REPORT OF THE SCIENTIFIC EXPERT GROUP (SEG)

FINAL REPORT TO BIOSECURITY AUSTRALIA

21 NOVEMBER 2005
REPORT OF THE SCIENTIFIC EXPERT GROUP (SEG)

EXECUTIVE SUMMARY

The Scientific Expert Group (SEG) reviewed:

- evidence (written and oral) presented to the Federal Court in relation to post-weaning multisystemic wasting syndrome (PMWS) by each expert called to give evidence;
- the Federal Court’s view in relation to that evidence; and
- new international scientific knowledge and understanding of PMWS published since the completion of the IRA Report.

The outcome of this review is that the SEG considers that the level of quarantine risk of PMWS through imported pig meat has not changed from that assessed and documented in the IRA Report completed in February 2004.
INTRODUCTION

The Director of Animal and Plant Quarantine requested Biosecurity Australia (BA) to review whether the level of quarantine risk in relation to post-weaning multisystemic wasting syndrome (PMWS) had changed since the completion of the final import risk analysis report of pig meat in February 2004 (the IRA Report).

The Chief Executive of BA established a Scientific Expert Group (SEG) to:

- review new scientific knowledge and understanding of PMWS published since February 2004;  
- consider whether the matters reviewed would affect the conclusions and/or recommendations of the IRA Report in relation to PMWS;  
- provide a report to the Chief Executive of BA on the SEG’s conclusions and recommendations.

The Terms of Reference, including the SEG membership, objectives, scope and working procedures, are in the Attachment.

The SEG met on 31 August, 2 September, 20 September, 26–27 September, 19 October and by teleconference on 7 November 2005.
REVIEW NEW SCIENTIFIC KNOWLEDGE AND UNDERSTANDING OF PMWS PUBLISHED SINCE FEBRUARY 2004

The SEG reviewed scientific literature relevant to the understanding of PMWS published subsequent to the IRA Report. Although a significant number of scientific papers had been published subsequent to the IRA Report, the SEG considered that there was no profound change in the understanding of PMWS. The new information has been taken into account in assessing the criticisms of the IRA Report. Key elements are discussed below.

Epidemiology of PMWS

The distribution of PMWS worldwide remains the same as documented in the IRA Report. The disease has not been reported in Australia. Recent investigations into two possible cases of PMWS in South Australia and New South Wales concluded that Australia remains free from PMWS (Media release DAFF05/9D, 10 November 2005).

Differences in prevalence may reflect variation in the case definition of PMWS used by individual countries. While some countries follow Sorden’s definition (Sorden 2000) — characteristic clinical signs and histopathological lesions and detection of porcine circovirus type 2 (PCV2) antigen in tissues — based on an individual animal within a herd. Others, such as New Zealand, made their PMWS diagnosis on the basis of at least a 15% post-weaning mortality rate together with characteristic histopathological lesions and the demonstration of PCV2 antigen in tissues (Stone 2004). Denmark diagnoses the disease in a herd based on histopathology and demonstration of PCV2 antigen in pigs with or without clinical signs indicative of PMWS and regardless of the number of animals affected (Vigre et al. 2005).

The prevalence of PMWS is poorly recorded as generally the disease is not notifiable. Individual studies and data collation suggest a herd prevalence of up to 20% in the UK (Gresham and Thomson 2001) although recent information suggests that over 70% of pig farms in England and Scotland have been affected by PMWS at some stage (DEFRA 2005). Other reports include a prevalence of 4–21% in the USA (USDA 2002), 15–50% in the Netherlands (de Jong et al. 2003), less than 1% in Sweden (Wallgren et al. 2004) and about 4% in Denmark (Vigre et al. 2005). The herd prevalence in Ireland, Norway, France, Spain and Canada are unreported although it is believed to be widespread. There appear to be few reports of the disease in Belgium (Meerts et al. 2004), a country surrounded by PMWS.

