A review of *Mycobacterium paratuberculosis* and its control in South Australia

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The cover image is of an impression smear (Ziehl-Neelsen stain) of the lining of the ileum in a 2 year-old South Australian Merino sheep with multibacillary OJD. The abundant *M* *ptb* organisms are stained red against the blue-stained intestinal cells.
TERMS OF REFERENCE

Purpose
1. To provide advice to Biosecurity SA and the South Australian Sheep Advisory Group on the most effective future disease management plan for Ovine Johne’s Disease (OJD).

2. To provide a current summary of the scientific knowledge of this disease, which can be used to make evidence based decisions for disease management at a low disease prevalence level. The review is to provide a summary of the epidemiology of the disease and comment on a management strategy to maintain low disease prevalence in South Australia.

Review parameters
1. Consult key subject matter specialists, including those with practical experience to develop a snapshot of the history of Johne’s disease in sheep in South Australia.

2. Review available published scientific papers on the epidemiology and ecology of Johne’s disease in sheep. Specifically, but not exhaustively, the review should address the transmission, incubation periods, disease occurrence (within and between flock prevalence) and shedding of bacteria and the impact of vaccination to identify the greatest risk periods and pathways to address in a control program. The scientific review should also provide comment on the current published science on links between Johne’s and Crohn’s disease.

3. Comment on both the current control program as well as the proposed program and the likely effectiveness of each of these programs. This should also include any limitations of current surveillance and testing. Comment on the SA industry’s preference that Johne’s in sheep remain a regulated disease.

4. Meet with the SA Sheep Advisory Group (and SA OJD committee) to consider industry views on the disease and the Chief Veterinary Officer before making recommendations for future management of the disease

5. Provide recommendations for a future path forward for the South Australian OJD control program based on science included in the review process.

All scientific facts, conclusions and comments should be framed according to the environment and disease conditions found in South Australia (ie low to high variable rainfall; low prevalence of disease).

Deliverables
The report is to be written in plain language that can be understood by producers who may not have a scientific background.
FOREWORD

Previous drafts of this report have been reviewed by staff of PIRSA and members of SASAG and the SA OJD Advisory Committee. In response to comments received in those reviews, several changes and additions to the report have been made for this, the final report. The recommendations, however, have not changed.

These changes include, but are not limited to the following:

- Clarification of the endemicity of OJD in SA (Appendix B).
- Expansion of the discussion around vaccination and the sale price of vaccinated ewes.
- Clarification of the estimates of the numbers of undetected but infected flocks which may be ‘missed’ by abattoir surveillance – please note that this estimate was intended to be indicative only and not a precise prediction.
- Further commentary of the current control program, the PIRSA proposed program and an approach proposed in this report (Appendix C).

All comments and suggestions have been considered and, in some cases, further changes have been made.

The principle recommendation in this report is for a de-regulation of OJD and a move towards individual responsibility for biosecurity. There is not one single compelling factor leading to this conclusion but a number of factors which have led me to consider this to be the best way forward. These reasons are listed in Section D-2 and are based on an understanding of the disease informed by the published science which is reviewed in this report. The conclusion does not infer that the program to date has been unsuccessful or inappropriate. In fact, it is likely that the relatively slow spread of OJD within mainland SA is in some part at least the result of the program’s activities. The recommendation is strongly influenced by the increase in the number of flocks in SA which have been detected with OJD and the fact that the funds available for OJD control in SA remain largely unchanged. The current program will not be able to sustain the same level of regulatory control as the number of infected flocks increases and a course of action more aligned with the developing prevalence and available funding is now required.

In addition to the limit on funding, knowledge about the behaviour of OJD and the external (national) environment which sees Johne’s disease de-regulated in the sheep and cattle industries in other states, have also influenced the decision to recommend a different approach.

The continuing spread of OJD through mainland SA is regrettable. OJD can have significant impacts on both productivity and animal welfare in flocks in which the disease becomes established. While it can be readily controlled with vaccination, vaccine is relatively expensive and has other drawbacks. Unlike diseases of sheep which can be eliminated from farms at a relatively moderate expense (virulent footrot, for example), elimination of OJD from a farm carries a very high price. Producers who are free of OJD have never had a better reason to introduce sound biosecurity protocols for their flocks in an attempt to prevent the introduction of OJD. To do so, they will need reliable, credible advice about the risks associated with sheep trading. The template provided in Appendix A is intended to contribute to the development of a revised assurance scheme for OJD in SA which can better inform the decisions such producers can make.

KA Abbott
October 2016
EXECUTIVE SUMMARY

1. Ovine Johne’s disease (OJD) is a chronic, progressive disease of the intestine of sheep caused by a bacterium - *Mycobacterium avium* subspecies *paratuberculosis* (abbreviated here as *M ptb*).

2. Sheep which are infected with OJD shed the bacteria in their faeces. These faeces contaminate soil, pasture and water and susceptible sheep become infected by ingestion of the bacteria from the contaminated environment.

3. Infection can occur by other routes, including through milk or across the placenta to foetal lambs. These alternate routes are more likely from ewes which are in the advanced stages of clinical disease than from ewes in the earlier stages of disease.

4. If infection establishes in the intestine of a sheep, it may start to shed the bacteria in faeces within 12 months and start to show clinical signs within 24 months of first exposure. Some cases take longer to develop and, uncommonly, some cases take less time.

5. Sheep develop a cell-mediated immune response to *M ptb* which, in some cases, is effective in preventing the onset of more severe disease and may overcome the infection. In other cases, the cell-mediated immune response is overwhelmed by the infection and the disease progresses to a clinical phase.

6. The clinical phase is characterised by an inability to absorb nutrients which at first reduces weight gain, relative to disease-free flock-mates, then causes increasing degrees of weight loss. The clinical course persists for a period of about eight months before the sheep dies or is euthanased, but may only be obvious to casual observation over the last one to three months of life.

7. Once the clinical phase occurs, the condition is always fatal.

8. Sheep which develop clinical disease are usually affected by the multibacillary form of the disease, and are shedding *M ptb* in their faeces in enormous numbers for weeks or months before death or removal from the flock.

9. Antibodies against *M ptb* can be measured in the blood of infected sheep but do not develop usually until the sheep is in the early stages of clinical disease and typically about six months after faecal shedding of bacteria commences.

10. In a flock in which OJD is established at a high level, over half the sheep may be infected with the organism. Most of these infections are sub-clinical; there is no discernible weight loss and shedding of bacteria from sub-clinical cases occurs at low levels or intermittently. A proportion of the sub-clinical cases progress to clinical disease. Some progress to this stage quickly while some may not progress until one, two or more years later.

11. In an unvaccinated flock, the proportion of adult sheep which develop clinical disease each year varies between near-zero and 15%.

12. Several factors influence the severity of disease seen in an unvaccinated flock with established infection. One of the most important is the level of exposure of young sheep to an *M ptb*-contaminated environment. The younger the sheep and the heavier the contamination, the greater the proportion of sheep which develop the disease and the younger the average age at which deaths occur.
13. When the bacteria are first introduced into a susceptible flock, it takes some years before the infection is so well established that clinical cases become apparent. In one case in a region favourable for OJD development, it was seven years between a low-level of introduction and the recognition of clinical cases.

14. In regions favourable to the transmission of OJD, the disease can lead to a high level of clinical disease and mortality of adult sheep. Annual losses may be as high as 15% but 5% to 7% is more usual. Strategies (other than vaccination) can be implemented to reduce the mortality rates but these strategies themselves have a cost and are not necessarily readily available to all sheep producers. Cropping parts of the farm, for example, will help provide low contamination pastures.

15. The economic cost of OJD infection varies with the level of clinical disease but mortality rates in the range of 6% to 8% are associated with decreases in sheep-enterprise gross margins of 6% to 8.5%.

16. Vaccination with Gudair vaccine is an effective method of controlling the disease within an infected flock and can eventually reduce the prevalence of infected, M ptb-shedding sheep to undetectable levels.

17. The number of years taken to achieve a very low prevalence of infection depends on a number of factors including the within-flock prevalence when vaccination commences, the steps taken to reduce contamination levels and the rigour of biosecurity applied to the farm.

18. While vaccination combined with complementary management strategies have been shown to reduce the level of disease below that detectable in surveillance or monitoring strategies, it has not been shown that vaccination has led to the elimination of M ptb from a farm.

19. In the absence of information to the contrary and with evidence of the incomplete protection afforded by vaccination, it must be assumed that a high proportion of vaccinated flocks remain infected, albeit at very low levels, and therefore remain a potential source of infection to other flocks, through boundary fences or trade.

20. Gudair vaccination is relatively expensive compared to other vaccines and, in a flock vaccinating all lambs produced, may reduce the sheep-enterprise gross margin by 3%.

21. The cost of OJD nationally has been estimated to be $35m, with most of the losses arising from infected flocks in the high OJD prevalence regions of Australia.

22. Three distinct strains of M ptb exist in Australia; an S strain which predominantly affects sheep, a C strain which predominantly affects cattle, deer and alpacas, and a B strain, restricted to one outbreak in cattle in Queensland.

23. These strains demonstrate a host preference, rather than a host specificity. Under conditions of high challenge, cattle can become infected with S strains and sheep with C strains. Goats can also be affected by S strain infections.

24. S strain infection in cattle remains uncommon in Australia but has been reported from over 20 beef herds in south-eastern Australia where sheep and beef cattle share pastures.

25. The situation in Australia may be moving towards that of New Zealand, where the S strain of M ptb is responsible for 80% of cases of Johne’s disease in beef herds, even although there is evidence that S strains are less virulent for cattle than for sheep.
26. Despite evidence from other countries that wildlife, particularly rabbits, can become infected with *M. ptb*, the evidence from Australia is that rabbits and macropods are not an important source of transmission or reservoir of infection in this country.

27. In decreasing importance, it would appear that the most important non-ovine sources of *S. strain* *M. ptb* infection in Australia are fibre goats, beef cattle and, possibly, macropods, but none of these appear to be important reservoirs of infection in the absence of infected sheep. Nevertheless, any of them could be involved in transmission to a susceptible flock by movement from an infected farm or by short-term maintenance of infection during an attempt to eliminate OJD by destocking.

28. The evidence is very strong that *M. ptb* has a finite life in the environment (outside an infected host) of less than 18 months.

29. After contamination of an environment there is a very rapid decline in the numbers of viable (infectious) *M. ptb* bacteria over the first two to three months, followed by a more gradual decline over subsequent months. The rate of decline is strongly influenced by the presence of shade and moisture, which prolong survival.

30. There are regions of South Australia (South-East, Adelaide Hills/Fleurieu, Kangaroo Island) with rainfall, climate and grazing practices similar to the high prevalence regions of NSW and Victoria and where similar flock prevalences of OJD can be expected if controls are ineffective. Around 42% of the state’s sheep are grazed in these regions.

31. In drier parts of SA or on farms which integrate cropping enterprises with sheep production, the number of *M. ptb* on sheep pastures will be less than in the high-rainfall, sheep-dominant farms, with subsequent effects on the levels of challenge to which sheep are exposed.

32. Abattoir surveillance serves two purposes in relation to OJD; one is to detect infected flocks so that regulatory controls can be imposed (if appropriate) or flock owners notified of the disease detection, and the other is to allow estimation of the regional prevalence of OJD infection.

33. Abattoir surveillance has a limited sensitivity influenced strongly by the line size, within-flock prevalence and other factors influencing the inclusion of affected sheep in an abattoir line.

34. It may be possible to make reasonably accurate predictions of the prevalence of infected flocks based on abattoir surveillance of a limited proportion of the region’s flocks,

35. In the absence of other surveillance strategies, it is necessary to have abattoir surveillance of a very high proportion of the region’s flocks if regulatory control is to be effective.

36. Investigations of flock OJD status, either following an abattoir surveillance detection or to permit the removal of an Order, are based on the testing of faeces from a large sample of the adult flock. Testing is done on pools of faeces from 50 sheep.

37. When seven pools (350 sheep) are tested, there is a very high probability that at least one infected sheep will be included in the sample, provided the within-flock prevalence is 2% or greater. For lower prevalences or larger flocks, the probability may decline to 95% or less.

38. The laboratory tests themselves (HT-PCR or culture) have less than 100% sensitivity so not all positive pools will necessarily be detected. The sensitivity is high if a multibacilliary case is included but substantially lower if only paucibacilliary cases are included.
39. The combination of these two probabilities mean that some infected flocks will be mis-diagnosed as free of infection, particularly if the within-flock prevalence of infection is low, or if there are no sheep in the sample with multibacilliary infection.

40. Flocks with a multibacilliary case detected at abattoirs are likely to contain other sheep with multibacilliary infection, particularly if infection is well-established in the flock. Such flocks are very likely to be detected by PFC tests.

41. Flocks which have been vaccinating for several years may have no sheep with multibacilliary infection or so few sheep with that degree of infection that, by chance, there is none included in the sample.

42. *M ptb* has been linked to Crohn’s disease in humans. There is evidence that the organisms occurs in some cases of the human disease, but not all cases. There is no evidence for a causative role in the disease and the nature of the association remains debated within the medical field.

43. OJD has been recognised in South Australia for 20 years but, given the high prevalence of infected flocks found on Kangaroo Island in 1998 and subsequent years, the disease is likely to have been present in that region for some years before detection.

44. The prevalence of known-infected flocks in South Australia remains low. Assuming all flocks which have ever been detected are still infected, there are about 190 known-infected flocks in SA.

45. The two regions of SA with the highest prevalence of flocks detected with OJD are Kangaroo Island (26%) and the South-East (2.6%). Kangaroo Island has been subject to much higher levels of structured disease surveillance than other regions of SA.

46. Over the past decade, most new detections have been in the South-East regions.

47. Of the 190 flocks detected in SA to date, 140 have been released from quarantine on the basis of a PDMP or PDEP and subsequent clearance test, based on testing of pools of faeces by HT-PCR, culture, or both.

48. Around 460,000 doses of Gudair vaccine are used in SA each year. Roughly one third of these doses are distributed by PIRSA under a subsidy arrangement with SASAG using the Industry Fund, and two thirds are used independently by producers outside the subsidy arrangement.

49. The proportion of sheep bred each year and retained beyond 12 months of age which are vaccinated with Gudair is relatively low compared to Tasmania, Victoria and NSW. A crude estimate is that 14% of the sheep for which vaccination is appropriate, based on their likely longevity, are vaccinated in SA.

50. Abattoir surveillance in South Australia is carried out at two abattoirs, Murray Bridge and Lobethal.

51. For the South-East regions, where the most new detections have occurred, 21%, 41% and 45% of each region (lower, mid, upper respectively) of flocks were subject to abattoir surveillance in the four years 2012 to 2015.

52. In 2015, an estimated 419 flocks in the South-East regions had sheep inspected at abattoirs. This represents 22% of the flocks which had sheep activity in 2015.

53. Interpretation of the available data suggests that detection of five new cases of infected flocks in the South-East resulted from inspection of lines from those 419 flocks.
54. One could assume therefore that a further 15-20 new cases were missed because the remaining 
78% of flocks did not submit sheep to an abattoir where surveillance was occurring.

55. Abattoir surveillance is not a highly sensitive tool for detecting OJD in low prevalence flocks. If one
assumes that abattoir surveillance detected 50% of the previously undetected flocks then it
follows that a further 20-25 flocks with new infections in the South-East remained undetected in
2015.

56. The combination of limited penetration and insensitivity of abattoir surveillance leads to the
conclusion that the prevalence of OJD in the South-East region is significantly under-estimated
(based on abattoir surveillance) at present.

57. The current South Australian Control Program has been effective and has reduced the rate of
spread of OJD within the state.

58. The control program is, however, dependent for its efficacy on abattoir surveillance (to detect
new infections) and vaccination (to eliminate infections from flocks). Both of these strategies are
less than 100% effective, allowing new infections to go undetected for extended periods, and for
vaccinated flocks with undetectably low prevalences to resume trading.

59. Both the trading patterns for sheep in SA and the climate are likely to have contributed to the
currently low prevalence of OJD in regions of the state other than the South-East and Kangaroo
Island.

60. The prevalence of OJD in SA is underestimated but does remain low.

61. OJD infections in flocks remain clustered in three medium to high rainfall zones; Kangaroo Island,
the South-East and the Adelaide Hills/Fleurieu regions.

62. While infections on Kangaroo Island are manageable, new infections in the South-East and other
medium/high rainfall regions will continue to occur, probably at increasing rates, even while
regulatory controls continue.

63. If regulatory controls are removed, it is expected that OJD infection will spread through the state’s
medium and high rainfall zones at a higher rate and progress towards the levels of flock infection
seen in Victoria, New South Wales and New Zealand.

64. OJD will present a significant disease threat to producers in the medium and high rainfall regions,
requiring preventive vaccination to control the disease.

65. In the lower rainfall zones of SA, OJD will spread more slowly and will in some cases be manageable
on farm without vaccination.

66. The Bio-economic model developed by AusVet (2006) indicated that, if one assumes a relatively
low rate of spread of OJD across the state, de-regulation of OJD had a cost to the industry similar
to the costs of continuing the current control approach.

