5. Sheep Health in Meat Production Enterprises

Kevin Bell

Learning objectives

At the end of this topic you should:

• Be aware of the most important diseases of sheep in Australia
• Have a particular appreciation of diseases impacting on enterprises in which sheep meat production is a significant component
• Understand and be able to implement treatment, control and prevention measures for these diseases.

Key terms and concepts

Disease, animal welfare, grazing management, chemical use, integrated pest management, loss of production, clinical, sub-clinical, immunity, chemical resistance.

5.1 Introduction

Profitable sheep production systems are largely based on converting grass into either food or fibre. Diseases (defined below) essentially interfere with this process, rendering it less efficient by impairing the function of the “converter” (the sheep). In most cases (but not necessarily all) the welfare of the sheep is also compromised. This is of singular importance; it must be redressed in its own right, apart from the economic issues historically given primary and sometimes sole consideration.

There are a great number of separately definable disease conditions which can afflict sheep in Australia, but the great majority of these are uncommon, sporadic in nature, and although of serious consequence to individual sheep, often inconsequential in their effect on enterprise economics. By and large these diseases are not preventable. Since sheep were introduced into Australia this range of diseases has arisen or been introduced, in many cases the disease has been noticed and defined following novel environmental influences. This may be as simple as an increase in pasture stocking rate, modified fertiliser usage or altered nutrition.

Where these diseases have had significant impact, research has generally produced information and/or products to economically and effectively control the conditions. As a result disease is relatively uncommon, but continuing health depends on maintenance of appropriate preventive measures including vaccination and drenching programmes.

5.2 What is disease?

Disease is a condition where an organism has its function compromised to the extent that health, and in the case of animals, welfare, is impaired. This altered state may be obvious or it may be very difficult to detect. In examples of the latter, a minor reduction in growth or reproductive rate may be the only discernable effect. This may not compromise the welfare of the animal, but may impact negatively on the enterprise, through the loss of system efficiency – the so-called “loss of production” syndrome.

Clinical disease refers to conditions where the altered state is detectable. This can include altered appetite, gastro intestinal dysfunction, lesions and even death.
• **Sub-clinical** diseases are not readily detectable. In animal enterprises they are often the most important.

• **Acute** disease is characterised by rapid onset and development of clinical symptoms, often severe in their effect, and resolution over some days or weeks. The animal’s immune system or therapeutic intervention may be a part of this resolution; death may also be a consequence. The acute phase may resolve to a chronic state.

• **Chronic** disease refers to a condition persisting for months, sometimes indefinitely. The disease is not fatal, but continues to afflict the animal. Often the disease’s causative agent persists in the animal, in a manageable state of fluctuating nature. This state is modulated by the ability of the animal to maintain resistance. Impairment or reduction of resistance, commonly by nutritional or hormonal means, may result in a re-appearance of the disease in the acute and clinical state.

• **Infectious** diseases are caused by living agents which proliferate in the host animal to the extent that the damage can’t be contained by the host’s immune system. The immune response itself may be a significant aspect of the overall effect of the disease on the animal. Lice infestations and gastro-intestinal parasites are examples of this.

• **Non-infectious** diseases result from physical or chemical agents or from specific nutrient deficiencies. For example: algal poisoning, nitrate poisoning, selenium deficiency.

### 5.3 Animal welfare

Animal welfare has always been a concern for farmers because it makes both financial and ‘moral’ sense. It is now becoming a more public issue and will undoubtedly become an increasingly important marketing issue in the future. It is also a legal obligation. Section 9 of the *Prevention of Cruelty to Animals Act* defines cruelty, and in addition to requiring that proper and sufficient food and shelter being made available for animals, specifies that disease be treated and prevented. State *Codes of Practice* also deal with the welfare of livestock. The Victorian Department of Agriculture has developed a Code of Practice for the welfare of sheep. (Reading 5.1: Bureau of Animal Welfare 2001).

The Code lists the basic requirements for the welfare of sheep as:

1. A level of nutrition adequate to sustain good health and vigour.
2. Access to sufficient water of suitable quality to meet physiological needs.
3. Social contact with other sheep; but with sufficient space to stand, to lie down and stretch their limbs.
4. Protection from predation.
5. Protection from pain, injury and disease.
6. Protection from extremes of weather which may be life threatening.
7. Provision of reasonable precautions against the effects of natural disasters (e.g. firebreaks and fodder storage).
8. Handling facilities which under normal usage do not cause injury and which minimise stress to the sheep.

### 5.4 Major diseases of sheep

It is the author’s experience that the majority of sheep losses in many areas of Australia arise from nutritional inadequacy and predation. In addition, sporadic extreme climatic events are the cause of losses, large at times, predominantly from hypothermia. These are health, production and welfare issues, but are not discussed here. It is presumed that every reasonable effort is made, making allowance for local conditions, to manage the circumstances associated with such causes of loss. Information pertinent to these conditions can be found on the various websites of state agricultural bodies, details of which are appended.
In the context of the comments above, some of the most important diseases likely to afflict sheep in Australia are listed for description in this section. Information on other conditions which arise either often but are of low severity, or infrequently, can be found in a number of excellent references, details of which are appended. Amongst these the following are quite helpful as initial information sources, listed in order of increasing detail and complexity:

- **“Sheep diseases”** (A. Brightling) gives a fairly comprehensive, good concise summary with conditions listed in alphabetical order.
- **“Sheep medicine”** (Proceedings 335, Post Graduate Foundation in Veterinary Science) has a section of sheep diseases grouped by system or syndrome which is helpful in diagnosis.

This unit deals with sheep health specifically in meat production enterprises. Most of the diseases discussed are common to all sheep enterprises, and in fact most sheep meat production comes from dual (meat and wool) systems. A number of diseases are more important for the meat production aspect of the business, and some of these are given particular attention here. (See below). Using the classification of a national expert workshop on disease impact (MLA, 2006), pre-eminent sheep diseases in Australia have been grouped as to national economic impact.

### Table 5.1 Diseases of Sheep in Australia classified by economic impact.

**Source:** MLA (2006)

<table>
<thead>
<tr>
<th>High economic impact</th>
<th>Medium economic impact</th>
<th>Low economic impact</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lice</td>
<td>Fluke</td>
<td>Nitrate poisoning</td>
</tr>
<tr>
<td>Arthritis</td>
<td>Foot abscess</td>
<td>Grain poisoning</td>
</tr>
<tr>
<td>Plant poisons</td>
<td>Trace element</td>
<td>Mycoplasma ovis</td>
</tr>
<tr>
<td></td>
<td>Deficiency</td>
<td></td>
</tr>
<tr>
<td>Blowfly strike</td>
<td>Caseous lymphadenitis</td>
<td>Pneumonia</td>
</tr>
<tr>
<td>Post weaning mortality</td>
<td>Redgut</td>
<td>Clostridia</td>
</tr>
<tr>
<td>Ovine Johne’s disease</td>
<td>Yersinia</td>
<td>Hypocalcaemia,</td>
</tr>
<tr>
<td></td>
<td></td>
<td>pregnancy toxemia</td>
</tr>
<tr>
<td>Perinatal mortality</td>
<td>Scabby mouth</td>
<td>Ovine Brucellosis,</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Actinobacillosis</td>
</tr>
<tr>
<td>Abortion, stillbirth</td>
<td></td>
<td>Dermatophilosis</td>
</tr>
<tr>
<td>Internal parasites</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Scouring</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

The presence of a disease in this list does not mean that it is necessarily of common occurrence. In fact, many are not often reported. This is because, as mentioned above, effective preventive measures are in place where they are to be expected. However the cost, in economic and management terms, is substantial and relaxation of preventive strategies is frequently associated with severe and costly disease incidence. Blowfly strike is a good example. An effective jetting program largely eliminates the condition – at a cost (chemical, labour, management calendar disruption, potential wool residues, threat of chemical resistance developing in target flies). Internal parasite management presents a similar situation.

Another approach to determining major sheep diseases is to look at submissions to state government laboratories. However this is not a representative cross section of disease, as it represents a biased sample of initially obscure field cases for which additional diagnostic means were necessary. It does give an insight into disease prevalence from a government surveillance perspective. An example is shown (Table 5.2).
### Table 5.2 Sheep disease submission diagnosis, WA 2005

Source: M. Kabay, pers. comm.

<table>
<thead>
<tr>
<th>DIAGNOSIS</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vitamin E deficiency</td>
<td>16</td>
</tr>
<tr>
<td>Enterotoxaemia</td>
<td>12</td>
</tr>
<tr>
<td>Salmonellosis</td>
<td>10</td>
</tr>
<tr>
<td>Helminthosis</td>
<td>9</td>
</tr>
<tr>
<td>Rumenitis/Acidosis - grain overload</td>
<td>9</td>
</tr>
<tr>
<td>Lupinosis</td>
<td>7</td>
</tr>
<tr>
<td>Polioencephalomalacia</td>
<td>7</td>
</tr>
<tr>
<td>Annual ryegrass toxicity</td>
<td>6</td>
</tr>
<tr>
<td>Erysipelas</td>
<td>6</td>
</tr>
<tr>
<td>Listeriosis</td>
<td>6</td>
</tr>
<tr>
<td>Nutritional myopathy</td>
<td>5</td>
</tr>
<tr>
<td>Bacterial septicaemia</td>
<td>4</td>
</tr>
<tr>
<td>Copper toxicity</td>
<td>4</td>
</tr>
<tr>
<td>Urolithiasis</td>
<td>4</td>
</tr>
<tr>
<td>Mastitis</td>
<td>3</td>
</tr>
<tr>
<td>Parasitic infestation</td>
<td>3</td>
</tr>
<tr>
<td>Pasteurellosis</td>
<td>3</td>
</tr>
<tr>
<td>Bacterial meningitis</td>
<td>2</td>
</tr>
<tr>
<td>Clostridial Disease</td>
<td>2</td>
</tr>
<tr>
<td>Cobalt deficiency</td>
<td>2</td>
</tr>
<tr>
<td>Copper deficiency</td>
<td>2</td>
</tr>
<tr>
<td>Echinococcosis (hydatid disease)</td>
<td>2</td>
</tr>
<tr>
<td>Hypocalcaemia</td>
<td>2</td>
</tr>
<tr>
<td>Oxalate poisoning</td>
<td>2</td>
</tr>
<tr>
<td>Pregnancy toxaemia</td>
<td>2</td>
</tr>
<tr>
<td>Toxic algae</td>
<td>2</td>
</tr>
</tbody>
</table>

Other diseases/conditions likely to be more important for those enterprises with meat as a specialist product are listed below. They include conditions which may have negligible effect on health or welfare at farm level, but assume importance as a cause of downgrading meat quality.