The distribution of PMWS does not necessarily parallel the distribution of PCV2; neither in Denmark where PCV2 is widespread and PMWS prevalence is just 4%, nor in Australia where PCV2 is present but PMWS is not diagnosed.
Various studies report morbidity rates on farm of 5–30% (Rodriguez-Arrioja et al. 2002; Sorden 2000). The case mortality rate may reach over 80% (Harding et al. 1998). Over the period that clinical signs are evident, the overall mortality rate is variable but may be quite low. In Denmark, mortality rates on PMWS-affected farms were within the range of 4 to 18% (Hassing et al. 2002). The mortality rates on affected farms in Sweden were 2–9% (Wallgren et al. 2004) and 9% in the UK (Armstrong and Bishop 2004).

Although it seems that PMWS has been present in Denmark since 1989 (Vigre et al. 2005), the disease affected only about 4% of herds between 2001 and 2003. It is difficult to explain why the disease did not spread more widely between 1989 and 2001 unless some new co-factor allowed the disease subsequently to ‘break out’. Vigre et al. (2005) concluded that PMWS was caused by the introduction of a new, as yet unidentified, pathogen into the Danish pig industry.

In Sweden, PMWS is a very mild disease with 16 farms diagnosed as infected in the first year after the initial diagnosis. A PCV2 isolate was recovered in 1993 before PMWS was first reported in Sweden in 2004. Experiments conducted on the isolate in Ireland demonstrated that this isolate was as pathogenic as other isolates from other PMWS cases when evaluated in an experimental model (Allan et al. 2003). Opinion in Sweden (Wallgren et al. 2004) suggests that a new microbe is not involved with spread of the disease. Although there is some suggestion that PCV2 might be transmitted in semen, there is no suggestion that PMWS in Sweden was transmitted by this route (Wallgren et al. 2004).

In New Zealand, PMWS has been diagnosed in only 14 herds (positive or suspect) since the disease was first diagnosed in late 2003 (Loth and Stone 2005). It has even been suggested that the disease may have been present since about 2000 (Stone 2004).

Although PCV2 appears to be an organism essential in the aetiology and pathogenesis of PMWS, opinion about other interacting or contributing pathogens and factors is divided. Some, for example, favour an ‘Agent X’, necessarily and invariably involved but others believe multiple co-factors, which may include non-infectious and infectious agents, may be implicated. The literature includes a range of studies proposing the association of many different co-factors with PCV2 in the aetiology of PMWS (Allan et al. 2004; Ellis et al. 2004; Ha et al. 2005).

In 2004, Kristensen et al. reported transmission of PMWS from pigs originating on affected farms to pigs sourced from unaffected farms by placing them in direct contact. Clinical signs, typical PMWS histopathological lesions and demonstration of PCV2 antigen were all evident in affected pigs. However, the SEG is of the view that the conclusion that clinical PMWS was transmitted in this preliminary study remains unsubstantiated until repeated in further experiments with adequate control groups.
EVIDENCE PRESENTED TO THE FEDERAL COURT

Overview

In reviewing the evidence presented to the Federal Court, the SEG identified various categories of criticisms of and/or comment on assumptions that were made during the IRA Panel's assessment or on procedures used. These categories may be summarised as follows:

(a) The aetiology of PMWS, in particular:
   (i) whether Agent X was properly considered;
   (ii) use of PCV2 as a proxy for the ‘trigger’ for PMWS.

(b) The failure to conduct studies to examine the transmission of PMWS through ingestion of 'infected' meat or to examine whether viruses existed in pig meat muscle.

(c) Methodological concerns, particularly:
   (i) use of a risk assessment model in the IRA Panel's decisions;
   (ii) the use of expert opinion;
   (iii) use of the 50th percentile rather than the 95th percentile, to summarise risk distributions;
   (iv) use of ‘annual risk’, that is annual volume of trade.

(d) Criticisms directed to the various steps within the risk analysis.

(e) Criticisms aimed at the effectiveness of risk management measures.

The above categories have been defined for ease of presentation. The SEG had regard to each individual criticism arising from both the judgment of the Federal Court and the expert evidence heard in the Federal Court proceedings.
Aetiology

Whether ‘Agent X’ was properly considered

There is still debate and diversity of opinion on the aetiology of the disease. It is generally accepted that PCV2 is responsible for the clinical disease and pathological lesions observed in typical cases of the syndrome. It also appears that one or more co-factors are necessary to trigger PCV2 pathogenicity, and the manifestation of PMWS, by inducing some form of immune modulation. Alternatively, it could be that only particular strains of PCV2 are responsible for the syndrome.

