Investigating mortality in sheep and lambs exported through Adelaide and Portland
Literature Review
Abstract

This *Review of the literature* examines mortality rates and causes of mortality that occur during the live sheep export process. Mortality rates for sheep exported live by sea have been shown to vary with time of year, port of loading and line and class of sheep. A progressive reduction has been observed in mortality since the industry began in the 1970s when mortality rates were approximately 4%, this compares to 1% in 2003. Certain lines of sheep have been shown to suffer higher mortalities than others and it is also known that different groups of sheep from the same property are likely to suffer similar mortality when exported in different years. Investigations conducted in sheep exported from Western Australia in the 1980s and early 1990s have demonstrated that inanition and salmonellosis account for the majority (>60%) of the mortalities seen during the live export process. Other causes of mortality are described but generally account for less than 10% of deaths. More recently, two different salmonellosis syndromes have been described in the live sheep trade; classical outbreaks of salmonellosis in pre-export assembly depots with carry over mortality on ship and the Persistent inappetence-Salmonellosis-Inanition (PSI) complex. Significant changes have occurred in the industry over the last 20 years. These changes have been associated with a reduction in mortality. It is unknown if the previously identified causes of mortality are equally applicable to the current industry.
Executive summary

The objective of this document is to provide a synopsis of existing literature on mortality in the live sheep export trade. Extensive mortality investigations were conducted in the 1980s and 1990s in both Victoria and Western Australia and have consistently identified inanition and salmonellosis as the most common causes of death in sheep exported live by sea. Salmonellosis and inanition were identified as the primary cause of death for more than 75% of mortalities, while other causes of death generally account for less than 10% of mortalities. However, much of this data was collected almost 20 years ago and it is not known if current causes of mortality in the live sheep trade still follow these patterns.

Two salmonella syndromes have been shown to occur during the live sheep export process, classical (or feedlot-related) salmonellosis and the PSI complex. Classical salmonellosis occurs during the feedlot period and can cause significant losses, manifesting either as sporadic cases or an outbreak. Classical salmonellosis may also contribute to early on-ship mortality. The PSI complex has been identified as the most common and important cause of mortality during the on-ship phase. Persistent inappetance predisposes sheep to disease and mortality, with those that do not develop fatal salmonellosis ultimately perishing due to inanition.

It is apparent that failure to eat pelleted feed or persistent inappetance is an important problem in the live sheep trade, predisposing to death by either salmonellosis or inanition. Groups of sheep which have a higher risk of developing inanition and subsequently dying, are mature and overfat wethers. In addition to this, there is also a higher risk of inanition and death in the second half of the year. This is because older and fatter sheep are less able to cope metabolically with periods of stress or feed deprivation, particularly in the second half of the year.

It has also been shown that certain groups of sheep have a much higher risk of dying during export than others. The factors that predispose certain sheep to mortality remain unclear. Reports from Western Australian investigations suggest that sheep from areas with longer pasture growing season and higher rainfall may have a higher risk of mortality.

These mortality investigations have provided detailed information on the causes of mortality and many of the factors that predispose to mortality. However, mortality in the live sheep trade remains incompletely understood, in particular those factors at farm of origin, transport, feedlot and ship levels that predispose to mortality. In addition to this, the decision to place restrictions on the export of pastoral sheep and all lambs appears to have been based on anecdotal reports that these classes suffer higher mortalities than other sheep; currently there is little scientific evidence to suggest such differences exist. The LIVE.123 Project aims to clarify this scenario by:

- Investigating rates, causes and predisposing factors of mortality by region and time of year
- Developing an information management system that may be used by industry on an ongoing basis to monitor performance from farm to discharge
- To use real time data to define relationships between risk factors, disease processes and mortality
- Formulate management recommendations, for producers and industry, aimed at reducing the incidence of the common causes of mortality in the live sheep trade.
# Contents