**Additional diseases significant in sheep meat production**

- Cysticercosis (sheep measles)
- Grass seed infestation

It is also likely that in a sheep meat enterprise, the degree of economic impact will be raised. For example, grain poisoning may be a more common syndrome on account of feedlotting for production being much more common. Again, caseous lymphadenitis assumes greater significance because of the potential for affected carcases to be downgraded. In summary, selected disease conditions are discussed to a degree of detail in this module; they are considered those which a sheep meat specialist enterprise may encounter and need to deal with on farm. Some diseases not discussed but merely referred to are important, but are ones which can be eradicated from a property. Footrot and lice are examples. That is, the majority of enterprises will not be dealing with these diseases on a day to day basis, or are unlikely to encounter them. Where they do, these diseases assume high priority and significance. Detailed references are appended.
5.5 Blowfly strike (cutaneous myiasis)

This condition is one of the “big three” diseases of sheep in Australia. Since it was acknowledged as a problem around the year 1900, it has continued to plague sheep nationally. Even with strategic chemical treatments and a range of management modifications, losses continue. It has been estimated that of a total annual cost of $161 million, $130 million is spent on control, and up to 3 million sheep still die from flystrike.

Blowfly strike is caused by the feeding of fly larvae on the skin of sheep. It generally is of short duration (days to 2 weeks), but unapparent (covert) strikes may persist for longer if secondary flies are not involved. With the right conditions strike can be fatal within days.

**Conditions for blowfly strike to occur:**

**a) Susceptible sheep**

Flies do not initiate a strike on normal skin; it must become attractive to the flies. The attraction is associated with odour, generated in a variety of ways, often by bacteria. The skin must also have a source of liquid protein, necessary for the newly-hatched larvae.

Strikes are customarily categorized in relation to their position on the sheep’s body; in discussing these, the various predisposing factors become evident.

- **Breech strike** is the most common form. The wool and skin easily become wet with urine (in the case of ewes) or moist faecal material (dags, scouring). The tail is also likely to be struck.

- **Body strike** occurs if the fleece and skin are wet for a prolonged period. It is particularly likely where there is fleece rot or dermatophilosis (lumpy wool).

  *Fleece rot* is a bacterial skin infection, arising where the skin is moist for a week or more. These conditions are favorable for the rapid growth of the bacteria *Pseudomonas aeruginosa*, and other bacteria, which generate odours attractive to blowflies. The dermatitis provides the liquid protein. The bacteria also produce pigment which stains the wool, the colours blue/green to brown being characteristic.

  *Lumpy wool (Dermatophilosis, “Dermo”) is another bacterial skin infection, caused by Dermatophilus congolensis.* Signs are crusty scabs together with matted wool. These scabs start on the skin, and eventually lift and lift away from the skin with the wool growth. The dermatitis while active provides the fly attractant and protein, and as little as 4 mm of rain can make the lifted dry scabs conductive to eggs lays and strike. Dermo is of extra significance for those consigning sheep for slaughter, as it downgrades the value of skins.

- **Poll strike** describes the condition around the head, typically of rams with horns growing close to the skull, and acquiring wounds in fighting.

- **Pizzle strike** occurs when the urine-soaked wool and skin around the prepuce of rams or wethers becomes attractive to flies.

- **Wound strike** can occur anywhere. Mulesing is an obvious possibility at certain times. Skin damage associated with conditions such as scabby mouth and footrot provides suitable conditions.

**b) Blowflies**

The primary strike fly in Australia is *Lucilia cuprina*, the Australian sheep blowfly. This is a small green fly with green forelegs. It is responsible for initiating 90% of strikes.

The mature female fly is attracted to the susceptible sheep and may lay 2 to 3 batches of 50 to 250 eggs. These normally hatch in 12 to 24 hours, and the larvae (maggots) move to the skin surface and begin feeding. In 3-6 days after hatching the larvae moult twice; the second and third instars have abrasive mouth parts and inflict more damage. After this time the larvae drop off the sheep and pupate in the soil.
In ideal summer conditions this pupal stage may be as short as 7 days; in winter, however it may last as long as 4 months (this is the means of flies persisting between periods conducive to strike). The minimum time from egg to adult is 14 to 17 days. Fly trapping has revealed that the first appearance of *L. cuprina* in a trap is ample notification that strikes are imminent. Low numbers are sufficient to initiate a problem.

Once *L. cuprina* has initiated a strike, the resultant dermatitis and skin damage attracts **secondary flies**. The most severe of these is *Chrysomya rufifacies*, the **hairy maggot blowfly** (so called because of the appearance of the larvae). These flies cannot initiate a strike, but once they are involved the existing damage rapidly gets much worse. The maggots have abrasive mouth parts and are larger and more vigorous than those of *L. cuprina*, which are soon driven out.

**Figure 5.1** The lifecycle of *Lucilia cuprina*, the Australian sheep blowfly.

### c) Moisture
Both soil and fleece moisture are required for the fly life cycle. As little as 4 mm of rainfall can render lumpy wool scabs suitable sites for egg lay. Depending on the region of the body, moist fleece or skin results from rainfall, sweat, dermatitis, urine or faeces.

### d) Environmental factors
Other than rainfall, factors playing a critical role in the initiation of flystrike are:

**Wind** This is a key variable. Wind speeds above 5m/sec substantially lower the activity of adult flies, and encourages more rapid drying of susceptible lesions. Strike is therefore far less likely. All blowfly activity ceases with wind speeds greater than 10 m/sec.

**Temperature** The maximum temperature needs to reach at least 17°C before adult female flies are actively seeking and ovpositing on attractive sites. Above 35°C *L. cuprina* is much less active, and at temperatures above 40°C activity ceases completely. Soil temperatures between 13 and 30°C are ideal for pupal development.

**Carrion** Although blowflies are attracted to and lay eggs on carrion, this is not as great an issue with regard to multiplication of the population as might be thought. The more vigorous and destructive larvae of the secondary flies out-compete the *L. cuprina* larvae as described.
Detection
The irritating nature of the strike causes the sheep to bite at or twitch the affected area. Affected sheep have a reduced appetite, and become weak and dull. Respiration rate is increased, and toxemia causes increased temperature and heart rate also. The wool in the affected area is wet, darkened and foul-smelling. The sheep stands or lies apart from the flock. Death supervenes without treatment, especially when secondary strike has taken place. The generalized toxemia is severe enough to result in a complete break in the wool; the whole fleece may be seen to be detached and progressively lost in sheep that survive.

Treatment and control
At present this depends mainly on the use of a numbers of chemicals with insecticidal activity.

Struck sheep need to be treated quickly; ideally the wool over the struck area, and surrounding 10 cm, should be removed. To the surrounding wool and maggot-infested area is then applied one of a number of insecticides:

- Spinosins (eg Spinosad)
- Macrocylic lactones (eg ivermectin)
- Cyromazine and dicyclanil are insect growth regulators.
- Diflubenzuron and triflumuron are insect development inhibitors.
- (Organophosphates, commonly used in the past, are not now recommended due to wool residue, occupational health and safety, and fly resistance issues.)

Control is affected by strategically applying to the fleece one of the above compounds by hand or race jetting, dipping, backline or tip spray. Instructions and withholding intervals should be carefully observed. There are meat export slaughter intervals as well as withholding periods.

Shearing at a time of the season just before expected fly activity is effective, although other management activities may well take priority.

Genetics may in the future play a dominant role in combating fly-strike; large differences exist between Merino genotypes in their susceptibility to flystrike. In general, fine wool genotypes are more resistant, as are less wrinkled sheep. British breed sheep are less likely to be struck than Merinos.

Crutching is an essential husbandry for Merino sheep to reduce the likelihood of breech strike. Merino sheep are generally mulesed. The effectiveness of this in the prevention of flystrike has been well demonstrated. The decision by the sheep industry to cease this practice will require sound alternatives to be in place.

Fly trapping, although effective in reducing fly population numbers, has not proved uniformly effective in preventive fly strike.

5.6 Internal parasites (“worms”)

Gastro-intestinal parasites are one of the major on-going problems, indeed threats, for the sheep industry in much of Australia. Because of the life cycle and the acquisition of infective larvae from pasture, there exists a continuous state of flux in the balance between parasite and sheep. Most sheep management systems are intensive enough that some chemical intervention is necessary to aid the sheep in this balance, and the escalating rate at which most important parasites are developing resistance to these chemicals is cause for concern.

Of the internal parasites of sheep, most inhabit the gastro-intestinal tract. Lungworm infection can be important locally but is not a major disease under Australian conditions and will not be considered further. It is very adequately controlled by all common anthelmintics.
Table 5.3 Broad classification of the major parasites of sheep. Source: Bell, (2007).