67. The approach proposed by Biosecurity SA includes a de-regulation of OJD, with some regulatory
activity directed towards clinical cases of OJD. This approach should be tempered so that it is
consistent with falls in line with the approach taken for other serious diseases managed under the
Livestock Act and the Welfare Act.

68. There is confusion in the industry about the terms ‘low risk’ and ‘low prevalence’ in relation to
OJD. An alternate approach to describing the OJD status of farms is suggested (Appendix A).
69. The importance of a regional low prevalence on the sale value of breeding sheep is acknowledged but the specific value is unknown.

70. A summary of 13 important facts about OJD is presented with the suggestion that these be included in messages to sheep producers in SA (Section D).
RECOMMENDATIONS

Recommendation 1;
Planning for a transition to a de-regulated environment should begin now.

Recommendation 2;
OJD management funds should be directed away from vaccine subsidy and farm investigation costs towards a well-structured epidemiological survey to provide a reliable estimate of the prevalence of OJD-infected flocks within regions of SA.

Recommendation 3;
Steps should be taken to provide better information about OJD to producers. The 13 points in Section D, Summary, should be a key part of the information packages. Some of the funds currently spent on control strategies should be allocated to industry extension and education.

Recommendation 4;
Based on better knowledge about OJD prevalence across the state, producers in some areas should be encouraged, if appropriate, to create regional biosecurity areas or cooperative biosecurity groups in order to manage the disease-control status of their flocks and reduce the risk of OJD establishment. Some of the funds currently spent on control strategies could be allocated to supporting the creation of such groups.

Recommendation 5;
To aid OJD management and to assist control of other endemic diseases, abattoir surveillance should be continued and expanded, and the data should be analysed and reported to the industry.

Recommendation 6;
Plans to adapt the Sheep Health Statement to a different regulatory environment and to provide OJD assurance levels for flock managers should consider using the guide produced in Appendix A of this report, and to moving away from the term ‘low-risk’ for ‘low prevalence’ flocks.
SECTION A TECHNICAL REVIEW OF OJD

A-1  The disease and its time-course
OJD is a chronic, progressive disease of sheep caused by *Mycobacterium avium* subspecies *paratuberculosis* (abbreviated here as *M ptb*). There are different strains of the organism, categorised broadly into three groups (S, C and B). OJD in Australia is usually caused by S strains but C strains have also caused disease in sheep in Australia. In this report, OJD refers to Johne’s disease in sheep, whatever strain is responsible. Host specificity or host preferences of the strains are discussed further below.

Sheep usually become infected following ingestion of large numbers of the organisms\(^1\) \(^2\) (an ‘infective dose’\(^a\)) deposited in the environment in the faeces of infected animals. Thus, pasture and water are considered to be the main vehicles for transmission, but the contaminated skin of an infected ewe may also be important for establishing infection in lambs. In ewes with advanced infections, *M ptb* may be present in colostrum and milk and lambs may be infected *in utero*\(^3\).

Following ingestion of the bacteria an infection may establish - usually in the lower small intestine (the ileum) and associated lymph nodes. If infection establishes and develops, the intestinal wall becomes progressively thicker, ultimately interfering with absorption of nutrients to the extent that the animal can no longer maintain body condition. The disease causes chronic wasting – loss of body condition - and, eventually, profound weakness and death. Generally appetite is maintained until the animal is recumbent and unable to rise. Diarrhoea is not a common feature of the disease in sheep as it is in cattle. In some cases the faeces become soft but in many cases the faeces remain normal. There is no effective treatment for the disease.

The time course of the disease is prolonged. Following infection in the first few weeks of life, most sheep which develop the disease do not show any signs until they are aged two years or more. In flocks in which the infection is established, mortalities continue in all adult age groups. Under some circumstances, mortalities can occur in sheep less than two years of age. This is discussed further below.

During the course of the disease, there are several distinct stages which have relevance to the ability of diagnostic tests to determine if a sheep is infected, and which are relevant to the transmission of infection from an infected animal to a susceptible, non-infected animal. Four overlapping stages which are described here are

- Cell mediated immune response (CMI)
- Humoral response with antibody production or seroconversion
- Faecal shedding.
- Weight loss.

A-2  Cell mediated immune response
The first immune response of sheep to OJD infection is of the cell-mediated form. This type of immune response is not detectable with standard tests for OJD but can be detected with tests used in research studies. (The tuberculin test for tuberculosis is an example of a test for cell-mediated immunity.) The CMI may, in some cases, successfully overcome the infection but, if it does not, it usually becomes weaker late in the course of OJD progression.

\(^{a}\)In Australian pen trials, \(10^4\) organisms were insufficient to produce detectable infection. Infection could be established with \(>10^7\) organisms. Uncommonly, lower doses have resulted in infection studies in other countries.
A-3  Antibody production
Sheep infected with OJD eventually develop an immune response based on the production of antibodies which are detectable in blood tests, such as the ELISA or AGID test. This immune response (called a humoral response to differentiate it from the cell-mediated response) does not develop early in the course of infection and is usually ineffective in protecting the animal against the progression of OJD. The development of antibodies against OJD is also termed ‘seroconversion’ because tests of serum for antibodies change from being negative to positive.

A-4  Faecal shedding
In the context of OJD, faecal shedding refers to the passing of \( M ptb \) bacteria in the faeces of an infected sheep and at a level which is detectable by laboratory culture techniques. The term has become used in a colloquial way by those involved in OJD management and research. ‘High shedders’ are sheep which are passing \( M ptb \) at very high levels – up to \( 10^8 \) organisms per gram of faeces or \( 10^{11} \) organisms per day. ‘Low shedders’ are sheep which are passing \( M ptb \) at a rate several orders of magnitude lower and may also be shedding bacteria only intermittently. Animals which are high shedders have the multibacillary form of the disease; low shedders the paucibacillary form. These terms are described further below. Note also the term patency in relation to faecal shedding; an infection is said to be \textit{patent} once bacteria appear in the faeces.

A-5  Weight loss
As the disease progresses sheep begin to lose weight and condition, relative to their healthy flockmates. Weight loss or poor condition is the most reliable and consistent sign of OJD which can be observed without laboratory tests. At first, the weight loss is detectable only with structured, objective measurement using scales but, once the degree of weight loss is visually obvious to the careful observer, the sheep is considered to be in the clinical phase of the disease.

The relative timing of these stages is illustrated diagrammatically in Figure 1.

![Figure 1](image)

\textit{Figure 1: There are several stages of a progressive OJD infection. The cell-mediated immune (CMI) response occurs soon after infection commences but usually wanes late in the disease. Faecal shedding occurs usually before antibodies are detectable and weight loss (clinical disease) begins about the same time, but may not be obvious for some months.}
A-6 Intestinal pathology of OJD

*M. ptb* infects the wall of the small intestine, the lymph nodes draining the intestine and some other tissues. The pathological changes in the lymph nodes and intestinal wall are characteristic of the disease, but do vary in type. Broadly, there are two types described by pathologists. In one form, the reaction is based on the presence of macrophages – large, phagocytic white blood cells – which contain numerous *M. ptb* organisms. Because there are many bacteria present, this form of the disease is called *multibacilliary OJD*. Note that the multibacilliary nature of this form of the disease is characterised by both high shedding rates in the faeces and by distinct pathology. Pérez *et al* (1996)\(^5\) categorised this type of OJD lesion as type 3b.

The other form of the disease differs in the type of inflammatory cell present in the lesions – lymphocytes predominate over macrophages. There are few *M. ptb* bacteria present in the cells and this form of the disease is called *paucibacilliary*. Pérez classified this type as 3c. Types 1, 2 and 3a are also described by Pérez *et al* (1996)\(^5\).

A-7 Clinical cases

In endemically-infected flocks\(^b\), faecal shedding of *M. ptb* can be occurring in some animals by 12 months of age; antibody responses developing some six months later\(^6\). Within an age cohort, deaths from OJD typically begin in animals aged two years or more but occasional animals will die at younger ages. Deaths of sheep from OJD at 17 months of age have been recorded in at least two separate Australian studies\(^7\).

Weight loss, relative to flock-mates, begins about the same time as the onset of faecal shedding such that, eight months before death, they have lost about 4% of bodyweight, then continue to lose bodyweight at the rate of about 4% per month. At the time of death sheep with OJD are, on average, over 30% lighter than expected if in good health.

During the clinical phase of the disease, the gradually discernible weight loss is generally accompanied or preceded by a dramatic increase in the number of organisms shed in the faeces as animals move from the paucibacilliary form of the disease to the multibacilliary form. Once the infection is multibacilliary, recovery does not occur and the sheep will die of OJD, usually within 12 months\(^8\). Sheep can develop clinical OJD while affected with paucibacilliary infection of the intestine, but that is a relatively uncommon occurrence. In Australia at least, most sheep dying from OJD have multibacilliary infection (Pérez type 3b)\(^7\).

The clinical phase is also accompanied by a reduction in serum albumin levels, probably as a consequence of interference with protein digestion and absorption\(^9\). Some sheep may develop diarrhoea but, more commonly the faeces are normal or soft, but not fluid.

A-8 Sub-clinical cases

A sheep is said to be infected with *M. ptb* if the organism can be cultured from faeces, intestinal tissue or lymph node, or if organisms are detectable in characteristic histopathological lesions in the intestine or lymph nodes\(^8\).

Sheep which are infected but have not begun to lose weight (or show other clinical signs) are in the sub-clinical phase of the condition. There are several possible outcomes of sub-clinical infection. Based on a study\(^8\) to three years of age of 77 young Merino sheep from a heavily infected flock\(^8\), the following outcomes and the percentage of cases may be proposed:

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\(^b\)The term ‘endemic’ is described in Appendix B.
\(^c\)Goulburn, NSW, average annual rainfall 643 mm, uniformly distributed through the year.
- No infection develops before three years of age (40%)
- Sub-clinical infection develops (60%), which in turn leads to;
  - Rapid progression to clinical disease and death, within 12 months of the development of an infection in the intestine (16%)
  - Progression slowly and at variable rates towards clinical disease, some animals remaining with sub-clinical infection for extended periods (36%)
  - Full recovery, with complete freedom from infection (6%)
  - Apparent recovery then relapse to sub-clinical infection (1%)

Sheep in the early sub-clinical phases of infection generally have paucibacilliary infections; in other words, they shed *M. ptb* in faeces but at low levels. Sheep in the later phases of a sub-clinical infection may occasionally remain with paucibacilliary infections but more commonly the infection becomes multibacilliary. Clinical disease rarely occurs unless the infection is multibacilliary. Multibacilliary disease is always accompanied by clinical disease and death.

The progression to clinical disease accompanied by multibacilliary infection is linked to a failure of the cell-mediated immune response to contain the infection. Consequently, in a typical case, CMI responses diminish and humoral responses (antibodies) develop as the disease becomes multibacilliary and clinical signs develop.

**A-9  Asymptomatic infection**

A small proportion of sheep which are exposed to *M. ptb* on infected farms may develop minor infections, detectable by culture of intestinal tissues, with no evidence of intestinal pathology. The infection is clearly sub-clinical but this group has also been described as asymptomatic. In one study, asymptomatic infection is distinguished from both clinical and other forms of sub-clinical infection by the type of immune response that developed. The outcome of asymptomatic infection is not clear – the infection may progress to a clinical form, may persist as a permanent low level of infection, or resolve.

In a large Australian study, 12% of three-year old sheep, challenged since birth and slaughtered at the end of a field study, had no detectable histopathological lesion but had positive intestinal-tissue culture. Because the field site was *M. ptb*-contaminated, it is possible that at least some positive results could have been from contaminated ingesta in the intestine, but the figure puts an upper limit on the possible frequency of asymptomatic infection. The distinction of asymptomatic infection from other forms of sub-clinical infection is somewhat technical. From a clinical and field management viewpoint, all sub-clinical cases appear to be asymptomatic.

**A-10  Relationship between age of sheep at first challenge, level of challenge and prevalence of disease and age of onset.**

Sheep of all ages appear to be susceptible to infection with *M. ptb* but lambs are much more likely to develop patent infections (shedding *M. ptb* in faeces) following exposure than adults, and infections developed by lambs are more likely to lead to severe infections and clinical signs at shorter intervals post-infection than infections developed by adult sheep.

In an experiment with Merino sheep in NSW, sheep challenged as lambs were seven times more likely to shed *M. ptb* in faeces than sheep challenged for the first time as adults.

Earlier first-challenge also increases the risk of mortality from OJD. Sheep exposed to *M. ptb* before three months of age were found to be 2.5 times more likely to die from paratuberculosis before 36 months of age than sheep exposed for the first time after three months of age. McGregor *et al* (2012) found that sheep exposed to *M. ptb* for the first time at five to six months of age were five
times more likely to die of paratuberculosis in the subsequent 30 months, compared to sheep aged 2.5 years or more. Delgado et al (2012) found that lambs challenged with a high dose of M ptb at 1.5 months of age were more likely to develop multifocal lesions (Pérez type 2 and type 3a) than adult ewes, which developed predominantly focal lesions (type 1) under the same challenge conditions. The different age-related responses were associated with a more rapid and more efficient cell-mediated immune response in adult sheep than lambs.

The level of challenge also has an effect on the outcome in individual sheep. McGregor et al (2012) found that sheep challenged with highly contaminated pastures were 18 times more likely to die than those exposed at lower levels, while Abbott et al (2004) found three times as many sheep with paratuberculous lesions at 36 months of age in the high-exposure group compared to the low exposure group.

### A-11 Levels of infection in OJD-infected flocks

The prevalence of infection in a flock of sheep influences the likelihood that the flock will be detected by abattoir surveillance or by flock-level tests, such as pooled faecal culture (PFC). It also influences the level of clinical disease occurring in the flock and the likelihood of the disease being transmitted from infected farms through boundary fences or by the trading of infected sheep.

When M ptb is introduced at a low level (such as by the introduction then removal of a few infected sheep, or by pasture contamination through a boundary fence) into a non-infected flock, many years may pass before the prevalence of infection within the flock increases to a level which is detectable by clinical observations or even by routine testing. Initially, the disease is most likely to be transmitted to lambs born on the property and adult sheep are least likely to develop infections. Furthermore, because of the low levels of contamination to which they are exposed, only a few lambs will develop infections which become patent and the infections will tend to become patent at ages over three years, rather than at younger ages. The contamination caused by this first generation of infected sheep will also remain at low levels for a year or more, leading to a second generation of infected sheep which, likewise, do not shed significantly until they are also three years of age or more. Patent infection is likely to remain clustered in one or two age groups and at a low frequency for up to seven years after the organism is introduced into the flock, and clinical cases of OJD are unlikely to appear within that time.

Once infection becomes established in a flock, however, lambs are routinely exposed to M ptb organisms from birth and, unless steps are taken to reduce their exposure, the level of challenge is a consequence of the level of infection and shedding in the adult flock. Ultimately it is likely that, in flocks in higher rainfall zones in which OJD has become established, over 40% of the flock will be infected with M ptb. There are some examples reported in the literature which support this conclusion including that referred to in the previous section discussing sub-clinical disease.

In a non-vaccinating Merino flock in NSW (Gunning, average annual rainfall 640 mm) with a high level of M ptb infection, the prevalence of sero-positive (ELISA test for antibodies) adult sheep was between 8% and 18%, depending on the age group, over 25% of the two year old sheep were excreting M ptb in faeces, and over 13% of the adult sheep were dying each year from OJD.

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\(^d\) The relationship between climate and level of challenge, and its implication for regions of SA, is discussed in Section C.
In another flock with established OJD infection, the seroprevalence of three, four and five year-old ewes tested before entering a field study was 5.8%. In another, 18% of two year old sheep were seropositive and one third of them were shedding *M. ptb*.

The sensitivity of serological tests is known to be low and the true prevalence of infected sheep is always significantly higher than the seroprevalence.

A survey in Spain to estimate the prevalence of *M. ptb* infection in small ruminants found a seroprevalence of 11.7% across 546 sheep and goats in 91 herds in the Madrid province. The survey authors estimated the animal-level prevalence of *M. ptb* infection to be up to 44%.

**A-12 Levels of clinical disease and mortalities in infected, non-vaccinating flocks**

In Australia, for 10 to 15 years after the disease was first described in sheep in New South Wales in 1981, there was a belief amongst many producers and disease control officers that the mortality rate from the disease was low (0.4 – 4% of adult sheep) and likely to remain so because of the climatic conditions common to sheep raising areas of Australia.

Subsequent investigations, however, indicated that this was not always the case and, before OJD vaccination became available, a number of Merino flocks reported high levels of clinical disease and mortalities. One of these flocks (referred to in the previous section) was studied closely over a two-year period from 1999-2001 to determine the contribution that OJD was making to the annual sheep mortality in the flock. Mortalities in adult sheep from all causes were estimated to be 21.5% and 17.8% in each of the two years. It was concluded that paratuberculosis caused or contributed to the deaths of 14.5% of the adult sheep in the first year and 13.2% in the second year. The sheep, despite being adult and exposed to *M. ptb*, were vaccinated between Year 1 and Year 2 and, not unexpectedly, vaccination had no significant effect on mortalities in the first year post-vaccination. This report is, to my knowledge, the highest level of OJD mortality that has been substantiated through any form of structured study. It is possible that the form of management on this property (rotational grazing) contributed to a high level of exposure of young sheep to *M. ptb*.