<table>
<thead>
<tr>
<th>Page</th>
<th>1 Introduction</th>
<th>6</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.1</td>
<td>The LIVE.123 Project</td>
<td>6</td>
</tr>
<tr>
<td>1.2</td>
<td>Review of Current Literature</td>
<td>6</td>
</tr>
<tr>
<td>2</td>
<td>Mortality in the live sheep export industry</td>
<td>7</td>
</tr>
<tr>
<td>2.1</td>
<td>Mortality Rates</td>
<td>7</td>
</tr>
<tr>
<td>2.1.1</td>
<td>Overview</td>
<td>7</td>
</tr>
<tr>
<td>2.1.2</td>
<td>Stage of export process</td>
<td>7</td>
</tr>
<tr>
<td>2.1.3</td>
<td>Port of loading</td>
<td>7</td>
</tr>
<tr>
<td>2.1.4</td>
<td>Time of year</td>
<td>9</td>
</tr>
<tr>
<td>2.1.5</td>
<td>Line and class of sheep</td>
<td>9</td>
</tr>
<tr>
<td>2.1.6</td>
<td>Ship</td>
<td>9</td>
</tr>
<tr>
<td>2.2</td>
<td>Causes of Mortality</td>
<td>11</td>
</tr>
<tr>
<td>2.2.1</td>
<td>Overview</td>
<td>11</td>
</tr>
<tr>
<td>2.2.2</td>
<td>Fremantle mortality investigations</td>
<td>11</td>
</tr>
<tr>
<td>2.2.3</td>
<td>Portland mortality investigations</td>
<td>12</td>
</tr>
<tr>
<td>3</td>
<td>Salmonellosis and live sheep exports</td>
<td>13</td>
</tr>
<tr>
<td>3.1</td>
<td>Overview</td>
<td>13</td>
</tr>
<tr>
<td>3.2</td>
<td>Classical Salmonellosis</td>
<td>14</td>
</tr>
<tr>
<td>3.2.1</td>
<td>Overview</td>
<td>14</td>
</tr>
<tr>
<td>3.2.2</td>
<td>Pathogenesis of classical salmonellosis</td>
<td>14</td>
</tr>
<tr>
<td>3.2.3</td>
<td>Clinical signs and pathology</td>
<td>15</td>
</tr>
<tr>
<td>3.2.4</td>
<td>Treatment and control</td>
<td>16</td>
</tr>
<tr>
<td>3.3</td>
<td>The persistent inappetance-salmonellosis-inanition complex</td>
<td>17</td>
</tr>
<tr>
<td>3.3.1</td>
<td>Overview</td>
<td>17</td>
</tr>
<tr>
<td>3.3.2</td>
<td>Determining primary cause of death in the PSI complex</td>
<td>17</td>
</tr>
<tr>
<td>3.3.3</td>
<td>Persistent inappetance</td>
<td>18</td>
</tr>
<tr>
<td>3.3.4</td>
<td>Risk factors for persistent inappetance</td>
<td>18</td>
</tr>
<tr>
<td>3.3.5</td>
<td>Inanition in persistently inappetant sheep</td>
<td>20</td>
</tr>
<tr>
<td>3.3.6</td>
<td>Salmonellosis in persistently inappetant sheep</td>
<td>20</td>
</tr>
</tbody>
</table>
4 Conclusions .......................................................... 21
5 Bibliography .......................................................... 22
1 Introduction

1.1 The LIVE.123 project

Although extensive mortality investigations were carried out in WA in the 1980s and early 1990s and to a lesser extent in Victoria in the same time period, mortality in the live sheep trade, remains incompletely understood, in particular those factors at farm of origin, transport, feedlot and ship levels that predispose to mortality. In addition to this, the decision to place restrictions on the export of pastoral sheep and all lambs appears to have been based on anecdotal reports that these classes suffer higher mortalities than other sheep; currently there is little scientific evidence to suggest such differences exist. The LIVE.123 Project aims to clarify this scenario by:

- Investigating rates, causes and predisposing factors of mortality by region and time of year
- Developing an information management system that may be used by industry on an ongoing basis to monitor performance from farm to discharge
- To use real time data to define relationships between risk factors, disease processes and mortality
- Formulate management recommendations, for producers and industry, aimed at reducing the incidence of the common causes of mortality in the live sheep trade.

1.2 Review of current literature

The objective of this document is to provide a synopsis of existing literature on mortality in the live sheep export trade. The majority of the existing literature on the live export trade was published from the late 1970s to the early 1990s. At this time extensive research was carried out to investigate mortality in live sheep exports. These investigations involved sheep exported from Western Australia (WA), although some investigation was directed at the eastern ports (Kelly 1996). Previous studies identified common causes and seasonal patterns in mortality rate.
2 Mortality in the live sheep export industry

2.1 Mortality rates

2.1.1 Overview

In the live export industry, mortality rates are routinely recorded for all voyages. Mortality rates are recorded by stage of export process, port of loading, time of year, class of sheep and ship (Norris and Norman 2004). During every voyage, daily reports are completed that include daily mortalities as well as information on health, feed, water and environmental conditions. If shipboard mortality reaches or exceeds the reportable level of 2%, AQIS must be notified and contingency plans implemented where appropriate (DAFF 2005). Overall mortality rates have steadily declined to a record low of 1% in 2003 (Norris and Norman 2004). If the unusually high mortalities that occurred on the Cormo Express (9.82%) are excluded, the overall mortality for 2003 was 0.88% (Norris and Norman 2004). With the exception of the Cormo Express, mortality rates for all shipments during 2003 remained below the reportable level (Keniry et al. 2003). Mortality were similarly low in the first half of 2005, averaging 0.67% (DAFF 2005).

2.1.2 Stage of export process

Each shipment is divided into three stages; loading, voyage and discharge. The discharge stage begins on arrival at the first port of unloading. Norris described changes in mortality rate over time for several shipments and demonstrated that the number of mortalities occurring at different stages is dependant on the disease processes which are occurring. (Norris et al. 1989b) Richards suggested that salmonellosis was responsible for a majority of deaths early in the export process while a significant number of deaths later in the voyage were due to inanition (Richards et al. 1989).

