



Sheep,
Camelid
and Goat
Veterinarians
Conference

Conference Proceedings

Stamford Grand Adelaide, Glenelg
22 – 24 June 2022



What's Hot and What's Not
2022 Sheep, Camelid and Goat
Veterinarians Conference
with a Sheep Reproduction Stream

KNOWLEDGE



SHEEP, CAMELID
AND GOAT
VETERINARIANS

Welcome



The SCGV Executive is proud to welcome you this week to the 2022 Sheep, Camelid and Goat Veterinarians Conference in Adelaide, South Australia!

We believe this is the first time that the special interest group has decided to go with an annual conference, and we have put together a list of quality speakers covering a range of topics. There has been no shortage of speakers wanting to be a part of the program and as a result we have big picture topics such as reproductive management of flocks, a focus on pain relief management, case reports on trace element toxicities and deficiencies, post bushfire management, insecticide resistance and is castration required for terminal lamb production. There is something of interest for all small ruminant and camelid veterinarians.

This week there's plenty of time for socialising and networking with your colleagues which we know is one of the main reasons you attend these conferences.

We look forward to reconnecting with you this week and welcoming you to Adelaide for another great SCGV Conference.

Regards,

Andrew Whale, SCGV President





Proceedings

2022 Sheep, Camelid and Goat Veterinarians Conference

Contents

| | |
|--|----|
| Allworth, Bruce - AVA Audit of AWI's Flystrike Prevention Research, Development and Extension program | 5 |
| Allworth, Bruce - Comparison between ram and wether lamb production - do we need to castrate lambs destined for slaughter? | 10 |
| Clune, Tom - Insights into campylobacteriosis in maiden ewe flocks..... | 12 |
| Dobrijevic, Tatjana - Investigating the cause and prevention of red gut in lambs grazing lucerne | 20 |
| Glanville, Elsa - Acute selenium toxicity in lambs: two overdose scenarios | 22 |
| Glanville, Elsa - Post-mortem findings from periparturient non-Merino ewes across southern Australia | 28 |
| Glanville, Elsa - <i>Toxoplasma gondii</i> exposure and associated risk factors in Victorian ewes | 35 |
| Gole, Tim - Pre-joining ram exams: a few tricks and an update of MLP ram mating success project | 44 |
| Graham, Kelly - What drench advice should I provide my clients? Anthelmintic resistance update: Results of 50 FECRTs conducted across Australia from July 2018 – March 2021..... | 45 |
| Hutchinson, Mark - Measurement enabled pain solutions for livestock | 48 |
| Jackson, Bruce - General surveillance for endemic and emergency animal diseases | 51 |
| Jacobson, Caroline - Evidence-based approach to investigating poor reproductive performance in maiden ewes..... | 56 |
| Jacobson, Caroline - Reproductive performance for maiden and multiparous ewes on Australian farms | 70 |
| Kirk, Bea - Certification programs available to Australian woolgrowers | 79 |
| Lehmann, Deb - Appropriate analgesia saves sheep with burnt feet on Kangaroo Island | 83 |
| Marini, Danila - Update on pain assessment and relief in sheep | 87 |
| McGrath, Sean - Bushfire Assistance: how to best help your clients in the immediate aftermath | 88 |

| | |
|--|-----|
| McGrath, Sean - Case Study: Ewe lamb foetal loss in South East South Australia | 105 |
| McGrath, Sean - Using disease investigation subsidies: a South Australian private practitioner perspective | 108 |
| McQuillan, Mary - Pneumonia in south Australian lamb feedlots | 110 |
| Pfeiffer, Caitlin - Health, welfare and biosecurity of sheep and cattle exposed to Australian bushfires | 112 |
| Plant, John - Epidemic Catarrh: what was it and why did it disappear? | 114 |
| Rast, Luzia - Investigating kidney lesions in lambs at Gundagai Meat Processors (GMP) . | 118 |
| Sales, Narelle - Sheep Blowfly Insecticide Resistance: Perpetual Motion | 120 |
| Slattery, Shaun - Plant poisonings of sheep in summer rainfall non-temperate eastern Australia | 123 |
| Suter, Robert - Arboviruses, flaviviruses and the foetus | 136 |
| Trengove, Colin - Impact of ewe nutrition on healthy bones and disease prevention | 141 |
| Trengove, Colin - Is regenerative agriculture a myth or critical to the survival of domestic herbivores? | 145 |
| Watt, Bruce - Clinical manifestations of selenium deficiency in sheep (and goats) | 146 |
| Whale, Andrew - Optimising reproduction success through pre joining ewe assessments | 148 |
| Williams, Scott - What's hot in sheep industry sustainability reporting | 150 |
| Windsor, Peter - Welfare impacts of plant intoxications on extensively-raised livestock in southern Australia | 153 |

AVA Audit of AWI's Flystrike Prevention Research, Development and Extension program

Bruce Allworth
Charles Sturt University
Wagga Wagga NSW

Introduction

Australian Wool Innovation (AWI) has undertaken research projects over the past 18 years around breech flystrike. Initially the focus was on finding an alternative to mulesing, but more recently the focus of the research has broadened to flystrike prevention (including breech flystrike) and animal welfare more generally. The AVA provides two auditors to independently review the program.

AVA Audit

The AVA audit is a consequence of an undertaking in 2005 to overseas manufacturers and retailers of clothing incorporating Australian wool by the Australian wool industry to provide an independent report on their research into alternatives to mulesing. AVA was approached to provide two auditors in recognising that veterinarians representing the AVA were independent and had technical expertise to assess the AWI Flystrike Research program

Initially the auditors were appointed directly by the AVA Board, and audits were conducted biannually. More recently, the AVA board has requested the SCGV SIG recommend the two auditors, and audits are now conducted annually. The appointed auditors meet with AWI staff to review the research projects and extension initiatives carries out over the previous 12 months, then provide a report to AWI, via the AVA President, on their performance. This is a brief 1-2 page report, which is then provided to Wool Producers Australia to inform industry and overseas interested parties of the work being carried out. It should be noted that while in discussion auditors may make technical comments on specific projects, the audits are not aimed at assessing the scientific validity or outcomes of these projects, but rather to assess that the work is undertaken to complete the objectives set for the AWI research program.

In addition to the annual audit and subsequent report, AVA auditors attend an annual or biannual Animal Welfare forum, where AWI host representatives from numerous Animal Welfare agencies to inform them of their current work and engage with these organisations. AVA auditors also attend the biennial Researchers' Forum, and are generally involved in stakeholder meetings that involve the development of the Flystrike research objectives, which are generally reviewed every 5 years. Auditors are contracted by AWI and paid a per diem rate for the audit and their attendance to the various AWI events, as well as travel and accommodation reimbursement.

AWI Research Program

The current 5 year program, known as the Flystrike Prevention Research, Development and Extension Program, has 5 Pillars - Breeding and Selection, Breech Modification Procedures, Non-Invasive Management Practices, Analgesia and Anaesthesia, and Education, Extension and Promotion. Each of these pillars has various specific milestones/outcomes, with a total of 18 outcomes specified across the 5 pillars (Table 1).

AVA Audit 2022

In April 2022, the AVA auditors met with AVA staff and completed an annual audit. The full report is available from the AWI website www.wool.com. The audit found that AWI continues to invest in research aimed at decreasing breech strike and specifically to decrease the reliance of wool producers on mulesing. The planned AWI Sheep Health and Welfare program budget for 2021-22 was \$A1.5 m, of which 80% is directed at activities to better manage flystrike. Over the past year, AWI managed a total of 14 projects (Table 1), including 2 projects

that commenced and 2 projects completed during the reporting period. This work, while still focused on finding alternatives to mulesing, continues to support much broader sheep welfare outcomes. In this reporting period there is continuing emphasis specifically on vaccine development, and a strong focus on extension of control methodologies. A number of the projects have resulted in the production of papers for publication in scientific journals and extension articles for sheep producers.

Non-Invasive Management Practices (5 projects - 2 completed, 3 ongoing) is an important area of current research activity, with emphasis on developing an effective vaccine against flystrike (3 projects). **Breeding and selection** (1 project) research work is now focussing on acquiring and utilising genomic data, with the commencement of a project to gather data from the Merino Lifetime Project dataset and the planning of a second project to establish the ability to collect genomic data from other flocks. **Breech Modification Procedures** projects are not currently being funded by AWI. **Analgesia and Anaesthesia** remains a focus for the Program but there are currently no research projects being undertaken. **Education, Extension and Promotion** (8 projects – 2 completed, 4 ongoing, 2 commenced) continues to focus on the support of the successful ParaBoss website and extension activities with advisors and producer groups. A series of Workshops and webinars are being either held or designed to support producer decision-making in the managing of flystrike, including moving to a non-mulesed system. Three webinars for one of these initiatives (It's Fly Time) was attended by 900 producers. AWI continues to support the Sheep Sustainability Framework, and its first Report is due in July 2022. This provides some objective data on level of mulesing, availability of non-mulesed wool, and adoption of pain relief.

Conclusion

The AVA, through its auditing of the AWI Flystrike Research program, provides an independent report which informs the industry and in particular, overseas processors and retailers, of the progress in research on breech flystrike. While initially the audited AWI research program was focussed on finding alternatives to mulesing, the program has expanded to deal with broader flystrike and welfare issues. The involvement in several pain relief products now being available to sheep producers, and the current work on bringing a vaccine against flystrike to sheep producers, as well as the extensive Extension activities, are examples of the success of the program.

Table 1. AWI FLYSTRIKE RESEARCH, DEVELOPMENT, EDUCATION, EXTENSION AND COMMUNICATION STRATEGY AUDIT TABLE, 19 April 2022

NON-INVASIVE MANAGEMENT PRACTICES

THE AIM: Improved management practices to reduce the risk of flystrike

| Stated Outcome | Status during Reporting Period - 5 projects | Comment – 2 completed, 3 ongoing | Outcome Achieved |
|---|--|---|------------------|
| Monitor and define blowfly resistance to chemicals. | Work completed previously. No current activity. | Publication in IJP: Drugs and Drug Resistance (2020). Commercial activity available. | Yes |
| Refine blowfly chemical resistance best practice management advice. | 00651 -Honours modelling project - completed -Final Report available Further work planned | Summer shearing, crutching increased resistance, winter shearing not cost-effective. FlyBoss updated. | Yes |
| Invest in early trials of new potential actives and parasitic | 00549 - nanotechnology-completed-Final Report available | Commercial application of nanotechnology being discussed. | Yes |

| | | | |
|---|--|---|-----|
| control treatments and vaccines. | 00619 - vaccine – ongoing-final report due end 2022 00801 – vaccine – new project to fast track vaccine development | 50 prototype vaccines tested in 45 flocks. New project to get to commercialisation faster -narrow 50 to 2 candidates. | |
| Complete a population study of blowflies to identify potential genetic differences to inform blowfly management programs. | 00624 – Informing on vaccines-ongoing - final report due mid 2022 | Important work to understand potential for differing fly populations - Tas, Qld and WA different to others | Yes |

ANALGESIA AND ANAESTHESIA

THE AIM: Improved provision of analgesia and anaesthesia for surgical husbandry practices.

| Stated Outcome | Activity during Reporting Period – No current projects | Comment | Outcome Achieved |
|--|---|---|------------------|
| Investigate longer acting, cost effective anaesthesia and analgesia options. | 00550- Gap Evaluation-completed previously. Open access publication. No current activity. | Small, A.; Fisher, A.D.; Lee, C.; Colditz, I. Analgesia for Sheep in Commercial Production: Where to Next? <i>Animals</i> 2021, 11, 1127. https://doi.org/10.3390/ani11041127 | YES |
| Extend advice on analgesia and anaesthesia to woolgrowers. | Updated Anaesthetics and Analgesics Factsheet | https://www.wool.com/globalassets/wool/sheep/research-publications/welfare/breeding/btb_march2021_p34-35.pdf | YES |

BREEDING AND SELECTION

THE AIM: Long term solutions to advance lifetime welfare.

| Stated Outcome | Activity during Reporting Period– 1 Project | Comment– 1 commenced, 1 in planning stage | Outcome Achieved |
|---|---|--|------------------|
| Understand the performance and economic impacts of breeding for reduced flystrike. | Work completed previously. No current activity. | Beyond the Bale article Dec 2021 | YES |
| Investigate the, as yet unknown, factors that cause flystrike. | Work completed previously. No current activity. | | YES |
| Improve the accuracy of selection for flystrike resistance traits through phenotyping and genotyping. | 00775- AGBU MLP-genomics of flystrike – Commenced 00820 – flystrike genomic flock-planning stage | Focus on acquiring and using genomic data. | YES |

| | | | |
|---|---|--|-----|
| Better understand how to reduce the incidence of dags and urine stain through breeding. | Work completed previously. No current activity. | Greeff,J; Karlsson, L; Schlink,A. Are breechstrike, dags and breech wrinkle genetically the same trait in crutched, uncrutched and mulesed Merino sheep? Animal Production Science, 2019, 59, 1777-1782 https://doi.org/10.1071/AN18461 | YES |
| Track genetic trends for breech wrinkle, breech cover, dags and higher productivity | 00775 also has application in this area | Beyond the Bale article | YES |

BREECH MODIFICATION PROCEDURES

THE AIM: Breech modification procedures to improve lifetime resistance to flystrike.

| Stated Outcome | Activity during Reporting Period – 0 Projects | Comment | Outcome Achieved |
|---|---|---------------------------------|------------------|
| Undertake further R&D of the animal welfare impacts of breech modification procedures. | No activity | | |
| Undertake further R&D to refine the application protocols for breech modification procedures. | No activity | | |
| Support best practice mulesing training. | | See extension activities below. | YES |

EDUCATION, EXTENSION AND PROMOTION

THE AIM: Adoption of best practice strategies to improve the lifetime welfare of sheep, reduce reliance on mulesing and support transparency in the supply chain.

| Stated Outcome | Activity during Reporting Period – 8 Projects | Comment- 5 ongoing, 1 commenced | Outcome Achieved |
|--|---|---|------------------|
| Develop and implement education, training and extension strategies to improve lifetime welfare of sheep. | 00790-ParaBoss III-commenced (with MLA) 00651 Lamb Marking Online Course-development | | YES |
| Monitor, evaluate and improve the success of education, training and extension strategies. | Within projects below | | |
| Engage with woolgrower advisors on the RD&E program. | 00651 Blowfly Chemical Resistance (DemystiFly)-publication 00765 Flystrike Extension Package (It's Fly Time! and SimpliFly) – webinar/workshop | Three It's Fly Time events held (900 participants) Other "Flies" due late 2022/2023 AmpliFly aimed at advisors. | YES |

| | | | |
|---|--|---|-----|
| | 00815 Breeding for Flystrike Resistance Workshops (ClassiFly) 00818 Moving to a Non-Mulesed Enterprise (StrateFly and AmpliFly) | | |
| Ongoing engagement with domestic and international stakeholders to ensure they understand best practice management of flystrike and the welfare implications. | 00816 - Sheep Sustainability Framework 00651 Welfare forum held 19 April 2022 | SSF launched April 2021, first report due July 2022 | YES |

Comparison between ram and wether lamb production - do we need to castrate lambs destined for slaughter?

Bruce Allworth¹, Shawn McGrath¹, Benjamin Holman² and James Stephens¹
Charles Sturt University¹ and NSW Department of Primary Industry²
Wagga Wagga NSW

Introduction

Sheep production in Australia includes routine castration of male lambs, at a young age, unless the lambs are to be used for breeding. Due primarily to welfare concerns, it is becoming more common to leave male lambs uncastrated in many countries, and it is now illegal to castrate lambs in some countries on welfare grounds. Leaving ram lambs entire may have positive welfare and production benefits, but it may also require changes to management such as ensuring lambs reach slaughter age at a young age to avoid meat taint.

On-farm Study

A study was conducted at Charles Sturt University (CSU) Farm, Wagga Wagga to assess the effects on management and production of leaving male Composite lambs uncastrated. In brief, Composite breed lambs were systematically allocated to one of two treatment groups at lamb marking, with every other male lamb allocated to the alternate group. Group 1 (n=133) were left uncastrated and Group 2 (n=132) were castrated using the Numnuts® system; all other procedures at lamb marking (tail docking, ear tagging and vaccinations) were the same. Lambs were weighed immediately after marking, again at weaning and prior to slaughter (after curfew). Ewes and lambs grazed monocultures of dual-purpose wheat then lucerne prior to weaning, and lambs grazed lucerne-dominant pastures post-weaning without supplementation. Male lambs (both castrated and uncastrated) were run as a single cohort through until lambs were sent to the abattoir for slaughter.

Ram lambs grew faster ($P < 0.001$) than wether lambs (315 v. 277 g/hd/day), resulting in heavier mean live weights of ram lambs at weaning. There was no difference ($P = 0.678$) in post-weaning growth rates (190 v. 187 g/hd/day) and the final live weight for rams was greater than for wethers (Table 1).

Table 1. Liveweight data (kg) for ram and wether lambs

| | Rams | Wethers |
|----------------------|-------------------|-------------------|
| Marking weight | 14.4 | 14.4 |
| Weaning weight | 31.1 ^a | 29.1 ^b |
| Pre-slaughter weight | 44.6 ^a | 42.5 ^b |

Row-wise superscripts indicate different means ($P < 0.05$).

Abattoir assessment

A cohort of 50 lambs (25 ram lambs, 25 wether lambs), balanced on liveweight, were slaughtered as a single flock, at a commercial Australian abattoir. Carcasses were eviscerated, dressed and inspected as per standard industry practice. Individual hot carcass weight (kg) was recorded at ~ 30 min post-slaughter and immediately prior to their scan with a commercially installed dual energy x-ray absorptiometer (DEXA, Scott Automation and Robotics), to estimate percentage of lean, fat and bone in the carcasses. The change in weight between the live animal and HCW was calculated as the dressing percentage. Medium voltage electrical stimulation was applied to each carcass before its entry into the holding chiller, whereupon the tissue depth at the girth rib (mm) site was measured. At 24 h post-slaughter, carcasses were fabricated and the loin saddles collected. Eye muscle area (cm²) and subcutaneous fat depth (mm) was measured from the exposed LL surface, at the 12th rib.

Table 2. Carcass parameters for ram and wether lambs

| | Rams | Wethers |
|------------------------------------|-------------------|-------------------|
| Hot Carcase Weight (kg) | 25.5 | 25.4 |
| Dressing% | 53.3 | 53.4 |
| Lean % | 56.8 ^a | 55.4 ^b |
| Fat % | 28.8 ^a | 30.5 ^b |
| Bone % | 14.5 | 14.2 |
| GR tissue depth (mm) | 21.7 | 21.6 |
| Eye muscle area (cm ²) | 14.8 | 13.5 |
| Subcutaneous fat depth (mm) | 3.1 | 3.8 |

Row-wise superscripts indicate different means ($P < 0.05$).

Ram lamb carcasses had a lower fat percentage, more lean muscle, and provided comparable yields to wether lambs.

Eating quality

Eating quality characteristics are also being assessed, using a untrained sensory (tasting) panel of 64 Australian consumers. Preliminary results suggest wether lambs performed better, being ranked significantly better on both tenderness and overall liking (Table 3).

Table 3. Preliminary sensory testing scores for ram and wether lambs

| | Rams | Wethers |
|----------------|-------------------|-------------------|
| Tenderness | 54.6 ^a | 61.0 ^b |
| Juiciness | 56.6 | 57.3 |
| Flavour | 63.7 | 66.3 |
| Overall liking | 61.5 ^a | 65.1 ^b |

Row-wise superscripts indicate different means ($P < 0.05$).

However, both ram and wether lambs had suitable scores for all parameters assessed, suggesting both were palatable.

Conclusion

This study demonstrated that ram lambs can be effectively managed for meat production on a commercial farm and achieved higher finished weights than wether lambs. Meat performance, based on both meat quality and eating quality assessments, was satisfactory for ram lamb meat, although meat from wether lambs was considered superior.

Insights into campylobacteriosis in maiden ewe flocks

Tom Clunea^a, Elsa Glanville^c, Angus Campbell^d, Amy Lockwood^a, Serina Hancock^a, Andrew N. Thompson^a, Sue Beetson^a, Mieghan Bruce^b, Daniel Brookes^c, Colin Trengove^e, Ryan O’Handley^e and Caroline Jacobson^a

^a Centre for Animal Production and Health, Murdoch University, Murdoch, WA

^b Centre for Biosecurity and One Health, Murdoch University, Murdoch, WA

^c Mackinnon Project, University of Melbourne, Werribee, Victoria

^d Nossal Institute for Global Health, University of Melbourne, Melbourne, Victoria

^e School of Animal and Veterinary Sciences, University of Adelaide, Roseworthy, SA

Summary

Campylobacteriosis is an important reproductive disease of sheep in Australia and maiden ewes are considered at greatest risk. However, the impacts of *Campylobacter* spp. on reproduction are poorly quantified. Here we show that exposure to *C. fetus* or *C. jejuni* was widespread on Australian sheep farms, but associations between titre and reproductive outcome were inconsistent in a case-control study of 22 flocks of maiden ewes. *Campylobacter fetus* abortions were confirmed with microbial culture in one flock, and *C. fetus* titres fluctuated and often waned by lamb marking in this flock. This has implications for using serology for diagnosis of campylobacteriosis. Serological evidence of exposure to *Campylobacter* spp. should be considered in the context of timing of sampling and risk factors specific to the flock. Apart from serology, strategies to investigate the impact of *Campylobacter* exposure on ewe reproductive performance includes monitoring ewes for evidence of abortion, lamb necropsies to determine aetiological diagnosis for abortions and perinatal mortalities, and vaccination trials.

Key points for practitioners

- Campylobacteriosis is an important cause of abortion and lamb mortalities in Australia and should be considered as a differential diagnosis during investigations into poor reproductive performance in ewes
- *Campylobacter* spp. serology should be interpreted with care; consider in the context of the timing of sampling and risk factors specific to the flock
- Bacterial culture or PCR detection along with supportive histopathological changes of aborted tissues remains the gold standard for diagnosis of campylobacteriosis
- Practitioners should always remember that lamb mortality is multifactorial – even in flocks where campylobacteriosis is diagnosed, there will be other causes of lamb mortality occurring simultaneously.

Introduction

Ovine campylobacteriosis caused by *Campylobacter fetus* or *C. jejuni* is one of the most frequently diagnosed causes of ovine abortion in Australia.^{1, 2} Maiden ewes are considered most susceptible due to a lack of immunocompetency afforded by previous natural infection. *Campylobacter* abortions occur both sporadically and in outbreaks, with up to 50% of ewes aborting in the latter.³ Naïve flocks are at greater risk of outbreaks. As well as abortion, campylobacteriosis also reduces the survival of neonatal lambs, increasing losses in the perinatal period. Some studies estimate annual lamb mortalities due to campylobacteriosis of around 10% in flocks where *Campylobacter* spp. are endemic.⁴ However, the extent of abortion and perinatal mortalities attributable to campylobacteriosis will vary between flocks and is difficult to quantify, particularly in extensively-managed sheep flocks.

Transmission of *Campylobacter* spp. occurs via ingestion of feed and water contaminated by faeces or aborted material, with no evidence of venereal spread in sheep.⁵ Risk factors for clinical campylobacteriosis on Australian farms have not been well defined, but may include

rainfall, stocking density, ewe age, feeding management, whether the flock is open or closed, previous natural exposure and vaccination status.

This paper will summarise the findings of a recent study investigating the role of *Campylobacter* spp. in abortion and lamb mortalities for maiden ewe flocks and described in more detail by Clune, et al. ⁶. Strategies to investigate suspected campylobacteriosis outbreaks and management of campylobacter outbreaks will also be discussed.

Methods

The methods for this study are described in more detail by Clune, et al. ⁶

Animals and study sites

A case-control study was performed between 2018 and 2020. Twenty-two flocks of approximately 200 non-Merino ewe lambs or maiden Merino hoggets in South Australia ($n=6$), Victoria ($n=5$) and Western Australia ($n=11$) were monitored during pregnancy and lambing. Study flocks were not vaccinated against *Campylobacter* spp.. However, on some farms, there were other sheep cohorts that had received Coopers Ovilis® Campyvax® *Campylobacter* vaccine for sheep (Coopers, MSD Animal Health, Australia). Each farm ran self-replacing flocks and maiden ewes were managed as per standard farm practice.

Reproductive outcome

Pregnancy scanning was conducted twice to determine foetal number and viability. Scan 1 was conducted at an average of 85 days from the start of joining. Scan 2 was conducted at an average of 118 days from the start of joining and on average 33 days after scan 1. Loss of pregnancy or foetal viability between scans was categorised as *in utero* foetal loss and confirmed with lambing records and ewe lactation status at marking. Ewes and lambs were monitored daily over the lambing period. On most farms, lambs were tagged within 24 hours of birth, recorded as live or dead and their dam was identified. Lamb survival per ewe was assessed to marking. Lambs that were dead at lambing rounds (died between birth and tagging) were categorised as 'born' and therefore mortality was included in the birth - marking period (*i.e.*, born but did not survive to marking). Ewe udders were assessed as wet (lactating) or dry (not lactating) at marking.

Serology

Serum samples were collected from all maiden ewes in the study at five timepoints; pre-joining, scan 1, scan 2, pre-lambing and lamb marking. Subsets of ewes that raised lambs ($n = 10$) and ewes that failed to raise lambs ($n = 10-20$) were selected to determine antibody titres to both *C. fetus* and *C. jejuni* using Agar Gel Immunodiffusion (AGID) test, performed by ACE Laboratories, Bendigo, Victoria. Serology was performed using blood samples collected at lamb marking. For cases where a marking sample was not available, samples from the latest available timepoint were screened instead. Titres $\geq 1:10$ were categorised as 'exposed' and $\geq 1:80$ categorised 'positive' (Table 1) as previously described.⁷⁻⁹

Statistical analyses

Ewe flock and *Campylobacter* spp. titre categories are shown in Table 1. Lamb mortality was calculated based on the number of foetuses identified at scan 1 and the number of lambs marked. Mid-pregnancy abortion was expressed as a proportion (%) using the number of ewes with pregnancy loss between scan 1 and scan 2. A farm or flock was classified as seropositive if at least one ewe had a titre above the specified threshold (Table 1). Seroprevalences were compared using a Pearson Chi-squared test (two-tailed) and 95% confidence intervals were determined using Jeffrey's method.¹⁰ Odds ratios for failing to raise a lamb were calculated for (i) 'exposed' maiden ewes compared to ewes that were not exposed (titre $< 1:10$), and (ii) 'positive' maiden ewes compared to non-positive ewes (titre $< 1:80$) using logistic regression with flock included as a fixed effect, and are presented as odds ratio OR, 95% confidence interval CI. Odds ratios were determined for failing to rear for

different *Campylobacter* spp. titre categories ((i) 1:10 to 1:40; (ii) 1:80; and (ii) \geq 1:160) compared to titres <1:10 using logistic regression with flock included as a fixed effect.

Table 1: Ewe flock and *Campylobacter* spp. titre category definitions

| Category | Definition |
|------------------------|---|
| Ewe lambs | Primiparous ewe mated at 7-10 months of age |
| Maiden hoggets | Primiparous ewe mated at 18-20 months of age |
| Mature ewes | Multiparous ewes aged 3-years of age or older |
| Mid-pregnancy abortion | Foetal loss detected between scan 1 and scan 2 |
| Fail to rear | Maiden ewe determined to be pregnant at Scan 1 that subsequently failed to rear a lamb to lamb marking |
| Exposed ewe | Ewe with <i>C. fetus/C. jejuni</i> titre \geq 1:10 |
| Exposed flock | Ewe flock (ewe lamb, maiden hogget or mature ewe flock) with <i>C. fetus/C. jejuni</i> titre \geq 1:10 detected in at least one ewe |
| Exposed farm | Farm with <i>C. fetus/C. jejuni</i> titre \geq 1:10 detected in at least one ewe (regardless of age) |
| Positive ewe | Ewe with <i>C. fetus/C. jejuni</i> titre \geq 1:80 |
| Positive flock | Ewe flock (ewe lamb, maiden hogget or mature ewe flock) with <i>C. fetus/C. jejuni</i> titre \geq 1:80 detected in at least one ewe |
| Positive farm | Farm with <i>C. fetus/C. jejuni</i> titre \geq 1:80 detected in at least one ewe (regardless of age) |

Results and Discussion

Reproductive outcomes in study flocks

Overall foetal and lamb mortality in maiden ewes ranged from 18 - 66% for ewe lambs and 20 - 53% for hoggets. Mid-pregnancy abortion was detected in 11/12 ewe lamb flocks and 6/10 hogget flocks. Mid-pregnancy abortion was detected for 5.7% of ewe lambs that were pregnant at scan 1 (220/4351). The frequency of mid-pregnancy abortion for ewe lamb flocks ranged from 0 - 23.8%. For hogget flocks, mid-pregnancy abortion was detected in 1.5% of pregnant ewes (16/1886), with the frequency ranging from 0 - 4.4%.

Lamb mortality between scanning and marking in this study was consistent with a recent Australian survey that reported lamb mortality of 33% for non-Merino ewe lambs and 25% for maiden Merino hoggets.¹¹ Lamb mortality was an important contributor for poor marking rates observed in these flocks, and included both abortions and perinatal mortality. Lamb necropsies and laboratory investigation were performed for a subset of cases to determine aetiological diagnosis.^{6, 12} Lamb mortalities in the perinatal period were mainly due to dystocia, stillbirth and starvation-mismothering¹², with both infectious and non-infectious aetiologies contributing to lamb mortalities.¹

Detection of seropositivity to *Campylobacter* spp.

The frequency of individual ewe seropositivity detected at the 'exposed' (titre \geq 1:10) and 'positive' (titre \geq 1:80) antibody titre cut-offs for *C. fetus* and *C. jejuni* are outlined in Table 2, and described in more detail by Clune, et al. ⁶.

There was evidence of widespread exposure to *Campylobacter* spp. across the study farms with *C. fetus* 'exposed' ewes detected in 82% of maiden ewe flocks and *C. jejuni* 'exposed' ewes detected in all (100%) of maiden ewe flocks. The level of exposure to *C. fetus* was variable between flocks. There was no serological evidence of 'exposure' to *C. fetus* in 4/22 flocks, all of which were from Western Australia. There were no 'positive' maiden ewes detected in 14/22 flocks.

C. fetus titres ranged between 0 and 1:80 for ewes in most (18/22) maiden flocks. Titres $\geq 1:160$ were detected in 4/22 flocks; 3/5 flocks from Victoria and 1/6 flocks from South Australia. None of the flocks from Western Australia had maiden ewes with titres $\geq 1:160$.

Table 2: Individual seroprevalence with 95% confidence interval (CI) for *C. fetus* and *C. jejuni* in maiden ewe lambs or hoggets selected based on reproductive outcome (raised lambs vs failed to rear), and randomly selected mature ewes (*C. fetus* only) across all farms. Table adapted from Clune, et al. ⁶

| | Tested | | Exposed (titre $\geq 1:10$) | | Positive (titre $\geq 1:80$) | | |
|-------------------------|------------|------------|------------------------------|-------------------|-------------------------------|-------------------|-------------------|
| | (n) | n | % | 95% CI | n | % | 95% CI |
| <i>C. fetus</i> | | | | | | | |
| Maiden ewe lambs | 260 | 84 | 32.3 ^a | 26.8, 38.2 | 37 | 14.2 ^a | 10.4, 18.9 |
| Maiden hoggets | 202 | 47 | 23.3 ^b | 17.8, 29.4 | 20 | 9.9 ^a | 6.3, 14.6 |
| Mature ewes | 210 | 114 | 54.3 ^c | 47.5, 60.9 | 65 | 31.0 ^b | 25.0, 37.4 |
| TOTAL | 672 | 245 | 36.5 | 32.9, 40.2 | 122 | 18.2 | 15.4, 21.2 |
| <i>C. jejuni</i> | | | | | | | |
| Maiden ewe lambs | 260 | 248 | 95.4 ^a | 92.3, 97.4 | 121 | 46.5 ^a | 40.5, 52.6 |
| Maiden hoggets | 202 | 195 | 96.5 ^a | 93.3, 98.4 | 83 | 41.1 ^a | 34.5, 48.0 |
| TOTAL | 462 | 443 | 95.9 | 93.8, 97.4 | 204 | 44.2 | 39.7, 48.7 |

^{abc} Values within *Campylobacter* species and titre category with different superscript letters are significantly different using two sample z-test to compare sample proportions (two-tailed) $P < 0.05$
CI: confidence interval

Our findings were consistent with previous studies where ‘exposure’ was detected in 80% (170/218) of flocks for *C. fetus* and 95% (198/208) of flocks for *C. jejuni*.^{9, 13} This suggests *Campylobacter* spp. ‘exposure’ is widespread on Australian sheep farms and often occurs from a young age.

The risk of exposure likely varies between states and regions, with lower rates of seropositivity observed in Western Australian flocks compared with South Australian and Victorian flocks. This was consistent with variable frequency of diagnosis of campylobacteriosis by state veterinary laboratories.¹ Differences between regions and flocks may be related to rainfall, stocking density, ewe age and feeding management.

Immunological naivety is a risk factor for abortion due to campylobacteriosis, with previous studies indicating that convalescent ewes develop protective immunity.^{14, 15} The low level of seroconversion for *C. fetus* detected in some flocks suggests a low level of immunity towards *C. fetus* is in these flocks and a greater risk of clinical campylobacteriosis if exposure occurs via contaminated feed, water or contact with infectious material. However, the degree of protection relative to antibody titre is unclear and requires further research.

Association between seropositivity and reproductive outcome: *Campylobacter fetus*

There were inconsistent associations between pregnancy outcome and *C. fetus* seropositivity. ‘Exposed’ maiden ewes had higher odds of failing to rear compared to ewes with no evidence of exposure when adjusted for farm effects (OR 2.0, 1.09, 3.77; $P = 0.027$). However, there was no difference in the odds of failing to rear between maiden ewes with ‘positive’ titres and those with titres $< 1:80$ ($P = 0.19$). In ewe lamb flocks ($n=12$), ‘positive’ ewe lambs had higher odds of failing to rear than ewe lambs with titres $< 1:80$ (OR 2.59, 1.03, 6.78; $P = 0.047$). There was no association between ‘positive’ titre and failing to rear a lamb for maiden hogget flocks ($n=10$ flocks; $P=0.440$).

There was no significant increase in the odds of failing to rear a lamb for ewes with *C. fetus* titres of (i) 1:10 to 1:40, (ii) 1:80, and (iii) $\geq 1:160$ compared to ewes with titres $< 1:10$.⁶ However, there were insufficient ewes with titres $\geq 1:160$ across all study flocks to reliably determine whether titres $\geq 1:160$ at lamb marking were associated with increased abortion or lamb mortality rates.

The interpretation of antibody titres for *C. fetus*. and determining the association with the reproductive outcome is challenging. Seropositivity does not confirm current infection status or causality of abortion or lamb mortality, and 'exposed' or 'positive' titres may be detected in clinically healthy sheep.^{16, 17} This may reflect persistent antibodies, infections outside of the period of risk for reproductive disease, insufficient intensity of infectious challenge, variation in ewe immunity and strain pathogenicity^{8, 18, 19} In our study, *C. fetus* 'exposure' increased the odds of failing to rear in maiden ewes, and 'positive' titres increased odds of failing to rear only in the subset of ewe lambs. However, overall *C. fetus* titre was a poor indicator of failure to rear for the flocks in this study. This was confounded by the infrequent detection of high titres for *C. fetus* in maiden flocks, and multifactorial causes of abortion and lamb mortality.

As well as serology, other strategies for investigating the impact of *C. fetus* exposure on flock reproductive performance could include monitoring ewes for evidence of abortion, lamb necropsies to determine aetiological diagnoses and/or a vaccination trial.^{4, 8}

Association between seropositivity and reproductive outcome: *Campylobacter jejuni*

There was no increase in the odds of failing to rear a lamb in *C. jejuni* 'exposed' maiden ewes ($P = 0.506$).

Maiden ewes that were *C. jejuni* 'positive' had lower odds of failing to rear a lamb compared to ewes with *C. jejuni* titre of <1:80 (OR 0.46; 0.24, 0.87; $P=0.018$). This was the reverse of what we would expect to observe if *C. jejuni* was an important contributor to abortions and lamb mortalities in these flocks.

Similarly, there was no evidence of increased odds of failure to rear for ewes with *C. jejuni* titres of (i) 1:10 to 1:40, (ii) 1:80, and (iii) $\geq 1:160$ compared to a titre of <1:10.⁶

Our findings show that whilst ewes were commonly exposed to *C. jejuni*, there was no evidence that seropositivity was associated with poor reproductive outcomes at any of the titre thresholds. *Campylobacter jejuni* is a common commensal organism of the ovine gastrointestinal tract, and the aetiopathogenesis of *C. jejuni* abortion requires further elucidation.

Fluctuation of *C. fetus* titres

Campylobacteriosis was diagnosed in one of the 22 flocks in this study by microbial culture using opportunistically recovered aborted material. Testing of aborted and stillborn tissue samples was not performed in all flocks. It is therefore possible that campylobacteriosis causing foetal and lamb mortality occurred undetected in other flocks.

The ewe lamb flock where campylobacteriosis was confirmed was located in Victoria. This was the only flock in the study where the proportion of *C. fetus* "positive" ewes was higher for those that failed to rear compared to ewes that raised lambs (85% vs 20%, $P < 0.001$). Furthermore, this was one of four flocks where titres $\geq 1:160$ were detected.

Campylobacter fetus serology was conducted on serum samples for a subset of ewes with different reproductive outcomes with titres $\geq 1:10$ detected at the latest available timepoint (Table 3). Titres between mating and marking fluctuated in ewes with mid-pregnancy abortion and in many cases had declined by marking. This included 6/8 ewes that had *C. fetus* titre $\geq 1:320$ at scan 2 or pre-lambing. This indicates that *C. fetus* titres may rise transiently, and serology conducted at lamb marking (or later) could result in low or moderate titres in flocks where campylobacteriosis abortions occurred in mid-late pregnancy. The short and inconsistent window for detection of rising titres has implication for practitioners because the rise in titre may be missed by the time abortions or lamb mortalities due to *C. fetus* are detected by producers. Ultimately, while serology can be supportive of diagnosis, a definitive diagnosis of campylobacteriosis abortion and perinatal mortality should be based on

detection of *Campylobacter* spp. at necropsy of the foetus or lamb and not on serology from a single timepoint alone.⁷

Ewes with high titres at marking were evident in this flock (Table 3). However, even in the event of a *Campylobacter* spp. abortion outbreak, there will be multiple causes of perinatal lamb mortality and a random selection of ‘fail to rear’ ewes at marking will capture ewes that have lost lambs due to causes other than *C. fetus*. Hence, sufficient ewes must be sampled to detect high titres suggestive of recent infection. Based on assumptions of disease prevalence of 25% and test sensitivity of 85%, the required sample size from a mob of 200 is 13 ewes to be 95% confident disease has been detected. The sample size increases where expected prevalence is lower, with required sample size of 32 ewes at assumed prevalence of 10%. Further investigation to determine ‘typical’ prevalence and validate the sensitivity and specificity for AGID serology with natural *Campylobacter* spp. infections in Australian sheep is required and will better inform sampling protocols.

Table 3: Serial *Campylobacter fetus* titres for maiden ewes in a ewe lamb flock with campylobacteriosis confirmed by microbial culture. Table adapted from Clune, et al. ⁶

| Ewe ID | Mating | Scan1 | Scan 2 | Pre-lambing | Marking |
|---|--------|-----------------|--------|-------------|---------|
| Mid-pregnancy abortion (scan 1 – scan 2) | | | | | |
| 18650 | ≤1:10 | ≤1:10 | 1:320 | 1:320 | 1:160 |
| 18621 | ≤1:10 | ≤1:10 | 1:160 | 1:320 | 1:160 |
| 18698 | ≤1:10 | ≤1:10 | 1:160 | 1:160 | 1:80 |
| 18648 | ≤1:10 | ≤1:10 | 1:80 | 1:160 | 1:80 |
| 18551 | ≤1:10 | ≤1:10 | 1:80 | 1:320 | 1:160 |
| 18654 | ≤1:10 | ≤1:10 | 1:40 | 1:320 | 1:320 |
| 18557 | ≤1:10 | ≤1:10 | 1:40 | 1:320 | 1:40 |
| 18611 | ≤1:10 | ≤1:10 | 1:20 | 1:20 | 1:10 |
| 18595 | ≤1:10 | 1:40 | 1:160 | 1:160 | 1:160 |
| 18538 | 1:20 | 1:160 | 1:320 | 1:640 | 1:80 |
| Fail to rear (late abortion or perinatal lamb death) | | | | | |
| 18546 | <1:10 | NT ^A | 1:320 | NT | 1:160 |
| 18549 | <1:10 | NT | 1:80 | NT | 1:80 |
| 18548 | <1:10 | NT | 1:80 | NT | 1:80 |
| 18563 | <1:10 | NT | 1:80 | NT | 1:80 |
| 18553 | <1:10 | NT | 1:40 | NT | 1:80 |
| 18643 | <1:10 | NT | 1:40 | NT | 1:80 |
| 18610 | <1:10 | NT | 1:20 | NT | 1:160 |
| 18691 | <1:10 | NT | <1:10 | NT | 1:80 |
| 18571 | 1:80 | NT | 1:320 | NT | 1:320 |
| 18615 | 1:160 | NT | 1:80 | NT | 1:40 |
| Raised lambs | | | | | |
| 18628 | ≤1:10 | NT | NT | NT | 1:160 |
| 18690 | ≤1:10 | NT | NT | NT | 1:80 |
| 18632 | ≤1:10 | NT | NT | NT | 1:40 |
| 18703 | ≤1:10 | NT | NT | NT | 1:20 |
| 18708 | ≤1:10 | NT | NT | NT | 1:20 |
| 18709 | ≤1:10 | NT | NT | NT | 1:20 |
| 18663 | ≤1:10 | NT | NT | NT | 1:10 |
| 18555 | 1:160 | NT | NT | NT | 1:40 |
| 18642 | 1:160 | NT | NT | NT | 1:40 |
| 18625 | 1:160 | NT | NT | NT | 1:10 |

^A NT: not tested

Conclusion

Exposure to *Campylobacter* is variable, but widespread across Australia. This study confirms that exposure to *Campylobacter* during pregnancy can result in abortions and perinatal lamb mortalities on some farms in some years. However, interpreting *Campylobacter* serology is challenging as the association between the titre at marking and reproductive outcome is inconsistent. The relationship may be more consistent on farms with *Campylobacter* abortion outbreaks but insufficient outbreaks occurred on participating farms to confirm this. Therefore, serology should be used in combination with other strategies to determine an association with reproductive disease such as monitoring ewes for abortions, determining aetiological diagnoses for foetal and lamb mortality using necropsies, or vaccination trials. An improved understanding of *Campylobacter* antibody dynamics and risk factors for disease at region- and farm- levels will support veterinarians in making sound interpretation of *Campylobacter* serology to inform evidence-based recommendations for risk management and diagnosis of disease. Bacterial culture or PCR detection along with supportive histopathological changes of aborted tissues currently remains the gold standard for confirming the role of *Campylobacter* in reproductive losses in sheep.

Acknowledgments

This research was funded by Meat and Livestock Australia (B.AHE.0318). Tom Clune was supported with post graduate scholarships from Meat and Livestock Australia, Sheep Industry Business Innovation (Department of Primary Industries and Regional Development, Western Australia) and the 2020 Science and Innovation Awards for Young People in Agriculture, Fisheries and Forestry (Australian Government Department of Agriculture in partnership with Australian Wool Innovation). Some of the laboratory diagnostic testing was supported through the DPIRD Abortion Surveillance Scheme.

We thank the participating farmers who provided access to their animals and facilities, conducted lambing rounds, and collected and stored lambs for necropsy.

We thank the team at ACE Laboratory, and Celia Smuts, Janine Simmonds and the staff at VetPath. We also thank Shane Besier, Sam Hair, Richmond Loh and Anna Erickson at DPIRD.

We thank Tom La and Nyree Phillips (Murdoch University), Louis Lignereux and Rob Paterson (University of Adelaide), Andrew Whale, Mary McQuillan and Patrick Hannemann (Livestock Logic, Hamilton, Victoria), Sean McGrath (Millicent Veterinary Hospital), Simon Edwards and Michelle Smart (Willunga Veterinary Hospital), and Lauryn Stewart and Deb Lehmann (Kangaroo Island Veterinary Hospital) for assistance with sample collection and feedback on project design.

We thank Johann Schroder for helpful feedback on the original manuscript and project design.

Conflict of Interest

None of the authors of this paper have a financial or personal relationship with other people or organisations that could inappropriately influence or bias the content of the paper. The funding body (Meat and Livestock Australia) approved the manuscript for publication but was not involved in the collection, analysis or interpretation of data, or in the writing of the manuscript.

References

1. Clune T, Beetson S, Besier S et al. Ovine abortion and stillbirth investigations in Australia. *Aust Vet J* 2021;99:72-78. DOI: <https://doi.org/10.1111/avj.13040>
2. Refshauge G, Atkinson T, Robertson SM et al.: Reducing Kid Loss – Select and Project. Phase 1 Final report. Meat and Livestock Australia; North Sydney: 2020: Available at:

<https://www.mla.com.au/research-and-development/reports/2020/reducing-kid-loss-select-and-protect--phase-1/> Accessed: October 2021

3. Clough W. A review of ovine *Campylobacter* in Australia. *Proceedings of the Australian Sheep Veterinarians Annual Conference*. Australian Sheep Veterinarians, Cairns, 2003:181-184.
4. Anderson P. The implications of *Campylobacter* infections in ewe flocks. *Proceedings of the Society of Sheep and Beef Cattle Veterinarians of the New Zealand Veterinary Association*. The Society of Sheep and Beef Cattle Veterinarians of the New Zealand Veterinary Association, 2001.
5. Mearns R. Other Infectious Causes of Abortion. In: Aitken ID, editor. *Diseases of Sheep (fourth edition)*. Blackwell Publishing, Oxford, 2007:127-137.
6. Clune T, Briuce M, Glanville E et al. Seropositivity to *Campylobacter* and association with abortion and lamb mortality in maiden ewes from Western Australia, South Australia and Victoria. *Aust Vet J* 2022;In press
7. Dempster RP, Wilkins M, Green RS, de Lisle GW. Serological survey of *Toxoplasma gondii* and *Campylobacter fetus fetus* in sheep from New Zealand. *N Z Vet J* 2011;59:155. DOI: 10.1080/00480169.2011.579240
8. Glanville EJ, 2017. The effect of vaccination against *Campylobacter* on maiden ewe reproduction in Victoria (thesis). University of Melbourne.
9. Walsh J. *Campylobacter* infection in sheep: Field survey results and vaccination outcomes. *Proceedings of the Australian Sheep Veterinarians Annual Conference*, Dubbo, 2016:158-163.
10. Brown LD, Cat TT, DasGupta A. Interval estimation for a proportion. *Stat Sci* 2001;16
11. Hutchison D, Clarke BE, Hancock S et al. Lower reproductive rate and lamb survival contribute to lower lamb marking rate in maiden ewes compared to multiparous ewes. *Animals* 2022;12:513. DOI: 10.3390/ani12040513
12. Clune T, Besier S, Hair S et al. *Chlamydia pecorum* detection in aborted and stillborn lambs from Western Australia. *Vet Res* 2021;52:84. DOI: 10.1186/s13567-021-00950-w
13. Wills F. Survey of *Campylobacter fetus* and *Campylobacter jejuni* seroprevalence in Australian sheep. Sheep Goats and Camelid Veterinarians (Australian Veterinary Association), Australian Sheep Goats and Camelid Veterinarians Annual Conference, Wagga Wagga, 2021.
14. Jensen R, Miller VA, Hammarlund MA, Graham WR. Vibrionic abortion in sheep. I. Transmission and immunity. *Am J Vet Res* 1957;18:326-329
15. Frank FW, Waldhalm DG, Meinershagen WA, Scrivner LH. Newer knowledge of ovine vibriosis. *J Am Vet Med Assoc* 1965;147:1313-1318
16. Yang R, Abraham S, Gardner GE, Ryan U, Jacobson C. Prevalence and pathogen load of *Campylobacter* spp., *Salmonella enterica* and *Escherichia coli* O157/O145 serogroup in sheep faeces collected at sale yards and in abattoir effluent in Western Australia. *Aust Vet J* 2017;95:143-148. DOI: 10.1111/avj.12572
17. Yang R, Jacobson C, Gardner G et al. Longitudinal prevalence, faecal shedding and molecular characterisation of *Campylobacter* spp. and *Salmonella enterica* in sheep. *Vet J* 2014;202:250-254. DOI: 10.1016/j.tvjl.2014.08.001
18. Sahin O, Fitzgerald C, Stroika S et al. Molecular evidence for zoonotic transmission of an emergent, highly pathogenic *Campylobacter jejuni* clone in the United States. *J Clin Microbiol* 2012;50:680-687. DOI: 10.1128/jcm.06167-11
19. Grogono-Thomas R, Blaser MJ, Ahmadi M, Newell DG. Role of S-layer protein antigenic diversity in the immune responses of sheep experimentally challenged with *Campylobacter fetus* subsp. fetus. *Infect Immun* 2003;71:147-154. DOI: 10.1128/IAI.71.1.147-154.2003

Investigating the cause and prevention of red gut in lambs grazing lucerne

Tatjana Dobrijevic
School of Animal and Veterinary Sciences, The University of Adelaide
Roseworthy SA 5371

Introduction

Red gut is an enteric disease of sheep that occurs on high protein low fibre diets, predisposing to gastro-intestinal instability.¹ Volvulus of the intestinal mass may follow leading to an intensely red coloured obstruction, shock, and sudden death.² Currently, fibre supplementation and rotational grazing are the only recommended preventative strategies.³ The highest risk is for lambs grazing lucerne pastures, particularly monocultures grown for seed production.² Often these pastures are 'winter cleaned' which involves the application of herbicides to remove grasses and weeds with a consequence of a higher protein lower fibre diet for lambs.⁴ Red gut prevalence has been reported at up to 10% in lambs in the upper south east of South Australia.