<table>
<thead>
<tr>
<th>Helminths (Worms)</th>
<th>Roundworms (Nematodes)</th>
<th>Flukes (Trematodes)</th>
<th>Tapeworms (Cestodes)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Haemonchus contortus Trichostrongylus spp. Ostertagia circumcincta Numerous other spp.</td>
<td>• Fasciola hepatica</td>
<td>• Monezia spp.</td>
<td></td>
</tr>
</tbody>
</table>

The roundworms (nematodes) are by far the major problem. Liver fluke is a significant threat, but in a restricted area. The tapeworms, although the most apparent, have no significant effect on sheep and are not considered a problem.

Gastro-intestinal nematode infection
This is the major disease problem of the sheep industry in all but the low rainfall pastoral areas of Australia. It is a chronic disease, with loss of animal production being a major feature. However, infection can be acute and also fatal.

Species and life cycle
Table 5.4 lists the common species in Australian sheep. The three most important species in Australia are:

*Haemonchus Contortus* (Barber’s Pole worm). The mature worm lives in the abomasum, attaching to the stomach lining and feeding by sucking blood. It thrives in warmer, wetter regions and seasons.

*Trichostrongylus* spp. (Black Scour worm). The mature worm lives in association with the epithelium of the first 3-4 metres of small intestine. Several species occur depending on climate, so it is the most widespread of the worms. These worms can handle cooler, dryer conditions better than Haemonchus.

*Ostertagia spp.* (also known as *Teladorsagia* spp. Small Brown Stomach worm). The mature worm lives deep in the crypts of the abomasum (fourth stomach). This is a parasite of cool/cold wet regions.

Table 5.4 Common gastrointestinal nematodes of sheep in Australia and their prevalence in the different rainfall zones. Source: Bell, (2007).

<table>
<thead>
<tr>
<th>Specific name</th>
<th>Common name</th>
<th>Location</th>
<th>Rainfall zone</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Summer</td>
</tr>
<tr>
<td><em>Haemonchus contortus</em></td>
<td>Barber’s Pole worm</td>
<td>Abomasum</td>
<td>+++</td>
</tr>
<tr>
<td><em>Trichostrongylus</em> spp.</td>
<td>Black Scour worm</td>
<td>Small intestine</td>
<td>+++</td>
</tr>
<tr>
<td><em>Ostertagia circumcincta</em></td>
<td>Small Brown Stomach worm</td>
<td>Abomasum</td>
<td>++</td>
</tr>
<tr>
<td><em>Nematodirus</em> spp.</td>
<td>Thin Necked Intestinal worm</td>
<td>Small intestine</td>
<td>++</td>
</tr>
<tr>
<td><em>Oesophagostomum</em> spp.</td>
<td>Large Bowel worm/Nodule worm</td>
<td>Large intestine</td>
<td>++</td>
</tr>
<tr>
<td><em>Chabertia</em> spp.</td>
<td>Large Mouthed Bowel worm</td>
<td>Large intestine</td>
<td>+</td>
</tr>
<tr>
<td><em>Trichuris</em> spp.</td>
<td>Whip worm</td>
<td>Large intestine</td>
<td></td>
</tr>
</tbody>
</table>

The life cycle is very similar for these species and involves phases in the host animal (parasitic) and in the environment (Figure 5.3). This poses a major challenge in control, because while it is relatively easy to target the parasite while it is in the host, the environmental stages of the life cycle are much more difficult to attack.
The life cycle is direct, with no intermediate hosts. Sheep become infected with nematodes when feeding on contaminated pastures, consuming infective larvae while grazing. The larvae pass into the gastrointestinal tract where they develop into mature adults. These adults lay eggs which pass out in the faeces of the host. Once the eggs hatch, the larvae undergo three stages of development (without multiplication), with the L3 stage being the infective stage. The L1 and L2 larvae live on bacteria and fluids in the faeces and soil but the infective L3 larvae are encased in a protective sheath and cannot feed until ingested by a host. Large numbers of eggs, L1 and L2 larvae perish from desiccation and low oxygen tension and typically only 0-5% of eggs develop into infective L3 larvae. Once ingested, L3’s undergo exsheathment and develop into adults over a 16 day period.

Figure 5.2 Lifecycle of typical gastro-intestinal nematode parasite of sheep. This lifecycle is common to all of the major genera such as Haemonchus, Trichostrongylus and Ostertagia.

Source: Bell, (2007).

In Nematodirus, the L1 to L3 development occurs within the egg making it particularly resistant to adverse environmental conditions. In Trichuris, the eggs hatch once they are eaten by the host. The duration of the lifecycle varies widely, depending on environmental conditions and worm species. But under optimum conditions it can be as short as 3 weeks from egg to egg (16 days in the host and 5 days in the environment). When environmental conditions are unfavourable some L4 larvae enter into hypobiosis (arrested development) in the wall of the gut and remain in this state, only resuming development when conditions improve.

Factors affecting distribution and prevalence

Environmental factors

Temperature. An air temperature of >10°C is required for larval development of most species and >15°C for Haemonchus. Rate of development and the speed of the lifecycle increases with increasing temperature. Very high temperatures are lethal, but the effects of high temperatures are mainly mediated by increased desiccation.

Moisture. Desiccation is the major cause of losses in the environment. Eggs, L1 and L2 are most affected.

The most favourable combinations of temperature and moisture are often found in during spring and autumn although summer rainfall areas have a summer problem with Haemonchus.

Parasite factors

Egg production. This varies greatly between species and to some extent is largely a function sheep immune status. High levels of egg production allow rapid build up in parasite numbers and explosive outbreaks of disease to occur (eg. Haemonchus).

Pathogenicity of adult worms. Haemonchus adults are approximately 6-8 times more pathogenic than Ostertagia, Nematodirus and Trichostrongylus adults and L4 larvae.
Resistance to cold and desiccation. *Haemonchus* eggs and larvae are most susceptible to desiccation, *Trichostrongylus* and *Nematodirus* most resistant. Similarly, *Haemonchus* and *Oesophagostomum columbianum* eggs and larvae are most susceptible to cold, with *Ostertagia* and some *Trichostrongylus* species most resistant.

**Anthelmintic resistance.** This is a very serious and growing problem! It is widespread for the levamisole (“clear drench”) and benzimidazole (“white drench”) groups and closantel, and is increasing rapidly with the macrocyclic lactones

**Host factors**

**Immunity.** Sheep develop acquired or “age immunity” as they grow. Young sheep in the first 18 months of life are the most susceptible to infection. Immunity is influenced by:

*Worm species.* *Haemonchus* induces less immunity than the other species and remains a threat to adult sheep.

**Degree of challenge.** Immunity requires exposure to infection.

**Host genotype.** There is within and between breed variations in resistance to helminth infection. The heritability of faecal worm egg count is approximately 0.25.

**Physiological state.** Immunity is almost totally lost during late pregnancy and lactation. This leads to a “peri parturient rise” in faecal egg counts.

**Host nutrition.** This affects both resistance to infection (numbers of worms in the host) and resilience (ability to continue to produce for a given number of worms).

**Stocking rate/grazing management.** This has profound effects on the level of pasture contamination with eggs and infective L3 larvae (see section on control).

**Pathogenesis**

Worms exert their detrimental effect on the sheep in the following ways:

**Reduction in feed intake.** This is the major problem. The extent varies with all the factors mentioned above, but reductions of up to 20% have been seen in young animals showing no clinical signs of disease. Intake generally returns to normal after immunity is acquired, or the sheep are treated.

**Reduced efficiency of protein digestion.** Loss of protein from the gut wall is a feature. Although much of this protein is reabsorbed further along the gut, the process is associated with a cost in energy and protein.

**Diversion of nutrients from production to immune response.** Overall there is a reallocation of nutrients, particularly protein, from productive processes such as weight gain, wool growth and lactation, to repairing damage and mounting an immune response. The latter may require a cost up to 15% that of maintenance.

**Clinical Signs and Diagnosis**

The major clinical signs of nematode infection in sheep are: ill-thrift in young stock, anaemia and scouring (diarrhoea).

All nematode worm species induce ill-thrift and most induce scouring. Few are associated with anaemia, the major species being *Haemonchus contortus*. Scouring and ill-thrift can also be caused by other factors other than worm burdens and for this reason, are very poor indicators of infection. Also, by the time these clinical symptoms are evident, substantial sub-clinical losses will already have occurred. Infection with *Trichostrongylus spp.* is often (but not always) associated with dark green to black scours. This provides a suitable environment for the development of larvae of the sheep blowfly (i.e. breech strike). Severe infection with *Haemonchus* can result in the accumulation of fluid under the lower jaw, known as “bottle jaw”, due to the loss of protein from plasma.

Note that *Haemonchus* can be fatal, even in adult sheep. Very severe infestations of all worms associated with poor nutrition can kill sheep. Diagnosis is aided by reference to worm egg counts (WEC) from a number of sheep in the affected mob. 10 to 20 samples are generally recommended. The interpretation of the number of worm eggs in the sheep faeces (measured in eggs per gram of faeces,) requires skill and experience, and is used as a decision aid with regard to the need for treatment, moving to alternate pastures or other management interventions. Where necessary to diagnose individual worm species, larval culture to hatch the eggs and allow larval differentiation is performed. Total worm counts from sheep after slaughter can indicate adult numbers, but unless special techniques are employed infective larval presence is overlooked. The latter may be the major cause of losses.
Management and control
The objective of worm control is to limit to manageable levels the numbers of infective larvae on pasture, combined with allowing young sheep sufficient exposure to larvae to safely stimulate immunity. This is done with a combination of anthelmintic treatments (drenches) and judicious use of pastures with less larvae. The latter can arise through planned management such as:

i) Prior grazing with dry sheep (eg adult wethers) with low worm burdens and low WEC (needs to be confirmed).

ii) Prior grazing with cattle.— Alternating grazing between cattle and sheep has been demonstrated to be an effective means of reducing worm infection. This process relies on the fact that most worms are host specific – most worms are only able to successfully infect either sheep or cattle, not both, and ingestion by the non-preferred host results in death of the larvae. An exception is the ability of Haemonchus contortus and Trichostrongylus axei to successfully reproduce within sheep and young cattle (i.e. pre-weaning). Using adult cattle for cross grazing is preferable.

iii) Use of crop residues.

iv) Strategic anthelmintic treatment associated with summer hot and dry conditions where present, as in southern Australia. The timing of treatments will vary with regional climate. Each state and region has recommended overarching programs, guided by local knowledge and informed interpretation of WEC. It cannot be stressed enough that there is no overall “prescription” that can be relied upon.