2.1.3 Port of loading

Historically, sheep exported from the eastern ports (Adelaide and Portland) have suffered higher mortality rates than those exported from Fremantle (figure 2.1). Mortality data collected in 2002 showed that sheep exported from Portland, were more than twice as likely (relative risk = 2.4) to die during the voyage when compared with sheep exported from Fremantle. Voyages from Adelaide also had higher mortality compared to Fremantle, however, the differences between these groups were not as marked (relative risk = 1.5) (Norris and Norman 2003).

These data indicated that while mortality rates for voyages from Fremantle were consistently reaching record lows, mortality rates for Adelaide and Portland voyages were relatively static (Norris and Norman 2003). The reasons for this disparity in mortality rate between ports have not been clearly defined. It has been suggested that, differences in feeding and management between the Western Australian shed based pre-export assembly depots and the eastern States’ paddock based pre-export assembly depots may affect mortality outcomes (Norris et al. 1989b).
Figure 2.1 – Annual mortality rates for sheep exported from Adelaide, Portland and Fremantle from 1985 to 2003 (Norris and Norman 2004).
2.1.4 Time of year

Since the implementation of the mortality surveillance system in 1985, mortality rates have consistently been higher in the second half of the year than in the first (figure 2.2). It has been demonstrated that season has a significant affect on sheep metabolism, feeding behaviour, disease occurrence and mortality (Higgs et al. 1991; Richards et al. 1991).

2.1.5 Line and class of sheep

Mortality rates in sheep exported live by sea have been shown to vary widely between farm groups, with certain lines of sheep suffering significantly higher mortality than others. Norris found that 54% of all deaths during export occurred in only 25% of lines. (Norris et al. 1989b) In addition to this, Higgs found that mortality rates in different farm groups ranged from 0 to 28.2%, with half of all sheep that died being traced to 14.2% of consignments. Furthermore, this study found that groups of sheep from the same farms, exported in different years were likely to suffer similar mortality rates, suggesting that skewed mortality rate between farms are not due to chance (Higgs et al. 1999). In addition to this, different classes of sheep vary in their ability to adapt to sea transport. Wether hoggets consistently have lower mortality rates than adult wethers. Higgs analysed 50 shipments and found lower mortality rates in wether hoggets (0.52%) than adult wethers (1.63%).(Higgs et al. 1991)

2.1.6 Ship

Mortality records collected between 1997 and 2002 have shown that certain ships have higher mortality voyages than others. However, high and low mortality voyages are known to occur on individual ships under the same management conditions indicating that this variation is most likely due to sheep factors rather than ship factors (Norris and Norman 2003). Several studies have investigated spatial patterns of mortality on board ships. Kelly found a significant difference between tiers, with upper tier sheep having mortality rates 1.5 to 2.4 times greater than sheep in lower tier pens.(Kelly 1996) However, analysis of mortality records has shown that these differences are not consistent, with no difference existing between tiers on 61% of voyages (Norris and Norman 2003).
Figure 2.2 – Mortality Rates (%) for sheep exported from Fremantle, Adelaide and Portland to the Middle East for the first and second half of each year from 1997-2003 (Norris and Norman 2004).
2.2 Causes of mortality

2.2.1 Overview

Richards found that inanition was the most important causes of mortality in sheep exported live by sea, followed by salmonellosis. Together the two conditions accounted for more than 60% of deaths occurring during the export process (Richards et al. 1989). Inanition is defined as the exhausted state due to prolonged under-nutrition or starvation (Blood and Studdert 1999). These results are consistent with those of a Portland based mortality investigation by Kelly in which, inanition and salmonellosis were shown to account for 61-75% of mortalities (Kelly 1996). Richards provided further evidence that inanition is the most common cause of mortality in live sheep exports in a follow up study in which inanition was responsible for 65% of all deaths. (Richards et al. 1991)

2.2.2 Fremantle mortality investigations

RICHARDS, NORRIS et.al 1989

In this mortality investigation post-mortem examinations were performed on sheep that died in the Fremantle pre-export assembly depot and during the voyage to the Middle East. Differences were noted between causes of death during the assembly period and the voyage, as shown in table 2.1. The most common causes of mortality in the depot were salmonellosis, miscellaneous diseases, trauma, inanition, acidosis and enterotoxaemia. On-ship inanition was the most common cause of death followed by salmonellosis, trauma, miscellaneous diseases and enterotoxaemia.

Table 2.1 – Causes of mortality in live export sheep (Richards et al. 1989; Norris et al. 1989b).

<table>
<thead>
<tr>
<th>Causes of death</th>
<th>During feed-lotting</th>
<th>During export by sea</th>
<th>Overall</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>% deaths</td>
<td>Number of cause-specific deaths per 100,000 sheep</td>
<td>% deaths</td>
</tr>
<tr>
<td>Inanition</td>
<td>10.2</td>
<td>6.3</td>
<td>43.4</td>
</tr>
<tr>
<td>Salmonellosis</td>
<td>53.4</td>
<td>33.1</td>
<td>20.2</td>
</tr>
<tr>
<td>Trauma</td>
<td>12.6</td>
<td>7.8</td>
<td>10.6</td>
</tr>
<tr>
<td>Miscellaneous diseases</td>
<td>23.8</td>
<td>14.8</td>
<td>5.9</td>
</tr>
<tr>
<td>Enterotoxaemia</td>
<td>3.4</td>
<td>2.1</td>
<td>1.0</td>
</tr>
<tr>
<td>Acidosis</td>
<td>3.9</td>
<td>2.4</td>
<td>-</td>
</tr>
<tr>
<td>Inconclusive³</td>
<td>3.4</td>
<td>2.1</td>
<td>19.0</td>
</tr>
</tbody>
</table>