This study aimed to identify any significant associations between farming practices and the prevalence of red gut as well as recording the impact of a calcium-based foliar spray on disease prevalence. It was hypothesised that farm management factors would be found that influence the prevalence of red gut in lambs grazing lucerne. It has also been postulated that nutrient deficiencies, especially calcium, associated with the characteristic sandy soils in the study area predisposes to red gut through reduced plant fibre content.⁵

Methods and Materials

The study concentrated on lucerne seed producers that were in the upper south east of South Australia. A total of 27 producers participated in a survey to collect information regarding their farm management practice, particularly concerning lambs grazed on lucerne pastures. Study participants were asked to promptly report lamb deaths that occurred while grazing on lucerne in July and August 2021 so that a post-mortem could be conducted within 48 hours of death. Post-mortem findings and grazing history provided by the farmer, were used to assign a cause of death to each animal.

A calcium-based foliar was also applied to selected grazed paddocks and compared to paired untreated grazed paddocks to see if this influenced red gut prevalence. Plant mineral content and feed quality of the treated and untreated paddocks were also monitored. All data was entered into Excel and imported for analysis in SPSS, using frequencies and a series of multiple linear regression analyses using the MIXED procedure.

All participating producers were contacted to answer a short questionnaire at the conclusion of the study to gather data on the number of weaned lamb and ewe deaths, environmental conditions, lucerne management and any other notable events occurring during the trial period.

Results

A total of 17 deaths were investigated: 71% (n = 12) due to enterotoxaemia, 24% (n = 4) red gut, and 6% (n = 1) ruminal acidosis (Figure 1). It was noted that red gut is not restricted to weaned lambs as it was also diagnosed as the cause of death in one ewe. Additionally, it was noted to occur in sheep grazing both pure lucerne and composite pastures ranging from very short to lush growth. Insufficient sample size prevented statistical investigation of associations between red gut and farming practices. No differences were found in the nutrient analyses between the treated and untreated paddocks. All trial paddocks recorded high crude protein and metabolisable energy content, coinciding with lower fibre content and were considered high risk pastures for grazing sheep.

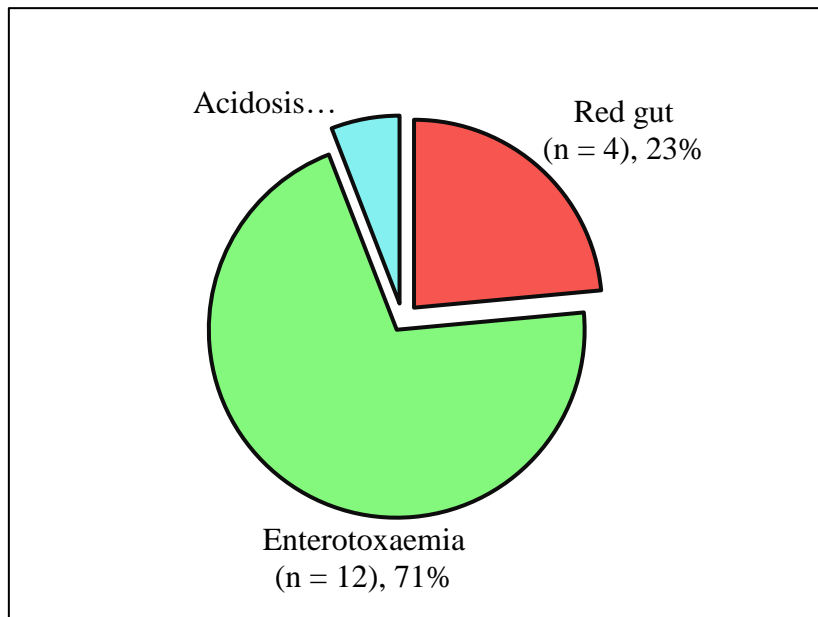


Figure 1: Distribution of lamb and ewe deaths during July – August 2021 based on post-mortem findings.

25 of the original 27 producers completed the concluding questionnaire and forty two percent reported no lamb or ewe deaths on lucerne during the July - August study period. Results from the concluding questionnaire show that of the producers that did record deaths, 25% had less than 6 deaths, 21% had 6-10 deaths and the remaining 13% experienced more than 10 deaths during this period. Many producers suggested that a reason for the lack of lamb deaths may have been due to a late start to the season delaying lucerne growth by up to a month.

Conclusions

Low sample size and low mortality rate were the main limitations to the investigation. It was concluded that red gut was less prevalent in this study compared to previous reports, likely due to a range of factors including previous misdiagnosis, seasonal differences, and modified management practices. Producers appear to have reduced the occurrence of red gut through improved feed supplementation and grazing management strategies. Similar limitations were evident in the foliar trial where no deaths were experienced and there were no significant differences in mineral or other feed components between control and treated paddocks based on the soil, plant, and feed analyses conducted. Further studies on these farm management practices including the use of foliar sprays would help to elucidate red gut prevention strategies.

References

1. Gumbrell RC, Redgut in sheep: A disease with a twist. *New Zeal Vet J* 1997;45:217-221.
2. Jagusch KT, Gumbrell RC, Dellow DW, Red gut in lamb lucerne grazing trials at Lincoln. *New Zealand Society of Animal Production* 1976;36:190-197.
3. Jagusch KT, Gumbrell RC, Mobley MC, Jay NP, (1977) Effects on growth of early weaning lambs on to pure stands of lucerne. *New Zeal J Exp Agr* 1977;5:15-18.
4. dpi.nsw.gov.au. Lucerne for pasture and fodder, NSW Agriculture.; c2003 [Cited 2022 May 9]. Available from: https://www.dpi.nsw.gov.au/data/assets/pdf_file/0010/164737/p2225pt1.pdf
5. Mintz C, Dal Grande E, Trengove CL. Epidemiology of red gut in lambs grazing lucerne. *Proceedings of the 33rd Australian Association of Animal Sciences conference, Fremantle.* 2021;33:195

Acute selenium toxicity in lambs: two overdose scenarios

Elsa Glanville¹, Bruce Watt²

¹Mackinnon Project University of Melbourne, ²NSW Local Land Services

¹Faculty of Veterinary & Agricultural Sciences, Werribee VIC, ²66 Corporation Drive, Bathurst NSW

Introduction

Selenium is an essential trace element, with deficiency resulting in clinical disease and/or subclinical production loss.¹ The potential growth and production benefits associated with selenium have resulted in its inclusion in many livestock products including drenches, vaccines, lick blocks and water supplements. However, selenium is not benign, and overdose can result in acute or chronic toxicosis. Acute toxicity in lambs results in cardiac failure causing lethargy, respiratory distress, neurological disturbance and death, mostly within 72 hours of exposure.² Fatal dose rates of 1-2.2 mg/kg (oral)³⁻⁵ and 0.45-1 mg/kg (injected)² are reported. As selenium is often included in products routinely administered to livestock, several overdose scenarios are possible, including overdosing one product or multiple products given simultaneously. Two cases of selenium intoxication are presented from the Central Tablelands of NSW that illustrate these scenarios and serve as a reminder of the risks associated with selenium supplementation and the ease with which fatal, costly toxicity can occur.

Case 1

In mid-December 2021 near Bathurst NSW, two mobs of September drop Merino lambs (3-4 mth old, 470 <18 kg 'light draft', 630 >18 kg 'heavy draft') were weighed (8-24 kg), vaccinated (5-in-1 only), drenched (Startect® 10 mg/ml derquantel and 1 mg/ml abamectin, to 25 kg) and weaned into containment yards (feed: Lucerne hay, started at 50 g/head/d pellet/barley mix, mixed weed; water: bore water to high-flow, low-volume troughs; 10 ml/head/day liquid mineral supplement containing 1500 mg/L sodium selenate for three days after weaning). The producer noted intermittent lamb deaths pre-weaning, shortly after imprint feeding. Twenty-four hours after weaning, four lambs were found dead across both pens, with no obvious external abnormalities. The next day, the producer noticed increasing numbers of lambs foaming from the mouth and nares, panting, walking with a stilted gait and another dead with foam around the nares. Seven more dead lambs were found in the 'light' mob and one more in the 'heavy' mob. A private veterinarian was called to investigate.

Clinical examination

At least six lambs with respiratory distress, lethargy and/or neurological signs were noted in each mob. These lambs were tachypneic, with foam at the mouth, and had pink mucous membranes with rapid refill. They stood with a high head carriage and walked with a stiff, stilted gait. Unaffected lambs were calm, bright and alert.

Necropsy

One moribund lamb from the 'light' mob and five recently deceased lambs were presented for necropsy. In total, three lambs were presented from each mob.

'Light' mob lambs

Lamb 1: 10 kg emaciated, non-responsive lamb. Normal respiratory excursions. Humanely euthanased by producer. Necropsy revealed an extensive abscess extending from Gudair® vaccine site on right neck proximal to base of ear. Purulent material tracked through fascial planes into atlas/axis, extending cranially and caudally through spinal canal. The carcass was malnourished, but the gross necropsy was otherwise within normal limits.

Lamb 2: small lamb dead in lateral recumbency. White foam from both nares. No other external abnormalities. Carcass in fair condition. Diffuse muscle pallor with flabby texture, most notable in hindlimbs. Abundant straw-coloured fluid in peritoneal, pericardial and pleural cavities. Stable foam through trachea to the bifurcation. Lungs heavy and wet, lesions consistent with pulmonary oedema. Cardiac muscle was mottled with areas of pallor.

Lamb 3: light lamb in poor body condition. No obvious external abnormalities. Gelatinous atrophy of pericardial and perirenal fat, blunt rumen papillae and little rumen fill. No other gross abnormalities. A most likely diagnosis of malnutrition was made, but fresh liver and kidney samples were taken.

'Heavy' mob lambs

Lambs 4 and 5: heavy lambs, good condition, dead in lateral recumbency. Similar to Lamb 2 with foam from nares and no other external abnormalities. Abundant straw-coloured pleural, pericardial and peritoneal fluid. The main bronchi and trachea were filled with stable foam. Lungs were congested, heavy and wet with lesions consistent with pulmonary oedema. The heart of lamb 4 was noticeably discoloured, with regions of pallor extending out from coronary arteries. Skeletal muscles were pale with a flabby texture, especially through the hindlimbs. The liver of each lamb was congested and moderately enlarged. Fresh liver and kidney samples were taken.

Lamb 6: dead more than four hours. Abdominal distension. A provisional diagnosis of ruminal acidosis was made based on gross necropsy findings. Liver and kidney were sampled. The preliminary diagnosis for the main presenting syndrome, based on gross necropsy findings and clinical signs, was selenium intoxication. The time that had elapsed between initial access to the likely source of the excess selenium and the presentation of signs, 12-24 hours, was consistent with previous investigations of oral selenium intoxication.⁶

Laboratory finding

Samples were unable to be taken for histopathology. Fresh liver samples were submitted for selenium and copper concentrations based on a strong suspicion of selenium intoxication and a potential for elevated copper based on the potential quantity of supplement ingested. Kidney samples were taken but not analysed due to the conclusive results from the liver. Liver selenium from clinically consistent cases (lambs 2, 4, 5) was markedly elevated (102-164 umol/kg wet weight; range: 1.3-19 umol/kg wet wt). The non-clinically consistent case, lamb 3, had a normal liver selenium concentration (4.8 umol/kg wet wt). Liver copper was normal.

The laboratory findings from case 1 confirmed that the cause of death for lambs with a history of sudden death, respiratory and neurological signs was selenium intoxication. Based on the management history, the intoxication occurred due to excess intake of supplement provided in the water troughs. The likely amount of selenium ingested relative to toxic levels is discussed below.

Case 2

Lambs from two mobs of spring lambing ewes from the Central Tablelands NSW were marked in October 2021. Mob 1, 209 Merino x White Suffolk lambs, was marked on 8 October. The marking procedure included ears marked and tagged, rings on tails and scrotums, Clik® on breach, Gudair® injected below the ear (ewe lambs only) and clostridial vaccination containing selenium. All lambs were drenched with 2 ml Triguard® (Boehringer Ingelheim, Ryde, Australia) for sheep (1 ml/ 5 kg; 1.0 g/L abamectin, 22.7 g/L oxfendazole, 33.9 g/L

levamisole, 0.5 g/L selenium, 2.2 g/L cobalt). No deaths occurred in the first mob. Mob 2, 227 Merino x Border Leicester lambs, was marked on 17 October. In addition to the previously procedure, lambs from Mob 2 also received 0.2 ml Multimin Copper-free® injectable supplement for sheep and cattle into the right axilla (5 mg/ml selenium, 10 mg/ml manganese, 40 mg/ml zinc; axilla is not the recommended injection site).

At least 24 (10.6%) lambs died over the following 48 hours. Two lambs died with reports of respiratory distress and excess 'drool' when returned to their paddock. Nineteen lambs were found dead in the paddock the next day (18th October), and another three on 19th October. More dead lambs may have been missed in the paddock. This case was investigated by Central Tablelands Local Land Services.

Clinical examination

Observation of ewes and lambs in the paddock revealed that most lambs were lethargic and lame on the right-fore leg. Some were lame on the left-fore.

Necropsy

The three dead lambs found on the 19th October were necropsied.

Lamb 1: 8 kg male. Lungs heavy, pink and wet especially cranioventrally. White foam in trachea. Otherwise no abnormalities.

Lamb 2: 10 kg male. Lungs heavy, dark pink and wet with multiple red blotches up to 1 cm diameter across the surface of all lung lobes. Abundant white foam in trachea. Otherwise no abnormalities.

Lamb 3: 5 kg male. Lungs normal, pink. No foam in trachea. Gelatinous subcutaneous tissue in right axilla. Otherwise no abnormalities.

Serum, fresh liver and fresh kidney were submitted for selenium analysis. Fixed samples of lung, liver and kidney were submitted for histopathology.

Laboratory and histopathology findings

Liver selenium levels were elevated in all three lambs, markedly so in lambs 1 (147 umol/kg wet wt) and 2 (203 umol/kg wet wt). Levels were slightly lower in lamb 3 (67.3 umol/kg wet wt). Serum selenium was slightly elevated in lamb 1 (7.4 umol/L, range: 0.5-6.5 umol/L), markedly elevated in lamb 3 (12 umol/L) but normal in lamb 2 (5.4 umol/L). Kidney selenium was only slightly elevated in lamb 1 (43.7 umol/kg wet wt, range: 8-38 umol/kg wet wt) but was high normal in lamb 2 (33.9 umol/kg wet wt) and 3 (35 umol/kg wet wt). The kidney levels likely reflects the longer time taken for selenium to accumulate in the kidneys compared to the liver, and the death of the lambs before such time had elapsed.

Histopathology from all three lambs revealed periacinar hepatocyte degeneration, with possible early transition to necrosis in Lamb 2, and congested sinusoids, most severe in Lamb 3. The lungs were congested with variably severe haemorrhages, variable evidence of intra-alveolar oedema (mostly Lamb 2) and variable minimal to mild neutrophilic inflammatory response (Lamb 1, and 2). No significant changes were noted in the kidney. In general, the changes in the lung and liver were acute and non-specific, with acute cardiac failure a potential cause.^{6, 7}

Discussion

These two cases demonstrate the relative ease with which young sheep can receive an accidental selenium overdose with fatal consequences. Here we report acute, fatal selenium

intoxication. Cardiac insufficiency underlies the pathological and clinical features including but not limited to myocardial necrosis and pulmonary vasculitis, oedema and haemorrhage presenting as tachypnea, respiratory distress and/or sudden death.^{6,8} Chronic toxicosis may also occur following prolonged ingestion of diets high in selenium, causing a syndrome of hoof abnormalities, weight and hair loss.⁹ An explanation of the quantity of selenium to which the lambs in case 1 and 2 were potentially exposed, relative to the reported toxic doses together with a discussion of the potential risk factors for intoxication follows.

In the first case, intoxication occurred when excess mineral supplement was added to troughs. The liquid supplement had been previously used by this operator with no complications. However, the producer had started using an in-house weaning protocol with a typographical error. The dose of the liquid supplement exceeded the label recommendation (label: lambs <30 kg, 5 ml/head over 3 days; protocol: 10 ml/head for 3 days). The label instructions suggested the total volume each day be added to troughs either once or twice a day. Having used the product previously, the producer elected to add it once daily. Hence, at a rate of 10 ml/head/day, 4.7 L (470 head) was added to the trough in the light draft pen on day one of weaning. A total 7050 mg of Se (1500 mg/L Se in supplement) was added to the 40 L trough, resulting in an initial concentration of 176 mg/L. As water was drawn out of the trough, that concentration would have decreased. However, as Se intoxication in lambs has occurred with oral doses of 1-2.2 mg/kg, or 18-40 mg for an 18 kg lamb, the initial concentration easily exceeded that dose. On a warm day, a lamb may drink 5-10% bodyweight, 0.9 L for 18 kg lamb. On this occasion, 0.9 L would have contained 158 mg selenium, or 8.8 mg/kg for that 18 kg lamb. Hence, early drinkers may have received over four times the toxic dose. In the heavy draft, the total dose of the liquid supplement was divided between two troughs, with 4725 mg selenium added to each and an end concentration of 118 mg/L. At a similar level of water ingestion, the early drinkers in the heavy mob would have ingested over two times the toxic dose. The lower selenium concentration in the water and the heavier lambs may explain the lower mortality in the heavy mob. Selenium administered via the oral route may be more available to young lambs, likely over-represented in the light mob, due to incomplete rumen development.

Following consultation, advice was given to remove access to the supplement. Troughs were dumped and flushed with clean water, and we recommended that no further doses be offered. These recommendations likely reduced total mortalities. Lambs were monitored over the ensuing days for further cases, as published reports suggest cases may present five or more days after exposure, though most cases occur over the initial 72 hours. No further cases occurred after four days.

In addition to the lambs that died peracutely, some had less immediately fatal respiratory and neurological signs. As no specific antidote to selenium intoxication is available, the recommendation was made to manage symptoms and avoid stress, excess exercise and provide fresh water and good roughage. Six of these lambs died after several days. However, those with less severe respiratory signs recovered well. A total of 20 lambs died with signs consistent with selenium intoxication (1.8%). Due to other contemporaneous disease processes, this was an estimate. There are reports of sublethal selenium intoxication resulting in scouring and pneumonia after pulmonary damage. No such signs were seen in this case nor case 2.

With the verbal consent of the producer, the manufacturers of the liquid supplement were notified. The instructions for use of the supplement in lambs has subsequently been modified and now includes a discussion of the risks of adding the supplement to low-volume, high-flow troughs and a recommendation to further split doses to reduce the risk. The case highlights

the importance of considering the dilution effect when providing supplements to livestock via water sources. In this case, lambs were not provided with additional selenium via drench or vaccines at weaning and were running on country with marginal selenium levels. If they had been additionally supplemented or in an environment with high selenium, mortality rates may have been higher still.

In contrast, to case one, no single selenium supplementation product was administered in excess in case 2. However, the cumulative effect of providing three sources of selenium to young, stressed lambs resulted in intoxication sufficient to cause 10.7% mortality in exposed lambs. Importantly, the lambs in case 2 were inappropriate candidates to receive two of the three selenium sources they were administered, being too light for Triguard® and too stressed for Multimin®. The Triguard® label states it is 'contraindicated for use in lambs less than 15 kg bodyweight' and both labels state that the products are not for use when 'selenium is provided by other means'. Each lamb in case 2 received 2 ml of Triguard®, a dose of 0.1 mg/kg selenium for a 10 kg lamb. Whilst lower than other toxicity cases, as above, oral selenium is more available to young lambs due to incomplete rumen development. Multimin® copper-free was provided at 0.2 ml/10 kg, the dose on the label, providing 0.1 mg/kg selenium to 10 kg lambs. The Multimin® label recommends avoiding administration to stressed animals, which marking results in. Vaccination with a clostridial vaccine including selenium provides an additional 0.1 mg/kg selenium for a 10 kg lamb. In total, a 10 kg lamb would have received 0.2 mg/kg injectable selenium and 0.1 mg/kg oral selenium. A 5 kg lamb would have received 0.4 mg/kg injectable, close to reported toxic doses, and 0.2 mg/kg oral selenium. These doses approach but do not breach the reported toxicity thresholds.^{2, 6} However, the use of all but the vaccine is contraindicated in young lambs at marking and the label of each states that use is contraindicated where selenium is provided by other means. Additionally, each product recommends avoiding use in environments where selenium is sufficient and blood selenium levels are high.

Unlike case 1, deaths due to selenium toxicity ceased 48 hours after administration. No chronic effects of toxicity were reported. The producer was advised to avoid using multiple selenium sources contemporaneously in the future, and to adhere to label recommendations for appropriate sheep age and size for treatment. Anecdotally, it is not uncommon for lambs to receive two sources of selenium contemporaneously at weaning, for example a vaccine and a drench both containing selenium. Importantly, most lambs are then older, with more mature rumens, and heavier. Hence the dose of injected selenium received from the vaccine is likely more than half that a lamb receives at marking.

These cases highlight the need for increased awareness of selenium intoxication especially at times when multiple sources may be provided, including around marking and weaning. They serve as a reminder for veterinarians to include selenium intoxication in a list of differentials for acute onset respiratory distress, lethargy, neurological signs and sudden death, especially where a stable foam is noted either on clinical exam or necropsy. In such cases, key diagnostic samples include fresh liver (Se concentration) and liver, lung and heart muscle (histopathology).⁶ However, if sampling potential is limited, fresh liver, the gross post-mortem lesions described and a history of access to or administration of an overdose will suffice. If investigating an antemortem case, serum will suffice. Preventing access to the supplement and ensuring no further supplementation is provided over the ensuing month (for short acting sources), is the best means of preventing subsequent mortalities in the survivors.

Finally, in case one, the cervical abscess, ruminal acidosis and malnutrition cases highlight the importance of conducting a suite of necropsies. This is especially so in scenarios where

multiple disease processes are likely to present contemporaneously including at weaning, in periparturient animals or in feedlots.

Conflict of interest

Neither author has a financial or personal relationship with other people or organisations that could inappropriately influence the content of the paper. There is no conflict to declare.

References

1. Hosking WJ, Caple IW, Halpin CG et al. Selenium. In: Hosking WJ, Caple IW, Halpin CG, et al., editors. *Trace Elements for Pastures and Animals in Victoria*. Victorian Government Printing Office, Department of Agriculture and Rural Affairs, Melbourne, Victoria, Australia, 1986:20-26.
2. Blodgett DJ, Bevill RF. Acute selenium toxicosis in sheep. *Vet Hum Toxicol* 1987;29:233-236.
3. Gabbedy BJ. Toxicity in sheep associated with the prophylactic use of selenium. *Aust Vet J* 1970;46:223-226.
4. Caravaggi C, Clark FL, Jackson ARB. Experimental acute toxicity of orally administered sodium selenite in lambs. *Research In Veterinary Science* 1970;11:146-149.
5. Lambourne DA, Mason RW. Mortality in lambs following overdosing with sodium selenite. *Aust Vet J* 1969;45:208.
6. Tiwary AK, Stegelmeier BL, Panter KE, James LF, Hall JO. Comparative toxicosis of sodium selenite and selenomethionine in lambs. *Journal of veterinary diagnostic investigation* 2006;18:61-70.
7. McKenzie CM, Al-Dissi AN. Accidental selenium toxicosis in lambs. *Can Vet J* 2017;58:1110-1112.
8. Glenn MW, Jensen R, Griner LA. Sodium selenate toxicosis: pathology and pathogenesis of sodium selenate toxicosis in sheep. *Am J Vet Res* 1964;25:1486-1494.
9. Yaeger MJ, Regg DN, Larry H et al. The Effect of Subclinical Selenium Toxicosis on Pregnant Beef Cattle. *Journal of veterinary diagnostic investigation* 1998;10:273-268.

Post-mortem findings from periparturient non-Merino ewes across southern Australia

Elsa Glanville¹, David McGill¹, Mary McQuillan², Caroline Jacobson³,
Leanne Sherriff⁴, Andrew Whale²

¹Mackinnon Project, ²Livestock Logic, ³Murdoch University, ⁴Pinion Advisory
¹Faculty of Veterinary & Agricultural Sciences Werribee VIC, ²Portland Rd, Hamilton VIC

Introduction

Mortality risk increases over the periparturient period for ewes and their progeny.¹ Periparturient ewe mortality results in substantial economic and welfare costs and managing ewe mortality is a challenge for producers.^{2,3} Decreasing ewe mortality would both decrease these costs and increase perinatal lamb survival.⁴ However, reducing ewe mortality requires understanding causes of death, and the associated risk factors. Hence 51 non-Merino producers across southern Australia enrolled in a Meat and Livestock Australia supported study exploring periparturient mortality using both producer records⁵ and post-mortem (PM) investigation.

Methods

Study design, location and PM methodology

Over two years (2019-2020), 595 deceased ewes from participating farms located in Victoria, New South Wales, South Australia and Western Australia were submitted to project veterinarians for gross PM (Figure 1). On average, eight PMs were conducted per participating farm (range: 1-32), 18% of all recorded mortalities in 2020 and 17% in 2019.

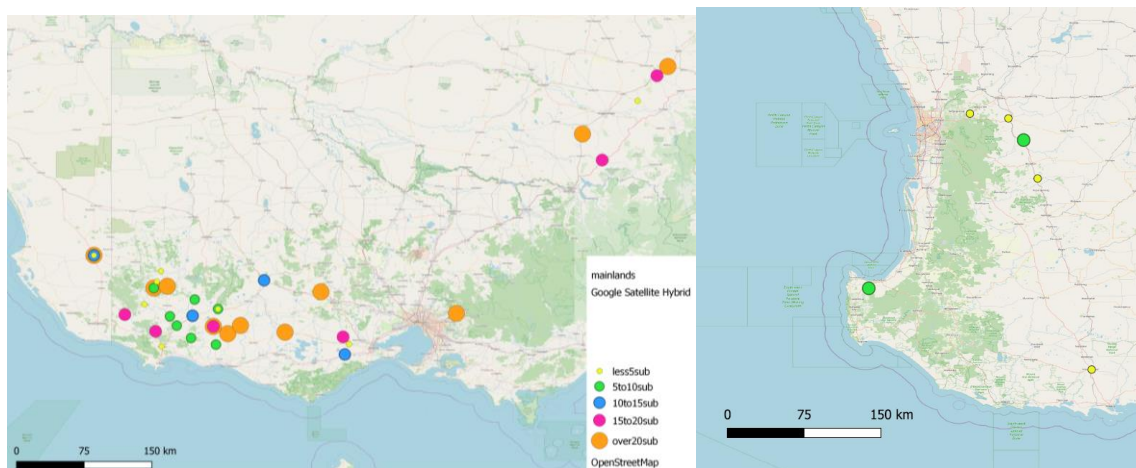


Figure 1 Location of properties that submitted ewes for post-mortem over two years (2019-2020) in South Australia, Victoria and New South Wales (left) and Western Australia (right). The number of submissions from each property is signified by circle size (QGIS).

A standard history was taken for each submitted ewe, and a project specific PM protocol was followed.⁶ The history and PM observations were recorded in a custom-built app within an online platform (CommCare®, Dimagi, Inc. 2022). Observations were recorded according to each major body system, with veterinarians recording if the findings were grossly normal or abnormal, and if the latter, a description was requested. The app was designed to facilitate streamlined data collection across the widely distributed project veterinarians, and interrogation of cases after the event. Aqueous humour was sampled when possible for calcium, magnesium and beta-hydroxybutyrate analysis. Faeces were sampled, if present, for

individual worm egg counts (WEC). Additional samples were submitted from a subset of cases that were investigated under Victoria's Significant Disease Investigation program and/or the national Transmissible Spongiform Encephalopathy (TSE) surveillance program. The most likely diagnosis recorded at the time of gross PM was checked against laboratory results and adjusted if necessary.

Statistical analysis

Causes of death are presented as the mean proportion of submissions diagnosed with each condition by farm (mean \pm standard error of the mean, SEM) in both years of the project (year 1: 2019, year 2: 2020). Multiple diagnoses were possible per case, so the total count for diagnoses exceeds 1.0 per farm.

Features of the submitted population including age, condition score, litter size and BCS range at death are reported using the mean and SEM (where appropriate).

Logistic regression was used to analyse the relationship between the probability of four common diagnoses and risk factors related to the ewe (age, condition score at death, litter size, litter weight), environment (rainfall zone, state) and management (pasture type and feed on offer estimates) accounting for farm of origin. Where an odds ratio was found to be significant, the interpretation is that the odds of that diagnosis are either increased or decreased for the deceased ewes exposed to that risk factor compared to ewes in the comparator groups (*i.e.* deceased ewes not exposed to the risk factor).

Odds ratios were expressed relative to a reference group for body condition score (BCS 3.0), age (3 years of age), litter size (twins), estimated feed on offer (1500 kg DM/ha) and rainfall zone (medium). BCS reference group was based on Lifetime Ewe targets. Other reference categories were based on the most common response.

Rainfall zone was derived from the mean annual rainfall (mm) recorded at the nearest Bureau of Meteorology station.⁷ Each submitted ewe was allocated to either the low (<575 mm annual), medium (575-700 mm) or high (>700 mm) zone based on her farm of origin.

Categories were formed to reduce the number of groups with low numbers. Age was analysed by year from 1-5 years old and then all >5 years old. Body condition score (BCS) at death was recorded to the nearest quarter. All ewes less than 2 were grouped into one category ('2') and all >3.75 fell into '3.75'. Age and BCS were also analysed as young (<5) versus old (\geq 5) and mid (2.5-3.5) versus low (\leq 2.25) and high (\geq 3.75) BCS. Estimated feed on offer (FOO, kg DM/Ha) in the paddock where the ewe died was rounded to the nearest 500 and reduced to four groups, \leq 1000, 1500, 2000 and \geq 2500 kg DM/Ha. Pasture type was categorized into seven groups (improved, annual, native, Lucerne, Lucerne-mix, improved-mixed and crop). Total litter weight fell into \leq 5, 5-10, 10-15 and >15 kg.

Results and Discussion

Cause of death – overall findings

Periparturient trauma and/or septicaemia and dystocia were the most common causes of death (Figure 2). Hypocalcemia was the third most commonly diagnosed cause of death and was the predominant cause on some farms. Dorsal vaginal wall rupture, uterine and vaginal prolapse were important causes of death on some farms in some years. This likely reflects annual and farm variation in expression associated with disease specific risk factors.⁸⁻¹¹ Pregnancy toxemia was diagnosed relatively infrequently.¹² The timing of most submissions,

after lambing start date, may have resulted in some pregnancy toxemia deaths being missed.

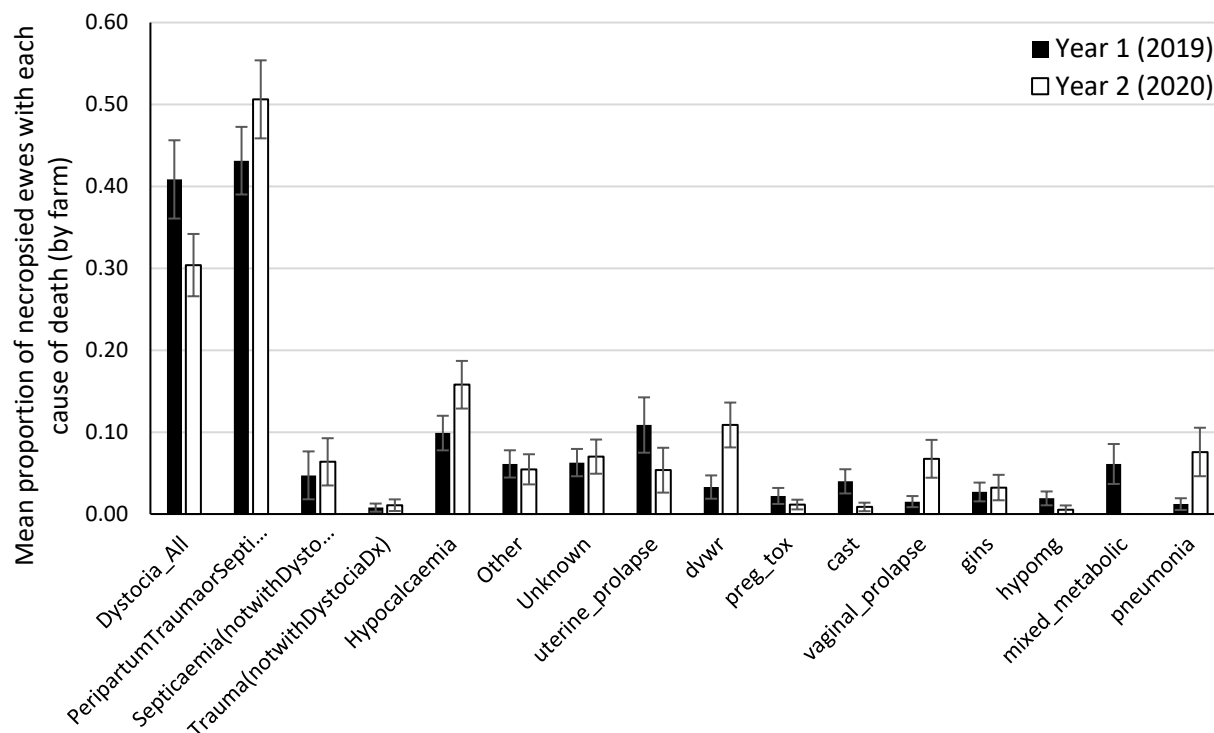


Figure 2 Mean proportion of necropsied ewes diagnosed with each cause of death by farm (\pm SEM). Peripartum trauma or septicaemia includes all causes directly related to late pregnancy or parturition. 'Septicaemia' and 'trauma' does not include any case directly related to late pregnancy or parturition.

Pregnancy status, litter size, BCS and age of submitted ewes

Sixty-four percent of submitted ewes were pregnant at time of death. Of those ewes, 47% had lamb/s engaged in the birth canal, implying death occurred during stage 2 of parturition.

Litter size determined at mid-gestation transabdominal ultrasound scanning or at PM examination was recorded for 404 of the ewes submitted for PM. Sixty-one percent of PM cases were twin-bearing, and 15% triplet-bearing. Litter size was not recorded for the remainder (mob of origin not reported and death occurred post-partum or ewe was not scanned and death occurred post-partum, so no fetuses were present to inform litter size). More multiple (twin or more, 58%) than single (31%) pregnancies were reported on participating properties. However, multiple-bearing ewes were over-represented in the PM sample, most likely due to higher cumulative mortality of multiple-bearing ewes during lambing.⁵

Most ewes submitted were in BCS 2.75 to 3.25 at death. The proportion of ewes that fell into the lower and higher BCS categories differed between years. In 2019, 25% of ewes were <2.5 compared to only 11% in 2020. In contrast, 14% of cases in 2019 and 27% in 2020 were in BCS 3.5 or greater. This likely reflects the tighter season in 2019 compared to 2020 across many of the participating properties. Across both years of the project, ewes submitted from NSW were in significantly higher BCS (mean: 3.43, SEM: 0.03) compared to those submitted from VIC (2.95, 0.02), SA (2.99, 0.10) and WA (2.82, 0.11; $P < 0.01$).

Over both years, 54% of the ewes submitted for PM were five years or older. The proportion of ewes submitted in the older age cohorts likely reflects the increased risk of mortality within these cohorts.^{5, 13}

Cause of death - dystocia

Of the 227 cases of dystocia diagnosed at PM across both years, only 56% were reported as 'obvious dystocia' based on history or external evidence recorded by the attending veterinarian, including protruding head/s or limb/s. The remaining cases were identified during the PM process. Hence dystocia is likely under-reported in the absence of internal examination or PM.

Dystocia was recorded as the cause of death in most cases that died in stage 2 parturition. Those that died in stage 2 but were not recorded as dystocia had hypocalcaemia and significant trauma recorded as cause of death. Some of these cases may have suffered dystocia without it being recorded as the primary cause of death due to the presence of other fatal disease processes. Differences in allocation of cause of death between attending veterinarians is an acknowledged limitation.

In submitted ewes, single-bearing ewes were more likely to be diagnosed with fatal dystocia than twin-bearing ewes. There was no difference in the likelihood of a dystocia diagnosis across the different age groups submitted. Ewes in BCS 2.5 or less at death were less likely to have a dystocia diagnosis than ewes in BCS 3.

The most common type of dystocia recorded was malpresentation (57%), followed by foetal size (14%) and uterine inertia with failure to progress for unknown reason (13%). Across all litter sizes, the most common malpresentations were leg/s back (34%), breech (tail or hindlimbs first, 27%), two lambs simultaneously (18%) and head back (14%). When litter size was considered, ewes that died carrying triplets (35%; OR 9.47, 2.52 - 35.55) and singles (17%; OR 3.13, 0.9 - 10.85) lambs had higher odds of 'head-back' malpresentations than twins (5%). In the ewes submitted with malpresentations, those carrying twins had higher odds of leg/s back malpresentations (41%) than triplets (9%; OR 6.08, 1.19 - 31.01).

Sixty-two percent of all dystocia cases had evidence of substantial periparturient trauma and/or septicaemia. The odds of trauma and/or septicaemia was increased by a factor of 2.59 in ewes diagnosed with dystocia (95% CI 1.84 to 3.64). This is discussed below.

Dystocia is not necessarily a fatal condition, only a proportion of ewes with dystocia succumb. The breakdown of dystocia types here only describes the proportions in fatal dystocias. Additionally, dystocia type was predominantly recorded for ewes that died during stage 1 or 2 of parturition, where the foetus/es were observed by the attending veterinarian. Hence the proportion of each type of dystocia in surviving ewes may differ. However, these results emphasise the contribution malpresentations make to dystocia deaths and are consistent with findings reported in a recent dystocia review.¹⁴

Cause of death – periparturient trauma and/or septicaemia

Over both years, 49% of all ewes submitted had evidence of considerable trauma and/or septicaemia directly related to late pregnancy or birth (periparturient trauma and/or septicaemia, PTS). Cases were submitted with a PTS diagnosis from 33 of 35 participating flocks in 2019 and 33 of 38 in 2020. Up to 100% of cases submitted were diagnosed with PTS as a contributing cause of death. The most likely origin of septicaemia in cases diagnosed with PTS was metritis and peritonitis while the major traumas recorded were uterine and/or bladder rupture (reported previously for these results).⁵ Interestingly, traumas and injury to the genitourinary tract are reported to be largely the consequence of forceful intervention in parturition.¹⁵ However, no such assistance was rendered to the majority of the

ewes submitted to this project. Hence the observed trauma was likely a sequelae to disordered parturition.

Accounting for farm of origin, the odds of a PTS diagnosis were lower in ewes submitted from WA properties than those from VIC but did not differ between any other states and VIC. There was a trend toward an increased odds of PTS diagnosis in ewes submitted with singleton pregnancies compared to those carrying twins. This may be associated with the previously described increased likelihood of a dystocia diagnosis in the single-bearing ewes submitted. Ewes in the low BCS category (<2.5) at death had significantly lower odds of a diagnosis of PTS (OR 0.45) than those in the middle BCS category (2.5-3.5). Again, the lower likelihood of a dystocia diagnosis in ewes submitted in the low BCS category may explain this finding.

Within cases diagnosed with PTS, dystocia was more likely to be diagnosed in those that were pregnant at death (85%) than those that were not (38%, OR 9.61, 95% CI 5.42 - 17.02). For some of the cases that were not pregnant at death, the traumatic consequences of dystocia are observed at PM and inform the cause of death. However, the lamb that caused the dystocia was birthed by the ewe prior to death and the ewe succumbs post-mortem from the related trauma. Hence the root cause of the PTS was dystocia, but without the presence of the foetus in utero or in the birth canal, the attending veterinarian may have insufficient information to record dystocia as a cause of death.

Cause of death – hypocalcaemia

Sixteen percent of all ewes submitted had a diagnosis of hypocalcaemia. The mean proportion of cases per farm diagnosed with hypocalcaemia was 0.10 ± 0.02 in 2019 and 0.16 ± 0.03 in 2020. Hypocalcaemia diagnoses were unevenly distributed across participating farms. No cases were recorded in the ewes submitted from 51% of participating farms in 2019, and 39% of farms in 2020. On farms with cases, the proportion of submitted ewes confirmed with hypocalcaemia as a cause of death was as high as 75%.

Hypocalcaemia was less likely to be diagnosed in ewes submitted from medium rainfall zones of NSW compared to medium rainfall zones in VIC, accounting for farm of origin. Considering state alone, submissions from NSW were less likely to receive a hypocalcaemia diagnosis as cause of death than those from VIC. The higher BCS of ewes submitted from NSW compared to VIC likely explains this finding, as ewes in lower BCS at death (<2.5) were more likely to be diagnosed with hypocalcaemia than those in mid-BCS at death (2.5 - 3.5). Ewes submitted with singleton pregnancies were less likely to be diagnosed with hypocalcaemia as a cause of death than those submitted with twin pregnancies ($P < 0.05$). Finally, older ewes were more likely to be diagnosed with fatal hypocalcaemia than younger ewes, flagging a target population in which management could be altered to reduce the cases of fatal hypocalcaemia.

Cause of death – dorsal vaginal wall rupture

As for hypocalcaemia, dorsal vaginal wall rupture (DVWR) was the predominant cause of death in ewes submitted from some flocks. However, most farms had very low or no cases of DVWR.

Accounting for farm of origin, ewes submitted from NSW medium rainfall zones had a significantly higher likelihood of a DVWR diagnosis than those from VIC medium rainfall zones ($P < 0.005$). Odds of diagnosing DVWR was higher in cases from NSW than those from VIC, considering farm of origin (OR 6.44, $P < 0.005$). High BCS ewes at death (>3.5) increased the odds of a DVWR diagnosis compared to those submitted in mid-BCS (OR 2.64, $P < 0.05$).

The state of origin risk factor is likely explained by the increased diagnoses of DVWR in high BCS ewes, and the fact that ewes from NSW were in higher BCS than those from VIC. Additionally, ewes submitted pregnant with >10 kg total foetal weight had higher odds of a DVWR diagnosis than ewes that died with 5 - 10 kg total foetal weight. Single bearing ewes had lower odds of a DVWR diagnosis than twin-bearing ewes submitted (OR 0.07, P < 0.05). Ewes grazing \leq 1000 kg DM/ha at death had a lower likelihood of a DVWR diagnosis than ewes grazing 1500 kg DM/ha FOO (P < 0.05).

These findings contribute to the understanding of DVWR, a spontaneous rupture of the dorsal vaginal wall with evisceration of abdominal organs that is fatal. The condition is one of high BCS ewes carrying multiple fetuses of high total foetal weight.¹⁶ Minimising the range of BCS, and the proportion of the mob >3.75, is suggested as a strategy to reduce the risk of a number of fatal diagnoses, including DVWR. This may be especially pertinent following droughts, when properties may have lower stocking rate relative to pasture availability (feed on offer).

Key findings for veterinarians and consultants

The most commonly diagnosed causes of death in periparturient non-Merino ewes across Southern Australia were dystocia and periparturient trauma and/or septicaemia. Genitourinary tract tears and organ rupture, catastrophic haemorrhage, metritis and peritonitis were common findings in the latter. They are likely sequelae to dystocia. These causes of death were found in ewes from most farms, in up to 100% of ewes submitted. Important implications of these results for veterinarians and producers involved in acute obstetrical management of ewes include consideration of antibiotics and anti-inflammatories, extension of appropriate obstetric intervention and frequency of surveillance.

The cases presented represent only ewes with fatal obstetric complications. A substantial proportion likely survive dystocia. Survivors may have ongoing production and welfare consequences stemming from the PTS sequelae to dystocia. Potential production implications of non-fatal dystocia and PTS could include decreased milk yield hence reduced lamb growth, increased lamb mortality and subsequent reproductive failure.

Caution is advised in interpreting the risk factors described, as they describe exposures and risk only in dead ewes. Exposure to the factors reported as significant either increased or decreased the likelihood of a diagnosis in dead ewes compared to those that were not exposed. The identified risk factors highlight some management strategies that could be adjusted in ewes more likely to have that diagnosis as a cause of death. Hence our findings support modified management for older non-Merino ewes (> 5 yo) including increased surveillance, careful attention to calcium requirements and minimizing time off feed in late gestation. For older ewes, increased surveillance and management in separate mobs to age groups with lower risk may result in earlier detection of hypocalcaemia and modification of management to decrease the magnitude of loss. Strategies to reduce DVWR cases include early identification of high BCS ewes (>3.5) and careful nutritional management to an appropriate BCS of both high and low BCS ewes, especially in multiple bearing ewes.

Half the dystocia cases required PM for diagnosis, suggesting that mortalities attributable to dystocia are likely to be underestimated by producers. Furthermore, peripartum trauma and/or septicaemia were frequent diagnoses and also only evident at PM. This emphasizes the importance of PMs in understanding mortality and informing targeted strategies to reduce the magnitude of ewe mortality. This presents an opportunity for veterinary engagement with producers, especially those concerned about ewe morbidity and mortality.

A recommended starting point based on this study is a case series of ewe PMs over the periparturient period, with the goal of providing property specific descriptions of key causes of death. This could readily be combined with lamb necropsies to address overall reproductive inefficiency. An explanation of the disease processes underpinning losses, together with a bespoke treatment and management plan addressing those diseases would help reduce subsequent ewe mortality. Extending this information offers short term solutions to decrease periparturient morbidity and mortality. Over the longer term, this information is critical to ram selection, ewe and lambing management decisions and would inform longer term property specific plans to reduce susceptibility to the relevant causes of death.

Conflict of Interest

The authors of this paper do not have financial or personal relationships with people or organisations that could inappropriately influence the content of the paper. The funding body (Meat and Livestock Australia) approved the manuscript for publication but was not involved in the collection, analysis or interpretation of data, or manuscript writing.

References

1. Pfeiffer CN. Improving disease surveillance in Australia's sheep industries: investigations of syndromic surveillance, farmer behaviour and sheep trade networks. 2019.
2. Doyle R. Assessing and addressing on-farm sheep welfare. *Meat & Livestock Australia*, 2018:210.
3. Scott PR. The management and welfare of some common ovine obstetrical problems in the United Kingdom. *The Veterinary Journal* 2005;170:33-40.
4. Bruce M, Young JM, Masters DG et al. The impact of lamb and ewe mortality associated with dystocia on Australian and New Zealand sheep farms: A systematic review, meta-analysis and bio-economic model. *Preventive veterinary medicine* 2021;196.
5. McQuillan M, Glanville EJ, Jacobson C, Sherriff L, Whale A. Unlocking the keys to ewe survival. *Wagga Wagga*, 2021.
6. Jubb T, Perkins N. *Veterinary Handbook*. Meat and Livestock Australia and Australian Livestock Export Corporation Ltd. 2009.
7. Anonymous. Bureau of Meteorology Climate Data. <http://www.bom.gov.au/climate/data/>. 2017. Retrieved 01-March 2017.
8. Hansen AT, Plant JW. Tearing of the vaginal wall with intestinal prolapse in pregnant ewes. *Aust Vet J* 1980;56:510.
9. Knottenbelt DC. Vaginal rupture associated with herniation of the abdominal viscera in pregnant ewes. *Vet Rec* 1988;122:453-456.
10. Knottenbelt DC. Cervico-vaginal prolapse in the preparturient ewe : an epidemiological and clinical study. University of Edinburgh, 1990.
11. Hosie BD. Vaginal prolapse and rupture in sheep. *In Practice* 1989;11:215-218.
12. Suter R. Causes of periparturient ewe deaths 2009-2010. *Ewe & Lamb mortality survey*. Department of Primary Industry VIC, 2011.
13. Turner H, Dolling C, Sheaffe P. Vital statistics for an experimental flock of Merino sheep I. Death rates in adult sheep, in relation to method of selection, age and sex. *Australian Journal of Agricultural Research* 1959;10:581-590.
14. Jacobson C, Bruce M, Kenyon PR et al. A review of dystocia in sheep. *Small Ruminant Res* 2020;192:106209.
15. Mavrogianni VS, Brozos C. Reflections on the causes and the diagnosis of periparturient losses of ewes. *Small Ruminant Res* 2008;76:77-82.
16. Watt B, Denman A. Spontaneous vaginal rupture in multiparous late pregnant first cross ewes. *Flock & Herd Case Notes*. Local Land Services New South Wales, 2007.

***Toxoplasma gondii* exposure and associated risk factors in Victorian ewes**

Elsa J Glanville¹, JWA Larsen and Angus JD Campbell²

¹Mackinnon Project University of Melbourne, ²Melbourne School of Population and Global Health

¹Faculty of Veterinary & Agricultural Sciences Werribee VIC, ²University of Melbourne, Parkville VIC

Summary

Sheep are especially susceptible to the protozoan parasite *Toxoplasma gondii*. Infection of a naïve pregnant ewe can result in reproductive loss, and it is the third most commonly diagnosed cause of infectious ovine abortion in Australia. Additionally, undercooked meat from infected sheep is a source of human toxoplasmosis. The prevalence of *T. gondii* infection in Victorian ewes is not known, and the risk factors for infection are not described. Our aim was to determine the prevalence of antibodies to *T. gondii* in self-replacing ewe flocks across Victoria and describe associated risk factors. A commercial antibody-ELISA was used on serum samples from 50 mature ewes from 50 self-replacing flocks across Victoria ($n = 2500$). A questionnaire from each flock informed risk analyses. At a flock level, 78% of farms had at least one positive ewe. Within-flock prevalence was not normally distributed, as only 14% of farms had >25% seropositive ewes. Low-level exposure was common across Victorian ewes, meaning many flocks are relatively naïve. Hence for most flocks, there is a risk of a toxoplasmosis abortion if ewes are exposed to oocysts during pregnancy. Few definitive risks for 'positive' flock or individual status were identified in early risk factor analyses.

Introduction

Toxoplasma gondii is a common protozoan parasite with a complex lifecycle involving both definitive and intermediate hosts. Felids are the definitive host, and can shed millions of infective oocysts in their faeces.^{1,2} All other endotherms are potential intermediate hosts but sheep are particularly susceptible, and *T. gondii* cysts are more common in sheep meat than beef.³

Toxoplasmosis is both a potentially important cause of ovine reproductive loss and a significant zoonosis.^{4,5} It is the third most commonly diagnosed cause of ovine abortions in Australia.^{6,7} However, it is relatively infrequently diagnosed compared to *Campylobacter* spp. and *Listeria* spp.⁸ The consequence of infection in the ewe is greatest when naïve ewes are infected during pregnancy, with the outcome depending on gestation at infection. Importantly, early to mid-gestation infection results in foetal death followed by resorption, mummification or abortion.⁹

Toxoplasmosis of sheep is a zoonotic concern because raw and undercooked sheep meat is a route of human exposure. Human infections are most pathogenic when infection occurs during pregnancy, or in the immunocompromised.¹⁰ Internationally, it is recognized as an important foodborne pathogen warranting intervention.¹¹ Abattoir and mince-meat surveys suggest Australian sheep meat is commonly contaminated with *T. gondii* cysts.^{12, 13} Toxoplasmosis is not a notifiable disease in Australia with the public health response emphasizing education to decrease risk of exposure in those at risk of disease.