Some recognised “programs”, and a summary of their main characteristics, are:

**Summer rainfall regions** – “WormKill” (Love, 2005) eg northern NSW

- Mid October: closantel (see below under “Anthelmintic resistance” for comment) to all sheep
- Late December: closantel to all sheep, broad-spectrum drench to all sheep
- Late February: closantel to all sheep, broad-spectrum drench to young sheep
- April/May: broad-spectrum drench to young sheep

The widespread development of closantel resistance necessitates considerable variation with the above: substitution of other drenches, monitoring by WEC to lessen the need for drenches, use of safe pastures, cattle etc.

**Summer/winter rainfall regions** – “DrenchPlan” (Love, 2005) eg central and southern NSW, and winter rainfall regions, much of Victoria.

One or two summer drenches. The first in November/December when pastures are drying off, the second in mid-summer, generally February. The necessity for the second is governed by climate, WEC and the availability or otherwise of low-risk pastures or crop residues. Young sheep may need a winter drench as indicated by WEC and experience.

Lambs should be drenched at weaning. Other drenches as indicated by WEC eg young sheep, ewes pre-lambing, lamb-marking.

**Smart grazing for winter rainfall districts** – A simple and reliable strategy for the control of worms in weaner sheep during their first winter in winter rainfall districts. An example of a smart grazing timetable is shown in Table 15.4 below.
Table 5.5 Smart grazing timetable. Source: AWI/Sheep CRC (2005).

<table>
<thead>
<tr>
<th>Month</th>
<th>Strategy</th>
</tr>
</thead>
<tbody>
<tr>
<td>October</td>
<td>Select the 'smart grazing' paddock – choose one with a history of good winter pasture</td>
</tr>
<tr>
<td>November</td>
<td>Give the first summer drench (MUST be an effective product), then intensively graze the paddock at 2.5-3 times the normal stocking rate</td>
</tr>
<tr>
<td>December</td>
<td>Remove the sheep to another part of the farm after 30 days intensive grazing. Ideally, the pasture residue should be 800-1000 kg DM/ha (2.2-3 cm)</td>
</tr>
<tr>
<td>January</td>
<td>Paddock remains unstocked until the second summer drench</td>
</tr>
<tr>
<td>February</td>
<td>Give the second summer drench, then intensively graze the 'smart grazing' paddock with the drenched sheep (again, not &gt; 30 days)</td>
</tr>
<tr>
<td>March</td>
<td>Paddock remains de-stocked until the autumn break</td>
</tr>
<tr>
<td>Autumn break (March-April)</td>
<td>Drench weaners and set-stock on the 'smart grazing' paddock when pasture &gt; 600 kg DM/ha (1.5 cm). Weaners can remain there until spring but monitor their worm egg counts every 4-6 weeks</td>
</tr>
</tbody>
</table>

Merino weaners are very susceptible to worms in their first winter. Consequently, they need to graze pastures that have as few worm larvae as is practicable. 'Smart grazing' combines intensive grazing for 30 days with each of the two 'summer' drenches to ensure that virtually no worm eggs are deposited on a chosen pasture from the first summer drench (November) until after the autumn break (March-April), when the weaners are put into these pastures.

Results from a controlled experiment over 2 years in western Victoria (as quoted in WormBoss (AWI/Sheep CRC 2005) show that, compared to weaners grazing paddocks prepared the usual way (grazed by wethers over the summer/early autumn), weaners grazing 'smart grazing' plots: grew 13% more clean wool (2.29 vs. 2.03 kg) which was 3.5% broader (17.1µm vs 16.5µm) and were 3 kg heavier in October (46.5 vs. 43.2 kg).

“Mediterranean” climate regions – more concentrated winter rainfall pattern, more reliably dry and hot summers. eg much of the sheep raising regions of Western Australia and South Australia.

A modified version of the above, taking into account the extreme pressure on worm resistance development likely when drenching in hot dry summers and onto crop residues. The principal is to avoid drenching in hot dry conditions when few worms are “in refugia” and therefore can contribute chemically unexposed members to subsequent worm generations. Generally one summer drench is adequate, either early (November/December) or late (March onwards) as indicated by WEC. If drenching early in summer, leave a small proportion (5%) of adult sheep undrenched as a source of refugia.

Drench all young sheep (weaners) at weaning, and early summer if these are not coincident.

Because of the nationally universal presence of drench resistance (see below), it is essential that the effectiveness of any drenches used be established with a reasonably current drench resistance test, within the last 3 years at least (see below).

Long term management of sheep worms will depend upon breeding worm resistant sheep. The success of this approach has been amply demonstrated for Merino sheep, and it is taken for granted that future control will incorporate this strategy.

See “Management of Helminth Parasites of sheep in Australia” (B. Besier, webCT reading), for detailed programs applicable in different regions.
Special considerations for prime lamb production
Most prime lamb production enterprises are in medium to high rainfall regions and hence parasite control is essential. Prime lamb enterprises are very often ewe dominant; that is they are unlikely to have significant numbers of dry sheep such as wethers to use in rotation as an aid to reduce pasture infective larval numbers. Extra parasite control may be needed to ensure maximum lamb growth rate for lambs either whilst still with the ewes and/or after weaning. WEC can be used as a guide to drenching.

Parasites may limit the growth of prime lambs, with no apparent signs (particularly in unweaned prime lambs). The foregone weight is simply not appreciated, but significant loss of potential income is the result. The possibility of this was amply demonstrated in a survey of sheep and lambs arriving at an abattoir in Western Australia (Bath et al. 2005).

This survey was instigated in response to concerns of abattoir operators about scouring sheep, particularly lambs in winter and spring months. Samples were collected from both adults and lambs on arrival at a Western Australian abattoir. Results demonstrated that significant numbers of lines of lambs had a WEC high enough to assume that some production loss had occurred (Table 5.6). The fact that these sheep on farm had reached a weight and condition suitable for consignment without any signs prompting anthelmintic intervention is a salutary message for producers in intensive lamb producing enterprises.

Table 5.6 WEC in sheep sampled in lairage
(Source: Bath et al, 2005)

<table>
<thead>
<tr>
<th>WEC Description</th>
<th>Lambs (&lt; 12 months)</th>
<th>Adults (&gt; 2 years)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lines &gt; 1000 epg</td>
<td>43%</td>
<td>13%</td>
</tr>
<tr>
<td>Lines &gt; 2000 epg</td>
<td>22%</td>
<td>6%</td>
</tr>
<tr>
<td>Lines scouring</td>
<td>9.5%</td>
<td>9.3%</td>
</tr>
<tr>
<td>Average WEC (all worms excluding Nematodirus)</td>
<td>1525 epg</td>
<td>486 epg</td>
</tr>
<tr>
<td>Average WEC (Excluding Haemonchus)</td>
<td>1150 epg</td>
<td>364 epg</td>
</tr>
</tbody>
</table>

Anthelmintic resistance
Discussion of worm control in sheep cannot take place without highlighting that throughout Australia, worms are continuing to become more resistant to the chemicals used. This degree of resistance has continued to progress at an alarming rate, such that control programs now must take it into account with regard to the chemical used and the timing of treatments. Anthelmintic resistance is defined as a situation where there is a greater frequency of individuals within a population able to tolerate doses of a compound than in a normal population of the same species and is heritable (Pritchard et al, 1980).

Currently, of the 3 distinct chemical classes of broad-spectrum drenches commonly used, 2 (BZ and LV groups, see below) are now so ineffective that they can rarely be used successfully on their own, although used in combination they may be suitable.

The three groups of drenches are:

Benzimidazole group (“BZ”, “white” drenches)
Levamisole group (“LV”, “clear” drenches)
Macrocylic lactone group (“ML” drenches)

In addition the organophosphate Naphthalophos has a useful but variable effect, depending on worm species and circumstance. Closantel is a narrow-spectrum drench for use only against Haemonchus contortus. It is part of control programs in summer rainfall regions, but widespread resistance means that its effectiveness can never be presumed. The effectiveness of the BZ and LV groups is so poor as to be not generally worth testing, but a local example of the effectiveness of a number of drenches and drench combinations can be gleaned from Table 5.7.
Table 5.7 The average % reduction over time in WEC for selected drenches and combinations, south-western Australia. Source: Bell, (2005) unpublished

<table>
<thead>
<tr>
<th>Drench/mixture</th>
<th>1998</th>
<th>1999</th>
<th>2000</th>
<th>2001</th>
<th>2002</th>
<th>2003</th>
<th>2004</th>
</tr>
</thead>
<tbody>
<tr>
<td>BZ/LV</td>
<td>87</td>
<td>90</td>
<td>87</td>
<td>71</td>
<td>82</td>
<td>85</td>
<td>83</td>
</tr>
<tr>
<td>BZ/LV/naphthalophos</td>
<td>99</td>
<td>94</td>
<td>97</td>
<td>88</td>
<td>97</td>
<td>94</td>
<td>96</td>
</tr>
<tr>
<td>ML - abamectin</td>
<td>98</td>
<td>97</td>
<td>98</td>
<td>99</td>
<td>99</td>
<td>99</td>
<td></td>
</tr>
<tr>
<td>ML - moxidectin</td>
<td>100</td>
<td></td>
<td></td>
<td>99</td>
<td>100</td>
<td>99</td>
<td></td>
</tr>
<tr>
<td>ML - ivermectin</td>
<td>95</td>
<td>92</td>
<td>93</td>
<td>90</td>
<td>80</td>
<td></td>
<td></td>
</tr>
<tr>
<td>BZ/LV/ivermectin</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>96</td>
<td></td>
</tr>
</tbody>
</table>

**Drench resistance testing**

This test is at present the only practicable method of estimating the effectiveness of any drenches, alone or in combination, used as part of a sheep worm control program. It relies upon measuring the WEC of young sheep before and after administration of the drenches, hence the description of the test as a “Faecal Egg Count Reduction Test” (FECRT). It is acknowledged that the test is imprecise, but if sufficient numbers of young, immunologically naïve sheep are used, with high enough initial WEC, and the treatments done with care, the presence or absence of a significant degree of resistance can be discovered. The convention in Australia is that resistance is deemed to be present when a drench fails to remove at least 95% of a particular parasite (or rather, when the WEC fails to be reduced by at least 95%).