Other OJD-infected Australian flocks reported OJD mortality rates less than 10%. The authors of a survey of 155 flocks in the Central Tablelands of NSW were able to prepare the following table (Table 1) based on the estimates of flock owners of the level of mortalities in their flocks, related to the number of years since OJD was diagnosed in the flock.

**Table 1: A survey of flock owners in the Central Tablelands of NSW showed the range of mortality rates caused by OJD, based on the owner’s opinion. Vaccination was not available at that time.**

<table>
<thead>
<tr>
<th>Years since diagnosis</th>
<th>Number of properties</th>
<th>Percent annual mortalities attributed to OJD</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Mean</td>
</tr>
<tr>
<td>0-2</td>
<td>70</td>
<td>2.4</td>
</tr>
<tr>
<td>3-5</td>
<td>54</td>
<td>4.3</td>
</tr>
<tr>
<td>6-9</td>
<td>19</td>
<td>5.4</td>
</tr>
<tr>
<td>10+</td>
<td>12</td>
<td>5.9</td>
</tr>
<tr>
<td>Total</td>
<td>155</td>
<td>3.7</td>
</tr>
</tbody>
</table>

A number of management strategies (other than or in addition to vaccination) can be employed to reduce the severity of OJD in a flock. These strategies were proposed following a three-year long field study which showed that steps that are feasible for flock managers under commercial conditions can make a substantial difference to the number of sheep which develop clinical disease by three years of
Review of the management of OJD in South Australia. October 2016

These steps include the immediate removal from the flock of any sheep showing the early signs of OJD, short joining periods, early weaning and the preparation of pastures for lambing and, especially, for weaned lambs which are likely to be low in contamination.

At least one producer in the central Tablelands of NSW was able to achieve very good control of OJD by adopting strategies such as these - independently and preceding the trial discussed above - despite a long history of OJD in the flock and a similar environment to other producers with rampant clinical disease in their flocks. While not studied in detail, this farm was subject to a number of visits and reviews by producer representatives and research scientists.

The need to manage OJD does, however, further limit and constrain the operation of a sheep flock so the level of control described above is not without cost. Control of the disease through management is rarely used in Australia now that vaccine is available but could remain a consideration for flock managers to use in conjunction with vaccination when OJD is first diagnosed in a flock, to hasten the progression to a low-prevalence status, or for producers in environments which are not favourable to OJD spread and who wish to exercise control without vaccination.

There are no published data on mortality rates of OJD in South Australian flocks. Reports from workers in the field (D Lehmann, Kangaroo Island, P Nosworthy, South-East) include cases where mortality rates from OJD were estimated to exceed 10% of adult sheep annually but the factors which contributed to these losses are not known.

Published data on paratuberculosis-attributable mortality rates in other countries are relatively scarce. In the most comprehensive study, Sigurdsson presented data from over 6000 sheep in 141 flocks in Iceland indicating annual death rates of 8% to 12% attributable to paratuberculosis in 1950-51. Most other published figures are unsubstantiated estimates. In Cyprus, paratuberculosis death rates were estimated to be 4% to 5% in sheep flocks. In the UK, OJD mortality rates are generally considered to be low but Cranwell suggested that, in an outbreak in one flock, paratuberculosis may have caused the deaths of 6% of the adult ewes per year. In New Zealand, reported mortality rates from paratuberculosis in sheep are typically around 1% per annum although some flocks have reported losses of 4% of adult ewes.

Cost of uncontrolled OJD infection

A study of OJD on 12 endemically-infected farms in NSW in 2002-2004 found that, in the absence of Gudair vaccination, OJD led to an average annual mortality rate of 6.2%, 7.8% and 6.5% in the three consecutive years. In 2004, based on necropsy examination of a sample of all sheep dying, the range between farms was 2.0% to 11.9%. The average decrease in gross margin as a consequence of the mortality rate was calculated by economic modelling of those 12 farm enterprises and was estimated to be 6.4%, 8.5% and 7.4% in each of the three years. The cost per dry sheep equivalent was estimated to be $7.68.

It should be noted that this study was performed in areas of southern NSW (Bungendore, Harden, Gunning, Taralga) where OJD was endemic and infection was long-standing on the farms. The data are included because the results indicate the levels of mortality that can occur in areas where the disease is established in farm environments which are apparently favourable to its spread and in order to relate the level of disease expression – the mortality rate – to the estimated cost of the disease.

* Mr Cliff Kelly, producer of Blayney, NSW and Member of the OJD Industry Advisory Committee, 2002
A-14  Vaccination

Vaccination with Gudair vaccine is the most common tool used in Australia to reduce the level of disease and losses from OJD in infected flocks. Vaccination is also practised in some non-infected flocks to provide some protection from introduction of the disease.

Vaccination does not prevent the infection of animals but modifies the inflammatory response, reducing the progression of lesions in the intestine and reducing the shedding of \( M \text{ptb} \) within a flock. Vaccination appears to work by stimulating immune responses much more strongly than occurs in response to natural infection, particularly during the early stages of natural infection\(^{23} \).

Vaccination can be effective even if administered to animals which are already exposed and infected with \( M \text{ptb} \) at a sub-clinical level. When lambs aged four to six weeks were experimentally infected by oral dosing with \( M \text{ptb} \), then vaccinated two weeks later, the number of lambs which developed clinical disease, and the severity of OJD lesions was substantially reduced (by over 80%), compared to unvaccinated lambs\(^{26} \). In infected flocks, vaccination of adult sheep, presumably including some that are already infected, leads to a reduction, but not elimination, of the occurrence of new clinical cases\(^{19} \).

Vaccination of lambs as young as two or four weeks leads to the development of a measurable immune response, although there is evidence that older animals (20 weeks, for example) develop a stronger and more persistent immune response\(^{25,27} \). The practical importance of the different strength of immune response between lambs vaccinated at very young ages, and those vaccinated at greater ages is unclear.

Currently, Australian recommendations are that lambs in infected flocks are vaccinated between four and sixteen weeks of age. From the published data, this recommendation appears to be sound because any lambs which have already been infected by that age will still be in an early stage of infection, when vaccination can be expected to have a beneficial therapeutic effect, and the youngest lambs will be old enough to mount a useful immune response. While vaccination at greater or lesser ages may still be effective, for producers who wean lambs 13 to 16 weeks after the lambing start-date, the weaning event may provide the best opportunity to vaccinate lambs at an age when vaccination will be as effective as possible.

Research trials show that, in infected flocks experiencing significant OJD mortalities, vaccination will, within the first 4-5 years

- reduce mortalities from OJD amongst vaccinated sheep by 90%
- reduce the number of sheep with sub-clinical infections at 3½ to 4½ years of age by 66%
- reduce the number of sheep which shed bacteria by 90%
- delay by 10 months the onset of shedding of bacteria in those that do become infected

Sheep which develop clinical disease despite vaccination can develop multibacilliary infections and shed bacteria at high levels\(^{28} \). The reduction in shedding at a flock level following vaccination is principally achieved by a reduction in the number of sheep which develop disease.

There is mounting evidence that the benefits of vaccination accumulate in the years beyond the first four or five years, presumably because the vaccinated progeny of vaccinates are exposed to increasingly diminishing levels of pasture infectivity.

Windsor (2013)\(^{29} \) summarised the experience of medium to long term Gudair vaccination of sheep in Australia, based particularly on three studies. Some of the information from that summary is presented in Table 2.
The results of those trials suggest that vaccination of all sheep on the farm for a period of five or more years can reduce the prevalence of infected, *M. ptb*-shedding sheep to undetectable levels. Factors which reduced the likelihood that vaccination would lead to undetectably-low prevalence were:

- Having sheep stray onto the farm
- Purchasing sheep other than approved vaccinates\(^ {7}\)
- Leaving some sheep (wethers for example) unvaccinated, on the basis that they will be sold as 1½ year old sheep. At least in the first six years after vaccination commences, this strategy is unsuccessful\(^ {10}\).
- It is likely that progress to undetectably-low levels would be slower in flocks with an initially high prevalence of infection. There is some evidence of this from experience in SA.

The high levels of control of OJD achieved on KI are probably associated with the additional strategies put in place in those flocks to achieve an early and immediate reduction in the level of shedding\(^ {31,32}\).

The question remains as to whether the vaccination program in those flocks which have achieved undetectably low levels of shedding has resulted in complete clearance of the bacteria from any flock and farm. The answer will only be clear when the owners of those flocks choose to cease vaccination altogether and then to institute surveillance activities for six or more years into the future. In the absence of information to the contrary and with evidence of the incomplete protection afforded by vaccination, it must be assumed that a high proportion of vaccinated flocks remain infected, albeit at very low levels, and therefore remain a potential source of infection to other flocks, through boundary fences or trade.

### Table 2: A summary of three trials in NSW and KI reported the benefits of Gudair vaccination. (SR = self-replacing). When infection is endemic in the flock before vaccination commences, it must be practised for at least five years before significant improvements in flock-levels of disease are seen. Efficacy at the flock level is improved when other disease-control strategies are combined with vaccination\(^ {29}\).

<table>
<thead>
<tr>
<th>Identifier</th>
<th>Number of flocks in study</th>
<th>Vaccination history</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trial 033 or 0565</td>
<td>11 flocks, SR Merino, NSW</td>
<td>Vaccination in lambs &lt;16 wks. Sheep older than 2 years were sampled every two years starting two years after vaccination commenced, and repeated at 4 years and 6 years.</td>
<td>No vaccinated sheep in first test. All sheep in third test were vaccinates. Vaccination reduced the prevalence of shedding by 62%. Shedding still detectable with PFC350 in 10 of the 11 flocks.</td>
</tr>
<tr>
<td>0309</td>
<td>37 SR Merino, NSW</td>
<td>Vaccination practised for at least 5 years.</td>
<td>PFC350 used. 30 flocks had detectable infection, 7 did not.</td>
</tr>
<tr>
<td>KI study</td>
<td>16 flocks.</td>
<td>Vaccination practised for at least 6 years. Vaccination initially included the whole flock. Additional strategies put in place to reduce the number of infected sheep present.</td>
<td>PFC600 used, if possible. 14 flocks had no detectable infection.</td>
</tr>
</tbody>
</table>

\( ^{7}\) Approved vaccinates are sheep which are vaccinated with Gudair before 16 weeks of age or are vaccinated as adults with no history of exposure to *M. ptb* prior to vaccination.
**Cost of vaccination**

The Gudair vaccine is relatively expensive, compared to other vaccines used in sheep flocks. Unlike the clostridial or cheesy gland vaccines, however, the Gudair vaccine need only be administered to sheep once in their lives. Because of the cost of the vaccine and the cost of administering the vaccine, producers who wish to use the vaccine in their flocks will seek to minimise the number of animals vaccinated to those which will be retained beyond 12 months of age, on the assumption that younger animals will not shed *M. ptb* bacteria even if they become infected.

Flocks with the lowest vaccination cost will be those which sell a high proportion of their home-bred animals as lambs. Flocks with the highest vaccination cost will be those which retain lambs of both sexes beyond 12 months of age or vaccinate sheep which are intended to be retained by other breeders beyond 12 months of age. To place the vaccination cost in the context of a typical sheep farm enterprise, I refer to a gross margin budget for a self-replacing 20 µm Merino flock of 1000 breeding ewes prepared and published by NSW DPI (October 2015)\(^{13}\).

For such a flock, containing breeding ewes aged two to five years and marking 890 mixed-sex lambs, the cost of Gudair vaccination ($2.64 each) of all lambs at marking (both sexes) adds $2348 to the annual expenses. Ignoring any additional labour costs, the cost of vaccinating all lambs annually is $2.35 per ewe present, and reduces the gross margin of the flock by 3% (Table 3).

**Table 3: Gudair vaccine is a significant animal health cost. When administered to all lambs born in a Merino enterprise it will reduce the gross margin of a sheep flock by around 3%.**

<table>
<thead>
<tr>
<th></th>
<th>Without vaccination ($)</th>
<th>Including vaccination ($)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gross income per 1000 ewe flock</td>
<td>155076</td>
<td>155076</td>
</tr>
<tr>
<td>Gross margin per 1000 ewe flock</td>
<td>80211</td>
<td>77863</td>
</tr>
<tr>
<td>Gross margin per ewe</td>
<td>80.20</td>
<td>77.90</td>
</tr>
<tr>
<td>Gross margin per dse</td>
<td>30.60</td>
<td>29.70</td>
</tr>
<tr>
<td>Gross margin per ha (10 dse/ha)</td>
<td>306</td>
<td>297</td>
</tr>
</tbody>
</table>

Gross margin analysis does not include all costs associated with the farm business because it ignores fixed costs. Depending on the fixed costs of the farm, profitability of the sheep enterprise may be reduced by 7% to 10% by the 3% decrease in gross margin.

Based on the NSW DPI budget figures, the cost of Gudair vaccination in such a flock similar in magnitude to anthelmintic administration, dipping for lice or crutching.

This estimate of the financial impact of Gudair vaccination is based on a flock in which no OJD mortalities occur. It represents the cost that would be incurred by a producer to protect a flock from OJD or to maintain at a near-zero level the impact of an OJD infection on the flock survival rate.

A comparison of this result to the estimated cost the disease (see previous section) indicates that vaccination is financially justified, based on the likely reduction in mortality alone, in flocks where OJD mortality rates are contributing to a reduction in gross margin greater than 3%. For example, in a flock with a mortality rate due to OJD of 6.5% (the average of those 12 flocks in 2004), vaccination will (eventually) replace the predicted loss of 7.4% of gross margin due to disease with an annual cost of around 3% of gross margin as a consequence of the cost of vaccine.

The return on the investment in vaccination is not immediate. Toribio et al\(^4\) suggested that the cost of vaccination is recovered in two to three years for breeding enterprises if the level of OJD is high. If
the level of OJD is low, the breakeven point is achieved in three years for a crossbred enterprise and seven years for a Merino ewe enterprise.

There may also be other financial benefits from vaccination such as improved access to markets for sale sheep, and intangible benefits, including the personal satisfaction that comes from a healthier, more productive flock.

**Human health risks associated with vaccination**

The inadvertent exposure to Gudair vaccine to humans through a needle-stick injury can cause serious injury requiring medical and often surgical treatment, with prolonged recovery periods. Scratching of the skin or needle-stick with the vaccinating needle can occur to the person performing the vaccination of sheep (self-inoculation) or to people assisting with the handling or restraint of sheep during the vaccination procedure. The lesions described for Gudair needle-stick injuries are substantially more severe and more serious than those associated with other, more commonly used sheep vaccines. Despite developments to improve the safety of the methods used for Gudair vaccination of sheep, it can be expected that accidental human injuries will continue as a significant and serious risk to those using the vaccine with sheep.

**A-15 Cost of disease nationally**

A recent report has estimated the cost of OJD nationally to be $35m, consisting of $21m in lost productivity and $14m in prevention costs, based on estimates of national OJD prevalence and OJD control strategies in place in 2014. The report acknowledged that losses associated with movement restrictions were particularly severe for studs in states with on-going regulatory control, but did not assign a direct cost to trading restrictions. The model used in the study included an uneven distribution of costs across Australian flocks, with around 80% of the losses arising from 15% of the sheep population in OJD-infected flocks in the regions of Australia with a high OJD prevalence. The estimated cost of OJD nationally was of a similar order of magnitude to that estimated for mastitis, virulent footrot, arthritis, clostridial diseases, liver fluke, pneumonia and cheesy gland (all in the range from $18m to $52m), and significantly lower than that for lice ($81m), perennial ryegrass toxicity ($105m), flystrike ($173m), weaner illthrift and mortality ($188m), dystocia ($219m), internal parasites ($436m) and post-natal mortalities ($540m).

**A-16 Strain types and host specificity**

Using IS1311-PCR analysis, three distinct genotypes of *M ptb* occur in Australia: an S strain that predominantly affects sheep, a C strain that predominantly affects cattle and a B strain reported only once in this country, from cattle in Queensland. There are sub-types of each strain – a number of which are recognised in Australia and some which have not been reported in this country.

Using different genotyping techniques, the S strain types found in Europe have been further subdivided into Type I and Type III strains. With those techniques, C strains are considered to be Type II strains.

S strains are particularly difficult to culture with techniques that are widely used successfully for C strains but the application of the technique of radiometric culture was widely used in Australia from the 1990s until 2012 and facilitated epidemiological studies in this country. Because of the lack of appropriate culture techniques for S strains in many other countries there are very few reports which shed any light on the epidemiology of S strain infections in countries other than Australia. Publications

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6 With a low level of OJD, mortality rates were predicted to fall from 1.5% to 0.2% annually following vaccination. A high level of OJD was modelled with mortality rates initially set at 8%, eventually declining to 0.6% following vaccination.
by Juste and colleagues from Spain (for example, Juste and Perez, 2011) are perhaps the best source of non-Australian information on this topic. Recent information from New Zealand is discussed below.