Infection of sheep occurs through the consumption of feed or water contaminated by oocysts shed by infected felids.³ Oocysts can remain infective in the environment for years,⁵ and a dose of just 200 is sufficient to cause congenital disease in a naïve, pregnant ewe.¹⁴ Once ingested, sporozoites form into tachyzoites, which replicate rapidly within host cells before emerging to damage tissue and stimulate a strong inflammatory response.¹⁵ The immune response stimulates tachyzoites to transform into bradyzoites, which replicate slowly within

cysts in the brain or muscle. The tissue cysts persist indefinitely and are the infective stage for any potential host that ingests the tissue.¹⁶ IgG antibodies are persistently increased following infection, facilitating serological prevalence studies and the use of seropositivity as a proxy for historic exposure and infection.¹⁷

Worldwide, a considerable body of evidence demonstrates high prevalence of *T. gondii* seropositivity in sheep.¹⁸ Reported risk factors include definitive host (cat) factors, sheep density, climate, altitude and water source.^{4, 18, 19} However, there is a lack of consensus between studies in the extent and direction of risk associated with some factors.¹¹ Historically, 9% mean prevalence was reported across NSW rams (41% flocks positive), 61.7% overall prevalence in sheep from Tasmanian abattoirs, and 7.4-25.2% prevalence in South Australian sheep.²⁰⁻²² Some risk factors are described in these studies. However, they are over 30 years old and the dynamics of the sheep flock, sheep management and cat populations have since changed. More recently, 57% to 97% of flocks were detected positive.²³⁻²⁵ The current prevalence in Victoria is unknown.

A vaccine that protects ewes against toxoplasmosis abortion is available.²⁶ Vaccination both increases lamb marking rates, where infection is causing reproductive disease, and decreases the presence of infective cysts in meat with potential public health benefits. The vaccine is not currently available in Australia. Extrapolating the risk of toxoplasmosis or benefits of vaccination to the Australian sheep flock ignores potentially important differences in exposure due to differences in production systems, climate and cat populations, all of which potentially influence prevalence. Australian studies of risk factors for exposure to *T. gondii* in sheep are inconclusive and there is minimal current evidence regarding the situation in Victoria, the largest lamb producing state in Australia.^{21, 22} Hence our aim was to determine the prevalence of seropositivity in mature ewes across Victoria, and to explore risk factors for flock-, within-flock and individual-exposure to *T. gondii*.

Method

Summary

A multi-stage cross-sectional cohort study was used to determine exposure to *T. gondii* in mature ewes (≥ 3 years) from self-replacing flocks in Victoria, Australia. Only ewes reared on the property were included, to ensure serological status reflected infection that occurred on that property. This allowed farm factors to be paired with the serological status of ewes.

Sample size and participating farms

The study sample size of 50 flocks and 50 ewes/flock ($n = 2500$) was calculated assuming: a within-flock prevalence of 15%; 25% of flocks with at least one positive ewe; and an intra-cluster (i.e., farm) correlation coefficient (ICC) of 0.2.^{27, 28} This sample size estimates within-flock prevalence with a precision of $\pm 12\%$ points.

Flocks were selected from across Victoria (Figure 1) from different Natural Resource Management regions (NRMs) weighted according to the proportion of the total Victorian sheep flock present in each NRM. Within areas, convenience sampling was used. There are ten NRMs across Victoria. Two, Glenelg Hopkins and Corangamite (south-west region) fall within the same climate (Zone 6) and agroecological zone (Agroecological Landscapes of Victoria, DPI 2008) and were grouped together for this study.

Serology

Mature ewes on each farm in the study were randomly selected and a single sample of jugular venous blood was collected (minimum 5 mL). Sera were separated via centrifugation within 48 hours of sampling, removed from the vacutainers using sterile pipettes, and stored in Eppendorf tubes at -20°C prior to submission to an external NATA-accredited commercial laboratory (ACE Laboratory Services, Benalla, Australia) where *T. gondii* antibody levels were

measured using a commercially available Enzyme Linked Immunosorbent Assay (ELISA; Chekit Toxotest, IDEXX).

Toxotest ELISA results are reported as a ratio between sample optical density and positive control optical density (S/P%) together with cut-offs. An S/P% <20% is negative, 20-30% suspect, 30-100% weak positive and >100% positive. Ewes with >30% S/P% were considered 'positive' for *T. gondii* antibodies. 'Suspect' ewes were classified as 'negative' for analyses ($n = 125$ across all flocks).

A flock was characterised as positive if one or more ewes tested positive. A negative flock was one with no test positives. Within-flock apparent prevalence was calculated as the proportion of positive ewes across all ewes sampled per farm. Following the calculation of apparent prevalence, estimated true prevalence was calculated to correct for the imperfections of the serological test. Published sensitivity (91.6%) and specificity (97%) for the IDEXX Toxotest ELISA were used in the calculation [true prevalence = (apparent prevalence + specificity - 1) ÷ (sensitivity + specificity - 1)].^{29, 30}

Questionnaire

A questionnaire was distributed to each flock owner or manager to collect property and flock information and inform risk analysis. Factors associated with increased or decreased prevalence in overseas studies were addressed by this survey, including flock factors (enterprise, age structure, reproductive success and size of flock), cat demographics (domestic and feral, litters); environmental factors (altitude, rainfall, proximity to bush and urban areas) and management factors (feed types, water sources and rodent control).^{18, 19, 31}

Statistical analysis and mapping

Overall flock-level and within-flock estimates of the prevalence of *T. gondii* seropositivity were calculated with 95% confidence intervals (CI). Within-flock seroprevalence was calculated as the mean of estimated true prevalence in each of the 50 flocks (95% CI). The distribution of flocks with negative, low-medium and high within-flock seroprevalence (0%, 1-25% and >25%, respectively) was mapped using QGIS.³²

Pearson's Chi-squared test was used to assess differences in prevalence across NRMs, and within the four NRMs with the most sheep. At a flock level, the relationship between potential risk factors and flock-level status (positive or negative for *T. gondii*) was assessed using logistic regression. The relationship between potential risk factors and the serological status of an individual ewe (positive/negative) was also explored using logistic regression, accounting for the effect of farm. Poisson regression was used to assess the relationship between within-flock prevalence (count of positive and negative ewes) and exposure to potential risk factors.

Farm level factors included rainfall zone (<500, 500-599, 600-699 and ≥700 mm), altitude (m asl), enterprise type (wool, meat and wool/meat), ewe base (Merino/non-Merino), proximity to urban and bush areas (proportion of property bordering each), use of stock containment areas, flock size (<2000, 2000-4000, 4000-10000, >10000), age at first joining (ewe lamb or hogget), mean three year lamb marking rate (lambs marked/ewes joined), presence of cats (house or barn cats), recent litters, rodent activity around feed stores and primary water source. Reproductive concerns (yes/no) and a history of abortions (yes/no) were also recorded.

Statistical analysis was completed in Stata 17.³³

Ethics

This study was approved by the Human (1545893.2) and Animal (1513763) Research Ethics Committees of the University of Melbourne.

Results and discussion

Overall prevalence

Overall, 203 of 2500 ewes were seropositive to *T. gondii* (8.1%, 95% CI 7.1-9.3%). This estimate for Victoria is similar to estimated prevalence's from three recent Australian surveys over different regions. McGregor and Harvey reported a 6.7% overall prevalence in 489 ewes from 10 farms in south-eastern NSW.²⁴ Clune *et al.* estimated an overall prevalence of 8.1% in 558 mature ewes from 28 flocks distributed across Western Australia (WA), South Australia (SA) and New South Wales (NSW).²³ Abattoir surveillance in 401 sheep from across Australia revealed that 11.5% were positive for *T. gondii*.¹⁷ Overall prevalence in South Australia alone was estimated to be higher, at 23.9%.²⁵

Flock-level serological prevalence

The flock-level prevalence (proportion of flocks with ≥ 1 test-positive ewe) in Victoria was 78% (95% CI 64–88%; 39/50). This is consistent with recent serological surveys across other Australian states or regions, with 60% of NSW flocks positive and 57% of 28 flocks from WA, Victoria and SA positive.^{23, 24} In SA, a higher proportion of flocks were positive (97%).²⁵ There was no difference in the relative number of positive flocks across the Victorian NRMs ($P = 0.34$; Table 1). Additionally, there was no difference in the proportion of positive flocks between the four NRMs where most sheep, and hence sample flocks, were located ($P = 0.92$; Figure 1).

Table 1 Flock status and mean within-flock prevalence by Victorian Natural Resource Management region.

| Natural Resource Management region | Total sheep population | Flock sample size | | Mean apparent within-flock prevalence | Proportion of positive flocks |
|------------------------------------|------------------------|-------------------|----------------|---------------------------------------|-------------------------------|
| | | Intended | Actual | | |
| Glenelg Hopkins and Corangamite | 6,233,037 | 21 | 24 | 5% | 0.79 |
| North Central | 2,759,372 | 9 | 8 ^a | 2% | 0.75 |
| Wimmera | 2,432,101 | 8 | 5 ^b | 3% | 0.80 |
| Goulburn Broken | 1,718,030 | 6 | 6 | 19% | 0.83 |
| Mallee | 580,227 | 2 | 2 | 2% | 1.00 |
| West Gippsland | 358,077 | 1 | 2 ^c | 17% | 1.00 |
| North East | 290,973 | 1 | 1 | 0% | 0.00 |
| East Gippsland | 191,095 | 1 | 1 | 0% | 0.00 |
| Port Phillip & Westernport | 110,233 | 0 | 1 ^d | 35% | 1.00 |
| Overall | 14,673,145 | | 50 | 7% | 0.79 |

^aOne flock was located on the border of North Central and Goulburn/Broken and was allocated to NC. ^bOne flock was located on the border of Wimmera and Glenelg, it was allocated to Wimmera. ^cTwo flocks were sampled in West Gippsland to account for different agroecological zones. ^dOne flock was sampled from Port Phillip and Westernport to cover all regions.

Within-flock serological prevalence

The true prevalence within flocks ranged from 0% to 71%. Mean within-flock prevalence was 6.7% (95% CI 2.8% - 10.6%). Nine flocks had only one ewe with a positive titre (>30% S/P). Most farms were negative or had a low within-flock prevalence (<10%). Only seven (14%) flocks had a within-flock prevalence of more than 25% but they were distributed across Victoria (Figure 1). Mean within-flock prevalence was greater in Goulburn Broken than Glenelg Hopkins/Corangamite, North Central and Wimmera but this was not significant ($P = 0.09$; Table 1). The mean within flock prevalence in Victoria is consistent with the within-flock prevalence reported in South-East NSW.²⁴ The uneven distribution of within-flock prevalence is also similar to SE NSW.

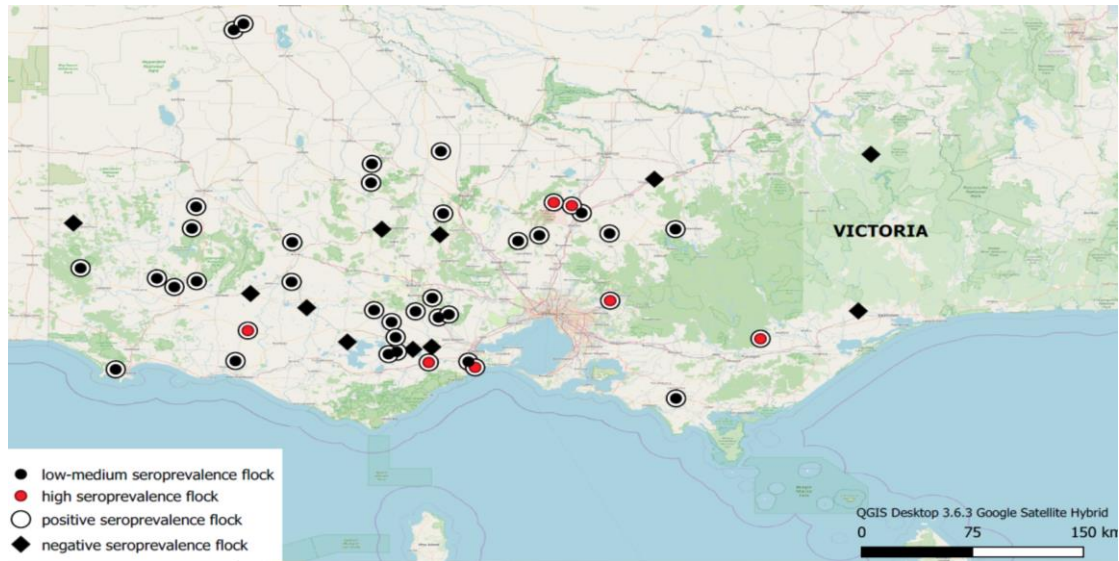


Figure 3 Geographical distribution of the 50 self-replacing flocks sampled, and their status as either negative (0%), low-medium (1-25%) or high (>25%) within-flock prevalence of *T. gondii* antibodies.

Description of flocks sampled and potential risk factors

Forty-six of the 50 flocks returned surveys, 44 of which were complete. Completed responses from farms with incomplete surveys were included in the analysis where possible. The four flocks without surveys were excluded.

Sampled flocks were from farms at an altitude of 10 m above sea level (ASL) to 550 m ASL. There was a statistically significant decrease in the odds of a flock having $\geq 10\%$ positive ewes at higher farm altitude (odds ratio for 50 metre increase in altitude 0.80, 95% CI 0.67–0.97 $P < 0.05$). Mean average 10-year rainfall across participating properties was 577 mm (95% CI 541-614 mm), with a minimum of 329 mm and a maximum of 950 mm. Flocks from properties with 500-599 mm mean annual rainfall tended to have a higher within-flock prevalence than flocks from properties in other rainfall zones (Figure 2). A small sample size in the >700 mm rainfall category limits interpretation.

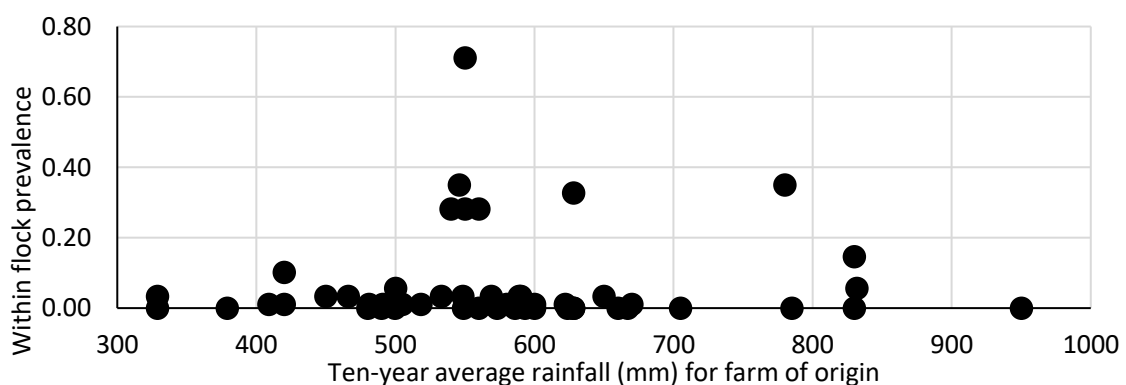


Figure 4 Within-flock prevalence of *T. gondii* seropositive ewes by mean annual rainfall on the farm of origin.

Most flocks (42/46, 91%) had <25% bush bordering the property and only six bordered areas identified as 'urban'. Around half reported using feedlots (45%) and half also ran cattle (57%). Twenty-seven of the 44 properties (61%) had domestic or semi-domestic (barn) cats and 41/44 (93%) reported cat activity of any type, including feral cats. A smaller proportion, 39%, reported litters of kittens. Despite international surveys reporting these factors as risks, in

univariable analyses there were no significant relationships between exposure to these factors and the likelihood of either a flock being positive, an individual ewe being positive (accounting for farm of origin) or the within-flock prevalence.

Across the participating flocks, 38% were wool, 36% meat and 26% mixed (wool/meat) enterprises. No relationship was detected between enterprise type and likelihood of a flock being positive, nor within-flock prevalence. Non-Merino flocks were slightly less likely to be positive than Merino flocks, though this was not significant (OR 0.31, 0.07-1.41; $P = 0.13$). Mean grazing area across all farms was 1406 Ha (998-1813 Ha) with the smallest being 130 Ha and the largest 5945 Ha. Mean total flock size was 6427 (range: 250-26706) and mean total ewes joined was 3597 (range: 250-13300), culled on average at 6.3 years of age (range: 4.5-8). Sixty-seven percent of properties first joined ewes as hoggets, the rest as ewe lambs. Flocks that joined ewes first as hoggets had a significantly greater odds of being positive than those that joined ewes first as ewe-lambs (OR 12.99, 1.72-98.29; $P < 0.05$).

Mean lamb marking rate (lambs marked per ewe joined) over the three years prior to the visit was 1.00 (range: 0.48-1.37). There was no relationship between marking rate and within-flock prevalence of *T. gondii* seropositivity for either Merino or non-Merino ewes (Figure 3).

Only three of the participating flocks had previously had confirmed *T. gondii* abortions (7%), but 50% had a history of abortions and 80% reported concerns of poor reproductive performance. Reproductive performance is notoriously multifactorial, and infectious agents are only one potential cause of poor performance. Additionally, *T. gondii* infection detected via positive serology only indicates exposure occurred at some point in the ewe's life prior to sampling. Toxoplasmosis resulting in reproductive loss can only occur if initial exposure happens to coincide with gestation. Hence the lack of a relationship between lamb marking rates and within-flock prevalence is not surprising. However, it is in contrast to the negative linear relationship reported between lamb marking percent and within-flock *T. gondii* seropositivity in South Australian flocks.²⁵

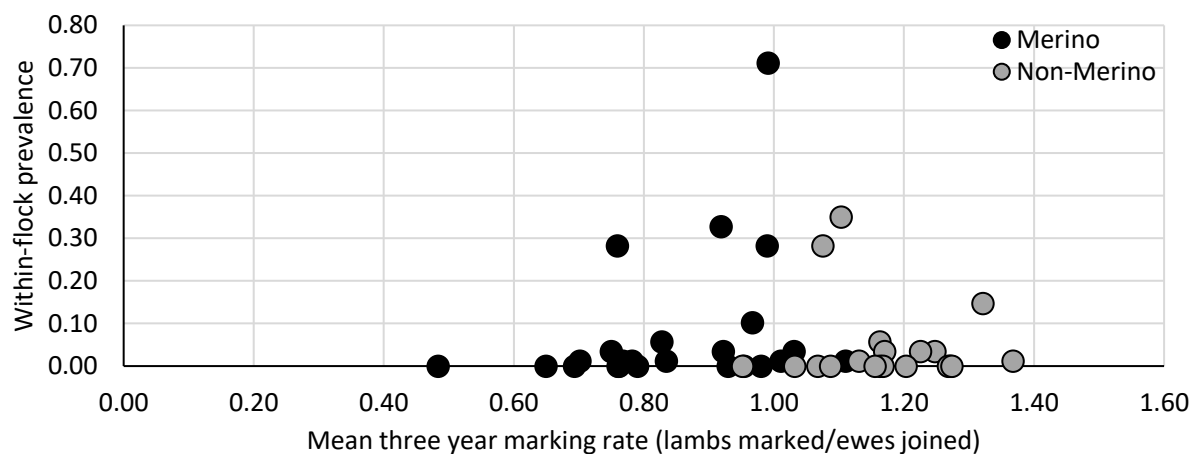


Figure 5 Estimated true prevalence and mean flock lamb marking rate (three years prior to sampling) for flocks with a Merino (black) and non-Merino (grey) ewe base.

Implications of findings for veterinary practitioners

Toxoplasma gondii infection is common at a flock level, but within-flock exposure is highly variable. Most flocks have a low level of exposure, but occasionally flocks have much higher levels of exposure. None of the ewe, cat, management or environmental factors explored resulted in a significant increase in the risk of high within-flock prevalence. Some factors, including age at first mating and altitude, influenced the likelihood of a flock or ewe being positive. For veterinarians investigating reproductive failure in ewes, these results are a reminder to keep *T. gondii* on the list of differentials across all ages of ewes and in the

absence of conventionally accepted risk factors for toxoplasmosis such as the presence of breeding cats on a farm.

The ewes sampled were at least 3 years of age, and 86.9% were naïve. Hence for most mature ewes across Victoria, there remains a risk of toxoplasmosis if sheep are exposed to infective oocysts during pregnancy. The within-flock prevalence results suggest that in Victorian ewes, exposure to *T. gondii* is typically sporadic and exposure is not uniform. If intermittent and unpredictable exposure to *Toxoplasma* also causes intermittent pregnancy losses, farmers might be less likely to notice these and investigate them. However, in the smaller proportion of flocks with higher within-flock prevalence, infection has been more extensive across the flock. This may be the result of more uniform exposure associated with a point source of contamination to which a greater proportion of ewes were exposed, for example supplementary feed or water contaminated with oocysts. It is difficult to identify from this study when exposure occurs in these flocks, but if ewes on these farms are first exposed when they are pregnant, there is a greater risk of a costly abortion storm.

Importantly for practitioners, water sources, pasture or supplementary feed suspected to be contaminated with *T. gondii* may remain infective for months to years. Unsporulated oocysts can survive in the environment for at least three months, whilst sporulated oocysts remain infective in moist soils for up to 18 months.^{34, 35} Oocysts can persist in fresh water for years.³⁶ Hence if a source is identified on high-prevalence farms or in cases of toxoplasmosis abortions, it may be appropriate to advise restricting access by pregnant ewes for the duration of oocyst survival. As most ewes sampled in this study were immunologically naïve to *T. gondii*, any suspected source of *T. gondii* infection on any farm should be avoided by pregnant ewes given the widespread lack of protective immunity in Victoria breeding flocks. Whilst a risk associated with cat presence was not identified, they are the definitive host of *T. gondii* and there is a considerable body of evidence describing infections in ewes after ingestion of feed contaminated with cat faeces.³⁷ It is advisable to prevent access of cats to hay and other supplementary feeds, or avoid feeding these to pregnant ewes.

Finally, expression of toxoplasmosis in ewes is determined by the gestational stage at which trans-placental transmission occurs. The potential range of presentations should be noted by investigating veterinarians. Early gestation infection results in foetal death and resorption, easily mistaken for infertility that could result in fertile ewes being culled. Infection from mid-gestation onwards likely results in abortion, stillbirth or weak lambs that fail to thrive.⁴ Late gestation infection may result in the birth of clinically normal but infected lambs. Multiple bearing ewes may give birth to different sized lambs or one live and one deceased lamb. Gross pathology in aborted fetuses is limited to the well-described changes in cotyledons.³⁸ As placenta recovery rates are relatively low⁸, submitting foetal samples including heart, brain and blood for histology and further testing including immunohistochemistry, PCR and foetal serology, is required to rule out *T. gondii*.⁴

Conflict of Interest

None of the authors have a conflict to declare. Elsa Glanville was supported by a Meat & Livestock Australia residency position for part of this project and part of the funding for the testing component of the project was supported by MSD Animal Health Australia. Neither MLA nor MSD Animal Health had any role in the design, analysis and interpretation of the work.

References

1. Hutchinson WM. Experimental transmission of *Toxoplasma gondii*. *Nature* 1965;206.
2. Frenkel JK, Dubey JP, Miller NL. *Toxoplasma gondii* in cats: faecal stages identified as coccidian oocysts. *Science* 1970;167:893-896.
3. Tenter AM, Heckeroth AR, Weiss LM. *Toxoplasma gondii*: from animals to humans. *International Journal for Parasitology* 2000;30:1217-1258.
4. Dubey JP. Toxoplasmosis in sheep-The last 20 years. *Vet Parasitol* 2009;163:1-14.

5. Dubey JP. *Toxoplasmosis in Humans and Animals*. 2 edn. CRC Press, 2010.
6. Plant JW, Acland HM, Beh KJ. Laboratory findings from ovine abortion and perinatal mortality. *Aust Vet J* 1972;48:558-&.
7. Plant JW, Richards N, Moyle GG. Toxoplasma infection and abortion in sheep associated with feeding of grain contaminated with cat faeces. *Aust Vet J* 1974;50:19-21.
8. Clune T, Beetson S, Besier S et al. Ovine abortion and stillbirth investigations in Australia. *Aust Vet J* 2021;99:72-78.
9. Benavides J, Fernández M, Castaño P et al. Ovine Toxoplasmosis: A New Look at its Pathogenesis. *J Comp Pathol* 2017;157:34-38.
10. Montoya J, Liesenfeld O. Toxoplasmosis. *Lancet* 2004;363:1965-1976.
11. Opsteegh M, Maas M, Schares G, van der Giessen J, consortium obot. Relationship between seroprevalence in the main livestock species and the presence of *Toxoplasma gondii* in meat (GP/EFSA/BIOHAZ/2013/01) An extensive literature review. Final Report. European Food Safety Authority supporting publication, 2016:294.
12. Dawson AC, Ashander LM, Appukuttan B et al. Lamb as a potential source of *Toxoplasma gondii* infection for Australians. *Australian and New Zealand journal of public health* 2020;44:49-52.
13. Kiermeier A, Hamilton D, Smith G, SARDI. National serological baseline survey of *Toxoplasma gondii* in lambs and sheep. *Meat and Livestock Australia Final Report*, North Sydney, NSW 2008:1-28.
14. McColgan C, Buxton D, Blewett DA. Titration of *Toxoplasma gondii* oocysts in non-pregnant sheep and the effects of subsequent challenge during pregnancy. *Vet Rec* 1988;123:467-470.
15. Dubey JP, Lindsay DS, Speer CA. Structures of *Toxoplasma gondii* tachyzoites, bradyzoites, and sporozoites and biology and development of tissue cysts. *Clinical Microbiology Reviews* 1998;11:267-+.
16. Montoya JG, Liesenfeld O. Toxoplasmosis. *Lancet* 2004;363:1965-1976.
17. Hamilton D, Hodgson K, Howard A et al. Investigation of the viability and national serological prevalence of *Toxoplasma gondii* in Australian sheep. South Australian Research & Development Institute, 2021:58.
18. Stelzer S, Basso W, Benavides silvan J et al. *Toxoplasma gondii* infection and toxoplasmosis in farm animals: risk factors and economic impact. *Food and Waterborne Parasitology* 2019:e00037-e00037.
19. Afonso E, Germain E, Poulle M-L et al. Environmental determinants of spatial and temporal variations in the transmission of *Toxoplasma gondii* in its definitive hosts. *International Journal for Parasitology: Parasites and Wildlife* 2013;2:278-285.
20. Munday BL. Prevalence of Toxoplasmosis in Tasmanian meat animals. *Aust Vet J* 1975;51:315-316.
21. Odonoghue PJ, Riley MJ, Clarke JF. Serological survey for *Toxoplasma* infections in sheep. *Aust Vet J* 1987;64:40-45.
22. Plant JW, Freeman P, Saunders E. Serological survey of the prevalence of *Toxoplasma gondii* antibodies in rams in sheep flocks in New South Wales. *Aust Vet J* 1982;59:87-89.
23. Clune T, Lockwood A, Hancock S et al. *Toxoplasma gondii* is not an important contributor to poor reproductive performance of primiparous ewes from southern Australia: a prospective cohort study. *BMC Veterinary Research* 2022;18:1-10.
24. McGregor H, Harvey R. A pilot survey of the prevalence of serum antibodies against *Toxoplasma gondii* and risk factors for transmission of *T. gondii* to sheep in the Tumbarumba shire of New South Wales. *Flock and Herd Case Notes* 2011;March.
25. Lanyon SR, O'Handley RM. Relationship between *Toxoplasma gondii* seroprevalence and lamb marking in South Australian sheep flocks. *Aust Vet J* 2020;98:525-528.
26. Buxton D, Innes EJP. A commercial vaccine for ovine toxoplasmosis. *Parasitology* 1995;110:S11-S16.
27. Otte M, Gumm I. Intra-cluster correlation coefficients of 20 infections calculated from the results of cluster-sample surveys. *Preventive veterinary medicine* 1997;31:147-150.
28. Cenci-Goga BT, Ciampelli A, Sechi P et al. Seroprevalence and risk factors for *Toxoplasma gondii* in sheep in Grosseto district, Tuscany, Italy. *BMC veterinary research* 2013;9:25.
29. Opsteegh M, Teunis P, Mensink M et al. Evaluation of ELISA test characteristics and estimation of *Toxoplasma gondii* seroprevalence in Dutch sheep using mixture models. *Preventive Veterinary Medicine* 2010;96:232-240.
30. Rogan WJ, Gladen B. Estimating prevalence from the results of a screening test. *American Journal of Epidemiology* 1978;107:71-76.

31. Alvarado-Esquivel C, Silva-Aguilar D, Villena I, Dubey JP. Seroprevalence and correlates of *Toxoplasma gondii* infection in domestic sheep in Michoacán State, Mexico. *Preventive veterinary medicine* 2013;112:433-437.
32. QGIS.org. QGIS Geographic Information System. <http://www.qgis.org>. 2022.
33. StataCorp. Stata Statistical Software: Release 15. StataCorp LLC., College Station, Texas, 2017.
34. Lindsay DS, Blagburn BL, Dubey JP. Survival of nonsporulated *Toxoplasma gondii* oocysts under refrigerator conditions. *Vet Parasitol* 2002;103:309-313.
35. Frenkel JK, Ruiz A, Chinchilla M. Soil survival of *Toxoplasma* oocysts in Kansas and Costa Rica. *American Journal of Tropical Medicine and Hygiene* 1975;24:439-443.
36. Lindsay DR, Dubey JP. Long-term survival of *Toxoplasma gondii* sporulated oocysts in seawater. *Journal of Parasitology* 2009;95:1019-1020.
37. Innes EA, Bartley PM, Buxton D, Katzer F. Ovine toxoplasmosis. *Parasitology* 2009;139:1887-1894.
38. Buxton D. Ovine toxoplasmosis: a review. *Journal of the Royal Society of Medicine* 1990;83:509-511.

Pre-joining ram exams: a few tricks and an update of MLP ram mating success project

T Gole

For Flocks Sake Pty Ltd, Dubbo, NSW

With a booming sheep industry and rising ram prices there has been renewed interest in what drives ram success and failure. Utilising sheep handling equipment and electronic identification (EID) allows the private practitioner to conduct pre-joining ram exams including semen evaluation in a professional, safe and efficient manner. An update from the research findings of the Merino Lifetime productivity program at the Trangie research station will be given.

Pre-joining ram exams were conducted based on the industry standard 4 Ts, age and body condition score, and semen was collected with electro ejaculation. Semen was assessed as progressively motile and semen morphology was undertaken at independent labs. The Tricks and tips are a system in commercial use by For Flocks Sake Pty Ltd

Although sample sizes are small there is an indication that semen morphology has value as a guide of ram success and further research is warranted.

There is a unique opportunity for sheep vets to serve sheep producers at a higher level. This authors opinion is that pre-joining ram exams is the cornerstone of that opportunity.

What drench advice should I provide my clients? Anthelmintic resistance update: Results of 50 FECRTs conducted across Australia from July 2018 – March 2021

Kelly Graham
Associate Director – Livestock Veterinary Operations, Zoetis Australia
Rhodes NSW

Introduction:

In 2015 MLA estimated that internal parasites cost the Australian sheep industry \$435.9M annually¹.

Drench selection decisions are hugely important as drench resistance is common throughout Australia. Ineffective drenching due to using a product with efficacy less than 95% will result in reduced average daily gains, reduced wool growth, scouring, anaemia and even death, with the compounding issue of contaminating pasture with the resistant worms that have survived the drench. Pasture contamination with resistant worms due to ineffective drenching, results in more rapid reinfestation and increased resistance pressure.

Study Objective:

The purpose of this multi-site field study was to obtain current Australian efficacy data on the various actives against gastrointestinal nematodes of sheep. The field efficacy and resistance status of 5 active ingredients abamectin, moxidectin, levamisole, and albendazole, administered as single active drenches, as well as Startect (derquantel and abamectin combination), were tested on 50 commercial farms across Australia. Efficacy was measured by faecal egg count reduction (FECR) 14 days after treatment.

Study Outline:

Fifty commercial sheep properties with a pre study mob average worm egg count (WEC) of 200 eggs per gram (epg) or greater were enrolled in the study. On Day 0, 100 – 200 sheep (depending on the number of treatment groups per study location as additional products were often added) were drafted into a clean yard. Forty-five (45) fresh faecal samples were collected from the ground and sent to the laboratory. All sheep were then run up the race and sequentially allocated to treatment groups until 20 animals had been allocated to each group, i.e., 1st sheep to group 1, 2nd sheep to group 2 and so on. After allocation each animal was uniquely identified to their group and given a single oral dose of treatment (according to the label) as per their allocated treatment group. At the completion of Day 0 activities, all study sheep were returned to the paddock and grazed as a single mob until the end of the study. On Day 14, all sheep were brought back into the yards and faecal samples were collected, from the rectum, from the first 15 animals within each treatment group to come up the race. All samples were then sent to the laboratory. Pooled WECs and larval differentiation were performed on samples obtained on Day 0 and Day 14 (+/- 1 day). The 45 Day 0 samples were pooled into 5 composite samples, and the 15 Day 14 samples were also pooled into 5 composite samples. Counts were conducted using Mini-FLOTAC at a sensitivity (Limit of Quantification) of 10 epg.

At each study location, overall efficacy was assessed as the percentage reduction in the arithmetic mean (AM) of the pooled strongyle WECs for each treatment group relative to the pre-treatment sample WEC (Day 0), based on the WEC data collected on Day 14 (+/- 1 day). Pre-treatment (Day 0) larval cultures from pooled faeces were listed using percentages of each parasite genus identified. Larval culture results from the pooled faeces collected on Day 14 (+/- 1 day) were also listed for each treatment group separately using percentages of each parasite genus identified. The percentage reduction in AM for each genus was then estimated for each treatment group but only when the Day 0 mean pooled WEC per genus was at least 50 epg.

Study Results:

94% of the farms in this study showed evidence of resistance. Only the results of the actives/products that were administered at all study locations are reported below.

Across the 50 study locations, the overall mean AM efficacy for abamectin, benzimidazole, levamisole and moxidectin was less than 95%. Only Startect had a mean AM efficacy greater than 95%.

Drench resistance (efficacy less than 95%) to the single active drenches was shown to be prevalent in all major sheep producing regions of Australia despite them still being widely used by producers.

Every farm in NSW (22/22), SA (6/6) & WA (6/6) had evidence of resistance. In VIC, 12 out of 14 farms had evidence of resistance, and 1 out of 2 farms in Tasmania.

Of particular concern was that overall, 70% of farms enrolled in the study showed evidence of resistance to 3 or more actives:

- 72.7% of farms in NSW
- 83.3% of farms in SA
- 50% of farms in TAS
- 78.5% of farms in VIC
- 33.4% of farms in WA

And all the drench actives used in the study showed evidence of resistance:

- 70% of farms showed evidence of resistance to abamectin
- 84% of farms showed evidence of resistance to BZs (white drenches)
- 60% of farms showed evidence of resistance to levamisole (clear drenches)
- 38% of farms showed evidence of resistance to moxidectin

Resistance was also seen across all the major production limiting nematodes: Barber's Pole worm (*Haemonchus*), Black Scour worm (*Trichostrongylus*) and Brown stomach worm (*Teladorsagia*).

NOTE: Zoetis has now conducted 280 FECRTs with Startect. In these studies, the overall mean Australian efficacy is calculated to be 99.6% (SheepTRAX.com.au).

Drench selection advice for producers:

Choose an effective drench. Advise all your sheep producers to conduct either a DrenchCheck or DrenchTest (FECRT) to understand the resistance status on their farm given the prevalence of anthelmintic resistance. Every property is different therefore only by testing can producers know what drench works. More information on these tests can be found on www.wormboss.com.au or from your parasitology laboratory. The most expensive drench is the one that doesn't work!

Advise all your sheep producers to give a proper quarantine drench when bringing new sheep onto their property or when bringing sheep back from agistment. A quarantine drench (containing 4 actives with at least one component being a new active such as derquantel or monepantel) is an essential component of induction protocols if your clients are to successfully minimise the chance of bringing resistant worms onto their farms. Quarantine sheep for at least 72 hours post drenching with access to food and water. Post quarantine, release sheep onto a paddock with an existing larval burden to dilute the population in case some worms have survived. Ideally recommend a WEC 14 days post drenching to determine the success of the quarantine drench.

Always advise producers to use combination drenches. Combination drenches must be considered best practice by all producers based on the prevalence of resistance to single actives and also to delay the onset of resistance. If you know the efficacy of individual actives on your client's farm after completing a DrenchTest (FECRT), the efficacy of a combination product can be calculated by using the Combination Drench Efficacy Calculator on Wormboss (<http://www.wormboss.com.au/sheep-goats/tests-tools/management-tools/drenches/combination-drench-efficacy-calculator.php>).

Always recommend short-acting products. Restrict the use of long-acting or persistent products for specific purposes or high worm-risk times of the year. If using a long-acting treatment, use priming and tail cutter drenches (ensure the drenches chosen are effective on the property) to improve efficacy and help manage the onset of resistance.

Responsible use Startect + Sheepguard LA at pre-lambing²



Conclusion:

Drench resistance is highly prevalent in Australia and internal parasites are costing Australian sheep producers money. If your clients are not regularly conducting WECs and do not have an understanding of the drench resistance status on their property (by doing a DrenchCheck or a DrenchTest (FECRT)), production losses associated with worms should be expected.

References:

1. GHD Pty Ltd (Joe Lane) with Tristan Jubb, R.S., John Webb-Ware, Geoffry Fordyce, B.AHE.0010 Final Report: Priority list of endemic diseases for the red meat industries. 2015, Meat & Livestock Australia. p. 101-107

Measurement enabled pain solutions for livestock

Prof Mark R Hutchinson
ARC Centre of Excellence for Nanoscale BioPhotonics
University of Adelaide
Adelaide, SA

Pain as a Multidimensional Problem

Pain is complex, occurring at multiple levels from molecular and cellular (physiology = bio-) to psychological and behavioural (mind and brain = psycho-) and at both individual and social levels: hence a biopsychosocial model of pain is the most compelling and best aligns with the Five Freedoms ¹. But understanding the biopsychosocial pain nexus (the interface and connections of these inputs that leads to pain experiences) is largely quantitatively unexplored in animals. This knowledge gap presents a major obstacle to understanding our responsibilities and developing and implementing ethical practices for the care of domesticated, companion, captive animals, and wildlife. Animals frequently experience acute pain due to management procedures; injuries from fighting, environmental or housing factors; diseases or infections; and birth ². These acute experiences can result in persistent pain, which profoundly impacts animal well-being and health, including suffering and increased costs. Repeated acute and persistent pain episodes are chronic, life-long stressors, leading to reduced quality of life. In the animal production setting where we have preliminary data, persistent pain leads to reduced well-being (e.g., lower food intake and daily average weight gain), reducing productivity and generating significant economic losses ².

This discussion of what pain is and how we are to deal with it is timely, as Australian public and corporate concerns about captive and farm animal welfare are growing (71% identify it as a moderate or serious issue), and understandings of animal sentience are changing dramatically ³. However, we are currently unable to reliably diagnose or quantify either chronic or acute animal pain experiences ⁴. Even when animals in pain are identified, we typically cannot verify the success of interventions ². These limitations arise from our inability to understand what animal pain is, the prevalence of unproven assumptions about how to treat it, and the lack of tools to measure it ^{1,5}. These unmet needs are of immense social and economic importance, as seen in the \$3.2B liability related to pain in the Australian red meat industry ⁶, and form an integral part of the United Nations Sustainable Development Goals ⁷.

What are we to do?

By 'simply' acknowledging that pain is multidimensional the untapped opportunities we can access in animal production increase profoundly. These fall into three domains: 1) mechanistic understanding; 2) understanding enabled measurement; 3) measurement guided precision interventions and enhanced resilience.

Firstly, we can learn from the mistakes in human medicine and the leaps and bounds the field has taken in understanding pain. The aetiology of persistent pain in humans is known, but often overlooked as being comprised of a complex, twisted and multi factorial journey that culminates in a "cancer of the soul". This biopsychosocial challenge of pain is embodied in the recently announced update of the definition of pain by the International Association for the Study of Pain ⁸. Here, pain is acknowledged to be multidimensional and not solely a sensory experience, but one that has critical emotional components. This complexity is also embodied within recent advances in the basic science underpinning our mechanistic

understanding of persistent pain, which have embraced "the other brain" as an integrator of multiple life stimuli 4. This complex integration of life experiences, which are translated into neuro and immune signals cause the neuroimmune cells of the central nervous system to adapt and change the environment in which the neuronal system operates. If these adaptations present in the somatosensory neuroanatomical locations then this can present as hypernociception and eventual persistent pain.

Our appreciation for this neuroimmune signalling and its contributions to the health and disease of the brain has its origins in the study of the illness response 9. It is now apparent that these specialised brain-immune processes are engaged in a range of other disparate responses, including the innate rewarding drive to seek food. Additionally, this immune signal of pain also leads to novel "windows into the body" and the potential that a pain blood test might be possible 4,5. Through this objective quantification of pain state, we may be able to move away from empirical 'guesses' to objective measurement of this complex pain state. If this pain measurement is possible, either as a research tool, or en masse in production settings it will be possible to benchmark best practice. Moreover, this will fast track development of novel pain mitigation therapies that stop the development of chronic pain. Importantly, this also allows for the optimisation of breeding values and genetic lines to allow selection for chronic pain resilient traits. Critically, this is not to eliminate pain or stress all together. But rather minimise the likelihood of the maladaptive processes being engaged that lead to unnecessary and unwanted pathology.

Conclusion

It is clear that a greater understanding pain and an ability to quantify it will have profound benefits across animal husbandry practices. The field exploring the biopsychosocial multidimensional state of pain is rapidly moving, and will only make profound steps forward with new technologies and engagement across disciplines that can capture the information in 4 dimensions. Transdisciplinary collaborations that imagine, design, create and curate these technologies are needed. The convergence of human and animal biological sciences with common experimental and measurement technologies will enable a more rapid translation of clinical discoveries across multiple species, culminating in better health outcomes and productivity overall.

References

1. Mellor DJ. Updating Animal Welfare Thinking: Moving beyond the "Five Freedoms" towards "A Life Worth Living." *Animals* Multidisciplinary Digital Publishing Institute, 2016;6:21.
2. Williams S, Page S. *Literature review of pain and welfare impacts associated with on-farm cattle husbandry procedures*. Meat and Livestock Australia, 2014.
3. Futureye. *Australia's Shifting Mindset on Farm Animal Welfare (commissioned by the Commonwealth Department of Agriculture and Water Resources)*. 2019. <https://futureye.com/resources/>.
4. Grace PM, Hutchinson MR, Maier SF et al. Pathological pain and the neuroimmune interface. *Nat Rev Immunol* 2014;14:217–231.
5. Hutchinson MR, Terry R. Review: What innovations in pain measurement and control might be possible if we could quantify the neuroimmune synapse? *Animal* 2019;13:3000–3008.
6. *Red Meat Industry Strategic Plan 2020*. Red Meat Advisory Council, 2015.

7. Keeling L, Tunón H, Olmos Antillón G et al. Animal Welfare and the United Nations Sustainable Development Goals. *Front Vet Sci* 2019;6:336.
8. Raja SN, Carr DB, Cohen M et al. The revised International Association for the Study of Pain definition of pain: concepts, challenges, and compromises. *Pain* 2020;161:1976–1982.
9. Devlin BA, Smith CJ, Bilbo SD. Sickness and the social brain: How the immune system regulates behavior across species. *Brain Behav Evol* 2021; <http://dx.doi.org/10.1159/000521476>.

General surveillance for endemic and emergency animal diseases

Bruce Jackson and Rob Barwell
Animal Health Australia
Canberra ACT

Introduction

General surveillance is a “citizen science” form of passive surveillance, where members of the public gather and report information on the geographic spread and occurrence of weeds, pests and diseases.

General Surveillance – animal health

General surveillance may be used to provide evidence of freedom from trade limiting diseases, allow early response to incursions, monitor prevalence and trends of endemic diseases, increase understanding of the epidemiology of a disease and provide an early warning system for endemic disease outbreaks.

Currently most Australian jurisdictions rely on reporting from veterinary practitioners, government veterinary field staff, veterinary laboratories and hotlines to alert authorities to cases of a suspected Emergency Animal Disease (EAD). These kinds of arrangements can be slow to pick up outbreaks as was demonstrated in England in 2001 where foot and mouth disease (FMD) spread for some weeks before being detected in an abattoir.

General surveillance seeks to broaden the surveillance network and break down some of the barriers to reporting. Greater knowledge on the presence, prevalence and seasonality of outbreaks of endemic disease may also be captured at reasonable cost by such systems, and the trust and established communication channels can be invaluable if an outbreak does occur.

The important barriers to producers requesting investigation of significant endemic disease outbreaks and suspicion of EADs to veterinarians and regulatory authorities are:

1. Fear of regulators – many producers equate regulators with restrictions such as quarantine or animal welfare investigations.
2. Producers are concerned that they will be embarrassed amongst their peer group if regulators are seen on their property.
3. Producers report that the cost of calling in veterinary practitioners (including laboratory fees) can be significant and sometimes a definitive diagnosis is not reached. Some practices will not allow producers to speak with the veterinarian on the telephone, but insist they make an appointment for the veterinarian to visit.
4. Producers complain that local veterinarians or department veterinarians do not have sufficient expertise in sheep/goat/camelid medicine.
5. Producers are ignorant of the clinical signs of EADs, some of which can be not that spectacular, for example FMD in sheep and goats.
6. Producers and lay advisors will not usually include EADs in a differential diagnosis.
7. Producers can diagnose and treat the condition themselves without referral to a veterinarian (for example ryegrass staggers).
8. Many producers prefer to consult with people they know and trust, such as local rural merchandisers and contractors.

General surveillance uses these trusted local advisors as ‘notifiers’ to gather the information.

The features of successful general surveillance programs include¹:

1. A dedicated technically competent and trusted coordinator who can manage data, notifier and stakeholders' engagement
2. Notifiers derive 'in-kind' benefits, such as easy access to the expertise of the coordinator who can help them better assist their clients
3. Simple flexible reporting formats
4. Using non-regulatory language (avoid using the term "surveillance") when communicating with notifiers or producers
5. Clear program objectives/scope, and
6. Adequate funding.

A very important feature of using such a system to gather animal health information is that the notifier does not normally reveal the identity of the producer to the coordinator, and it is also best if the coordinator is not a regulator.

The importance of early detection of an EAD

Early detection is essential if an EAD is to be quickly eradicated or controlled². Even a small outbreak of FMD in Australia will cost billions of dollars and severely affect the livelihood of producers and the veterinarians servicing them for a number of years, and every additional day it is present will cost millions of dollars.

Veterinarians should include EADs in their differential diagnostic list when dealing with cases where an obvious endemic disease diagnosis is not apparent. The most significant EADs for sheep, goats and camelids are detailed in the AUSVETPLAN disease specific manuals³ and are listed below:

Table 1: Obvious clinical signs of significant Emergency Animal Diseases of Sheep, Goats and Camelids

| EAD | Sheep | Goat | Camelid | Obvious clinical signs |
|------------------------|-------|------|---------|--|
| FMD | Y | Y | Y | Usually mild. Vesicles on tongue, coronary band, interdigital space - rupture easily to form shallow ulceration and lameness. Young lamb deaths. |
| Anthrax | Y | Y | Y | Sudden death, blood-stained discharge from external orifices. |
| Sheep and Goat pox | Y | Y | N | Pox lesions most obvious on areas with short hair/wool. Deaths. |
| Peste petits ruminants | Y | Y | Y | Fever, depression, nasal and ocular discharge/crusting, oral mucosal necrosis/ulceration, diarrhoea/dysentery, deaths. |
| Scrapie | Y | Y | N | Neurological signs |
| Bluetongue | Y | Y | Y | Fever, nasal discharge, oedema of lips/tongue/face, mouth ulcers, red coronary band, lame, abortions. Goats, camelids milder. |

There are also a number of other exotic diseases where sheep and/or goats and/or camelids are incidental hosts (for example Aujeszky's disease, rabies, Surra) or are unlikely to occur in Australia due to presumed lack of vectors (heartwater, louping ill), very limited world-wide

distribution (Borna disease, Nairobi sheep disease), or are only likely to enter through live animal movement (*Brucella melitensis*, maedi-visna, contagious caprine pleuropneumonia, pulmonary adenomatosis and sheep scab)⁴. However, veterinarians should still include such diseases in the differential diagnosis when appropriate.

New diseases could also appear in sheep, goats and camelids as they have in horses and bats.

Veterinarians could consider downloading a digital copy of the 'Emergency animal diseases: A field guide for Australian veterinarians' onto their phone⁵.

The Surveillance and Biosecurity Extension in Tasmania project.

After a successful surveillance pilot project in 2018/2019, Animal Health Australia, with support from WoolProducers Australia and Sheep Producers Australia, partnered with the Tasmanian Department of Primary Industries, Parks Water and Environment (DPIPWE) to fund continuation and expansion of the project, aligned closely with the National Sheep Industry Biosecurity Strategy (NSIBS) 2019–2024 for 2020/2021 and then again for 2021/22.

Objectives of the project

- 1) Maintain a network of rural service providers ('notifiers'), who cover most of Tasmania, with an emphasis on the sheep industry, reporting surveillance information to a coordinator who can initiate timely disease investigations if EADs are suspected.
- 2) Produce general surveillance reports able to support claims of disease freedom for the Tasmanian sheep industry and assist rural service providers and their clients to control endemic diseases more efficiently.
- 3) Promote good biosecurity practices within the Tasmanian sheep industry, consistent with the NSIBS.

Notifier network

A network of rural veterinarians, merchandising agents, sheep contractors (foot-paring, pregnancy scanning, lamb marking), shearing contractors, agricultural advisors, livestock agents, pet food processors, livestock transporters, saleyard operators and abattoir operators has been established and maintained.

The coordinator provides support and advice to the notifiers who can ring any time, sending photos and videos is encouraged and triage advice given, often with a differential diagnostic list and recommendation that a local veterinarian is called in. Occasional cases are referred to the coordinator, especially if the problem has broader implications such as a feed manufacturer including excessive amount of copper in sheep pellets.

In return the notifiers supply de-identified information on a monthly basis. Reporting is by telephone, email, text or in person, using common names for the diseases and conditions observed.

