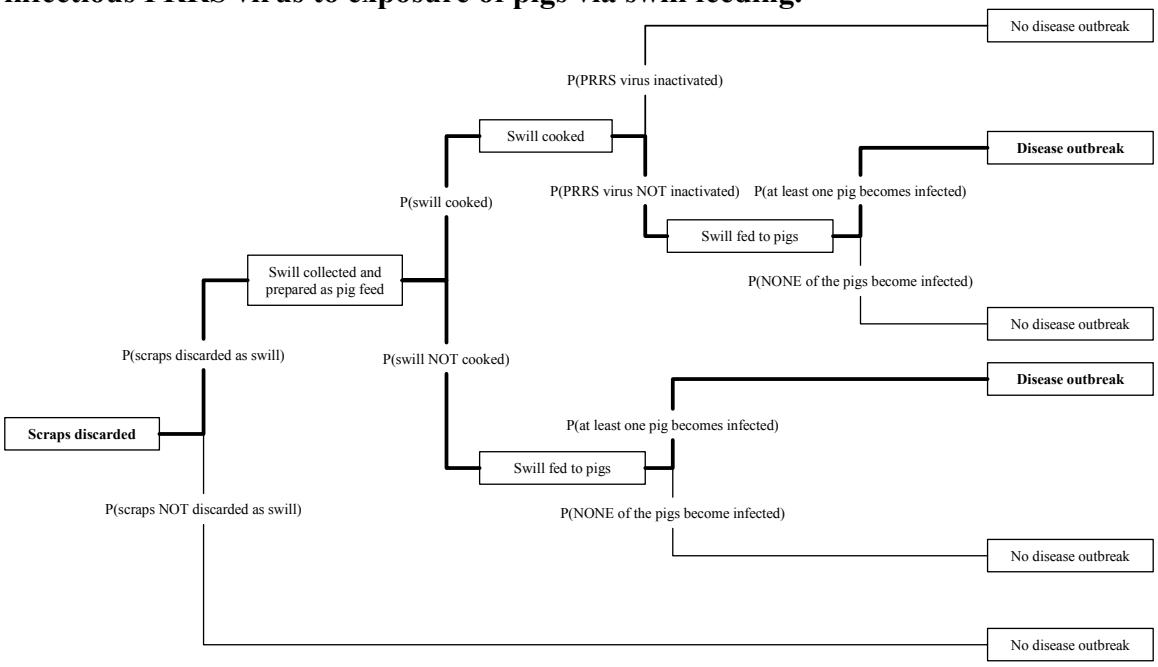
Thus, the scientific literature shows that meat from slaughter-aged pigs must be considered as a vehicle for the transmission of PRRS, if that meat is fed to pigs.

4.2.1 Effect of cooking on PRRS virus

Arteriviruses are relatively heat labile, and their survival decreases rapidly with temperatures above 4°C. PRRS infectivity persists for less than 24 hours at 37°C and less than 20 minutes at 56°C (Benfield et al, 1999).

Therefore, normal cooking[1] can be considered to inactivate PRRS virus, so that exposure of susceptible pigs to the virus in imported pig meat would require that pigs consume uncooked meat. As shown in Figure 6, effective exposure depends on the likelihood of scraps being generated prior to the imported pig meat being cooked, and the likelihood of raw scraps being fed to pigs.

Figure 6. The biological pathways leading from the disposal of scraps contaminated with infectious PRRS virus to exposure of pigs via swill feeding.



4.2.2 Likelihood of generation of infectious scraps prior to cooking

Scraps of raw pig meat could potentially be generated from any source where imported meat is purchased and trimmed prior to cooking for human consumption; this could include domestic households, restaurants, retail outlets, processors and manufacturers. However, even if a pig were to consume raw pig meat that was harbouring PRRS virus, infection could not be initiated in that pig unless the virus was present in a sufficient amount.

To estimate the likelihood of exposure through scraps of raw meat, in particular to allow the quantitative modelling of this, it would be necessary to consider the likelihood of infection occurring by the oral route with different quantities of virus (the infectious dose), the titre of

[1] Normal cooking is recommended at 75°C to 85°C. The USDA recommend that an internal temperature of at least 71°C be achieved – http://www.fsis.usda.gov/OA/pubs/pork.htm

the virus in meat of slaughter age pigs, and the likelihood of raw meat scraps of various sizes being generated during preparation of imported pig meat for human consumption.

The information on these matters is limited.

4.2.2.1 Infectious dose

In general, the infectious dose for viruses is known to vary with route of exposure. In the case of the lactic dehydrogenase-elevating virus of mice (also an arterivirus), transmission by oral exposure requires much higher doses than other routes. Notkins and Scheele (1963) reported that only 18% to 50% of animals were infected by oral exposure with doses of $10^{8.1}$ to $10^{8.8}$ ID_{50} per ml, while Cafruny and Hovinen (1988) found that the minimum infectious dose by intraperitoneal or tail cartilage injections approached a single virus particle. The literature contains little information on the infectious dose for PRRS virus, and only one study on the infectious dose by the oral route.

Yoon et al (1999) reported that 2 ml of an inoculum containing 10 fluorescent units of PRRS virus per ml were sufficient to infect young pigs by the intranasal or intramuscular routes. Benfield et al (2000), in a study on the titre of PRRS virus 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 (2005) estimated the infectious dose (ID_{50}) for PRRS virus to be $10^{5.3}$ $TCID^{50}$ (CI_{95} $10^{4.6} – 10^{5.9}$) by the oral route. The probability of infection by the oral route administration of different quantities of PRRS virus is shown in Table 15.

Table 15. Logit estimate of the probability of PRRS virus infection by dose by the oral route(TCID50).

Probability of infection	Oral dose ($TCID_{50}$)
0.01	$10^{1.7}$
0.10	$10^{3.6}$
0.20	$10^{4.2}$
0.25	$10^{4.4}$
0.30	$10^{4.6}$
0.40	$10^{5.0}$
0.50	$10^{5.3}$
0.60	$10^{5.6}$
0.70	$10^{5.7}$
0.75	$10^{6.1}$
0.80	$10^{6.4}$
0.90	$10^{7.0}$
0.99	$10^{8.9}$

Source: Hermann et al (2005).

The infectious dose by the intranasal route, by comparison, was estimated as $10^{4.0}$ $TCID_{50}$ (CI_{95} $10^{3.0} – 10^{5.0}$) 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 (Hermann et al, 2005). However, these studies were done 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 (Johnson, 2003).

In estimating a dose-response curve for oral PRRS virus, Hermann et al (2005) essentially rejected the notion of minimum infectious dose. The limit of detection of PRRS virus 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 Table 15 can be seen to be enough virus to infect 25-30% of pigs receiving this dose by the oral route. The finding that meat containing virus below detectable levels can result in infection when 500-900 g of meat is fed (Van der Linden, 2003; Magar and Larochelle, 2004) is consistent with the dose-response relationship estimated by Hermann et al (2005).

4.2.2.2 Titre of virus in meat of slaughter age pigs

Neither of the recently reported feeding trials (Van der Linden et al, 2003; Magar and Larochelle, 2004) addressed the titre of virus present in meat of slaughter age pigs. This can be expected to vary between production systems (Dee, 2003), with the lowest titre expected in animals from 'stabilised herds'.

Similarly, neither feeding trial investigated the minimum quantity of meat that can transmit infection orally. While Van der Linden et al (2003) fed a constant 500g to each recipient pig, Magar and Larochelle (2004) fed recipients a variable amount of meat, ranging from approximately 500g to approximately 900g[1].

Nevertheless, in both feeding trials the meat fed to pigs was thawed, and this has been shown to reduce by 75% the frequency of samples being positive by virus isolation, so it is reasonable to assume that a freeze/thaw cycle reduces the titre of virus present by 75% from the titre present at slaughter (Van der Linden et al, 2003).

The titre of virus present in chilled meat will depend on the ultimate pH of meat, the chilling temperature, and the time held at that temperature. Approximately 90% of PRRS infectivity is lost within 1 week at 4°C (Benfield et al, 1999).

In both chilled and frozen/thawed meat, the titre of virus in the meat at the time of feeding to recipient pigs would depend on the length of time (if any) that the meat has been held at ambient temperature. If the meat were held at 37°C for 24 hours, it could be considered free of infectious PRRS virus (Benfield et al, 1999).

4.2.2.3 Likelihood of generation of raw scraps during preparation for human consumption

Surveys have not been carried out on the size and quantity of raw scraps that may be generated from raw pig meat during preparation for human consumption in different situations in New Zealand. In the absence of such survey information, it is assumed that the likelihood and level of scraps generated would be inversely proportional to the level of processing of the pig meat prior to purchase. Therefore, at one extreme, the likelihood of raw scraps being generated from cuts such as chops, steaks and roasts is considered to be very

[1] Individual meat samples from the 11 pigs weighed from 1.05 to 1.8 kg. Each sample was cut into four equal parts. Two of these parts were fed to the two recipient pigs on the first day, and the other two parts were fed to the same recipient pigs on the second day.

low, while at the other extreme the likelihood of generating of raw scraps from whole carcasses is considered to be high. Since the majority of meat purchased for human consumption in households is likely to be in the form of ready to cook cuts, it is concluded that the overall likelihood of generation of raw scraps of pig meat from households is low. The likelihood of raw pig meat scraps being generated by restaurants, retail outlets, processors and manufacturers is considered to be higher than for households.

4.2.2.4 Conclusion

Despite the recent estimation of the infectious dose by the oral route, since the titre of PRRS virus in pig meat is not known, it is not possible to calculate the minimum size of meat scrap necessary to initiate infection by the oral route. However, the low pH of the porcine stomach would rapidly destroy PRRS virus, so it is considered that infection via the oral route would require the virus making contact with the mucosal surface of the oropharynx and tonsils (Van der Linden et al, 2003). Therefore it is likely that chewing of meat scraps is necessary if infection is to result, which means that there is probably a minimum scrap size below which infection is unlikely to occur, regardless of the amount of virus present. However, the size of such scraps is not known.

Feeding trials have demonstrated that PRRS virus can be transmitted by the feeding of both experimentally- and naturally-infected frozen/thawed meat, provided sufficient meat is fed. However, the titre of PRRS virus in meat at slaughter remains unknown, and there has been no attempt to explore the effect of size of scraps and infectivity. Indeed, the infectious dose approach explored by Hermann et al (2005) supports the notion that scraps of any size have the potential to infect an animal orally, and that the likelihood of infection occurring is directly related to the amount of meat fed.