**Note:** Holding sheep off feed prior to and following treatment – Fasting sheep for up to 24 hours before treatment can be used to improve the efficacy of treatments with BZ, ML or closantel products as it slows the flow of digesta through the gut of the sheep and therefore keeps the drench in the gut for a longer period of time, allowing greater absorption of the active ingredient. Keeping sheep off feed for up to 6 hours after treatment can also assist drench efficacy. However, fasting strategies should not be used when treating with levamisole or naphthalophos as this could increase the risk of toxicity with these products. Fasting should not be used in heavily pregnant, stressed or poor sheep and they should have access to water during the fasting period.

**Liver fluke disease (Fascioloisa)**

This parasitic condition is potentially severe, although occurring in restricted localities throughout Australia. The regions where this disease is found coincide with the distribution of a snail, *Lymnaea tomentosa*, the intermediate snail host. This includes much of Victoria, the eastern half of NSW and parts of South Australia, Queensland and Tasmania. The cause is the liver fluke, *Fasciola hepatica*, which can infect sheep and cattle (as well as horses, deer and goats). Sheep are more susceptible than cattle.

A flat, leaf-like parasite, liver fluke has a complex life cycle. Adult flukes are pale brown or greyish-brown in colour and when mature vary from 15 mm to 40 mm in length and up to 12 mm in width. A liver fluke burden can result in deterioration in wool quality, reduced meat and milk production and ill-thrift in young stock. Acute infestations can be fatal. If flukes are detected in livers at the abattoir, the livers are condemned as unsuitable for human consumption.

**Pathogenesis**

After ingestion of infested pasture by a host animal, the young flukes penetrate the animal’s intestinal wall and enter the peritoneal cavity. From there they penetrate the liver where they mature and cause damage as they move around. The damage done may be so severe as to cause a rupture of the liver capsule and haemorrhage into the peritoneal cavity, which may result in sudden death. The adult flukes may also cause inflammation to and blockage of the bile ducts leading to jaundice and cirrhosis of the liver.

**Life cycle**

Liver flukes mature and live in the bile ducts of the host where they lay large numbers of eggs. The eggs are then passed down the bile ducts and enter the intestine to eventually be excreted with the faeces. With favourable circumstances – water and moist conditions – the eggs hatch into larvae (miracidia) which invade the intermediate host snail. To survive and reach the host snail, which must be within 24 to 30 hours as they have a short life span, the larvae need temperatures above 5°C with the optimum temperature being 15°C to 24°C. After five to eight weeks and several larval
stages later, depending on the temperature, larvae (cercariae) emerge from the snail. These larvae form cysts (metacercariae) on herbage and are eaten by cattle and sheep. The larvae then migrate to the animal’s liver before entering the bile ducts. Occasionally a fluke may migrate through other organs or may infect the unborn foetus. The liver is damaged during this migration. This damage alone may kill the animal or may make it susceptible to black disease (see clostridial diseases).

**Diagnosis**

Chronic liver fluke can be diagnosed by worm egg count. The eggs have a characteristic shape. At autopsy the adult flukes can be seen in the bile ducts. The acute form, with extreme liver damage caused by the migrating immature fluke, can only be diagnosed at autopsy. The snail can be identified by its characteristic cone-shaped shell and clockwise spiral when viewed from the apex.

**Treatment and control**

In fluke regions, strategic use of closantel may help, but the most effective chemical is Triclabendazole. This is the only drug with an effect on all stages. As for the nematodes worms, regional control programs are utilised. Control of the snail’s habitat, or restricting sheep access, is valuable if at all practicable.

**5.7 Arthritis**

Arthritis, from a number of causes, is common in Australian sheep flocks. In larger wool-growing enterprises the extent of infection is often not truly appreciated until weaning, or as hoggets, but in fact most cases begin in lambs before weaning. Apart from death and loss of production, arthritis causes additional economic loss for meat enterprises, being responsible for carcase condemnation and downgrading in abattoirs.

**Cause**

Arthritis is inflammation of joint surfaces. A number of different bacteria gain entrance to the body early in life. The umbilical cord of the newborn lamb, and skin wounds including lamb marking activities, are very likely routes. It has been found that a common cause is the bacteria *Erysipelothrix rhusiopathiae*. This is the same bacteria which causes disease in pigs, for which a vaccine is available.

**Signs**

When the bacteria first lodge in the joints (circulating in the bloodstream, they have a natural predilection for this site) there is local heat and swelling, very likely with a generalized fever. The knees and hocks are the joints most commonly affected. The heat and swelling subside after a few days, but there remains restriction of movement to varying degrees, associated with thickened joint capsules. The joint fluid becomes thickened and loses its lubricative qualities. Damage to the joint cartilage follows, and pain further restricts movement. With the chronic lameness the sheep feeds less, and suffers ill-thrift often permanently. Well fed lambs before weaning can tolerate the effects of arthritis to some degree, but the lesions at slaughter are often severe enough to result in carcase trimming, downgrading and in some cases rejection.

**Prevention**

Hygiene at all lamb interventions is essential. Any knives etc. used should be thoroughly sterilized between lambs. A Western Australian study looked at factors associated with arthritis incidence. The authors concluded that to reduce the incidence of arthritis lambs raised for meat production should not be mulesed or shorn. Navel infection is impossible to forestall. If the incidence of arthritis is unacceptably high, vaccination with Erysipelas vaccine may well be successful, as that organism is quite likely to be involved. As in the field, the bacteria poses a threat immediately after birth, the lamb needs passive immunity transferred from the ewe in the colostrum. This is produced by giving the ewe two doses of vaccine, at least 4 weeks apart, the second ideally about 2 weeks before lambing is due to start. It is satisfactory to give the first dose at joining time.
5.8 Scouring

Scouring (diarrhoea) of sheep is never a normal state. Whereas cattle often have very fluid faecal material this is not customary for sheep. The condition of scouring in sheep is compounded by the soiling of breech wool (“dags”), inevitable with more than a few months growth. This soiling creates a predisposing state for flystrike, and in the case of sheep for meat production contamination of carcasses at slaughter and soiled, stained skins which are downgraded at cost to both farmer and abattoir. Adult sheep in southern Australia are commonly affected by soiling of the breech area during winter and spring. This occurs on well-managed farms adopting best practice in all areas of management, and remains a cost to the industry.

Because this scouring is associated with lush pasture, and high worm egg counts are not a common feature, internal parasites were not thought to be involved. However it has been well demonstrated that the scouring is essentially a function of a hypersensitivity reaction to worm larvae in the intestine of some sheep, not related to the level of intake. Scouring is also associated with high levels of larval intake and adult worm burdens. The larvae of worms of winter rainfall regions, *Ostertagia circumcincta* and *Trichostrongylus spp.*, are implicated in this reaction. Some sheep are susceptible to this condition, and very small levels of larval intake induce the problem. On the other hand in sheep not susceptible even larges doses fail to provoke scouring. Where lambs are scouring as a result of worm infestation, high levels of larval intake can be suspected.

Other causes of scouring
The scouring referred to above is not necessarily considered “pathological”, unless worm infestation of larval intake is sufficient to cause loss of production. There are, however, occasional disease outbreaks associated with infectious agents which are certainly of extra concern. Examples of these are merely listed here; information can be found in the references listed. This list is not exhaustive; other, not so common, causes of scouring do exist.

Salmonellosis, Coccidiosis, plant poisons – all ages
Weaner colitis, Campylobacteriosis - weaners
Yersiniosis – sheep > 1 year

Signs
The presence of dags on sheep is very obvious. The scouring can develop very quickly in a mob, affecting a considerable proportion within a week or less. This is nearly always on lush winter and spring pasture

Prevention and control
Best practice worm management is strongly advised, in spite of the failure to prevent the problem. This is discussed in a separate section of the module. Severe worm infestation does cause scouring along with considerable production loss. In the long term, culling of susceptible sheep and selection against such sheep in breeding programs is considered the most likely to succeed. Avoiding sudden diet changes, especially to highly digestibly diets, is recommended. In the case of scouring associated with infectious agents, the appropriate diagnosis and treatment is necessary. This is not dealt with in this module.

5.9 Mineral deficiencies

Introduction
At present nineteen of the naturally occurring mineral elements are known to be essential for normal sheep function. Mineral deficiencies may cause noticeable clinical disorders, but when marginal or transient are often associated with minor undetected but significant effects on production. Deficiencies of minerals usually occur within specific localities, influenced by soil type and climate, principally rainfall. Other influential factors are time of year, composition and age of pasture, and fertilizer usage. The age, sex and reproductive status of sheep will also influence mineral requirements. It is important to be aware of local mineral status, as ill-informed provision of supplements may be responsible for problems, either directly or indirectly by inducing a deficiency of other minerals. Although mineral supplementation may be associated with a measurable effect on production, this may not be necessary or economic.
Mineral deficiencies occur in all continents, and affect production of livestock. While severe deficiencies still do occur, they are becoming less frequent due to improvements in awareness, identification and treatment. Marginal or subclinical deficiencies are not so readily identified and can still cause losses in production. However local knowledge generally is responsible for preventive measures being instituted.