The co-factor(s) may be infectious or non-infectious. If infectious, they could be known infectious agents. Porcine reproductive and respiratory syndrome (PRRS) virus and porcine parvovirus are examples of agents that can be used to experimentally reproduce the disease, in the presence of PCV2. If non-infectious, they could include management factors that induce stress, vaccination regimens, or genetic factors that have rendered pigs more susceptible to PMWS.

The SEG was not persuaded by the argument presented in evidence before the Federal Court that there is a single unidentified infectious agent (‘Agent X’) that is invariably implicated, together with PCV2, in causing PMWS. While in some circumstances spread of the disease has appeared to be contagious (for example, in Denmark and the UK (Gresham and Thomson 2001; Vigre et al. 2005), this does not necessarily imply the presence of a novel agent. In other circumstances, including Sweden, the epidemiological evidence does not support contagious spread (Wallgren et al. 2004).

There is a growing consensus that any strain of PCV2 can cause PMWS in the presence of one or more co-factors (Grierson et al. 2004; Hasslung et al. 2005). There is some evidence that strains of PCV2 isolated from unaffected countries (for example, Sweden before the disease was identified) or unaffected farms, can cause PMWS. However, there is also molecular evidence that there is some variability in genomic sequence between PCV2 isolates and it is not yet known if there are differences in pathogenicity between them (Wen, et al., 2005). Thus, the hypothesis that strains of PCV2 may differ in pathogenicity has not been disproved.

The SEG considered that the best working hypothesis is that PMWS is caused by PCV2 in the presence of immunomodulation induced by one or a combination of either infectious or non-infectious co-factors. The possibility of particular pathogenic strains of PCV2 (that are not currently in Australia) being necessary cannot be discounted. Infectious co-factors could include known or novel agents not yet present in Australia and this possibility, together with exotic pathogenic strains of PCV2, are the scenarios in which introduction of the disease into Australia would represent a quarantine concern for importation of pig meat. Should non-infectious co-factors be demonstrated to be the trigger for PMWS, and PCV2 strains not to differ in pathogenicity, the importation of pig meat would clearly not represent a quarantine concern.

The SEG is satisfied that the possibility of one or more unknown agents (‘Agent X’), and its implications for quarantine risk, was adequately considered in the IRA Report.
Use of PCV2 as a Proxy

The IRA Panel used PCV2 as a surrogate for the necessary co-factor to trigger PMWS, when assessing the quarantine risk associated with it. The SEG considers that this was appropriate because:

- the identity of a contagious element of PMWS (at risk of potentially being transmitted in pig meat) is unknown but might be a pathogenic strain of PCV2 exotic to Australia;
- PCV2 is both a highly stable virus (more so than most other viruses) and known to persist in pigs for considerable periods after recovery from clinical or subclinical disease;
- PCV2 infection is often subclinical and hence infected pigs would not be detected and removed at ante mortem of post mortem inspection; and
- it would reflect an appropriately conservative approach in consideration of quarantine risk.

The SEG recognises that in using PCV2 as a proxy the IRA Panel acted very conservatively. It would have been open to the IRA Panel to use expert knowledge of the pathogenesis of viral infections for particular steps in the risk assessment, such as R2, R4 and L2 (this is explained in more detail under the individual steps in the Risk Assessment section).

Experimental transmission

The SEG considers that at this stage a scientific experiment to determine whether transmission of PMWS is possible by feeding meat would produce results that are uninterpretable because there is no robust experimental challenge model with adequate controls available. Moreover, such a model would require a large number of pigs to be statistically valid. It would also require reliable positive (known to be infected) and negative (known to be not infected) control groups, and this is not possible until the aetiology of PMWS is clarified. The SEG considered that the quarantine risk of PMWS associated with the importation of pig meat could be assessed without these experimental studies.
Methodology of the risk assessment process

Use of a risk assessment model in the IRA Panel's deliberations

The SEG considers that the use the IRA Panel made of a spreadsheet model during its risk assessment of various diseases was suitable because the model:

- facilitated the expression of expert opinion by translating the IRA Panel's judgments on each of the individual steps of the pathway to an overall assessment which was logically consistent with such judgement;
- provided a structured guide to assessing risk and added discipline to the assessment process.