In view of the above, it is not possible to accurately estimate the likelihood that scraps of a critical size will be generated prior to further processing (cooking) of imported pig meat, so the likelihood of generating infectious scraps prior to cooking must be considered to be non-negligible. However, it is improbable that the volume of scraps generated from a single household at any one time will approach the relatively large quantities (500 – 900g) that were used in the feeding trials.

4.2.3 Evaluation of historical imports

Although PRRS virus is considered to be endemic in most countries where there is a substantial pig industry, several countries in Europe have remain free, namely Sweden, Finland, Norway and Switzerland. Of these, at least Sweden and Finland maintain strict controls over the importation of live pigs and porcine genetic material, but at the same time they import substantial volumes of pig meat from countries where PRRS is endemic. Since 2000, annual imports of fresh and frozen pig meat have been approximately 40,000 tonnes for Sweden and almost 10,000 tonnes for Finland. However, this does not necessarily mean that food waste is not a vehicle for spread of PRRS, because EU Member States are obliged by legislation to impose and enforce a prohibition on the feeding of any mammalian protein to any farmed animals, so there would have been a negligible likelihood of exposure by this route within the EU.

As discussed earlier, more than 30% of the pig meat consumed in New Zealand is imported, and much of it comes from countries where PRRS is endemic. In the period 1999- 2000,

approximately 80% of imported pig meat came from infected countries, averaging about 9,000 tonnes per year (see Table 3). In addition, as discussed in section 2.1.2, from 1998 to mid-2005 there were no controls in this country on the feeding of food waste to pigs. Thus, since preliminary sanitary measures for PRRS were put in place only in 2001, there was a 3½ year period (1998 to mid-2001) during which pig meat was imported from PRRS-infected countries without any controls being imposed in the form of requirements for cooking or feeding of garbage. Despite this New Zealand has remained free from PRRS virus (Stone and Kittelberger, 2004).

About 30,000 tonnes of pig meat could have been imported from PRRS-infected countries over that 3½ year period without any controls on garbage feeding being in place in this country. The fact that this did not result in the introduction of PRRS suggests that the likelihood of effective exposure to PRRS virus via pig meat is remote. However, the possible explanations include the following:

i) about 97% of all imported pig meat is imported frozen, and although PRRS virus can be expected to survive in frozen meat, a single freeze/thaw cycle can be expected to reduce the amount of PRRS virus in pork by 75% (Van der Linden et al, 2003), such that the likelihood of an infectious dose being present in meat that was imported in frozen form is only 25% that of meat that was imported chilled.

ii) about 95% of imported frozen pork would have been further processed before being sold to consumers – in New Zealand such processing commonly involves cooking, which would inactivate any residual PRRS virus present.

iii) most chilled meat would have been held for a total of 7 days between slaughter and sale to consumers in New Zealand, over which time about 90% of any PRRS infectivity present would be lost.

iv) the increasing importation of higher value consumer-ready cuts would have meant that there would have been limited trimming prior to cooking and therefore limited generation of raw scraps.

Therefore it appears that even in the absence of any regulation of garbage feeding in New Zealand, the combination of international market conditions, handling practices and consumer preferences have ensured that the likelihood of effective exposure from PRRS in imported pig meat is remote in practice.

4.2.4 Evaluation of the effect of recent changes to regulation of garbage feeding

As discussed in section 2.1.2, under the Biosecurity (Meat & Food Waste for Pigs) Regulations 2005, which came into effect on 9 July 2005, the feeding to pigs of untreated meat or untreated food waste is prohibited. Clearly, if the regulations are complied with by all pig farmers, then there cannot be any exposure pathway by which PRRS virus in imported pig meat could cause infections in New Zealand pigs. It follows that the likelihood of exposure by this route is inversely proportional to industry compliance with the regulations. Nevertheless, even normal levels of compliance will mean that the likelihood of exposure is less than it was in the 3½ years discussed above.

4.2.5 Likelihood of exposure to uncooked scraps by compartment

Although accurate statistics on the frequency of garbage feeding are not available, the feeding of waste food to pigs is not an uncommon practice in New Zealand, particularly around the main urban centres in the North Island. In a study of farm-level risk factors for post-weaning multisystemic wasting syndrome (PMWS) in New Zealand, about 35% of pig farms reported feeding some form of food waste (Stone, 2004), but the likelihood of uncooked scraps of pig meat being in such food waste has not been investigated.

Both the likelihood of garbage feeding and the likelihood of compliance with the 2005 garbage feeding regulations may be expected to vary across the pork production sector, as the awareness of the general principles of biosecurity tends to be lower in smaller herds. Thus, the likelihood of exposure can be expected to vary in different compartments of the industry. Moreover, feral pigs are outside the pork production sector and constitute a special case. Therefore it is appropriate to consider the following groups separately:

- feral pigs
- backyard pigs
- commercial piggeries

In the following discussion it is important to note that an objective cut-off between the backyard and commercial sectors cannot be determined, as there may be a number of small herds that could be described as either backyard or semi-commercial.

4.2.5.1 Feral pigs

Feral pigs occupy about 93,000 sq km of New Zealand at an overall density of 1.2 animals per sq km (McIlroy, in press[1]). Assuming that kitchen waste containing scraps of uncooked pig meat is very likely to be disposed of into landfills, the likelihood that feral pigs could gain access to such scraps depends on the location and standard of management of landfills. Modern management practices for landfills under environmental legislation in New Zealand mean that feral pigs are unlikely to have access to dumped kitchen scraps in the majority of landfills, but there may be a small number of landfills in New Zealand, particularly in rural areas, where feral pigs might gain access. However, although PRRS virus retains infectivity for 30 days at 4°C, infectivity is lost after relatively short periods at ambient temperatures; after 1 to 6 days at 21°C, and after only 24 hours at 37°C (Benfield et al, 1999).

Although environmental temperatures above 20°C may be expected to occur quite commonly during summer months in many parts of New Zealand (Gerlach, 1974), temperatures above 30°C are very rare. However, for organic garbage that is buried within a landfill, composting effects could be expected to ensure that the temperature rises above 37°C relatively quickly. The likelihood of pig meat scraps being consumed by a feral pig within 24 hours of it being discarded in garbage is remote. Moreover, in view of the factors discussed in section 4.2.3, the likelihood of exposure of feral pigs to uncooked scraps that are infectious for PRRS is considered to be extremely low.

[1] McIlroy, in press, The Handbook of New Zealand Mammals. Oxford University Press.

4.2.5.2 Backyard pigs

Notwithstanding the limited recording of urban backyard pigs in *AgriBase*, as discussed in Section 2.1.2, about 7,000 farms are recorded as keeping at least one pig but are not commercial pig farms. It is considered very likely that kitchen waste would be fed to these pigs, whether or not the householders are aware of their obligations under the garbage feeding regulations of 2005. However, as discussed earlier, the likelihood that scraps of raw pig meat will be present in households kitchen waste, especially in quantities approaching the 500-900g used in the feeding trials, is considered to be low, so the likelihood of exposure of backyard pigs to uncooked pig meat scraps is correspondingly low. Considering the factors discussed in Section 4.2.3, the likelihood of exposure of this sector to scraps containing infectious PRRS virus is very low.

4.2.5.3 Commercial piggeries

AgriBase records about 350 commercial piggeries as users of garbage and food waste, including a small number of large commercial piggeries. However, most industrial food waste such as brewers grains, stale bread, fruit and vegetable waste from supermarkets or cooked food waste poses a negligible risk of transmission of any animal pathogens. A larger number of small commercial operators collect food waste from restaurants, institutions and supermarkets for use as pig feed. As discussed in Section 4.2.2, some of this material may contain uncooked meat scraps, but considering the factors discussed in Section 4.2.3, the likelihood of exposure to scraps containing infectious PRRS virus is very low. However, regardless of the source of the food waste, the risk is essentially zero for any operation that is complying with the 2005 garbage feeding regulations.

4.2.6 Exposure assessment conclusion

Considering that:

i) PRRS virus will be inactivated by normal cooking, so the only exposure pathway of relevance is the feeding of raw pork;

ii) although a recent study has estimated the oral infectious dose of PRRS virus for pigs, the titre of virus likely to be in meat of pigs at slaughter is still unknown, so the minimum scrap size likely to infect a pig by the oral route is unknown. Quantities used in transmission studies were 500–900 g and to be infectious, scraps probably have to be large enough so as to require chewing;

iii) there is a low likelihood that scraps of raw pork in quantities similar to those used in transmission studies will be present in kitchen waste generated from households;

iv) there is a moderate likelihood that scraps of raw pork will be generated in waste from restaurants, retail outlets, processors and manufacturers;

v) the form of pig meat likely to be imported into New Zealand and the likely processing that it is submitted to prior to being sold for human consumption means that it is very unlikely to contain infectious PRRS virus;

vi) it is illegal to feed raw meat scraps to pigs in New Zealand;

vii) compliance with the garbage feeding regulations is likely to be high in the commercial pig production sector, but is probably lower in other sectors;

it is considered that for piggeries complying with the garbage feeding regulations the likelihood of exposure to infectious PRRS virus in pig meat is essentially zero, and for other piggeries the likelihood of exposure is very low.

This page is intentionally blank

4.3 CONSEQUENCE ASSESSMENT

The consequence assessment examines the consequences on the people, the environment and the economy of New Zealand of entry, establishment or spread of the organism, in the event that it is introduced in the commodity and susceptible animals are exposed to it.

Since PRRS virus affects only pigs, its introduction would have no effects on the health of New Zealanders, and the only effect on the environment would be via its effect on feral pigs.

Moreover, since New Zealand pork production is not price-competitive internationally, the export of pork from New Zealand is limited, and the introduction of PRRS virus would have a negligible macro-effect on the economy. Therefore, the economic consequences of the introduction of PRRS virus would be restricted to the micro-economic effects arising from direct losses incurred at the level of individual pig farms.

The approach in this consequence assessment is to consider what might reasonably be expected if an animal or group of animals became infected as a result of exposure to the virus in scraps of uncooked pig meat. Considering that there is a non-negligible likelihood of exposure for feral pigs, backyard piggeries and those 'fringe' backyard/commercial piggeries that do not comply with the garbage feeding regulations, the consequence assessment will examine the consequences of PRRS becoming established in each of these sectors, including the potential spread from the sector concerned to other pig production sectors.