S strains and C strains lead to different immunological and pathological responses in sheep following infection. Experimental challenge in sheep has led to the inference that S strains are more likely to cause severe, persistent lesions leading to clinical disease while C strain infections are more likely to decrease in severity as the infection progresses. With Angora goats, the opposite may be true, with evidence that they are susceptible to infection with both strains but develop more severe disease, with higher shedding and clinical disease rates with C strain infections than S strain infections.

In this report, it is considered that OJD refers to the disease in sheep, whether it is caused by S strain, C strain or an unidentified strain.

**S strains**

In Australia, the epidemic of ovine Johne’s disease which began in the 1970s (or earlier, perhaps) and was first reported in 1980, has been caused by the S strain of *M. ptb*. There is a strong host preference of S strains for sheep but infection with this strain has been reported in fibre goats and beef cattle in Australia. Cattle appear to be at least partly refractory to infection with S strain but infection of beef cattle with *M. ptb* S strain has been reported from over 20 herds between 2003 and 2013 in regions of south-eastern Australia where sheep and beef cattle share pastures.

Transmission of S strain *M. ptb* from sheep to cattle under natural conditions has been reported in Iceland.

In New Zealand, the herd and flock prevalence of paratuberculosis is high; 76% of sheep flocks and 42% of beef herds are infected with *M. ptb*. Co-grazing of sheep and beef cattle is common in NZ and there is strong evidence for transmission between the two host species. A host preference of Type I strains (S strains) for sheep is reported. Sheep and beef cattle are more commonly affected with Type I strains than with Type II strains (over 80% of isolates from sheep and beef cattle are Type I strains) while deer and dairy cattle are most commonly infected with Type II strains (around 90% of isolates from these two species are Type II strains). The incidence of clinical Johne’s disease in beef cattle is uncommon in New Zealand, suggesting that Type I strains are less virulent for cattle than for sheep.

Considering that, the clustering of Type I strains in sheep and Type II strains in dairy cattle and deer and the difference in prevalence between sheep flocks and beef cattle herds, it seems probable that sheep are the principal source of infection with Type I strains for beef cattle in NZ.

Infection of goats with S strain is reported more commonly in other countries, including Cyprus, Czech Republic, Spain and Greece (reported and cited by Liapi et al., 2015).

In Europe, including Britain, it has long been known that some isolates from sheep are difficult or impossible to culture with techniques in common usage, so the strain of these isolates has not been determined. Given the known difficulties of culturing S strains of *M. ptb* it seems likely that many of these untyped cases are caused by S strains.

Despite the general lack of typing of sheep-derived isolates in Britain, the existence of pigmented strains of *M. ptb* in that country sheds some light on the likelihood that a host preference of S strains does exist there. In animals infected with the pigmented strain, the intestine appears yellow or orange, presumably due to the very large numbers of bacteria in the mucosal lesions. Johne’s disease accompanied by pigmentation of the lesions is reported commonly from sheep but there is only one

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This is the opinion of the author of the NZ study. The lower virulence in cattle compared to sheep may be related to agent or host factors, or both.
report of natural infection of cattle accompanied by pigmentation. A study in Scotland on five isolates of the pigmented strain have found all to be of the S strain, based on IS1311-PCR analysis. This result supports the proposition that genetic characteristics of the S strains which are detected by IS1311-PCR analysis are associated with the host preference of S strains for sheep, even in countries where the disease has been long endemic and where co-grazing of sheep and cattle is common.

C strains
In Australia, cattle, dairy-breed goats and alpacas are most commonly infected with C strains. Infections of sheep with C strains occur less commonly in Australia but are common in other countries. It is not yet clear what forms the basis for the relative host specificity seen so far in Australia. It could be due to limited opportunities for cross-species transmission but that seems unlikely given that bovine JD was reported in Australia over 90 years ago with only one report of an infected sheep before the beginning of the OJD epidemic was recognised in 1980.

The transmission of \( M \text{ ptb} \) from cattle to sheep has been reported in New Zealand although it was not shown that the sheep represented a source of infection to other cattle or sheep. Also in NZ, feral goats have become infected with \( M \text{ ptb} \) of bovine origin under conditions of natural grazing and develop clinical Johne's disease and excrete \( M \text{ ptb} \) in faeces.

A-17 Infections in non-ruminant wildlife
Natural infection of rabbits with \( M \text{ ptb} \) has been reported from Scotland. There was a strong association between the presence of infection in cattle and the detection of infection in rabbits on the same farm. The strains present in the two host species appeared to be the same and although the rabbits were excreting less than 1% of the number of organisms excreted by cattle, they were still considered a possible source of infection to susceptible cattle.

In the Scottish studies, no evidence was found of an association between infection in sheep and infection in rabbits although the usual difficulties in culturing \( M \text{ ptb} \) from sheep in the UK occurred. Infection in rabbits, but at a very low level, has been reported in other countries, associated with infection in cattle.

In Australia, two studies have examined the prevalence of paratuberculosis rabbits on farms carrying sheep infected with OJD. In one study of 300 rabbits in NSW, none was found to be infected. In Victoria 100 rabbits from an OJD-affected sheep farm were examined – again with all negative results. In the same study, 210 rabbits from two properties carrying JD-infected cattle were also examined with negative results.

Three studies have sought to estimate the prevalence of paratuberculosis in macropods exposed to pastures contaminated by OJD-affected sheep flocks. In Victoria and NSW, a total of 400 Eastern Grey kangaroos were examined. No evidence of active infection was detected but one kangaroo in NSW had a positive faecal culture, despite the absence of any detectable histopathological lesion in the intestinal tissue.

On Kangaroo Island, 785 Tammar Wallabies and 55 Western Grey kangaroos were examined. Two animals had histopathological lesions of paratuberculosis but all faecal cultures were negative. The authors concluded that excretion of significant numbers of \( M \text{ ptb} \) organisms from macropods is rare and that they do not represent a reservoir of infection for sheep.
A-18 Survival of the organism

Transmission of paratuberculosis between animals is by the faecal-oral route and so the length of survival of the organism in faeces or free in soil or water is important in determining the risk of transmission between animals.

*M. ptb* is considered to be an obligate parasite which cannot multiply outside an infected host. Unlike other members of the *M. avium* species, *M. ptb* cannot produce the iron-chelating compound *mycobactin* that enables it to acquire iron from the environment. It is believed that this mycobactin-dependence renders the organism dependent on the cells of an infected host, or mycobactin-enriched culture media, in order to multiply. (There is, however, some evidence that mycobactin is not always necessary for growth on media and that some media will support growth in the absence of mycobactin.)

*M. ptb* can survive, presumably without multiplication, for extended periods in faecal pellets and on soil, pasture and in water. In common with other *M. avium* subspecies, *M. ptb* is well-adapted to aquatic environments. Reports of its survival in spiked water samples, summarised by Collins (2003), indicate maximum survival times up to 517 days (17 months). The longest survival times were associated with favourable conditions – darkness, constant warm temperature and neutral pH.

Studies in Australia with the S strain of *M. ptb* indicate that the maximum survival time in exposed and dry sites is typically less than 32 weeks but up to 55 weeks in a dry, fully-shaded site. The organism was found to survive in the sediment of trough water, sourced from a dam, for 48 weeks. The implications of these findings for the South Australian environment are discussed further in Section C.

The likelihood that an environmental site has sufficient living *M. ptb* bacteria to cause an infection in a susceptible animal is influenced both by the environmental conditions and the number of bacteria which contaminate the site. The mortality of the bacteria in the environment follows a logarithmic curve, rather than a linear decline or a sudden contemporaneous disappearance after a critical duration. Estimates of the rate of decline vary around a rate of 1 log per month over extended periods but the possibility of a biphasic rate of decline – 5 logs in the first month then a much slower rate thereafter – has also been proposed. As a consequence of the pattern of logarithmic decline, it can be confidently proposed that the greater the number of bacteria present in a site when it is first contaminated, the greater the chance that an infective dose of bacteria could be ingested from the site at any time in the subsequent months.

Factors which increase the environmental survival time are water and aquatic sediments, shade and geographic area (NSW tablelands vs western NSW).

Factors which decrease the survival time are direct exposure to the sun, high environmental temperatures (western NSW), and a lack of vegetation or shade.

In summary, studies in Australia and overseas, with S strain and C strain examples of the species, support the view that survival of the organism outside a host and free in the environment is finite but prolonged. While the number of bacteria which survive declines very rapidly over the first few weeks, some organisms can survive for a year in favourable sites and represent potentially a source of infection for a susceptible host. Given that dams and water courses on farms are frequently heavily contaminated with sheep faeces and that the survival of the organism is prolonged in such environments, low-lying areas of farms should be considered as areas of high risk for survival of the organism.

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1 This observation is included only as an illustration of the maximum survival time under suitable conditions.
bacteria. Nevertheless, based on the studies reported to date, survival of the organism at levels which could lead to new infections is unlikely for periods greater than 18 months.

As a corollary, it is therefore widely accepted that the organism is not present in sites or on farms which have not previously been contaminated by infected hosts, or have not been grazed by infected hosts for periods greater than 18 months.

The presence of shade and moisture on farms is a characteristic of regions in the medium and high rainfall zones (>400 mm annual rainfall) of southern Australia (Figure 8). These regions also support the higher stocking densities which are likely to favour *M. ptb* transmission between sheep. It is not surprising, therefore that those regions of Victoria and NSW in the medium and high rainfall zones are the same regions in which OJD exists at a high flock prevalence, and suggests that the similar regions of SA (South-East, Adelaide Hills/Fleurieu and Kangaroo Island) are also suitable for the transmission of OJD. These regions of South Australia contain around 42% of the sheep in the state.

In contrast to NSW and much of Victoria, South Australia and southern Western Australia have a Mediterranean climate, characterised by hot, dry summers. This extended period of heat, without rainfall, is likely to also reduce the opportunities for survival of *M. ptb* in the environment on many farms in SA, particularly in the regions of medium and lower rainfall. In addition to the direct effects of rainfall, some SA farms in the medium and high rainfall zones conduct cropping enterprises in association with sheep production – a factor which is also likely to reduce the levels of *M. ptb* to which sheep are exposed.

### A-19 Methods of detecting paratuberculosis

**Abattoir surveillance**

Abattoir surveillance can serve two functions in OJD control programs. One is to detect flocks with OJD so that action can be taken to reduce the prevalence in the detected flock and to impose restrictions on trade with a view to reducing spread to uninfected flocks. The second is to allow an estimation of the prevalence of infected flocks in a region.

In a regulatory environment, abattoir surveillance will only be effective as a control measure if the surveillance includes a large proportion of the sheep flocks in the region. The effectiveness of the control is also influenced by the sensitivity of abattoir inspection in detecting infection in a flock, particularly when infection in the flock is recent and therefore at a low level of expression.

The second, however, is not dependent on achieving inspection of a high proportion of a region’s flocks, provided there are (a) sufficient numbers of flocks inspected to provide a reliable estimate and (b) there is randomness in the selection of flocks inspected in relation to the likelihood of OJD being present. Estimates of prevalence can then be made based on the expected sensitivity of abattoir surveillance.

**Sensitivity of abattoir surveillance**

Two Australian studies have reported on the sensitivity of abattoir surveillance for detection of OJD. It is clear that trained inspectors are able to detect gross pathology in a high proportion (74% and 87% in the case of two inspectors) of sheep which have OJD lesions detectable by histopathology. Training is important, and untrained inspectors perform with a significantly lower sensitivity.

Despite the relatively high sensitivity of detection of an infected sheep by a trained inspector, a number of other factors strongly influence the likelihood that an infected flock will be detected. These include the within-flock prevalence of the disease and the line size. Within-flock prevalence tends to be low in regions where the disease has been recently introduced because many infected flocks will
be in the early stages of within-flock spread. Line size is strongly related to flock size, so infected small flocks are less likely to be detected than large flocks, and less likely to be detected early in the development of a flock infection.

Abbott and Whittington (2003) concluded that abattoir surveillance will have a high probability (≥95%) of detecting an infected flock from the examination of one abattoir line only if the prevalence in the flock is ≥7%. When within-flock prevalence is ≤2%, detection is much less likely. They concluded that, unless abattoir surveillance strategies were amended or supported by other surveillance activities, it will fail to detect nearly half of the infected flocks in low-prevalence regions. As a consequence, a control program will fail to achieve adequate control of the spread of infection in a region in the early stages – when control is most likely to be effective.

One of the limiting factors identified by those authors for an acceptable flock-level sensitivity in the early stages of a disease was the limit on the number of specimens submitted for histopathology from a suspect line. In low prevalence flocks, the rate of false-positive gross lesions can rival or exceed the rate of true positive lesions. Provided line size is high, flock level sensitivity in low prevalence flocks (<2%) may be increased substantially by lifting the upper limit on submissions from three to six. If line size is low, little can be done to improve sensitivity at low prevalences.

The relationship between likely flock-level sensitivity, within-flock prevalence, line size and upper limit on submissions is illustrated in the following figure (Figure 2), reproduced from Abbott and Whittington (2003).

![Figure 2](image-url)

**Figure 2:** A computerised simulation of abattoir surveillance, based on the NSW abattoir surveillance program, examined the likelihood that an infected flock would be detected under varying conditions of within-flock prevalence, line size and the upper limit on specimen submission per line.
Bacterial culture
Bacterial culture is the most sensitive test for detection of animals infected with *M. ptb*. Radiometric culture (*Bactec™*) was widely used in Australia during the period when tests were being developed and reported and the estimates of sensitivity were done with that technique. Across Australia, radiometric culture has been replaced in the last few years by non-radiometric culture on liquid medium, reportedly of similar levels of sensitivity to radiometric techniques. Culture of the organisms from intestinal tissues is more sensitive than from faeces or soil.

Pooled faecal culture
The pooled faecal culture test (PFC), utilising multiple pools of faeces from 50 sheep per pool, is the preferred test for detection of OJD in sheep flocks in Australia and is the only test suitable for use in vaccinated flocks (where serology may give false positive results as a consequence of vaccination).

Sensitivity and specificity of PFC tests
The sensitivity of the PFC test is influenced by two independent probability functions. First, the probability that a pool does actually contain the faeces from an infected sheep (a true positive pool) and, second, the probability that the culturing process actually detects the organism if it is present in the pool.

The first of these probabilities is simply a matter of chance although the probability of including an infected animal in a pool can be increased if low-condition score animals are favoured in sampling. The relationship between the probability of including at least one infected animal in the tested sample and flock size, the number of animals tested (the size of the sample) or the prevalence of disease in the flock is illustrated in Figures 3 and 4. Increasing the number of animals tested, by increasing the number of pools, has a marked effect on increasing the likelihood that an infected animal is included, particularly at prevalences below 1%.

For comparison purposes, it should be noted that in large flocks with a prevalence of 2%, the probability of including at least one infected animal in a sample size of 350 is 99.9%.
Figure 3: The probability of including an infected sheep at a true prevalence of 1%. The probability of including at least one infected animal in the tested sample is influenced by flock size, but exceeds 99% if 450 sheep are tested in flocks up to 10000 sheep.

Figure 4: The probability of including an infected sheep in a flock of 10000 varies with the true prevalence. For low prevalence infections, the probability of including at least one infected animal in the tested sample is strongly influenced by the true prevalence when fewer than 600 animals are tested.

The second probability (that an infected animal is detected by the culturing process) is most strongly influenced by the number of organisms being excreted by the infected animal(s) in the pool.

This probability is highest when sheep with multibacillliary forms of OJD are included in the pools. The probability is lowest when the infected sheep included in the pools have only the paucibacillliary form of OJD or are in the early stages of disease. If only paucibacillliary forms of the disease are present in the flock, increasing pool size beyond 50 sheep may in fact reduce sensitivity by increasing the dilution of positive specimens in the pool73.
The sensitivity of the PFC test is effectively the combination of the two probabilities. In one study to estimate the sensitivity, flocks were grouped into high (≥2%) or low (<2%) prevalence categories. The sensitivity of the PFC350 (seven pools of 50 sheep) was estimated to be around 92% in the high prevalence group and 82% in the low prevalence group.²⁴

These results mean that there is a significant probability, perhaps greater than one in five, that a flock with a prevalence of infection below 2% will not be detected by the performance of one PFC350. If the sheep which are infected are only excreting *M. ptb* in low numbers (paucibacillary infection), the sensitivity is further compromised. Increasing the number of animals tested (more than seven pools) will increase the sensitivity of the flock test, and is especially relevant in large flocks if the prevalence is expected to be low.

The HT-PCR (High-Throughput direct faecal PCR assay) is now used as a screening test for OJD on faecal pools, with negative results being interpreted in the same way as negative PFC culture tests. The HT-PCR is thought to have a similar sensitivity for *M. ptb* as culture, although it may detect some animals not detected by culture and fail to detect some animals which are detected by culture.²⁵

This difference in the performance of the two tests means that any requirement that both tests are positive before a diagnosis is confirmed would further decrease the sensitivity of the (combined) testing procedure.