Too little or too much?
In any discussion on minerals it is necessary to deal with those which are not deficient, as much as those which may be deficient, and for which supplementation is prudent. Factors leading to unnecessary or overuse of mineral supplements are many, and include:

1. Historical deficiencies during early land use.
2. Seasonal nutritional fluctuation and deficiency throughout much of the state. Observed loss of production in livestock, attributed to minerals. Failure to acknowledge the overriding importance of energy and protein deficiencies.
4. Marketing of commercial products. Purchasing such supplements gives people a sense of having done something in times of sometimes serious nutritional inadequacy.
5. Misleading, unsubstantiated theories of mineral activities and requirements.
6. The theory that ‘a little is good, more must be better’. Unnecessary provision of minerals is not just uneconomic and wasteful; it may be dangerous for sheep health. Excess of one mineral may induce a deficiency in others.

Factors influencing mineral availability
Many Australian soils are extremely infertile due to their extensive weathering. Although historically major deficiencies existed, many of these have been rectified. Since clearing and sowing of improved leguminous pastures and a range of crops, the use of superphosphate has corrected in many instances three deficiencies - phosphorus, sulphur and nitrogen. Additionally, on soils derived from laterite, it was identified some time ago that the trace elements copper, zinc and molybdenum were required for pasture legume persistence and for crop production. In more limited areas, manganese is required for optimum plant growth. These have generally been applied, to the extent that plant growth is not impeded. In most cases animal requirements are met by these adequate plant levels, although high-producing animals (dairy cattle, rapidly growing lambs) may require more.

The ingestion of soil by sheep is a means by which minerals are acquired for significant periods of the year, even though plants may have levels which at the time are deficient for sheep. Soil pH affects availability; increasing pH (more alkaline soil) makes phosphorus and molybdenum more available, but decreases the availability of copper, zinc and manganese. The type of pasture species influences mineral supply to grazing sheep. Compared with grasses, clovers generally have higher concentrations of calcium, phosphorus, potassium, magnesium, iron, copper, zinc, molybdenum and cobalt. Grasses have higher selenium levels. Capeweed is a special case in that it accumulates many minerals.

Time of year can also be important because concentration of most minerals is lower in dry feed (calcium, iron and selenium are exceptions). However this lower concentration does not mean that deficiencies will result. With the associated lower energy and protein of these pastures, sheep growth and metabolism is less, and requirements for minerals are generally reduced. In spring the rapid pasture growth tends to dilute the concentrations of cobalt and selenium, and deficiencies can occur - more so with cobalt, as it is not stored, and required in the rumen for manufacture of Vitamin B12 by bacteria.

Finally, supplementary feeds have an influence. Grains in particular have high phosphorus and low calcium levels. High levels of feeding, including feedlotting, for prolonged periods (more than six weeks) will result in calcium deficiency, as mobilisation from bones may not be rapid enough to maintain adequate blood levels.
Effects and signs
Common features of many mineral deficiencies are reduction in feed intake and reduced efficiency of digestion and utilisation of feed. These effects, combined with impairment of specific metabolic functions, produce the classic signs of ‘illthrift’, so often associated with these mineral deficiencies. Ill-thrift refers to those non-specific signs of reduced growth of body tissues and wool, and direct and indirect effects on reproductive performance.

Diagnosis of mineral deficiencies
It can be appreciated that diagnosis may be difficult, given the many complicating factors involved, and the sometimes obscure and non-specific signs in the sheep. Mineral levels may be defined as deficient or marginal.

Deficient - Depleted animal reserves, loss of production and/or overt disease, response to mineral supplementation expected.

Marginal - Reduced reserves, production response may be observed.

By far the most reliable indicators of mineral deficiencies are those to do with the sheep – clinical signs combined with direct sheep measurements. Levels in blood and other body tissues can be measured and compared with figures calibrated against production and clinical responses. The type of tissue and the number of sheep to sample are also well documented. The best sheep measurement is a response trial, where a mineral or minerals suspected of being deficient in supply is administered to a significant number of sheep, and their response in terms of health and production compared with at least a similar number of unsupplemented sheep.

Soil and feed measurements are not consistently useful. Soil indicators, particularly of trace minerals, have a generally poor correlation with sheep needs. Feed measurements are not necessarily good indicators. This is because sheep are very selective grazers, and frequently have unknown soil intakes in a range of field circumstances. Transient, or even prolonged low levels in feed may not necessarily cause any problems to sheep; they naturally can accommodate feed source fluctuations by accumulating reserves at times of plenty, and mobilising these when feed levels are low. Liver and bone are examples of tissue which retains large reserves of many minerals and vitamins.

There are some discrepancies between published tables of sheep requirements, and tables of accepted sheep tissue levels. Confusion may be engendered by seeming differences between the various sources, all authoritative. Differences may arise for a number of reasons including:

- Inclusion of a safety margin in some tables - the extra amount may not always result in production increases.
- Different sheep breeds may have different requirements.
- Different units may be used.
- Sheep can tolerate periods of low intakes for many trace minerals, as mentioned above.

Minerals and sheep status - current awareness
To present a summary of the major sheep/minerals interactions in Australia, each mineral is referred to, and likely status discussed. For obscure situations and some obscure or small localities, it is strongly recommended that local expertise be consulted. Some minerals in the list already documented earlier can be ‘ticked off’, as being in generally high or adequate levels. Deficiencies are consequently most unlikely. Reasons are advanced; if these do not apply further investigation may be warranted if signs indicate a problem.

Sodium, Chlorine, Iron and Silicon
- these are all rarely an issue in Australia.

Phosphorus, Calcium and Sulphur
- Calcium is adequate in most soils, and is present in superphosphate (20%). Use of this fertilizer has provided plenty of these three minerals for plants and animals. Calcium deficiency may be induced by prolonged grain feeding (more than six weeks), due to a calcium:phosphorus imbalance (grains are high in P and low in Ca). Addition of fine limestone at 1% to the cereal grain will provide
a satisfactory calcium level. Feedlotting is a situation where this applies. The ratio of potassium to (calcium + magnesium) is important; excess potassium can lead to a combined calcium/magnesium deficiency, with recumbency and death. Pregnant and lactating ewes are most susceptible. Theoretically sulphur levels are below what is necessary to provide an ideal nitrogen:sulphur ratio, but responses to sulphur supplementation have not been demonstrated in the field.

**Potassium**
- Animal deficiencies not reported.

**Magnesium**
- Seldom in deficient levels, but excess potassium as described leads to deficiency (see above); grass-dominant pastures are mostly involved, associated with superbloom and unnecessary potassium fertilization.

**Copper, Zinc, Molybdenum and Manganese**
- These have nearly always been applied as fertiliser, and as plant and sheep requirements are similar, deficiencies are now unlikely. For sheep on typical pastures, a history of trace element application up to 20 years ago can be expected to provide adequate levels. Excessive lime application may lead to copper and zinc deficiency if levels are marginal. High molybdenum levels will induce a copper deficiency.

**Boron and Chromium**
- Deficiencies not reported. Generally thought to be in adequate levels in most soils, although little is known about chromium. Boron can be excessive in some soils and cause toxicity in plants.

**Iodine**
- Iodine is necessary for the production of thyroid hormone, essential for newborn lambs to survive the rigors of birth, develop and grow. Goitre (enlarged thyroid gland) is a visible symptom in extreme deficiency. Deficient lambs lack vigor and are very susceptible to cold stress. Deficiencies reported in parts of NSW, Victoria and Tasmania, notably hilly, high rainfall areas.

**Selenium**
- Selenium is necessary in preventing skeletal muscle damage (myopathy). It is not required by plants. The trace mineral most likely to be deficient for sheep in Australia. Areas where deficiency can be expected generally have an annual rainfall greater than 450 mm. In some areas lamb white muscle disease occurs, in the spring; throughout all deficient regions weaners are susceptible to the condition over the summer/autumn, typically on dry senesced pastures and stubbles. Ill-thrift and mortalities can occur. Diagnosis is by blood testing about 6 sheep; in cases of severe disease, typically a treatment is expected to provide about 6 weeks protection against deficiency.

![Note: With fertilised pastures and no other source of selenium, caution is necessary in cases where young sheep graze stubbles or crops not fertilised with selenium that season. If these sheep are on such untreated areas for more than one month, some form of selenium must be supplied. A drench can be given, or to avoid mustering the drench preparation can be appropriately diluted and mixed with any grain supplement being fed, such that each sheep receives the recommended dose.](#)
- Licks and blocks typically contain a range of other minerals and compounds, of doubtful or unknown effect. For selenium alone, they are an expensive and less reliable source.

**Cobalt**
- Vitamin B12 is made by rumen bacteria from dietary cobalt, and clinical signs reflect inadequate levels of this vitamin in tissues. Deficiencies mainly reported about the time of maximum spring pasture growth. At this time of the season cobalt levels are temporarily reduced by dilution, and as animals require a cobalt source at no more than weekly intervals, symptoms may quickly develop. Although cobalt deficiency is not common nationally, it is certainly a problem in defined localities, and local knowledge is the best indicator of likely need. It is typically difficult to diagnose, being often transient in nature, and occurring in some seasons but not others in the same locality. It is more common on calcareous soils, and excessive application of lime can induce a deficiency. Signs are often non-specific, but are characterised by loss of appetite, wasting and anaemia. Profuse watering of the eyes is a characteristic sign in extreme cases.
- Diagnosis is by blood
testing about six sheep from affected mobs. Because the condition is complex, it is wise to test some clinically normal sheep from the mob as well, and the indicators of cobalt deficiency compared.