Of central importance in considering the consequences of introduction of the virus into any particular group or compartment of pigs is an assessment of the likelihood that it would be confined to that particular group or compartment. The following assessment is made in light of the discussion in the hazard identification on the major routes of transmission between herds.

4.3.1 Consequences by compartment

4.3.1.1 Feral pigs

Many countries have found that the prevalence of PRRS is inversely proportional to pig density (Zimmerman, 2003). Wild boars in France and Germany are known to have been infected with the virus (Albina, 1997), but at rates less than 4% (Albina et al, 2000). Similar results have been reported from Mexico (Morilla et al, 2003), Puerto Rico and USA (Zimmerman, 2003), and there is no evidence of clinical signs of infection in wild pigs and no indication that they have any role in transmission to domestic pigs. Feral pigs in New Zealand are widely spread, and although the population density varies it is generally low; extremely low relative to the normal population density on a pig farm. Therefore the likelihood that PRRS virus would be maintained in wild pig populations is considered to be negligible. Although it is possible that nocturnally foraging feral pigs could come into contact with domestic pigs, particularly those raised outdoors, the likelihood of this event is considered to be low. Therefore, the consequences of introduction of PRRS into the feral pig population of New Zealand are considered to be negligible.

4.3.1.2 Backyard pigs

As discussed in section 2.1.2, over 95% of the 7,000 recorded pig herds in in New Zealand are considered to be non-commercial units, including semi-commercial herds, small backyard herds, and hobby-farms.

If PRRS were introduced into a herd of backyard pigs via the illegal feeding of raw scraps of pig meat harbouring the virus, the direct animal health consequences may be expected to vary considerably, depending on the size, age structure and management of the unit. As discussed earlier, the clinical signs in infected herds are highly variable; in very small herds comprising a few fattener pigs for home consumption there would probably be few clinical signs, whereas in backyard breeder units comprising tens of sows the clinical signs could include reproductive losses in sows, mortality in very young piglets and respiratory disease in older pigs. However, it is reasonable to assume that epidemic fadeout would occur in many herds after a variable period of time due to a lack of susceptible animals. Surveys of backyard herds in Mexico (Morilla et al, 2003) and Colombia (Mogollon et al, 2003) support this assumption. The likelihood that an incursion would be detected in a backyard herd is considered to be very low, as in the absence of overt clinical signs, particularly against a background of other endemic diseases and other production-limiting factors such as sub-optimal nutrition, there would be no reason for animal health professionals to become involved with the herd.

Spread from the index herd might be possible by several routes, but one route of inter-herd transmission that requires special discussion in this context is airborne spread. As discussed in the hazard identification section, in the early 1990s much of the so-called "area spread" up to 3 km was assumed to be by airborne aerosols of virus. If these assertions were to be taken at face value, then it could be argued that the introduction of PRRS virus into even a single backyard piggery could put the entire pork industry at risk, as spread by the airborne route would quickly result in a widespread epidemic. Indeed, *AgriBase* records that in June 2005 there were between 239 and 415 instances in New Zealand where there were two pig farms within 3 km of each other.[1] However, as discussed in the hazard identification, research carried out since the late 1990s has led to general agreement that in the field the transmission of PRRS virus by aerosols is probably a rare event, and that if it occurs at all it is likely to be possible only within infected barns. In that case the consequences of a single backyard piggery becoming infected would be limited to that establishment itself, unless there were movement of infected animals, genetic material or fomites from the index property to other piggeries. Thus, some spread to other herds within the backyard sector would be likely, particularly to herds that introduced live animals (including travelling boars) or used semen from the index herd, but possibly also to other herds that shared implements such as vehicles or other equipment. However, spread to commercial pig herds that observe standard biosecurity practices would be most unlikely.

Therefore the consequences of infection in a backyard piggery are considered to be low.

[1] This was based on 686 mapable piggeries registered in AgriBase as at June 2005, that were recorded as farm_type = 'PIG' or had >= 20 pigs listed in their enterprise data. The analysis technique created unique pairings (i.e. treated set [FarmA, FarmB] as identical to [FarmB, FarmA]) and then calculated inter-land parcel distances. There were 415 pairings when the calculation was based on farm boundaries, whereas there were 239 pairings when the calculation was based on centroids (representing a point approximating the centre of the largest block of contiguous land owned by each piggery).

4.3.1.3 Commercial piggeries

As discussed, the clinical signs of PRRS infection are extremely variable, and depend on the strain of the virus, the immune status of the herd, the presence of intercurrent disease and management factors. Even in the initial epidemic period, infections in finishing pigs, boars, unbred gilts and sows often go unnoticed, as in these animals infection is commonly characterised only by a transient fever and inappetence. However, in breeding herds the introduction of the virus could be expected to result in epidemics of acute porcine reproductive disease characterised by a marked increase in late-term abortions (up to 15% at peak of the epidemic), stillborn and weak pigs, lowered farrowing rates, high death rates among weaned pigs (increased from 1-2% to approximately 10-15%), and impaired sow fertility. Acute losses could last 4 – 6 months, after which the disease is likely to become endemic and lead to chronic production losses in weaners and fattening pigs. However, not all herds may experience such dramatic consequences, even in the epidemic phase. As a result the economic consequences of PRRS are extremely variable and range from very serious to no obvious consequences. Estimates of the cost of an acute outbreak in the USA vary from US$228-302 per sow (Hock and Polson, 2003).

In one of the few instances where the cost estimate was based on high quality records and rigorous analytical methods, Polson et al (1990) modelled the cost of production losses of PRRS on a pig farm in Minnesota over the epidemic period of 4 months. Statistical analysis of computerised production records showed that the major losses were the result of a large reduction in farrowing rate; this was reduced by 34% in first parity sows and by 18% overall. The production losses were modelled at US$236 per sow, and this translated to an 80% reduction in expected profits for the year of the outbreak. Statistical analysis of the available data did not indicate any significant losses in the endemic situation, notwithstanding anecdotal accounts of losses continuing in the endemic period for 15 – 24 months. Nevertheless, using assumptions on possible losses in the endemic period[1], the model projected total costs over a 3 year period of approximately US$500 per sow.

In the UK, the epidemic period lasted for 11 weeks in four herds studied in 1991, and after this period the production levels returned to normal levels (Hopper et al, 1992).

If PRRS were introduced onto commercial piggeries, the direct consequences at a herd level (that is, production losses) would probably be of a similar magnitude to what has been experienced in other countries. However, it is important to note that in countries where PRRS is now endemic, the virus was already widespread by the time the clinical syndrome was recognised, so eradication or even containment were never realistic control options, unlike the situation that currently exists in this country. For the purposes of this risk analysis, it is reasonable to accept the assertion of Albina (1997) that as long as the AI centres do not become infected the disease can be controlled by standard farm-level biosecurity measures.

The New Zealand Pork Industry Board defines a commercial herd as one having 80 sows or more[2], which means about 750 pigs in total. However, as shown in Table 4 and discussed in Section 2.1.3, there are only about 150 pig farms in New Zealand with more than 500 pigs, which means that there are fewer than 150 commercial pig farms in New Zealand. The direct

[1] Assuming a 4 month acute outbreak, a 24 month endemic breeding herd stage, a 2 month acute post-weaning stage and a 24 months endemic stage.
[2] Angus Davidson, NZPIB, email to HJ Pharo dated 11 July 2005.

costs at the farm level would depend on the number of sows infected. Costs of control and eradication would depend on the approach adopted (Dee, 2003).

The introduction of PRRS virus into pig herds in New Zealand would be unlikely to result in significant indirect costs in terms of domestic or international market reactions. The domestic market is unlikely to be affected as there are no zoonotic or food safety issues surrounding PRRS. Internationally, no major pork producing country in the Northern Hemisphere considers that PRRS can be transmitted by pork, as evidenced by the lack of regulations for intracommunity trade in pig meat among EU Member States, regardless of the fact that at least two of them (Sweden and Finland) are free from PRRS. Moreover, exports of pork from this country are limited to a few hundred kilograms annually to the Pacific Islands and Singapore[1].

4.3.2 Consequence assessment conclusion

The consequences of a herd becoming infected with PRRS virus would depend on the strain of the virus introduced and the nature of the herd. The predominant route of transmission is contact between viraemic and naïve pigs, but transmission by semen is also recognised. Therefore it is considered that for spread to occur off the index property, there would have to be movement of live infected animals, semen from infected boars, or possibly contaminated fomites. This could probably occur relatively easily within the smallholder sector. However, for any pig farm adhering to standard biosecurity measures[2] (especially the appropriate sourcing of genetic material and replacement live animals and the control of visitors and fomites onto the farm) the likelihood of becoming infected with PRRS virus is considered to be low.

If PRRS were introduced as a result of the illegal feeding of raw imported pig meat to pigs, the majority of impacts of PRRS virus would be the direct disease effects on small, non-commercial breeding herds. Spread from such herds to commercial herds would be likely in the case of lapses in biosecurity.

Apart from the direct losses on affected farms, the consequences of PRRS introduction on the economy, the people and the environment of New Zealand are considered to be negligible.

4.4 RISK ESTIMATION

As a result of this analysis, the conclusion of MAF's 2001 draft release assessment is unchanged. That is, there is a non-negligible likelihood of release of PRRS virus in imported pig meat.

The likelihood of exposure through the feeding of raw scraps of imported pork is considered to be negligible for any farm that is complying with the 2005 garbage feeding regulations. Although the likelihood of exposure is undoubtedly higher for farms that are not complying with those regulations, this likelihood cannot be estimated because the minimum amount of uncooked pork that contains an infectious dose of PRRS virus is not known. In the absence of that information the risk analysis must conclude that this likelihood is non-negligible.

[1] Angus Davidson, NZPIB, email to HJ Pharo dated 18 July 2005.
[2] For example, the "Farm Biosecurity Policy" developed by the NZ Pork Industry Board. See: http://www.pork.co.nz/technical_papers/6%20Pig%20Health%20and%20Welfare.pdf

If PRRS did become established in a sector of the pig industry through non-compliance with the garbage feeding regulations, the consequences of PRRS infection would be significant in the breeding herds that became infected. However, the likelihood of secondary spread to any units observing standard biosecurity measures would be negligible. There would be no significant impact on exports.