1 Based on overall mortality rate during feedlotting of 6.2 deaths per 10,000 sheep
2 Based on overall mortality rate during export by sea of 210.7 deaths per 10,000 sheep
3 Most of these animals were too decomposed at the time of necropsy to give meaningful results

Included in the miscellaneous diseases category, are a variety of common diseases of sheep which were most likely present on farm including, urolithiasis, pneumonia, non-salmonella enteritis and lupinosis. Deaths due to trauma are generally acute or sub-acute and follow transport injuries. The inconclusive category primarily consisted of sheep that were too decomposed at the time of necropsy to identify significant pathology, but did include a small number of sheep with no detectable abnormalities (Richards et al. 1989).
2.2.3 Portland mortality investigations

KELLY 1996

In this investigation, a representative cohort of sheep was selected from the consignment and monitored throughout the export process. Post-mortem examinations were performed on all cohort sheep and a selection of non-cohort sheep that perished. The causes of mortality reported in this study were similar to those reported by Richards and Norris in 1989. For this investigation, five diagnostic criteria were defined. The relative mortality for each category is shown in table 2.2.

Table 2.2 – Proportional mortality rates (%) in cohort and non-cohort sheep on board ship, adapted from (Kelly 1996).

<table>
<thead>
<tr>
<th>Diagnostic Category</th>
<th>Voyage 1 Cohorts</th>
<th>Voyage 1 Non-cohorts</th>
<th>Voyage 2 Cohorts</th>
<th>Voyage 2 Non-cohorts</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inanition Syndrome</td>
<td>56.2</td>
<td>60.4</td>
<td>58.4</td>
<td>58.9</td>
</tr>
<tr>
<td>Salmonellosis/Enteritis</td>
<td>21.9</td>
<td>24.8</td>
<td>21.4</td>
<td>15.4</td>
</tr>
<tr>
<td>Sudden Deaths</td>
<td>8.6</td>
<td>7.6</td>
<td>3.4</td>
<td>4.3</td>
</tr>
<tr>
<td>Trauma Induced Conditions</td>
<td>0.0</td>
<td>0.0</td>
<td>5.6</td>
<td>2.8</td>
</tr>
<tr>
<td>Background/Miscellaneous conditions</td>
<td>13.3</td>
<td>7.2</td>
<td>11.2</td>
<td>18.7</td>
</tr>
</tbody>
</table>

The inanition syndrome was considered a primary cause of death, as was salmonellosis. The sudden death category included bloat and suffocation, which have similar pathology and may be confused on post-mortem. Sheep in this mortality category had good body condition and rumen fill. Trauma induced conditions occurred relatively infrequently and were specifically due to injury, cellulitis and abscess formation at vaccine sites. The final diagnostic category contained causes of mortality that are common in all farmed sheep. These background/miscellaneous mortalities included enterotoxaemia, other gastrointestinal diseases, nephrosis syndrome and urinary tract diseases, pneumonia and hepatic diseases. The nephrosis syndrome accounted for a small number of deaths and was characterised by elevated blood urea, grossly large and pale kidneys at post-mortem and severe tubular degeneration seen on histopathology.
3 Salmonellosis and live sheep exports

3.1 Overview

Two salmonella syndromes have been shown to occur during the live sheep export process, classical (or feedlot-related) salmonellosis and the PSI complex. Figure 3.1 schematically illustrates the occurrence over time and crude mortality rates for the two salmonella syndromes. Clinically it is difficult to differentiate classical salmonellosis and the PSI complex as causes of death as post-mortem findings and salmonella serotype are often identical. The two syndromes are only distinguishable by identifying the risk factors that contribute to disease (More 2002).

Classical salmonellosis occurs during the feedlot period and can cause significant losses, manifesting either as sporadic cases or an outbreak. As shown below, classical salmonellosis may also contribute to early on-ship mortality.

The PSI complex has been identified as the most common and important cause of mortality during the on-ship phase. Persistent inappetance predisposes sheep to disease and mortality, with those that do not develop fatal salmonellosis ultimately perishing due to inanition.

Figure 3.1 – Onset, duration, and mortality rate for the two salmonella syndromes that occur during the live sheep export process (More 2002).