**A-20 Association between *M. ptb* in animals and Crohn’s disease in humans.**

Crohn’s disease in humans is a chronic inflammatory condition of the alimentary tract which most frequently involves the ileum and colon. Pathologically, the condition has many features in common with Johne’s disease of animals and *M. ptb* DNA has been identified in the intestinal tissues of some Crohn’s patients. Crohn’s disease does not, however, develop into a pathological form in which *M. ptb* bacteria are clearly evident in intestinal lesions or readily cultured from the affected tissues as is the case with Johne’s disease of ruminants. The disease in humans is marked by a particularly florid expression of an immune response in the intestinal tissue to an unknown antigen. A number of researchers and human clinicians consider that *M. ptb* is one of the possible triggers for the development of Crohn’s disease in humans.

The association of *M. ptb* with some cases of Crohn’s disease is clear. A recent systematic review and meta-analysis of 47 published studies found that *M. ptb* was detected more frequently in the gut of patients with Crohn’s disease than with patients without the disease. *M. ptb* is not, however, an essential part of the pathogenesis of Crohn’s disease - Crohn’s disease has occurred in parts of the world where *M. ptb* is absent and, in many cases of Crohn’s disease, there is no evidence that *M. ptb* is, or was, present.

The opportunity for *M. ptb* to be involved with some cases of Crohn’s disease is also clear. It is known that *M. ptb* is an occasional contaminant of human foods of animal origin, including milk, even if pasteurised and humans may be exposed to the organism through contaminated water or directly from infected animals.

Despite the association and the opportunity, there is no evidence that *M. ptb* is a cause of Crohn’s disease. It is a bacterium which is associated with the disease in some cases. The role of *M. ptb* in the pathogenesis of Crohn’s disease is still debated in the human medical field. The reader is referred to some recent reviews which present and discuss the evidence on both sides of the debate, including the zoonotic potential of the organism.²⁹,³⁰,³¹,³²,³³.
SECTION B  OJD IN SOUTH AUSTRALIA

B-1 Total detections in SA
OJD was first diagnosed in SA in 1997. In the 20 years since that time there have been 194 flocks detected with OJD in South Australia (Figure 5). A small number of those, estimated at 5 or 6, have been detected a second time after achieving a clearance test to allow freedom from restrictions. The number of unique property detections is therefore around 190.

In the 10 years since 2007, 62 flocks have been detected – an average rate of 6.2 per year. Assuming that there are 13,037 sheep flocks in SA, OJD has been detected in 1.5% of these.

Figure 5: The number of new detections of OJD in South Australia has risen at an average rate of around 10 per year since 1997.

The disease has been detected most frequently in two regions; Kangaroo Island and the South-East.

The pattern of detections suggests that the relatively high number found in Kangaroo Island in the first 10 years of the disease history in SA reflects the detection of an accumulation of infected flocks which had occurred over the preceding years. Kangaroo Island flocks have also been subject to more intense surveillance activities than other regions of the state, which is likely to have contributed to the higher detection rate there.

Eleven detections in the South-East in 2001 arose from trace-forwards from one South-East sheep stud. The occurrence of eight flocks with OJD (caused by C strain) in the mid-North region in 2001 and 2004 related to infection in uncontrolled deer and added to the early phase of the SA epidemic.

Since 2007, the rate of new detections has been low in all regions except the South-East which region accounts for most of the new detections in the past decade (Figure 6).

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1 Data gathered from Dr Nosworthy on 1 July 2016
In contrast to NSW and Victoria, South Australia has a very low number of known-infected flocks. In 2012, (the most recent data available from Animal Health Australia) there were 1286 infected flocks reported in NSW, 685 in Victoria, 64 in Tasmania and 41 in WA. While these data may not be directly comparable because of differences between states in recording and reporting strategies, they do indicate the high prevalence of infected flocks detected in NSW and Victoria at that time (4 to 5 years ago).

If all 190 flocks in SA that have been detected to date were considered to be still infected, the prevalence of infected flocks in the state as a whole is 1.5%. The figure is higher for the South-East region (2.6%) and for Kangaroo Island (26%) (Table 4).

**B-2 Release from Orders (quarantine) in SA**

In July 2016, 50 flocks with confirmed OJD remained under Order in SA. A further six flocks were under investigation and remained Suspect. Assuming that there are 13,037 sheep flocks in SA\(^k\), 0.4% are currently under Order.

Approximately 144 flocks have been released from Orders since 1997. The 50 flocks with confirmed OJD remaining under Order have been so for an average of 4.6 years (Figure 7). The median number of years in quarantine for the currently quarantined flocks is between two and three years.

Flocks which have been released from an Order in South Australia are those which have completed a PDMP-V or a PDEP and have subsequently ‘passed’ a clearance test – a negative result on a pooled faecal culture from 350 sheep in the flock (in flocks of that size or greater). In larger flocks, it has been customary to sample a greater number of sheep in the flock.

At the time of the collection of faecal specimens for clearance testing, flocks consist of approved vaccinates and the properties on which they run are likely to have very low levels of *M. ptb* contamination – possibly zero levels. The PFC test is a sensitive test for OJD but is not 100% sensitive. The likelihood of detecting infection is affected by the prevalence of OJD in the flock – and particularly the presence of at least one multibacillitary case of OJD amongst the sample population. Flocks which

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\(^k\) Number of active sheep PICS Jan 2012 to December 2015. Source, Dr Mary Carr
have pursued a PDEP or PDMP-V are likely to have very few, if any sheep which are shedding *M. ptb* at levels which are readily detectable by PFC testing. In such flocks, the sensitivity of PFC testing is low, and there is a significant chance that flocks in which infection persists are not detected at clearance testing and are released from Order despite the continuing presence of *M. ptb* in the flock – albeit at very low levels.

Once a flock has ‘passed’ a clearance test it is not subject to any further targeted surveillance. There is, therefore, no further opportunity to re-classify a ‘false-negative’ flock except through abattoir surveillance. If flocks released from Orders continue to vaccinate with Gudair, the probability of detecting the presence of a low level of infection through (untargeted) abattoir surveillance is very low.

For the purposes of this report, flocks which have been released from a quarantine Order will be considered to be still infected. It is accepted that these flocks, if infected, have a low prevalence of infected sheep and a very low prevalence of high-shedders. Nevertheless, the continuing presence of *M. ptb* in the flock and on the property constitutes an ongoing risk of spread to other properties. It is also accepted that some of the flocks released from an Order may be truly free of infection, particularly those which have undergone a PDEP through de-stocking. There is, however, no information from any part of Australia to support the proposition that any flock which has undergone a PDEP and re-stocked – without on-going vaccination – has remained free of OJD beyond three years. On the contrary, there is evidence that most flocks (68%) which have attempted to eliminate the organism by destocking and restocking failed within three years.\(^\text{45}\)

On this basis, in this report, the number of known-infected flocks in SA will be considered to be the number of total detections, rather than the number currently under Order. This may be an overly pessimistic conclusion but the evidence available to date suggests that any consequent overstatement of infected flocks is likely to be quite small.

### Table 4: Prevalence of infected flocks (PICS) in SA by region

<table>
<thead>
<tr>
<th>Region</th>
<th>Sheep active PICS</th>
<th>OJD detections since 1997</th>
<th>Currently under Orders</th>
<th>Currently suspect</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adelaide Hills/Fleurieu Barossa/Lower North/ Murray Mallee</td>
<td>5879</td>
<td>11</td>
<td>5</td>
<td>2</td>
</tr>
<tr>
<td>Eyre Peninsula</td>
<td>1278</td>
<td>1</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Kangaroo Island</td>
<td>378</td>
<td>100</td>
<td>14</td>
<td>2</td>
</tr>
<tr>
<td>South-East</td>
<td>2812</td>
<td>73</td>
<td>30</td>
<td>2</td>
</tr>
<tr>
<td>Northern/Pastoral</td>
<td>756</td>
<td>Presume zero</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yorke Peninsula/Mid North</td>
<td>1934</td>
<td>9</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>
B-3 Use of Gudair vaccine in South Australia
Sheep producers are likely to use Gudair vaccine to control OJD in their flocks after the disease has occurred, or because they consider the risk of contracting OJD in their flocks is sufficiently high to warrant the additional expenditure. In addition, some producers will vaccinate sheep in the absence of disease or high risk of disease because there is a market advantage in offering vaccinated sheep for sale. This latter group is probably substantially smaller than the group of producers who see OJD as a proximate threat. The amount of Gudair vaccine used in each state of Australia can therefore be a guide to the perceived (and actual) threat posed by OJD to sheep producers.

In the seven years since early 2009, 1.15 million doses of subsidised vaccine have been supplied to producers in South Australia – an average of around 160,000 doses per year.

In addition to the vaccine subsidised by the SASAG-controlled Industry Fund, an additional quantity of vaccine is purchased and used privately in SA. The total vaccine used in SA each year in 2013, 2014 and 2015 has been around 460,000 doses per year, including subsidised vaccine. This level of vaccine usage suggests that lambs from about 7% of the state’s ewes are vaccinated each year, or about 14% of the ewes mated to Merino rams each year (Table 5). (I recognise that some crossbred ewe lambs are likely to be vaccinated but, the numbers of pure Merino matings are used as an indicator of the differences between the level of vaccine usage in states with different numbers of breeding ewes and different levels of OJD prevalence.) For comparison, note that there is an apparently high frequency of vaccine usage in Tasmania and Victoria, both states with substantial sheep numbers in medium to high rainfall zones and with a relatively high flock prevalence of OJD infection. Usage in WA and SA is relatively low. NSW, despite the high prevalence of OJD in the east of the state, still has extensive areas of low OJD prevalence in the north and west.
### Table 5: Comparison of ewes mated with predicted breeding intentions in 2014 (from MLA and AWI Wool and Sheepmeat survey; [http://www.wool.com/market-intelligence/wool-and-sheepmeat-survey/](http://www.wool.com/market-intelligence/wool-and-sheepmeat-survey/)) to doses of Gudair vaccine sold in each state in 2015. The ratio is made between vaccine doses and ewes mated to Merino rams as a proxy for the likely number of lambs which will be born and retained beyond 12 months of age. Vaccine usage data are provided by Zoetis Animal Health.

<table>
<thead>
<tr>
<th></th>
<th>NSW</th>
<th>Vic</th>
<th>SA</th>
<th>WA</th>
<th>Tas</th>
<th>Qld</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ewes for pure Merino lambs (million)</td>
<td>7.8</td>
<td>3.2</td>
<td>3.2</td>
<td>5.5</td>
<td>0.5</td>
<td>1.5</td>
</tr>
<tr>
<td>Ewes for other types of lambs (million)</td>
<td>7.1</td>
<td>4.8</td>
<td>3.1</td>
<td>2.7</td>
<td>0.7</td>
<td>0.6</td>
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<tr>
<td>Vaccine doses sold (million)</td>
<td>1.76</td>
<td>2.13</td>
<td>0.46</td>
<td>0.44</td>
<td>0.40</td>
<td>-</td>
</tr>
<tr>
<td>Ratio of vaccine doses to ewes mated to Mo rams</td>
<td>23%</td>
<td>67%</td>
<td>14%</td>
<td>8%</td>
<td>80%</td>
<td>-</td>
</tr>
</tbody>
</table>

### B-4 OJD surveillance in South Australia

The rate of new detections in SA remains low at around six flocks per year. New detections occur largely as a result of two activities by the regulatory authority; abattoir surveillance and tracing of sheep movements to and from infected properties, which may include testing of neighbouring farms. In recent years, five flocks have been placed under Order following notification from private veterinarians, and two following a positive finding during testing for the MAP.

In the past four years (possibly longer) the rate of flocks being placed under Order has approximately matched the rate of new detections resulting from abattoir surveillance. In other words, abattoir surveillance has been the critical tool\(^1\) in detection of infected flocks.

### B-5 Abattoir surveillance in SA

In South Australia, abattoir surveillance for OJD is currently performed at two abattoirs – in Lobethal and Murray Bridge. Surveillance has been carried out for several years but training of inspectors specific to detection of OJD was not carried out until 2014. Training is reported to have increased the confidence of Biosecurity SA in the ability of inspectors to recognise OJD lesions.

In the four years 2012-2015 inclusive, at least one line from 4,585 unique PICS was inspected at SA or interstate abattoirs. This represents 38% of the 13,037 sheep-carrying properties in SA or 35% of the PICS reported to be sheep-active at that time (Table 6).

A high proportion of properties from Eyre Peninsula (70%) and Kangaroo Island (78%) have had some level of abattoir surveillance.

The proportion of sheep-owning properties which have been tested is at medium levels for the regions of the Upper South-East (45%), Yorke Peninsula/Mid-North (43%), Murray Mallee (43%), Mid-South-East (41%) and Northern Pastoral (40%).

The proportion of properties from which sheep have been inspected is relatively low for Adelaide Hills/Fleurieu, Barossa/Lower North and Lower South-East regions.

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\(^1\)Abattoir surveillance is considered the critical tool because without it, detections and subsequent investigations would be based only on the small number of notifications from animal health officers or private veterinarians investigating disease at the owner’s request.
Table 6: The proportion of South Australian PICS from which lines of sheep have been inspected at SA or interstate abattoirs in the four years 2012-2015. There is overlap in the number of PICS inspected in SA and PICS inspected interstate. For the period 2012 to 2015 the number of unique South-East PICS inspected in SA and interstate abattoirs was 75% of the sum of the two figures.

<table>
<thead>
<tr>
<th>Region</th>
<th>Active sheep PICS</th>
<th>PICS inspected in SA</th>
<th>PICS inspected interstate</th>
<th>PICS inspected at either</th>
<th>% PICS inspected</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adelaide Hills/Fleurieu</td>
<td>2411</td>
<td>253</td>
<td>21</td>
<td>265</td>
<td>11%</td>
</tr>
<tr>
<td>Barossa/Lower North</td>
<td>1764</td>
<td>262</td>
<td>12</td>
<td>267</td>
<td>15%</td>
</tr>
<tr>
<td>Eyre Peninsula</td>
<td>1278</td>
<td>891</td>
<td>24</td>
<td>896</td>
<td>70%</td>
</tr>
<tr>
<td>Kangaroo Island</td>
<td>378</td>
<td>294</td>
<td>75</td>
<td>295</td>
<td>78%</td>
</tr>
<tr>
<td>Lower South-East</td>
<td>1062</td>
<td>185</td>
<td>109</td>
<td>223</td>
<td>21%</td>
</tr>
<tr>
<td>Mid South-East</td>
<td>921</td>
<td>229</td>
<td>281</td>
<td>381</td>
<td>41%</td>
</tr>
<tr>
<td>Murray Mallee</td>
<td>1704</td>
<td>716</td>
<td>115</td>
<td>740</td>
<td>43%</td>
</tr>
<tr>
<td>Northern/Pastoral</td>
<td>756</td>
<td>298</td>
<td>24</td>
<td>303</td>
<td>40%</td>
</tr>
<tr>
<td>Upper South-East</td>
<td>829</td>
<td>289</td>
<td>205</td>
<td>376</td>
<td>45%</td>
</tr>
<tr>
<td>Yorke Peninsula/Mid North</td>
<td>1934</td>
<td>832</td>
<td>52</td>
<td>839</td>
<td>43%</td>
</tr>
<tr>
<td><strong>TOTAL</strong></td>
<td><strong>13037</strong></td>
<td><strong>4249</strong></td>
<td><strong>918</strong></td>
<td><strong>4585</strong></td>
<td><strong>35%</strong></td>
</tr>
</tbody>
</table>

As a consequence of these inspections in 2012-2015, 54 abattoir surveillance detections occurred leading to further investigations. Of those 54, 13 flocks were already under Order and a further 24 SA flocks were found to be infected and placed under Order (Table 7).

In 2012, there were five new abattoir detections and six properties were determined to be infected. In 2013, there were three new abattoir detections but 12 properties went under Order. No new abattoir surveillance detections occurred in the last quarter of 2013. In 2014 nine properties were new detections from abattoir surveillance. Five properties were placed under Order in 2014 so presumably at least four of the new abattoir detections were carried over into 2015 before confirmation. All five properties were in the south-east. In 2015, five properties were new detections from abattoir surveillance and two further properties remain under investigation. Nine properties were placed under order. Presumably the extra four were carried forward from 2014. Six of the nine properties placed under order were from the South-East.

Table 7: The outcome of abattoir surveillance for OJD in SA 2012-2015 inclusive. 54 PICS were detected through surveillance and, as a result, 24 flocks were placed under Order. Data provided by Dr Peter Nosworthy.

<table>
<thead>
<tr>
<th>Year</th>
<th>PICs detected</th>
<th>Already under order</th>
<th>Cleared after histopathology or on farm inspection</th>
<th>Outcome not stated or flock in Victoria</th>
<th>New detections</th>
</tr>
</thead>
<tbody>
<tr>
<td>2012</td>
<td>9</td>
<td>3</td>
<td>1</td>
<td>1</td>
<td>5</td>
</tr>
<tr>
<td>2013</td>
<td>8</td>
<td>2</td>
<td>3</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>2014</td>
<td>25</td>
<td>8</td>
<td>7</td>
<td>1</td>
<td>9</td>
</tr>
<tr>
<td>2015</td>
<td>12</td>
<td>0</td>
<td>3</td>
<td>2</td>
<td>7*</td>
</tr>
<tr>
<td><strong>TOTAL</strong></td>
<td><strong>54</strong></td>
<td><strong>13</strong></td>
<td><strong>14</strong></td>
<td><strong>3</strong></td>
<td><strong>24</strong></td>
</tr>
</tbody>
</table>

*2 of these detections not yet confirmed by follow-up testing at time of data collection
For the South-East region, where the highest level of new detections have occurred in recent years, it is instructive to examine the intensity of abattoir surveillance in that region specifically (Table 8).