The risk estimate for PRRS virus in imported pig meat is therefore considered to be non-negligible for small, non-commercial or marginally commercial breeding herds that are not complying with the garbage feeding regulations and for herds with inadequate biosecurity practices.

This page is intentionally blank

5. RISK MANAGEMENT

5.1 RISK EVALUATION

The risk estimate for PRRS virus in imported pig meat is non-negligible for farms where there is not compliance with the 2005 garbage feeding regulations, and for other pig farms if there are lapses in biosecurity.

In this situation risk management measures could be applied :

either:

i) to protect a sector of the pig industry (most likely small, non-commercial or semi-commercial breeding herds that are not complying with the swill feeding regulations) from a risk that they would not be facing if they were complying with the regulations

or:

ii) to protect other farms from secondary exposure to PRRS virus through contacts with farms that are not complying with the swill feeding regulations, on the basis that even for farms that do practise standard biosecurity measures, breaches in biosecurity can occur that may result in PRRS virus being introduced from farms that are infected by illegally feeding garbage.

5.2 OPTION EVALUATION

5.2.1 Risk management objectives

Risk management objectives in this situation fall into the following two categories:

(1) to reduce the likelihood of release, that is, to reduce the likelihood that imported pig meat harbours the virus when it is given a biosecurity clearance in New Zealand;

(2) to reduce the likelihood of exposure, that is to reduce the likelihood that uncooked imported pig meat would be fed to pigs in New Zealand.

5.2.2 Options available

Although PRRS is listed by the OIE as a notifiable disease, the *Terrestrial Animal Health Code* (OIE, 2005) does not include a chapter on PRRS, so there are no international guidelines in regard to this virus for the safe trade of pig meat.

The following discussion on risk management options available is divided into measures that reduce the likelihood of release and those that reduce the likelihood of exposure.

5.2.2.1 Measures that reduce the likelihood of release

Removal of high risk tissues

PRRS virus has a high affinity for lymphoid tissues, as discussed earlier (section 4.1.2.2). Replication may continue in pulmonary macrophages for up to 7-8 weeks post infection, and

as immunity develops the virus localises in lymphoid tissues where it has been found to persist for periods of up to 22½ weeks post infection. However, replication does not appear to be significant in bone marrow.

Therefore, although there is no reason to suspect that the removal of bone would reduce the risk, the removal of major carcass lymphoid tissues, especially those of the head and neck, and also the major regional lymph nodes, can be considered to significantly reduce the risk in meat.

One way of applying this principle as a safeguard would be to permit only the importation of specific cuts of meat from which lymph nodes had been removed. For example, whole carcasses might be permitted only if the head and neck are removed, or importation might be restricted to any cuts that have the major lymph nodes removed.

Stabilised herds

Although PRRS virus has been shown to persist for long periods in oropharyngeal and tonsillar tissues, in other tissues the likelihood and titre of viraemia at slaughter is dependent on when the animals became infected, their immune status at the time of infection, the duration of viraemia and the age at slaughter. In animals vaccinated with modified live vaccines, infection leads to a much lower viraemia of shorter duration than in unvaccinated animals, and animals that are naturally exposed to field virus after having recovered from a previous infection with the same virus strain are known to have shorter duration of viraemia than naïve animals exposed for the first time, providing the basis for the concept of the 'stabilised/inactive' herd (Dee, 2003).

Thus, in situations where infection is known to have occurred several months prior to slaughter, even if the animals have been recently re-infected, the likelihood of viraemia in slaughter age pigs can be considered to be significantly lower than in situations where a herd of naïve animals has been infected just a few weeks prior to slaughter (the peak of viraemia occurs 11 days post-infection). For example, if pigs became infected with PRRS at around 4 months of age and were slaughtered at 6 months, most market age pigs would be either clear of infection or have viraemia of a very low titre at the time of slaughter.

This notwithstanding, there is no accepted standard for herd stability in North America and protocols for monitoring stability have not yet been developed. Therefore the principle of the stabilised herd cannot be incorporated into risk practical management measures at this time.

Treatment of pig meat to inactivate PRRS virus

PRRS virus is known to be relatively sensitive to pH and outside the range of pH 6.0 to 7.5 the virus is rapidly inactivated (Benfield et al, 1999). On this basis a wide range of salamis can be considered to pose negligible risk of PRRS.

A single freeze/thaw cycle has been shown to reduce the titre of virus by up to 2 logs (Steverink, 2000) and the likelihood of virus isolation in thawed meat was shown to be only 25% of that prior to freezing (Van der Linden et al, 2003). Therefore, the risk posed by frozen meat is considerably lower than that posed by chilled meat. Similarly, holding meat chilled for a week has been shown to reduce the level of infectivity present by 90% (Benfield et al,

1999). Thus, curing for 12 months such as involved in the production of Parma ham is considered to result in insignificant levels of infectivity.

Benfield et al (1992) reported that the virus was completely inactivated in tissue cultures containing a starting titre of 10^5 $TCID_{50}$ per 100 µl by holding them for 45 minutes at 56°C, and Bloemraad et al (1994) reported a 3 log decline in virus titre after one hour at 56°C. Thus, pig meat that is cooked at this level or greater is considered to pose negligible risk.

5.2.2.2 Measures that reduce the likelihood of exposure

High value cuts

Any form of meat that minimises trimming or cutting during its preparation prior to cooking can be expected to pose a lower risk than whole carcasses because of the lower likelihood that scraps will be generated prior to cooking. For example, in the case of consumer-ready cuts of pork, it is considered that there is a negligible likelihood of meat scraps being generated prior to cooking.

5.2.3 Recommended sanitary measures

Pig meat must be:

either

- from a country free from PRRS

or

- treated prior to import or on arrival, in an officially approved facility, by approved cooking or pH change

or

- in the form of consumer-ready, high value cuts

or

- further processed on arrival, in an officially approved facility, into consumer-ready high value cuts

This page is intentionally blank

REFERENCES

Albina E (1997). Epidemiology of porcine reproductive and respiratory syndrome (PRRS): an overview. *Veterinary Microbiology* 55, 309-316.

Albina E, Mesplede A, Chenut G, Le Potier MF, Bourbao G, Le Gal S, Leforban Y (2000). A serological survey on classical swine fever (CSF), Aujeszky's disease (AD) and porcine reproductive and respiratory syndrome (PRRS) virus infections in French wild boars from 1991 to 1998. *Veterinary Microbiology* 77, 43-57.

Albina E, Blanquefort P, Vannier P, Le Potier MF, Mesplede A, Delzescaux D (2003). PRRS in France – an example of disease control and eradication. In: Zimmerman J, Yoon KJ (Eds). *2003 PRRS Compendium: Second Edition*. P 245-252. Des Moines, National Pork Board.

Allende R, Laegreid WW, Kutish GF, Galeota JA, Wills RW, Osorio FA (2000). Porcine reproductive and respiratory syndrome virus: description of persistence in individual pigs upon experimental infection. *Journal of Virology* 74(22), 10834-10837.

Allison G (2003). Control using oropharyngeal scraping inoculation. In: Zimmerman J, Yoon KJ (Eds). *2003 PRRS Compendium: Second Edition*. P 143-146. Des Moines, National Pork Board.

Batista L, Dee SA, Rossow KD, Deen J, Pijoan C (2002). Assessing the duration of porcine reproductive and respiratory syndrome virus persistence and shedding in a large population of breeding age gilts. *Canadian Journal of Veterinary Research* 66, 196-200.

Batista L, Pijoan C, Dee S, Olin M, Molitor T, Joo HS, Xiao Z, Murtaugh M (2004). Virological and immunological responses to porcine reproductive and respiratory syndrome virus in a large population of gilts. *Canadian Journal of Veterinary Research* 68, 267-273.

Benfield DA, Nelson E, Collins JE, Harris L, Goyal SM, Robinson D, Christainson WT, Morrison RB, Gorcyga DE and Chladek DW (1992). Characterisation of swine infertility and respiratory syndrome (SIRS) virus (isolate ATCC-VR2332). *Journal of Veterinary Diagnostic Investigation* 4, 127-133.

Benfield D, Rowland R, Nelson J, Neiger J, Rossow K, Collins J (1996). A model for the study of persistent PRRS infection. *Conference of Research Workers in Animal Disease*. Abstract 187.

Benfield DA, Collins JE, Dee SA, Halbur PG, Joo HS, Lager KM, Mengeling WL, Murtaugh MP, Rossow KD, Stevenson GW, Zimmerman JJ (1999). *Porcine Reproductive and Respiratory Syndrome*. In: Straw ES, D'Allaire S, Mengeling WL, Taylor DJ (eds). Disease of Swine (8th edition).Pp 201-232. Iowa State University Press, Ames.

Benfield D, Nelson C, Steffen M, Rowland R (2000). Transmission of PRRSV by artificial insemination using extended semen seeded with different concentrations of PRRSV. In: *Proceedings of the American Association of Swine Practitioners* pp 405-408.

Blaha T, Büker E (1995). Risk factors for the spread and severity of PRRS. *Proceedings of Second International Symposium on Porcine Reproductive and Respiratory Syndrome (PRRS),* Copenhagen, p51.

Bloemraad M, de Kluijver EP, Petersen A, Burkhardt GE, Wensvoort G (1994). Porcine reproductive and respiratory syndrome: temperature and pH stability of Lelystad virus and its survival in tissue specimens from viraemic pigs. *Veterinary Microbiology* 42, 361-371.

Bøtner A (2003). The PRRS situation in Denmark, Norway, Finland and Sweden. . In: Zimmerman J, Yoon KJ (Eds). *2003 PRRS Compendium: Second Edition*. P 233-238. Des Moines, National Pork Board.

Bøtner A, Strandbygaard B, Sorensen KJ, Have P, Madsen KG, Madsen ES, Alexandersen S (1997). Appearance of acute PRRS-like symptoms in sow herds after vaccination with a modified-live PRRS vaccine. *Veterinary Record* 141(19), 497-499.

Brockmeier SL, Lager KM (2002). Experimental airborne transmission of porcine reproductive and respiratory syndrome virus and *Bordetella bronchiseptica*. *Veterinary Microbiology* 89, 267- 275.

Brouwer J, Frankena K, Dejong MF, Voets R, Dijkhuizen A, Verheijden J, Komijn RE (1994). PRRS - Effect on herd performance after initial infection and risk analysis. *Veterinary Quarterly* 16(2), 95-100.