In the SEG’s view this ensured that an orderly approach was used to explore and scrutinise the components of risk. The SEG recognises that, in the case of PMWS, precise data were either not available or were considered of limited reliability so that the IRA Panel would have had to form an opinion based on its members’ collective judgement according to their knowledge and experience of viral pathogenesis.

The use of expert opinion

The SEG considers that the use of distribution bands to represent expert opinion was appropriate for the following reasons:

- expert opinion was formed at each step in the model through scrutiny of published studies and other reports and consideration of unpublished information from identified experts. The SEG notes that the IRA Panel met frequently to discuss and evaluate information, and to exchange views and opinions. A consensus decision on a value for each step in the assessed risk was made (if it had not been, then this would have been documented in the IRA Report) and this led to an assessment of overall risk as calculated by the simulation model;
- the elicitation of expert opinion was a complex process that allowed for full expression of the views of individual experts while providing a structured approach through the use of the model. As discussed above, precise data were generally not available and thus the SEG considers it reasonable to use distribution bands to reflect expert opinion. The SEG notes that in the one case where more precise information was available (for R3.2), a specific range was used rather than a semi-quantitative expression of expert opinion.
Use of the 50th percentile

The SEG considered that the use of the 50th percentile, rather than the 95th percentile, was appropriate. The SEG considers that:

- the choice of the 50th percentile provides a balanced view of risk, which is consistent with the way in which it was used;
- the IRA Panel incorporated a level of conservatism in the risk assessment (see, for example, earlier discussion on use of PCV2 as a proxy). Accordingly, it would have been inappropriate to force a further over-estimation of real risk by using the 95th percentile when evaluating risk.

Use of ‘annual risk’

The SEG considers that the use of annual risk — that is, assessing likelihood of at least one exposure in a year — was appropriate. This is for the following reasons:

- it is a realistic and accepted rate in which to express and assess risk. It does not force the IRA Panel to try to look too far into the future when new information might be available or risks for specific items might have significantly changed;
- the level of risk continues to be monitored once the IRA Report is complete. It is stated in the IRA Report that emerging information will continue to be monitored and the risk re-assessed as appropriate;
- it was the likelihood of entry and exposure that was considered on an annual basis. Consequences of any disease introduction and establishment or spread were assessed over an indefinite period.

The SEG considered it was not appropriate to estimate a 10-year risk (as had been presented in expert evidence before the Federal Court) by simply extrapolating results from the pig meat model, which were based on annual rates, because:

- in the assessment of risk, wherever the IRA Panel had to deal with unknown or unreliable data, it is clear that a conservative estimate of risk was made;
- it is probable that published information on disease incidence, which may be used in arriving at the risk estimate, often represents the ‘extreme case’;
- simply extrapolating from figures derived using annual volumes of trade to statements of risk 10 years into the future inevitably results in amplification of the over-estimate of risk built in by the conservatism of the original estimations;
- if it was decided to estimate risk over a 10-year time frame then the IRA methodology would need to be amended.
Risk Assessment of PMWS

Release assessment

R1

The SEG agrees with the IRA Panel's estimate of ‘moderate’ for R1 for the following reasons:

- since publication of the IRA Report, further data have become available for the herd prevalence of PMWS in different countries. Some of the estimates include: Denmark 4% (Vigre et al. 2005), Sweden 1% (Wallgren et al. 2004) and New Zealand 4% (Stone 2004);
- the SEG believes that it is sensible to discount individual isolated cases of disease as not representative of a contagious process. This would reduce the herd prevalence estimate where an individual case definition had been used;
- the SEG recognises that estimates of the prevalence of PMWS are often based on very weak data. Also, data for many countries are simply not available. PMWS is rarely categorised as a notifiable disease so that there is no strong basis for establishing a prevalence estimate and there is likely to be under-reporting of the disease.

Hence, the SEG considers it appropriate for the IRA Panel to have used an estimate that incorporated the highest reported prevalence.

The SEG notes that the IRA Report stated that individual countries could supply prevalence data to BA and the simulation could be re-run to refine the assessment for a specific country.