Cobalt deficiency can be remedied in a numbers of ways:

1. Weekly drench of cobalt sulphate.
2. Cobalt pellet - expected to last for three to four years.
3. Injection of Vitamin B12 - two months duration of effect.
4. Cobalt sulphate top dressed onto pasture - 0.25 kg/ha has been reported to be adequate for at least one year, and possibly two. Very little definitive information about pasture application.

Vitamin E
This is not a mineral, but is mentioned here because deficiencies are increasingly reported throughout the same areas as are deficient in selenium, as well as lower rainfall areas. Deficiency mimics to a large degree the signs of selenium deficiency, and the two may need to be supplied at the same time. To a very minor degree vitamin E and selenium can replace the other, but severe disease caused by a deficiency of one often occurs at a stage of high levels of the other. Diagnosis is by blood testing about six sheep from affected mobs. The most economical treatment or prevention is by oral administration of a vitamin E preparation. Cost is reasonable, and a dose of 2000 units can be expected to last for two months, if necessary.

5.10 Caseous lymphadenitis (cheesy gland)
Caseous lymphadenitis (CLA) is a highly prevalent disease in sheep and goats not just in Australia but throughout the world.

Cause
The causative agent is the bacteria Corynebacterium pseudotuberculosis. It is generally a chronic disease, associated with the formation of purulent or caseous abscesses in lymph glands, lungs and other organs. There is an acute phase, associated with initial infection, and during this production loss is a consequence. It has been shown that up to 0.2 kg of clean wool growth is foregone in infected sheep (Paton et al, 1988). CLA spreads mostly following shearing (95% in one trial) – a time of close contact between sheep and skin damage from shearing cuts. Also, most of this spread is in young (1 and 2 year old) sheep. This is as a result of shearing cuts providing a portal of entry for the organism, origination from externally ruptured lesions (direct contact) and possibly by aerosol transmission from lesions rupturing in lungs. The organism can even gain entry to the body through intact, wetted skin.

Signs
Sheep may be febrile at initial infection, but this is seldom observed. In cases where bronchial lymph nodes are infected and discharging to the bronchi, sheep may be noticed coughing. Enlarged lymph glands, particularly the pre-scapular glands, can be seen where these organs are infected. Ruptured abscesses are reasonably characteristic. f recently formed the pus is thick and liquid. Over time this solidifies to resemble an onion with concentric layers. CLA represents a significant form of loss in abattoirs, both from carcase rejection, trimming losses and the associated labour costs. Detection at slaughter in live export sheep is damaging to Australia’s reputation as a source of high quality sheep.

Prevention and control
CLA vaccine is readily available, in combination with the common clostridial vaccines. It does not cure sheep already infected, but prevents new infection. Two doses of the vaccine are given to lambs, at least 4 weeks apart, together with an annual booster not long before shearing, the known time of maximum disease transmission. Hygiene is naturally of worth to minimize transmission of the bacteria, at times where the skin is broached – shearing, crutching, marking and mulesing. If sheep are to be dipped for lice control after shearing, it is wise to dip the youngest first. The bacteria survive sufficiently in dipping fluid to spread infection.
5.11 Scabby mouth

Scabby mouth (contagious ecthyma) is a viral disease of sheep and goats that is transmissible to humans. It has been reported in sheep-raising countries all over the world. The virus is an Orf virus. Orf is the name of the disease in humans. Morbidity (the percentage of animals affected) may reach 100% in a mob, although mortality (deaths) is low in uncomplicated cases. A Western Australian survey reported that on 106 farms examined the proportion with evidence of scabby mouth in weaner sheep was 24%, and on those farms with the disease the overall prevalence rate was 6%. The disease is generally considered unimportant on farm in Australia, but assumes some significance on account of:

- Spread in feedlot conditions – sheep for export and slaughter (therefore more important in meat enterprises, where feedlotting more likely).
- Effect on sheep – loss of production, predisposition to flystrike in suitable conditions.
- Unsightly in sale sheep.
- Potential human transmission (zoonosis).
- Resembling exotic diseases – foot and mouth disease, blue tongue, sheep pox.

The virus is probably acquired mostly from carrier sheep; small scabby lesions are not easily perceived, but not uncommon on sheep. The virus is also quite persistent in the environment. The virus enters the skin in association with abrasions. Around the mouth, such abrasions may be caused by coarse, dry feedstuffs (grain, stubbles, grass seeds, burrs, thistles). The other common area involved is the lower legs, just above the coronet of the foot, and between the foot claws. Grass seeds are implicated here. (It can be initially confused with benign footrot).

**Signs**

Initial infection, commonly around the lips and nose, is associated with the production of clear fluid-filled vesicles. These become opaque pustules, rupture, and are replaced by scabs. These may drop off, but often are enlarged as the surrounding skin edges proliferate. The scabs usually drop off after about 3 weeks, leaving normal skin. Around the edges of the scabs, and underneath if dislodged, is moist and attractive to flies. The region around the lower legs is at risk in this regard; more so are regions of the sheep’s flank against which the moist foot rests when the sheep lies down. Thus an increased incidence of flystrike is a consequence in fly seasons. The scabby lesions may also be found in other regions of the body: udder and teats, ears and occasionally around the anus, vulva, prepuce and scrotum.

**Prevention and control**

There is no treatment. Sheep in an outbreak should be left undisturbed if possible, with the exception of where there is flystrike risk. In such situations walking the sheep through a footbath of flystrike jetting fluid is a wise precaution, or general jetting/dipping. A scabby mouth vaccine is available (Scabiguard). This is a live vaccine, and applied to the abraded skin by a safe scratching device. Vaccination on the brisket is recommended for lambs destined for slaughter, in case of infected scratch sites being responsible for skin or carcass impairment. This is convenient at lamb marking. Otherwise the inside of the thigh is a satisfactory site. For sheep entering a feedlot and scabby mouth is considered a risk, vaccination at least 2 weeks prior is necessary. Pustules at the vaccination site after 3 to 5 days indicate success. Immunity lasts at least 6 months, at most likely a year or more.

5.12 Clostridial diseases

Most of the clostridial diseases are of great importance to the sheep and cattle industries. In sheep meat producing enterprises, the most important is enterotoxaemia caused by Clostridium perfringens type D. The other clostridial diseases are mentioned as a context, but are far less common due to vaccination and improved management. The clostridia characteristically produce powerful toxins, each exerting its effect in a different way depending on the nature of the toxin and/or the site of infection.
a) Entertoxaemia (pulpy kidney)
Caused by the toxin of *Cl. perfringens* type D. *Cl. perfringens* type D is a normal inhabitant of the gastrointestinal tract of healthy sheep. Clinical disease is most commonly associated with rapid changes in diet, especially an increasing plane of nutrition with large amounts of rapidly fermentable carbohydrate. This might be grain, lush pastures and young cereal crops. Pastures may change in characteristics quite quickly even whilst being continuously grazed, giving a basis for otherwise inexplicable outbreaks of the disease. Clinical signs of entertoxaemia may include scouring, depression, anorexia, bloat, convulsions, paralysis or specific nervous signs. In young sheep the course of the disease is very short, often less than 2 hours, so clinical signs are not often seen. Death is to be expected. Adult sheep survive longer, up to 24 hours.

b) Tetanus
Caused by a toxin produced by *Cl. tetani*. This toxin interferes with the transmission of impulses between nerve fibres, producing muscle spasms, paralysis and death. All animal species, including man, can be infected. The spores of *Cl. tetani*, an inactive form, are present in the soil. These spores infect sheep by contaminating a skin wound, typically a shearing, marking or mulesing wound, dog bite or grass seed penetration wound. In healthy tissue they are inactive, but in dead or damaged tissue the spores germinate and the bacteria grow rapidly with production of the potent toxin. The clinical signs noticed include spasm of body muscles, with restricted jaw movement, pricked ears and dilated nostrils. As the condition progresses sheep become recumbent and are prone to convulsions, especially triggered by sudden noises. Death follows in the majority of cases, and treatment is rarely possible.

c) Malignant oedema and gas gangrene.
The toxins produced in these diseases are from *Cl. oedematiens* (novyi), *Cl. chauvoei* and *Cl. septicum*, amongst others. The disease is primarily due to wound contamination, typically where deep tissue damage has occurred. After infection and multiplication of the bacteria, intense swelling and skin discoloration develop. Depending on the bacteria present, gas may be produced in the tissues, further causing damage. Blood-stained fluid leaks from any wounds. The disease is so acute and severe that treatment is rarely practicable or successful.

d) Swelled head.
The bacteria involved in this local infection, typically of rams, is *Cl. oedematiens* Type A. It is presumed that the infective spores gain entry through wounds caused by fighting, the tissue damage providing the suitable environment for the bacteria to proliferate. Swelling starts about the face and involves the eyelids, nose, ears and lower jaw. It may spread along the neck. As swelling progresses there may be exudation of fluid through the skin.

Blackleg, Caused by *Cl. chauvoei* (feseri), Black disease, caused by *Cl. oedematiens*, and Botulism (*Cl. botulinum*), are further diseases having clostridia as the causative agents.

Prevention
Fortunately effective and cheap vaccines exist for all the Clostridial diseases. An effective vaccination program involves giving 2 doses at least 4 weeks apart, followed by annual booster doses. As a special note for lamb producers, vaccination should be administered near the edge of the pelt, for example behind the ear, so that any blemish can be trimmed if necessary.