Bush EJ, Corso B, Zimmerman J, Swenson S, Pyburn D, Burkgren T (1999). Update on the acute PRRS investigative study. *Swine Health and Production* 7(4), 179-180.

Cafruny WA, Hovinen DE (1988). The relationship between route of infection and minimum infectious dose: studies with lactate dehydrogenase-elevating virus. *Journal of Virological Methods* 20, 265-268.

Cho JG, Dee SA, Deen J, Trincado C, Fano E, Murtugh MP, Collins JE, Joo HS (Accepted for publication). An evaluation of different variables on the shedding of porcine reproductive and respiratory syndrome virus in aerosols. *Canadian Journal of Veterinary Research*.

Christianson WT, Choi CS, Collins JE, Molitor TW, Morrison RB, Joo HS (1993). Pathogenesis of porcine reproductive and respiratory syndrome virus-infection in midgestation sows and fetuses. *Canadian Journal of Veterinary Research* 57(4), 262-268.

Christopher-Hennings J, Nelson EA, Hines RJ, Nelson JK, Swenson SL, Zimmerman JJ, Chase CCL, Yaeger MJ, Benfield DA (1995). Persistence of porcine reproductive and respiratory syndrome virus in serum and semen of adult boars. *Journal of Veterinary Diagnostic Investigation* 7(4), 456-464.

Christopher-Hennings J, Nelson EA, Nelson JK, Benfield DA (1997). Effects of a modified-live virus-vaccine against porcine reproductive and respiratory syndrome in boars. *American Journal of Veterinary Research* 58(1), 40-45.

Christopher-Hennings J, Nelson EA, Nelson JK, Rossow KD, Shivers JL, Yaeger MJ, Chase CCL, Garduno RA, Collins JE, Benfield DA (1998). Identification of porcine reproductive and respiratory syndrome virus in semen and tissues from vasectomized and nonvasectomized boars. *Veterinary Pathology* 35(4), 260-267.

Dee SA (1996). The porcine respiratory-disease complex - are subpopulations important? *Swine Health and Production* 4(3), 147-149.

Dee SA (1997). Elimination of PRRS virus - is it possible? *Large Animal Practice* 18(5), 36-38.

Dee S (2001). PRRS virus drags down performance. *National Hog Farmer* October 2001, 6-12.

Dee SA (2003). Approaches to prevention, control and eradication. In: Zimmerman J, Yoon KJ (Eds). *2003 PRRS Compendium: Second Edition*. P 119-130. Des Moines, National Pork Board.

Dee SA (2004). Elimination of porcine reproductive and respiratory syndrome virus by test and removal on 30 farms. *Swine Health and Production* 12, 129-133.

Dee SA, Joo HS (1994). Prevention of the spread of porcine reproductive and respiratory syndrome virus in endemically infected-pig herds by nursery depopulation. *Veterinary Record* 135(1), 6-9.

Dee S, Joo HS (1996). PRRS serology - a critical component of the diagnostic work-up. *Swine Health and Production* 4(2), 100-101.

Dee SA, Joo HS (1997). Strategies to control PRRS - a summary of field and research experiences. Veterinary *Microbiology* 55(1-4), 347-353.

Dee SA, Molitor TW (1998). Elimination of porcine reproductive and respiratory syndrome virus using a test and removal process. *Veterinary Record* 143(17), 474-476.

Dee SA, Philips RE (1999). Use of polymerase chain reaction (PCR) to detect vertical transmission of porcine reproductive and respiratory syndrome virus (PRRSV) in piglets from gilt litters. *Swine Health and Production* 7(5), 237-239.

Dee S, Joo HS, Pijoan C (1994). Control of porcine reproductive and respiratory syndrome virus transmission - handling infected seedstock. *Compendium on Continuing Education for the Practicing Veterinarian* 16(7), 927.

Dee SA, Joo HS, Polson DD (1996a). Improved performance of a large pig complex after sequential nursery depopulation. *Veterinary Record* 138(2), 31-34.

Dee S, Collins J, Halbur P, Keffaber K, Lautner B, Mccaw M, Polson D, Rodibaugh M, Sanford E, Yeske P (1996b). Control of porcine reproductive and respiratory syndrome (PRRS) virus. *Swine Health and Production* 4(2), 95-98.

Dee SA, Joo HS, Henry S, Tokach L, Park BK, Molitor T, Pijoan C (1996c). Detecting subpopulations after PRRS virus-infection in large breeding herds using multiple serologic tests. *Swine Health and Production* 4(4), 181-184.

Dee SA, Joo HS, Polson DD, Park BK, Pijoan C, Molitor TW, Collins JE, King V (1997a). Evaluation of the effects of nursery depopulation on the persistence of porcine reproductive and respiratory syndrome virus and the productivity of 34 farms. *Veterinary Record* 140(10), 247-248.

Dee SA, Joo HS, Polson DD, Marsh WE (1997b). Evaluation of the effects of nursery depopulation on the profitability of 34 pig farms. *Veterinary Record* 140(19), 498-500.

Dee SA, Joo HS, Park BK, Molitor TW, Bruna G (1998). Attempted elimination of porcine reproductive and respiratory syndrome virus from a seedstock farm by vaccination of the breeding herd and nursery depopulation. *Veterinary Record* 142(21), 569-572.

Dee SA, Molitor TW, Rossow KD (2000). Epidemiological and diagnostic observations following elimination of PRRS virus from a breeding herd of pigs by the test and removal protocol. *Veterinary Record* 146, 211-213.

Dee SA, Bierk MD, Molitor TW (2001). An evaluation of Test and Removal for the elimination of PRRS virus from 5 swine farms. *Canadian Journal of Veterinary Research* 65, 22-27.

Dee SA, Torremorell M, Rossow K, Mahlum C, Otake S, Faaberg K (2001). Identification of genetically diverse sequences (ORF 5) of porcine reproductive and respiratory syndrome virus in a swine herd. *Canadian Journal of Veterinary Research* 65(4), 254-260.

Dee S, Deen J, Rossow K, Wiese C, Otake S, Joo HS, Pijoan C (2002a). Mechanical transmission of porcine reproductive and respiratory syndrome virus throughout a coordinated sequence of events during cold weather. *Canadian Journal of Veterinary Research* 66, 232-239.

Dee S, Deen J, Rossow K, Wiese C, Eliason R, Otake S, Joo HS, Pijoan C (2002b). Mechanical transmission of porcine reproductive and respiratory syndrome virus throughout a coordinated sequence of events during warm weather. *Canadian Journal of Veterinary Research* 67, 12-19.

Dee S, Jacobson L, Rossow K, Pijoan C (2003). Developing a model to re-evaluate aerosol transmission of PRRSV. *Proceedings of 84th Conference of the Research Workers in Animals Diseases*. Chicago, USA, November 9 to 11, 2003, p 19.

Dee SA, Deen J, Jacobsen L, Rossow KD, Mahlum C, Pijoan C (2005). Laboratory model to evaluate the role of aerosols in the transport of porcine reproductive and respiratory syndrome virus. *Veterinary Record* 156, 501-504.

Dewey CE, Wilson S, Buck P, Leyenaar JK (1999). The reproductive performance of sows after PRRS vaccination depends on stage of gestation. *Preventive Veterinary Medicine* 40(3-4), 233-241.

Done SH, Paton DJ, White EC (1996). Porcine reproductive and respiratory syndrome (PRRS): a review, with emphasis on pathological, virological and diagnostic aspects. *British Veterinary Journal* 152(2):153-174.

Edwards S, Robertson I, Wilesmith J, Ryan J, Kilner C, Paton D, Drew T, Brown I, Sands J (1992). PRRS ("blue-eared pig disease") in Great Britain. *American Association of Swine Practitioners Newsletter* 4(4), 32-36

Drew TW, Lowings JP, Yapp F (1997). Variation in open reading frames 3, 4 and 7 among porcine reproductive and respiratory syndrome virus isolates in the UK. *Veterinary Microbiology* 55, 209-221.

Duan X, Nauwynck HJ, Pensaert MB (1997). Effects of Origin and State of Differentiation and activation of Monocytes/Macrophages on Their susceptibility to porcine reproductive and respiratory syndrome Virus (PRRS). *Archives of Virology* 12, 2483-2497.

Fano E, Pijoan C, Dee S (2005). Evaluation of the aerosol transmission of a mixed infection of Mycoplasma hyopneumoniae and porcine reproductive and respiratory syndrome virus. *Veterinary Record* 157, 105-108.

Fauquet CM, Mayo MA, Manioff J, Desselberger U, Ball LA (eds) (2005). *Virus Taxonomy: Classification and Nomenclature of Viruses*. Eighth Report of the International Committee on Taxonomy of Viruses. San Diego, Elselvier Academic Press.

FitzSimmons MA, Daniels CS (2003). Control in large systems. In: Zimmerman J, Yoon KJ (Eds). *2003 PRRS Compendium: Second Edition*. P 137-142. Des Moines, National Pork Board.

Forsberg R, Oleksiewwicz MB, Petersen AMK, Hein J, Bötner A, Storgaard T (2001). A molecular clock dates the common ancestor of European-type porcine reproductive and respiratory syndrome virus at more than 10 years before the emergence of disease. *Virology* 289(2), 174-179.

Frankeena K, Brouwer J, De Jong MF, Voets R, Verheijden J, Kominj RE (1992). PEARS effect on herd performance and risk analysis. *Proceedings of Internationational Pig Veterinary Society* 12, 118.

Frey ML, Landgraf JG, Schmitt BJ, Eernisse KA, Pearson JE (1995a). Recovery of the porcine reproductive and respiratory syndrome virus from tissues of slaughter weight pigs. *Proceedings of the Second International Symposium on Porcine Reproductive and Respiratory Syndrome (PRRS).* P 28.August 9-10, Copenhagen.

Frey ML, Mengeling WL, Lager KM, Landgraf JG, Eernisse KA (1995b). Survival of the virus of porcine reproductive and respiratory syndrome (PRRS) in swine carcasses. *NVSL Development Project Final Report* Pp1-9. Ames, USDA.

Gerlach JC (1974). *Climatographs of New Zealand*. Ruakura Agricultural Research Centre Research Bulletin 74-1.

Gillespie TB (2003). Control with modified-live (MLV) PRRS vaccine. In: Zimmerman J, Yoon KJ (Eds). *2003 PRRS Compendium: Second Edition*. P 147-150. Des Moines, National Pork Board.