R2

The SEG considers that the IRA Panel's estimate of ‘moderate’ for R2 was conservative. The SEG notes that by adopting the working hypothesis proposed above under Aetiology, R2 could now be assessed as ‘low’ for the following reasons:

- it is likely that all strains of PCV2, including the Australian strain, can be triggered with appropriate co-factors resulting in PMWS;
- the necessary involvement of one or more presently unidentified, exotic infectious co-factor(s) is contestable;
- it is possible that some herds affected with PMWS do not have co-factors of quarantine concern because:
  - the co-factors are agents endemic to Australia;
  - the co-factor is exotic PRRSV, which would be managed by measures in place for that disease; or
  - they are non-infectious co-factors that would not be transmitted in pig meat;
- if the co-factor (or one of them) involved in a particular herd is an exotic agent, it is still not necessarily present in a particular pig at the time of slaughter.
The SEG re-ran the pig meat model, as originally used by the IRA Panel with this revised value, in combination with other revised values (see further below). The unrestricted risk of PMWS was still assessed as ‘low’, as had been assessed by the IRA Panel.

R3.1, R3.2

The SEG agrees with the IRA Panel’s estimates for these steps.

R4

The SEG agrees with the IRA Panel’s estimate of ‘moderate’ for R4.

It was argued during the Federal Court proceedings that it was unreasonable for the IRA Panel to make assumptions about the reduction in infectivity of a carcass by the dressing process, when the identity of the infectious agent is unknown. The SEG disagrees with this criticism. Whether the infectious agent is a pathogenic strain of PCV2 or another agent, it is likely to be present in highest concentration in affected organs and, since the effect appears to be an immunomodulating one, this is likely to include those lymphoreticular tissues (including spleen, mesenteric lymph nodes) that are mostly excluded from the dressed carcass. Further, the characteristic lesions associated with PMWS are in the lymphoreticular tissues.

R5, R6

The SEG agrees with the evidence provided to the Court on the assessment of these two steps of the release assessment. The SEG recognised that if PCV2 was used as a proxy then it would be valid to raise R5 and R6 from ‘high’ to 0.99–1 due to the highly stable nature of the virus. Given the SEG’s view on the likely aetiology of PMWS, this higher range represents a conservative approach.

As noted above, the SEG re-ran the pig meat model, as originally used by the IRA Panel with these (and other) revised values. The unrestricted risk of PMWS was still assessed as ‘low’, as had been assessed by the IRA Panel.
Exposure assessment

In light of criticism that the IRA Panel may have underestimated the number of backyard pigs that might be exposed to waste units, the SEG tested the sensitivity of the exposure assessment by increasing the figure used for backyard pigs by 50%. On re-running the simulation model with this value and other revised values, the unrestricted risk of PMWS was still assessed as ‘low’, as had been assessed by the IRA Panel.

L1

The SEG considers that equating L1 with the outcome of the release assessment as presented in the IRA Report is conservative. An assertion was made during the Federal Court proceedings that because meat from different carcasses was combined during the manufacture of small goods there is an increased likelihood that waste from a particular product might contain infectious agent. However, the SEG believes this to be offset by the conservative assumption that infection, if it were present, would be likely to affect all carcass cuts equally and, by extension, all waste units derived from those cuts.

It should be noted that in practice it is more likely that infected carcasses would contain non-uniform distribution of infectious agent that, on dilution by mixing with uninfected meat, would result in fewer waste units capable of causing infection thereby reducing L2. This factor was not included as the IRA Report maintained a conservative approach.

L2–L5

The SEG agrees with the IRA Panel's assessment of L2–L5 as documented in the IRA Report.

Consequence assessment

The SEG is of the view that criticisms of consequence assessment, as it was undertaken by the IRA Panel, were either:

- based on a misinterpretation of the IRA methodology in this regard; or
- otherwise misconstrued.