The coordinator also receives copies of the abattoir reports from the National Sheep Health Monitoring Project (NSHMP) abattoir in Tasmania. This data is summarised in the report and changes in the prevalence of sheep abattoir conditions are monitored.

Support for veterinarians

There are no medium to large sized practices in Southern Tasmania or on the Bass Strait islands that specialise in production animal work. The coordinator is available to support the practices that do this work by discussing cases, linking them up with other support,

researching specific problem cases and to mentor younger veterinarians who are interested in developing expertise in production animal medicine.

Outputs

A monthly report is collated by the coordinator and sent back to the notifier network, extension outlets, and the sponsors. Any stakeholder can register to receive a digital copy of the monthly report⁶ by email and a Facebook group has also been set up. The report contains a table of conditions reported that month with brief details on how to recognise, treat and prevent the condition, often with links to authoritative web sites such as ParaBoss. Sections on seasonal disease alerts, a ‘biosecurity story of the month’, a short section promoting national surveillance/prevention programs and links to a number of useful web sites such as Farm Biosecurity are also included.

The coordinator attends as many face-to-face producer events as possible, to gather information and to distribute biosecurity extension material.

Results

There were at least 900 reports of diseases/conditions made by the notifier network over the period 1 November 2020 to 30 October 2021. If a notifier reported that a condition is ‘widespread’, it was counted as 2 reports. Abattoir data is not included in this tally.

Table 2: Summary of the number of reports of diseases/conditions for each species from 1 November 2020 to 30 October 2021. These totals include multiple reports of the same disease/condition.

| Species | sheep | cattle | goats | pigs | camelids | deer | poultry |
|-------------------------------------|-------|--------|-------|------|----------|------|---------|
| Number of disease/condition reports | 649 | 175 | 39 | 18 | 6 | 0 | 13 |

The total number of different disease/conditions detected in livestock species since the project first began in March 2018 through to November 2021 is set out below. These totals will include the same condition reported using different terminology by notifiers as well as different conditions presenting in a similar fashion reported as one condition.

Table 3: Total number of different diseases/conditions reported by notifiers from March 2018 to November 2021

| Species | sheep | cattle | goats | pigs | camelids | deer | poultry |
|-------------------------------|-------|--------|-------|------|----------|------|---------|
| Number of conditions reported | 268 | 127 | 45 | 24 | 18 | 1 | 13 |

Abattoir monitoring has revealed that “nephritis” is becoming more prevalent in lambs and adult sheep in Tasmania.

Two cases were reported where some suspicion of an EAD was entertained by the coordinator. On both occasions the coordinator convinced the notifier to divulge the name of the manager and attended personally to rule out an EAD at no cost to the producer and without involvement of regulatory personnel. Both cases turned out to be common conditions presenting a little differently.

The first diagnosis of *Mycoplasma ovipneumoniae* in Tasmania was initiated through information gathered by this project. A new condition described as 'lumpy jaw' where the mandibular symphysis in adult sheep becomes an enlarged bony mass has also been detected.

Conclusion

General surveillance programs can be a very effective and economical way of broadening the reach of passive animal health surveillance programs, tapping into networks of industry stakeholders who do not usually communicate with regulatory authorities, enhancing endemic disease control, increasing the chances of early detection of EADs and establishing communication channels that will be very useful if an EAD outbreak did occur.

Acknowledgement

Funding for the Surveillance and Biosecurity Extension in Tasmania project is provided by Animal Health Australia (with support from Sheep Producers Australia and WoolProducers Australia) and by the Department of Natural Resources and Environment (NRE) Tasmania.

References

1. Kruger H, Ticehurst J and van der Meer Simo A (2022). Guidelines for General Surveillance Programs – Insights and considerations from systems thinking and nine case studies. ABARE, Research Report 22.03.
2. Anon (2011) Determining whether an EAD can be eradicated. Animal Health Australia. <https://animalhealthaustralia.com.au/nationally-agreed-standard-operating-procedures/> accessed on 7/05/2022.
3. Geering WA, Forman AJ & Nunn MJ (1995). Exotic animal diseases: a field guide for Australian veterinarians, Bureau of Resource Sciences, Department of Primary Industries and Energy, Australian Government Publishing Service, Canberra.
4. AUSVETPLAN: <https://animalhealthaustralia.com.au/ausvetplan/> accessed on 7/05/2022.
5. Department of Agriculture and CSIRO 2019, Emergency animal diseases: A field guide for Australian veterinarians, Canberra, August. CC BY 4.0.
6. Tasmanian Livestock Health Monitoring Report. <https://animalhealthaustralia.com.au/tas-health/> accessed on 7/05/2022.

Evidence-based approach to investigating poor reproductive performance in maiden ewes

Caroline Jacobson^a, Tom Clune^a, Elsa Glanville^c, Angus Campbell^d, Amy Lockwood^a, Serina Hancock^a, Sue Beetson^a, Mieghan Bruce^b, Daniel Brookes^c, Colin Trengove^e, Ryan O'Handley^e and Andrew N. Thompson^a

^a Centre for Animal Production and Health, Murdoch University, Murdoch, WA

^b Centre for Biosecurity and One Health, Murdoch University, Murdoch, WA

^c Mackinnon Project, University of Melbourne, Werribee, Victoria

^d Nossal Institute for Global Health, University of Melbourne, Melbourne, Victoria

^e School of Animal and Veterinary Sciences, University of Adelaide, Roseworthy, SA

Summary

Improving ewe reproductive performance remains a priority for the Australian sheep industry. Here we present a protocol for veterinary practitioners to investigate cases of poor reproductive performance. The proposed methodology can be readily adapted to account for resources available and risk factors specific to the farm in question. This protocol will allow practitioners to determine the (a) timing and (b) likely causes of foetal/lamb loss to inform strategies targeted at improving reproductive performance.

Key points for practitioners

- Investigation of ewe poor reproductive performance can be adapted for different clients ranging from basic (measuring reproductive rate and lamb survival) to comprehensive (including differentiating abortions from perinatal loss, and cause of foetal/lamb mortality)
- Encouraging clients to maintain good records on reproduction performance across years and age groups can highlight farms where investigation is warranted and will inform investigation protocols that appropriate for the client
- Practitioners can use the information from reproduction investigations to develop tailored management plans to improve reproductive performance specific to the client, and provides a point of differentiation from generic extension/adoption programs
- Consider utilising significant disease investigation or surveillance programs
- Practitioners can reduce risks to public health by advising clients on appropriate hygiene when handling ewes during pregnancy and lambing

Introduction

Ewe reproductive performance is one of the important determinants of sheep enterprise profitability. Lamb marking rates in Australia have increased by about 15% over the last 30 years.^{1,2} This has been attributed to changes in flock structure (including breed) and adoption of husbandry practices including differential management of single- and multiple-bearing ewes.^{3,4} Improving ewe reproductive performance is a priority for the Australian sheep industry, with survival of twin lambs and reproductive performance of maiden ewes identified as key targets for improving the performance of the national flock.⁵ Management strategies that focus on improving both reproductive rate and lamb survival can improve enterprise profitability and animal welfare.⁵

Survival of lambs between scanning and marking remains a challenge for the Australian sheep industry. Lamb mortalities have been estimated at 13.3 million head per year based on 38 million breeding ewes.⁶ Approximately 10% of single-born and 30% of twin-born lambs die before weaning under Australian conditions, with most lamb mortalities occurring in the perinatal period between birth and 3 days of age.^{7,8} Dystocia, starvation-mismothering-

exposure and stillbirths are the most important causes of death reported for lambs in the perinatal period.^{6, 8-10.}

The reproductive performance of maiden (primiparous) ewes on Australian sheep farms is often poorer and more variable compared to multiparous ewes.^{7, 11-13.} Lower marking rates for maiden ewes are due to both lower reproductive rate and lower lamb survival compared to multiparous ewes.¹¹ A recent Australian study identified mid-pregnancy abortion was an important contributor to lamb mortality in some ewe lamb flocks abortions were detected in $\geq 2\%$ ewes for approximately one in three flocks.^{7.} Notably, mid-pregnancy abortion in 5- 50% of ewes were detected in 4/19 ewe lamb flocks with no obvious external signs of illness that would alert a producer or veterinarian to the problem.

Differentiating perinatal mortalities from mid- or late-pregnancy abortion has important implications for improving flock reproductive performance. Implementing strategies to address lamb mortality in the perinatal period (primarily from dystocia and starvation-mismothering) are unlikely to have a meaningful impact on mid- or late-pregnancy abortion related to infectious aetiologies.

Foetal and lamb mortalities are typically multifactorial and may have infectious or non-infectious aetiologies, or a combination of both. Numerous endemic diseases may cause mid- and late-pregnancy abortion in Australian ewes, with campylobacteriosis, listeriosis and toxoplasmosis the most common aetiological diagnoses made for abortion investigations in Australia.^{14, 15} More recently, abortions and stillbirths associated with *Chlamydia pecorum* have been reported in ewe lambs across Australia.^{16, 17.} Important exotic diseases also cause abortion, including *Chlamydia abortus* and *Brucella melitensis*.

In addition to abortion and stillbirth, antepartum infections may result in low vigour lambs with increased perinatal mortality (e.g. starvation-mismothering). In general, younger, maiden ewes will be at higher risk of infectious reproductive disease as they are less likely to have developed protective immunity compared with mature, multiparous ewes.

Management strategies devised to improve poor reproductive performance resulting from foetal or neonatal mortality should be informed by an understanding of contributing causes of loss on the enterprise in question. Foetal and neonatal lamb necropsies, in addition to a thorough history, farm visit and examination of mobs concerned is standard. Where infectious aetiologies are suspected, an aetiological diagnosis should be sought where possible. Submitting appropriate samples, including placenta if possible, for laboratory testing, and careful storage and transport of samples aids in determining the aetiological diagnosis.¹⁴

Protocol for investigating sub-optimal reproductive performance

A protocol for on-farm investigation of poor reproductive performance in maiden ewes based on the findings from a national project that investigated the magnitude and causes of foetal/lamb mortality between pregnancy scanning and lamb marking are outlined in Table 2. This protocol can be adapted for multiparous ewes.

The proposed methodology was developed initially for lamb survival research, but can also be readily adapted for investigating poor reproductive on commercial sheep farms. This protocol allows practitioners to determine the (a) timing and (b) likely causes of foetal or lamb mortality to inform strategies targeted at addressing lamb survival. Practitioners should consider the available resources (facilities, labour), risk factors for infectious diseases specific to the farm system and location concerned, and risk:benefit ratio for additional procedures when determining which components of the protocol are warranted. Examples of modification of the procedures for investigation on commercial farms are outlined in Table 2.

Table 2: Examples of components of investigation for poor reproductive performance on commercial sheep farms

| Measure | Outcome | Activity needed | Useful in commercial field investigation? |
|-------------------------|--|--|--|
| Pre-joining inspection | Pre-empt issues that may impact reproductive rate that season | Pre-joining inspection of rams, ewes | Rams – yes Ewes - consider for flocks with history of poor reproductive rate |
| Reproductive rate | Potential marking rate Identify issues occurring at joining/early pregnancy | Scanning for multiples (scan 1) | Yes |
| Lamb survival | Quantify mortality scanning to marking without discriminating <i>in utero</i> and perinatal losses | Scanning for multiples Lambing singles and multiples in separate mobs | Yes |
| Mid-pregnancy abortion | Quantify contribution of mid-pregnancy abortion to overall lamb survival | Scan 2 | Consider for ewe lambs or properties where abortions suspected |
| Late pregnancy abortion | Quantify contribution of late-pregnancy abortion to overall lamb survival | Scan 3 and/or daily inspection to collect dead lambs | Consider for ewe lambs or properties where abortions suspected |
| Perinatal mortality | Quantify contribution of perinatal mortality to overall lamb survival Identify cause of death | Daily inspection to collect/count dead lambs Gross post mortem ± laboratory investigation | Consider for flocks with high mortality in single and multiple lambs to inform management and genetics |

Managing public health risks

Several endemic disease agents associated with abortion, stillbirths and reduced neonatal viability have zoonotic potential. These include *Toxoplasma gondii*, *Campylobacter* spp., *Listeria* spp., *Leptospira* spp. and *Coxiella burnetii*. Some exotic reproductive diseases have zoonotic potential (e.g. *Chlamydia abortus*, *Salmonella abortus* and *Brucella melitensis*).

Practitioners can reduce potential public health impacts by advising clients on recommendations for hygiene when handling pregnant and periparturient ewes, neonatal lambs or any aborted tissues. This includes:

- Use disposable gloves where possible and wash hands with soap or detergent after handling affected ewes, lambs or aborted tissues
- Goggles/eye protection and mask may be warranted in high-risk scenarios
- Pregnant women should avoid handling lambing ewes or any aborted tissues
- Cuts and needlestick injuries are a potential source of zoonotic transmission
- Q-fever (*C. burnetii*) can be transmitted through aerosols and dust. Residues contaminated by birth fluids, blood, faeces, or urine may remain infectious for months to years.
 - *C. burnetii* can be dispersed by windborne spread of contaminated dust over several kilometres. Activities generating dust, such as mustering, shearing, transport may represent a risk to susceptible people.
 - Transmission can occur to people handling clothing contaminated with blood, faeces, urine, birthing fluids etc. Family members do not have to be in direct contact with livestock to be at risk of zoonotic transmission

- People working closely with livestock should discuss vaccination for Q-fever and leptospirosis with their medical practitioner.

Reproductive disease surveillance programs

Practitioners may be able to access significant disease investigation programs when investigating abortions, lamb mortality and/or poor reproductive rate. Practitioners should contact the local district veterinarian to discuss eligibility of cases and for further details about subsidised disease investigations (Table 3).

Table 3: Disease investigation programs

| State | Contacts | Program | Website contact |
|-------|---|--|---|
| All | Animal Health Australia | National significant disease investigations program (NSDI) | https://animalhealthaustralia.com.au/collaborative-disease-investigations/ |
| WA | Department of Primary Industries and Regional Development (DPIRD) | Ewe abortion and newborn lamb deaths surveillance program | https://www.agric.wa.gov.au/livestock-biosecurity/ewe-abortion-and-newborn-lamb-deaths-surveillance-program |
| | Diagnostics and Laboratory Services (DDL) – Animal pathology | Significant Disease Investigation Program | https://www.agric.wa.gov.au/livestock-biosecurity/significant-disease-investigation-program |
| NSW | Department of Primary Industries (DPI NSW) | Abortion investigation | https://www.dpi.nsw.gov.au/about-us/services/laboratory-services/veterinary-abortion-investigation |
| | | Significant Disease Investigation Program | https://www.dpi.nsw.gov.au/biosecurity/your-role-in-biosecurity/veterinary-professionals/programs-and-projects |
| TAS | Departments of Natural Resources and Environment Animal Health Laboratory | Significant Disease Investigation Program | https://nre.tas.gov.au/biosecurity-tasmania/animal-biosecurity/animal-health/information-for-veterinary-practitioners |
| VIC | Agriculture Victoria AgriBio Laboratories | Significant Disease Investigation Program | https://agriculture.vic.gov.au/biosecurity/animal-diseases/significant-disease-investigation-sdi-program |
| SA | Department of Primary Industries and Regions South Australia (PIRSA) VETLAB/ Gribbles Veterinary Pathology | Significant Disease Investigation Program | https://pir.sa.gov.au/biosecurity/animal_health/veterinarians/livestock_disease_surveillance |

Table 2: Protocol for investigating poor reproductive performance at flock level, including determination of reproductive rate, abortion, and perinatal mortality

| Timing | Activity | Outcome |
|--------------|---|---|
| Pre-joining | Rams: check the number provides adequate ram power (ram:ewe ratio); ram team age demographics; which rams to which ewes | Investigate potential for ram infertility contributing to poor reproductive rate |
| | Rams: age, breeding soundness exam including penis, prepuce, testicle circumference and palpation, body condition score, teeth and feet | |
| | Rams: <i>Brucellosis ovis</i> exclusion (testicular palpation ± serology) | |
| | If joining out of season, check teaser preparation protocol | Investigate factors that may impact ewe attainment of puberty and cycling that contribute to poor reproductive rate |
| | Ewes: age, weight and condition score | |
| | Ewes: determine farm-level risk of campylobacteriosis and instigate vaccination if warranted (2 injections at least 3 weeks apart given pre-joining). Consider a vaccination trial (treated and untreated controls). | Address campylobacteriosis risk |
| | Optional: test for vitamin and trace element deficiencies relevant to region (e.g. vitamin E, vitamin A, selenium, copper, zinc). Consider a trace element supplementation trial (treated and untreated controls). | Determine if vitamin or mineral deficiencies could be impacting ewe fertility |
| | Optional: collect blood samples for ewes to determine pre-gestation exposure to infections that require primary exposure during pregnancy for reproductive disease (e.g. <i>Toxoplasma</i> and BDV/pesti-virus). Serum and plasma can be stored at -20°C. | Determine timing of primary infection in event that infectious disease is subsequently identified |
| Joining | Aim for 35-day joining | Schedule scanning to optimise accuracy of foetal counting |
| | | Plan monitoring during lambing by estimating start and duration of lambing based on joining dates |
| Post-joining | Confirm joining dates to schedule pregnancy scanning within target period | Accuracy for foetal counting is highest 80-100 days from start of joining |

| Timing | Activity | Outcome |
|---|---|--|
| Scan 1 | Scan with foetal count (empty, single, twin/triplet) at 50 days from removal of rams/85 days from start of joining (50-85 days gestation) | Record number of ewes scanned, number of foetuses counted and calculate reproductive rate |
| | Record litter size against ewe ID, or draft by litter size into mobs | Determine if poor reproductive rate (poor conception/embryo survival) is contributing to overall reduced reproductive performance |
| | Record ewe weight and condition score | Determine if sub-optimal condition score profile or inadequate growth rate (ewe lambs) are contributing to poor reproductive rate |
| | Ewes with suspected recent abortion should be identified and drafted off from pregnant ewes. Consider monitor/re-check to confirm abortion. | Reduce transmission of infectious disease by affected ewes Validate accuracy of scanning in detecting abortion |
| | Consider collecting blood samples and vaginal/rectal swabs from ewes with evidence of recent abortion Consider collecting blood samples again in 2-3 weeks (paired serology) | Serology can be conducted for <i>Toxoplasma</i> , <i>Neospora</i> , <i>Coxiella</i> , <i>Campylobacter</i> . Discuss recommended tests with regional laboratory Paired serology to detect rising/falling titre or IgM/IgG may indicate timing of infection Vaginal/rectal swabs can be used for point-of-care tests or molecular tests <i>Chlamydia</i> spp. |
| Check pasture for evidence of oestrogenic clover (seasonal – may need to be delayed until sufficient pasture growth for species identification) | Determine if phytoestrogens are contributing to infertility | |

| Timing | Activity | Outcome |
|--------|---|---|
| Scan 2 | <p>Mid-pregnancy abortion monitoring scan conducted 85 days from removal of rams/120 days from start of joining (85-120 days gestation)* to identify ewes with non-viable pregnancy (determined by lack of heartbeat or foetal movement, changed echogenicity of cotyledons) or pregnancy loss (absence of foetus).</p> <p>*Scan 2 should be conducted at least 30 days after scan 1. Scan 2 can be delayed to identify more ewes with foetal loss, but scanning becomes slower and less accurate later in pregnancy. Note that scanning later in pregnancy takes longer than standard pregnancy scanning – this should be considered when negotiating costs with the scanning provider and producer.</p> <p>Consider cost and risk associated with additional handling against potential benefit from discriminating abortion from perinatal losses.</p> | Detect mid-pregnancy abortion |
| | Ewes with suspected abortion should be identified and drafted off from pregnant ewes. These ewes should be monitored over lambing to confirm abortion. | <p>Reduce transmission of infectious disease by affected ewes</p> <p>Validate accuracy of scanning in detecting abortion</p> |
| | <p>Collect blood samples and vaginal/rectal swabs from ewes with evidence of mid-pregnancy abortion</p> <p>Consider collecting blood samples again in 2-3 weeks (paired serology)</p> | <p>Serology can be conducted for <i>Toxoplasma</i>, <i>Neospora</i>, <i>Coxiella</i>, <i>Campylobacter</i>. Discuss recommended tests with regional laboratory</p> <p>Paired serology to detect rising/falling titre or IgM/IgG may indicate timing of infection</p> <p>Vaginal/rectal swabs can be used for point-of-care tests or molecular tests <i>Chlamydia</i> spp.</p> |

| Timing | Activity | Outcome |
|--------------------|---|---|
| Post-Scan 2 | <p>If a high frequency of mid-pregnancy abortion is detected, increase the frequency of monitoring for all maiden ewe mobs for evidence of abortion (<i>e.g.</i> aborted lambs or foetal membranes, breech staining).</p> <p>Ewes with evidence of non-viable lambs at Scan 2 (<i>i.e.</i> lambs with no movement or heartbeat) should be monitored closely for evidence of abortion. If abortions are observed;</p> <ul style="list-style-type: none"> • People handling aborting ewes and aborted tissues should use PPE (<i>i.e.</i> gloves and mask) and practice good hygiene. Pregnant women and immunocompromised individuals should avoid contact where possible. • Any aborted tissue (<i>i.e.</i> aborted lamb or foetal membranes) should be collected into a clean plastic bag, sealed and stored at 4-10°C until submission for diagnostic testing. • If submission is likely to be delayed, discuss freezing samples with veterinarian/laboratory. <p>Aborted tissue should be submitted for abortion surveillance testing. This should include:</p> <ul style="list-style-type: none"> • Full necropsy and tissue collection where possible • Submission of fresh and fixed tissues where possible • Discussion of sample collection and submission protocol with the receiving laboratory in advance where possible | Disease investigation to determine aetiological diagnosis (cause) including infectious agents |

| Timing | Activity | Outcome |
|-----------------------------|---|---|
| Optional: Scan 3 | <p>If a high frequency of mid-pregnancy abortion has been detected, a third scan can be conducted at day 120-140 gestation to identify ewes with late-pregnancy foetal loss.</p> <p>The benefit of quantifying late-pregnancy abortion should be considered against the risk of metabolic disease when handling ewes during late-pregnancy. Feed and water should not be withheld from ewes in late pregnancy. Treatment for metabolic disease (pregnancy toxaemia, hypocalcaemia) should be available, and ewes should be monitored carefully during and after yarding.</p> <p>Note that scanning later in pregnancy takes longer than standard pregnancy scanning and additional technical skills are needed to detect foetal viability (foetal movement or heartbeat) during late pregnancy. This should be considered when negotiating costs with scanning provider and producer.</p> | Detect late-pregnancy abortion |
| | <p>Collect blood samples and vaginal/rectal swabs from ewes with evidence of late-pregnancy abortion</p> <p>Consider collecting blood samples again in 2-3 weeks (paired serology)</p> | <p>Serology can be conducted for <i>Toxoplasma</i>, <i>Neospora</i>, <i>Coxiella</i>, <i>Campylobacter</i>. Discuss recommended tests with regional laboratory</p> <p>Paired serology to detect rising/falling titre or IgM/IgG may indicate timing of infection</p> <p>Vaginal/rectal swabs can be used for point-of-care tests or molecular tests <i>Chlamydia</i> spp.</p> |
| | Ewes with suspected late-pregnancy abortion should be drafted off from pregnant ewes. These ewes should be monitored over lambing to confirm abortion. | <p>Reduce transmission of infectious disease by affected ewes</p> <p>Validate accuracy of scanning in detecting abortion</p> |

| Timing | Activity | Outcome |
|-------------|---|--|
| Pre-lambing | Allocate ewes to lambing paddocks based on litter size (single, multiple or twins/triplets; separate dry/aborted). Record the number of ewes allocated to each lambing paddock. | Lambing/marking in litter size groups allows for calculation of single/multiple lamb survival Accurate ewe head counts and foetal count (based on scanning) allows for calculation of lamb and ewe mortality Monitoring dry/aborted ewes over lambing used to confirm scanning records |
| | Ewes: record weight and condition score | Determine if sub-optimal condition score at lambing or condition score/weight profile in late pregnancy are contributing to poor lamb survival |
| | Ewes: check for evidence of abortion or premature lambing (e.g. breech staining, foetal membranes, aborted foetus) | Detect late-pregnancy abortion |
| | Ewes: Collect blood samples and rectal/vaginal swabs from any ewes with evidence of late-pregnancy abortion Consider collecting blood samples again in 2-3 weeks (paired serology) | Serology can be conducted for <i>Toxoplasma</i> , <i>Neospora</i> , <i>Coxiella</i> , <i>Campylobacter</i> . Discuss recommended tests with regional laboratory Paired serology to detect rising/falling titre or IgM/IgG may indicate timing of infection Vaginal/rectal swabs can be used for point-of-care tests or molecular tests <i>Chlamydia</i> spp. |
| | Collect any aborted tissues or dead premature lambs for sampling (refer to post-scan 2 for procedures) | Collect samples for disease investigation to determine aetiological diagnosis (cause) |
| | Undertake predator control ahead of lambing | Minimise losses due to predation and disturbance |
| | Habituate ewes to routine inspection in lambing paddock. Use a different vehicle for inspections to that used for mustering or feeding where necessary. | Minimise disturbance from inspections during lambing rounds |
| | Record paddock characteristics: shelter, feed-on-offer, mob size | Determine if paddock characteristics are contributing to perinatal mortalities |

| | | |
|--|---|---|
| | Check pasture for evidence of oestrogenic clover (seasonal) | Determine if phytoestrogens are contributing to reduced lamb survival |
|--|---|---|

| Timing | Activity | Outcome |
|----------------|---|--|
| Lambing | Inspect ewes in lambing paddock once or twice daily. Record the number of lambs born and/or collect dead lambs Record the number of lambs born or estimate based on the number of lambs marked and dead lambs recovered NOTE: Inspections during lambing should be balanced against risk of disturbance of ewes. Habituation of ewes to inspection should be considered and instigated prior to the commencement of lambing. | Use number of lambs born to calculate (a) foetal loss between scan 1 and lambing, and (b) perinatal loss |
| | Weigh dead lambs Optional: consider weighing and tagging lambs at birth (seedstock) | Determine whether birthweight is contributing to lamb mortality |
| | Necropsy dead lambs using method described by Holst ¹⁸ to assign cause of death category | Determine cause of perinatal death |
| | Collect appropriate samples from aborted or premature lambs, stillborn lambs, lambs with evidence of inflammatory changes at necropsy or small weak lambs for laboratory investigation to identify aetiological diagnoses. This should include <ul style="list-style-type: none"> • Full necropsy and tissue collection where possible • Submission of fresh and fixed tissues where possible Discuss of sample collection and submission protocol with the receiving laboratory | Disease investigation to determine aetiological diagnosis (cause) including infectious agents |
| | Record ewe mortalities and cause of death (if known). Consider necropsy for unexplained ewe mortalities or where mortality exceeds target | Determine cause of ewe death |
| | Monitor dry/aborted ewes periodically over lambing | Confirm scanning records |
| | Record daily temperature, wind speed and rainfall (measured or closes BOM station). Calculate chill index. | Relate chill index to lamb mortality pattern |

| Timing | Activity | Outcome |
|---------|---|---|
| Marking | Record the number of lambs marked | Use number of lambs marked to estimate number of lambs born Calculate (a) foetal loss between scan 1 and lambing (b) lamb mortality between birth and marking (c) overall lamb survival scan 1 to marking |
| | Ewe udder inspection (wet/dry) | Identify ewes that have failed to lamb (no udder development) or failed to rear |
| | Ewes: Collect blood samples from sample of ewes that failed to lamb or failed to rear in mobs with (a) high proportion of ewes that fail to lamb, or (b) very high perinatal lamb mortality | Serology can be conducted for <i>Toxoplasma</i> , <i>Neospora</i> , <i>Coxiella</i> , <i>Campylobacter</i> . Discuss recommended tests with regional laboratory |
| | Consider collecting blood samples again in 2-3 weeks (paired serology) | Paired serology to detect rising/falling titre or IgM/IgG may indicate timing of infection |

Acknowledgments

This research was funded by Meat and Livestock Australia (B.AHE.0318). Tom Clune was supported with post graduate scholarships from Meat and Livestock Australia, Sheep Industry Business Innovation (Department of Primary Industries and Regional Development, Western Australia) and the 2020 Science and Innovation Awards for Young People in Agriculture, Fisheries and Forestry (Australian Government Department of Agriculture in partnership with Australian Wool Innovation). Some of the laboratory diagnostic testing was supported through the DPIRD Abortion Surveillance Scheme.

The recommendations reported in this paper were the culmination of a larger project that could not have been achieved without the contributions of a large number of producers, scientists and veterinarians.

We thank the participating farmers who provided access to their animals and facilities, conducted lambing rounds, and collected and stored lambs for necropsy.

We thank the teams at the veterinary laboratories that conducted sample testing to inform the recommendations reported in this paper; Celia Smuts, Janine Simmonds and the staff at VetPath; Sam Hair, Cameron Loomes, Richmond Loh and Anna Erickson at DPIRD; and the team at ACE Laboratory,

We thank Tom La and Nyree Phillips (Murdoch University), Louis Lignereux and Rob Paterson (University of Adelaide), Andrew Whale, Mary McQuillan and Patrick Hannemann (Livestock Logic, Hamilton, Victoria), Sean McGrath (Millicent Veterinary Hospital), Simon Edwards and Michelle Smart (Willunga Veterinary Hospital), and Lauryn Stewart and Deb Lehmann (Kangaroo Island Veterinary Hospital) for assistance with sample collection and feedback on project design.

We thank our co-authors on publications for the broader project that informed the approach to disease investigation reported in this paper: Shane Besier (Department of Primary Industries & Regional Development, Western Australia), Graeme Knowles (Department of Primary Industries, Parks, Water and Environment, Tasmania), Roger Paskin (OMNI Animal Health Consultancy, South Australia), Grant Rawlin (Agriculture Victoria, Department of Jobs, Precincts and Regions), Robert Suter (Agriculture Victoria, Department of Jobs, Precincts and Regions) Gavin Kearney (Biometrics Consultant, Hamilton, Victoria), Martina Jelocnik (University of the Sunshine Coast) and Sam Hair (Department of Primary Industries and Regional Development, WA). We thank Johann Schroder for helpful feedback on the manuscripts and project design.

Conflict of Interest

None of the authors of this paper have a financial or personal relationship with other people or organisations that could inappropriately influence or bias the content of the paper. The funding body (Meat and Livestock Australia) approved the manuscript for publication but was not involved in the collection, analysis or interpretation of data, or in the writing of the manuscript.

References

1. Trompf J, Young J, Bowen E: Review of National Sheep Reproduction and Lamb Survival Sheep Producers Australia and Animal Health Australia; Canberra, Australia: 2018: Available at: Accessed:
2. Australian Bureau of Agricultural and Resource Economics. About my region. <https://www.awe.gov.au/abares/research-topics/aboutmyregion>. 2022. Retrieved 1 November 2021 2021.
3. Thompson AN, Hamill B, King E, Scott M, Trompf J. Impacts of the Lifetime Ewe Management training program on the Australian sheep industry *Anim Prod Sci* 2020;61:clxxxvii. DOI: <https://www.publish.csiro.au/AN/pdf/ANv61n3abs>
4. Trompf JP, Gordon DJ, Behrendt R et al. Participation in Lifetime Ewe Management results in changes in stocking rate, ewe management and reproductive performance on commercial farms. *Anim Prod Sci* 2011;51:866-872. DOI: <http://dx.doi.org/10.1071/AN10164>
5. Young JM, Trompf J, Thompson AN. The critical control points for increasing reproductive performance can be used to inform research priorities. *Anim Prod Sci* 2014;54:645. DOI: 10.1071/AN13269
6. Bruce M, Young JM, Masters DG et al. The impact of lamb and ewe mortality associated with dystocia on Australian and New Zealand sheep farms: A systematic review, meta-analysis and bio-economic model *Prev Vet Med* 2021;196:105478. DOI: 10.1016/j.prevetmed.2021.105478
7. Clune T, Lockwood A, Hancock S et al. Abortion and lamb mortality between pregnancy scanning and lamb marking for maiden ewes in southern Australia. *Animals* 2022;12:10. DOI: <https://doi.org/10.3390/ani12010010>
8. Hinch GN, Brien F. Lamb survival in Australian flocks: a review. *Anim Prod Sci* 2014;54:656-666. DOI: 10.1071/AN13236
9. Refshauge G, Brien FD, Hinch GN, van de Ven R. Neonatal lamb mortality: factors associated with the death of Australian lambs. *Anim Prod Sci* 2016;56:726-735. DOI: <https://doi.org/10.1071/AN15121>
10. Jacobson C, Bruce M, Kenyon PR et al. A review of dystocia in sheep. *Small Rumin Res* 2020;192:106209. DOI: <https://doi.org/10.1016/j.smallrumres.2020.106209>
11. Hutchison D, Clarke BE, Hancock S et al. Lower reproductive rate and lamb survival contribute to lower lamb marking rate in maiden ewes compared to multiparous ewes. *Animals* 2022;12:513. DOI: 10.3390/ani12040513
12. Kleemann DO, Walker SK. Fertility in South Australian commercial Merino flocks: sources of reproductive wastage. *Theriogenology* 2005;63:2075-2088. DOI: 10.1016/j.theriogenology.2004.06.017
13. Allworth MB, Wrigley HA, Cowling A. Fetal and lamb losses from pregnancy scanning to lamb marking in commercial sheep flocks in southern New South Wales. *Anim Prod Sci* 2017;57:2060-2065. DOI: <https://doi.org/10.1071/AN16166>
14. Clune T, Beetson S, Besier S et al. Ovine abortion and stillbirth investigations in Australia. *Aust Vet J* 2021;99:72-78. DOI: <https://doi.org/10.1111/avj.13040>
15. Clune T, Beetson S, Besier S et al. Sheep abortion and stillbirth investigations at Australian veterinary laboratories. Australian Veterinary Association, 2021 Sheep, Camelid and Goat Veterinarians Conference, Wagga Wagga, 30 June - 2 July 2021 2021.
16. Clune T, Besier S, Hair S et al. Chlamydia pecorum detection in aborted and stillborn lambs from Western Australia. *Vet Res* 2021;52:84. DOI: 10.1186/s13567-021-00950-w
17. Westermann T, Jenkins C, Onizawa E et al. Chlamydia pecorum associated sporadic ovine abortion. *Vet Pathol* 2021;58:114-122. DOI: <https://doi.org/10.1177/0300985820967451>
18. Holst PJ: Lamb Autopsy: Notes on a procedure for determining cause of death. NSW Agriculture; Australia: 2004: Available at: https://www.dpi.nsw.gov.au/__data/assets/pdf_file/0006/177783/lamb-autopsy.pdf Accessed: 13 May 2022

Reproductive performance for maiden and multiparous ewes on Australian farms: Opportunities for veterinary practitioners to provide on-farm investigations and advice

Caroline Jacobson, Dayna Hutchison, Bronwyn E. Clarke, Serina Hancock, Elise Bowen and Andrew N. Thompson

Centre for Animal Production and Health, Murdoch University, Murdoch, WA, 6150

Summary

Suboptimal reproductive performance of maiden ewes impacts the productivity of sheep enterprises, but the extent and causes of poorer reproductive performance for maiden ewes are not well understood. Here we show the reproductive performance of maiden ewes relative to their multiparous counterparts on the same farms across Australia using a cohort survey. There was a 58% difference in marking rate for non-Merino ewe lambs compared to multiparous ewes, and this was attributable to a 50% difference in reproductive rate and 16% difference in lamb survival. For maiden Merino hogget ewes, there was a 22% difference in marking rate compared to multiparous ewes and this was attributable to a 24% difference in reproductive rate and 3% difference in lamb survival. Our findings show that veterinarians can add value to sheep enterprises by working with managers to assess reproductive rate and lamb survival in different ewe cohorts and compare current performance against industry benchmarks. This will inform interventions targeted to ewe age groups and stages of the reproductive cycle with the greatest opportunity to deliver improvement in farm profitability and animal welfare.

Key points for practitioners

- Marking rate is lower in maiden ewes compared to multiparous ewes
- Poorer marking rate in maiden ewes is related to poorer reproductive rate and poorer lamb survival
- Reproductive rate and lamb survival are more variable in ewe lambs compared to maiden hogget ewes
- Investigation of disappointing marking rates should differentiate losses attributable to reproductive rate (*i.e.* issues occurring joining to scanning) and lamb survival (*i.e.* foetal and lamb mortality) to prioritise interventions to improve ewe reproductive performance
- Ewe lambs may need different strategies to adult ewes to improve reproductive rate and lamb survival
- Wide variability in reproductive rate and lamb survival, plus a high proportion of enterprises with maiden ewe performance below industry targets, indicates considerable opportunity for practitioners to provide services to sheep enterprises to (a) identify key drivers of poor reproductive performance for maiden ewes, and (b) targeted advice of strategies to improve ewe reproductive performance

Introduction

Improving the reproductive performance of maiden (primiparous) ewes lambing for the first time as ewe lambs (at approximately 12 months of age) or maiden hoggets (at approximately 24 months of age) has been identified as a priority for the Australian sheep industry.¹ It is widely accepted that the reproductive performance of maiden ewes is generally poorer and more variable than multiparous ewes, but the 'gap' between maiden and multiparous ewes has not been well quantified. Improved understanding of the reproductive performance of maiden ewes will inform benchmarks for reproduction and strategies aimed at improving whole-flock reproductive performance and animal welfare.

Marking rate has increased in Australia by about 15% over the last 30 years.^{2, 3} This has been attributed to changes in flock structure and greater adoption of husbandry practices including management to optimise condition score.⁴ However, industry data for marking rates are based predominantly on multiparous and mixed-age ewes and do not differentiate between maidens and multiparous ewes.^{2, 3}

Marking rate is a function of reproductive rate and foetal/lamb survival between scanning and lamb marking. Studies conducted predominantly in New Zealand have demonstrated that ewe

lambs have lower fertility and ovulation rates, higher embryo loss between conception and scanning (resulting in poorer reproductive rate), and lower foetal/lamb survival between scanning and marking compared to multiparous ewes.⁵ Recent Australian studies have reported variable reproductive rate and marking rates in ewe lamb flocks across Australia, with an average reproductive rate of 108% and foetal/lamb mortality between scanning and marking exceeding 35%.^{6, 7} However, the reproductive performance of ewe lambs relative to multiparous ewes, and the relative contributions of reproductive rate and lamb survival to the poorer and more variable marking rates for ewe lambs remains poorly defined for Australian flocks.

Surprisingly, even less is known about typical differences in reproductive rate and lamb survival for maiden hogget ewes compared to multiparous ewes. A large South Australian study on 43 farms reported that reproductive rate was about 13% lower for maiden hogget ewes, and survival of both single and twin lambs was about 10% lower compared to multiparous ewes on the same farm.⁸ Other studies reported no differences in lamb survival between maiden hoggets and multiparous ewes on Australian farms.^{9, 10} Whilst differences in lamb survival between maiden hogget ewes and multiparous ewes may be less than observed between maiden ewe lambs and multiparous ewes, even small differences will impact national marking rates due the large number of maiden hogget ewes joined annually.

This study aimed to determine the difference in reproductive performance between maiden and multiparous ewes across major sheep producing regions of Australia to inform strategies to improve the reproductive performance of maiden ewes. We hypothesised that (i) maiden ewes joined either as ewe lambs or hoggets will have lower marking rates than multiparous ewes, and (ii) this will be due to a combination of lower reproductive rate and lower lamb survival between scanning and marking.

Methodology

The study was conducted according to the guidelines of the Australian Code for the Responsible Conduct of Research, and approved by the Murdoch University Human Research Ethics Committee (2018/056).

Survey methodology

The survey methodology is described in more detail by Hutchison, et al.¹¹ Briefly, sheep producers from Western Australia, South Australia, Victoria, New South Wales and Tasmania completed a questionnaire focused on reproductive performance for ewes that lambed between 2018 and 2020. The questionnaire included data recorded at pregnancy scanning and lamb marking for mobs of maiden and multiparous ewes, plus farm and flock characteristics.

Respondents were selected for inclusion in the survey on the basis that; (a) they managed ewe lambs (lambing at approximately one year old) or maiden hoggets (lambing at approximately two years old), (b) utilised pregnancy scanning by transabdominal ultrasonography to determine the number of foetuses for maiden and multiparous ewes, and (c) were able to determine lamb survival to marking for maiden and multiparous ewes on the same property, which generally required managing maiden ewes separately from multiparous ewes during lambing. The most common reasons for exclusion from study were: (a) the number of foetuses was not determined at pregnancy scanning for some or all of the maiden or multiparous ewes, and/or (b) maiden ewes were mixed with multiparous ewes during lambing and thus lamb survival to marking could not be determined separately for maiden and multiparous ewes.

Very few responses were received for Merino ewe lambs and non-Merino maiden hogget ewes during the first 12 months of the study, so these were subsequently excluded from the survey and only non-Merino ewe lambs and Merino hogget ewes were targeted thereafter.

Statistical analysis

Reproductive traits were determined as shown in Table 3. Reproductive traits for maiden and multiparous ewes were compared using two-tailed non-parametric related-samples Wilcoxon signed rank test. Correlations between reproductive traits for maiden and multiparous ewes on the same farm were determined using bivariate Pearson correlation (two-tailed) and linear regression. Association between the three reproductive performance traits and potential risk

factors with were analysed with linear mixed effects models described in more detail by Hutchison, et al. ¹¹

Table 3: Determination of reproductive traits

| Reproductive trait | Method of calculation |
|----------------------------|---|
| Reproductive rate (%) | Number of foetuses identified at pregnancy scanning expressed relative to the number of ewes scanned |
| Lamb survival (%) | Number of live lambs present at lamb marking expressed relative to the number of foetuses identified at scanning |
| Marking rate (%) | Number of live lambs present at marking expressed relative to the number of ewes joined |
| Pregnant ewe mortality (%) | Number of ewe deaths between pregnancy scanning and lamb marking expressed relative to the number of ewes that were pregnant at scanning (<i>i.e.</i> excludes ewes not pregnant at scanning). |

Results

Survey responses and characteristics of study flocks

Valid survey responses were received for 79 respondents. Of these, 16 producers contributed data for two years, and three producers contributed data for three years to give a total of 103 survey responses representing 111,117 maiden ewes managed in 307 mobs from lambing to marking, and 302,585 multiparous ewes (Table 4). Mean farm size was 3,750 hectares (range: 230 - 115,000 hectares) and the mean number of breeding ewes per farm was 4,762 (range: 477 - 25,000 ewes).

Table 4: Distribution of eligible survey responses by state based on number of respondents (farms) and total number of maiden and multiparous ewes joined between 2018 and 2020.

| State | Respondents (n) | | Ewes (n) | | | | |
|-------|----------------------|----------------|----------------------|----------------|-------------|---------|---------|
| | Non-Merino ewe lambs | Merino hoggets | Maiden | | Multiparous | | Total |
| | | | Non-Merino ewe lambs | Merino hoggets | Non-Merino | Merino | |
| WA | 3 | 11 | 2,945 | 14,904 | 9,580 | 37,305 | 64,734 |
| SA | 8 | 4 | 8,974 | 4,645 | 27,338 | 19,520 | 60,477 |
| NSW | 8 | 13 | 15,712 | 8,818 | 38,216 | 29,226 | 91,972 |
| VIC | 19 | 11 | 34,387 | 13,728 | 80,448 | 43,931 | 172,494 |
| TAS | 2 | 0 | 7,004 | 0 | 17,021 | 0 | 24,025 |
| Total | 40 | 39 | 69,022 | 42,095 | 172,603 | 129,982 | 413,702 |

The mean length of joining was 39 days (Table 5), with a mean age at joining of 8 months for non-Merino ewe lambs and 18.5 months for maiden Merino hogget ewes. Non-Merino ewe lambs included a range of breeds such as composite, Suffolk and Dorper that were joined with maternal or terminal rams. Differential management of single- and multiple-bearing ewes during lambing was used for ewe lambs by 47% respondents (66% ewes) and for maiden Merino hoggets by 57% respondents (61% ewes).

Condition score before lambing was reported by 75 respondents (Table 5), of which 31 (41%) responses were based on direct measurement, 35 (47%) were based on estimation and 9 (12%) did not specify the method used. Feed-on-offer during lambing was reported by only 8 respondents, of which three were based on measurement, four were estimated and one did not specify method used. Eighty six percent (n=68) of respondents reported having been trained in assessment of condition scoring and feed-on-offer.

Table 5: Management of maiden ewes based on eligible survey responses.

| | Ewe lamb flocks | | | Merino hogget flocks | | |
|-------------------------------------|-----------------|-------------|-------------|----------------------|-------------|------------|
| | n | Mean ± s.e | Range | n | Mean ± s.e | Range |
| Joining length (days) | 43 | 40 ± 1 | 28 - 60 | 36 | 38 ± 1 | 28 - 56 |
| Condition score | | | | | | |
| Mating | 47 | 3.15 ± 0.04 | 2.5 - 4.0 | 30 | 2.90 ± 0.06 | 2.0 - 3.7 |
| Lambing | 47 | 3.19 ± 0.05 | 2.7 - 4.0 | 28 | 3.02 ± 0.07 | 2.5 - 4.0 |
| Feed-on-offer at lambing (kg DM/Ha) | 6 | 1700 ± 93 | 1500 - 2000 | 2 | 750 ± 250 | 500 - 1000 |

s.e: standard error

DM/Ha: dry matter per hectare

Reproductive performance in maiden and mature ewes

Mean marking rate, reproductive rate, lamb survival and pregnant ewe mortality are shown in Table 6. Maiden ewes had a lower marking rate than multiparous ewes on the same farm and this was attributable to both lower reproductive rate and lower lamb survival for both ewe lambs and Merino hoggets.

Lamb survival was considerably more variable for non-Merino ewe lambs compared to multiparous ewes on the same farm. Lamb survival was relatively more important as a contributor to poorer marking rate for non-Merino ewe lambs than for maiden Merino hoggets. Notwithstanding this, the range for lamb survival reported for top 25% ewe lambs (farm level) was comparable to that for maiden Merino hoggets and multiparous ewes of Merino or non-Merino breed (Table 6).

Table 6: Mean and range for top and bottom quartile respondents for reproductive traits (reproductive rate, marking rate and lamb survival) reported in maiden and multiparous ewes.

| | Ewe age group | | | | | | Age group comparison ^a | |
|-----------------------------|---------------|--------------|-----------|-------------|--------------|-----------|-----------------------------------|---------|
| | Maidens | | | Multiparous | | | | |
| | Bottom 25% | Mean | Top 25% | Bottom 25% | Mean | Top 25% | Difference | p-value |
| Non-Merino ewe lambs | | | | | | | | |
| Marking rate (%) | 24 - 62 | 73.8 | 92 - 119 | 101 - 124 | 131.9 | 140 - 159 | -58.1 | <0.001 |
| Reproductive rate (%) | 61 - 94 | 108.6 | 129 - 149 | 128 - 151 | 159 | 167 - 187 | -50.5 | <0.001 |
| Lamb survival (%) | 14 - 59 | 67.3 | 89 - 99 | 75 - 82 | 83.4 | 87 - 96 | -16.0 | <0.001 |
| Ewe mortality (%) | 3.2 - 5.4 | 2.6 | 0 - 1.5 | 3.4 - 5.3 | 2.8 | 0 - 1.7 | -0.2 | 0.378 |
| Merino hogget ewes | | | | | | | | |
| Marking rate (%) | 56 - 71 | 80.1 | 92 - 110 | 70 - 93 | 102.3 | 112 - 136 | -22.3 | <0.001 |
| Reproductive rate (%) | 83 - 96 | 107.6 | 118 - 144 | 96 - 118 | 131.9 | 146 - 165 | -24.4 | <0.001 |
| Lamb survival (%) | 52 - 69 | 74.5 | 82 - 98 | 61 - 73 | 77.7 | 83 - 97 | -3.4 | 0.026 |
| Ewe mortality (%) | 2.4 - 4.0 | 1.7 | 0.1 - 0.8 | 0 - 1.0 | 2.4 | 2.6 - 4.8 | -0.7 | 0.006 |

^a Non-parametric related-samples Wilcoxon signed rank test**Comparison of reported lamb survival against industry targets**

Lamb survival for single- and twin-bearing maiden ewes relative to industry targets are shown in

Table 7. Mean twin-lamb survival was similar for non-Merino ewe lambs (64%) and Merino hoggets (61%). None of the respondents reported lamb survival for ewe lambs exceeding industry targets for single- or twin-bearing ewes (

Table 7). For maiden Merino hoggets, 20% of respondents reported lamb survival above the industry target for single-bearing ewes and 9% respondents reported lamb survival above the target for twin-bearing ewes (

Table 7).

Table 7: Lamb survival for single- and twin-bearing maiden ewes and proportion of respondents reporting lamb survival above industry targets

| | Non-Merino ewe lambs | | Merino hoggets | |
|-----------------------------------|----------------------|---------------|----------------|---------------|
| | Single | Twin | Single | Twin |
| Respondents (n) | 20 | 22 | 25 | 23 |
| Mean lamb survival (%) | 76% | 64% | 82% | 61% |
| Industry target (%) | 90% | 80% | 90% | 70% |
| Survival ≥50% (n (% respondents)) | 20 (100%) | 21 (95%) | 25 (100%) | 20 (87%) |
| Survival ≥60% (n (% respondents)) | 20 (100%) | 15 (68%) | 24 (96%) | 15 (65%) |
| Survival ≥70% (n (% respondents)) | 16 (80%) | 6 (27%) | 23 (92%) | 2 (9%) |
| Survival ≥80% (n (% respondents)) | 6 (30%) | 0 (0%) | 17 (68%) | 0 (0%) |
| Survival ≥90% (n (% respondents)) | 0 (0%) | 0 (0%) | 5 (20%) | 0 (0%) |

Correlation between maiden and multiparous ewes on same farm for reproductive traits

The correlation between maiden and multiparous ewes for reproductive traits are shown in Table 8. There were moderate positive correlations between maiden Merino hoggets and their multiparous counterparts for all four traits. In contrast, there was a very weak positive correlation between ewe lambs and their multiparous counterparts for lamb survival but no correlation for reproductive rate, marking rate or ewe survival.