The major salmonella serotypes isolated from clinical salmonellosis cases in the live export trade included *Salmonella typhimurtium*, *S. bovis-morbificans* and *S. havana* (Higgs et al. 1993). *S. derby*, *S. adelaide* and *S. tennessee* have also been infrequently isolated (Richards et al. 1989; Kelly 1996). All these *Salmonella* sp. are non host adapted and are capable of causing disease in a range of animal species including humans (Radostits et al. 2000).
3.2 Classical salmonellosis

3.2.1 Overview

Salmonellosis refers to the clinical disease associated with salmonella infection. The majority of salmonella infections in livestock are subclinical. With subclinical infections animals may shed salmonella in faeces without appearing sick. Salmonella are commonly found in animal production systems, with the prevalence increasing with intensification and increasing herd size (Radostits et al. 2000). Salmonella sp. have been demonstrated to cause enteritis in sheep as early as 1919 (Wray and Linklater 2000). Salmonella are opportunistic pathogens; disease typically reflects a series of events that lead to compromised host immunity and increased pathogen exposure. Salmonellosis in sheep is commonly associated with assembly of sheep at high stocking rates. Individual sheep can be affected however, ovine salmonellosis commonly occurs as a sporadic outbreak of disease following a stressful event, with up to 50% of the flock being affected (Radostits et al. 2000).

3.2.2 Pathogenesis of classical salmonellosis

As demonstrated in Figure 3.2, the development of clinical salmonellosis is dependant on interaction between salmonella exposure and host resistance. Salmonella exposure, shown in the image above, is a function of size of the salmonella challenge (i.e. number of organisms) and the virulence of the salmonella serovar.

**Figure 3.2 - The balance between host resistance and exposure, and likelihood of clinical salmonellosis. (More 2002) – [Adapted from House and Smith 2000]**

![Diagram](image)

- **Maximal host resistance**
- **Decreased host resistance**

| Large exposure | +/-- | + | ++ |
| Small exposure | -- | +/- | + |

++ = consistently severe disease  + = usually causes disease  +/- = may cause illness  -- = usually does not cause illness

Infection with salmonella occurs most commonly via the faecal-oral route and animals can become infected via animal to animal transmission, through contaminated feed and water and from environmental exposure (Hodgson et al. 2003). Infected animals, including clinically infected animals and carriers, are an important source of infection, capable of transmitting infection directly and contaminating the environment, feed and water (Radostits et al. 2000).
Salmonella can survive for months to years in the environment and under suitable conditions, will proliferate to increase the level of environmental contamination (Murray 2000). In live export pre-export assembly depots, certain salmonella types are frequently isolated from different consignments, suggesting that salmonella will persist between consignments and infect new groups of sheep (Kelly 1996).

Most sheep are able to withstand salmonella challenge unless host resistance is decreased. Any event that produces stress, including transport, yarding, inappetance and inclement weather can decrease host resistance. Stressful events stimulate the adrenal glands to produce and secrete excessive amounts of corticosteroids. Corticosteroids actively suppress immune function, increasing the likelihood of clinical salmonellosis following a relatively low salmonella challenge (Dickson 1993). Higgs studied the effects of stress on salmonellosis in live export sheep and found that all sheep with salmonellosis lesions at post-mortem also had enlarged adrenal glands, indicating severe stress and increases corticosteroid production. (Higgs et al. 1993) This observation is likely to reflect the stress of the disease process.

Clinical salmonellosis may occur in healthy animals and has been recorded in live export sheep showing no signs of stress or host compromise (Higgs et al. 1993). A large challenge dose or particularly virulent challenge strain may overwhelm healthy animals. In experimental trials a challenge dose of $10^9$ salmonella was sufficient to cause clinical disease in healthy sheep (Wray and Linklater 2000).

Exposure to salmonella organisms in the assembly depot is common. Higgs found that on arrival at the depot, prevalence of faecal salmonella shedding is close to zero, ranging from 0% to 0.70%. (Higgs et al. 1993) Mixing of groups and high stocking rates associated with intensive feedlot management provide opportunity animal to animal and environment to animal while transport and other stressors may cause latent salmonella carriers to shed salmonella (Kelly 1996; Radostits et al. 2000). The sum of these events is an increase in the number of excretors and the quantity of salmonella shed by individuals. As feedlotting progresses, prevalence of faecal salmonella shedding has been shown to increase form close to zero to more than 15%, 83% and 93% on days 6, 14, and 22 of the feedlot period respectively (Higgs et al. 1993; Kelly 1996). This study demonstrated that a large proportion of sheep may be exposed to and shed salmonella during the feedlot period. Historically only a relatively small proportion of sheep subsequently develop clinical salmonellosis (Higgs et al. 1993).

3.2.3 Clinical signs and pathology

Clinical salmonellosis in sheep is often classified as septicaemic, acute enteritis, sub-acute enteritis or chronic enteritis based on severity of disease. Table 3.1 shows the clinical signs and pathology associated with the disease syndromes. All four syndromes are known to occur in live export sheep. The severity of lesions and site of the gastrointestinal tract affected is influenced by the progression of disease. Acute salmonellosis primarily affects the abomasum and upper small intestine where as sub-acute and chronic disease was more pronounced in the lower small intestine and upper large intestine (Richards et al. 1993).
Table 3.1 – Clinical signs and pathology associated with classical salmonellosis in sheep (Radostits et al. 2000).