The SEG considers that Table 8 of the IRA Report (in which the rules for the assessment of direct or indirect consequences on a national scale are set out) did not limit the IRA Panel's assessment of the consequences of any outbreak of a disease to no more than ‘minor’ at the State level. If this assertion were correct then the consequences of the establishment of any of the diseases considered would also be ‘minor’. However, the consequences of establishment of some of the other diseases were assessed as more than ‘minor’ at the State level (e.g. foot-and-mouth disease, classical swine fever, African swine fever, swine vesicular disease).
The purpose of the consequences assessment is to assess the likely ‘impact’ of a disease on the Australian nation as a whole. Therefore, in considering PMWS the IRA Panel commenced its consideration at a national level. On the data available to it, none of the indirect and direct consequences associated with PMWS was considered to be discernible at that level. The IRA Panel then considered such indirect and direct consequences at progressively lower levels (i.e. State then region or local level). It is important to note that in the IRA Report impact is a different issue from the extent of spread of disease: a disease may have serious consequences at the national level despite occurring in only a small area.

It has been estimated that PMWS causes losses of some €600–900 million in the EU (Armstrong and Bishop 2004; Allan cross examination, Federal Court Proceedings, 2004). It has been estimated that an epidemic of PMWS in Australia could add 15% to the cost of pig meat production in affected herds (Media release, Australian Pork Limited MR140, 7 June 2005). Recently, it has been estimated that if PMWS were endemic in Australia, and, on average 20 per cent of pig farms had active PMWS, the impact on the national herd would be to increase the cost of production by 4.4 per cent. This would equate to about $26 million in additional production costs each year with an economy-wide impact estimated to be a decline in GDP of approximately $85 million per year (Estimates of PMWS impact on pig production sector costs of production, ABARE, November 2005).

The SEG reviewed the data on the economic consequences of PMWS, as detailed in the IRA Report, and subsequent data, including the above, on the possible economic impact. It is satisfied that the consequence assessment of ‘minor’ at the State level was appropriate.

In making these statements the SEG recognises, as did the IRA Panel, that the impact of PMWS could be significant for individual pig producers and the Australian pig industry, but notes that its impact on the Australian community as a whole would be limited, especially as an outbreak of PMWS would have no effect on international trade.

The SEG notes that there is no new evidence available on which it would assess consequences at a higher level. In fact, the contrary appears to be the case. More recent information from countries with well managed industries, similar to Australia’s, indicates that the impact and spread of PMWS has been less than was originally reasoned in the IRA Report, from information available at the time.
Effectiveness of Risk Management Measures

The SEG considers that the risk management measures recommended in the IRA Report were sufficient to reduce the unrestricted risk of PMWS from ‘low’ to a restricted risk of ‘very low’, for the following reasons:

- if an ‘Agent X’ is involved in triggering PMWS, it is unlikely to be in high concentration in muscle tissue. There is no record of lesions (gross or microscopic) being observed in the muscle of PMWS-affected pigs. Lesions in tissues generally result where virus accumulates in high concentrations;
- if ‘Agent X’ is in lymphatic tissue, then the majority would be removed as a result of risk management measures (in particular modified carcass dressing). Based on current knowledge of infectious agents and the pathological effects recognised in PMWS-affected pigs, the SEG considers that any ‘Agent X’ is likely to be located in these tissues. In particular:
  - it appears that the effect of co-factors in the expression of PMWS is an immunomodulating one, and therefore the agent responsible is most likely to be located in the reticuloendothelial system\(^1\); and
  - transmission of PMWS appears to be oral or oronasal and therefore an infectious co-factor is most likely to locate in the lymph nodes draining alimentary and respiratory systems, as commonly occurs with other viral infections transmitted by such means. Such lymphatic tissue would be removed in the modified carcass dressing specified under the proposed risk management measures;
- if only a pathogenic strain of PCV2 is responsible for triggering PMWS, then the above arguments also apply, as PCV2 has been demonstrated to be located and cause lesions in lymph nodes and is considered to be transmitted oronasally. Thus the removal of lymphatic tissue with modified carcass dressing would reduce the risk of a carcass being infected;
- whether a pathogenic strain of PCV2 or an unknown infectious co-factor is responsible for triggering PMWS, reduction of waste (which comes about by requiring all imported pig meat to be cooked, or cured long-term, and deboned) would ensure that there is less waste that might contain PCV2 or an unknown infectious co-factor and hence reduce exposure opportunities;
- in the case of an unknown infectious co-factor, cooking and long-term curing measures are likely to reduce concentrations of the infectious agent, thus reducing the number of infectious waste units. This consideration was not included in the original modelling as the IRA Report maintained the conservative approach of using PCV2 (which is highly heat-resistant) as a surrogate.