Table 8: Bivariate Pearson correlation (two-tailed) between reproductive traits in maiden ewes and corresponding measure for multiparous counterparts on the same farm

| | Correlation coefficient | p-value |
|---|-------------------------|---------|
| Non-Merino ewe lambs vs multiparous ewes | | |
| Marking rate (%) | 0.223 | 0.096 |
| Reproductive rate (%) | -0.032 | 0.814 |
| Lamb survival (%) | 0.280 | 0.035 |
| Ewe mortality (%) | 0.180 | 0.222 |
| Merino hogget ewes vs multiparous ewes | | |
| Marking rate (%) | 0.541 | <0.001 |
| Reproductive rate (%) | 0.652 | <0.001 |
| Lamb survival (%) | 0.635 | <0.001 |
| Ewe mortality (%) | 0.659 | <0.001 |

Factors associated with reproductive performance of maiden ewes

Of the factors investigated for association with reproductive traits, only birth-type was associated with higher marking rates. Specifically, marking rates were higher for twin- and triplet-bearing ewes compared with single-bearing ewes (Table 9). This was due to greater number of fetuses in these mobs and not higher lamb survival, with survival of twin- and triplet-born lambs lower than that for single-born lambs (Table 9). Reproductive traits were independent of ABARE region, ewe age, ewe breed, ram breed, month of mating, condition score at mating, feed-on-offer at mating, pasture type at mating, condition score at lambing, duration of supplementary feeding, supplementary feeding method, type of supplementary feed or average feed-on-offer ($p < 0.05$)

Table 9: Predicted means \pm standard error for the marking (%) and lamb survival (%) according to ewe management group during lambing for non-Merino ewe lambs and maiden Merino two-tooth ewes.

| Management group during lambing | Marking % | | Lamb survival % | |
|---------------------------------|-----------------|-----------------|-----------------|----------------|
| | Ewe lambs | Two-tooth ewes | Ewe lambs | Two-tooth ewes |
| Mixed | 97.3 \pm 2.9 | 91.4 \pm 5.0 | NA | NA |
| Single | 75.2 \pm 2.3 | 83.3 \pm 3.3 | 76.0 \pm 1.6 | 82.7 \pm 1.8 |
| Twin | 129.8 \pm 2.3 | 122.0 \pm 3.6 | 64.6 \pm 1.6 | 60.5 \pm 2.0 |
| Triplet | 146.0 \pm 7.9 | NA | 43.7 \pm 3.9 | NA |

NA: not available (not measured or no eligible responses)

Discussion

The difference in marking rate between maiden and multiparous ewes was 58% lower for non-Merino ewe lambs and 22% lower for maiden Merino hoggets. Lower marking rates for maiden ewes were attributable, albeit to varying degrees, to both lower reproductive rate and lamb survival. Management strategies to improve marking rate for non-Merino ewe lambs should focus on

improving both reproductive rate and lamb survival as they contributed about equally to the differences in marking rate compared to multiparous ewes. The poorer marking rate of Merino hoggets was largely due to differences in reproductive rate compared to multiparous ewes. Nevertheless, the strategies to improve marking rate for Merino hoggets should address both reproductive rate and lamb survival because lamb survival was variable in both maiden and multiparous ewes. This highlighted that improving lamb survival for Merino ewes across all age groups remains an issue for the Australian sheep industry. The economic value of improving reproductive rate in Merino enterprises is greater when lamb survival is higher¹ and improving lamb survival has implications for improving animal welfare.

Over the last decade more than 5,000 sheep producers in Australia have participated in extension and adoption programs to improve ewe management and reproductive performance, such as Lifetime Ewe Management^{4, 12} and Bred Well Fed Well¹³, but these programs have focused on management of multiparous ewes. Similar extension and adoption programs to improve the reproductive performance of maiden ewes could have a significant impact on national marking rates and lamb supply. Non-Merino ewe lambs and Merino hogget ewes represent about 10 million breeding ewes in Australia.³ This represents substantial opportunity for veterinary practitioners to provide services to sheep enterprises to determine causes of reproductive inefficiency and inform targeted interventions to improve both productivity of sheep enterprises and animal welfare.

The weak and generally non-significant correlation between the reproductive performance of ewe lambs and their multiparous counterparts hinged on more variable performance of ewe lambs. In contrast, reproductive performance of maiden Merino hogget ewes was more consistent when compared with multiparous ewes. A number of studies have demonstrated that ewe lambs have a greater reproductive rate response to higher joining liveweight^{6, 14} and liveweight gain during the joining¹⁵⁻¹⁷ compared to older ewes. Age of mating also impacts the reproductive rate of ewe lambs⁶, although this was not apparent in the current survey data where almost 90% of the ewe lambs were 7 or 8 months of age at mating. Collectively, this implies that reproductive rate of ewe lambs is more sensitive to management before and during mating than is the case for multiparous ewes. Heritabilities for reproductive traits typically range from 5-15%¹⁸⁻²¹ indicating that whilst progress in reproductive traits can be made with genetic selection, reproductive performance is driven mostly by management.

The precise reasons for poorer and more variable survival of lambs born to maiden ewes, especially ewe lambs, compared with their multiparous counterparts were not able to be determined in this study. Maiden ewes were managed separately to multiparous ewes during pregnancy and lambing, and differences in nutrition or the lambing environment such as time of lambing, shelter and mob size could have been impacting lamb survival. Whilst the level of nutrition and liveweight change from pregnancy scanning to lambing is likely to influence the survival of lambs born to ewe lambs²², the average condition score of ewe lambs in the current study was reported to be 3.2 at lambing which is likely to be close to the optimum. It may therefore be that factors other than nutritional management during pregnancy and lambing contributed to the relatively large difference in survival of lambs between ewe lambs and multiparous ewes.

There are a number of factors that may contribute to foetal and lamb mortality between scanning and marking. Important causes of perinatal lamb mortality include dystocia, stillbirths and starvation-mismothering, but these conditions are usually multifactorial and the role of dam parity is not fully understood.²³⁻²⁶ Clune, et al.⁷ reported that lamb mortality between birth and marking was the major contributor to lamb mortality between scanning and marking for Australian maiden ewe flocks, but mid-pregnancy abortion caused significant *in utero* foetal loss in some flocks of ewe lambs. Immunological naïvety is considered a risk factor for infectious reproductive diseases and therefore maiden ewes have a higher risk for infectious causes of abortion because they have had less time to be exposed to infection and develop immunity before pregnancy.²⁷⁻³⁰ Sporadic abortion and perinatal lamb mortality due to infectious disease may explain some variability in lamb survival for maiden ewe flocks, and especially ewe lambs. Practitioners can contribute to investigation of poor reproductive performance through lamb necropsies to determine cause of death based on gross post mortem³¹ and submission of appropriate samples to determine the likely contribution of infectious disease.³²⁻³⁴

Only enterprises that utilised pregnancy scanning for litter size were eligible for inclusion in this study. Subsequently, the sampled population likely included a higher proportion of ewes that were differentially managed according to litter size compared to the general population. Differential management of single- and multiple bearing ewes was used for 66% maiden non-Merino ewe lambs and 61% maiden Merino two-tooth ewes which was consistent with adoption rate for producers that had undertaken Lifetime Ewe Management training. It is possible that respondents that have already adopted pregnancy scanning were more likely to adopt other management strategies that could impact reproductive performance compared to the broader population. As such, the findings of this study should only be generalised to Australian sheep producers that have adopted pregnancy scanning and should not be extrapolated across the national sheep flock.

Conclusion

Marking rates for non-Merino ewe lambs and maiden Merino hogget ewes were lower than their multiparous counterparts on the same farm. A combination of lower reproductive rate and lower lamb survival contribute to lower marking rates in maiden ewes. Wide variability in both reproductive rate and lamb survival indicates opportunity for improvement in reproductive performance for maiden ewes through adoption of strategies that increase the number of fetuses conceived, and the number of lambs surviving between pregnancy scanning and lamb marking. Strategies specific to ewe lambs may be required because their reproductive performance was not correlated with multiparous ewes on the same farm.

Acknowledgments

This research was funded by Meat and Livestock Australia (B.AHE.0318). Elise Bowen was supported with a scholarship from Sheep Industry Business Innovation (Department of Primary Industry and Regional Development, Western Australia).

We thank the sheep producers that provided responses for this study and feedback on the survey design. We thank participating consultants and service providers for providing assistance sourcing farmers eligible for inclusion in the study including Ken Solly (Solly Business Services), Meg Bell (MacKillop Group), Genstock Kojonup, Leanne Sherriff (Pinion Advisory), Bruce Watt (NSW Local Land Services), Mick O'Neill (Elders), Rex Luers (Nutrien) Pip Houghton (Thomas Elder Consulting), Colin Trengove (University of Adelaide), Gordon Refshauge (NSW DPI), Jim Gordon and Geoff Duddy (Sheep Solutions). We thank Maeve O'Brien for assistance collecting survey responses and data entry. We thank Andrew van Burgel for advice on the data collation and analyses. We thank Henry Annandale and David Miller for helpful feedback on an honours thesis describing this work. Changes made in response to their feedback was incorporated into the subsequent manuscript published in *Animals* and this article.

Conflict of Interest

None of the authors of this paper have a financial or personal relationship with other people or organisations that could inappropriately influence or bias the content of the paper. The funding body (Meat and Livestock Australia) approved the manuscript for publication but was not involved in the collection, analysis or interpretation of data, or in the writing of the manuscript.

References

1. Young JM, Trompf J, Thompson AN. The critical control points for increasing reproductive performance can be used to inform research priorities. *Anim Prod Sci* 2014;54:645. DOI: 10.1071/AN13269
2. Australian Bureau of Agricultural and Resource Economics. About my region. <https://www.awe.gov.au/abares/research-topics/aboutmyregion>. 2022. Retrieved 1 November 2021 2021.
3. Trompf J, Young J, Bowen E: Review of National Sheep Reproduction and Lamb Survival Sheep Producers Australia and Animal Health Australia; Canberra, Australia: 2018: Available at: Accessed:
4. Trompf JP, Gordon DJ, Behrendt R et al. Participation in Lifetime Ewe Management results in changes in stocking rate, ewe management and reproductive performance on commercial farms. *Anim Prod Sci* 2011;51:866-872. DOI: <http://dx.doi.org/10.1071/AN10164>
5. Kenyon PR, Thompson AN, Morris ST. Breeding ewe lambs successfully to improve lifetime performance. *Small Rumin Res* 2014;118:2-15. DOI: 10.1016/j.smallrumres.2013.12.022
6. Thompson AN, Bowen E, Keiller J et al. The number of offspring weaned from ewe lambs is affected differently by liveweight and age at breeding. *Animals* 2021;11:2733

7. Clune T, Lockwood A, Hancock S et al. Abortion and lamb mortality between pregnancy scanning and lamb marking for maiden ewes in southern Australia. *Animals* 2022;12:10. DOI: <https://doi.org/10.3390/ani12010010>
8. Kleemann DO, Walker SK. Fertility in South Australian commercial Merino flocks: sources of reproductive wastage. *Theriogenology* 2005;63:2075-2088. DOI: 10.1016/j.theriogenology.2004.06.017
9. Paganoni BL, Ferguson MB, Kearney GA, Thompson AN. Increasing weight gain during pregnancy results in similar increases in lamb birthweights and weaning weights in Merino and non-Merino ewes regardless of sire type. *Anim Prod Sci* 2014;54:727. DOI: 10.1071/AN13263
10. Lockwood AL, Hancock SN, Trompf JP et al. Data from commercial sheep producers shows that lambing ewes in larger mobs and at higher stocking rates reduces the survival of their lambs. *N Z J Agric Res* 2020;63:246-259. DOI: 10.1080/00288233.2019.1570945
11. Hutchison D, Clarke BE, Hancock S et al. Lower reproductive rate and lamb survival contribute to lower lamb marking rate in maiden ewes compared to multiparous ewes. *Animals* 2022;12:513. DOI: 10.3390/ani12040513
12. Thompson AN, Hamill B, King E, Scott M, Trompf J. Impacts of the Lifetime Ewe Management training program on the Australian sheep industry *Anim Prod Sci* 2020;61:clxxxvii. DOI: <https://www.publish.csiro.au/AN/pdf/ANv61n3abs>
13. Hancock S, Blumer S, Trompf J. Bred Well Fed Well: one day practical workshop delivers behavioural change and improved marking percentage in Australia. The International Symposium on the Nutrition of Herbivores, Clermont-Ferrand, France, 2-6 September 2018 2018.
14. Lindsay D, Knight T, Smith J, Oldham C. Studies in ovine fertility in agricultural regions of Western Australia : ovulation rate, fertility and lambing performance. *Aust J Agric Res* 1975;26:189-198. DOI: <https://doi.org/10.1071/AR9750189>
15. Adalsteinsson S. The independent effects of live weight and body condition on fecundity and productivity of Icelandic ewes. *Animal Science* 1979;28:13-23. DOI: 10.1017/S0003356100023011
16. Viñoles C, Glover KMM, Paganoni BL, Milton JTB, Martin GB. Embryo losses in sheep during short-term nutritional supplementation. *Reprod Fertil Dev* 2012;24:1040. DOI: 10.1071/RD11281
17. Thompson A, Bairstow C, Ferguson M et al. Growth pattern to the end of the mating period influences the reproductive performance of merino ewe lambs mated at 7 to 8 months of age. *Small Rumin Res* 2019;179:1-6. DOI: <https://doi.org/10.1016/j.smallrumres.2019.08.007>
18. Bunter KL, Brown DJ. Yearling and adult expressions of reproduction in maternal sheep breeds are genetically different traits. *Proceedings of the Association for Advancement in Animal Breeding and Genetics* 2013;20:82-85
19. Hatcher S, Atkins KD, Safari E. Lamb survival in Australian Merino Sheep: A genetic analysis. *J Anim Sci* 2010;88:3198-3205. DOI: 10.2527/jas.2009-2461
20. Everett-Hincks JM, Mathias-Davis HC, Greer GJ, Auvray BA, Dodds KG. Genetic parameters for lamb birth weight, survival and death risk traits1. *J Anim Sci* 2014;92:2885-2895. DOI: 10.2527/jas.2013-7176
21. Brien FD, Cloete SWP, Fogarty NM et al. A review of the genetic and epigenetic factors affecting lamb survival. *Anim Prod Sci* 2014;54:667-693
22. Griffiths KJ, Ridler AL, Heuer C, Corner-Thomas RA, Kenyon PR. The effect of liveweight and body condition score on the ability of ewe lambs to successfully rear their offspring. *Small Rumin Res* 2016;145:130-135. DOI: <https://doi.org/10.1016/j.smallrumres.2016.11.001>
23. Horton BJ, Corkrey R, Hinch GN. Estimation of risk factors associated with difficult birth in ewes. *Anim Prod Sci* 2018;58:1125-1132. DOI: <https://doi.org/10.1071/AN16339>
24. Hinch GN, Brien F. Lamb survival in Australian flocks: a review. *Anim Prod Sci* 2014;54:656-666. DOI: 10.1071/AN13236
25. Jacobson C, Bruce M, Kenyon PR et al. A review of dystocia in sheep. *Small Rumin Res* 2020;192:106209. DOI: <https://doi.org/10.1016/j.smallrumres.2020.106209>
26. Refshauge G, Brien FD, Hinch GN, van de Ven R. Neonatal lamb mortality: factors associated with the death of Australian lambs. *Anim Prod Sci* 2016;56:726-735. DOI: <https://doi.org/10.1071/AN15121>
27. Buxton D. Ovine toxoplasmosis: a review. *J R Soc Med* 1990;83:509
28. Katzer F, Brülisauer F, Collantes-Fernández E et al. Increased *Toxoplasma gondii* positivity relative to age in 125 Scottish sheep flocks; evidence of frequent acquired infection. *Vet Res* 2011;42:121-121. DOI: 10.1186/1297-9716-42-121
29. Jensen R, Miller VA, Hammarlund MA, Graham WR. Vibronic abortion in sheep. I. Transmission and immunity. *Am J Vet Res* 1957;18:326-329
30. Berri M, Souriau A, Crosby M et al. Relationships between the shedding of *Coxiella burnetii*, clinical signs and serological responses of 34 sheep. *The Veterinary Record* 2001;148:502-505. DOI: 10.1136/vr.148.16.502

31. Holst PJ: Lamb Autopsy: Notes on a procedure for determining cause of death. NSW Agriculture; Australia: 2004: Available at: https://www.dpi.nsw.gov.au/_data/assets/pdf_file/0006/177783/lamb-autopsy.pdf Accessed: 13 May 2022
32. Clune T, Beetson S, Besier S et al. Ovine abortion and stillbirth investigations in Australia. *Aust Vet J* 2021;99:72-78. DOI: <https://doi.org/10.1111/avj.13040>
33. Clune T, Besier S, Hair S et al. *Chlamydia pecorum* detection in aborted and stillborn lambs from Western Australia. *Vet Res* 2021;52:84. DOI: 10.1186/s13567-021-00950-w
34. Westermann T, Jenkins C, Onizawa E et al. *Chlamydia pecorum* associated sporadic ovine abortion. *Vet Pathol* 2021;58:114–122. DOI: <https://doi.org/10.1177/0300985820967451>

Certification programs available to Australian woolgrowers

Drs Bea Kirk & John Webb Ware
Mackinnon Project
Faculty of Veterinary and Agricultural Science, University of Melbourne
Werribee Victoria

Introduction

There are now several certification programs available to Australian woolgrowers which may enable them to receive a premium for at least part of their wool clip. The details of these programs vary, but all have a list of practices required to be carried out, or prohibited, on farms. However, some confusion exists amongst producers as to how the premiums received work, and whether they are likely to result in a profit. To attempt to resolve this, the details of four of the main wool certification programs currently available to wool producers (Responsible Wool Standards; ZQ; SustainaWOOL and Authentico) were evaluated, and a sensitivity analysis was conducted to examine the potential profit received by woolgrowers.

The sensitivity analysis

A sensitivity analysis was conducted using a model wool clip from a typical wool-producing enterprise, assuming that the farm already complied with the program requirements. The variables examined were the proportion of the clip attracting a premium, and the percentage premium (above the price for non-certified wool) received. The cost of certification for each program, and the cost of the producer's time to complete the certification process were also included in calculations.

The model flock in this analysis consisted of 2000 ewes cutting 5 kg greasy wool of 18 micron; 1800 weaners cutting 3.5 kg greasy wool of 17 micron; and 1000 wethers cutting 5.5 kg greasy wool of 18 micron. A 70% yield was assumed; and this was multiplied by 0.95 to obtain a 'sweep the board' wool price for each class of sheep. Wool prices of 2000 c/kg clean fleece weight (CFW) for 18 micron wool, and 2378 c/kg CFW for 17 micron wool, were used (November 2021)¹. The proportion of the clip that received a premium ranged from 20-80%, (assuming that oddments are unlikely to receive a premium).

For the purposes of this analysis, it was assumed that the certification and auditing process would take between one and five days (8-40 hours) of the producer's time, depending on the specific program, at a labour cost of \$40/hour. These analyses were conducted without a broker levy or wool handling costs being applied to the gross income, because of the variability of these figures. It was assumed that the wool was sold under one certification, though in reality wool may be sold as certified under more than one program.

Quantifying cost and benefit

Gaining certification does not automatically guarantee a premium on wool price: the premium depends on the wool's specifications, including length, micron, staple strength and vegetable matter (VM). For example, fleeces of 38N/kTex staple strength, with low VM are preferred². Not all wool in a clip would meet buyer specifications, so producers may not receive a premium for their whole clip (this is particularly the case with oddments, for example). The proportion of the clip receiving a premium varies depending on buyer requirements on the day of sale; on some occasions, most of the clip may receive a premium, but this is not always the case. Hence, a range of proportions of wool receiving a premium varied from 20-80% in our analysis.

The premiums generally vary in percentage for different microns³. The eventual additional wool income will depend on the particular wool broker and any fee involved with certification, including whether there is a percentage levy or a flat fee associated with the program. Therefore, because additional income is also dependent on the proportion of the clip that would meet specifications, and the degree of premium involved, it is worthwhile looking at a range of scenarios.

Part of what is driving the premium is the small percentage of certified wool offered for sale (3% of Merino wool sold in the 2021-22 wool selling season to mid-March has been RWS accredited⁴). This, along with the demand from buyers, will likely result in a premium, but this can be difficult to quantify, because of the many variables involved, not least the different certification programs available. The Chinese ban on South African wool that commenced on April 1, 2022, due to the Foot and Mouth disease outbreak in that country may also lead to increased premiums in the short to medium term⁵.

As well as the wool price premium, another benefit of certification is the larger range of sale opportunities it brings, including forward selling at higher than median prices; or direct sales rather than via auction. These sorts of sales have a number of benefits, including increased certainty for budgeting and satisfying bank requirements, as well as the potential for better prices. These benefits were not considered in this analysis because of their variability but should be part of the decision-making process.

Results

Table 1. Additional income (\$) with individual RWS certification at a cost of \$3,500 with different proportions of the clip attracting a premium, and premiums ranging between 5 and 20%.

| Premium | Proportion of clip receiving a premium | | | |
|---------|--|-----------|-----------|-----------|
| | 20% | 40% | 60% | 80% |
| 5% | -\$ 2,042 | \$ 1,016 | \$ 4,073 | \$ 7,131 |
| 10% | \$ 1,016 | \$ 7,131 | \$ 13,247 | \$ 19,362 |
| 15% | \$ 4,073 | \$ 13,247 | \$ 22,420 | \$ 31,593 |
| 20% | \$ 7,131 | \$ 19,362 | \$ 31,593 | \$ 43,824 |

Table 1 shows the results of a sensitivity analysis conducted using a range of premiums, between 5 and 20%, with a range of proportions of wool attracting a premium for RWS certification. Individual certification cost ranges between \$1,500 and \$5,500. For this analysis it was assumed that individual, rather than group certification was undertaken, for a cost of \$3,500 per annum, and that the process took 40 hours of producers' time. With the current average quoted premium of at least 10%, this certification provides good returns especially once 40% of the clip attracts the premium. Furthermore, the cost of certification is comparatively less important with a larger clip. It is worth noting that if group membership was undertaken through a broker, these figures may look better again, though this would depend on the broker levy applied. However, this table shows that individual certification may still be a cost-effective option for producers wishing to pursue this option.

Unsurprisingly, the premium received for accredited wool will be affected by supply and demand. In November 2021, comparatively greater premiums (in terms of percentage) were paid for some wool, particularly the broader Merino microns³. For example, on November 9 2021, 17.5 micron wool achieved a premium of just under 250c/kg clean, 18 micron received a just over 100c/kg clean, and 20 micron wool received a premium of 230c/kg clean. However, with such a small percentage of wool, particularly broader wool, being RWS accredited in Australia, it is difficult to draw conclusions³. Furthermore, with an increasing amount of wool being sold with RWS accreditation, these premiums may be eroded in the future due to increased supply³. The premiums received will also depend on the demand for this wool, and hence whether buyers are willing to pay premiums for certified wool².

It is worth noting that there is a shortage of certified Australian farms producing wool of 19 μ and higher, which would influence price; for example, in November South African sales data provided by Independent Commodity Services, there was a 17.7% premium for 19 μ wool, and this increased as the fleeces broadened, up to a 24.4% premium for 21 μ wool⁶.

Table 2. Additional income (\$) with ZQ certification with different proportions of the clip attracting a premium, and premiums ranging between 5 and 20%. Likely premiums are boxed.

| Premium | Proportion of clip receiving a premium | | | |
|---------|--|-----------|-----------|-----------|
| | 20% | 40% | 60% | 80% |
| 5% | -\$ 9,829 | -\$ 6,893 | -\$ 3,958 | -\$ 1,023 |
| 10% | -\$ 6,893 | -\$ 1,023 | \$ 4,848 | \$ 10,719 |
| 15% | -\$ 3,958 | \$ 4,848 | \$ 13,655 | \$ 22,461 |
| 20% | -\$ 1,023 | \$ 10,719 | \$ 22,461 | \$ 34,203 |

Note, 100% of the clip will be eligible to attract any RWS premiums available. Growers also have the option to forward contract any qualifying volumes according to their preference. These contracts can be between one and ten years forward.

Table 2 shows that with ZQ certification, at least 40% of the clip must receive a premium of at least 10%, for a profit to be made. However, given the structure of ZQ, it is likely that these minimum requirements will be met (the likely figures are boxed in a thick border), particularly because any wool not placed through ZQ will be eligible to attract an RWS premium. Furthermore, much of the strength of the program lies in its forward contract nature, which is not evaluated here.

Table 3. Additional income (\$) with SustainaWOOL® GREEN certification with different proportions of the clip attracting a premium, and premiums ranging between 1 and 3.5%.

| Premium | Proportion of clip receiving a premium | | | |
|---------|--|----------|----------|----------|
| | 20% | 40% | 60% | 80% |
| 1% | \$ 142 | \$ 753 | \$ 1,365 | \$ 1,976 |
| 1.50% | \$ 447 | \$ 1,365 | \$ 2,282 | \$ 3,199 |
| 2% | \$ 753 | \$ 1,976 | \$ 3,199 | \$ 4,422 |
| 2.50% | \$ 1,059 | \$ 2,588 | \$ 4,117 | \$ 5,646 |
| 3% | \$ 1,365 | \$ 3,199 | \$ 5,034 | \$ 6,869 |
| 3.50% | \$ 1,670 | \$ 3,811 | \$ 5,951 | \$ 8,092 |

A sensitivity analysis was conducted for SustainaWOOL® GREEN level membership (Table 3), assuming eight hours were spent on the process. This showed less risk compared to some other programmes. Some profit can still be made at the current quoted premium of 2-3%, even if only small proportions of the clip attract this premium, owing to the very modest fee involved; however, there is also a lower potential for financial benefit when compared to RWS. SustainaWOOL® BLUE membership is the only certification program which allows mulesing; the premiums here can be expected to be less than the figures in this table.

Table 4. Additional income (\$) with Authentico certification with different proportions of the clip attracting a premium, and premiums ranging between 1 and 5%.

| Premium | Proportion of clip receiving a premium | | | |
|---------|--|----------|----------|-----------|
| | 20% | 40% | 60% | 80% |
| 1% | \$ 292 | \$ 903 | \$ 1,515 | \$ 2,126 |
| 2% | \$ 903 | \$ 2,126 | \$ 3,349 | \$ 4,572 |
| 5% | \$ 2,738 | \$ 5,796 | \$ 8,853 | \$ 11,911 |

It was difficult to assign a premium to Authentico certification because of the many variables involved. However, assuming a very modest premium, as seen in Table 4, the financial benefit from Authentico was less than the other certification programmes examined; returns would obviously increase if true premiums were higher. Eight hours were assumed to be spent on certification, because this qualification may be gained in addition to another certification, making the process quicker. The benefit of this programme is that there is little risk involved in joining because of the

lack of application fee; therefore, it could be a good 'add on' for wool producers already doing all the practices required.

Intangibles

Though the profit for some of these certifications can look attractive in some scenarios, the reality is that may not be right for every producer.

One of the most prominent requirements of most of these types of certification (the exception being SustainaWOOL® Blue) is the commitment to a non-mulesed (or ceased-mulesed) status. There are a number of other criteria involved in certification, and these may not be possible to be met. The certification and auditing process also requires farm practices to be documented, and this must be kept in mind when making the decision.

Given that there may be a reasonable investment of time and effort involved with the certification process, even if the certification criteria are being met, not all producers may have the time or inclination to pursue this. Nevertheless, the option should be explored by woolgrowers who currently fit, or could fit, the criteria, and produce wool meeting buyer specifications which is likely to attract a premium.

Conclusion:

Certification programmes may be highly profitable for wool growers and can generate positive returns at current premiums particularly if a larger proportion of the clip attracts a premium, or the cost of certification is low or zero. Lower cost programmes generally carried less risk of producing a loss, but maximum profits were also lower. Overall, the likely profit was variable depending on the proportion of the clip attracting a premium, the percentage premium offered and the fee structure of the certification programme.

Growers should investigate whether the wool they have produced in the past is likely to attract a premium, and the proportion likely to do so. Furthermore, the possibility of forward contracts should be investigated. A role exists for veterinary consultants in assisting clients with conducting these analyses as well as helping clients meet the animal welfare requirements of such programmes.

References:

1. Innovation AW. Weekly price report - Week 18, 2021/22: Australian Wool Innovation; 2022 [Available from: <https://www.wool.com/market-intelligence/weekly-price-reports/20212022/week-18-november-2021/>].
2. Woods A. Premiums on RWS certified wool in Australia. In: Kirk B, editor. 2021.
3. Woods A. RWS premiums in absolute terms 2021 26/4/22. Available from: <https://mecardo.com.au/rws-premiums-in-absolute-terms/>.
4. Woods A. Quality systems volume and premiums. Wool and Fibres [Internet]. 2022 26/4/22. Available from: <https://mecardo.com.au/quality-systems-volume-and-premiums/>.
5. Myers D. Foot & mouth outbreak in South Africa. Wool and Fibres [Internet]. 2022 26/4/2022. Available from: <https://mecardo.com.au/foot-mouth-outbreak-in-south-africa/>.
6. Woods A. RWS premiums in the merino market 2021 3/5/22. Available from: <https://mecardo.com.au/rws-premiums-in-the-merino-market/>.

Appropriate analgesia saves sheep with burnt feet on Kangaroo Island

Debra Lehmann BVMS
Kangaroo Island Veterinary Clinic
252 Playford Highway, Kingscote SA

Summary

I am presenting a study of my assessment, palliative treatments and nursing care of sheep with burnt feet, sloughed hooves and burns to the groin and udder from a Merino stud on Kangaroo Island (KI). We minimised suffering with pain relief¹ and provided an environment to promote healing over the 6-week hospitalisation period such that 973 sheep that could have been destroyed on welfare grounds were saved. Most sheep saved were ewes which allowed the preservation and perpetuation of elite fine wool genetics in this flock as well as continued income from wool and sale of excess hogget rams. I would like the information presented to be added to a database of similar treatments to assist assessors of burnt livestock on other Australian farms in the future to reduce the number of burnt sheep hastily euthanized in the early days after fires^{2,3}.

History

Most farms on the western end of KI were severely impacted by a large out of control bushfire on the evening of 3rd January 2020. The fire had started over a week before in the northwest corner of Flinders Chase National Park. Catastrophic fire conditions led to the development of a massive fire front that swept through native vegetation and Blue Gum plantations then exploded onto neighbouring farms with little or no warning. The fire caused destruction of almost 100 farms, their homes and associated farm buildings, fodder and fences. No homes had been lost in any fires on KI prior to this one. Two people lost their lives.

This highly productive fine wool merino flock with about 50% DSE's as dry wethers and weaners thrives most years on 485ha of improved sub-clover pasture and other annuals with an average annual rainfall of 720mm. A lot of the farm has kikuyu included in the pasture mix now which responds readily to any summer rain. The flock has elite genetics and has been full pedigree recording since closing the flock in 1998. Rams used to join the 1700 flock ewes each year are selected progeny from AI sires used over a nucleus of 300 elite ewes. The farm had an incursion of virulent footrot in the spring of 2013. Sheep across all age groups were culled following custom vaccination in 2014⁴. Footrot was eradicated but it took 5 years to regain a balanced age structure in the flock thus in November 2019 many flock ewes were culled for genetic merit as well as age. Full pedigree lower ranked nucleus ewes were moved to the commercial flock and more young but higher ranked hoggets moved into the nucleus.

Discussion

On the 3rd January 2020 over half (55%) of the 300 stud ewes and a further 155 stud ewe lambs (all with FP+ ASBV's >150) perished in the fire. All rams in the team recently selected for the 2020 joining in February were ranked in the top 5% ASBV's on sheep genetics FP+ Index. All 31 perished in the fire. Fortunately, some of these and older deceased sires had semen in storage at the KI Vet Clinic. Most of the 140 ram weaners in another paddock had not been burned at all. Almost all the 1,558 mixed sex flock and stud ewe weaners were found in a huge pyre of bloated or ruptured bodies interspersed with those of similarly affected kangaroos and wallabies.

When I arrived on farm the next morning, I was made aware of the decimation of the stud rams and ewes. A decision had been made by the family team to ask my help to save as many of the surviving burnt ewes as possible so that the flock could rebuild numbers as quickly as possible. A quick audit of facilities showed that the undercover yards remained reasonably intact so were still useable. The adjacent shearing shed was unusable (45bales of wool from the recently culled sheep were incinerated to ash). The home of the flock owners burned as they rushed to try and escape the fire front. The husband and the dogs in the ute were burned over on the road. John crawled to safety. His wife found refuge on a nearby airstrip. The home of their daughter and her husband who were not at home was left intact probably because of the large kikuyu lawn around it within the house-yard. We made a quick decision to have the fence repaired around this as a hospital area. Shearing was due to start later that month. The shearing team turned up to help. Farmers from the eastern end of the island unaffected by the fires arrived with tools and assorted machinery

including a Bob-cat and Tip-truck that were used to help gather dead livestock ready for burial. Some hammered droppers in against damaged boundary posts to keep surviving sheep on the property. The silos almost full of barley or lupins were undamaged. All the hay was burned – some rolls had still been in the paddock. Hay was generously donated from another farm unaffected by the fires. More would be needed.

With the knowledge that we had sufficient resources to proceed with more intensive assessment and hospitalisation I went with my shooter to immediately euthanize severely affected sheep such as those with swollen faces and limbs, laboured breathing with froth coming from the nostrils and those with obvious extensive full thickness burns. Lambe sheep were euthanized if hooves had already sloughed. The sheep that were already dead were counted. The numbers that died in the fire or were euthanized immediately after are listed as fire deaths (Table 1). Sheep that were recumbent but fit enough to hospitalise were given a spray mark on their back that could be seen from the air so that resources weren't wasted coming to check on them. Volunteers came to the paddocks after shooting was finished and loaded the remaining sheep onto a ute and took them to the hospital area. The balance of the sheep that could walk were mustered and moved slowly to the yards. Water troughs were set up for the sheep. The sheep were more closely examined in the force area leading to the draft. The shearers worked all day tipping sheep over for assessment of pizzles and udders where required. Ones to be euthanized were raddled and drafted off to be dealt with as soon as possible.

Table 1. Total sheep numbers and mob breakdowns pre-fire; fire deaths within 24hrs; numbers of sheep hospitalised; hospital deaths; percent saved; actual deaths (fire + hospital) over the 6wks and potential deaths if the hospital sheep had been euthanized.

| Sheep mob | Before 2.1.22 # | Fire Deaths # | Hospital Mob # | Hospital Deaths # | Hospital Saved % | Actual Deaths # | Actual Deaths % | Potential Deaths # | Potential Deaths % |
|-------------------------|-----------------|---------------|----------------|-------------------|------------------|-----------------|-----------------|--------------------|--------------------|
| TOTAL | 5,573 | 2,218 | 1,290 | 317 | 75.4 | 2535 | 45.5 | 3508 | 63.0 |
| Flock Ewes | 1,264 | 141 | 503 | 209 | 22.8 | 350 | 6.3 | 644 | 11.6 |
| Stud Ewes | 300 | 165 | 135 | 9 | 9.8 | 174 | 3.1 | 300 | 5.4 |
| Hoggets MS [†] | 900 | 312 | 588 | 80 | 39.4 | 392 | 7.0 | 900 | 16.1 |
| Weaners [‡] MS | 1,558 | 1,500 | 58 | 13 | 3.4 | 1,513 | 27.1 | 1558 | 28.0 |
| Wnr [§] Rams | 140 | 0 | 6 | 6 | 0.0 | | | 6 | 0.1 |
| Adult Rams | 31 | 31 | 0 | N/A [¶] | N/A | 31 | 0.6 | 31 | 0.6 |
| Wethers | 1,380 | 69 | 0 | N/A | N/A | 69 | 1.2 | 69 | 1.2 |
| TOTAL | 5,573 | 2,218 | 1,290 | 317 | 75.4 | 2,535 | 45.5 | 3,508 | 63.0 |

† MS = mixed sex; ‡ Weaners = flock MS + stud ewe wnr; § Wnr = Weaner; ¶ N/A = Not Applicable

All sheep forming the 1290 hospital mob were given blanket treatments of intramuscular Alamycin LA and Flunixin – both dosed according to body weight and repeated every 3 or 4 days until the end of January. Flunixin is a potent analgesic, anti-pyretic and anti-inflammatory drug taking effect within an hour of injection. Its use in sheep is off label. The sheep that could walk were then put in a small holding paddock with water in troughs and kikuyu pasture which helped to prevent soil contamination of many of the burns. The sheep that couldn't walk very well were put in the intensive hospital area around near the remaining house. The daughter elected to nurse them with visits from me every few days when we euthanized any sheep not making progress or evidence that injury was more extensive than when first assessed. After 2 weeks 169 adults and 5 weaners had been euthanized, with a further 59 adults and 7 weaners by 3 weeks. By 6 weeks another 86 adults and 6 weaners were euthanized. A total of 317 sheep were euthanized over the hospitalisation period leaving 973 survivors (75.5% of the 1290 selected for treatment).

Burns were sprayed daily with a product developed for mulesing wounds called Trisolfen and it seemed to stop the wounds from drying and promote healing. Extinosad was mixed up in a hand-held pressure sprayer and applied around wounds to keep the flies away. Wound care was difficult with the sheep in full wool and healing proceeded more rapidly in the worst affected sheep once they had been shorn starting on the 12th February. Recumbent sheep with sore feet were stood up

twice daily and given food and water within easy reach so that they weren't forced to walk. Once sheep became mobile and no longer needed daily wound treatment, they were moved to the small holding paddock. The main problem encountered during the hospitalisation period was flystrike of wounds where exudation into the wool became an attractant. All sheep had left the intensive hospital paddock by the end of shearing only 6 weeks after the fire event.

A new Norbrook product Hexasol LA injection was released in Australia for use in cattle in January 2020. Some was donated by the company and used on other properties under my supervision on KI where cattle and sheep were hospitalised with burns. Each ml of this product contains 300mg oxytetracycline and 20mg flunixin meglumine. The dose is 1ml per 10kg body weight and provides 6 days of activity. The withhold for cattle is 21 days. Most animals were given 1 dose to control and prevent infection. In animals with more severe burns 2 doses were given. This product makes hospital treatments quicker and easier with less stress on the animal due to less handling. There is more owner compliance with fewer treatments with the outcome of improved animal welfare and saved lives.

Table 2. Sample of sheep production data for some years before and after 2020 fires.

| Farm data | 2014 footrot† | 2015... | 2017... | 2019 | 2020 fire 2.1 | 2021 | 2022 to 10.5 |
|--|------------------|---------|------------|---------|-------------------|---------------|-----------------|
| Grazed ha | | 521 | 521 | 521 | 485 | 485 | 485 |
| Season prior‡ | | good | good, long | average | good | late, wet | poor |
| #Sheep shorn Feb (#survived the fire) | | 5,579 | 5,906 | 6,002 | 5,573§ (3,038) | 4,021 | 4,917 |
| DSE's/ha | | 11 | 13 | 15 | 8 | 12 | 13 |
| Wool clean kg/ha | | 35 | 41 | 38 | 34¶ | 30 | 33 |
| #Joined Total Feb | | 1,765 | 1,858 | 1,578 | 1,294 | 1,676 | 2,380 |
| “ “ Adult Flock | | “ | “ | “ | “ | 1,041 | 1,588 |
| “ “ Adult Stud | | | | | | 126 | 1,94 |
| “ “ Lambs Flock | | | | | | 446 | 499 |
| “ “ Lambs Stud | | | | | | 73 | 99 |
| %Pregnant Total | | N/A | N/A | N/A | 98 | 84 | |
| “ “ Adult Flock | | | | | “ | 96 | |
| “ “ Adult Stud | | | | | | 98 | |
| “ “ Lambs Flock | | | | | | 50 | |
| “ “ Lambs Stud | | | | | | 81 | |
| %Weaned Total | | 78 | 73 | 67 | 85 | 83 | |
| “ “ Adult Flock | | “ | “ | “ | 85 | 85 | |
| “ “ Adult Stud | | | | | 123 | 119 | |
| “ “ Lambs Flock | | | | | | 41 | |
| “ “ Lambs Stud | | | | | | 80 | |
| #Lambs weaned (includes orphans) | | 1,369 | 1,352 | 1,052 | 1,093 (58) | 1,390 (82) | |
| Contribution to # | | | | | 5.3% | 5.9% | |

† footrot eradication in 2014 reduced flock numbers for 2015 and affected flock structure for next 5 years

‡ season prior is described to represent Food On Offer for the wool growing year

§ includes 1213 cull sheep shorn then sold prior to the fire on 2.1.20

¶ Includes 5675kg clean wool in bales from the culls burnt in the shearing shed

For the 2020 joining the 134 weaner rams remaining were fed an ad lib diet of lupins, barley and hay to increase their body weight and testicle size to gain sufficient body size and semen quality to join the 1294 ewes that survived the fires. The 900 ewes that survived the intensive hospital treatment with pain relief and antibiotics were able to be joined in late February. Of these 98% became pregnant and 85% lambs were weaned to ewes joined by the ram lambs. The stud ewes joined to 10 of the highest ranked ram lambs (all in the top 1% of Sheep Genetics ASBV's FP+ Index) rather than attempt AI and 123% lambs were weaned. In order to further increase numbers weaned, 58 lambs were hand reared due to the mother's inability to feed them for example triplets, abandoned in terrible weather or burnt teats. They contributed 5.3% to the lambs weaned.

In 2021 all ewes were again joined with the same 2019 drop rams. At joining many flock lambs were not suitable for mating due to age (born in August/September) and the very poor start to the season but were kept in the mob for ease of handfeeding. Stud lambs by comparison were well fed on ad lib lupins, barley and hay. Most important to the outcome was the use of teasers prior to the introduction of the rams in the stud ewe lambs. The weaning rate was 83% overall with 85% for adult flock ewes, 119% for adult stud ewes, 50% for flock lambs and 81% for stud ewe lambs.

In the 2022 joining flock ewe lambs as well as studs were fed daily with 550g/hd lupins and barley with access to ad lib hay. Teasers were used in both groups. They are yet to be scanned but the expectation is that the flock lamb pregnancy rate will approach the 81% achieved by the 2021 stud lambs with a resultant 80% lambs weaned.

I admire the grit, determination and focus of this farming family team to save many breeding sheep from destruction after the 2020 fire and annually increase flock numbers and farm production on the pathway towards optimum potential while at the same time ensuring continued genetic improvement. It was an honour to help them on their journey.

Acknowledgements

Dr Jack Reddin provided a significant donation to KI Vet Clinic used to provide medication for fire affected livestock on a number of KI properties including the case study property.

Dr Andy Doube (who was on KI at the time of the fire) provided voluntary assistance as my shooter to humanely euthanize assessed unsalvageable burnt sheep.

References

1. Paull DR, Lee C, Colditz IG, Atkinson SJ, Fisher AD. The effect of a topical anaesthetic formulation, systemic flunixin and carprofen, singly or in combination, on cortisol and behavioural responses of Merino lambs to mulesing. *Aust Vet J* 2007;85:98-106.
2. McAuliffe PR, Hucker DA, Marshall AN. Establishing a prognosis for fire damaged sheep. *Aust Vet J* 1980;56:126-132.
3. Cowled BD, Bannister-Tyrrell M, Doyle M, Clutterbuck H, Cave J, Hillman A, Plain K, Pfeiffer C, Laurence M, Ward MP. The Australian 2019/2020 Black Summer Bushfires: Analysis of the Pathology, Treatment Strategies and Decision Making About Burnt Livestock. *Frontiers in Vet Sc* 2022;9 Article790556.
4. Lehmann DR. Footrot: challenges with eradication from Kangaroo Island sheep flocks. *Proceedings of the National Workshop on Footrot Diagnosis and Research. (2017) RJ Whittington and OP Dhungyel (Eds). University of Sydney, Camden, Australia*

Update on pain assessment and relief in sheep

Dr Danila Marini
Livestock and Aquaculture
The Commonwealth Scientific and Industrial Research Organisation
FD McMaster Laboratory, Armidale NSW

Introduction

In Australia's production system, livestock such as sheep undergo painful procedures as part of common husbandry practice. As concern for livestock welfare increases the need for mitigating pain also increases. The ability to assess pain in sheep and provision of pain relief options has improved over the last few years. This presentation will cover the assessment and alleviation of pain in practical situations as well as update on recent research outcomes in sheep.

Pain and its assessment

The assessment of pain in sheep is determined by the approach used. Using indicators such as physiological (sympatho-adrenal activation, inflammation), behavioural or production measurements can help us determine if an animal is in pain. Each of these measures has their own challenges and can vary with the type of pain being experienced. In addition, while studies in humans have shown sex differences in response to painful events¹, little is known in relation to sex differences in sheep. A recent study we conducted in lambs that underwent tail-docking, highlighted the effect of sex on the expression of some of these measures such as behaviour.

Analgesia

The most commonly used drugs to alleviate pain in sheep include anti-inflammatory drugs (NSAID) and local anaesthetics. With the industry having access to an injectable local anaesthetic, topical local anaesthetic combination as well as a buccal and injectable NSAID. There are still challenges in the use of these analgesics with unknowns about longer term pain in sheep and the need to re-administer analgesics. There are also potential toxicity aspects of lambs receiving multiple doses of anaesthetics when undergoing castration, tail docking and mulesing. As well as limitations of currently registered for use in sheep such as the anaesthetic lignocaine.

There are opportunities to develop new methods of assessing pain in sheep that are practical and validated. As well as finding alternative therapeutic options.

Reference

1. Mogil, J.S., Qualitative sex differences in pain processing: emerging evidence of a biased literature. *Nature Reviews Neuroscience*, 2020; 21(7): p. 353-365.

Bushfire Assistance: how to best help your clients in the immediate aftermath

S McGrath
Millicent Veterinary Clinic, Millicent SA

Abstract

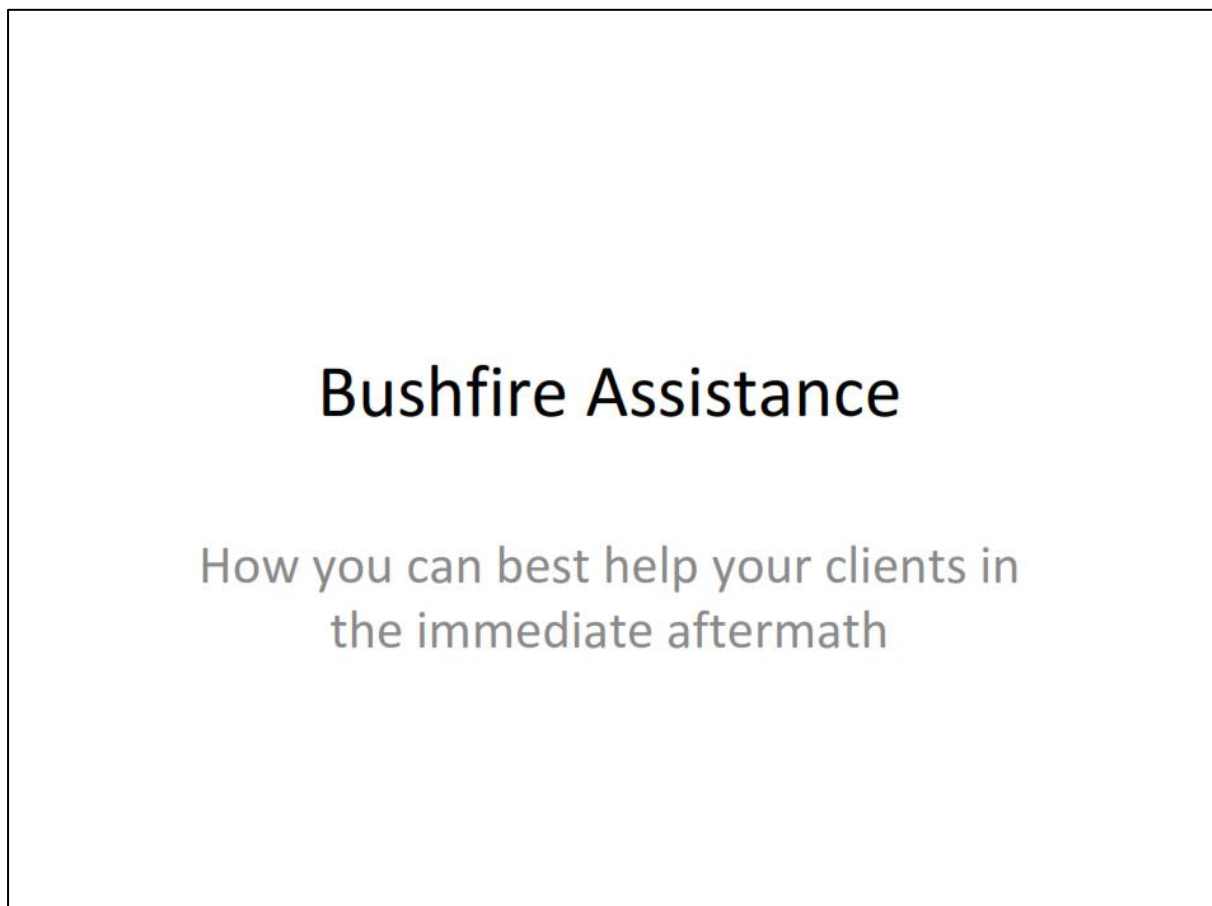
This paper will outline how the private practitioner can assist their clients immediately after a bushfire and is based on recent firsthand learnings from the farmer's perspective.

This paper comes from the perspective of a private veterinary practitioner who recently had to manage the immediate fire response on their own family property, which experienced significant stock losses. It is a summary of learning from that first hand experience of being in the farmer's position but viewed through the lens of the vet.

In the immediate aftermath of a fire a farmer has many things they need to do, coordinate and get right. Time is of the essence and in most cases the farmer does not know what, how, where or when to do things such as, stock assessment and destruction, stock burial, co ordination of people and logistics.

The paper will outline how with our training we can provide the correct advice and leadership to assist our clients through such an event. Having some preparedness of private practitioners can allow them to be effective at taking control of a situation where the client is likely to have no experience.