<table>
<thead>
<tr>
<th>Classification</th>
<th>Clinical Signs</th>
<th>Pathology</th>
</tr>
</thead>
<tbody>
<tr>
<td>Septicaemic</td>
<td>Severe depression, dullness, prostration and high fever followed by death within 24-48 hours</td>
<td>Widespread haemorrhage, organ congestion, multifocal hepatic necrosis and enteritis</td>
</tr>
<tr>
<td>Acute Enteritis</td>
<td>High fever and severe diarrhoea which is putrid and may contain blood or casts. Up to 75% mortality if untreated</td>
<td>Haemorrhagic enteritis, especially in the ileum, caecum and proximal colon</td>
</tr>
<tr>
<td>Chronic Enteritis</td>
<td>Succeeds acute disease, intermittent fever, chronic weight loss and ill thrift</td>
<td>Enteritis with submucosal thickening and mucosal hyperplasia and ulceration</td>
</tr>
</tbody>
</table>

3.2.4 Treatment and control

Appropriate and prompt fluid therapy is critical in the treatment of clinical salmonellosis. Antimicrobial use is considered by many to be of great value in treatment of salmonellosis but there also exists strong arguments against the use of antimicrobials in these cases. Antimicrobial use has been associated with emergence of antimicrobial resistant salmonellae and increased duration of salmonellae shedding (House and Smith 2000) and can actually interfere with normal digestive microflora, predisposing sheep to further gastrointestinal disease (Radostits et al. 2000). The potential for adverse consequences means that blanket prophylactic antimicrobial use in the live export trade is inappropriate and that prior to using antimicrobials to treat clinical cases, culture and antimicrobial sensitivity testing is warranted.

Control of salmonellosis in the live sheep trade is aimed at maximising host resistance to infection and minimising exposure to salmonella organisms. Host resistance can be maximised by maintaining adequate nutrition and minimising stress (More 2002). Management guidelines for achieving these goals throughout the export process are given in the Australian Standards for the Export of Livestock. The key to minimising exposure to salmonella pathogens is to reduce environmental salmonella contamination. Management procedures to reduce salmonella contamination include paddock rotations, isolation of sick animals, feed and water management to prevent faecal contamination and control of flies and rodents (More 2002). Disease prevention and control strategies need to be tailored to each facility to account for differences in variables such as soil type, drainage, and environmental conditions. It is also important to recognise the potential for positive and negative consequences to changes in management. For example the risk of salmonellosis is increased if sheep fail to eat therefore it is important that changes in feed presentation to reduce faecal contamination does not adversely impact feed intakes.
3.3 The persistent inappetance-salmonellosis-inanition complex

3.3.1 Overview

A schematic overview of the PSI complex is given in figure 4.2. There are several factors that contribute to the development of the PSI complex, including farm, feedlot and ship related factors as well as factors that produce stress and salmonella challenge. Two mortality outcomes occur in the PSI complex, with persistently inappetant sheep eventually dying from inanition, provided they don’t first succumb to salmonellosis. Mortality studies conducted by Richards determined that inanition is the most common cause of death, accounting for 42.2% of mortalities, while salmonellosis is responsible for 21.1 % of mortalities. (Richards et al. 1989)

![Figure 4.2 – PSI Complex Overview (More 2002).](image)

3.3.2 Determining primary cause of death in the PSI complex

Diagnosis of inanition as a primary cause of death is achieved by identifying characteristic post-mortem findings including: low weight of rumen solids, absence of other significant lesions and presence of widespread fat necrosis. Criteria used to diagnose salmonellosis as the primary cause of death are presence of characteristic enteric lesions and isolation of *Salmonella* sp. from tissues. Differentiating inanition and salmonellosis as primary cause of death is often difficult, because post-mortem examinations commonly reveal evidence of both inanition and enteric salmonellosis. To assist diagnosis in these cases, Richards developed diagnostic criteria, which used weight of rumen solids, expressed as a percentage of body weight (BW) to determine if inanition or salmonellosis was the primary cause of death. Findings of rumen solids less than 1.5% BW and acute enteric lesions were taken to indicate that inappetence preceded development of salmonella lesions and inanition was diagnosed as the primary cause of death. A diagnosis of salmonellosis was made in sheep with longer-term enteric salmonellosis lesions, with or without low (but higher than 1.5% BW) rumen solids (Richards et al. 1989)
3.3.3 Persistent inappetance

Persistent inappetance is defined as complete, but voluntary refusal to eat (More 2002). A study by Norris found that the percentage of non-feeders at the end of the feedlot period ranged from 0.2% to 23.3%. However, most feedlot non-feeders will begin to eat early during the ocean voyage, with 82.4% of feedlot non-feeders eating by day 5 of the voyage and the number of non-feeders continuing to steadily decrease throughout the voyage (Norris et al. 1990). Despite the fact that a majority of feedlot non-feeders will eventually begin to eat during the voyage they remain more likely to die during the export process. Extending the length of the feedlot period (Norris et al. 1992) and preferential feeding management of inappetant sheep on ship (Norris et al. 1990) have been shown to be ineffective at stimulating feed intake in persistently inappetant sheep.