Table 8: The intensity of abattoir surveillance in the South-East in 2015. While 1933 flock PICS had recorded sheep movement, only (an estimated) 419 of these were subject to abattoir surveillance.

<table>
<thead>
<tr>
<th>Region</th>
<th>Flocks with sheep</th>
<th>Flocks with NLIS movements in 2015</th>
<th>PICS with mutton lines inspected in SA in 2015</th>
<th>PICS inspected interstate in 2015*</th>
<th>PICS inspected in SA and interstate (75% of the sum)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lower SE</td>
<td>1062</td>
<td>585</td>
<td>73</td>
<td>50</td>
<td>92</td>
</tr>
<tr>
<td>Mid SE</td>
<td>921</td>
<td>695</td>
<td>80</td>
<td>145</td>
<td>169</td>
</tr>
<tr>
<td>Upper SE</td>
<td>829</td>
<td>653</td>
<td>121</td>
<td>90</td>
<td>158</td>
</tr>
<tr>
<td>TOTALS</td>
<td>2769</td>
<td>1933</td>
<td>274</td>
<td>285</td>
<td>419</td>
</tr>
</tbody>
</table>

* Interstate lines include lines of lambs in an unknown proportion

If one assumes that five of the six South-East new detections confirmed in 2015 were based on abattoir surveillance carried out in 2015, then five of the (estimated) 419 SE PICS inspected in 2015 were found to be infected based on abattoir surveillance and subsequent flock testing (1.2%). Interstate inspection lines include an unknown number of lines of lambs so the estimated detection rate in lines of adult sheep could lie between 1.2% and 1.8% (5/274).

The sample size (419 flocks) is sufficiently large for the purpose of estimating of the incidence of new flock infections in the South-East – provided an assumption is made about the sensitivity of abattoir surveillance as practised for SA flocks and, additionally, that there is no relationship between the likelihood of OJD infection and the submitting of sheep to abattoir surveillance.

It is beyond the scope of this report to make a refined estimate of the prevalence of infected flocks in SA and particularly in the South-East region. Furthermore, the available data lack granularity – particularly with regard to the total numbers of adult sheep from each flock which are inspected in any one year. These are data which could provide additional insight into the likely sensitivity of abattoir surveillance across the region. Nevertheless, some crude estimates are possible:

Given the fact that less than one quarter of the flocks in the South-East were subject to surveillance one could predict that a further 15 to 20 South-East flocks remained undetected by abattoir surveillance.

The sensitivity of abattoir surveillance (the likelihood of detecting an infected flock) is strongly affected by the within-flock prevalence and the line size. If the within-flock prevalence is low and line size is small, the sensitivity of detection could be as low as 20%. If a sensitivity of 50% is assumed, the five detected flocks and 15-20 undetected flocks are matched by a further 20-25 flocks which are infected but escaped detection through abattoir surveillance.

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* It is possible, of course, that producers with infected flocks that have not been detected may actively avoid sending sheep to abattoirs where surveillance for OJD occurs. There is no evidence for this, and the opinion of Mr Laryn Gogel, stock agent of Southern Australian Livestock, Naracoorte, and member of the SA OJD Advisory Committee is that Victorian abattoirs are chosen as a destination for livestock by SA producers on the basis of their farm location and the price offered by the abattoir.
In summary, based on the following assumptions,

- five South-East flocks were detected with OJD in 2015 based on abattoir surveillance
- about one quarter of the flocks in the South-East were subject to abattoir surveillance in 2015
- abattoir surveillance of South-East flocks has a sensitivity of 50%
- the flocks which were subject to abattoir surveillance did not represent a biased sample with respect to OJD status

one could conclude that, while five flocks were newly detected in the South-East in 2015, a further 35 to 45 remained undetected. The five new flock infections in the South-East represented around one eighth of the total number of new cases that would have been detected in the region if all flocks had been subject to a more highly sensitive form of surveillance.

This conclusion does not imply that the prevalence of infected flocks in the South-East is eight times higher than current detections. A number of these infected but undetected flocks will be detected in the following few years, as a consequence of abattoir surveillance detections or trace-back investigations. The conclusion does imply, however, that a significant number of flocks in the region will remain undetected for prolonged periods, during which they will represent a source of infection to other uninfected flocks.
SECTION C COMMENTARY

C-1  The current approach to OJD control in SA

The evidence attesting to the true prevalence of OJD on mainland South Australia is limited, and broad inferences only can be made, largely based on abattoir surveillance – the limitations of which have been discussed and are discussed further below.

The prevalence of OJD on Kangaroo Island (around 26% of flocks are infected) is known with some confidence, as a result of a relatively high level of active surveillance in past years. The prevalence of OJD-infected flocks in the regions of the South-East is not known with any confidence because surveillance has been limited. The true prevalence is likely to be substantially higher than the prevalence of known-infected flocks (2.6%, including flocks released from Orders). By comparison to Victoria and NSW, the prevalence in the South-East is probably still relatively low.

Despite the limited evidence, the information that is in existence does indicate that the prevalence of infected flocks in other parts of mainland SA remains very low.

The question that can be asked, therefore is, “Is the prevalence low because of the regulatory activity of Biosecurity SA, the trading patterns in SA (largely from north to south and east) which do not favour disease spread from the higher prevalence zones and states, or because of environmental and sheep management factors which are not favourable for the transmission of the disease?”

The answer to this question will inform the debate about the best way to manage OJD in South Australia in future. It is likely that all of these factors have contributed to some degree to the current situation in SA but it is not possible to conclude with any confidence how much each factor is responsible. It will be useful to examine these factors one by one.

Regulatory activity has led to the detection of OJD in around 90 mainland flocks. Once infection is known to exist in the flock, trading in sheep is restricted such that the opportunity for further spread (to other South Australian flocks) is dramatically reduced. Neighbouring flocks are inspected and tested if deemed necessary. Tested or not, neighbouring flock owners are made aware of the risk of the proximity of the infected flocks. A PDMP or PDEP is put in place on infected farms and restrictions on trade are not removed until the flock is tested and the level of infection found to be undetectably low, as a consequence of a de-stocking program, vaccination program or both. These regulatory activities have dampened the disease in SA flocks when it has been detected – actions which have reduced the impact of the disease on affected farms and have contributed to a slowing in the rate of OJD spread to uninfected flocks.

Furthermore, regulatory activity has restricted trade in sheep from high prevalence regions in other states of Australia into South Australia.

There is no proof that these strategies have reduced spread but the conclusion is logical and the experience of similar control approaches with other diseases supports the fact. Modelling by AusVet in 2006 supported the effectiveness of the current program. One could infer from that model that, even if OJD was de-regulated immediately, the prevalence of infected flocks in 2035 would be 30 percentage points lower in 2035 than if it had been de-regulated in 2006. The past 10 years of regulatory control have bought an extended period of freedom from OJD for many SA producers, while the industry has gained considerable experience in the management of the disease locally, regionally and nationally.

On the basis of the trade restrictions and disease-limiting strategies that have been applied to date, it is reasonable to conclude that the current SA control program has reduced the rate of spread of OJD
to uninfected flocks in SA, compared to the rate of spread which would have occurred in the absence of any regulatory control.

**Trading patterns** for sheep in South Australia have generally followed a north to south and south-east pattern, with sheep moving from the Merino breeding flocks of the pastoral regions to the sheep-wheat (medium rainfall) zones and the high rainfall. There is also substantial local movement within regions.

Traditionally, there has been very limited movement of sheep from the high prevalence OJD zones in the eastern states to the wheat-sheep or pastoral zones of South Australia and these zones in SA appear to remain largely free of OJD. Movement of sheep from the northern and western zones of SA has therefore presented a very low risk of OJD introduction to the central and south-east regions.

**The climatic characteristics of SA** vary significantly across the state and are different from the high-prevalence OJD areas of NSW and Victoria in a number of respects. Parts of South Australia have an average annual rainfall similar to that of the central and southern Tablelands and western slopes of NSW and the higher rainfall parts of Victoria where OJD is endemic and at a high flock-level prevalence (Figure 8). The pattern of rainfall is, however, different from most of NSW. South Australia has a winter rainfall pattern (Figure 9) and extended hot dry periods during summer.

![Figure 8: Areas of southern Australia with average annual rainfall greater than 500 mm form a zone of medium and high rainfall which is conducive to the transmission and survival of *M. ptb*. Sheep run in these areas are managed at stocking densities which also favour disease transmission between sheep and between flocks. Source: Australian Bureau of Meteorology.](image-url)
Regional climatic factors are likely to be relevant to the establishment, development and persistence of OJD infection in a sheep flock. The organism survives for shorter periods in a hot dry environment than in cooler, shaded and moist environments. Furthermore, sheep stocking densities are lower in regions of low annual rainfall and, in the medium rainfall zones of all states, sheep grazing is often done in association with cereal grain production, further lowering average sheep densities. The transmission rate of OJD within flocks is likely to be lower when sheep densities are lower.

These factors – both low annual rainfall and hot, dry summers – are likely to reduce the rates of transmission of OJD within flocks and reduce the level of challenge faced by young sheep compared to that seen in cooler or higher rainfall zones. As a consequence, flock infection rates will be relatively low and it can be expected that fewer sheep will develop clinical disease - and will do so at greater ages – on farms in lower rainfall zones, compared to farms in high rainfall zones. It should be noted, however, that OJD is still likely to persist in such flocks. While survival of the organism free in the environment is adversely affected by the heat and dryness of the South Australian summer climate, it will persist in infected sheep through the summer and its passage from infected sheep onto pasture during autumn, winter and spring will represent a source of infection for young sheep every year.

In the higher rainfall zones of SA (over 600 mm annually, for example), the impact of the seasonal distribution of rainfall on OJD transmission is likely to be less than in the lower rainfall zones because (a) high stocking densities of sheep, particularly at lambing, favour disease transmission and high stocking densities are common in the high rainfall zones, (b) shade is more commonly available, (c) areas of permanent water or moisture such as dams and soaks are common and (d) summer rainfall events are more common. These factors probably provide a summer environment sufficiently similar
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to that of NSW Tablelands and south-west Victoria to make little difference to the persistence of *M ptb* in the environment on farms in those different regions.

C-2 Current effectiveness of OJD control in SA

All three factors just described are likely to have contributed to the current low prevalence of OJD in SA and to the clustering of infection in the high rainfall zones of Kangaroo Island and the South-East.

Despite the regulatory activity, however, the disease has spread into SA and is continuing to spread. The principal reasons for the continuing spread are that (1) detection of infected flocks is based largely on detection through abattoir surveillance and (2) detections are followed by PDMPs which, most frequently, rely on vaccination as the principal disease-reduction measure.

Unfortunately, both abattoir surveillance and control by vaccination have critical weaknesses if they form the basis of attempts to eliminate infection from flocks or to hold flock-level infection rates static.

Effectiveness of abattoir surveillance in SA is limited

Abattoir surveillance is a relatively blunt surveillance tool. It is unlikely to detect an infected flock until the disease has been present in the flock for some years, by which time there have been opportunities for spread to other flocks. Abattoir surveillance tends to indicate the regional prevalence of the disease some years prior to the present.

Compounding the insensitivity of abattoir surveillance in SA is the incomplete surveillance of all flocks in a region. In SA, where the South-East regions present the greatest threat to control, between one half and three quarters of flocks have not been subject to abattoir surveillance in the past four years.

**PDMP-V will not reliably eliminate infection from a farm**

Flocks which are found to have OJD are required to go through a PDEP or PDMP-V – strategies which reduce the prevalence of OJD in the flocks. Ultimately these flocks are assessed with a clearance test and, if the test is negative for *M ptb*, the flocks are released from their trading restrictions. The release is consistent with the rules published in the *Standard Definitions, Rules and Guidelines for Sheep and Goats* and is not intended to imply freedom from *M ptb* – only that the organism has not been detected in the clearance test. (Note that Biosecurity SA staff do not state that the property is free of *M ptb* when an Order is lifted.)

Knowledge of the effect of vaccination in infected flocks, the behaviour of the disease and the limitations on current testing protocols lead to the conclusion that many of the flocks which are released from Orders are probably not free of *M ptb*. It is likely that, in most cases, the infection remains present at sub-clinical levels in a small proportion of sheep in the flock. If vaccination in the flock ceases, the low level of contamination caused by the few remaining infected sheep will be sufficient to lead to infection at higher levels in co-grazing unvaccinated animals.

C-3 Future changes in prevalence of OJD across the state

The reasons that two areas (KI, SE) have a higher prevalence than elsewhere in the state are well understood. First, both regions have a relatively high rainfall and high sheep densities; two conditions which favour survival and transmission of *M ptb*. Second, on Kangaroo Island the disease spread locally after an introduction decades ago, and before monitoring for the disease was in place. The disease spread widely on the island before any control strategies were adopted. In the SE, the prevalence of the disease is higher than other mainland regions of SA as a consequence of its proximity to Victoria and to trading patterns within SA and from the east.

The presence of OJD on KI appears to be manageable and to present a limited risk to mainland flocks. Control of the disease on the island is principally achieved by vaccination. The disease is relatively
widespread and the local farming community are well aware and well informed about OJD risks and management. The risk of OJD transmission from KI flocks is well understood by producers and livestock agents in other regions of SA.

The situation in the South-East is different. The disease is spreading, the prevalence is underestimated, detections are lagging behind the development of new cases and the proportion of flocks which undergo abattoir surveillance each year is well under 50% - insufficient to allow adequate improvement in detection rates. Furthermore, the level of concern, awareness and knowledge about OJD amongst sheep producers remains low, based on reports from other producers, including those interviewed for this review.

OJD infection in the South-East will spread from two groups of farms. First, it will spread from some of the 50 farms which have had a clearance test and have been released from infected status. Some of these flocks will still contain infected sheep and will continue to trade. The fact that these sheep are vaccinated is likely to mean that the disease prevalence within the flock is at a low level and only a few sheep have patent infections, and those infections are likely to be paucibacilliary. A risk-based approach to trade (under current guidelines) might lead a buyer to consider such a flock a low risk for transmission but in fact the purchase of a line of young sheep from such a flock might include a significant risk of purchasing at least one infected animal which is shedding $M_{ptb}$.

Second, infection will spread from flocks which are infected but not yet detected. Because abattoir surveillance is not sufficiently widespread, flocks will spread OJD to neighbours or trading partners before they are detected by abattoir surveillance or by trace-back activities.

Given the indicative prevalence of infected flocks in the South-East (both detected and undetected) and the incomplete and insensitive surveillance of flocks in the region, it can be expected that the rate of new infections and new detections will increase in that region over the next few years, even if current regulatory activities continue. The increased incidence is predicted as a consequence of the increasing number of flocks which can act as a source of transmission to uninfected flocks. Successful transmission contacts will therefore occur more frequently and the rising rate at which new cases occur will soon exceed the capacity of regulatory authorities (or SASAG funds) to act in a timely fashion.

It is also likely that other, higher rainfall regions of the State, notably Adelaide Hills/Fleurieu, will see a similar increase in the rate of new detections as the infection spreads from infected farms before they are detected and from farms released from Orders following a clearance test.

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In epidemiologic terms, for a disease which spreads from infected individuals (farms in this case) to non-infected individuals (farms), the rate of spread is initially low because it is at first limited by the low number of infected individuals. As an epidemic continues the rate of spread increases from the low base because there are in increasing number of sources of infection but still many susceptible individuals. Eventually, the epidemic wanes because there are a limited number of non-infected and susceptible individuals.
C-4  Bio-economic model (AusVet 2006)

It is instructive to review the report prepared by AusVet Animal Health Services for PIRSA in 2006. This report predicted that

- the ‘current’ regulatory approach would maintain the proportion of infected flocks below 5% for 30 years (until 2035) at least.
- a removal of controls would result in a prevalence of infected flocks of 50% to 80% by 2035, depending on the assumed rate of spread.

It should be noted that the model used in the report assumed that 4000 lines of sheep would be subject to abattoir surveillance each year. In fact, only half that number have been inspected each year since 2012, and the regional representation in the sample has been patchy, with the highest representation from two regions of very low prevalence (Eyre Peninsula and Yorke Peninsula/mid-North) and lower representation in the higher risk South-East regions. Consequently, the value of abattoir surveillance may have been over-estimated in the model.

Commentary on the AusVet report has noted that the model assumed a similar level of disease transmission across all regions of South Australia. This assumption is unlikely to be valid, given the range in average rainfall across the sheep-carrying parts of the state. Nevertheless, nearly half of the state’s sheep population are run in regions with average annual rainfall exceeding 400 mm and 30% are run in high rainfall zones (Figures 8 and 10) so the predicted prevalence of infected flocks in the absence of control measures could be appropriate for those regions of the state.