PowerPoint Slides from presentation:



Perspective

- Mixed practice vet
- Family farm affected
- Unknown unknowns
 - Most farmers will not have had to go through this process before
- Personal experience with the process of the immediate aftermath

Aim of Presentation

- Tips and tricks on what worked
- What can vets do best
 - Understand the process
- Look at key pillars of
 - Assessment
 - Destruction
 - Burial

Poolaijelo Fire 2021/22

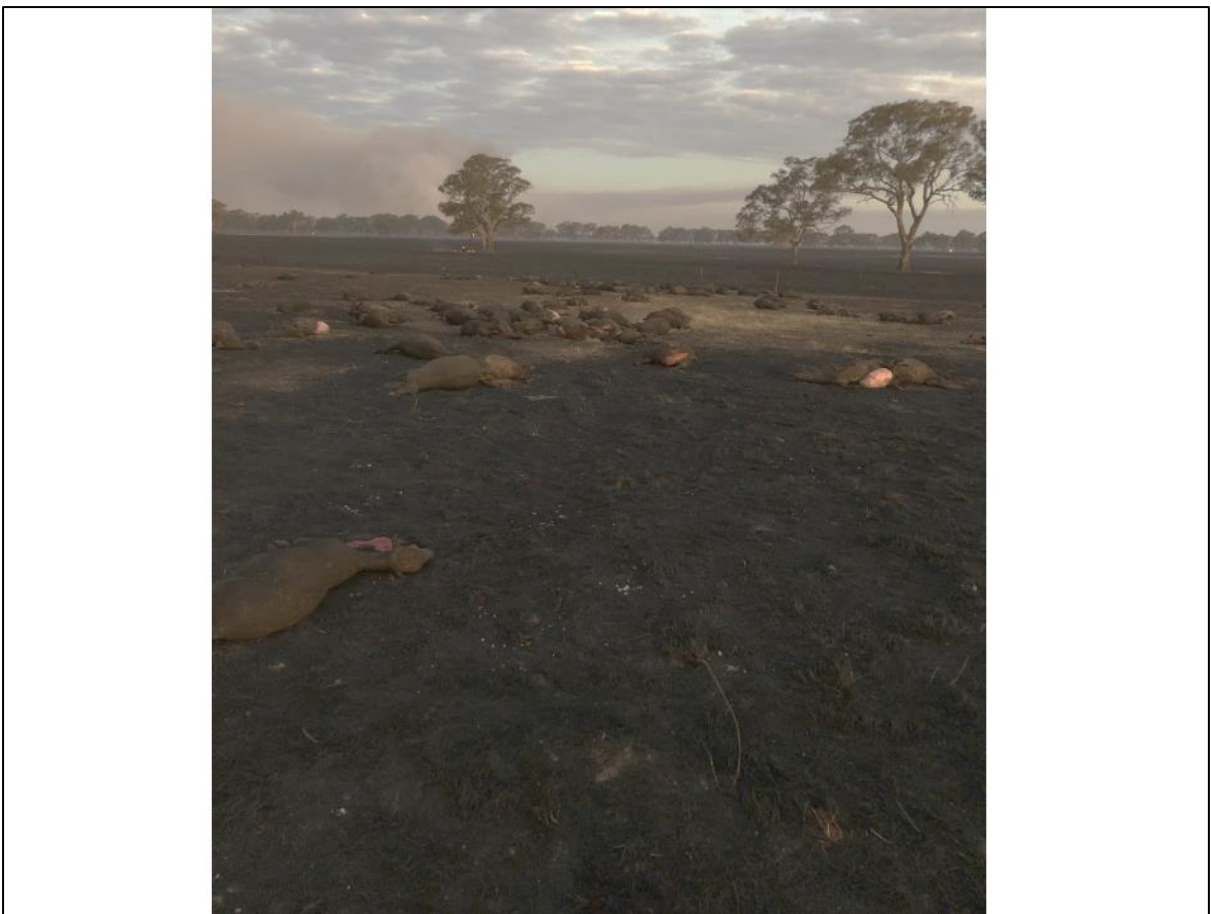
- New Years Eve
- >7,000ha burnt
- Estimated 10,000 sheep lost
- Very fast timeframe

Jalna

- 750ha burnt
- 2,600 sheep dead
- 21 cattle dead

- Survivors
 - 50 cows
 - 130 steers
 - 300 lambs
 - 700 ewes





The Reality

- The scale is daunting
- Knowing where to start
- Phone calls
- Offers for help
- Offers for feed
- You don't know what you need
- A focus on the healthy livestock

What Is Needed

- A leader/control person
 - Co ordinate people/help
 - Guide the process
- Accept the help
 - Need feet on the ground

3 Main Processes

- Assessment
- Destruction
- Burial

Assessment

- How
 - Yards
 - Paddock
- Who
 - Vets
 - Stock agents
 - Helpers

Assessment

- Cut Offs for Euthanasia
 - Dictated by insurance
 - Logistics
 - Scale
 - Ability of stock to move
- Non affected animals
 - Stocktake
 - Plan for them

Assessment

- Insurance cover
 - Is it adequate
- Scale
 - The magnitude of the affected stock can dictate how many to destroy
- Logistics
 - Ability to move and ease of assessment
- Look ahead
 - Can salvaged stock be cared for

Logistics of Saving Stock

- Feed
 - Availability
 - Ability to determine requirements
- Land
- Facilities
- Human Resources
- Emotional resources
- Can they handle trucking

Stocktake

- Unaffected animals
 - Important for final numbers
 - Involve stock agents
- What is done with them
 - Trucked off/agistment/sell







Destruction

- How
 - Guns
 - Captive bolts
 - Training – on the spot
- Who
 - Helpers
 - Work in small groups

Destruction

- Lots of resources
 - People
 - Guns
 - Ammunition
 - Yards
- Safety
 - Lots of people with guns



Burial

- How
 - Machinery
- Who
 - Helpers
 - Machinery
- When
 - ASAP
 - Pile into heaps
- Regulations
 - BIG limiting step in the process







What Role Can Vets Play

- Leadership
- Be the expert
- Train in destruction
- Train in assessment
- Lay out the process and steps that will be required
- Make decisions

Preparedness

- Networks
 - People to help
- Guns, ammunition, captive bolts
- Know the process
- Each event and property will be different
- Be prepared to go, don't wait for the call

Case Study: Ewe lamb foetal loss in South East South Australia

Dr Sean McGrath
Millicent Veterinary Clinic
62 Mt Gambier Rd, Millicent SA

Introduction

This case study is from a Composite ewe flock in the South East of South Australia. The farm manager had records of variable reproductive performance in ewe lambs on the property over a nine year period. Most recently they identified a significant loss of foetuses from scanning to lamb marking that triggered further investigation. The farm was enrolled in the MLA funded Murdoch University project on Reproductive Wastage In Maiden Ewes.

Investigation was undertaken in 2020 and followed the project guidelines. During the course of the project, it was found that 50% of ewes that were scanned in lamb at the first scan, then had aborted by the time of the second scan. Further investigation into these aborted ewes was then undertaken outside the project guidelines.

Method

A monitor mob of 200 ewe lambs was followed from pre joining through to lamb marking. To be eligible they needed to be 40kg liveweight pre joining. They were then followed through the year and data was collected at the following timepoints. In addition, the farm manager was required to undertake lambing rounds to match up lambs born alive to foetuses scanned.

The timepoints for data collection were as follows:

1. Pre joining
2. 1st scan 42-50 days from removal of rams
3. 2nd scan 110-117 days after rams in, at least 30 days after scan 1
4. Pre lambing
5. Post lambing

The data collected at each timepoint is as follows:

- Bodyweight
- Body Condition Score
- Serology for infectious disease
- Feed On Offer

After Scan 2, there was such a high number of aborting ewes identified, samples were taken from the aborted ewes. Vaginal swabs were taken from the aborted ewes for PCR and culture. An aborted foetus and placenta was found in one of these ewes and submitted for PCR, culture and histopathology. Two ewes were sacrificed and the entire uterus was submitted for culture.

Results

The results of the losses from Scan 1 to Scan 2 are shown below in Tables 1 and 2. Table 3 shows the breakdown of single and twin bearer status of the two groups of ewes, Pregnant and Aborted.

Table 1: Results of pregnancy Scan 1

| | Total Number | % |
|-----------------------|--------------|------|
| Scanned in Lamb (SIL) | 122 | 61% |
| Single | 53 | 44% |
| Twin | 69 | 56% |
| Total Fetus | 191 | 156% |

Table 2: Results of pregnancy Scan 2

| | Total Number | % |
|------------------|--------------|-----|
| SIL | 60 | 49% |
| Aborted | 61 | 50% |
| Loss of Foetuses | 92 | 48% |

Table 3: Breakdown of Single and Twin bearing in Aborted and Pregnant ewes.

| | Total | Single | Twin | Single % | Twin % |
|----------|-------|--------|------|----------|--------|
| Aborted | 61 | 30 | 31 | 49% | 51% |
| Pregnant | 60 | 23 | 37 | 38% | 62% |

Tables 4 and 5 show the difference in body weight and body condition score for at each time point for the two groups of ewes, those that aborted and those that maintained pregnancy.

Table 4: Body Weight of aborted and pregnant ewes

| Bodyweight (kg) | Pre Joining | Scan 1 | Scan 2 | Pre Lamb |
|----------------------------------|-------------|--------|--------|----------|
| Aborted ewes | 43.0 | 51.6 | 46.2 | 46.0 |
| Pregnant ewes | 44.5 | 53.0 | 55.7 | 56.8 |
| Difference (aborted vs pregnant) | -1.5 | -1.4 | -9.5 | -10.8 |

Table 5: Body Condition Score of aborted and pregnant ewes

| Body Condition Score (1-5) | Pre Joining | Scan 1 | Scan 2 | Pre Lamb |
|----------------------------|-------------|--------|--------|----------|
| Aborted Ewes | 3.4 | 3.4 | 3.3 | 3.2 |
| Pregnant Ewes | 3.4 | 3.4 | 3.4 | 3.1 |

The results of infectious disease testing is summarised in table 6 below.

Table 6: Summary of results from infectious disease testing

| Samples | Results |
|---|---|
| Serology of study mob | No significant findings |
| Vaginal swabs of 30 aborted ewes | Negative to campylobacter 4 positive to <i>Chlamydia pecorum</i> on qPCR |
| Intact fresh uterus x2 for culture | No growth |
| Aborted foetus | Culture – <i>Truepurella pyogenes</i> PCR – <i>Campylobacter sputorum</i> Negative for <i>Chlamydia</i> spp |
| Vaginal swab of ewe with aborted foetus | PCR positive to <i>Chlamydia sputorum</i> |

Discussion

There was clearly a significant abortion event in that 50% of ewes initially scanned in lamb were subsequently scanned not in lamb at Scan 2. There was no difference in the abortion rate between those that were single bearing and those that were twin bearing.

There was a significant decrease in average bodyweight of the ewes that aborted, from Scan 1 to Scan 2, that being 5.4kg. This weight loss plateaued and average weight remained similar from Scan 2 to the Pre Lamb timepoint. The average weight of the ewes that remained pregnant increased from Scan 1 through to Pre Lambing, presumably driven by conceptus growth. As a group the ewes that aborted were 1.5 kg lighter Pre Joining and 1.4kg lighter at Scan 1.

In terms of infectious disease the evidence was unclear on which pathogens may be playing a role. From vaginal swabs 4 of 30 ewes (13%) were positive on qPCR for *Chlamydia pecorum*. The aborted foetus that was discovered cultured True *Purella pyogenes*, which is generally considered an opportunistic pathogen. PCR of the aborted foetus and the vaginal swab of that ewe revealed the presence of *Campylobacter sputorum* but was negative for *Chlamydia pecorum*.

Chlamydia pecorum has recently been associated with late term lamb abortions in primiparous ewes, however given these abortions were mid-term, the significance of the vaginal PCR results is unclear.

This process has identified that abortion happened soon after Scan 1 and can be characterised as mid-term abortions. There was a large loss of average weight in a short amount of time between Scan 1 and Scan 2, indicating an adverse health event in these ewes. Further research on infectious causes of mid-term abortions in ewes is warranted, as is further analysis of ewe lamb liveweight during joining and pregnancy.

Using disease investigation subsidies: a South Australian private practitioner perspective

Dr Sean McGrath
Millicent Veterinary Clinic, Millicent, SA

Introduction

This is a brief description on how existing Disease Investigation subsidies are used from a private practitioners perspective in South Australia. It will describe what disease investigation subsidies exist in the state and how the practice best uses them to achieve good outcomes for clients, the practice and the industry. It will give some pros and cons to those that are available.

Existing Subsidy Programs

The existing programs and how they are used by our practice are outlined below.

Disease Surveillance

This program subsidises laboratory fees at the state vet lab facility to the amount of \$700. It does not pay for any professional time for the work up of the case. Any disease outbreak that has an infectious differential diagnosis will be considered or eligible for the program. If follow up testing needs to be done to achieve a diagnosis, such as multiple post mortems, then this can be accommodated. Approval should be gained verbally from the local PIRSA district vet, but then there is no further paperwork to perform.

Significant Disease Investigation

This program is part of a national program and pays for laboratory fees and also professional fees to the vet for the work up and reporting. Approval needs to be gained from PIRSA representatives and formal standardised reports need to be completed after the outbreak as well as invoicing to PIRSA for payment.

TSE submission

This program is part of the national TSE program that provides proof of TSE freedom. It will pay for all laboratory fees, a rebate to the producer and a fee to the vet for the submission. No approval is required but the case must fit the criteria of the TSE scheme. No further paperwork is required, however an invoice must be sent to PIRSA for payment.

The most commonly used program in our practice is the Disease Surveillance Program. The reasons for this are multiple

- Simplicity – there is no paperwork required and quick approvals
- Broad application – we can use it to subsidise lab fees for investigations for a variety of disease presentations such as but not limited to Sudden Death, Ill Thrift, Abortion, Infertility, High Morbidities, High Mortalities, Respiratory Disease
- Cost of lab fees does not limit the investigation which means we usually get a good diagnosis
- No restriction on professional fees – we charge our clients for our time to do the investigations without restriction

The downfall of the program is mainly the amount of the subsidy. Whilst it usually covers what is needed, sometimes it does not and the balance needs to be paid. This is rarely an issue though, especially when pointed out as being a subsidy.

The SDI program is almost never used by our practice for the main reason that it is too cumbersome in terms of approval and paperwork. The main benefit is to the producer who has the vet time subsidised, however our clients have always been happy to pay for our time and rarely question that.

The TSE scheme is one that is used occasionally, mainly for cases that fit the criteria or those opportunistic individual illnesses or deaths that fit the criteria but may not be an outbreak. This scheme is quite attractive given the ease of approval and paperwork.

Practice Usage of Disease Surveillance Program

A brief review of the lab submission book for an 18-month period showed that out of 50 disease outbreaks, 25 went through the Disease Surveillance program. This does not include things like calf scours submissions, BVD serology for monitoring or trace element testing.

Some examples of cases that it can be used on are as follows:

- Dead and dying lambs, 25 dead, 6 moribund out of 400. Diagnosis was worm burden.
- Weaner death and ill thrift in cattle, 3 dead and poor growth rates in 250 weaners. Diagnosis was trace elements.
- Calf still birth, 50 out of 1000. Diagnosis was iodine deficiency.

Benefits of a Good Disease Investigation

When the financial constraints are minimal for lab fees, I feel we can generally get good outcomes in terms of a diagnosis. Alternatively with the above examples, whilst the diagnoses were not purely infectious, we were able to exclude infectious diseases as a cause or those that may have other herd or flock ramifications, such as BVD in cattle.

By achieving good outcomes for clients we can charge well for our time but it most importantly helps build good relationships with clients and shows that we are the experts. It can also be a foot in the door to lead into other services or advice, such animal health programs.

Pneumonia in south Australian lamb feedlots

Dr Mary McQuillan

Shawn McGrath, Luzia Rass, Marta Hernandez-Jover, Bruce Allworth
School of Animal and Veterinary Science
Charles Sturt University, Wagga Wagga NSW

Pneumonia is a disease of the lower respiratory tract of sheep with a range of causative agents including parasites, viruses and bacteria ^{1, 2 3}.

The terms used to classify pneumonia in sheep can be confusing, owing to the range of descriptors and terms used to encompass the condition in various countries.

Enzootic pneumonia is commonly used as a term to encapsulate the whole spectrum of lower respiratory tract infections ², but is also used to describe only the most severe, acute and exudative forms of pneumonia often associated with the bacteria *Manheimia haemolytica* ⁴.

Ovine respiratory complex (ORC) ⁵ and pasteurellosis, have been used as terms synonymous with pneumonia ². However, pasteurellosis is also used to describe pneumonia caused by *M. haemolytica* ⁶, owing to the bacteria previously being termed *Pasteurella haemolytica* type B ³.

In New Zealand two clinical forms of pneumonia are recognised; chronic non-progressive pneumonia (CNP) of lambs or hoggets between 3-10 months of age and acute fibrinous pneumonia in sheep of all ages ². CNP is a subclinical infection associated with the bacterium *Mycoplasma ovipneumoniae*, which is enzootic in lambs of slaughter age in New Zealand ^{7, 8}. It is likely that this is the case in Australia and work in this area is currently underway in Australian abattoirs ⁹. The condition appears to have similar characteristics to the condition “summer pneumonia” in Australia, which occurs in weaned sheep over the summer months ³.

CNP is complex, involving the interaction of a range of different micro-organisms and environmental factors and the sheep’s physical and immune mediated responses to these organisms and environmental factors ². In general, viral lung infections predispose to secondary bacterial infections, most notably *Mycoplasma ovipneumoniae* ⁹, *Mannheimia haemolytica*, *Pasteurella multocida* or *Histophilus somni* ¹. *M. haemolytica* is a commensal organism found in the upper respiratory tracts of healthy sheep. It is thought that infections with certain viruses (parainfluenza virus type 3 and bovine herpes virus type 1) damage respiratory clearance mechanisms and lung tissue, which facilitates the translocation of bacteria from the upper respiratory tract and the establishment of infection in compromised lungs ¹⁰. However, it is unclear if all viruses involved in pneumonia act in this way and so further studies are needed to determine the role of respiratory virus infections in sheep ¹¹.

Difficulty breathing, fever, anorexia, weakness, nasal discharge and coughing are the classical clinical signs of pneumonia ^{12, 13}. According to the presentation of these signs, three main clinical forms can be discerned, these being peracute, acute and chronic ⁶. Sudden deaths are the most frequent expression of peracute disease⁶. By contrast, in chronic forms, lambs will not necessarily show respiratory related clinical signs but often have a poor body condition and reduced average daily liveweight gain⁶.

Lambs entering feedlots are likely to be at risk of pneumonia, given the impact of stress, young age and adverse environmental conditions which are known risk factors that can predispose to pneumonia in sheep ^{11, 14-16}.

Currently an Animal Health Australia (AHA) and Meat and Livestock Australia (MLA) funded lamb feedlot disease surveillance project is underway to better understand the incidence of animal health issues specific to lamb feed lotting in Australia. Animal health and performance information from established feedlots, post mortem examinations on a cohort of lambs which die in these feedlots and abattoir surveillance data in relation to lambs from the selected feedlots is being collected.

A case series of pneumonia related deaths within South Australia have been compiled for this presentation with the findings of the diagnostic tests discussed.

Acknowledgements

Animal Health Australia and Meat & Livestock Australia for funding this work

Rob Barwell from Animal Health Australia and Melanie Smith from Meat and Livestock Australia for project insight and input.

Darren Trott, Farhid Hemmatzadeh and Lucy Woolford from The Roseworthy Veterinary Diagnostic Laboratory for processing and interpreting the findings.

References

1. Caswell J, Williams K. Respiratory system In Jubb, Kennedy, and Palmer's Pathology of Domestic Animals. 5 edn. Ed M. Maxie. Philadelphia, PA, Elsevier Saunders, 2007.
2. West D, Bruere A, Ridler A. *The Sheep: Health, Disease and Production. 4th Edtn.* Massey University Press. Auckland, New Zealand, 2017.
3. Abbott K. *The Practice of Sheep Veterinary Medicine.* University of Adelaide Press, 2018.
4. Hore D. A review of respiratory agents associated with disease of sheep, cattle and pigs in Australia and overseas. *Australian veterinary journal* 1976;52:502-509.
5. Gonzalez JM, Bello JM, Rodriguez M et al. Lamb feedlot production in Spain: most relevant health issues. *Small Ruminant Research* 2016;142:83-87.
6. Radostits OM, Done SH, Blood DC. *Veterinary medicine : a textbook of the diseases of cattle, horses, sheep, pigs, and goats.* 10th ed. edn. Elsevier Saunders, Edinburgh, 2007.
7. Alley M, Clarke J. The experimental transmission of ovine chronic non-progressive pneumonia. *New Zealand veterinary journal* 1979;27:217-220.
8. Alley M, Ionas G, Clarke J. Chronic non-progressive pneumonia of sheep in New Zealand – a review of the role of *Mycoplasma ovipneumoniae*. *New Zealand Veterinary Journal* 1999;47:155-160.
9. Lloyd J, Barchia I, Schroder J, Rutley D. Preliminary observations on pneumonia-pleurisy in slaughter lambs at one abattoir in South Australia. The Australian Veterinary Association Ltd., 2016.
10. Radi ZA, Meyerholz DK, Ackermann MR. Pulmonary cyclooxygenase-1 (COX-1) and COX-2 cellular expression and distribution after respiratory syncytial virus and parainfluenza virus infection. *Viral immunology* 2010;23:43-48.
11. Navarro T, Ramos J, de Arcaute MR, González J. Predisposing factors inducing ovine respiratory complex in intensive-reared lambs. *Small Ruminant Research* 2019;180:106-111.
12. Alley M. Pneumonia in sheep in New Zealand: an overview. *New Zealand veterinary journal* 2002;50:99-101.
13. Bell S. Respiratory disease in sheep. 2. Treatment and control. *In Practice* 2008;30:278-283.
14. Goodwin-Ray KA. Pneumonia and pleurisy in sheep: Studies of prevalence, risk factors, vaccine efficacy and economic impact. *PhD, Massey University, Palmerston North, New Zealand* 2006.
15. Mukasa-Mugerwa E, Lahlou-Kassi A, Anindo D et al. Between and within breed variation in lamb survival and the risk factors associated with major causes of mortality in indigenous Horro and Menz sheep in Ethiopia. *Small Ruminant Research* 2000;37:1-12.
16. Nash ML, Hungerford LL, Nash TG, Zinn GM. Risk factors for respiratory disease mortality in lambs. *Small Ruminant Research* 1997;26:53-60.

Health, welfare and biosecurity of sheep and cattle exposed to Australian bushfires

Dr Caitlin Pfeiffer

Faculty of Veterinary and Agricultural Sciences, The University of Melbourne, VIC

Introduction

In 2019/2020, Australia experienced severe bushfires, with tens of thousands of livestock killed or euthanized. At the time, there was little systematic research available describing the long-term effects of fire exposure on surviving animals. While recommendations for assessment of burnt stock were available, there were limited other resources specific to sheep and beef cattle farm systems to support recovery efforts. This project aimed to investigate the impacts of bushfire exposure on cattle and sheep health and welfare, and to suggest mitigation strategies for future fire seasons.

Lessons learnt from fire-affected farms

A major activity in this project was a series of detailed investigations of fire-affected beef and sheep farms in Victoria, southern New South Wales and Kangaroo Island, South Australia. Data were collected between July 2020 and March 2021, through farm visits and an online survey. All farms were asked to report their observations of animal health, welfare and biosecurity challenges experienced since their farm was burnt. In addition, during farm visits, detailed information on the fire preparation and response activities were collected. Additional studies not detailed in these proceedings investigated the areas of farms burnt for association with livestock losses, and samples collected from surviving fire-exposed cattle to assess trace element, disease and immune fitness status.

Over 50% of fire-affected farmers in these studies identified health problems in their livestock post-fire, including plant toxicities, respiratory and eye problems, lameness and unexplained deaths. Smoke exposure was believed to have contributed to the respiratory and eye diseases reported. The majority of other health issues appeared to be associated with changes to farm management rather than direct fire injuries. Biosecurity breakdowns (largely infectious diseases causing abortions such as campylobacteriosis in ewes or pestivirus in cows) were only reported from a small number of farms but had substantial economic impacts on those farms affected. The specific health issues encountered varied widely between farms. This suggests that supporting on-farm decision making around key farm management and recovery work such as fencing, provision of appropriate feed and water, and regular monitoring of livestock in response to local seasonal conditions are likely the most effective mitigation strategies to avoid disease outbreaks.

Data on fire preparation and response collected during farms visits were analysed for association with avoiding livestock losses to identify potentially effective risk mitigation strategies.¹ Of the 46 farms included, 21 (46%) reported bushfire injured or killed stock. Being prepared for fire by having a fire plan and more than two farm bushfire fighting units was associated with a lower odds of livestock deaths from fire. Other protective factors identified included backburning and receiving assistance from fire authorities. Although only 9 farms in the dataset had sheep (while all had cattle), these farms were more likely to have fire-injured livestock. This study provides an evidence-base to support the importance of having a farm fire plan and being prepared for fire. As a result, a template farm fire plan specific to sheep and beef cattle farms has been developed within the project.

Assessment of burnt livestock

A qualitative study² was undertaken to investigate the pathology observations and decision-making of veterinarians assessing burnt livestock during 'Black Summer'. While guidelines for assessment of burnt livestock are widely available, prognostic doubt occurred because it can take several days for the full extent of burns to become apparent. This doubt can be mitigated through clinician experience and the ability to re-assess stock, which was difficult to achieve given the number of farms impacted in fire-affected areas during the 2019-20 fire season. In future, resourcing regular revisits of injured livestock to manage the risk of progressing burn pathology may improve prognostic assessment accuracy, as long as adequate pain management and other supportive care can be provided for affected stock.

Analysis of abattoir data

To investigate whether smoke exposure in sheep was associated with an increased prevalence of lung disease, National Sheep Health Monitoring Program records were analysed for association between bushfire exposure and pneumonia or pleurisy at slaughter.³ A modest increase in pneumonia at slaughter was detected in sheep exposed to high intensity fires. Although the risk of pneumonia was increased 10-fold in animals with close proximity to fire slaughtered within the first week after the fire, the incidence of pneumonia was 3.78 cases per 1000 sheep in these worst-exposed animals, compared to a baseline of 0.387 cases per 1000 sheep across the whole dataset. No association was detected between fire exposure and pleurisy. The economic consequences of such an increase are considered very limited, although these impacts may vary between farms.

Implications for industry extension

A key project finding is the importance of farm management during the recovery period (which often extends for multiple years after the fire). A holistic approach to farm decision-making and management during recovery is needed, to help mitigate and reduce animal welfare and health impacts post-fire. This approach can also support farmer wellbeing, economic recovery and getting the farm business 'back on track'.

The project has developed a bushfire preparation and recovery manual for livestock producers, containing recommendations based on study results and expert recommendations. This manual will be available through Meat and Livestock Australia later in 2022 and is expected to be a useful industry resource to underpin future extension work on livestock farm bushfire preparation, response and recovery.

Acknowledgements

We acknowledge all participating fire-affected farmers who shared their stories and experiences. This project was undertaken as a collaboration between the University of Melbourne (Caitlin Pfeiffer, John Webb Ware, Megan Thomas, Carolina Munoz, Andrew Fisher, Robyn Warner, Melindee Hastie, Mark Stevenson, Simon Firestone & Peter Mansell), Ausvet Pty Ltd (Brendan Cowled, Alison Hillman, Rohan Sadler and Melanie Bannister-Tyrrell) the University of Sydney (Karren Plain, Auriol Purdie, Melanie Smith, Marina Gimeno, Anna Waldron, John House, Russell Bush, Ruth Zadoks and Mark Krockenburger), as well as external collaborators including NSW LLS veterinarians and private veterinarians in Victoria. This work was funded by Meat and Livestock Australia (project B.AHE.2102; final project report and producer manual forthcoming in late 2022 <https://www.mla.com.au/>).

References

1. Cowled BD, Hillman A, Ward MP, Clutterbuck H, Doyle M, Webb Ware J, Thomas M, Plain K, Barwell R, Laurence M, Pfeiffer C. The black summer bushfires: impacts and risk factors for livestock bushfire injury in south-eastern Australia. Aust Vet J 2022. <https://doi.org/10.1111/avj.13165>
2. Cowled BD, Bannister-Tyrrell M, Doyle M, Clutterbuck H, Cave J, Hillman A, Plain KM, Pfeiffer CN, Laurence M, Ward M. The Australian 2019/2020 black summer bushfires: analysis of the pathology, treatment strategies and decision making about burnt livestock. Front Vet Sci. 2022; 9:790556. <https://doi.org/10.3389/fvets.2022.790556>
3. Hillman A, Sadler R, Smith M, Pfeiffer C, Barwell R, Lee A, Fraser C, Lau J, Cowled B. Livestock exposure to bushfires and meat, offal and carcass quality: Is there an association? Prev Vet Med 2022:105655. <https://doi.org/10.1016/j.prevetmed.2022.105655>

Epidemic Catarrh: what was it and why did it disappear?

Dr John Plant
Veterinary Specialist (Sheep Medicine)
1/54-56 Barclay Road, North Rocks NSW

Introduction

In the early 1800s, ovine catarrh was described as an irritation of the mucous membrane of the nasal passages, produced by inhalation of dust or by exposure to cold or wet after shearing or dipping. Parasitological nasal catarrh caused by the larvae of *Oestrus ovis* was also recorded, together with infectious nasal catarrh. Infectious nasal catarrh was considered the scourge of the sheep industry in the middle of the 1800's. It was sometimes referred to Australian catarrh¹.

The cause of the disease was never established, but recent reports^{2,3} suggested a possible role for black disease. This paper will refute that suggestion and provide evidence that the disease was pneumonic pasteurellosis, caused by *Mannheimia haemolytica*.

History

The first documented outbreak in NSW was in June 1834 at Boorowa in NSW¹. The origin of the disease is not clear, but there is a report "that the sheep first observed to be affected with Catarrh, although then in a comparatively low lying and warm part of the colony, had been brought from a run on the Coast range in the County of Argyle, some 12 months previous to the disease breaking out"¹. The initial losses commenced after the onset of rain and despite being moved from the affected area, losses persisted for some time.

The spread of the disease in the late 1830's is summarised by Cameron⁴. Further outbreaks occurred in the area and in attempts to control the disease, unaffected sheep were moved away from the affected flock, contributing to the spread of the disease to other districts. By mid-August, 2500 deaths were recorded from one flock of 4200 sheep. Some owners experienced the loss of thousands of sheep in the one year. Mortality rates of 5 to 75 per cent were recorded in individual flocks. Many outbreaks occurred shortly after sheep were moved to a new location and many outbreaks could be linked to the introduction of sheep from infected flocks to a new district.

The disease rapidly spread so that by the 1850's it had spread from southern Queensland to central Victoria. In 1868³ it was noted that the disease was always traceable to sheep or the progeny of sheep brought from a cold exposed upland country. Catarrh was rarely seen in the saltbush country. Then by the mid 1860's it disappeared as a major problem. The disease was still present in some flocks in the late 1860's. The last outbreak reported was in 1870⁵.

The disease was of such concern that in 1838 a Catarrh Act was promulgated in Victoria (still part of NSW) in an attempt to control the disease. In New South Wales, there was some reluctance to institute legislative controls because of experiences with the Scab Act. Investigations by the Committee set up to investigate the disease led to the conclusion that it was contagious, but the method spread was not known. Controls under the Catarrh Act remained until the Stock Diseases Act was passed in 1923.

Clinical Signs

The early reports indicate that it affected sheep of all ages, sex and condition and that losses were more common in the cooler months. In some cases, the course was as short as 6 hours and seldom more than 3 days^{1,7}. Few sheep recovered. Clinical signs reported included malaise, inappetence, sneezing, nasal discharges, laboured respiration, swollen face and sometimes nervous signs. There are reports of cases where 75% of the flock were dead within 24 hours⁴.

Animals affected with pneumonic pasteurellosis caused by *Mannheimia haemolytica* show dullness and anorexia with respiratory distress followed by sudden death⁶. They often show serous and ocular discharges. Pneumonic pasteurellosis can occur sporadically in individual sheep but it can also occur as an outbreak in a flock.

Pathology

Bennett⁷ was one of the few people to report on the disease. He was a surgeon with a long-standing interest in comparative anatomy. His detailed post-mortem findings indicate the lesions were confined to the respiratory system. He considered that “the immediate cause of death existed in the larynx, trachea, bronchiae or the substance of the lung”. He reported no changes in the alimentary canal, liver and other organs. Lesions in the liver were not a feature of the disease⁷. The pathology described is similar to that seen with pneumonic pasteurellosis.

With pneumonic pasteurellosis, necropsy will often show extensive ecchymotic haemorrhages in the throat, with clear yellow pleural and pericardial exudates. The lungs show congestion and oedema. In peracute cases, the lungs will be swollen and cyanotic with bright purplish-red solid areas that exude a frothy haemorrhagic fluid when excised. Lesions in the liver are not a feature of this disease⁶.

Epidemiology

In the UK, environmental factors are important predisposing causes of outbreaks of pneumonic pasteurellosis. A change to cold wet weather and the practice of folding sheep on improved pastures are recorded as possible stress conditions leading to outbreaks. In England, where flock sizes were also much smaller, the practice was to move the yard to a fresh position each day⁸.

In the 1800's, sheep flocks in New South Wales were usually managed by one shepherd, with each shepherd looking after flocks of 1500 to 3000 sheep. These sheep were enclosed in folds (yards) at night to prevent straying and to prevent losses from dingoes and indigenous people or thieves. Unlike the UK, the folds were more permanent in nature with the sheep being yarded in the same area night after night. In wet winters these yards became a quagmire, providing an ideal environment for the spread of disease. Cameron⁴ cites from the report by Bennet that “unhealthy sheep and in-bred flocks were more prone to attack, especially if folded in cold, damp valleys”. Early reports suggested that the major predisposing cause was the muddy yards in which shepherded sheep were confined at night⁵.

The spread of the disease was linked to rivers and creeks. This could be expected as sheep had to be moved to new locations where there was access to water. At this stage in the development of the pastoral industries there were no dams. Losses also appeared to be associated with the movement of sheep from infected flocks and commenced shortly after contact with sheep from the infected flocks

The practice of yarding sheep overnight ceased with the loss of labour associated with the gold rushes in the 1850's. There was also the advent of fencing and with the availability of strychnine to control dingos³. Fencing allowed the sheep to spread out and find their own camps, thus avoiding the overcrowding and wet muddy conditions that predisposed the sheep to infection with catarrh.

Possible Causes

There are few reports of detailed investigations on affected sheep, apart from the report of the Committee set up in NSW in 1934⁷. This was in part due to the nature of the country where the sheep were being run, the poor communications at the time and the problems with travelling to investigate outbreaks. There were also few people available with scientific training to report on the disease⁹.

An early report suggested that catarrh, like scab, was contagious and spread by physical contact⁷. The spreading by the agency of water or air was also mentioned as a possibility. There have been several other suggestions made as to possible causes. These included that it may have been a virulent respiratory infection in a susceptible sheep population⁹. Others have suggested that it may have been due to other diseases in association with the presence of respiratory diseases¹⁰. This view was supported by the small number of post-mortem reports on affected animals.

Recent reports have suggested that epidemic catarrh was black disease, with a probable contribution from acute fascioliasis in some outbreaks^{2,3}. However, all the information suggests that it was most unlikely that this was the case. The early reports do not mention liver disease, even where there are reports of the post-mortem findings which describe the lesions observed in

some detail¹. In one report the liver is described as “usually soft and easily broken, infiltrated with fat and of a clay colour, or it may be dark coloured and filled with blood and the gall bladder distended with thick, dark bile. There is no mention of either mature or immature fluke. Bennet was described as a man with a long-standing interest in comparative anatomy who performed several post-mortems on affected sheep. It is unlikely that he would not have observed and reported these lesions in at least some of the affected sheep¹.

Black disease is associated with rapid decomposition of the carcass, but the lesions of catarrh that are described are not typical of those seen with black disease or the other clostridial diseases. There may be blood-stained frothy discharges from the nostrils in cases of black disease, but these are seen after death. There are several reports that refer to some sheep surviving for up to three days before succumbing. They also refer to sheep that live longer than this and that these sheep usually survive. Again, this is not consistent with black disease.

The pattern of outbreaks does not fit with the epidemiology of liver fluke. There were no outbreaks in Queensland or on the western slopes in New South Wales where liver fluke is rarely seen. The spread of the disease in flocks in the winter of 1834 as sheep returned to their home stations is not consistent with the epidemiology of black disease. If it was associated with fluke, then one would have expected signs of liver fluke infestations to have been seen.

In later years sheep were run in areas of the Southern Tablelands where fluke still occurs. Despite this, catarrh has not been seen since the 1850's. Similarly, one would have expected cases of catarrh in other areas where liver fluke was a major problem before the advent of drenches and vaccines e.g., the New England region of New South Wales. In the New England, there were areas where sheep could not be run because of liver fluke, but there are no reports of catarrh being involved. Further, if the disease was associated with black disease, then there is no logical reason for the apparent disappearance of catarrh in the 1840's and 1850's. At this time, there were no effective treatments available for controlling fluke and there were no vaccines.

The extent of the losses is not typical of black disease with mortalities affecting most of the flock. For losses from black disease to occur to the extent reported, there would have had to have been a massive contamination with metacercariae of pastures around the snail habitat and a mass intake of this pasture by the affected flock. The fact that the losses in the flock occurred in large numbers over a short period of days is not consistent with either acute or chronic fluke infestations or black disease. The early reports indicate that in some flocks, losses were first seen shortly after the introduction of sheep from affected flocks. There would not have been enough time for the liver fluke cycle to have been completed in sufficient numbers to affect the number of sheep reported affected in some flocks.

The case against black disease being involved is further supported by the failure of the early reports to make any mention of liver fluke in the livers of affected sheep. The small number of detailed reports available make specific mention of no lesions being observed in the liver.

The association between the disappearance of the disease and the cessation of the practice of folding sheep at night provides further evidence for a respiratory disease. The change in this husbandry practice removed the main factor setting up the conditions ideal for the transmission and development of pasteurellosis.

Does it still occur?

Outbreaks of pneumonic pasteurellosis are still seen in flocks, all associated with shedding or yarding of sheep in wet conditions or in situations where ventilation in sheds is poor. In a case investigated on the Southern Tablelands in the mid-1960's, significant losses were seen in a mob of sheep that had been brought in for drenching for fluke (with a new drench). After drenching, the sheep were shedded because of impending cold and wet weather. Losses occurred over the next 3-4 days. In retrospect, the history and signs were consistent with the pneumonic pasteurellosis (epidemic catarrh).

Conclusions

The brief post-mortem reports, the clinical findings, the history of the outbreaks and the epidemiology leads to the conclusion that most of the losses attributed to epizootic catarrh were in fact pneumonic pasteurellosis, most likely caused by *Mannheimia haemolytica*.

The information available does not support the contention that catarrh was a manifestation of black disease^{1,2}.

References

1. Bruce A (1868)- Catarrh. Journal of the Agricultural Society of NSW 1(3):35-381.
2. Parsonson IM (1998)- The Australian Ark- a history of domesticated animals in Australia (1788-1998). CSIRO Publishing pages 161-174.
3. Parsonson IM (2007)- Epidemic catarrh of sheep: the possible role of black disease. Australian Veterinary Journal 85(1 and 2):78-83.
4. Cameron JMR (1984)- The Catarrh Act of 1838. The Push from the Bush, No. 18:49-65.
5. Bruce A (1870-71) New South Wales Legislative Assembly- Votes and Proceedings- Report of the Chief Inspector Of Stock for the year 1870.
6. Donachie W (2007)- Pasteurellosis. In Diseases of Sheep 4th Edition. Edited by I.D. Aitken pages 225-234.
7. Bennett G (1835)- in Bennett G, Gibson A and Sherwin W (1935)- Reports on the Epidemic Catarrh or influenza prevailing among the sheep in the Colony of New South Wales 1835, Stephens and Stokes, Sydney:1-32.
8. Dutton WH (1985)- in Merinos, Myths and Macarthurs 1788 1900- edited by J.C. Garran and L White page 155.
9. St George TD and Sullivan ND (1973)- Pneumonia in sheep in Australia. University of Sydney Post-Graduate Foundation in Veterinary Science Veterinary Review No 13.
10. Mylrea PJ (1992)-Catarrh in sheep. Australian Veterinary Journal 69(12):298-300.

Investigating kidney lesions in lambs at Gundagai Meat Processors (GMP)

Drs Luzia Rast, Jennifer Manyweathers, Prof. Marta Hernandez-Jover, Lynne Hayes
Charles Sturt University, School of Agricultural, Environmental and Veterinary Sciences
Wagga Wagga NSW

Introduction

The Australian National Sheep Health Monitoring Project (NSHMP) commenced in 2007 to monitor diseases of sheep that are associated with production losses, reduced flock profitability and wastage of meat¹. Data collected from lines of slaughter sheep are used to assess national and regional animal health status and to provide farmers with feedback to inform proactive animal health planning and reduce the effects of endemic disease on flock productivity and profitability. Twenty diseases or conditions are monitored at 10 domestic and export abattoirs across Australia¹. Nephritis, defined as inflamed or damaged kidneys, is one of the conditions monitored and is mostly (95.3%) detected in lambs (<2-year-old)^{1 2}. Nephritis prevalence as part of the NSHMP has increased from 1.8% in 2018/19 to 3.0% in 2020/21 and was the most frequent condition identified both in NSW and QLD with 5.1% and 7.6% in 2020/21, respectively³. Nephritis has been the only condition monitored as part of NSHMP that is increasing in prevalence³.

Gundagai Meat processors (GMP) in southern NSW was identified as having particular high levels of nephritis detection in lambs at slaughter, which initiated this research project. Nephritis detected at abattoirs does lead to condemnation of kidneys and sometimes other parts of the carcass. Aetiology, true prevalence, risk factors for this condition and risk to public health and food safety are uncertain, as are associated production losses aside from carcass condemnation.

The aims of the project are to improve our understanding of the epidemiology (aetiology, prevalence and associated risk factors) of kidney lesions in lambs processed at Gundagai Meat Processors from 2018 to 2022. This project is ongoing and finishes in late 2022.

Methods

A retrospective study using data from GMP from 2018-2022 was conducted in May 2022 to estimate the prevalence and associated risk factors of kidney lesions. Statistical methods used Bayesian Network model (BN)⁴ followed by a tree-based approach to identify the relative importance of 12 predictor variables. The BN and tree-based models analysis results were then used as a guide to develop a logistic regression model by excluding the least influential variables from the 12 candidate predictor variables. A Generalised R squared was used for assessment of the model prediction performance^{5 6}.

In early June 2022, kidneys condemned due to nephritis will be sampled at GMP to determine the pathology and potential aetiologies of the lesions. This will be followed by a cross-sectional study using an online questionnaire to collect data that aims to further elucidate risk factors for nephritis.

Interim Results

Data were collected from 9,428 mobs that ranged in size from one to 2,099 animals, with a mean of 292.6 (median = 243.0, SD = 209.8). The sex of the majority of mobs of sheep was unknown (96.7%, n = 9,114), with the remainder divided into female (1.8%, n = 170), male (0.04%, n = 4) and mixed mobs (1.5%, n = 141).

Most mobs consisted of lambs less than two years old (90.2%, n = 8,504), with 6.4% being recorded as mixed aged mobs (n = 602) and 3.4% mobs recorded as consisting of sheep older than two years (n = 321). Most mobs were from New South Wales (96.1%, n = 9,057), followed by Victoria (3.3%, n = 308), Queensland (0.4%, n = 40), Western Australia (0.2%, n = 15) and South Australia (0.1%, n = 9). Almost three quarters (71.0%, n = 6,708) were received directly via contract to the processing plant, with the remainder being through organic producers (2.8%, n = 266) and through saleyards (26.0%, n = 2454). Overall, prevalence of nephritis ranged from 7.6 to 11.0% per annum between 2018 to 2022.

Modelling results suggest that there was a significant event in Summer 2018 that appeared to contribute to a dramatic decrease in the prevalence observed by GMP, but that the remaining season/years were not significantly different to each other in prevalence of nephritis.

Data suggest that there may be important differences between female and male mobs when considering prevalence of nephritis. Due to the high number of mixed and unknown mobs, however, it is not possible to make any further comments on the association between sex and nephritis.

Further, modelling suggests that there may be differences between mobs with animals of less than two years and more than two years when considering prevalence of nephritis. However, like with sex, given the high number of mixed and unknown mobs, no conclusions can be drawn in relation to age and its association with nephritis.

When comparing the prevalence of nephritis with the prevalence of other pathology within the mobs, a small positive correlation was found between prevalence of nephritis and pleurisy ($P=0.106$), arthritis ($P=0.076$) and bladder worm ($P=0.088$) (*Cysticercus tenuicollis* or *Taenia hydatigena*).

Discussion

Whilst detection of nephritis in sheep at abattoirs, including at GMP, is reported as relative common^{2 7}, clinical renal diseases in sheep appears to be less commonly diagnosed^{8 9}. This may reflect that renal disease in sheep is usually subclinical and result in minimal obvious production losses and are difficult to diagnose in live sheep in commercial farming systems.

Our data analyses suggest that using the available dataset, there is not a simple statistical answer to the most influential factor in prevalence of nephritis detection at abattoirs. Results suggest that there may be an association between the prevalence of nephritis in lambs presenting at GMP, arthritis, pleurisy and cysticercosis. This could be explained as many pathogens can spread by the hematogenous routes in infected animals and also because some pathogens have tropism for renal tissues^{7 8 9}.

Given no significant associations were found between demographic characteristics and prevalence of nephritis, a random approach to sample collection at the abattoir will be used.

Other risk factors that may need to be examined are individual Property Identification Codes and meat inspection at the abattoir.

References

1. Animal Health Australia. The National Sheep Health Monitoring Project 2021 [Available from: <https://animalhealthaustralia.com.au/national-sheep-health-monitoring-project/>].
2. Animal Health Australia. Nephritis Fact Sheet 2021 [Available from: <https://animalhealthaustralia.com.au/download/9019/>].
3. Animal Health Australia. The National Sheep Health Monitoring Project: Annual report 2020-2021 2021 [Available from: <https://animalhealthaustralia.com.au/download/9074/>].
4. Crawley MJ. The R book. 2nd ed. Hoboken, N.J: John Wiley & Sons Inc.; 2013.
5. Norsys software Corp. Netica 6.05. 2018.
6. Wood S. Generalized Additive Models: an introduction with R. Florida: Chapman and Hall/CRC press; 2017.
7. Dutta S, Rahman S, Azmi S, Prawez S, Kour N, Wani H. Patho-morphological Changes in Kidneys of Slaughtered Sheep and Goats in Jammu Region. Journal of Animal Research. 2016;6(4):705
8. Sargison ND, Angus KW. Diseases of Sheep: Blackwell Publishing; 2007.
9. Abbott K. The Practice of Sheep Veterinary Medicine. Adelaide: Aderlaide Press; 2018.

Sheep Blowfly Insecticide Resistance: Perpetual Motion

Narelle Sales
NSW Department of Primary Industries
Elizabeth Macarthur Agricultural Institute, Menangle NSW

Introduction

The Australian sheep blowfly, *Lucilia cuprina*, is an economically significant insect which has developed resistance to a number of insecticides. Despite this, there is a long history of insecticide use which is still an essential component of flystrike management. Over the last four decades cyromazine and dicyclanil have arguably been the most utilized insecticides for flystrike prophylaxis in Australia. Cyromazine was first released in 1979 as Vetrazin™ with resistance confirmed in the field in 2012¹. Resistance to dicyclanil, first marketed as Clik™, was reported in 2020² to be present, associated with cyromazine resistance, in 100% of submissions from NSW. This cross-resistance is unsurprising given cyromazine and dicyclanil belong to the insect growth regulator (IGR) group and have similar chemical structures. In addition, NSW submitters confirmed their long term and almost exclusive use of these two insecticides for flystrike prevention. This was not the case for submitters from other states but is understandable given the long performance history of cyromazine and the increased length of protection claimed by the spray-on dicyclanil based products.

Cyromazine and Dicyclanil Resistance

The responses of 100 strains of the Sheep Blowfly submitted from NSW (n=55), Victoria (n=11), South Australia (n=12), Western Australia (n=21), and Tasmania (n=1) were determined to both cyromazine and dicyclanil. Dicyclanil resistance was not found in the absence of cyromazine resistance but both cyromazine only resistance and strains susceptible to both insecticides were found in states other than NSW. As a result, strains were categorised as:

- a) DSus - Susceptible to dicyclanil and cyromazine (n=12)
- b) CyR- Resistant to Cyromazine only (n=15)
- c) DRL- Low level resistance to dicyclanil + cyromazine (n=25)
- d) DRM- Mid level resistance to dicyclanil + cyromazine (n= 20)
- e) DRH - Higher level resistance to dicyclanil + cyromazine. (n=28)

An *in vivo* trial determined the flystrike protection periods provided by marketed products when challenged by combined dicyclanil resistant (DRH) and combined dicyclanil susceptible (DSus) strains³. The development and survival of DRH 1st instar larvae, through to the fly stage, on sheep treated with one of the three dicyclanil based spray-on products (active ingredient 12.5 g/L, 50 g/L and 65 g/L) was used to demonstrate that protection periods were reduced by 73%, 78% and 69% respectively. In addition, sheep jetted with either a cyromazine based or ivermectin based product had their protection periods reduced by 50% and 33% by dicyclanil resistance. In contrast, these treatments all delivered protection against the DSus susceptible strain according to the manufacturers claims.

Imidacloprid to prevent flystrike

The most recent insecticide to obtain registration for flystrike prophylaxis was the neonicotinoid, imidacloprid, which is marketed as AVENGE + FLY®. Despite only being released to prevent flystrike since 2019 this insecticide had previously been used for 10 years to control the sheep body louse, *Bovicola ovis*. The registration of insecticidal products for multiple parasite targets on sheep is not unique and includes the OPs (e.g. Coopers® Diazinon), the ML, ivermectin (e.g. ZINJET®), and the spinosyn, spinosad (e.g. Extinosad™). However, such prior use of the same insecticide or group of insecticides for lice control was thought to have hastened the development of organochlorine resistance in the sheep blowfly.⁴

We investigated the response of field strains (n=100) to imidacloprid, which were expressed relative to a laboratory susceptible strain (LS). The susceptibility of these strains to imidacloprid ranged from a minimum ratio of 3.2-fold in a strain from Tasmania to a maximum of 42.5-fold in a strain from Victoria. The imidacloprid susceptibility of strains submitted from NSW were

significantly different ($P < 0.05$) to all other states, with WA significantly different to Victoria but SA not significantly different to either WA or Vic.

A frequency distribution plot of the response of the strains to imidacloprid, when expressed as Log (LC50) values, did not represent a “normal” distribution, and tended towards less susceptibility. In addition, the Log (LC50) value of the LS strain fell outside the observed very broad distribution. Both of these factors indicate that selection pressure has been applied by imidacloprid lice treatments to the sheep blowfly.