In three separate studies, feedlot non-feeders have been shown to be 3.2 (Norris et al. 1989a), 6.83 (Higgs et al. 1996) and 13.9 (Norris et al. 1990) times more likely to suffer mortality on ship than sheep that ate during the feedlot period. However, sheep that remain persistently inappetant during the ocean voyage are at even higher risk of dying and account for a majority of mortalities during the export process (Richards et al. 1989). Differences in the proportion of inappetant sheep identified in each farm group have been shown to exist and also to have a significant effect on the mortality outcomes for the farm group. Norris found that the percentage of non-feeders per line of sheep ranged from 0 to 59.1%, with a majority of feedlot non-feeders originating from a limited number of farm groups. (Norris et al. 1989b)

3.3.4 Risk factors for persistent inappetance

Intensive investigation of sheep and on-farm risk factors, including season, age and adiposity, has been carried out to identify the reasons for disparity in mortality rates between farm groups. Higher proportions of inappetant sheep and higher mortality rates have been identified in sheep with greater fat reserves (Richards et al. 1989), in mature sheep (Norris et al. 1989b), in sheep from areas with a long (greater than 7 months) pasture growing season (Higgs et al. 1999) and in sheep exported during the second half of the year (Norris and Norman 2004). Higgs found that in lines of sheep with high condition score there was a higher proportion of feedlot non-feeders and higher mortality on ship, with fat sheep being almost twice as likely to die on ship when compared with sheep that are not fat. (Higgs et al. 1991)

It has been proposed that the influences of season, age and adiposity on feeding behaviour and subsequent mortality are driven by naturally occurring seasonal-cycles that affect sheep. As strongly seasonal animals, sheep are subject to annual variation in bodily functions (known as homeorhesis) including metabolic rate, appetite and reproduction. This variation occurs due to hormonal responses to change in photoperiod and aims to compensate for changing environmental conditions including rainfall, pasture availability and temperature.

Fat metabolism and appetite of adult sheep are significantly affected by seasonal variation. During the first half of the year when pasture availability is lowest sheep have a strong appetite drive and are in negative energy balance. During this period, the sheep is metabolically accustomed to utilising body fat stores and will cope well with prolonged inappetance or feed deprivation. Conversely, in the second half of the year when pasture availability is high, sheep have a diminished appetite drive and are in positive energy balance. At this time of year, sheep are metabolically geared to acquiring energy and laying down fat stores. As a result, sheep are less able to adapt to prolonged inappetance or feed deprivation by metabolising body fat and instead will start to mobilise amino
acids from muscle to meet energy needs (Richards et al. 1991). The overall effect is that, in comparison to the first half of the year, adult sheep exported in the second half of the year are more likely to become persistently inappetant and less likely to cope metabolically, resulting in higher mortality rates. Metabolic profiles conducted on sheep during the feedlot period indicate that sheep that fail to eat mobilise fat, oxidize mobilised fats to produce ketones, and develop evidence of fatty liver as indicated by elevations in liver enzymes (Richards et al. 1991). High levels of ketones lead to depressed mentation and appetite, the subsequent reduction in feed intake will confound the problem. The evidence indicating that this process occurs during the feedlot period reaffirms the importance of getting sheep onto feed at this time.

Young sheep are much less likely to become persistently inappetant and therefore have lower mortality rates, historically average mortality rate for adult wethers is 1.63% while hogget wethers have an average mortality of just 0.52% (Norris and Norman 2004). The reason for this is that younger animals maintain a strong appetite drive year round, independent of seasonal influences in order to meet their higher energy demands for tissue growth (Higgs et al. 1991).

It is likely that other risk factors contribute to the development of persistent inappetance and mortality. This is evident in the fact that some lines of sheep suffer above average mortality rates independent of the above mentioned risk factors (Higgs et al. 1999). Norris investigated on-farm management factors that may contribute to mortality, including prior experience with transport, yarding, supplementary feeding, time of shearing and social interactions. (Norris et al. 1989a) No consistent associations were identified between on-farm management and mortality during export so the effect of these factors on outcomes in the live sheep trade remains unclear. The power of this study was limited by the number of property of origin consignments of sheep that could be studied.

The influence of feedlot management on mortality outcomes has also been investigated. In Western Australian shed based depots, sheep are housed indoors and only fed the pelleted ration that will be used on ship. Paddock based systems often feed hay as well as pellets early in the feedlot period (More 2002). Several studies have investigated the effect each system has, on level of inappetance, however findings have varied between studies.

Norris found that early in the feedlot period, sheep held in sheds and exclusively fed pellets had higher levels of inappetance than those held in paddocks and fed hay. (Norris et al. 1989a) Despite significant differences in feeding behaviour early in the feedlot period, the number of non-feeders at the end of the feedlot period was not significantly different between shed and paddock based feedlots. In contrast to this, feeding hay has been shown to significantly increase the number of sheep not eating pelleted feed at the time of loading. In this study, the percentage of sheep that had not eaten pellets at the time of loading, was much higher in hay fed sheep (23.3%) than it was in sheep fed only pellets (0.2-4.4%) (Norris et al. 1989b).