The assumption, however, is critical to the conclusions that are drawn from the model. The report describes a range of scenarios, from a continuing application of the ‘current’ approach, an expanded regulatory approach (‘maximum control’), and other scenarios including a totally deregulated environment.

Over a 30 year period (2005 to 2035), the predicted lowest cost of OJD arose from a continuation of the ‘current’ program and, almost equal in cost, a ‘voluntary vaccination’ program in which subsidisation of vaccine ceased and regulatory activity continued at a reduced level. The net present value (NPV) of each of these two scenarios was around $32m over 30 years. By contrast, the NPV of the ‘deregulated’ scenario was $45m.

When, however, the assumptions about the rate of spread of OJD were changed to a lower figure (slower rate of spread), the proportion of infected flocks in SA in 2035 in a deregulated environment was predicted to be significantly lower and the disease was predicted to spread more slowly. Under these assumptions, the model predicted that the ‘current’ approach, the ‘voluntary vaccination’ approach and a ‘deregulation’ would all have similar costs, with NPVs around $30m over 30 years.

Given earlier comments about the distribution of sheep flocks in SA and the climate variation across the state, it is reasonable to propose that the ‘slower-spread’ assumptions may offer a more realistic prediction of the development of OJD endemicity in SA. If so, the model provides weight to an argument that deregulation is a rational economic choice for South Australia and equally rational to a continuation of the current program.

A move from the ‘current’ program to a ‘deregulated’ environment would have the effect of moving costs from some sectors of the industry to other sectors. Under the ‘current’ program, most costs are shared across the industry through the funding provided by SASAG and the sheep industry fund levy. Individual producers share in this industry cost, of course, but the small number of affected producers contribute directly to the cost through the impact of the disease in the affected flock, the unsubsidised
portion of vaccine costs and the lost marketing opportunities consequent on the imposition of trading restrictions.

In a ‘deregulated’ environment, the industry-wide contribution to costs is withdrawn and affected producers, in steadily increasing numbers, take on the responsibility for the cost of the disease. They bear this cost through the economic impact of JD on their flock’s productivity and/or the cost of preventive management strategies including vaccination. The cost of lost marketing opportunities diminishes (under the model but not necessarily in reality, depending on the extent to which OJD freedom influences markets for sale sheep).

Under this scenario, the sheep industry fund contribution is no longer spent on OJD control, but is ‘freed-up’ for other purposes which, hopefully, return benefits to the industry greater than the expenditure.

The net (economic) effect of a move from the ‘current’ to a ‘deregulated’ model therefore is the increased contribution to OJD costs by (some) individual producers and the consequent availability of funds for alternative investment by the industry.

C-5 Removal of regulatory control of OJD in South Australia

Prevalence
OJD will increase in prevalence in SA whether the current regulatory program is continued or not. It is predicted to increase faster if the current regulatory approach is abandoned.

It is also predictable, based on the climate in South Australia, that the disease will become endemic at a moderately high flock prevalence in the South-East region as it has on Kangaroo Island and that the prevalence of infected flocks will be in the range predicted in the AusVet (2006) model and evident though the high prevalence regions of Victoria, New South Wales (30% to 80%). New Zealand – which has had OJD longer than Australia - also serves to illustrate the prevalence that will occur in time. The Adelaide Hills/Fleurieu region is expected to become a relatively high prevalence region because of its climate which is considered favourable to OJD spread.

In the other, lower rainfall zones of South Australia, the disease will occur at a more limited prevalence and is likely to spread more slowly. Its spread will be limited by the hotter, drier climate, the lower sheep densities, the lower intensity of sheep production systems and the fact that sheep trading patterns generally follow a pattern of movement from the drier regions to the wetter regions rather than the reverse.

Impact
In the medium and high rainfall areas of the state, OJD is predicted to present a significant threat to animal health and welfare unless it is controlled by vaccination. In the high rainfall zones, the disease is likely to have an effect on the health and productivity of the flock similar to that reported from NSW endemic areas and discussed in Section A of this report. If flocks in those regions become infected and no control is enacted (vaccination particularly), levels of sub-clinical and clinical disease will be similar to levels reported from NSW studies, with mortality rates due to OJD of the order of 6% annually, but variable between farms. OJD vaccination is likely, therefore, to become normal practice for flock managers in those regions.

In the low rainfall zones, the impact of OJD within a flock may be relatively minor and flock owners may choose to adopt control measures other than vaccination, if their flocks become infected.
In the medium rainfall zones (400 mm to 500 mm annual rainfall), the impact of OJD in an infected flock will be intermediate; OJD mortality rates are expected to be low (1% to 3% perhaps), the levels of clinical and sub-clinical disease will be similarly low and management strategies which reduce the exposure of animals to *M. ptb* will have a significant impact on the prevalence, and clinically-observed effect, of the disease. Vaccination, if used as a control strategy in flocks in these regions, is likely to be very effective in preventing mortalities and reducing the level of shedding of *M. ptb* in the flock.

**Figure 10:** Around 30% of sheep in South Australia are run in high rainfall regions, 44% in medium rainfall regions and 26% in low rainfall regions.

**C-6 The proposed approach to OJD control in SA**

Biosecurity SA, through the Chief Veterinary Officer, favours a change in the way OJD is managed in South Australia, to bring the South Australian approach in line with the National approach to OJD and BJD.

It is also proposed that flocks in which clinical cases of OJD are detected will be subject to an Order, requiring the removal of all clinical cases and the implementation of a PDMP and two years of vaccination of all lambs born. These requirements are subject to funding arrangements being put in place.

This approach is considered to be similar to the approach taken to control of some endemic diseases of sheep. It does, however, assume that (advanced) clinical cases of OJD are the principal source of risk of transmission of OJD, rather than sheep with sub-clinical disease or sheep in the early clinical stages. Sheep in these categories also shed *M. ptb*, albeit at lower levels than clinical cases, so the proposed regulatory action and requirement is out of proportion to the level of risk of transmission presented by clinically-affected sheep from an infected flock, compared to sub-clinical or pre-clinical cases.
If the disease is deregulated, a more effective and equitable strategy may be to provide information and advice to producers with clinical cases of OJD, rather than to impose an Order. As is the case with other diseases of sheep in SA (virulent footrot for example), clinical cases of OJD should not be presented to saleyards, permitted to stray or pose an undue disease threat to neighbouring flocks.

The PIRSA-proposed strategy is further discussed in Appendix C.

Risk and prevalence – a need for clarity

If a new approach to OJD management is adopted and individual producers are given a greater share of the responsibility for disease control (biosecurity) on their properties, it is important that the terminology around ‘risk’ and ‘prevalence’ are made clear.

Currently, there is often confusion over the term ‘risk’ in relation to OJD.

A flock which has a low prevalence of OJD-infected sheep may be described as a ‘low risk’ flock in relation to the likelihood of spread to another flock. This is not always valid. Consider, for example, a flock of 1000 sheep in which the prevalence of infected sheep is 0.1%. This is a very low prevalence – only one sheep in the flock is infected.

It is true that a producer who buys one single sheep from that flock has a low risk of buying the one and only infected sheep.

On the other hand, a producer who buys the whole flock, or who agrees to agist the whole flock, is guaranteed to introduce OJD to the property – that is a very high risk! It is true that the disease will be introduced at a low level and may take some years to establish and be recognised, but the disease has been transmitted and is now present on the property.

On this basis, it is misleading to label low-prevalence flocks as low-risk flocks, because the degree of risk varies with the number of sheep (the consignment size) transferred from the infected flock to the uninfected flock. Note that flocks with very low prevalence of infection (<0.5% for example) have a high likelihood of escaping detection through tests such as PFC350, or abattoir surveillance. This may be a level achieved by many flocks which have returned a negative clearance test after a PDMP-V.

The issue of risk, prevalence and consignment size is discussed in more detail in the report by Shepherd and Williams (2014), in relation to false-negative MAP flocks. As they suggest, ‘larger consignments of sheep purchased from false-negative MNV (monitored negative vaccinated) flocks present a high risk of disease across the feasible range of within-flock prevalence (for example, more than 20% probability of infection for a consignment of 50 sheep where within-flock prevalence is 0.5%).’

For a flock to be considered truly low risk, it should be necessary to have a high level of confidence that there are zero infected sheep in the flock. This would be true for a sheep flock with good biosecurity practices, in a region of low OJD prevalence, and with a history of negative abattoir surveillance or other surveillance testing, in the absence of vaccination AND with no history of OJD ever being present. Such a flock is truly a low-risk, no matter how many sheep are traded. There is always a risk that the disease is present but not detected – such as might happen with a recent undetected introduction of M ptb. This is a risk related to events, rather than the prevalence of disease.

For this reason, the classification of sheep flocks in South Australia that is recommended in this report is that provided in Appendix A. The terminology used in that table is intended to give information to producers which is more useful for guiding sheep management decisions than one which conflates risk with prevalence.
Impact of OJD on the market value of sheep in SA

In the interviews conducted while preparing this report, there were several suggestions made that the current regulatory environment in South Australia, combined with its low OJD prevalence, contributed to a positive reputation for South Australian sheep in the market place, which significantly added to their value when trading, particularly when trading into Victoria. 

It is difficult to place a specific value on this but perhaps the most useful comment on the subject was made by Mr Laryn Gogel who commented that saleyard competition in the South-East region is often greater for lines of young ewes which are vaccinated, and that he has observed that at any one sale some buyers show disinterest in unvaccinated sheep but bid competitively for vaccinated sheep.

It is logical and rational for buyers to act this way, depending on the OJD status in their ‘home’ flocks. The benefit may be just a few dollars per head and may be a consequence of the vaccinated status at least as much as the low-prevalence regional status, but should be kept in mind when considering the value of creating regions in South Australia of known low flock prevalence.

An analysis conducted in 2007°, based on sales of crossbred ewes and crossbred female lambs at the 2007 Naracoorte first-cross ewe sales, revealed that there was a statistically significant relationship between OJD score (on the ABC-scoring system then in place) and sale price of crossbred ewe lambs. The strength of the relationship was $3.33 (around 4% of total price) per score across the range of scores from 3 to 9. There was no significant relationship between score and price for adult ewes. In the analysis, there was no examination of the effect of state of origin on price, nor any examination of confounding between OJD score and other variables which may have influenced the price.

Under the ABC score system, vaccination could add 1 to 4 points to the score, depending on the level and duration of vaccination practised in the flock of origin.

The limitations of the analysis mean that there is perhaps some uncertainty around the true value of vaccination in the saleyard, but the results do support the notion that vaccination contributes to the premium paid for well-presented and well-marketed sheep.

° Analysis and report from Mr Ian Sanderson, Biosecurity SA
SECTION D SUMMARY

D-1 Important facts about OJD
There are a number of important facts about OJD which are not widely understood within the industry but are important messages which should be incorporated in producer-awareness extension messages;

1. OJD in flocks in the higher rainfall zones of South Australia is likely to be a significant disease in terms of the impact on the health, welfare and productivity of sheep flocks. The impact of OJD in infected flocks in high rainfall zones should not be under-estimated. On the basis of experience in NSW and until evidence is presented to the contrary, the disease should be considered to be capable of causing significant mortality rates in adult sheep (of the order of 6% annually) once it becomes established in a flock and if no control strategies are put in place.

2. Control strategies, particularly vaccination, will be necessary to prevent unacceptable levels of mortality in most infected flocks in the higher rainfall zones.

3. After introduction at a low level, OJD takes years to establish in a flock. Initially, clinical disease will be hard to detect but, in the absence of control strategies, it will become more apparent over time.

4. OJD in the lower rainfall zones of South Australia is expected to have a low impact on the health, welfare and productivity of sheep flocks but, if introduced and established, may continue to persist and present a risk of transmission to other flocks. The stocking density, management practices and climate are expected to have a very strong influence on the level of disease apparent in flocks in such regions.

5. While vaccination can be very effective in reducing the number of clinical cases of OJD, it does not reliably prevent infection being introduced and established in a non-affected flock.

6. Non-infected flocks in which sheep are vaccinated pre-emptively will have a high level of protection against clinical signs of OJD should the organism be introduced onto the property.

7. Flocks which have been detected with OJD and have followed a PDMP-V to achieve a negative clearance test are still likely to contain infected sheep which present a risk of transmission to other flocks.

8. De-stocking for a period including two summers is likely to eliminate *M. ptb* from a property but steps also need to be taken to ensure that infection is not persisting in other animals on the property, particularly cattle, goats, deer and alpacas, nor re-introduced when re-stocking or from neighbouring flocks.

9. There is confusion over the notions of low-prevalence and low-risk. A flock with a low prevalence of OJD, even a level which is undetectable using standard testing strategies, still presents a high risk of transmitting OJD to another property with the movement of large consignments of sheep.

10. Producers whose flocks are free of OJD can be offered three alternative pathways. They can (1) attempt to maintain freedom from OJD with or without vaccination; (2) they can accept the possibility of contracting OJD but opt to prevent a significant level of disease by the use of management and sheep-purchasing strategies which may include vaccination; (3) they can disregard attempts to maintain freedom and implement surveillance strategies (abattoir surveillance, necropsies of thin adult sheep, PFC testing of selected thin sheep) so that they become quickly aware if and when vaccination should commence.
11. Producers who wish to breed and retain sheep, or sell to other flocks with a view to retention, but who do not wish to vaccinate, must take deliberate actions (a rigorous biosecurity strategy) to ensure that M ptb is not introduced to their flocks.

12. The most secure method of identifying flocks which are a low-risk source of OJD-free sheep is to identify flocks which are not vaccinating, have a sound biosecurity plan in place, and are testing for OJD at frequent intervals and to levels equivalent to those of the MAP.

13. Many producers who buy replacement female sheep for their flocks will be satisfied to buy sheep which are vaccinated (as lambs) and from a flock with a National Sheep Health Statement indicating undetectable levels of OJD in the flock, even if there is likely to be M ptb in the sheep at a low level. The history and vaccination status provide a promise of very low levels of clinical disease even if M ptb is introduced and this may be acceptable to many producers.

D-2 Reasons for recommendation

My recommendation with regard to OJD management in South Australia is that the approach move away from regulation and a restriction on trade for affected producers towards a more de-regulated approach, shifting responsibility for disease management to individual producers, but that steps be taken to capitalise on the current low prevalence of infected flocks through stimulating and encouraging producer awareness. My reasons are these:

1. Attempts to prevent the spread of OJD in NSW and Victoria, based on regulatory controls, have failed, and the control of the disease passed to individual producers very late in the epidemic within the high-prevalence regions.

2. As the funds available for vaccine subsidisation decline, the effects of regulatory control following an OJD detection will place a greater financial cost burden on individual producers through vaccine costs and restrictions on trade. Control programs which are perceived to lack equity are poorly supported by producers, engender strong anti-government sentiments and ultimately do not succeed without intensive and very expensive regulatory activity.

3. The current strategy in South Australia is providing only partial control (slowing, not stopping the spread) and is not providing good epidemiological data which could inform good strategies developed regionally and locally to control the disease.

4. Information about the disease – particularly its regional prevalence – is limited and unreliable. There is substantial misinformation, particularly in regard to the existence of OJD on farms released from Orders and the reliability of vaccine.

5. The current expenditure on control strategies – subsidised vaccine and farm investigations - is competing with the allocation of funds toward collection of better data, on which good management decisions depend.

6. Currently, control of OJD in SA through regulation places responsibility for disease management on Government agencies.

7. Producers are neither encouraged (point 6) nor sufficiently well-informed (point 5) to be able to make good business decisions about their plans for management of OJD.

8. If, as expected, there are marked differences in the prevalence of OJD-infected flocks between regions of the state, the most effective way to slow the spread of disease is to apply different strategies in different regions.
9. These strategies should be producer-led and based on reliable information on disease prevalence and the effectiveness of control practices.

10. In order to conduct the necessary survey to estimate regional prevalence, it will be necessary to have producer support for structured surveillance testing. This will not be practical if detection leads to the placing of an Order. In order for a reliable survey to be performed, regulatory control of the disease must be relaxed.

11. Ultimately, regardless of the continuation of the current program or not, OJD will spread to its natural limits in SA. At some point in its future spread, the decision will be taken to deregulate control of the disease.

12. The AusVet (2005) analysis indicated that, under certain conditions, deregulation is as cost-effective as the current approach in managing OJD in SA. The information in this review supports the proposition that those conditions (limited rate of spread in low rainfall regions) are likely to exist in SA. A reduction in the direct industry expenditure on OJD control would permit the industry to reconsider the way that its current expenditure on OJD control is spent and to identify better investments for its funds.

13. In the short-term, some of the funds saved should be allocated to an epidemiological survey, data analysis, producer-awareness and information packages, and support for the establishment of producer-led regional OJD-control strategies.

14. In the long-term, some of the funds saved should be allocated to expansion of the abattoir surveillance program.

15. A de-regulation of OJD in SA brings the state in line with the approaches used in other states in which OJD is becoming or has become endemic and established and in line with approaches now in place for management of bovine Johne's disease.