Potential for Multi-Resistance

Since the introduction of neonicotinoids in 1990 they are reputed to have become the most common foliar, seed or root insecticide treatments used worldwide. As a result, a number of plant pests have developed high levels of resistance, which includes the brown planthopper *Nilaparvata lugens* with a reported resistance ratio of up to 1298 to imidacloprid.⁵ Of additional concern are neonicotinoid resistant plant pests which display multi-resistance to a number of insecticide groups that include the avermectins (MLs). These studies identified increased activity of the cytochrome P450, glutathione-S-transferase, and carboxylesterase detoxification systems⁶ which are responsible for resistance to, for example, the OPs and carbamates.

The potential for multi-resistance, or “response associations”, between the insecticides currently registered for flystrike prevention and control was considered. Correlation coefficients were calculated in a pair wise fashion, using LC50 values for imidacloprid and ivermectin, according to the cyromazine and dicyclanil resistance categories. Categories of DRALL, all strains resistant to dicyclanil regardless of level and ALL, every strain tested, were also included. Associations were evident between imidacloprid and ivermectin for some of the dicyclanil/cyromazine categories. The most statistically significant correlation occurred between Imidacloprid and Ivermectin (0.8750 $p < 0.000019$) in the cyromazine resistant only (CyR) category ($n=12$). To a lesser extent, correlations were demonstrated in the DRALL (0.3762, $P= 0.00104$) ($n=73$) and ALL (0.3321, $p=0.0007$) ($n=100$) categories. The strains categorized as DRL displayed a less statistically significant correlation (0.5857, $p < 0.0218$) ($n=25$) while significant correlations ($P < 0.05$) did not exist in the remaining categories, most importantly the DSus strains ($n=12$). The literature and these data suggest that further investigation of the toxicological response to imidacloprid and the contribution of existing cyromazine and dicyclanil resistance is warranted in the Australian Sheep Blowfly.

Conclusion

In the case of the Australian Sheep Blowfly, the concurrent or contiguous use of insecticides and the subsequent development of resistance, cannot be considered in isolation or according to treatment targets. Determining the toxicological response of field strains to the range of insecticides used to control flystrike has proven to be a useful tool in providing valuable information on the potential risks and/or presence of “multi-resistance” and ultimately to inform on appropriate resistance management options.

References

1. Levot G. Cyromazine resistance detected in Australian sheep blowfly. *Aust Vet J* 2012; 90:433–437.
2. Sales N. Final Report (2020): Sheep Ectoparasite Resistance Update 2018-2020. NSW DPI Available at <https://www.wool.com/globalassets/wool/sheep/research-publications/welfare/flystrike-control/200911-on-00491-awi-project-final-report-for-publication-final.pdf> (Accessed 9.05.2022)
3. Sales N, Suann M, and Koeford K. Dicyclanil resistance in the Australian sheep blowfly, *Lucilia cuprina*, substantially reduces flystrike protection by dicyclanil and cyromazine based products. *Int J Parasitol: Drugs Drug Resist.*; 2020 ;14:118-125.
4. McKenzie J and Whitten M. Estimation of the relative viabilities of insecticide resistance genotypes of the Australian Sheep Blowfly *Lucilia cuprina*. *Aust. J. Biol. Sci.*; 1984, 37:45-52.
5. Wang Y, Wu S, Zhu Y, Chen J, Liu F, Zhao X, Wang Q, Li Z, Bo X, and Shen J. Dynamics of imidacloprid resistance and cross-resistance in the brown planthopper, *Nilaparvata lugens*. *Entomologia Experimentalis et Applicata* 2009; 131: 20–29.

6. Zhang B-Z, Su X, Xie L-F, Zhen C-A, Hu G-L, Jiang K, Huang Z, Liu R-Q, Gao Y-F, Chen X-L, and Gao X-W. Multiple detoxification genes confer imidacloprid resistance to *Sitobion avenae* Fabricius. *Crop Protection*; 2020; 128:105014

Plant poisonings of sheep in summer rainfall non-temperate eastern Australia

Shaun Slattery
Local Land Services
PO Box 466 Narrabri NSW

Introduction

Plant poisonings of sheep are a major animal health and management problem in the cropping and pastoral summer rainfall parts of eastern Australia. For the purposes of this paper the author defines this area as the NSW plains and lower slopes north of Dubbo and the non-temperate sheep areas of Queensland.

In this paper, the author draws on his 30 years' experience as a District Veterinarian in north-west NSW and the observations of colleagues, to describe the current field presentation, key clinical signs, key diagnostic tools, case management and prevention of plant poisonings in this part of Australia.

Many of the poisonings seen in this region are very sporadic in occurrence, not being seen for years or decades, only to return with poisonings in multiple flocks when the necessary circumstances align. This often creates a loss of corporate knowledge between poisoning years, both in the regional farming community and the veterinarians that serve them. This paper attempts to address this challenge.

The toxic principles and pathology of plant poisonings are not addressed, as these are generally well addressed in the veterinary literature.

The paper aims to include plant poisonings of sheep that have been reported in the region in recent decades. This list was determined by the following process. Firstly, all plant poisonings in John Plant's *Vade Mecum*¹ were listed. Poisonings were then included if any of the following applied: they had been investigated by the author or the author knew directly of confirmed cases in the region, Ross McKenzie's *Toxicology for Australian veterinarians*² included a report of poisoning in the region, there was a credible report in Hurst's *The Poisonous Plants of New South Wales*³, or colleagues consulted by the author reported they had seen cases.

This paper does have a weakness in that only colleagues with primarily NSW experience were consulted, relying on the literature to confirm poisonings that are limited to Queensland.

The poisonings are listed in alphabetical order, generally on genus name.

A list of plant poisonings not reported by the above process, despite the plant growing in the region, is at the end of the paper. This further emphasizes that plant poisonings need multiple circumstances for poisoning to occur, not just the presence of the plant.

The notes for each plant poisoning were then developed using the experiences of the author and consulted colleagues (if no direct author experience noted in section heading), information in McKenzie², Hurst³, Chris Bourke's guide for differentiating nervous and muscular locomotor disorders of sheep⁴ and case reports in the literature and on the District Veterinarians of New South Wales Flock and Herd case notes website⁵.

Hypocalcaemia in Ewes on Grazing Fodder Oats (*Avena Sativa*)

Circumstances of poisoning – Introduction of ewes in the last month of pregnancy to lush, rapidly growing fodder oats pasture. More likely if lime/salt licks have not been provided.

Clinical signs – Sternal recumbency, hind legs extending behind in “frog legs”, tremors.

Key diagnostic tools – History and clinical signs. Response to treatment. Reduced pupillary reflex. Low blood calcium variable diagnostic tool.

Case management – Calcium, Magnesium and Glucose injections.

Prevention – Avoid introducing late pregnancy ewes to risk crops. Provide lime/magnesium oxide/salt licks.

Photosensation in Lambs Grazing Fodder Oats (*Avena Sativa*)

Circumstances of poisoning – Unpigmented lambs. Typically well growing, heavily fertilised, lush crops. Cases often seen after few sunny days following period of several cold and overcast days.

Clinical signs – Photosensitisation. Drooping of oedematous ears, especially when viewed from back of mob. Lesions vulva docked sheep often may be used differentiate photosensitisation from other causes of face swelling.

Key diagnostic tools – History and Clinical Signs.

Case management – Remove from oats. Provide as much shade as possible (wool shed superior to shaded paddock), non-green feed. Pasture often safe within few days. Use small number test mob.

Prevention – Awareness with prompt action if cases observed.

Blue-Green Algae (Cyanobacteria)

Despite confirming several cases in cattle, the author has not investigated any confirmed cases of sheep. Other regional colleagues confirm this pattern. In contrast to reports in southern areas of Australia, a search of the literature found only one report of poisoning in sheep in the region, near Gilgandra in 1966⁶. This minimal incidence of poisoning in northern sheep may be due to climatic factors, and / or a reflection that in this zone, sheep are regularly watered from sources that are unlikely to be affected by blue green algae (troughs and dams supplied by windmills, artesian bores including pipe and trough schemes).

Circumstances of poisoning - Blue-green algae bloom producing a surface scum that is blown by wind and concentrated on a shore line where sheep drink. While blue green algae blooms are relatively common, livestock poisonings are uncommon.

Clinical Signs – Sudden death, jaundice, ill thrift, photosensitisation, blue-green stains mouth and legs.

Key diagnostic tools – History, liver gross and histopathology, identification of toxic species in water and scum samples.

Case management – Remove access to water, supportive including shade, providing non-green feed.

Prevention - Awareness and avoiding circumstances of poisoning.

Rock Fern (*Cheilanthes Sieberi*) - Polioencephalomalacia

Circumstances of poisoning – Usually when naïve sheep are exposed to pastures where *C.sieberi* is the only green feed. Classical circumstance is dry winters with dry stands of summer grasses with *C.sieberi* growing through it, with a small amount of rain sufficient to produce suddenly abundant green *C.sieberi* but no pasture green. Introduced, weaner and hoggets at most risk.

Clinical Signs –Blindness, reduced awareness and “star grazing” stance, progressing to lateral recumbency and paddling, maybe nystagmus.

Key diagnostic tools – History and clinical signs, response to thiamine, histopathology brain (may be false negative).

Case management – Treat with thiamine, usually good response, remove from pasture, corticosteroids may also assist.

Prevention - Awareness and avoiding circumstances of poisoning

Turkey Bush (*Eremophila deserti*) Poisoning

Circumstances of poisoning – Historically travelling hungry stock placed in paddocks where *E.deserti* only available feed. Recent investigations are sporadic low prevalence mortality where sheep have consumed *E.deserti* during winters when other pasture is short.

Clinical signs – Maybe found dead, photosensation and jaundice in mild cases, nervous signs.

Key diagnostic tools – Grazed shrubs present. Gross and liver histopathology.

Case management – Removal of mob from paddock. Affected cases place in wool shed (darkness) with no green feed.

Prevention – Awareness and prompt actions if poisoning occurs.

Fuchsia Bush (*Eremophila Maculata*) Cyanogenic Glycosides Poisoning

Circumstances of poisoning – Consumption of new shoots after dry period when *E.mucalata* has shed most of it leaves. Can occur with sheep already grazing paddock. *E.mucalata* can occur in stands of a dozen plus plants causing 10-20 deaths. Sheep die near plants.

Clinical signs – Dead sheep near plants. Respiratory distress, may recover.

Key diagnostic tools – History. Plant parts in rumen. Exclusion other causes sudden death.

Case management – Affected cases usually recover quickly if received non-lethal dose. Remove sheep from *E.mucalata* until shoots harden. Fencing off stands an option, especially coming out of drought.

Prevention – Awareness, checking *E.mucalata* for dangerous growth and exclude sheep.

Flood Plain Staggers (Author No Direct Experience With Sheep)⁷

Circumstances of poisoning –Pastures dominated by *Agrostis avenacea* (blown or blow away grass). In the 1990-91 outbreak this was associated with autumn and winter flooding in 1990 and two previous years of wet winters and dry summers (opposite to usual pattern). Cases began in spring. Sheep cases occur later in outbreak than cattle.

Clinical signs – Convulsive episodes, wide-based stance, ataxia, and hypermetria of the fore limbs and sometimes, less severely, of the pelvic limbs.

Key diagnostic tools – History, Clinical Signs, presence of toxic galls in *A.avenucea* inflorescences.

Case management – Remove sheep to safe pastures. Shade, water and minimal disturbance. A few cases and deaths will continue to occur.

Prevention – Awareness. As soon as cases seen in cattle in district, remove sheep to safe pastures.

Heliotrope (*Heliotropium Europium*) Poisoning (Author No Direct Experience, Consulted Colleague)⁸

Circumstances of poisoning – Reported from the southern part of the region, usually lambs, grazing stubble with thick covering of *H.europium* with no other feed. Acute hepatopathy after few weeks to months of grazing.

Clinical signs – Weight loss over weeks, less commonly photosensitisation.

Case management – Remove from pasture. Any sheep with photosensation place in shed and provide non-green feed.

Prevention – Awareness of risk pastures and avoid grazing.

Humpy Back⁹

Author believes this is not primarily a toxicity but rather hyperthermia due to mustering in hot conditions in full wool. The hyperthermia maybe accentuated by other minor ailments or toxicities such as respiratory disease, marginal hypocalcaemia due to oxalates etc. Increasing reports in recent years maybe due to use of motorised vehicles for mustering and increasing sheep size and fleece weight.

Circumstances of poisoning – Full woolled adult sheep being mustered distances in hot and often humid conditions. Classically for shearing during heat waves. Usually low prevalence within mob

Clinical signs – Not present when sheep first disturbed. Requires an extended period of driving (usually over one kilometre). Affected sheep lag behind mob with increasingly short and stilted gait, almost walking on the spot. Affected sheep then lower their head, bringing their hind legs forward and arching their back (humpy back) before collapsing. After brief rest (few minutes to twenty minutes) the sheep rise again to follow the flock. Problem recurs within shorter distance. Sheep will die if forced to make repeated attempts to walk. Elevated rectal temperatures.

Key diagnostic tools – . History, hyperthermia, recovery with removal of wool and exclusion of other causes.

Case management – Affected sheep picked up urgently and transported to yards for shearing as soon as possible, shade and water. Picking up sheep at first recumbency critical. These sheep generally recover. Sheep that have multiple recumbencies have a significantly poorer prognosis

Prevention – Minimise mustering in risk conditions. Shorter staged movements. Early morning. Sufficient staff for pick up vehicle with mob.

Flat Billy Button, *Leiocarpa Brevicompta* (*Ixiolaena Brevicompta*) Poisoning (Author No Direct Experience)¹⁰

Circumstances of poisoning – Locally dense stands may occur on the flood plains of the Darling River and its tributaries following flooding. Poisoning occurs when sheep are constrained to graze mature plants with intact seed heads over summer for more than 14 days. Mustering sheep precipitates clinical signs. Younger sheep more affected.

Clinical signs – On mustering, affected sheep show exercise intolerance, recumbency, weakness and sudden death.

Key diagnostic tools – History, clinical signs and gross and histopathology.

Prevention – Awareness of risk of dense local stands following flooding. Removal of stands at green stage by cropping or intensive grazing. Restricting grazing of mature plants to less than 14 days.

Marshmallow Staggers (*Malva Parviflora*)¹¹

Circumstances of poisoning – Two classic circumstances. Firstly, weaner sheep held in yards containing abundant growth of *M.parviflora*. On release, or if trucked from yards on arrival, weaners show classic staggers syndrome. Relatively common in wet winters. Second circumstance, in severe years ewes and lambs grazing pastures with extensive stands of *M.parviflora*. Lambs at foot are classically affected as yarded for marking, weaning etc with possible extensive losses at marking due to exercise stress and mismothering. Occasional adult sheep are also affected. In 2021 *M.parviflora* grew through winter until late spring with sheep readily consuming the seeds. As a result, weaners grazing pastures were also affected when mustered. Some weaner mobs took several weeks to recover after removal from *M.parviflora* pastures. Historically severe years occur with the break of major droughts in the autumn.

Clinical signs – In weaners and adult sheep, after walking a short distance, take increasingly short steps, before arching their backs and collapsing. After a short period, rise and walk short distance before repeating. Repeated attempts will lead to death. Tremors and knuckling seen in severe cases. In young lambs mustered with their ewes, sternal recumbency, refusal to move and generalised muscle fasciculations.

Key diagnostic tools – History and Clinical Signs. Histopathology of myocardium and limb muscles. May need multiple samples of myocardium and animals.

Case management – Awareness of risk pastures and have vehicles and staff available to pick up affected sheep at first recumbency. These sheep generally recover. Sheep that have multiple recumbencies have a significantly poorer prognosis.

Prevention – Remove *M.parviflora* from yards (mow or wiper-snip, graze other stock) prior to yarding with at risk animals. Awareness of risk pastures and manage risk with shorter staged movements, Staff and vehicles to pick up recumbent sheep, moving to safe paddock for a couple weeks before marking.

Nardoo (*Marsilea Drummondii*) Poisoning¹²⁻¹³

Circumstances of poisoning – While *M.drummondii* is a frequent part of many flood plain pastures on in the region, inhabiting drains, gilgais and other shallow depressions, poisonings are relatively uncommon. Mass poisonings of sheep grazing *M.drummondii* dominated pastures on floodplains have been recorded. However, poisonings are generally limited to circumstances where some factor attracts the sheep to the *M.drummondii*. These include during drought conditions when rain is sufficient to produce *M.drummondii* growth in depressions without other pasture growth and when paddocks are sown to fodder crops leaving unsown low patches containing areas of *M.drummondii*. Sporadic cases can also be seen in mobs grazing *M.drummondii* dominated paddocks.

Clinical Signs – Blindness, reduced awareness and “star grazing” stance progressing to lateral recumbency and paddling, maybe nystagmus.

Key diagnostic tools – History and clinical signs, response to thiamine, histopathology brain (may be false negative).

Case management – Treat with thiamine, usually good response, remove from pasture, corticosteroids may also assist.

Prevention - Awareness and prompt action when cases occur.

Photosensitation Grazing Burr Medic (*Medicago Polymorpha*)

Circumstances of poisoning – Lambs grazing abundant stands of lush *M.polymorpha*. Usually requires good winter rains after dry summers that reduced ground cover. Generally cases occurring throughout district. Aphids present in most but not all toxic pastures.

Clinical signs – Photosensitisation. Drooping of oedematous ears, especially when viewed from back of mob. Lesions vulva docked sheep often useful to differentiate photosensitisation from other causes of face swelling.

Key diagnostic tools – History and Clinical Signs.

Case management – Remove from pasture. Provide as much shade as possible (wool shed superior to treed paddock), non-green feed. Maybe extended period until pasture safe. Use small number test mob.

Prevention – Awareness with prompt action if cases occur.

Photosensitation Grazing Lucerne (*Medicago Sativa*)

Circumstances of poisoning – Classically young sheep grazing well growing, well managed stands. May have been grazing for extended period. Aphids may or may not be present.

Clinical signs - Photosensitisation. Drooping of oedematous ears, especially when viewed from back of mob. Lesions vulva docked sheep often useful to differentiate photosensitisation from other causes of face swelling.

Key diagnostic tools – History and Clinical Signs.

Case management – Remove from lucerne. Provide as much shade as possible (wool shed superior to treed paddock), non-green feed. Pasture often safe within 10 days. Use small number test mob.

Prevention – Awareness with prompt action if cases occur.

Mycotoxin Poisoning Associated with Feeding Grain or Plant Material¹⁴

Circumstances of poisoning – Author's experience suggests sheep are very resistant to mycotoxin poisoning from contaminated cereal grains (feeding of mouldy grain common in droughts). Cases with cereals are generally limited to sheep consuming solid caked mould grain residue from cleaned out silos or sheep given access to the caked exterior of grain piles stored long term in the open. Drought feeding of mould contaminated peanuts or other alternative feeds is higher risk.

Clinical Signs – dependent on toxin, inappetence at next feeding (drought).

Key diagnostic tools – Gross and histopathology, feed assay.

Case management – Cease feeding, supportive including shade, easy access water.

Prevention – Avoid caked mould grain from cleaned out, dilute mould contaminated cereals with clean sources, feed to most resistant class of stock, avoid or test non-cereals with mould.

Nitrate-Nitrite Poisoning

A wide variety of plants are potential nitrate accumulators, however in this region and the author's experience the majority of cases are due to pigweed (*Portulaca spp*) and marsh mallow (*Malva parviflora*).

Circumstances of poisoning – Specific circumstances are required for sheep to eat large amounts of high nitrate plant material in a short period of time. High nitrate plant material is classically from post dry period growth in high nitrogen soils in yards and holding yards. After the 2017-20 drought there had also been sufficient absence of growth and presumably nitrogen fixing for pasture paddocks to also produce high nitrate plants. Hungry and empty sheep is essential. This is often produced by trucking (and then unloading in yards with high nitrate plants) or yarding for management procedures. The longer off feed the greater the risk. Particular plants seem to be easily quickly consumed by sheep. These include pigweed (*Portulaca spp*) in yards and paddocks and marsh mallow (*M.parviflora*) in yards, especially with breaks that occur in winter months.

Clinical signs – Mostly sudden deaths. Affected sheep show rapid respiration and ataxia, either dying or recovering to walk away with 30 minutes of severe signs.

Key diagnostic tools – Nitrate-Nitrite test strips on eye fluid.

Case management – Rarely encounter live cases.

Prevention – Awareness. Trucked sheep, remove risk plants from yards by cutting and removing. Yarded sheep, supply good quality forage to fill before allowing access to paddocks. If early in outbreak remove from pasture or yards.

Poisoning Lambs Grazing Bambatsi Panic (*Panicum Coloratum*) Pasture

Circumstances of poisoning – Lambs preferentially grazing actively growing new shoots following rain. Maybe either short pasture regrowth or shoots from the base of mature clumps.

Clinical signs - Photosensitisation, possible severe, jaundice, initial cases signalled by shade seeking in overcast or cool weather.

Key diagnostic tools – History, Gross and histopathology liver.

Case management – Remove from pasture. Provide as much shade as possible (wool shed superior to treed paddock), non-green feed. See prevention for return to pasture.

Prevention - Only graze lambs on mature pastures that are not actively growing. Remove sheep from the pasture following rainfall. Initially graze actively growing pasture with ewes to remove green shoots. Delay grazing lambs until 2–3 weeks after any rain. Graze lambs at high stock densities to reduce the potential for lambs to preferentially graze any new green shoots.

Poisoning Lambs from *Panicum Effusum* (Hairy Panic) (Author No Direct Experience)¹⁵

Circumstances of poisoning – Reported from southern part of region. Lambs grazing pasture dominated by *Panicum* with green shoots, either from short growth or lower shoots in mature plants. Often in fallow cultivation paddocks.

Clinical signs - Photosensitisation, possible severe, jaundice, initial cases signalled by shade seeking in overcast and cool weather.

Key diagnostic tools – History, Gross and histopathology liver.

Case management – Remove from pasture. Provide as much shade as possible (wool shed superior to treed paddock), non-green feed. See prevention for return to pasture.

Prevention - Only graze lambs on mature pastures that are not actively growing. Remove sheep from the pasture following rainfall. Initially graze actively growing pasture with ewes to remove green shoots. Delay grazing lambs until 2–3 weeks after any rain. Graze lambs at high densities to reduce the potential for lambs to preferentially graze any new green shoots.

Acute Oxalate Poisoning with Pigweed (*Portulaca* Spp)

While kidneys with evidence of oxalate damage are a not uncommon incidental finding to investigations into other causes of death, the author has not seen clinical chronic oxalate poisoning.

Circumstances of poisoning – Sheep moved from pastures with no or little *Portulaca*, held off feed so hungry, and moved to pasture dominated by lush *Portulaca*. Classical risk pastures are often holding paddocks around yards that were bare following a dry summer, and that following late summer-autumn rains now contain abundant lush *Portulaca*. Cases can occur both in mobs grazing the *Portulaca* pasture (usually found within 12 hours) or only when the mob is moved within 48 hours.

Clinical signs – Initial sternal recumbency, hind legs extending behind in “frog legs”, tremors. Can quickly progress to lateral recumbency and death. If on moving, exercise intolerance followed by sternal recumbency and “frog legs”.

Key diagnostic tools – History and clinical signs. Response to treatment (can be immediate) Reduced pupillary reflex. Low blood calcium variable.

Case management – Calcium, Magnesium and Glucose injections.

Prevention – Awareness and avoiding risk circumstances.

Wild Turnip (*Rapistrum Rugosum*) Poisoning

Circumstances of poisoning – Cases are limited to specific circumstances where hungry sheep are suddenly given access to lush and rapidly growing stands of *R.rugosum* at the rosette stage. Paddock shifts, holding in yards and historically release from overnight Travelling Stock Route breaks are common situations. *R.rugosum* risk pastures occur when dry summers and autumns have reduced ground cover and late autumn-winter rains produce *R.rugosum* germination and growth.

Clinical Signs – Blindness, reduced awareness and “star grazing” stance progressing to lateral recumbency and paddling, maybe nystagmus,

Key diagnostic tools – History and clinical signs, response to thiamine (even though sulphur associated), histopathology brain (may be false negative)

Case management – Treat with thiamine, usually good response. As the sudden access is a trigger removal from pasture is not essential, corticosteroids may also assist

Prevention - Awareness and avoiding circumstances of poisoning, especially sudden access while hungry.

Rumen Impaction from Feeding High Oil Drought Feeds

Circumstances of poisoning – High level feeding of high oil drought feeds (nuts, palm kernel) with low digestibility roughage feed (straw etc). Sporadic deaths after day or so of standing away from mob. Often the better condition sheep. May be triggered by change in roughage, feeding regime.

Clinical signs – Affected sheep often found recumbent and or dead. Difficult to differentiate from shay feeders,

Key diagnostic tools – Gross pathology of dry, impacted rumen.

Case management – Cease feeding high oil feed, increase grain and pellets feeding, ensure roughage less than 20% diet.

Prevention – Ensure ration oil content less than 6%.

Acute Cyanogenic Glycoside Poisoning Grazing Forage and Grain Sorghums

Circumstances of poisoning – Requires both sorghum with high cyanogenic glycoside levels and hungry sheep able to consume large amount sorghum quickly. With modern varieties of forage sorghum, poisonings in actively growing, even re-growth crops is rare. Poisoning generally occur in crops that have been extensively grazed to stubble, with some short regrowth that is moisture stressed or frosted.

Clinical signs – Mostly found dead. Affected sheep show rapid respiration and ataxia , either dying or recovering to walk away within 30 minutes of initial severe signs.

Key diagnostic tools – History and exclusion of other causes. Testing of plant material.

Case management – Rarely encounter live cases.

Prevention – Don't graze risk stages. Avoid introducing hungry stock to sorghum paddocks, including accidental access. Hay feeding before introduction, use small test mob.

Sorghum Associated Ataxia (Author No Direct Experience)¹⁶

Circumstances of poisoning – Extended grazing of forage sorghums with elevated cyanogenic glycoside levels. Typically crops grown in droughts with just sufficient rain to produce occasional new shoots.

Clinical signs – Weakness, ataxia, head shaking, knuckling of fetlocks, inability to rise, and opisthotonos.

Key diagnostic tools – History, clinical signs and brain and spinal cord histopathology.

Case management – Remove from pasture. Provide feed and water to affected cases until recover, may take several months for full recovery.

Prevention – Avoid grazing risk pastures. Supplement with sulphur while grazing.

***Trachymene* (Wild Parsnip) – Bentleg (Author No Direct Experience)¹⁷**

Circumstances of poisoning – Late summer or autumn rains following drought producing *Trachymene*. Preferential grazing of flowers with seeds in early summer by pregnant or lactating ewes. Suitable circumstances for district wide outbreaks limited to every 10-15 years.

Clinical signs – Lambs with front legs bowed.

Key diagnostic tools – History and Clinical Signs.

Case management – Proportion of lambs fully recover

Prevention – Awareness and avoiding circumstances of poisoning

Photosensitisation from *Tribulus Spp*¹⁸

Circumstances of poisoning – *Tribulus* does not generally cause this poisoning when lush and rapidly growing. In the author's experience, cases have been associated with sufficient rain for initial germination and growth of a carpet of *Tribulus*, but low soil moisture then produces wilted and stressed plants. Young sheep are more susceptible.

Clinical signs – Photosensitisation, possible severe, jaundice, initial cases signalled by shade seeking in overcast and cool weather.

Key diagnostic tools – History, Gross and histopathology liver.

Case management – Remove from pasture. Provide as much shade as possible (wool shed superior to treed paddock), non-green feed. Pasture may be toxic until rain produces new growth. Use small number test mob.

Prevention – Awareness with prompt action if cases observed.

Coonabarabran Staggers (*Tribulus Terrestris*) – Cat Head, Caltrop¹⁹⁻²¹

Circumstances of poisoning – Summer rains after drought or on fallow paddocks, producing *T. terrestris* dominated pastures. Cases appear at least 3 months after initial exposure and may continue to appear months after removal from *T. terrestris*. Meat breed sheep and their crosses significantly more susceptible. Usually low prevalence (<3%). Clinical course lasts average 8 months. Usually adults, but occasionally hoggets may be affected. Note *T. terrestris* fruit has segments that form a typical caltrop long spiked form. *T. micrococcus* has globular fruit without long spikes.

Clinical Signs – Chronic, progressive, and irreversible. Initial mild hindquarter paresis. Next asymmetry develops with one hind limb showing more weakness. This results in affected sheep moving sideways from the hindquarters as they run. In the final stages, forelimb weakness develops leading to recumbency and death.

Key diagnostic tools – History and asymmetrical hindlimb ataxia.

Case management – No treatment. Consider euthanasia affected sheep. Remove mob from *T. terrestris* pastures.

Prevention – avoid grazing *T. terrestris* dominated pastures, especially fallow paddocks.

Yellow-Vine Staggers (*Tribulus Micrococcus*)²²

Circumstances of poisoning – Early autumn rains after drought, with follow up falls, producing *T. micrococcus* dominated pastures. Cases appear 3-5 weeks later in adults. Meat breed sheep and their crosses significantly more susceptible. Usually low prevalence (<3%). Deaths occur due to secondary issues such as flystrike, pregnancy toxemia, misadventure. Otherwise affected sheep recover a few weeks after removal from pasture or when *T. micrococcus* no longer dominates the pasture. Note *T. micrococcus* has globular fruit without long spikes, unlike *T. terrestris*.

Clinical Signs - Bilaterally symmetrical, transient, hind- quarter ataxia.

Key diagnostic tools – History and clinical signs.

Case management – Removal mob from pasture. Carry affected sheep to yards to ensure watering, feed and shelter.

Prevention – Avoid grazing meat breeds and cross bred adult sheep on *T. micrococcus* dominated pastures.

Swainsonia (Darling Pea) Poisoning (Author No Direct Experience, Consulted Colleague)²³

Circumstances of poisoning – Large number of historical records in northern NSW but few in recent decades. Recent reports after the 2013 Coonabarabran fires when timing of fires and rains produced regrowth pastures that were dominated by *Swainsonia*. Poisoning occurs where sheep eat *Swainsonia* as large proportion of diet for over 4 weeks. Noted in literature as occurring after summer droughts and floods with winter rain. Small holding or house paddocks feature in recent goat reports.

Clinical signs – Difficulty in mustering due to failure of mob behaviour an early sign, ataxia, tremor, hyperexcitability and weight loss (sheep may progress to emaciation and death without nervous signs).

Key diagnostic tools – History, clinical signs and histopathology.

Case management – Removal from pasture leads to gradual recovery unless severe.

Prevention – Avoid grazing risk pastures for more than four weeks.

Crown Beard (*Verbesina Encelioides*) Poisoning (Author No Direct Experience, Consulted Colleague)²⁴

Circumstances of poisoning – Hungry sheep where *V. encelioides* only feed or only green feed. Large number of historical records in northern NSW but few in recent decades, despite *V. encelioides* stands under tress being common in the region. Recent case reported in southern part of region.

Clinical Signs – Rapid death, respiratory difficulty.

Key diagnostic tools – History, gross pathology.

Case management – Remove from access.

Prevention – Awareness, avoid circumstances.

Bloat From Faba Bean (*Vicia Faba*)²⁵

Circumstances of poisoning – Generally dorper lambs or weaners given ad lib access to faba beans, especially after period off feed.

Clinical Signs – Death, bloat

Key diagnostic tools – History, gross pathology of rumen foam, forequarters congestion

Case management – Rarely opportunity to treat with critical period. As per pasture bloat, oral oils etc

Prevention – Limit feeding of beans including introductory ration, ensure self-feeders not faulty, generally large amount of consumption required for poisoning.

Plants that occur in Region but no Recent Reports of Poisoning in Literature

Copper poisoning from chronic pyrrolizidine alkaloidosis, *Nerium oleander*, Mother of Millions - *Bryophyllum* spp) despite grazing pastures poisoning cattle, Cycad, Green Cestrum (*Cestrum parqui*) - despite grazing pastures poisoning cattle, Gossypol toxicity despite extensive feeding of lambing ewe mobs with white cottonseed, *Ipomoea* poisoning, Noogoora Burr (*Xanthium chinense*), facial eczema (cases seen in cattle grazing oats), Nitrate-Nitrite from sorghum hay (fed same hay sources poisoning cattle), Stagger weed (*Stachys arvensis*), *Stypandra glauca*, *Solanum* spp,

Acknowledgements

I would like to acknowledge the assistance of Megan Davies, Steve Eastwood, Judy Ellem, Jillian Kelly, Greg McCann and Bob McKinnon in providing input on regional poisonings initially omitted and the key elements of poisonings, especially those outside the author's experience.

References

1. Plant J. Diagnosis of diseases of sheep. University of Sydney Post-Graduate Foundation in Veterinary Science. 1992.
2. McKenzie RA. Toxicology for Australian veterinarians. RA McKenzie; 2002.
3. Hurst E. The poison plants of New South Wales. Compiled under direction of the Poison Plants Committee of New South Wales. The poison plants of New South Wales. Compiled under direction of the Poison Plants Committee of New South Wales. 1942.
4. Bourke CA. The clinical differentiation of nervous and muscular locomotor disorders of sheep in Australia. Australian Veterinary Journal. 1995 Jun;72(6):228-34. Available from: <https://doi.org/10.1111/j.1751-0813.1995.tb03528.x>
5. Flock and Herd, Case notes on veterinary investigations in sheep, cattle and other species [Internet]. [Place unknown]: Association of District Veterinarians of New South Wales; [cited 9 May 2022]. Available from: <http://www.flockandherd.net.au>
6. [McBarron EJ, May V. Poisoning of sheep in New South Wales by the blue-green alga *Anacystis cyanea* \(Kuetz.\) Dr. and Dail. Australian Veterinary Journal. 1966 Dec;42\(12\):449-53. Available from: <https://doi.org/10.1111/j.1751-0813.1966.tb14471.x>](#)
7. Davis EO, Curran GE, Hetherington WT, Norris DA, Wise GA, Roth IJ, Seawright AA, Bryden WL. Clinical, pathological and epidemiological aspects of flood plain staggers, a corynetoxicosis of livestock grazing *Agrostis avenacea*. Australian Veterinary Journal. 1995 May 1;72(5):187-90. Available from: <https://doi.org/10.1111/j.1751-0813.1995.tb03187.x>
8. Salmon D. Pyrrolizidine alkaloid poisoning of sheep. Flock and Herd. March 2012. [cited 9 May 2022]; Available from: <http://www.flockandherd.net.au/sheep/reader/pyrrolizidine-alkaloid-poisoning.html>
9. Pratap B. T Humpty Back in Sheep [webinar]. Queensland: Leading Sheep; 7 March 2017 [cited 9 May 2022]. Available from: <http://www.leadingssheep.com.au/2017/03/humpty-back-in-sheep/>
10. Walker K. *Ixiolaena brevicompta* intoxication in sheep post-flooding and its control by environmental and grazing management. 1991 13-17 May, Darling Harbour, Australia; Australian Veterinary Association, 2009. Available from: https://www.ava.com.au/library-resources/library/conference-proceedings/1991/ixiolaena-brevicompta-intoxication-in-sheep-post-flooding-and-its-control-by-environmental-and-grazing-management/Ixiolaena_brevicompta_intoxication_in.pdf
11. Kelly J. Small flowered mallow (marshmallow, *Malva parviflora*) intoxication (marshmallow staggers) in the Coonamble district following drought-breaking rain. Flock and Herd. February 2021. [cited 9 May 2022]; Available from: <http://www.flockandherd.net.au/sheep/ireader/marshmallow-intoxication.html>
12. Slattery S. Unusual presentations of *Cheilanthes sieberi* and *Marsilea drummondii* poisonings in the Narrabri RLPB district. Flock and Herd. 2005. [cited 9 May 2022]; Available from: <http://www.flockandherd.net.au/archive/transcribed/2005/reader/rockfern-nardoo.html>
13. Chick BF, Eggleston GW, McCleary MB. The Epidemiology of Nardoo Poisoning in Sheep. The Association of Veterinary Inspectors of NSW, 62nd Annual Conference; 1979 April 2-6; Sydney. Place of publication: Assoc of veterinary Inspectors of NSW; 1979. [cited 9 May

2022] p. 55-60. Available from:
<http://www.flockandherd.net.au/archive/transcribed/1979/reader/nardoo-poisoning.html>

14. Davies M. Mycotoxicosis in a merino flock. Flock and Herd. February 2015. [cited 9 May 2022]; Available from: <http://www.flockandherd.net.au/sheep/reader/mycotoxicosis.html>
15. Litchfield NH. Yellow big-head in lambs. Institute of Inspectors of Stock of N.S.W. Year Book 1948 Annual Conference; 1948 March 15-19; Sydney, Australia : Institute of Inspectors of Stock of N.S.W, 1948 [cited 9 May 2022]. p. 75. Available from:
<http://www.flockandherd.net.au/archive/transcribed/1948/reader/yellow-big-head.html>
16. Davies M. Sorghum-associated neuroaxonal degeneration in dorper lambs. Flock and Herd. December 2018. [cited 9 May 2022]; Available from:
<http://www.flockandherd.net.au/sheep/ireader/neuroaxonal-degeneration-lambs.html>
17. Clark L, Carlisle CH, Beasley PS. Observations on the pathology of bent leg of lambs in South-Western Queensland. Australian Veterinary Journal. 1975 Jan 1;51(1):4-10. Available from:
<https://doi.org/10.1111/j.1751-0813.1975.tb14489.x>
18. McCann G. Crystal associated cholangiopathy (yellow big head) in lambs grazing *Tribulus terrestris*. Flock and Herd. July 2013. [cited 9 May 2022]; Available from:
<http://www.flockandherd.net.au/sheep/reader/yellow-big-head.html>
19. Bourke CA. Staggers in sheep associated with the ingestion of *Tribulus terrestris*. Australian Veterinary Journal. 1984 Nov;61(11):360-3. Available from: <https://doi.org/10.1111/j.1751-0813.1984.tb07156.x>
20. Eastwood S. Tribulus staggers. Flock and Herd. February 2011. [cited 9 May 2022]; Available from: <http://www.flockandherd.net.au/edition/poisonousplants2005/tribulus-staggers.html>
21. Maher S. *Tribulus terrestris* staggers in the Coonabarabran area. Flock and Herd. December 2019. [cited 9 May 2022]; Available from:
<http://www.flockandherd.net.au/sheep/ireader/coonabarabran-staggers.html>
22. Bourke CA, Macfarlane JA. A transient ataxia of sheep associated with the ingestion of *Tribulus micrococcus* (yellow vine). Australian Veterinary Journal. 1985 Aug 1;62(8):282. Available from: <https://doi.org/10.1111/j.1751-0813.1985.tb14253.x>
23. Irwin T. Neurological cases in ruminants. Flock and Herd. September 2010. [cited 9 May 2022]; Available from: <http://www.flockandherd.net.au/other/reader/neurological.html>
24. McCann G. Crownbeard toxicity. Proceedings of the Australian Sheep Veterinarians Annual Conference The Australian Veterinary Association Proceedings of the 7th World Congress on Medical Informatics. 2012 Sep; Barossa Valley, South Australia; 2012. Available from:
https://www.ava.com.au/library-resources/library/conference-proceedings/2011/crownbeard-toxicity/Crownbeard_toxicity.pdf
25. Morrice G. Faba bean bloat in weaned lambs. Flock and Herd. October 2010. [cited 9 May 2022]; Available from: <http://www.flockandherd.net.au/sheep/reader/bloat-faba-beans.html>

Arboviruses, flaviviruses and the foetus

Robert Suter
Apiam Animal Health
Piper Lane, East Bendigo

Summary

An overview of the arboviruses affecting sheep, camelids & goats, discussing the pertinent epidemiology that leads to episodic outbreaks of arboviral disease in vertebrates.

Introduction

The climatic variability of south-eastern Australia has been immortalized in the line from Dorothea Mackellar's poem, 'My Country', first published in 1908, as being...

"A land of droughts and flooding rains".

When discussing arboviruses, especially those spread by flying insects who breed in warm, moist conditions, it is the distribution of those flooding rains over warmer months that delimits the geographical extent of their distribution and enables us to forecast the likelihood of disease events in naive and susceptible populations of animals.

Sporadic but Episodic Outbreaks of Arboviral Disease

The chief influence on the temporal occurrence of arboviral disease outbreaks in SE Australia is the climate variation ascribed to the *El Niño* Southern Oscillation (ENSO) phenomena which attempts to describe the regular alteration between hot- and cold- water- at either side of the Pacific Ocean: when there is hot water off the coast of Peru, there is cold water in the western Pacific and *vice versa*. Warm water in the Coral Sea increases the likelihood, in conjunction with other climate influences such as a similar, somewhat synchronous, but reverse, effect in the Indian Ocean, of increased spring and southern rainfall over south-eastern Australia. Conversely, droughts will grip that region, as Dorothea Mackellar described.

(Interestingly, the relationship between ENSO in the Pacific and the adjacent Indian Ocean means that when there are droughts in eastern Australia, the risk of the arboviral disease Rift Valley Fever occurring in Eastern Africa is heightened.)

The influence of this cycle of approximately seven-years length is most apparent in the Murray-Darling Basin (MDB), and Australia's native plants, animals, birds, and insects have adapted to this variability. Marsupials have adapted by utilizing embryonic diapause to accelerate population increase in good years (abundant rainfall and vegetation growth); similarly native waterbirds can rear several clutches of chicks in good seasons. In the drought years as the MDB dries up, these waterbirds retreat to where there is always water: The Coorong and Lake Alexandra at the mouth of the Murray in South Australia.

In the meantime, the *Refugia* for insects is Australia's tropical north, where it is almost continuously wet and warm, and can be viewed as an ecological continuum with SE Asia. Virologists are continually discovering more arboviruses in the Top End, with the consensus being that the newly discovered viruses have been blown in carried by their insect vectors (rather than having been overlooked in previous exploratory surveys). In this way, over the past five decades, the number of bluetongue (BTV) serotypes described in Australia has expanded to 16 (of the 24 recognised).

The viruses that have established themselves in northern Australia have found suitable vertebrate hosts in which to replicate (reservoirs), usually asymptotically, and locally adapted vectors to perpetuate spread (competent vectors). Their incubation period and the duration of viraemia and infectivity to their vectors varies between virus and reservoir host, influencing the pattern of disease to spill-over vertebrate species, who may exhibit clinically apparent disease. It is these spill-over events that are of interest and concern to medicos, veterinarians, and the wider human population, especially as humans may be the unsuspecting spill-over host, sometimes with life threatening consequences.

In *La Niña* years, the potential range of the insect vectors expands down the east coast of Australia as graphically illustrated by the National Arbovirus Monitoring Program's (NAMP) maps of the range of a principal vector of BTV: *Culicoides brevitarsis*. As the insects spread southwards, they bring with them their virus passengers, which has been historically described for Bovine Ephemeral Fever (BEF) as a progressive extension that usually requires successive *La Niña* seasons to reach cattle herds in northern Victoria. Here is a further example of the complexity: *C.brevitarsis* is a much more efficient vector of Akabane than of BTV, so doesn't require swarms to be spread. Thus, BTV would be more likely in a *La Niña* year with high insect numbers, whilst BEF is one of the more commonly occurring arbovirus diseases in SE Australia (Table 1).

Overlaying that, the potential for arboviral disease is influenced by livestock management practices and their inherent physiology. Livestock production in eastern Australia is concentrated on the western slopes of the Great Divide, which is the source of the rivers and streams that form the margin of the MDB. Arboviruses with a foetal tropism such as the flaviviruses and bunyaviruses can cause disease in animals that are pregnant during the late spring and summer, so disease is more likely to be seen in autumn lambing flocks. By contrast, Akabane disease is rarely seen autumn calving herds, as the arthrogryptic effects on the bovine foetus occur early in the cow's nine-month pregnancy, peaking at about 3-4 months, prior to the onset of the midge season. Veterinarians attending such flocks and herds should be attentive to the potential of arboviral disease in *La Niña* seasons, especially when confronted with uncommon neurological, reproductive, or teratogenic signs.

The relative immunity of populations of these spill-over species also affects disease expression. After exposure, many animals are asymptomatic, but develop protracted immunity, which could be perpetuated if subject to constant exposure. The nature of ENSO means that this exposure doesn't occur, and by the next *La Niña* there is often an entire population of naive and thus susceptible animals, should that vector and virus be reintroduced.

A note of caution: many of these arboviruses are known to cause disease in people, usually through infection via the vector, but caution should be the catchcry when handling any aborted or malformed foetus.

Arboviral Disease and the Foetus

For arboviral disease to occur, a triad of predisposing factors are necessary: the presence of a viraemic animal (often in abundance), and of a competent vector (in abundance), as well as a naive spill-over population. This interplay of factors tends to result in different arboviruses causing disease in different vertebrate species in each *La Niña* year, and it may be several ENSO cycles later before a reoccurrence is recognised.

In multiparous species (those having litters) such as pigs, the reproductive impact of intrauterine infection is described by the acronym SMEDI: stillbirth, mummification, embryonic death. In sheep, which have small litters, and which tend to be managed extensively in Australia, the opportunity to observe this peculiar range of clinical signs is limited.

There are four pathophysiological mechanisms proposed to account for these SMEDI outcomes: a severe maternal infection leading to pregnancy loss (as seen with Bluetongue virus – BTV); placental infection alone which may result in pregnancy loss or low viability foetii; both placental and foetal infection (resulting in congenital defects as may be seen with Akabane or flaviviruses); or infection at the time of birth (leading to high perinatal mortality). Given that, in general, the period of viraemia is short-lived, how this combination of effects manifests in a flock naturally mated may mean that this overall picture is overlooked.

A study infecting ewes by the intramuscular injection with ovine Herpes virus (Border disease) at day 68 of pregnancy gives some clues as to the range of outcomes that could be expected. In these twin bearing ewes, the outcome was seven mummified foetii, four that might be classified as stillborns (or prematures in lay language) and one viable lamb that was a 'hairy shaker'. If assessed at lamb marking, there would be a large discrepancy between the results at lamb marking and the expectations after pregnancy scanning in these ewes.

The Arboviruses of Concern

This listing is not exhaustive; just as we are recognising evermore viruses present in Australia's Top End, we will undoubtedly discover more foetal impacts over time due to other viruses. Any described as exotic are Emergency Animal Diseases, and thus Notifiable immediately upon suspicion.

Togaviruses

Ross River virus

- Australia-wide
- Marsupials, unconfirmed vectors
- Zoonoses, man, horses
- Neurological disease

Bunyaviridae

Rift Valley Fever virus (exotic)

- Eastern Africa and Arabian Peninsula
- Cattle, *Aedes* mosquitoes, drought persistence infected eggs.
- Sheep, goats, cattle, buffalo, equids, dogs and cats, camels
- Abortion
- Zoonoses, abattoir workers and vets at high risk through contact with fluids

Akabane

- Asia and Australia
- Uncertain reservoir, *Culicoides brevitarsus*
- Cattle, sheep and goats
- Arthrogryposis

Aino

- Closely related to Akabane (Orthobunyavirus)
- Similar epidemiology
- Disease in cattle - Arthrogryposis

Schmallenberg (exotic)

- Emerged in Europe in 2011
- Uncertain reservoir, *Culicoides* Obsoletus complex
- Sheep, goats, cattle
- Arthrogryposis

Nairobi sheep disease (exotic *Nairovirus*)

- East and Central Africa
- Reservoir is ticks, *Rhipicephalus appendiculatus*
- Sheep and goats
- Systemic illness resulting in abortion

Cache Valley virus (exotic)

- North and Central America
- Ruminants (including deer) and horses, mosquitoes
- Sheep, goats, cattle
- Infertility, arthrogryposis, CNS pathology – 36-45 days of gestation
- Human encephalitis

Reoviridae

Bluetongue virus (Orbivirus; Emergency Animal Disease – EAD)

- Top End of Australia and the bluetongue zone around the globe (equatorial regions)
- Cattle and buffalo, *Culicoides* midges
- Sheep and goats, camelids

- Abortion amongst systemic signs of vasculitis, rarely CNS defects

Rhabdoviruses

Bovine Ephemeral Fever

- No recorded foetal effects

Flaviviruses

Wesselbron virus (exotic)

- Southern Africa
- Domesticated herbivores, *Aedes* mosquitoes
- Ruminants, pigs, equids, ostriches
- Hepatitis in lambs, abortion, arthrogryposis, CNS defects.

Japanese encephalitis complex (includes Kunjin – West Nile virus and Murray Valley encephalitis virus)

- SE Asia and tropical Australia
- Waterbirds, *Culex* mosquitoes
- Mammalian species
- Encephalitis in horses and man, SMEDI in pigs

Both Zika virus and Venezuelan equine encephalitis viruses are mosquito-borne Flaviviruses that cause congenital defects in humans. Both are exotic to Australia.

Table 1. Outbreaks of arboviral disease in south-eastern Australia and the associated *La Niña* years.

| <i>La Nina</i> years | Viruses causing diseases (Summer period seen) | Species of vertebrate affected |
|----------------------|---|--|
| 1903-04 | | |
| 1906-07 | | |
| 1909-11 | | |
| 1916-18 | | |
| 1924-25 | | |
| 1928 | | |
| 1938-39* | BEF - 1936-37 | Cattle |
| 1942-23* | | |
| 1949-51 | MVE -1951 Akabane - 1951 | People Cattle |
| 1954-57 | Akabane - 1955 BEF - 1955-56 RRV - 1956 | Cattle Cattle People |
| 1964-69 | Akabane - 1964 & 1968 BEF - 1967-68 | Cattle Cattle |
| 1973-76^ | MVE -1974 Akabane - 1974 BEF - 1972-74, 1974-76 Wallal orbivirus 1975 Aino 1975 | People Cattle Cattle Kangaroo blindness Cattle |
| 1988-89 | | |
| 1994-95 | Wallal orbivirus | Kangaroo blindness |
| 1998-99 (but weak) | | |
| 2007-09 (also weak) | | |
| 2010-12 (wettest) | BEF - 2010 MVE -2011 Kunjin & RRV | Cattle People, horses Horses |
| 2016 (mild) | RRV | Horses and people |
| 2020-22 | JE - 2022 | Pigs, people. An alpaca. |

| <i>La Nina</i> years | Viruses causing diseases (Summer period seen) | Species of vertebrate affected |
|----------------------|--|--------------------------------|
| | BEF - 2022 | Cattle |

* Weak *La Nina* years with little rainfall; 1937-47 drought throughout WWII. ^ Wettest period of the 20th century.

BEF: Bovine ephemeral fever; MVE: Murray Valley encephalitis; RRV: Ross River virus; JE: Japanese encephalitis virus.