Goldberg TL, Weigel RM, Hahn EC, Scherba G (2000a). Associations between genetics, farm characteristics and clinical disease in field outbreaks of porcine reproductive and respiratory syndrome virus. *Preventive Veterinary Medicine* 43(4), 293-302.

Goldberg TL, Hahn EC, Weigel RM, Scherba G (2000b). Genetic, geographical and temporal variation of porcine reproductive and respiratory syndrome virus in Illinois. *Journal of General Virology* 81(1), 171-179.

Halbur PG, Paul PS, Frey ML, Landgraf J, Eernisse K, Meng XJ, Lum MA, Andrews JJ, Rathje JA (1995). Comparison of the pathogenicity of two US porcine reproductive and respiratory syndrome virus isolates with that of the lelystad virus. *Veterinary Pathology* 32(6), 648-660.

Hermann JR, Muñoz-Zanzi CA, Roof MB, Burkhart K, Zimmerman JJ (2005). Probability of porcine reproductive and respiratory syndrome (PRRS) virus infection as a function of exposure route and dose. *Veterinary Microbiology* 110, 7-16.

Hirose O, Shibata I, Kudou H, Samegai Y, Yoshizawa S, Ono M, Nishimura M, Hiroike T, Kageyama K, Sakano T (1995). Experimental infection of SPF piglets with porcine reproductive and respiratory syndrome (PRRS) viruses isolated from two farms. Journal of Veterinary Medical Science, 57:6, 991-995.

Holck JT, Polson DD (2003). Financial impact of PRRS. In: Zimmerman J, Yoon KJ (Eds). *2003 PRRS Compendium: Second Edition*. P 51-58. Des Moines, National Pork Board.

Hopper SA, White ME, Twiddy N (1992). An outbreak of blue-eared pig disease (PRRS) in four pig herds in Great Britain. *Veterinary Record* 131, 140-144.

Horter DC, Pogranichnyy R, Chang C-C, Yoon K-J, Zimmerman JJ (2000). Evaluation of PRRS diagnostics in detecting persistently infected PRRSV carrier. *43rd Annual Meeting of American Association of veterinary Laboratory Diagnosticians.* Pp 92. 21-23 October, 2000.

Jensen T (1993). PRRS influence on productivity and economy. *Veterinärinformation* 3, 35-38.

Johnson BJ (2003). OSHA infectious dose white paper. *Applied Biosafety* 8(4), 160-165.

Kristensen CS, Bøtner A, Takai H, Nielsen JP, Jorsal SE (2004). Experimental airborne transmission of PRRS virus. *Veterinary Microbiology* 99, 197-202.

Lager KM, Mengeling WL (2000). Experimental aerosol transmission of pseudorabies virus and porcine reproductive and respiratory syndrome virus. *Proceedings of the American Association of Swine Practitioners* 2000, 409-410.

Lager KM, Mengeling WL, Brockmeier SL (1996). Effect of post-coital intrauterine inoculation of porcine reproductive and respiratory syndrome virus on conception in gilts. *Veterinary Record* 138(10), 227-228.

Lager KM, Mengeling WL, Brockmeier SL (1997a). Duration of homologous porcine reproductive and respiratory syndrome virus immunity in pregnant swine. *Veterinary Microbiology* 58(2-4), 127-133.

Lager KM, Mengeling WL, Brockmeier SL (1997b). Homologous challenge of porcine reproductive and respiratory syndrome virus immunity in pregnant swine. *Veterinary Microbiology* 58(2-4), 113-125.

Larochelle R, Magar R (1997). Evaluation of the presence of porcine reproductive and respiratory syndrome virus in packaged pig meat using virus isolation and polymerase chain reaction (PCR) method. *Veterinary Microbiology* 58, 1-8.

Le Potier M-F, Blanquefort P, Morvan E, Albina E (1997). Results of a control programme fro the porcine reproductive and respiratory syndrome in the French "Pays del la Loire" region. *Veterinary Microbiology* 55, 355-360.

MAF (1996). Porcine reproductive and respiratory syndrome suspected. *Surveillance* 23(2), 8.

MAF (1999). Teschen disease excluded. *Surveillance* 26(2), 18.

MAF (2001a). Feeding of Food Waste to Pigs. *MAF Discussion Paper No. 01/26* November 2001. Ministry of Agriculture and Forestry, Wellington, New Zealand.

MAF (2001b). Porcine exotic disease excluded. *Surveillance* 28(1), 11.

MAF (2003). Porcine reproductive and respiratory syndrome ruled out. *Surveillance* 30(3), 33.

Magar R, Robinson Y, Dubuc C, Larochelle R (1995). Evaluation of the persistence of porcine reproductive and respiratory virus in pig carcasses. *Veterinary Record* 137(22), 559-561.

Magar R, Larochelle R (2004). Evaluation of the presence of PRRS virus in pig meat and experimental transmission following oral exposure. *Canadian Journal of Veterinary Research* 68(4), 259-266.

McCaw M (2003). McRebel management. In: Zimmerman J, Yoon KJ (Eds). *2003 PRRS Compendium: Second Edition*. P 131-136. Des Moines, National Pork Board.

Meng XJ (2000). Heterogeneity of porcine reproductive and respiratory syndrome virus: implications for current vaccine efficacy and future vaccine development. *Veterinary Microbiology* 74(4), 309-329.

Mengeling WL, Lager KM, Vorwald AC (1994). Temporal characterization of transplacental infection of porcine fetuses with porcine reproductive and respiratory syndrome virus. *American Journal of Veterinary Research* 55(10), 1391-1398.

Mengeling WL, Lager KM, Vorwald AC (1995). Diagnosis of .porcine reproductive and respiratory syndrome. *Journal of Veterinary Diagnosis and Investigation* 7(1), 3-16.

Mengeling WL, Vorwald AC, Lager KM, Brockmeier SL (1996a). comparison among strains of porcine reproductive and respiratory syndrome virus for their ability to cause reproductive failure. *American Journal of Veterinary Research* 57(6), 834-839.

Mengeling WL, Vorwald AC, Lager KM, Brockmeier SL (1996b). Diagnosis of porcine reproductive and respiratory syndrome using infected alveolar macrophages collected from live pigs. *Veterinary Microbiology* 49(1-2), 105-115.

Mengeling WL, Lager KM, Vorwald AC (1998). Clinical effects of porcine reproductive and respiratory syndrome virus on pigs during the early postnatal interval. *American Journal of Veterinary Research* 59(1), 52-55.

Mengeling WL, Vorwald AC, Lager KM, Clouser DF, Wesley RD (1999). Identification and clinical assessment of suspected vaccine-related field strains of porcine reproductive and respiratory syndrome virus. *American Journal of Veterinary Research* 60(3), 334-340.

Molitor T, Shin J (1995). Porcine reproductive and respiratory syndrome in boars. *Proceedings of Allen D Lemen Swine Conference* 22, 101-102..

Moore C (1992). Biosecurity and minimal disease herds. In: Leman AD, Tubbs RC, eds. *Veterinary Clinics of North America: Swine Reproduction*. WB Saunders, Philadelphia, PA, 8, 461-474.

Mogollon JD, Rincon MA, Arbelaez G, Ruiz S, Rodrriguez V (2003). PRRS virus in Colombia. In: Zimmerman J, Yoon KJ (Eds). *2003 PRRS Compendium: Second Edition*. P 231-232. Des Moines, National Pork Board.

Morilla A, Gonzalex-Vega D, Diosdada F, Estrada E (2003). Seroepidemiology of PRRS in Mexico. In: Zimmerman J, Yoon KJ (Eds). *2003 PRRS Compendium: Second Edition*. P 261-263. Des Moines, National Pork Board.

Mortensen S, Madsen KS (1992). The occurrence of PRRS in Denmark. *American Association of Swine Practitioners Newsletter* 4(4), 48.

Mortensen S, Stryhn H, Søgaard R, Boklund A, Stärk KDC, Christensen J, Willeberg P (2002). Risk factors for infection of sow herds with porcine reproductive and respiratory syndrome (PRRS) virus. *Preventive Veterinary Medicine* 53, 83-101.

Motha J, Stark K, Thompson J (1997). New Zealand is free from PRRS, TGE and PRCV. *Surveillance* 24(1), 10-11.

Nielsen TL, Nielsen J, Have P, Baekbo P, Hoffjorgensen R, Bötner A (1997). Examination of virus shedding in semen from vaccinated and from previously infected boars after experimental challenge with porcine reproductive and respiratory syndrome virus. *Veterinary Microbiology* 54(2), 101-112.

Nodelijk G, DeJong MCM, VanNes A, Vernooy JCM, VanLeengoed LAMG, Pol JMA, Verheijden JHM (2000). Introduction, persistence and fade-out of porcine reproductive and respiratory syndrome virus in a Dutch breeding herd: a mathematical analysis. *Epidemiology and Infection* 124(1), 173-182.

Notkins AL, Scheele C (1963). Studies on the transmission and excretion of the lactic dehydrogenase agent. *Journal of Experimental Medicine* 118, 7-12.

OIE (2004a). *Handbook on Import Risk Analysis for Animals and Animal Products*. World Organisation for Animal Health. Paris.

OIE (2004b). *Porcine reproductive and respiratory syndrome*. In: Manual of Standards for Diagnostic Tests and Vaccines. 5th Edition. Pp 802-815. Office International des Epizooties. Paris.

OIE (2005). *Terrestrial Animal Health Code*. 14th Edition. World Organisation for Animal Health. Paris.

Otake S, Dee SA, Rossow KD, Moon RW, Pijoan C (2002a). Mechanical transmission of porcine reproductive and respiratory syndrome virus by mosquitoes, *Aedes vexans* (Meigen). *Canadian Journal of Veterinary Research* 66, 191-195.

Otake S, Dee S, Jacobson L, Torremorell M, Pijoan C (2002b). Evaluation of aerosol transmission of porcine reproductive and respiratory syndrome virus under controlled field conditions. *Veterinary Record* 150, 804-808.

Otake S, Dee SA, Rossow KD, Joo HS, Deen J, Molitor TW, Pijoan C (2002c). Transmission of porcine reproductive and respiratory syndrome virus by needles. *Veterinary Record* 150, 114-115.

Otake S, Dee SA, Rossow KD, Deen J, Joo HS, Molitor TW, Pijoan C (2002d). Transmission of porcine reproductive and respiratory syndrome virus by fomites (boots and coveralls). *Journal of Swine Health and Production* 10(2), 59-65.