16. SA has the opportunity to apply an innovative, cost-effective and epidemiologically-sound approach to reducing the rate of spread of OJD because it can begin the program while the prevalence is still relatively low.

17. Industry funds, currently spent on OJD control, could be re-directed towards gathering better information on OJD prevalence in regions of the state, developing education and extension packages, developing realistic pathways to managing OJD, to place the state’s industry in a strong position to manage the disease effectively.

18. Depending on the estimates of OJD prevalence found in each region of SA, it is likely that the best approach to management will vary with the region. It is likely, for example, that producers on Kangaroo Island, with an already established high prevalence, will adopt one strategy, while producers in the South-East, with a rising prevalence and imminent threat from Victoria will choose another, and producers in low prevalence regions of SA will choose yet another. It is possible that producers in the low prevalence areas, for example, could develop and adopt a Cooperative Biosecurity Plan to reduce the risk of OJD introduction.

19. While abattoir surveillance is not an effective tool for preventing the spread of OJD, it is a useful tool for alerting producers to the presence of disease and for estimating disease prevalence, particularly in a de-regulated environment.

20. There is confusion in the industry about the terms risk and prevalence, and the table produced in Appendix A should be considered as a guide to future strategies for categorising SA flocks with respect to OJD.
21. The Sheep Health Statement provides useful information to SA producers about OJD and should remain a requirement in SA. It could be modified in line with recommended changes to terminology with respect to risk.
SECTION E NOTES

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7. Ms Leonie Mills, sheep producer, Chair SASAG, member SA OJD Advisory Committee
8. Mr Allan Piggott, former member of SASAG, current Chair Blueprint SA
9. Mr Rohan Giles, Lucindale, member of SASAG
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18. Dr Greg Johnson, Private Veterinary Practitioner, Kangaroo Island
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20. Dr Elise Matthews, Disease Surveillance, Biosecurity SA
21. Mr Andrew Ewers, Senior Animal Health Advisor, Kangaroo Island, Biosecurity SA
### Abbreviations

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>CMI</td>
<td>Cell mediated immunity</td>
</tr>
<tr>
<td>EAS</td>
<td>Enhanced abattoir surveillance</td>
</tr>
<tr>
<td>IS1311-PCR</td>
<td>Insertion sequence 1311 - polymerase chain reaction; a test for a specific element of DNA</td>
</tr>
<tr>
<td>KI</td>
<td>Kangaroo Island</td>
</tr>
<tr>
<td>MAP</td>
<td>Market Assurance Program</td>
</tr>
<tr>
<td>M ptb</td>
<td><em>Mycobacterium paratuberculosis</em></td>
</tr>
<tr>
<td>OJD</td>
<td>Ovine Johne's disease</td>
</tr>
<tr>
<td>PDEP-V</td>
<td>Property Disease Eradication Plan (vaccinating)</td>
</tr>
<tr>
<td>PDMP</td>
<td>Property Disease Management Plan</td>
</tr>
<tr>
<td>PIRSA</td>
<td>Department of Primary Industries and Regions South Australia</td>
</tr>
<tr>
<td>PFC</td>
<td>Pooled faecal culture</td>
</tr>
<tr>
<td>PFC350</td>
<td>Pooled faecal culture of faeces from 350 sheep</td>
</tr>
<tr>
<td>SA</td>
<td>South Australia</td>
</tr>
<tr>
<td>SASAG</td>
<td>South Australian Sheep Advisory Group</td>
</tr>
<tr>
<td>SE</td>
<td>South-East; three regions in the south-east of South Australia</td>
</tr>
</tbody>
</table>
### Appendix A

**Recommended strategy for categorising sheep flocks in South Australia with respect to OJD**

<table>
<thead>
<tr>
<th>Description and point score</th>
<th>High assurance</th>
<th>Low prevalence flocks</th>
<th>Non-assessed</th>
<th>Suspect or infected</th>
</tr>
</thead>
<tbody>
<tr>
<td>Flock description with respect to OJD</td>
<td>Low risk flocks (8)</td>
<td>Evidence of very low prevalence (7)</td>
<td>Evidence of low prevalence (6)</td>
<td>Progressing towards evidence of low prevalence (4)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Risk rating</th>
<th>Low</th>
<th>Risk variable, depending on the number of sheep purchased</th>
<th>Unknown, dependent on regional plan</th>
<th>Uncontrolled, high</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>History of OJD detection</th>
<th>Never detected or 5 years without vaccination and not detected</th>
<th>Possibly</th>
<th>Possibly</th>
<th>Possibly</th>
<th>Not assessed</th>
<th>OJD confirmed</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>Vaccine use</th>
<th>Restricted to certain sale animals, if any. Portion of the flock remains unvaccinated.</th>
<th>Possibly</th>
<th>Possibly</th>
<th>Possibly</th>
<th>Possibly</th>
<th>Possibly</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>Biosecurity plan in place</th>
<th>Yes</th>
<th>Yes</th>
<th>Possibly</th>
<th>Possibly</th>
<th>Possibly</th>
<th>Irrelevant</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>-ve Abattoir 150 last 1 year</th>
<th>Yes</th>
<th>Yes</th>
<th>Yes</th>
<th>Yes</th>
<th>No</th>
<th>Irrelevant</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>-ve Abattoir 500 last 2 years</th>
<th>Yes, or alternative</th>
<th>Yes, or alternative</th>
<th>Yes, or alternative</th>
<th>No</th>
<th>No</th>
<th>Irrelevant</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>-ve PFC350 last 2 years</th>
<th>Possibly, and an acceptable alternative to Abattoir 500</th>
<th>Possibly, and an acceptable alternative to Abattoir 500</th>
<th>Possibly, and an acceptable alternative to Abattoir 500</th>
<th>No</th>
<th>No</th>
<th>Irrelevant</th>
</tr>
</thead>
</table>
NOTES

- Words such as ‘low risk’ and ‘assurance’ must be used very carefully to avoid overstating the safety of some trading decisions.
- One cannot say a flock is a low risk just because it is of low prevalence. The degree of risk depends on the number of animals traded.
- Flocks cannot progress from low prevalence to high assurance if they continue to vaccinate without particular practices in place. Strategies to allow progression need to be developed.
APPENDIX B

Clarification of terminology

Endemic

The term ‘endemic’ is used in this report to mean that infection is established and maintained within a sheep population without introduction from outside the region of interest (farm, region, state or nation) and at a prevalence which is relatively stable. Endemicity does not imply a high prevalence; diseases can be endemic at low, medium or high prevalence. Endemic diseases may be kept at a particular prevalence by the nature of the disease itself and how readily it transmits, the resistance of the population, the environment and by control measures which are applied.

To illustrate, OJD could be considered to be endemic in the central Tablelands of NSW and in some other regions of NSW and Victoria where it has been established for 30 years or more. It could also be considered endemic on Kangaroo Island. In mainland SA, the view proposed in this report is that the disease is still spreading to previously uninfected flocks and has not yet achieved a relatively stable flock prevalence – and therefore is not yet endemic, but is established and will become endemic.

The term endemic can also be applied to the existence of OJD within a flock. It could be endemic in a flock long before it is considered to be endemic in a region.

Control

In this report, the term ‘control’ in relation to OJD has the following meaning;

(a) At state level, achieving a reduction in the rate of disease spread from flock to flock, region to region, or maintaining a low rate of spread
(b) At flock level, achieving a reduction in the number of clinical cases of OJD in an infected flock and a reduction in the rate of transmission with the flock.

The term ‘control’ does not imply progress towards elimination of the disease from a flock, farm or region. Control strategies are often applied to a degree at which the cost of activities to achieve further disease reduction will exceed the benefit of any further disease reduction.

Managing OJD

In this report, the term ‘managing OJD’ has the following meaning:

(a) Exerting control of the disease – as above
(b) Monitoring and reporting the spread of the disease
(c) Providing mechanisms for producers to identify low risk and low prevalence flocks for trading purposes.
APPENDIX C

Comment on the current OJD control program

<table>
<thead>
<tr>
<th>Stated objectives of SA OJD Control program</th>
<th>Comment on current progress</th>
</tr>
</thead>
<tbody>
<tr>
<td>To slow the rate of spread among SA sheep flocks so that the incidence of OJD remains at less than 5% by 2025.</td>
<td>While the evidence of current true prevalence is weak, OJD has been detected in around 1.5% of the State’s flocks (July 2016).</td>
</tr>
<tr>
<td>To detect OJD-infected farms by feedback from abattoir surveillance and tracing sheep movements to and from infected properties. In addition conducting risk assessment on properties that neighbour infected farms and, if it is determined that there has been a significant risk of disease spread, testing of these properties.</td>
<td>While this activity has been performed to a creditable standard, it has inherent limitations due to (a) the incomplete nature of abattoir surveillance in the state, (b) the delay between introduction of OJD onto a farm and its detection by abattoir surveillance and (c) the sensitivity of abattoir surveillance for disease detection in an infected flock.</td>
</tr>
<tr>
<td>To assist OJD-infected sheep properties in attempting to eliminate the disease from their flocks through the use of vaccine and on-farm management changes detailed in customised PDMPs.</td>
<td>While clinical disease may be eliminated from farms by vaccination, it is unlikely that vaccination alone will eliminate <em>M ptb</em> from farms or flocks. Release from Orders after a clearance test encourages the view that the disease has been eliminated from some farms. This proposition remains substantially untested. There may be unrealistic expectations about the level of control of <em>M ptb</em> spread achieved in the program to date.</td>
</tr>
<tr>
<td>To participate in national discussions with industry on OJD policy development and implementation.</td>
<td>No comment.</td>
</tr>
</tbody>
</table>
Comment on the proposed OJD control program

The PIRSA-proposed program has a ‘disease focus’ rather than a ‘bacterium-level focus’.

The program proposal makes the following claims;

Focussing on the infection (all cases of OJD, clinical or sub-clinical) is required for a control program that aims to eradicate the infection or reduce it to a very low prevalence.

Focussing on the disease (clinical disease), rather than the infection, targets the population which will experience the greatest financial impact and pose the greatest risk of spread. There will be more spread of infection with a disease focus (than with a program focussed on infection at all levels).

The question to be asked, then, is ‘how does the proposed program differ from the current program?’

The answer is that they differ from each other only in degree. Like the proposed program, the current program bases most of the new detections on the appearance of clinical disease (abattoir surveillance, voluntary reporting, private practitioners) but the current program also bases some of the new detections on infection-level investigations (trace-forward, trace-back, MAP breakdowns, surveillance testing on KI). With the current program, flocks are placed under Orders and required to reduce the level of infection to very low levels before they are able to sell sheep to other producers in SA again. With the proposed program, producers will be required only to eliminate clinical disease before resuming open trade. The presence of sub-clinical disease in the flock will not be a barrier to resumption of normal business.

With both programs, OJD will continue to spread through SA but the additional rigour applied in the current program means that the rate of spread will be slower than would be the case in the PIRSA-proposed program.

It is argued that both programs have an arbitrary hurdle for resumption of open trading of sheep when an Order is lifted. For the current program, the hurdle is a clearance test which, as proposed in this report, proves only that the level of disease in the flock is very low, not that the infection is eliminated. The hurdle in the proposed program is more relaxed but perhaps more obviously an arbitrary one. Effectively, if the disease cannot be seen by a keen observer, the presence of infection in the flock will be ignored.

The view formed through this report is that the arbitrary nature of the hurdle (for a clearance test) is a problem for both the current program and the PIRSA-proposed program when the programs are directed at stopping the spread of infection. In both cases, restrictions are placed on farms with OJD with the intention of stopping further spread from their flocks. Then, after actions are taken to reduce the prevalence of infected sheep in the flock, restrictions are removed but, in most cases, the infection will still be present.
With both programs, the lack of thorough surveillance is a further impediment to the efficacy of control measures. Only some farms with OJD are detected early enough to prevent spread to other farms. With the current program, detection of new cases of OJD is led by abattoir surveillance then on-farm investigations. On-farm investigations (trace-forward, trace back, neighbours) reveal some of the infected flocks which are not surveyed through abattoir lines, but still do not detect all infected flocks in a timely fashion. With the PIRSA-proposed program, the level of surveillance would be lower, because on-farm investigations would not occur or would be based only on the detection of clinical disease.

Both programs have significant limitations. One will slow the spread of OJD more than the other, but both involve the imposition of restrictions on producers when OJD is detected at some level. Neither program, therefore, adequately moves the responsibility for retaining freedom from, or controlling OJD at flock level to the individual producer. Both programs encourage the notion that a lack of restrictions following a clearance test or the absence of clinical disease means that a flock is ‘safe’ to trade. This notion is counter-productive to a move towards individual biosecurity responsibility. It is much more desirable that producers report their OJD status on a meaningful scale (see Appendix A) which requires some level of proof that the disease is absent (level 8) or at a particular prevalence (levels 4 to 7) rather than simply that clinical disease was not seen in the flock, or not detected by laboratory tests which have a non-zero rate of false negatives.

This report does not, therefore, support the proposal to place restrictions on producers when clinical OJD is detected in a flock. That is not to say that PIRSA should not take action under the Livestock Act, for example, with straying stock or when infected flocks pose an uncontrolled threat to other producers, or under the Welfare Act, where uncontrolled disease is adversely affecting the welfare of animals. These regulatory responsibilities remain important with OJD, as with other diseases of sheep.

There are other elements of the PIRSA-proposed program which are strongly supported in this report. These include;

- Expanded surveillance and disease reporting, with more informative epidemiological analysis and reporting.
- Preventative actions, including assistance with development of biosecurity plans, tools for buyers, sheep health statements, collaborative biosecurity groups.
- Compliance activities, particularly in relation to reported disease status, saleyard inspections, oversight of interstate movements.
## Comment on the approach proposed in this review.

<table>
<thead>
<tr>
<th>Continue current program</th>
<th>De-regulate, complete epidemiological survey, introduce an 8-point OJD assurance scheme, increase producer-awareness activities.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unaffected producers remain relatively unaware of the risks of introducing OJD because they are aware that there is (a) an existing control program <em>(true)</em> and (b) only 50 flocks in SA 'have OJD' <em>(untrue)</em>.</td>
<td>Producers are made aware that there is no longer a control program and that they are individually responsible for protecting their flocks from infection.</td>
</tr>
<tr>
<td>Current prevalence of infected (undetected) flocks remains uncertain because estimates are based on abattoir surveillance of less than half of the state’s flocks and regulatory activity suppresses producer cooperation with testing programs which could provide more reliable estimates.</td>
<td>Increased accuracy of regional prevalence estimates provide a stronger basis for regional biosecurity plans, and provides more reliable indications to producers of the status of their region and trading partners.</td>
</tr>
<tr>
<td>OJD will continue to slowly spread in mainland regions of SA, limited in part by the current regulatory activities.</td>
<td>OJD will spread more quickly from flock to flock in mainland SA as a consequence of reduced regulatory activity.</td>
</tr>
<tr>
<td>OJD will remain well-controlled on Kangaroo Island because of wide adoption of sound, producer-led strategies.</td>
<td>No change to strategies on KI but reliable information about true prevalence in other regions may lead to the adoption by individual producers of biosecurity and flock-management strategies aimed at protecting against introduction of OJD, or at managing the disease.</td>
</tr>
<tr>
<td>An OJD assurance scheme (based on voluntary testing mechanisms) is difficult to introduce if the detection of disease leads to the imposition of an Order, restricting trade and requiring a PDMP. Assurance Adoptions</td>
<td>Adoption of an 8-point OJD assurance scheme (or equivalent) will provide producers with a more dependable opportunity to control the disease status of their flocks compared to dependence on the SHS alone. <em>(The MAP has a relatively low penetration and an uncertain future.)</em></td>
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documentation remains limited to disclosure on SHS or the MAP. (The MAP has a relatively low penetration and an uncertain future.)

| Producers whose infected flocks are detected will potentially suffer the negative emotional impact of ‘contracting’ a notifiable disease and the financial impact of lost trading opportunities. | De-regulation will reduce the stigma of OJD-detection and improve the level of communication, based on trust, between producers and animal health officers – a relationship which is very important for early detection of exotic disease and management of endemic disease.

In a de-regulated environment, some producers will still suffer a financial impact following detection of the disease because they may lose some trading opportunities. The impact, however, is likely to be less than occurs when an Order is served because some trading opportunities will still be present. |

| The ‘cost’ of an OJD detection to a producer is the cost of lost trading opportunities imposed by the Order plus the cost of future vaccinations, reduced by the level of subsidisation. | The ‘cost’ of an OJD diagnosis is the cost of lost trading opportunities, if any, plus the cost of all vaccinations.

The industry as a whole carries the cost of the vaccine subsidy plus some costs associated with regulatory activity. Vaccine costs are roughly one quarter of the annual (approximately) $1m expenditure of Industry funds on OJD in SA. |

| The industry as a whole is relieved of the cost of subsidised vaccination – only the affected producer pays. |

| Funds will continue to be allocated to vaccine subsidisation, at decreasing levels as the number of detected-infected flocks increases. | Funds currently used for vaccine subsidisation and other regulatory activities can be re-directed in the short to medium term to (a) survey activities and (b) producer-awareness activities and then, in the longer term, to other activities not necessarily related to OJD. |
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