Dates from Wikipedia (accessed 3 May 2022) [Effects of the El Niño–Southern Oscillation in Australia - Wikipedia](#)

Impact of ewe nutrition on healthy bones and disease prevention

Colin L. Trengove

School of Animal and Veterinary Sciences, The University of Adelaide, Roseworthy, SA

Preamble

This paper will discuss how grazing management and supplementation affects bone health and consequent disease susceptibility in ewes and lambs. Diseases associated with suboptimal bone health include uterine inertia, dystocia, osteoporosis, osteomalacia, bone fractures, hypocalcaemia, hypomagnesaemia, and pregnancy toxaemia. Dystocia alone is estimated to cost the Australian sheep industry \$780 million annually or \$23 per ewe joined based on a lamb sale price of \$6.50/kg carcass weight. This cost includes an estimated 35% of all ewe deaths and 54% of all lamb deaths attributed to dystocia. While the cause of dystocia is multifactorial failure to recognize and address mineral deficiency in the diet of late pregnant ewes is a major contributing factor. The flow-on effect on reduced lamb growth rate and bone fractures is conservatively estimated to cost the sheep industry a further \$20 million annually. Calcium is vital to bone, muscle and neural development and function in all mammalian species. Approximately 70% of lamb calcium and phosphorus requirements are derived from maternal bone resorption and 30% from the diet during early lactation. It is facilitated by an estimated 20% of accreted maternal bone calcium being mobilised during pregnancy to compliment dietary intake in meeting the needs of the developing foetus. The consequences of failure to mobilise or absorb sufficient calcium during mid to late pregnancy include hypocalcaemia, osteoporosis, osteomalacia and uterine inertia in the ewe as well as poor growth rates and weak bones in lambs. The epidemiology of these sequelae and strategies to prevent them will be discussed.

Introduction

Growing pasture to feed livestock is by far the cheapest option for a livestock enterprise all year round being approximately 15 times cheaper than feeding grain or hay. However, essential to long term profitability and sustainability is a practical knowledge of livestock nutrition combined with grazing management skills. Climate variability, topography, rainfall, and soil type all add to the complexity of successfully managing livestock in a grazing system. Profit drivers such as wool and/or meat produced per hectare can be used as a gauge of enterprise performance in the short term and longer-term measures may include regular strategic soil analyses and pictorial records to assess the sustainability of a farm.

The more immediate concern addressed in this paper is the failure to meet the nutritional needs of the late pregnant ewe and impending lambs. In particular, the calcium requirements so that diseases associated with suboptimal bone health including uterine inertia, dystocia, osteoporosis, osteomalacia, bone fractures, hypocalcaemia, hypomagnesaemia, and pregnancy toxaemia can be prevented.

Discussion

Calcium homeostasis

Osteoporosis has been defined as a disorder apparent from two to twelve months of age in lambs grazing pasture in south-eastern Australia evident as reduced bone mass per unit of bone volume.¹ The complex aetiology includes response of bone to minerals (calcium, phosphorus, and copper), vitamin D, malnutrition, and parasitism.^{2,3}

It is recognized that foetuses have a much higher plasma calcium concentration than the ewe during the last trimester of pregnancy enabling a placental calcium gradient to be established. This differential is from 2.15 mmol/L in the ewe to 2.58 mmol/L in the lamb from as early as 90 days of gestation.⁴ Studies have shown that approximately 200 mg Ca/kg foetal weight/day is transported across the placenta in late pregnancy.⁵ Field and Suttle⁶ found that up to 97% of Ca was deposited in the foetal skeleton after day 80 of gestation. This has been demonstrated by thyroparathyroidectomized foetuses having a 50% reduction in placental calcium transport resulting in foetal hypocalcaemia.⁷ Normal bone development in foetuses requires a placental calcium gradient to be established through the influence of parathyroid hormone (PTH) and a

related protein (PTHrP) as well as foetal hypercalcaemia. This is independent of the ewe's Ca status and the foetal plasma calcium ideally remains elevated for several months after birth.

However, young lambs remain susceptible to osteoporosis if their milk and calcium intake is compromised in their first few months of life. Prior to birth PTHrP from the foetal parathyroid gland acts on a placental pump to maintain high plasma calcium. Following birth maintenance of high plasma calcium is at the expense of bone quality if milk and calcium intake are inadequate leading to osteoporosis.

In ruminants, with sufficient dietary Ca supply, vitamin D stimulates bone formation and mineralization, whereas if Ca serum level drops, vitamin D enhances mobilization from the bones and intestinal Ca absorption.^{8,9,10} Ruminant Ca absorption plays an important role in Ca homeostasis in ruminants, but the underlying mechanisms are still not fully understood. Assuming that Ca homeostasis in ewes has already been severely challenged before parturition, one would expect a calcitriol (active hormonal form of vitamin D) response occurring even earlier in these animals. However, this is not the case. It has been shown for sheep that the maximal efficiency of gastrointestinal Ca absorption is reached two months after parturition, although bone resorption has already been stimulated before lambing.⁵

Naturally occurring rickets is uncommon in sheep in the United Kingdom.^{11,13,14,15}, but outbreaks associated with high latitude, low sunshine hours, and poor-quality pasture low in phosphorus have occurred in Scotland.^{11,15} In Ireland, rickets has occurred in sheep fed oats in winter months and, in northern England, in sheep grazing lush ryegrass swards over winter, most likely because of the rachitogenic effect of carotenes.^{13,15} These same circumstances are also regularly reported in southern Australia.^{16,17}

The serum vitamin D concentration of lambs has been shown to be closely related to the vitamin D concentration of the dam.^{18,19} Lambs at 3 to 4 weeks predominantly rely on milk, which may have low vitamin D concentrations if the vitamin D status of the dam is inadequate.²⁰ Vitamin D is intimately involved in calcium and phosphate homeostasis, and it plays a crucial role in bone formation and remodeling. Deficiency of calcitriol, whether due to environmental or genetic factors, may result in rickets or osteomalacia secondary to hypocalcaemia and hypophosphataemia. The hypocalcaemia may also lead to hyperparathyroidism and concurrent fibrous osteodystrophy.

Nemeth et. al.²¹ were able to demonstrate that, like cows, growing dairy goats and sheep are able to compensate a vitamin D-reduced diet by producing vitamin D within their skin while exposed to UVB irradiation and therefore to keep a high vitamin D status and a balanced Ca homeostasis. Milk can also be an adequate source of vitamin D for newborn ruminants under the condition of a high vitamin D status in combination with a moderate milk yield of the dam.^{22,23,24} The calcitriol can then be stored in the body tissue (primary fat, muscle, and liver) to help prevent a fast drop of vitamin D status.^{25,26} A sufficient vitamin D status, combined with a normal Ca availability, leads to the embedment of minerals in the bone and these lead to an increase of bone mineral density.²⁷ This ensures good bone mineral density and strength in the growing lamb and enables ewes to accrete bone mineral reserves post-weaning ready for the next pregnancy.

Feed associated calcium disorders

In southern Australia pregnant ewes are predominantly grazed on dry standing grasses and/or cereal stubbles for an autumn lambing or green feed consisting of grasses and legumes for a winter/spring lambing. This will vary with seasonal conditions and annual rainfall. Containment feeding has also become increasingly popular when autumn feed is scarce and low quality, or there is a late break to the winter season.

The availability of minerals important for ewe health (especially calcium, magnesium, phosphorus, and copper) as well as vitamin D also varies with seasonal conditions and grazing management. The dietary cation anion difference (DCAD) also influences mineral balance, especially plasma Ca, due to excess cations ($\text{Na}+\text{K}-\text{Cl} > 50 \text{ meq}/100\text{g DM}$) inducing a metabolic alkalosis and lowering urinary and blood Ca.²⁸ Legumes and broadleaves are also alkalogenic due to a relatively high DCAD (high cation (Na & K) and low anion (Cl & S) content) compared to

conserved fodders and silage.^{29,30} Block³¹ found spring lambing ewes had lower plasma Ca compared to autumn lambing ewes like the impact of feeding cationic and anionic salts. However, late pregnant ewes absorb Ca with similar 50% efficiency whether it is green fresh forage or conserved hay or grain. He also found that diets low in Ca and with a low DCAD may contribute to hypocalcaemia due to Ca mobilization as well as poor absorption and loss of Ca in urine. Vitamin D given to ewes pre-lambing did not raise plasma Ca and P but did improve lamb survival at 12 weeks old.

Other causes of hypocalcaemia at pasture include grazing oxalates such as sour sob (*Oxalis pes-caprae*) and sorrel (*Rumex spp.*); grazing green cereals low in Ca, Mg, and Na; high sugar and water plus low fibre diets predisposing to osmotic diarrhoea as well as less salivary Na and P to encourage rumen absorption of Ca and Mg; and vitamin D deficiency. In addition, grazing winter pastures high in carotene are known to cause osteodystrophy's in lambs.³²

Dietary supplements including dicalcium phosphate, magnesium oxide and calcium carbonate equivalent to 10g Ca effectively raised plasma Ca six hours post administration.³¹ This resulted in the recommendation to monitor DCAD and ensure ewes are supplemented pre-lambing if this is low. In addition, lambs should also be supplemented if their growth rate is less than 150 g/day from birth to weaning.

Conclusion

Ewe and lamb susceptibility to the various diseases mentioned can be prevented by attention to detail requiring regular monitoring of condition score to assess their energy stores and mineral balance especially during mid to late pregnancy. Pregnant ewes on dry feed diets including predominantly grain diets should be supplemented to ensure sufficient Ca is available (maintain Ca:P \geq 1.5). Ewes on green feed during pregnancy should have their DCAD monitored, and minerals and fibre supplemented to ensure Ca homeostasis. Lamb growth rate should be monitored from birth to weaning and supplements provided if they are not achieving at least 150 g/day. Ideally, vitamin D status should also be checked and supplemented if ewes and lambs are found to be at risk.

References

1. Palmer NC. Nutritional imbalances relating to bone disorders in large domestic animals. *Vic Vet Proc* 1969-70; 55-57.
2. Franklin MC. Vitamin D requirements of sheep with special reference to Australian conditions. *Aust Vet J* 1953; 29:302-309.
3. Suttle NF, Angus KW, Nisbet DI, Field AC. Osteoporosis in copper depleted lambs. *J Comp Path* 1972; 82:93-97.
4. Mellor DJ, Matheson IC. Variations in the distribution of calcium, magnesium, and inorganic phosphorus within chronically catheterized sheep conceptuses during the last eight weeks of pregnancy. *Quarterly J Exp Physiol*. 1977; 62:55-63.
5. Braithwaite GD, Glascock RF, Riazuddin SA. Studies on the transfer of calcium across the ovine placenta and incorporation into the foetal skeleton. *Brit J Nutr*. 1972; 27:417-424.
6. Field AC, Suttle NF. Retention of calcium, phosphorus, magnesium, sodium, and potassium by the developing sheep foetus. *J Agr Sci* 1967; 69:417-423.
7. Care AD, Dutton A, Mott JC, Robinson JS, Ross R. Studies of the transplacental calcium gradient in the sheep. *J Physiol*. 1979; 290:19P-20P.
8. Liesegang A, Risteli J. Influence of different calcium concentrations in the diet on bone metabolism in growing dairy goats and sheep. *J Anim Physiol Anim Nutr (Berl.)*. 2005; 89:113-119. 15787981.
9. Schröder B, Breves G. Mechanisms, and regulation of calcium absorption from the gastrointestinal tract in pigs and ruminants: Comparative aspects with special emphasis on hypocalcemia in dairy cows. *Anim Health Res Rev* 2006; 7:31-41. 17389052.
10. Dittmer KE, Thompson KG. Vitamin D metabolism and rickets in domestic animals: A review. *Vet Pathol*. 2011; 48:389-407. 20634407.
11. Bonniwell MA, Smith BSW, Spence JA, Wright H, Ferguson DAM. Rickets associated with vitamin D deficiency in young sheep. *Vet Rec*. 1988; 122:386-388.
12. Ewer TK. Rickets in sheep: field trials in East Anglia. *Vet Rec*. 1950; 62:603-606.

13. Mearns R, Scholes SFE, Wessels M, Whitaker K, Strugnell B. Rickets in sheep flocks in northern England. *Vet Rec.* 2008; 162:98–99.
14. Nisbet DI, Butler EJ, Smith BSW, Robertson JM, Bannatyne CC. Osteodystrophic diseases of sheep: II. Rickets in young sheep. *J Comp Pathol.* 1966; 76:159–169.
15. Crowley JP. Rickets in November-born lambs. *Vet Rec.* 1961; 73:295–297.
16. Grant I. Disorders of calcium and bone metabolism in the ewe. PhD thesis, Uni Melb. 1998.
17. Watt B. Bone problems in lambs on grazing cereals. *Flockandherd.net.au.* August 2008. Accessed 09.06.22.
18. Smith BSW, Wright H. Seasonal variation in serum 25-hydroxyvitamin D concentrations in sheep. *Vet Rec.* 1981; 109:139–141.
19. Smith BSW, Wright H, Brown KG. Effect of vitamin D supplementation during pregnancy on the vitamin D status of ewes and their lambs. *Vet Rec.* 1987; 120:199–201.
20. Van Saun RJ. Vitamin D-responsive rickets in neonatal lambs. *Can Vet J.* 2004; 45:841–844.
21. Nemeth MV, Wilkens MR, Liesegang A. Vitamin D status in growing dairy goats and sheep: Influence of ultraviolet B radiation on bone metabolism and calcium homeostasis. *J Dairy Sci.* 2017; 10:8072-8086.
22. Kohler M, Leiber F, Willems H, Merbold L, Liesegang A. Influence of altitude on vitamin D and bone metabolism of lactating sheep and goats. *J Anim Sci.* 2013; 91:5259-5268. 24045489.
23. Weiss WP, Azem E, Steinberg W, Reinhardt TA. Effect of feeding 25-hydroxyvitamin D3 with a negative cation-anion difference diet on calcium and vitamin D status of periparturient cows and their calves. *J Dairy Sci.* 2015; 98:5588-5600. 26051311.
24. Hymøller L, Jensen SK, Kaas P, Jakobsen J. Physiological limit of the daily endogenous cholecalciferol synthesis from UV light in cattle. *J Anim Physiol. Anim. Nutr. (Berl.).* 2017; 101:215-221. 27421247.
25. Hidioglou M. Kinetics of intravenously administered 25-hydroxyvitamin D3 in sheep and the effect of exposure to ultraviolet radiation. *J Anim Sci.* 1987; 65:808-814. 3667442.
26. Hidioglou M, Karpinski K. Providing vitamin D to confined sheep by oral supplementation vs ultraviolet irradiation. *J Anim Sci.* 1989; 67:794-802. 2542212.
27. Liesegang A, Hüttenmoser D, Risteli L, Leiber F, Kreuzer M, Wanner M. Influence of high-altitude grazing on bone metabolism of growing sheep. *J. Anim Physiol Anim Nutr (Berl.).* 2013; 97:58-66. 21992062.
28. Caple I, Shovelton J. unpublished.
29. Scott D. Changes in mineral, water and acid-base balance associated with feeding and diet. In *Digestion and Metabolism in The Ruminant.* Ed. McDonald IW, Warner ACI. Proc 4th International Symp on Ruminant Physiol, Uni of New England Publ, Armidale NSW. 1975; 205-215.
30. Fredeen AH, DePeters IJ, Baldwin RL. Characterization of acid-base disturbances and effects on calcium and phosphorus balances of dietary fixed ions in pregnant or lactating does. *J Anim Sci.* 1988; 66:159-173.
31. Block E. Anion-cation balance and its effect on the performance of ruminants. In *recent advances in animal nutrition.* Eds Haresign W, Cole DJA, Butterworths, London. 1991; 163-179.
32. Grant AB, O'Hara PB. The rachitogenic effect of vitamin A. *NZ J Sci Tech.* 1957; 38A:548-576.

Is regenerative agriculture a myth or critical to the survival of domestic herbivores?

C. L. Trengove

School of Animal and Veterinary Sciences, The University of Adelaide, Roseworthy, SA 5371, Australia.

This paper will discuss how grazing management affects mineral and vitamin nutrition of plants and domestic herbivores. The focus will be on how the grazer can sustainably maintain soil, plant, and animal health through both organic and supplementary means. Domestic herbivores are vital to the livelihoods of more than 10% of the global population through the conversion of low-quality plant material into valuable meat, milk, fibre, and labor. Careful attention to their nutritional management underpins their environmental, social, and economic value. The nutrient consumed while grazing is dependent on access to sufficient feed as determined by the grazer, the animal's physiological state, and the variety plus physical and chemical characteristics of plants available. Herbivores need to consume sufficient dry matter to meet physiological demands as well as a balance of essential nutrients and negligible toxins. Supplements are used to meet these needs when grazing options are limited either due to suboptimal grazing management or seasonal and climatic conditions. Most experienced graziers recognize the quantitative nutritional needs of livestock, but few know the specific nutritive needs or how best to provide them. It is all too common to see domestic herbivore health suffer due to human failure to appreciate and provide their nutritive needs over a few weeks to several months. The merits of various grazing management strategies will be discussed as well as the consequences for nutrient availability and how the grazer can be trained to proactively recognize and address these needs before livestock health and productivity are compromised. This will include key nutrient guidelines for optimal health maintenance in soil, plants, and herbivores.

Clinical manifestations of selenium deficiency in sheep (and goats)

Bruce Watt,
Central Tablelands Local Land Services, Bathurst NSW

Introduction

Much of the Central Tablelands of NSW is selenium deficient causing ill-thrift, nutritional muscular dystrophy ((NMD) or white muscle disease) and potentially, embryonic mortality¹. NMD, the most obvious manifestation, is uncommon but was seen in lambs, calves and kids in the late spring and early summer of 2020.

Case Presentations

The two cases of NMD in lambs occurred in November 2020. In the first case, 12 of 280 merino lambs mustered for marking were clinically affected and another 6 were found dead. In the second case², 12/120 composite lambs, also mustered for marking, were clinically affected and another 3 were found dead. Affected lambs were reluctant to walk and when moved, walked with short steps and an arched back.

In the first case, 5 affected lambs had glutathione peroxidase (GSHPx) levels ranged from 5-9 umol/L while in the ewes GSHPx levels ranged from 4-11 U/gHb. Creatinine kinase (CK) levels ranged from 10, 311 to 25,301 units. In the second case GSHPx levels ranged from 10-36 in the lambs and 26-103 in the ewes. CK levels ranged from 3492 to 4053 U/L in the affected lambs.

In mid-December 2020 between 10-30 deaths in Boer kids were attributed to WMD. Necropsy revealed chronic myocardial and skeletal muscle necrosis and mineralisation and peri acinar liver necrosis consistent with congestive heart disease. This case was not able to be confirmed by testing the selenium status of the does or kids.

Conclusions

Ill-thrift is probably the most common manifestation of selenium deficiency seen in sheep on the Central Tablelands but is often sub-clinical. Clinically normal merino lambs have showed an improved liveweight, wool growth and reduced worm egg count as demonstrated by a response trial we conducted about 45 km east of Bathurst in March 2010 on wether merino weaners. This farm has improved cocksfoot, phalaris and subterranean clover pastures on granite derived soils. Superphosphate is applied regularly. While the merino weaners were only marginally deficient in selenium (GSHPx levels < 40 U/gHb) at the onset of the trial they had improved growth rates (with a maximum difference of 2.86 kg bodyweight), fleece weights (2.75 versus 2.93 kg per head and a lower mean worm egg count³.

Embryonic mortality has been demonstrated in some years in Merino ewes in the New England region of NSW⁴. While it no doubt occurs occasionally it not been proven on the Central Tablelands.

NMD was common on the Central Tablelands in the mid-1970s following the widespread application of superphosphate in favourable, clover dominant springs⁵. but is now rare despite widespread selenium deficiency. In general, pastures are now perennial grass dominant although in the spring of 2020, following the breaking of a lengthy drought, pastures were temporarily subterranean clover dominant.

The cardiac form of NMD presented as sudden death in both lambs and calves in 2020 and has been seen in calves on the Central Tablelands previously⁶. Of interest, the (unconfirmed) case in kids presented as chronic heart failure. As mentioned, the skeletal muscle form seen in lambs and calves in 2020 presented as reluctance to walk, an arched back and a stiff, short stepping gait. The other causes of myopathy in lambs on the Central Tablelands, vitamin E deficiency and marshmallow poisoning have the same presentation but can be differentiated on history and laboratory findings. All have markedly increased CK levels but in selenium deficiency induced NMD, GSHPx levels are low in both the lambs and the accompanying ewes.

References

1. Watt BR, and Eppleston J, Selenium nutrition of sheep and cattle. Posted on Flock and Herd in March 2011, <http://www.flockandherd.net.au/other/reader/selenium-nutrition.html>
2. Bellingham P and Watt BR. White muscle disease in spring born lambs on clover dominant Central Tablelands pasture. Posted on Flock and Herd in May 2021.<http://www.flockandherd.net.au/sheep/ireader/white-muscle-disease.html>
3. Celi P, Watt B, Eppleston J, and Armstrong AB, (2010). The influence of selenium supplementation on parasite infection and sheep and wool production in weaner sheep. *Animal Production Science*, 50, pp 688-692.
4. Wilkins, J F, Kilgour R J, Gleeson A C, Cox, RJ, Geddes, S J, & Simpson I H, (1982). Production responses in selenium supplemented sheep in northern New South Wales. 1. Infertility in ewes and associated production. *Australian Journal of Experimental Agriculture and Animal Husbandry*, 22(114), 18-23 www.publish.csiro.au
5. Watt BR and Staples P, The myocardial form of nutritional muscular dystrophy in calves on clover dominant pastures. Posted on Flock and Herd in December 2012.<http://www.flockandherd.net.au/cattle/reader/cardiomyopathy-calves.html>
6. Dellow B, Personal Communication

Optimising reproduction success through pre joining ewe assessments

Andrew Whale
Livestock Logic, Apiam Animal Health
Hamilton, Victoria

Background to the fit-to-join resources

Often sheep producers are frustrated they cannot achieve their marking percentage goals, even though they're implementing industry best practice in the lead up to, and during, lambing. An underlying reason for this may be they are joining ewes unfit to rear a lamb.

A review of existing literature and industry information, and coordinated ewe selection case studies on three commercial farms, confirmed an opportunity to improve lamb and ewe survival outcomes by rigorous ewe assessment and selection before joining.

Project methodology

Ewes were classed pre-joining and then followed through scanning and lamb marking. Ewe and lamb mortality, lamb marking rate and scanning percentages were analysed, along with the economic cost-benefit of ewe selection pre-joining.

The results

The investigation found that unfit ewes had a 4.4x higher risk of scanning empty and a 3x higher risk of dying between joining and scanning. Lambs from unfit ewes had a 21% higher risk of dying (Table 1). These translated into significant modelled economic benefits for classing and culling ewes as unfit to join of between \$4 and \$8 per ewe.

Table 1. Average ewe mortality and marking results by ewe class

| | Average ewe mortality | Average marking percentage (%)* |
|---------------|-----------------------|---------------------------------|
| Fit to join | 2.2 | 137 |
| Unfit to join | 2.7 | 123 |

* marking percentage is calculated as lambs marked from ewes joined

Assessing ewes as 'fit to join' offers sheep producers an opportunity to increase ewe and lamb survival rates, productivity and profitability while improving animal welfare outcomes. On top of that, the process can be fast, efficient and cost-effective.

Risk factors

The range of risk factors that impact of ewe and lamb survival include: body condition, udder health and structure, lameness, teeth and age.

While many producers have adopted routine condition scoring as a key management tool, fewer producers routinely assess and address issues such as udder health and structure, lameness and age-related risks.

Udder issues

Udder health and structure is a key indicator of a ewe's ability to successfully rear a healthy lamb. Ewes with unsound udders are not fit to join as they are unlikely to raise a healthy lamb, which has significant animal welfare and productivity implications. Producers can assess udders quickly in the race through physical palpation and visual assessment.

Foot health

Foot health plays an important role in fitness of ewes to join, with significant production losses attributed to lameness, including loss of body condition and predisposition to metabolic disease during late pregnancy. Identifying and addressing foot-related conditions in ewes prior to joining will increase animal welfare outcomes and improve productivity and profitability of the sheep enterprise.

Mouth and teeth

While there is insufficient evidence to supporting culling ewes based solely on a broken or gummy mouth, loose and/or uneven incisors are likely to impact body condition and reproductive performance. Following an initial assessment of body condition, investigating the mouths and teeth of ewes with a BCS 0.5 less than the mob average could identify issues that exclude them as fit to join. It is generally worth focusing on the mouths and teeth of older ewes (>5yrs) as younger ewes in good condition (BCS ≥ 3.5) are unlikely to have loose, broken or uneven teeth, unless they have been subject to prolonged drought conditions.

Risks associated with age

The standard industry practice of 'cast-for-age' culling is not supported by evidence. When assessed on their merits, with age as one, but not the 'overriding', factor in the mix, healthy and sound older ewes offer a valuable breeding proposition to any sheep enterprise, particularly for producers trying to build their breeding flock

Older sheep with no other underlying issues (body condition score, feet, teeth) can have the same reproductive performance as sound, younger ewes, providing risk factors are managed.

While older ewes can be at increased risk of mortality (e.g. hypocalcaemia), this can be largely addressed by improving nutrition and monitoring for symptoms of declining health (e.g. condition score).

What's hot in sheep industry sustainability reporting

Dr Scott Williams
Chair, Sheep Sustainability Framework Steering Group
c/- Meat & Livestock Australia
North Sydney NSW

Introduction

The Sheep Sustainability Framework (SSF or The Framework) was launched in April 2021. The SSF is a joint initiative of Sheep Producers Australia (SPA) and WoolProducers Australia (WPA) and is managed by Meat & Livestock Australia (MLA) with co-funding from Australian Wool Innovation (AWI). The process to develop the SSF and the material issues identified during the process were described at last year's Sheep, Camelid and Goat Veterinarians Conference.¹ This paper describes main developments of the SSF since its launch.

Implementation of The Framework

The SSF is overseen by a skills-based steering group, whose membership was refreshed in late 2021. Members have expertise in wool and lamb production, meat processing, the wool value chain, retail, and the finance sector. The SSF Steering Group has already been invaluable in providing sustainability perspectives on the sheepmeat and wool value chain.

The SSF Steering Group reports to the SSF Board that comprises representatives of SPA and WPA, and an independent chair. The service bodies, MLA and AWI, participate as observers on the SSF Board.

The SSF Board recently approved a three-year strategic plan for the implementation of The Framework. The plan comprises three strategies:

- Stakeholder Engagement, which seeks to ensure that the SSF is both understood and informed by a broad range of stakeholders throughout the value chain – including sheep vets;
- Data Collection and Reporting, to provide easily-accessible, user-friendly and regularly updated data that reports industry progress against key measures; and
- Continuous Improvement, including review and adjustment of metrics, informed by periodic deep-dives into contemporary and emerging trends in sustainability.

The first annual report of the SSF will be launched in July 2022 at the Australian Sheep and Wool Show in Bendigo. The report will present data on the SSF metrics as well as case studies on highly material issues such as protection against breech flystrike.

Collaboration with other Sustainability Frameworks

Sustainability frameworks are becoming a common feature of the Environmental and Social Governance (ESG) landscape. Within agriculture, these include the Australian Beef Sustainability Framework (ABSF)² (also managed by MLA) and the Australian Grains Industry Sustainability Framework ('Behind Australian Grain')³.

The Australian agricultural sectors share many common areas of interest when it comes to describing their sustainability performance. The livestock industries, for example, all seek to report on the welfare of the animals under their care and the health of the natural environment in which they operate. All of agriculture is concerned for the wellbeing of its people and communities.

The SSF Steering Group is very conscious that sheep enterprises commonly co-exist with cropping, beef cattle or other enterprises. Multi-enterprise farms want, as far as possible, a single set of metrics to describe all aspects of their operations. Also, having common metrics across industries provides harmonisation and economies of scale in collecting and reporting data.

For these reasons, the SSF collaborates closely with the ABSF and will increasingly do so with other sustainability frameworks.

At the whole-of-agriculture level, the Australian Farm Institute has been commissioned by the National Farmers' Federation with funding from the Commonwealth Government to develop the Australian Agricultural Sustainability Framework (AASF)⁴. The AASF is not intended to replace individual industry frameworks. Instead, it is intended to harmonise existing schemes, identify high-level principles and goals, and provide guidance for the development and refinement of industry frameworks. The SSF is working closely with the AASF.

Major Recent Developments

Three major projects to support the SSF are in progress:

1. Project Proof

Project Proof is a survey of 2000 sheep producers conducted during May 2022. The survey will collect and track data for on-farm husbandry practices, including several that form metrics in the SSF, such as the national incidence of pain management, pregnancy scanning and vaccination. The survey will be repeated every three years. It is a collaboration with the ABSF and funded by MLA and AWI.

2. Life Cycle Assessment for the Australian Flock

AWI and MLA have jointly commissioned the first life cycle assessment (LCA) for the Australian flock. A working model of the Australian sheep industry has been developed to understand and report carbon emissions and emissions intensity from sheep meat and wool on an industry-wide scale.

The LCA is a critical piece of work. The debate about the role of ruminants and their products in climate change is complex, contested and poorly understood. A recent publication by CSIRO⁵ concluded that the Australian sheepmeat industry is climate-neutral. That means that the sector is making no contribution to global temperature increases and, further, its impact is in decline. Notwithstanding, the wool industry currently holds great concerns about an EU initiative, the Product Environmental Footprinting (PEF) project. The PEF will assist consumers to make purchases based upon a product's environmental sustainability. The PEF ratings are built on a methodology that currently suggests synthetic fibres – including polyester, which is derived from petrochemicals – are more sustainable than natural fibres including wool⁶.

3. Groundcover Mapping

It is challenging to identify metrics, to demonstrate how well the sheep industry (and indeed other grazing sectors) manage their natural resources, that are able to be monitored easily and cost-effectively and across all production systems.

Vegetation cover is one such metric. Whilst not perfect surrogate for soil protection, it provides a partial indication of the management of our soil resource. The SSF has partnered with the ABSF to visualise and analyse 30 years of satellite imagery over Australian grazing land. The technology informs the fractional groundcover metric for the SSF, demonstrates the impact of management on long term trends in ground cover and tree cover and assesses trends in land condition to guide decisions on long-term sustainable carrying capacity.

What's next?

ESG is dominating the discussions of many companies in the modern economy and there is little doubt that it will continue to grow in importance. Consumers are increasingly looking beyond the feature, function and benefit of a product or service, and routinely evaluate sustainability credentials such as the environmental and social impacts of the offering. Lamb and wool are now premium products rather than the everyday staples they used to be. Superior sensory characteristics such as eating quality and tactility must be supported by a value proposition of highly ethical production.

Agricultural sustainability frameworks, including the SSF, are in their relative infancy. There remain significant challenges in ascribing measures to even core elements of the four SSF themes: Caring for our Sheep; Enhancing the Environment and Climate; Looking after our People, our Customers,

and the Community; and Ensuring a Financially Resilient Industry. For example, how do we measure biodiversity? Or the physical and mental health of our people?

Any new concept such as sustainability reporting must start somewhere, though. The SSF provides a robust platform on which to build a comprehensive picture of the sustainability credentials of Australian sheepmeat and wool production. Working with other frameworks, it will strengthen over time.

References

1. Allworth B. The Australian Sheep Sustainability Framework – a world first. In: Proceedings of 2021 Sheep, Camelid and Goat Veterinarians Conference; 2021 Jun 30 – 02 Jul; Wagga Wagga. Sydney: Sheep, Camelid and Goat Veterinarians; 2021. p. 3-4.
2. The Australian Beef Sustainability Framework. The Australian Beef Sustainability Framework [Internet]. Sydney: The Australian Beef Sustainability Framework; 2022 [cited 2022 May 17]. Available from <https://www.sustainableaustralianbeef.com.au/>
3. GrainGrowers. Behind Australian grain [Internet]. Canberra: GrainGrowers; 2022 [cited 2022 May 17]. Available from <https://www.behindaustraliangrain.com.au/>
4. National Farmers Federation. Australian Agricultural Sustainability Framework [Internet]. Canberra: National Farmers Federation; 2022 [cited 2022 May 17]. Available from <https://nff.org.au/programs/australian-agricultural-sustainability-framework/>
5. Ridoutt B. Climate neutral livestock production – A radiative forcing-based climate footprint approach. J Clean Prod [Internet]. 2020 Nov 01 [cited 2022 May 17];291(1):125260. Available from: [10.1016/j.jclepro.2020.125260](https://doi.org/10.1016/j.jclepro.2020.125260)
6. Sheep Sustainability Framework. Wool's eco rating challenge in the EU [Internet]. Sydney: Sheep Sustainability Framework; 2021 Sep 23 [cited 2022 May 17]. Available from <https://www.sheepsustainabilityframework.com.au/resources/news/wools-eco-rating-challenge-in-the-eu/>

Welfare impacts of plant intoxications on extensively-raised livestock in southern Australia

Peter A Windsor
Production Animal Welfare & Health Services
Scarborough, NSW

Introduction

This paper addresses the question of whether plant intoxications should become more of a priority welfare issue for extensively farmed livestock in southern Australia, particularly for sheep production as the impacts of increasing climate variability emerge.¹ It has been estimated that ~95% of people consider farm animal welfare as a concern, with 91% seeking regulations ensuring transparent practices occur in livestock production.² Achieving this requires optimal nutritional and disease prevention management, humane husbandry, handling and transport and shelter for reducing impacts of climatic extremes, particularly hyperthermia and hypothermia episodes and severe bushfires and floods.¹

Despite improved uptake of drought planning, recent welfare disasters occurred during and following droughts, with prevailing dry El Niño eventually replaced by La Niña conditions and precipitous rainfall causing mass flooding and immediate and delayed disease and toxin-induced losses that exceed the losses from the dry period. In 2022, unprecedented rainfall in March led to mass drownings of livestock and welfare issues of excessive floodwater exposure and emergence of toxic plant syndromes. Drought management requires producers to consider whether dried toxic plants were introduced in hay, straw and silage and further, whether herbicide resistant seed including weeds, was introduced in drought feed. This requires manual checking of feeds and use of feed-out areas restricted to 'sacrificial paddocks' located where more regular animal surveillance can occur.

Australia is blessed with a spectacular array of native and introduced plants containing toxins that can cause a considerable range of disease syndromes and sometimes massive outbreaks (eg *Phalaris* poisonings and Annual Ryegrass Staggers). Approximately a thousand species of plants here are known to be toxic to livestock and humans, with poisoning of livestock by the more important of these, costing about \$100 million annually from the 60% of the toxic plants in Australia belonging to about 70 of the over 200 native plant families in Australia.³ Families with more than 10 toxic species include the legumes (Fabaceae, Mimosaceae), the nightshades and tobaccos (Solanaceae), the spurge (Euphorbiaceae), the grasses (Poaceae), the cycads (Cycadaceae, Zamiaceae), the saltbushes (Chenopodiaceae), the rice-flowers (Thymelaeaceae) and the buttercups (Ranunculaceae). Some genera are generally toxic (e.g. *Macrozamia* and *Pimelea* spp.), although it is noted that a number of important toxic species (e.g., *Erythrophleum chlorostachys* and *Trema tomentosa*) have few or no known toxic relatives.

With plant intoxications and iatrogenic substance exposure event historically common in our livestock populations due to poor environmental awareness of risks (e.g., lead poisoning), combined with husbandry conditions that can readily induce metabolic diseases (e.g., pregnancy toxemia, polioencephalomalacia), an understanding of systematic investigation of suspected intoxication cases is important. A consideration is that such investigations if adequately reported, facilitate deeper understanding of endemic and emerging disorders that may have welfare implications, whilst also enhancing our surveillance for incursions of exotic diseases, particularly as several transboundary infectious disease continue to emerge in proximity to our shores (e.g., FMD, Lumpy Skin Disease). Further, the diagnosis of neurological disorders presenting in livestock, aids our surveillance for transmissible spongiform encephalopathies (TSEs).⁴ Neurological diseases of sheep and cattle in Australia are rated in the top five reported diseases, together with diarrhoea, sudden death, ill-thrift/weight loss and weakness/depression. TSE surveillance data compiled by Animal Health Australia between 2005 and 2010, showed that the most frequently diagnosed neurological diseases were, in decreasing order of occurrence: plant poisonings (especially *Phalaris* and *Tribulus* spp. and corynetoxicosis), non-suppurative (predominantly viral) meningoencephalitis, hepatic encephalopathy, hypocalcaemia and hypomagnesaemia, listeriosis,

thiamine deficiency-associated polioencephalomalacia, suppurative (mainly bacterial) meningoencephalitis, cerebral abscess, developmental anomalies, and trauma and neoplasia.⁵

Disease syndromes associated with plant poisonings commonly recorded in these periods of droughts and floods include consideration of differential diagnoses of: Sudden death (e.g., green drought pregnancy toxemia, Nitrate and Cyanide poisoning, *Cestrum* sp., *Trema* and Phalaris poisoning); Neurological (e.g., Pyrrolizidine alkaloids, Phalaris, Ryegrass: perennial & annual); Gastrointestinal, hepatic (e.g., Pyrrolizidine alkaloids) and Renal (*Panicum* sp. crystalopathy), and Dermatological (primary & secondary photosensitisation, with St John's Wort and Blue Heliotrope of considerable concern in 2022) system disorders. Proactive management of the apparently increasing risks from intoxications from pasture and weed species are required as producers seek to adapt in a changing climate to the demanding animal welfare standards expected by a society increasingly divorced from rural community activities.

Livestock Welfare in Australia is Reputational

The extensive livestock production industries are vital to the national economy of Australia. Continuing improvements to extensively-raised livestock welfare is desirable, necessary and in some situations mandatory, if the social license for animal sourced food and fiber production is to continue sustainably.¹ However, meeting increasingly high welfare standards is challenging. The changing climate in this millennium, has seen the occurrence of two of the most severe drought periods on record in Australia, resulting in complex welfare issues arising from unforeseen disease, trade and environmental catastrophes. The onset of the first drought coincided with an uncontrolled epidemic of ovine paratuberculosis. It ended just prior to a temporary ban on live export of tropical cattle to Indonesia that induced a major market failure and led to severe morbidity and mortality on some beef properties. The second drought period progressed in severity and culminated in the most extreme bushfires recorded, causing unprecedented levels of mortality, morbidity and suffering in farmed animals and wildlife. Temperature extremes have also caused periodic heat-associated or cold-induced hypothermia losses, requiring increased vigilance and careful management to reduce both temperature-induced stress during transport and the high ovine peri-parturient losses traditionally observed in extensive sheep farming. Several issues remain controversial, including surgical mulesing of wool sheep to manage flystrike, and the continuing live export trade of sheep and cattle.

In reviewing the increasingly complex welfare challenges for the extensive livestock population industries of Australia that are export trade dependent, it is clear they remain vulnerable to increasing welfare activism.² However, it also appears progress has been made, including development of prescribed livestock welfare Standards and Guidelines and the introduction of the Exporter Supply Chain Assurance System (ESCAS) to address export concerns. Further, the sheep mulesing crisis led to improved producer welfare attitudes and practices, including pain management during aversive husbandry procedures that is now occurring globally. Finally, innovations in animal welfare surveillance and assessment, are additional encouraging signs that suggest improving change management of extensive farm animal welfare is occurring that provides lessons well-beyond Australian shores.¹ However, should welfare considerations of plant intoxications be added to this list of concerns?

Livestock welfare from European Perspectives

Traditionally, research on farm animal welfare in Europe has mainly focused on welfare problems thought to be common in intensive systems, with the welfare of animals kept in extensive systems attracting much less attention.⁶ This may be due to the generally held belief that extensive systems are advantageous in terms of animal welfare. Although it is undeniable that extensive systems have many benefits in terms of animal welfare, they are by no means free of welfare problems. A recent review of animal welfare problems likely to be found in extensive systems followed the four animal welfare domains of: nutrition, environment, health and behaviour.⁶ Extensive environments have highly variable climatic conditions, food quality, and often, access to high-quality water, raising welfare concerns from chronic hunger, thirst and thermal stress, with variations depending on the location and time of year. Some diseases are more likely in extensive systems than in intensive ones, compounded by more difficult supervision in extensive systems. Painful husbandry procedures as well as neonatal mortality and predation, with infrequent handling and / or

potentially aversive handling that impairs human-animal relationships, have potential negative effects on the welfare of extensively raised livestock.⁶

Toxic plants are suggested as providing both detrimental and beneficial impacts in extensive grazing systems, with animals encountering a diversity of plants that contain plant secondary compounds (PSCs).⁶ PSCs are highly diverse chemical structures with a wide variety of actions on animal health and behaviour, depending on the form and the dose ingested, the duration of ingestion, and the species exposed. Amongst the negative impacts, PSCs can alter nutrient utilisation, digestive function, respiratory and cardiovascular function, immune function, as well as deteriorating nervous system and reproductive capacities. However, herbivores have acquired behavioural and metabolic adaptations that allow them to cope with PSCs. The main behavioural adaptation is the capacity of herbivores to develop food aversions when ingesting PSCs, because some of these compounds induce nausea. As not all PSCs cause food aversions and delayed toxic effects can limit the ability of herbivores to form food aversions, the ingestion of toxic plants by extensively kept animals can be a source of pain and suffering. Raising livestock breeds that are not adapted to tropical environments with subsequent exposure to unfamiliar plants can compromise their welfare and production. Further, if food is scarce, animals may eat less palatable plants, with some containing toxins causing death in animals that would normally avoid eating such PSCs.

Conversely, PSCs can have beneficial effects on health when ingested in the right quantity, for the right amount of time, or in the right combination. PSC have recognised actions on the control of gut pathogen load through different mechanisms, with suggestions that parasitised sheep and goats increase preferences for anti-parasitic PSCs and tannins in particular, when experiencing parasitic burdens relative to non-parasitised animals.⁷ Other studies suggest livestock self-medicate by grazing specific medicinal plants when ill and may learn of the benefits of specific PSCs for mitigating discomfort and pain associated with certain health pathologies. It is suggested that the medicinal effects of PSCs have potential to improve both the health and welfare of ruminants kept in extensive systems, with the biochemical diversity of plants offering animals the opportunity to enhance their health and well-being from ingestion of anti-parasitic agents and immune modulators. Management practices that promote plant diversity and enhance animals' diet selection offers ways to reduce the impact of some health pathologies.⁶

Intoxications causing Sudden death

Important differential diagnoses include 'green drought' pregnancy toxaemia when supplementary feeding is removed too early, nitrate and cyanide poisoning outbreaks, Phalaris poisoning, *Cestrum parqui* and *Trema aspera* poisoning, etc. Recent mention on the ASCGV e-list is "nitrate poisoning associated with ingestion of many weeds, crop & pasture plants that are mostly well-known accumulators of nitrate, including: capeweed, pigweed (*Portulaca oleracea*), & variegated thistle, with crop plants including: barley, linseed, lucerne, maize, millet, oats, rape, sorghum, soybean, sub clover, ryegrass and wheat."

Intoxications causing Dermatological disorders

Primary & secondary photosensitisation risks are high after heavy rains in warm sunny periods, with St John's Wort and Blue Heliotrope of considerable concern in early 2022. Primary intrinsic photosensitivity occurs when the rumen metabolises chlorophyll to phylloerythrin yet circulation of excessive phylloerythrin that is usually excreted in bile, leads to free radical damage of skin areas exposed to light, with visible tissue injury particularly occurring in unpigmented areas of skin and other tissues (e.g. conjunctiva). Extrinsic photosensitisation occurs when photodynamic agents in plants are ingested in plants. Recent mention on the ASCGV e-list is: "Photosensitisation; client has 2,500 head of crossbred lambs finishing on Pioneer 970 canola, a variety they've used in the past without problems. Expect to turn them off in about 2m, 3 days in, noticed a few dozen with photosensitisation; removed into shed. Apart from Zinc cream is there anything else? They have pasture in the next paddock but it has heavy worm larvae contamination, like most of NSW at the moment. Advised getting off canola and onto pasture for a few days then gradual reintroduction to the canola. Concerns expressed on the animal welfare implications of not "bothering to remove photosensitised lambs? Have you never suffered painful sunburn? As humans we can seek shade and pain relief but what are the poor sheep supposed to do, keep on suffering?" Eventual burr injury & Orf risk may be worth further consideration.

Intoxications causing Gastrointestinal and Hepatic disorders

Pyrrolizidine alkaloidosis causes hepatocellular lesions from acute hepatic necrosis, to fibrosis with biliary proliferation and potentially nodular hyperplasia with chronic exposure. Hepatocellular megalocytosis if present aids the diagnosis and cases may present with hepatic encephalopathy. Lupinosis/Phomopsis from *Diaportia toxica* (formerly *Phomopsis leptostromiformis*) contamination of lupins, especially stubbles; releases a mycotoxin that inhibits microtubule formation, resulting in excessive mitoses, hepatocellular degeneration, fibrosis and biliary proliferation. Recent mention on the ASCGV e-list is: "Bloat; in beef cattle country we have experienced wetter than normal conditions with the result that clover is shooting up in autumn rather than spring. Terric bloat blocks were around and worked well, as was Tympanyl for treatment, but are both products are no longer available and how well does 'bloat-oil' work?" Of further interest is the drought-associated disorder of congenital biliary atresia seen in some progeny of ovine and bovine dams restricted to grazing the exposed banks of Burrinjuck dam and the Hume Weir infested by the water weed *Dysphania glomerulifera*.⁸ Recent studies have identified this plant contains a new toxin labelled biliatresone that inhibits gall bladder formation.⁹

Intoxications causing Neurological disorders

Important differential diagnoses have been reviewed.⁵ These include Pyrrolizidine alkaloidosis, Phalaris staggers, Annual Ryegrass staggers, Perennial Ryegrass staggers etc. Recent mention on the ASCGV e-list is: "Annual Ryegrass Toxicity; ~100mm rainfall about 5wks ago, cranky cattle 3wks ago, today falling over & severely affected. Masses of ryegrass and getting hay 3x a week; now sharing the only non-ryegrass paddock. Mycofix in Weatherpro may help, with publicity given to magnesium oxide but not seen anything useful. Shade, water, keep away from creeks & dams suggested. Ryegrass staggers is cutting a fair path of trouble here. A delay in onset, so working out which paddocks are most toxic is tricky if they are on rotation; resolves ~a month if undisturbed. Test ryegrass tested to determine toxicity; new varieties have a non-toxic endophyte, providing a good long-term plan." Further mention is: "Phalaris staggers; rapid onset, lush feed so little soil ingestion and so Co intake is low. Head bobbing a very common presentation in sheep. Difficulties with prehension and swallowing with tongue 'stabbing' in cattle. Pathognomonic changes found in brain histopathology with Phalaris staggers; dark green pigmentation seen grossly on transverse section" (also may be found in the renal pelvis).

Strategic management of plant poisonings

In a changing climate, Drought, Bushfire and Flood planning has become an increasingly important component of livestock enterprise management considerations. Extensive production requires improved surveillance vigilance systems to enable prompt decisions on carrying capacity, recognising the triggers to de-stock by class of animal, enhanced nutritional and water reserves, and robust financial reserves and literacy, with preparedness to pay for independent professional advice. Investors in drought preparedness are disadvantaged by current assistance to non-investors that still insist on financial drought relief, with a need to cease drought hand-outs for the industry to encourage better manage of this recurrent risk. However, when events are unprecedented, as with the 2019-20 bushfires and the 2021-22 floods, many forms of assistance are essential for the welfare of all quadruped and biped sufferers. The severity of these events indicates that emergency welfare planning, especially for Drought, Bushfire & Flood Preparedness, Prevention, Response and Recovery (PPRR) is required. Planning for plant intoxication events is indicated for the Response and Recovery phases of on-farm and regional PPRR welfare planning.

Conclusions

Are welfare impacts of plant intoxications on extensively-raised livestock in southern temperate climates of Australia becoming a priority welfare issue? This question may be debatable and views likely reflect the relative importance of livestock industry reputational concerns, consumer preference opinions and decisions compared to on-farm health and production losses. However, any uncertainty should not hinder the need for promotion of increased vigilance, surveillance and emergency welfare response planning, especially for Drought, Bushfire & Flood PPRR. It is possible that welfare PPRR may well become mandatory if the necessity of proactive management of the apparently increasing risks from intoxications from pasture and weed species fails to become visible to consumers. The veterinary profession has an important role in assisting producers

seeking to adapt in a changing climate and meet the increasing demands of animal welfare standards expected by a consumer society increasingly divorced from rural community activities.

References

1. Windsor PA. Progress with Livestock Welfare in Extensive Production Systems: Lessons from Australia. *Front. Vet. Sci.* 2021, 8:674482. doi: 10.3389/fvets.2021.674482
2. Futureye Report. Commodity or Sentient Being. Available online 2019. <https://www.sheepcentral.com/wp-content/uploads/2019/05/190129-Commodity-or-Sentient-Being-Australias-Shifting-Mindset-on-Farm-Animal-Welfare-v.-7.0.pdf>.
3. MacKenzie R. Australian Native Poisonous Plants. <http://anpsa.org.au/APOL7/sep97-4.html>
4. Animal Health Australia. TSEs. Available online 2019; <https://www.animalhealthaustralia.com.au/what-we-do/disease-surveillance/tse-freedom-assurance-program/surveillance-of-tses>).
5. Finnie J, Windsor PA, Kessell A. Neurological diseases of ruminant livestock in Australia. II. Toxic disorders and nutritional deficiencies. *Aust. Vet. J.*: 2011, 89:247–253.
6. Temple D and Manteca X (2020) Animal Welfare in Extensive Production Systems Is Still an Area of Concern. *Front. Sustain. Food Syst.* 2020, 4:545902. doi: 10.3389/fsufs.2020.545902.
7. Juhnke, J, Miller J, Hall JO, Provenza FD, Villalba JJ. Preference for condensed tannins by sheep in response to challenge infection with *Haemonchus contortus*. *Vet Parasitol.* 2012, 188: 104-114
8. Harper PAW, Unger B, Plant J. Congenital biliary atresia in lambs and calves. *Aust Vet J* 1990 May;67(5):167.
9. Koo KA, Waisbourd-Zinman O, Wells RG, Pack M, Porter JR. Reactivity of Biliatresone, a Natural Biliary Toxin, with Glutathione, Histamine, and Amino Acids. *Chem Res Toxicol.* 2016, 29(2):142-9. doi: 10.1021/acs.chemrestox.5b00308.