Park BK, Joo HS, Dee SA, Pijoan C (1995). Evaluation of an indirect fluorescent IGM antibody-test for the detection of pigs with recent infection of porcine reproductive and respiratory syndrome virus. *Journal of Veterinary Diagnostic Investigation* 7(4), 544-546.

Pejsak Z, Markowskadaniel I (1997). Losses due to porcine reproductive and respiratory syndrome in a large swine farm. Comparative *Immunology Microbiology and Infectious Diseases* 20(4), 345-352.

Pejsak Z, Stadejek T, Markowskadaniel I (1997). Clinical signs and economic-losses caused by porcine reproductive and respiratory syndrome virus in a large breeding farm. *Veterinary Microbiology* 55(1-4), 317-322.

Pirtle EC, Beran GW (1996). Stability of porcine reproductive and respiratory syndrome virus in the presence of fomites. *Journal of the American Veterinary Medical Association* 208(3), 390-392.

Polson DD, Marsh WE, Dial GD (1990). Financial implications of mystery swine disease. *Proceedings of Mystery swine disease Meeting*. Livestock Convention Institute Denver, 1990, pp 8-28.

Prieto C, Castro JM (2005). Porcine reproductive and respiratory syndrome virus infection in the boar: a review. *Theriogenology* 63, 1-16.

Prieto C, Suarez P, Bautista JM, Sanchez R, Rillo SM, Simarro I, Solana A, Castro JM (1996). Semen changes in boars after experimental-infection with porcine reproductive and respiratory syndrome (PRRS) virus. *Theriogenology* 45(2), 383-395.

Rajic, CE Dewey, AE Deckert, RM Friendship, SW Martin, D Yoo (2001). Production of PRRSV-negative pigs commingled from multiple, vaccinated, serologically stable, PRRSV-positive breeding herds. *Journal of Swine Health and Production* 9(4), 179-184.

Roberts J (2003). Monitoring infected sow herds using genomic sequencing. In: Zimmerman J, Yoon KJ (Eds). *2003 PRRS Compendium: Second Edition*. P 103-116. Des Moines, National Pork Board.

Robertson IB (1992). Porcine reproductive and respiratory syndrome (blue-eared pig disease): some aspects of its epidemiology. *Proceedings of the Society for Veterinary Epidemiology and Preventive Medicine* 1992, 24-38.

Rossow KD, Bautista EM, Goyal SM, Molitor TW, Murtaugh MP, Morrison RB, Benfield DA, Collins JE (1994). Experimental porcine reproductive and respiratory syndrome virus-infection in one-week-old, 4-week-old, and 10-week-old pigs. *Journal of Veterinary Diagnostic Investigation* 6(1), 3-12.

Rossow KD, Collins JE, Goyal SM, Nelson EA, Christopher-Hennings J, Benfield DA (1995). Pathogenesis of porcine reproductive and respiratory syndrome virus infection in gnotobiotic pigs. *Veterinary Pathology* 32, 361-373.

Rossow KD, Benfield DA, Goyal SM, Nelson EA, Christopher-Hennings J, Collins JE (1996). Chronological immunohistochemical detection and localisation of porcine reproductive and respiratory syndrome virus in gnotobiotic pigs. *Veterinary Pathology* 33, 551-556.

Shibata I, Mori M, Uruno K, Samegai Y, Okada M (1997). In-vivo replication of porcine reproductive and respiratory syndrome virus in swine alveolar macrophages and change in the cell-population in bronchoalveolar lavage fluid after infection. *Journal of Veterinary Medical Science* 59(7), 539-543.

Shibata I, Mori M, Uruno K (1998). Experimental infection of maternally immune pigs with porcine reproductive and respiratory syndrome (PRRS) virus. *Journal of Veterinary Medical Science* 60(12), 1285-1291.

Sornsen SA, Zimmerman JJ, Polson DD, Roof MB (1998a). Effect of PRRS vaccination on average daily gain - a comparison of intranasal and intranasal-intramuscular administration. *Swine Health and Production* 6(1), 13-19.

Sornsen SA, Zimmerman JJ, Roof MB (1998b). Effect of various stocking methods and extra label prrs vaccination on average daily gain. Swine Health and Production 6(1), 7-11.

Sproule R, Fairley R (1998). Possible pantothenic acid deficiency in pigs. *Surveillance* 25(46-7.

Stevenson GW, van Alstine WG, Kanitz CL, Keffaber KK (1993). Endemic porcine reproductive and respiratory syndrome virus-infection of nursery pigs in two swine herds without current reproductive failure. Journal of Veterinary Diagnostic Investigation 5(3), 432-434.

Steverink P (2000). Transmission of porcine reproductive and respiratory syndrome virus through the oral uptake of infected porcine muscular tissue by naïve recipients. *Final report. Study 807-47041-00-99-09*. Lelystad, Netherlands.

Stone M (2004). PMWS in New Zealand: surveillance, diagnosis and risk factors for affected farms. *Dissertation in partial fulfilment of the requirements of MVS (Epidemiology),* Massey University. 150 pages.

Stone M, Kittelberger R (2004). Serological survey for PRRS during a response to PMWS. *Surveillance* 31(4), 15-17.

Swenson SL, Hill HT, Zimmerman JJ, Evans LE, Landgraf JG, Wills RW, Sanderson TP, Mcginley MJ, Brevik AK, Ciszewski DK, Frey ML (1994). Excretion of porcine reproductive and respiratory syndrome virus in semen after experimentally-induced infection in boars. *Journal of the American Veterinary Medical Association* 204(12), 1943-1948.

Teuffert J, Schluter H, Muller T (1998). Boar semen - a potential risk factor in the infectious cycle of the porcine reproductive and respiratory syndrome. *Deutsche Tierarztliche Wochenschrift* 105(9), 340-345.

Thacker E, Thacker B, Wilson W, Ackerman M (2003). Control with inactivated virus PRRS vaccine. In: Zimmerman J, Yoon KJ (Eds). *2003 PRRS Compendium: Second Edition*. P 151-155. Des Moines, National Pork Board.

Tirado SCM, Yoon KJ (2003). Antibody-dependent enhancement of virus infection and disease. *Viral Immunology* 16(1), 69-86.

Torremorell M, Pijoan C, Janni K, Walker R, Joo HS (1997). Airborne transmission of Actinobacillus pleuropneumoniae and porcine reproductive and respiratory syndrome virus in nursery pigs. *American Journal of Veterinary Research* 58(8), 828-832.

Torremorell M, Henry S, Christianson WT (2003). Eradication using herd closure. In: Zimmerman J, Yoon KJ (Eds). *2003 PRRS Compendium: Second Edition*. P 157-161. Des Moines, National Pork Board.

Trincado C, Dee S, Jacobson L, Rossow K, Otake S, Halvorson D, Pijoan C (2003). Studies about porcine reproductive and respiratory syndrome virus (PRRSV) transmission by aerosol and non-porcine vectors under filed conditions. *Proceedings of the American Association of Swine Veterinarians*. Orlando, USA, February 8 to 11, 2003, pp 503-505.

Trincado C, Dee SA, Rossow KD, Halvorson D, Pijoan C (2004). Transmission of porcine reproductive and respiratory syndrome virus by non-porcine vectors: A re-evaluation of Mallard ducks. *Veterinary Record* 154, 233-237.

USDA (1997). Prevalence of PRRS virus in the United States. Information Sheet. Veterinary Services, Animal and Plant Health Inspection Service, United States Department of Agriculture.

Van der Linden IFA, van der Linde-Bril EM, Voermans JJM, van Rijn PA, Pol JMA, Martin R, Steverink PJGM (2003). Oral transmission of porcine reproductive and respiratory syndrome virus by muscle of experimentally infected pigs. *Veterinary Microbiology* 97, 45-54.

Wallgren P (2000). Ethical, ecological and economic aspects of diseases among pigs in Sweden. *Svensk Veterinar Tidning* 52, 685-694.

Wang F I (1999). Minimal residues of porcine reproductive and respiratory syndrome virus in pig carcases and boar semen. *Proceedings of the National Science Council ROC(B)* 23, 167-174.

Wen-Bin Chung, Min-Wen Lin, Woon-Fa Chang, Moses Hsu, Ping-Cheng Yang (1997). Persistence of porcine reproductive and respiratory syndrome virus in intensive farrow-to-finish pig herds. *Canadian Journal of Veterinary Research* 61,292-298.

Wesley RD, Mengeling WL, Lager KM (1998). Prior infection of nursery-age pigs with porcine reproductive and respiratory syndrome virus does not affect the outcome of transmissible gastroenteritis virus challenge. *Journal of Veterinary Diagnostic Investigation* 10(3), 221-228.

Wills RW, Zimmerman JJ, Yoon K-J, Swenson SL, McGinley MJ, Hill HT, Platt KB, Christopher-Hennings J, Nelson EA (1997a). Porcine reproductive and respiratory syndrome virus: a persistent infection. *Veterinary Microbiology* 55, 231-240.

Wills RW, Zimmerman JJ, Yoon KJ, Swenson SL, Hoffmann LJ, Mcginley MJ, Hill HT, Platt KB (1997b). Porcine reproductive and respiratory syndrome virus - routes of excretion. *Veterinary Microbiology* 57(1), 69-81.

Wills RW, Zimmerman JJ, Swenson SL, Yoon KJ, Hill HT, Bundy DS, McGinley MJ (1997c). Transmission of PRRSV by direct, close or indirect contact. *Swine Health and Production* 5(6), 213-218.

Wilson W (2000). PRRS update. *Large Animal Practice* 21(1), 30-31.

Yoon KJ (2003). Virology. In: Zimmerman J, Yoon KJ (Eds). *2003 PRRS Compendium: Second Edition*. P 163-184. Des Moines, National Pork Board.

Yoon IJ, Joo HS, Christianson WT, Morrison RB, Dial GD (1993). Persistent and contact infection in nursery pigs experimentally infected with porcine reproductive and respiratory syndrome (PRRS) virus. *Swine Health and Production* 1(4), 5-8.

Yoon KJ, Wu LL, Zimmerman JJ, Hill HT, Platt KB (1996). Antibody-dependent enhancement (ADE) of porcine reproductive and respiratory syndrome virus (PRRSV) infection in pigs. *Viral Immunology* 9(1), 51-63.
Yoon KJ, Wu LL, Zimmerman JJ, Platt KB (1997). Field isolates of porcine reproductive and respiratory syndrome virus (PRRSV) vary in their susceptibility to antibody-dependent enhancement (ADE) of infection. *Veterinary Microbiology* 55(1-4), 277-287.