The combined risk management measures outlined in the IRA Report (reduction in volume of waste, deboning and removal of head and neck and major peripheral lymph nodes) when inserted in the revised model as discussed above, meets Australia’s Appropriate Level of Protection.

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\(^1\) Part of the immune system — located in lymph nodes, spleen, liver and bone marrow — comprising cells that engulf and destroy foreign substances (including bacteria and viruses).
THE SEG’S CONCLUSIONS

The SEG concludes that none of the criticisms raised in the Federal Court proceedings, nor any of the new evidence it reviewed, affects the conclusions and recommendations of the IRA Report in relation to PMWS.

The SEG concludes that:

- the level of quarantine risk of PMWS in imported pig meat has not changed from that described in the IRA Report and the risk management measures recommended in the Report adequately address this risk;
- Biosecurity Australia should continue to monitor developments in knowledge of PMWS and use significant new information to review the risk of PMWS in imported pig meat.
REFERENCES


Introduction

1. Biosecurity Australia has been requested by the Director of Animal and Plant Quarantine (the Director) to review whether the level of quarantine risk in relation to post-weaning multisystemic wasting syndrome (PMWS) has changed since the completion of the final import risk analysis report of pig meat in February 2004 (the IRA Report).

Background

2. In the course of a recent Federal Court action — Australia Pork Limited v Director of Animal and Plant Quarantine (the APL decision) - the science underpinning the IRA Report was questioned by some of the expert evidence presented to the Court, leading the Judge to criticise aspects of the risk assessment and management measures that had been recommended in relation to PMWS.

3. The APL decision is the subject of an appeal, but in the meantime the Director has requested that Biosecurity Australia undertake a review of the work done in relation to PMWS by the risk analysis panel (the IRA Panel) that prepared the IRA Report in the light of expert evidence presented to the Court and informed by the Judge's criticisms outlined above.

4. In undertaking the review, Biosecurity Australia will take account of any international learning about PMWS since the IRA Report was prepared and determine whether the level of quarantine risk in relation to PMWS requires reconsideration and, if so, how and to what extent.

5. The Chief Executive of Biosecurity Australia has determined that an expert group should be formed (the Scientific Expert Group — SEG) to inform the review by Biosecurity Australia to assist it provide any additional advice to the Director that may be required. The SEG will comprise a range of experts who can provide technical and scientific contributions to the review, including a knowledge of issues identified in the IRA Report.

SEG Objectives and Scope

6. The task of the SEG is to:
   a) Review:
      i. evidence (written and oral) presented to the Federal Court in relation to PMWS by each expert called to give evidence;
      ii. the Federal Court’s view in relation to that evidence; and
iii. any new international scientific knowledge and understanding of PMWS published since the completion of the IRA Report.

b) Consider whether the matters reviewed above would affect the conclusions and/or recommendations of the IRA Report in relation to PMWS.

c) Provide a report to the Chief Executive of Biosecurity Australia on the SEG's conclusions and recommendations.

SEG Membership

7. Membership of the SEG is attached.

Working Procedures

8. The SEG will be supported by:

- the Bureau of Rural Sciences of the Department of Agriculture, Fisheries and Forestry for any modelling issues arising from the APL decision that the SEG may require assistance on;

- the Product Integrity and Animal and Plant Health division of the Department of Agriculture, Fisheries and Forestry for any advice that the SEG may require relating to animal health, epidemiology and management issues;

- Biosecurity Australia’s legal advisers on legal issues arising from the Court proceedings and decisions; and

- other scientific technical and expert advice on particular issues that might be sought by the SEG.

9. Secretariat support for the SEG will be provided by Biosecurity Australia.

Deadline

10. The advice from the SEG is to be provided to the Chief Executive of Biosecurity Australia no later than 30 September 2005.

John Cahill
Chief Executive
Biosecurity Australia
SEG Membership

The SEG will comprise:

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<th>Name</th>
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<tr>
<td>Professor Colin Wilks (Chair)</td>
<td>Professorial Fellow (Veterinary Public Health and Virology), University of Melbourne</td>
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<td>Dr Ross Cutler</td>
<td>Consultant Specialist Veterinarian, Victoria</td>
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<td>Dr Tony Forman</td>
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