In addition to this, the provision of feed troughs located centrally rather than on fence lines has been shown to increase feed intake and decrease the number of inappetant sheep (McDonald et al. 1990). It has been suggested that shed based feedlot systems are preferable to paddock based systems because the provide shelter from environmental extremes and dust as well as having better sheep handling and management facilities.
3.3.5 Inanition in persistently inappetant sheep

In the live sheep export trade, death due to inanition follows a voluntary and absolute refusal to eat available feed. Inanition is a persistent problem in the trade and has been identified as the most common cause of mortality during live sheep export. When sheep become persistently inappetant, death due to inanition will occur 2-4 weeks later. Most commonly, death occurs as a result of energy deprivation, however complicating factors such as trauma to recumbent animals and tetany (secondary to hypocalcaemia or hypomagnesaemia) can expedite death due to inanition. Inanition deaths occur as a direct result of persistent inappetance and contribute to the observed higher mortality risk in persistent non-feeders. In addition to this, Higgs demonstrated that, 85% of inanition deaths on ship are a direct consequence of persistent inappetance that began in the feedlot, that is, the attributable risk from failure to eat in the feedlot is 85%.(Higgs et al. 1991)

3.3.6 Salmonellosis in persistently inappetant sheep

Exposure to salmonella organisms is required for colonisation to occur and clinical salmonellosis to develop. However, as described in the section on classical salmonellosis, challenge with salmonella organisms alone is rarely sufficient to result in clinical disease without the assistance of some other host compromise (Higgs et al. 1993). In light of these facts, development of salmonellosis within the PSI complex is considered to be an opportunistic infection (More 2002).

Higgs found that, in comparison to sheep that are eating normally, persistently inappetant animals are significantly more likely to develop clinical salmonellosis and die during live export (Higgs et al. 1993). Based on these findings, and other evidence linking inappetance to development of salmonellosis (Kelly 1996), persistent inappetance has been identified as the critical risk factor contributing to the development of salmonellosis within the PSI complex. Persistently inappetant sheep have been shown to have lower resistance to enteric colonisation with salmonella organisms and subsequent development of clinical salmonellosis. In non-feeding sheep, rumen pH will increase and volatile fatty acid content will decrease providing favourable conditions for the survival of salmonella organisms. These changes to the ruminal environment, allow increased numbers of salmonellae to survive passage through the forestomachs (Radostits et al. 2000).

In addition to persistent inappetance, it has been demonstrated that stress is also an important risk factor for the development of salmonellosis (Radostits et al. 2000). Important stressors encountered in the live export chain include; transport and feed deprivation, mixing of sheep and social disturbance, sudden changes to management such as high stocking densities in the feedlot, and exposure to noise and dust (More 2002). Adrenal corticosteroids are released in response to stress and increases levels of these hormones, following stress, may suppress immune function and increase susceptibility to disease (Dickson 1993). Higgs concluded that, degree of stress may have a greater influence on inanition, severity of lesions and prognosis than does inappetance. In his study, pathological salmonella lesions only developed in inappetant sheep; with all affected sheep also having evidence of severe stress (increased adrenal gland weight), indicating that both factors play an important role in the development of disease (Higgs et al. 1993). The only proviso to concluding which event comes first, stress, inanition or salmonellosis is that one of the first clinical signs of salmonellosis is inappetance and the disease process is sufficiently stressful to increase adrenal gland weight. Therefore when adrenal gland weight is measured after the onset of disease it is not possible to determine if the stress preceded or is part of the disease process.
4 Conclusions

Existing mortality investigations have consistently identified inanition and salmonellosis as the most common causes of death in sheep exported live by sea. Salmonellosis and inanition were identified as the primary cause of death for more than 75% of mortalities, while other causes of death generally account for less than 10% of mortalities. Two salmonella syndromes have been shown to occur during the live sheep export process, classical (or feedlot-related) salmonellosis and the PSI complex. Classical salmonellosis occurs during the feedlot period and can cause significant losses, manifesting either as sporadic cases or an outbreak. The PSI complex has been identified as the most common and important cause of mortality during the on-ship phase. Persistent inappetance predisposes sheep to disease and mortality, with those that do not develop fatal salmonellosis ultimately perishing due to inanition.

Failure to eat pelleted feed is an important problem in the live sheep trade, predisposing to death by either salmonellosis or inanition. The groups of sheep that have a higher risk of becoming persistently inappetant and subsequently dying include; mature and overfat wethers. In addition to this, there is also a higher risk of inanition and death in the second half of the year. This is because older and fatter sheep are less able to cope metabolically with periods of stress or feed deprivation, particularly in the second half of the year. In addition to this, groups of sheep from certain farms have a much higher risk of dying during export than others, but the factors that predispose these groups to mortality remain unclear. Reports from Western Australian investigations suggest that sheep from areas with longer pasture growing season and higher rainfall may have a higher risk of mortality.

These mortality investigations have provided detailed information on the causes of mortality and many of the factors that predispose to mortality. However, mortality in the live sheep trade remains incompletely understood, in particular those factors at farm of origin, transport, feedlot and ship levels that predispose to mortality. In addition to this, the decision to place restrictions on the export of pastoral sheep and all lambs appears to have been based on anecdotal reports that these classes suffer higher mortalities than other sheep; currently there is little scientific evidence to suggest such differences exist.
5 Bibliography