Yoon KJ, Zimmerman JJ, Chang CC, Tirado SMC, Harmon KM, McGinley MJ (1999). Effect of challenge dose and route on porcine reproductive and respiratory syndrome virus (PRRSV) infection in young swine. *Veterinary Research* 30(6), 629-638.

Zimmerman J (2003). Epidemiology and ecology. In: Zimmerman J, Yoon KJ (Eds). *2003 PRRS Compendium: Second Edition*. P 27-50. Des Moines, National Pork Board.

Zimmerman JJ, Yoon KJ, Pirtle EC, Wills RW, Sanderson TJ, McGinely MJ (1997). Studies on PRRS virus infection in avian species. *Veterinary Microbiology* 55, 329-336.

APPENDIX 1: QUANTITATIVE RELEASE ASSESSMENT

A model was developed to estimate the probability of pig meat harbouring infectious PRRS virus at the time of slaughter. The key probabilities in this model were as follows:

P1 the probability of a pig being infected prior to slaughter = 0.575

This estimate for P1 is based on the results from the NAHMS study discussed in section 4.1.1 of the risk analysis.

P2 the probability of infectious PRRS virus being present at the time of slaughter:

P2a the probability of infectious PRRS virus being present in oropharyngeal and tonsil tissue at the time of slaughter is determined by running a simulation on the following algorithm:

IF(age at slaughter > age when infected + persistence in oropharyngeal and tonsil tissue,0,1)

P2b the probability of infectious PRRS virus being present in meat (muscle, bone and regional lymph nodes) at the time of slaughter is determined by running a simulation on the following algorithm:

IF(age at slaughter > age when infected + duration of viraemia,0,1)

The variables in the algorithms for P2a and P2b are presented in Tables A1 to A4 together with their corresponding distributions in Figures A1 to A4.

V1 age at slaughter

The variable representing the age when a pig is slaughtered is based on the data in Table 14 (section 4.1.2.3). For each interval it is assumed that a pig has an equal chance of being slaughtered on any day within that interval. As a result a uniform distribution is applied to each interval (Table A1). The distribution for the age at slaughter is presented in Figure A1.

Table A1: The variable representing the age of a pig at slaughter.

	A	B	C
1	Age at slaughter (days)	Age	percent of pigs
2	< 160	ROUND(RiskUniform(150,160),0)	12.40%
3	160 - 165	ROUND(RiskUniform(160,165),0)	8.90%
4	166 - 180	ROUND(RiskUniform(166,180),0)	37.00%
5	181 - 209	ROUND(RiskUniform(181,209),0)	37.10%
6	>210	ROUND(RiskUniform(210,220),0)	4.60%
7	**Age at slaughter**	ROUND(RiskGeneral(150,220,B2:B6,C2:C6),0)	

Figure A1: A distribution of the age at slaughter. This distribution is obtained by running a simulation on cell B7 in Table A1.

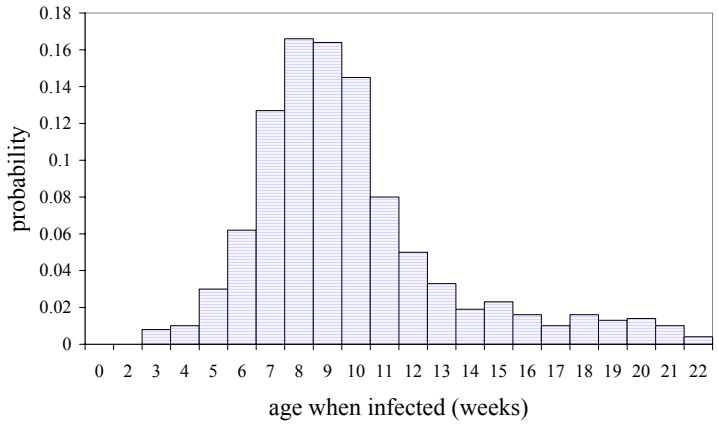
V2 age when infected

The variable representing the age when a pig is infected with PRRS virus is based on the data in Table 6 (section 4.1.2.1). The point prevalence estimate of viraemic pigs on a particular sampling occasion is likely to include some pigs that were viraemic on the day when the previous samples were collected as well as new cases that have arisen. Since it is not possible to differentiate between such animals, for the purposes of this analysis it is assumed that prevalence can be used as an approximate indicator of the relative probability of exposure to PRRS virus per fortnight The general distribution function in cell B in Table A2 automatically normalises the prevalence estimates in cells C2:C8, ensuring that the probability estimates are relative to each other (Figure A2).

Table A2: The variable representing the age when a pig becomes infected with PRRS virus.

	A	B	C
1	Age (weeks)	Age (days)	Prevalence of viraemic pigs
2	2	ROUND(RiskUniform(14,27),0)	3.3%
3	4	ROUND(RiskUniform(28,41),0)	7.8%
4	6	ROUND(RiskUniform(42,55),0)	55.6%
5	8	ROUND(RiskUniform(56,69),0)	64.4%
6	10	ROUND(RiskUniform(70,83),0)	20.0%
7	12	ROUND(RiskUniform(84,111),0)	5.6%
8	16	ROUND(RiskUniform(112,154),0)	5.6%
9	**Age when infected**	ROUND(RiskGeneral(14,154,B2:B8,C2:C8),0)	

Figure A2: A distribution of the age when a pig becomes infected with PRRS virus. This distribution is obtained by running a simulation on cell B9 in Table A2.



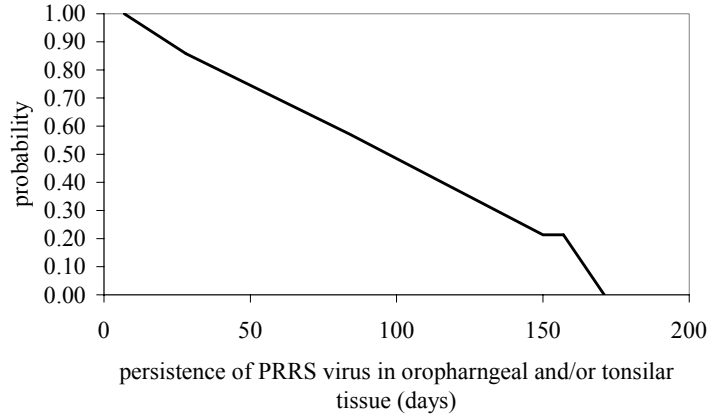
V3 persistence in oropharyngeal and tonsil tissue

The variable representing the persistence of PRRS virus in oropharyngeal and tonsil tissue is based on the survival data in Table 7 (section 4.1.2.2). In order to use a cumulative distribution function the survival data in column B needs to be converted to ascending cumulative probability values (cells C2:C7).

Table A3: The variable representing the persistence of PRRS virus in oropharyngeal and tonsil tissue.

	A	B	C
1	days post infection	survival	cumulative ascending
2	7	1	1-B2
3	28	0.86	1-B3
4	84	0.57	1-B4
5	150	0.21	1-B5
6	157	0.21	1-B6
7	171	0	1-B7
8	**Persistence in oropharyngeal and tonsil tissue**	ROUND(RiskCumul(7,171,A2:A7,C2:C7),0)	

Figure A3: A distribution of the persistence of PRRS virus in oropharyngeal and tonsil tissue. This distribution is obtained by running a simulation on cell B8 in Table A3.



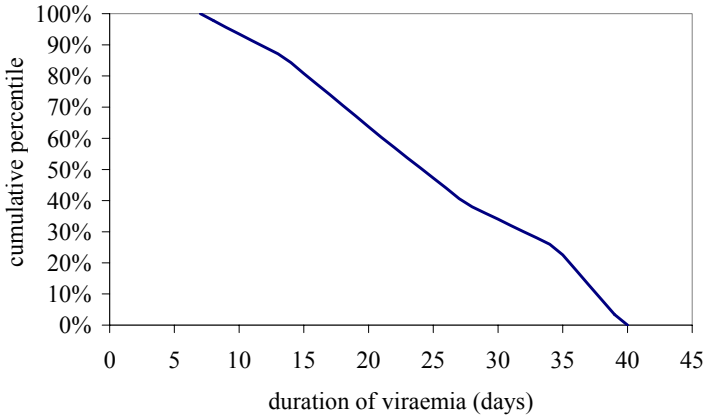
V4 duration of viraemia

The variable representing the duration of viraemia of PRRS virus is based on the survival data in Table 10 (section 4.1.2.2). In order to use a cumulative distribution function the survival data in column B needs to be converted to ascending cumulative probability values (cells C2:C7).

Table A4: The variable representing the duration of viraemia for PRRS virus.

	A	**B**	**C**
1	days post infection	Survival	cumulative ascending
2	7	1	1-B2
3	14	0.85	1-B3
4	21	0.61	1-B4
5	28	0.38	1-B5
6	35	0.24*	1-B6
7	40	0	1-B7
8	**Duration of viraemia**	ROUND(RiskCumul(7,40,A2:A7,C2:C7),0)	

Figure A4: A distribution of the duration of viraemia. This distribution is obtained by running a simulation on cell B8 in Table A4.



P3 the probability of a pig harbouring infectious PRRS virus in its tissues at slaughter:

P3 = P1 x P2

Model results

The model results, which are reported as expected values, are presented in Table A5.

Table A5: The results from running a model to estimate the probability of a pig harbouring infectious PRRS virus in its tissues at the time of slaughter.

P1: probability of a pig being infected prior to slaughter	**P2**: probability of infectious PRRS virus being present in the specified tissues at the time of slaughter	**P3**: probability of a pig harbouring infectious PRRS virus in its tissues at slaughter given that was infected prior to slaughter
P1 = 0.575	**P2a**: oropharyngeal or tonsil tissue P2a = 0.45	**P3a**: oropharyngeal or tonsil tissue P3a = P1 × P2a = 0.26
	P2b: meat P2b = 0.006	**P3b**: meat P3a = P1 × P2b = 0.003

These results indicate there is a moderate to high likelihood (0.26 or about 1 in 4) of infectious PRRS virus being present in oropharyngeal and tonsillar tissue at the time of slaughter, while there is a low likelihood (0.003 or about 1 in 300) of it being present in meat (muscle, bone and regional lymph nodes).