5.13 Sheep measles (ovine cysticercosis)
This condition is perhaps of minor concern to the majority of Australian sheep keepers. Yet it continues to rear its presence to those consigning sheep for slaughter, and for some provides a surprising and unpleasant reduction in income from consignments of sheep to abattoirs. Affected carcases are condemned from overseas markets, or may be totally rejected and condemned. Occasionally undetected, it is responsible for concern in overseas markets, where Australian sheep meat has built a reputation for unsullied product.
Cause
The responsible agent is a tapeworm, *Taenia ovis*, living in the intestines of dogs; reaching lengths of up to 2 metres it sheds eggs in the droppings, which not surprisingly are picked up by the intermediate host sheep (and goats) whilst grazing. The eggs hatch into larvae, which migrate through gut wall, and via the liver settle in any striated muscle. Whilst heart, diaphragm and cheek muscles are favored sites, all muscle is involved. In these muscles the larvae grow to clear fluid-filled, oval-shaped cysts up to 10mm long containing a single protoscolex. If eaten by dogs, these cysts are activated to invaginate and the protoscolex attach to the dog intestinal wall; the tapeworm grows and the life cycle of the dog tapeworm is completed. In the live sheep, in time the cysts calcify and remain as a blemish in meat destined for human consumption. The parasite provides no disease risk for humans, but is clearly unacceptable in sheep meat product. There is no apparent health effect on sheep (or dogs).

Prevention and control
The key is breaking the sheep-dog life cycle. This is not difficult or expensive, but does require planning and action.

Control dog movements
Prevent dogs from eating raw sheep or goat meat (thorough freezing or cooking destroys the cysts)
Treat dogs regularly with an effective worming product. To make this effective it is important to maintain dog worming at prescribed intervals short enough to guarantee mature egg-laying tapeworms are never present. *With the common products this is every 6 weeks.*

5.14 Grass seed infestation
That all sheep meat production in Australia benefits at some stage from the grazing of pastures is an accepted fact. However with many annual grass pasture species there exists a two-edged sword: beneficial nutritional attributes are tempered by seeds which attach to and penetrate sheep skin, causing irritation and loss of production in the live sheep. As few as 25 seeds in a lamb can reduce post weaning liveweight gain by up to 50%. The pain and irritation caused by the penetrating seeds inhibit sheep from moving and grazing. Damage to eyes, ears, feet and mouths is an associated consequence of particular severity. Wounds can become infected, increasing the severity of symptoms and making the sheep susceptible to flystrike. Increased vegetable matter in wool is another cause of loss.

It is at slaughter and processing of sheep that grass seed penetration can have severe economic effects. These include:

- Reduced skin prices due to seed contamination and damage
- Loss of carcase weight due to trimming
- Poor overall presentation of carcases due to trimming
- Products downgraded according to the extent of damage and trim required
- Cost of additional labour required
- Reduced throughput of carcases, slowing the whole chain

Cause
The main culprits are the mature seeds of some annual grasses. The actual species may vary between regions and soils types, but some common ones are:

- Spear grass
- Brome grass
- Silver grass
- Barley grass

In the eastern states *Chilean needle grass* is also a problem. In addition the broad leaf *Erodium (wild geranium)* is common in many regions across Australia.
Control and prevention

On each farm and in each region, the causative weed species will differ. The first part of a control strategy is to be well aware of the problem. The overall aim of any strategy is to maximize profit from the enterprise; grass seed control is done within this context. In the extreme case of inability to sufficiently control seed economically for a meat production enterprise, other grazing enterprises might be warranted. However this should seldom be the case.

Strategies include:

- Early weaning of lambs to safe pastures or special crops
- Changing lambing time
- Shearing lambs
- Fodder crops (eg oats, vetches,) – high quality, no seed problem.
- Feedlots
- Pasture management (see below)

Pasture management to reduce problem seeds might include:

- **Spray topping** – spring chemical treatment to inhibit grass seed set. Not always fully effective, but does control selected grass species and result in more digestible plants residues (until summer rain!)
- **Spray grazing** – early winter use of selective herbicide to substantially reduce broad leaf weeds. A sub lethal dose is used, which has the effect of making the target plants more palatable for a short time. In conjunction with high stocking rates, quite effective.
- **Winter cleaning** – grass selective herbicides used.
- **Fodder conservation** – cutting pastures for hay or silage will dramatically reduce grass seed numbers, as well as the added benefit of the conserved fodder. Regrowth can be grazed heavily, or herbicide used to control further seed production.

**5.15 Ovine Johne’s disease (OJD)**

This is a chronic, wasting, incurable intestinal disease that affects sheep, and occasionally goats and deer. OJD is caused by the bacterium *Mycobacterium paratuberculosis*.

First diagnosed in the eastern states in 1980, it has been the subject of much publicity, centred around a controversial and largely ineffective eradication campaign. This is no longer operative, although control of spread of the disease is no less important. It has been diagnosed in all states, although of greatest apparent prevalence in Victoria and parts of NSW. It is of special concern to those with meat specialist enterprises, as many purchase ewes regularly. This gives rise to the possibility of introducing the disease. A summary of the disease and its control is presented in this section. Students are referred to the readings provided for detail of varying degree.

See Web readings


The most likely cause of infection in a flock is through the introduction of sheep – purchased, agisted, or stray. Infected sheep shed the bacteria in their faeces for years before the disease becomes apparent. The disease can also spread through infected material in surface waters, including paddock runoff after rains, and through contaminated material on vehicles and equipment. The number of infected sheep on a property increases after the introduction of the bacterium. Infected sheep shed the bacteria, which in cool moist conditions can survive for more than a year. Other sheep become infected by ingesting contaminated feed. Sheep are most susceptible to the infection when they are young.

**Signs**

OJD is characterized by a chronic infection of the intestines, which causes mucosal thickening and reduced absorption of food. Affected sheep show severe wasting, but continue to eat and drink normally until they are too weak to feed; invariably they die. The average time from onset of clinical illness to death is 6 to 12 weeks. Scouring is not a common clinical feature. The average
annual incidence of disease cases in OJD-infected flocks is estimated to be about 2.5%, although this varies greatly between regions. The disease can be more severe on occasions, with odd reports of 10 to 15% mortalities per year. In affected flocks, sheep generally begin showing signs after 2 years of age, but often not until 4 or 5 years of age. The classic sign of the disease in a flock is a distinct “tail” comprising sheep in poor condition which all eventually die, when others in the mob may be in good condition. Although generally only seen in older sheep, sheep as young as 11 months have been affected. Factors such as high stocking rates and heavy contamination of the farm environment may shorten the incubation period.

**Diagnosis**

There exists a range of laboratory tests for OJD. However some of these tests have low sensitivity, failing to identify all infected animals and some running the risk of producing false positives. **Culture of the causative bacteria from sheep faeces is considered a definitive test for OJD.** A Market Assurance Program (MAP) has been developed to give some confidence to diagnosis, and aid in, for example, sheep trading decisions. (Australian Animal Health Council 2002). At autopsy, identification of the characteristic pathological changes by histology and identification of the organisms in tissue is a definitive test (gross changes are not always apparent).

**Control**

Control of the disease on an infected property is readily achieved, using a range of management strategies with the overall aim of minimizing pasture infectivity, and exposure of young sheep. A killed vaccine is available, which has been shown to be very effective in minimizing effects of the disease. A single dose is adequate. In summary every attempt should be made to minimize the likelihood of introducing OJD to a property; once introduced it is very difficult and prohibitively expensive to eradicate. Control is possible, and advice should be sought as to the components of a management program.

**5.16 Footrot**

Footrot is a disease of the feet of sheep associated with infection a complex mixture of bacteria. Of these the essential agent is the bacteria *Dichelobacter nodosus*. The disease involves infection of the interdigital skin, progressing in the virulent form to underrunning of the foot horn structures.

At present three clinically distinct forms are recognized:

**Virulent footrot** – rapid development of severe lesions under favorable conditions. Persistent and chronic, with extensive necrotic underrunning of the sole and wall of the hoof, and associated with an unpleasant-smelling grey exudate. Pus is not a feature of footrot.

**Intermediate footrot** – may be similar to virulent footrot in individual sheep. Many sheep self-cure, and the severity is not as great. Underrunning seldom involves the wall of the hoof.

**Benign footrot** – tends to involve only the interdigital area, and underrunning is rare. Self-cure usual with dry conditions.

**Signs**

To accurately diagnose these conditions, both clinical and laboratory evaluation is required. There are some differences of opinion and process between the states, and local information should be sought. The first sign of footrot is red, moist skin between the claws (a condition called **foot scald** looks similar at this stage). With further development the organism invades the skin-horn junction and progressively causes separation of the horn from the underlying tissues. Eventually the entire sole is underrun. More than one foot is usually involved. Lameness at this stage is severe; sheep may kneel to graze, where both front feet are infected.

**Eradication**

Given that the bacteria cannot survive for very long (only a few days) away from the sheep’s foot, eradication is very much possible, and is strongly recommended. The costs of living with the disease are too great. Eradication programs are administered by government departments in each state, and details can be found on the appropriate websites. Thus it is in the interest of producers to maintain biosecurity in sheep introductions, not just for footrot but most of the communicable diseases discussed.
5.17 Lice infestation

The sheep body louse *Bovicola ovis* is an obligate parasite of sheep that can also infect goats for a short period. It feeds on the external layer of the skin, causing irritation and a hypersensitivity reaction. This itchiness is the root problem with this disease. *B. ovis* is present in all states of Australia with 35-70% of properties being affected. It is a major disease of the wool industry. However it can be eradicated from a property, and as such eradication combined with biosecurity is recommended.

**Signs and effects**

Lice infestation causes a reduction in greasy fleece weight ranging from 15-30% depending on the severity of the infestation. The main mechanism is irritation (due to local immune system hypersensitivity responses) causing rubbing and biting of the fleece by the host, leading to loss of fibre. Yield also tends to be reduced by up to 5% (absolute units) while cotting and discolouration results in increased classing of fleeces into cast lines. Fibre length and fibre diameter are unaffected.

**Factors affecting lice infestation**

*Shearing* removes most of the eggs, 30-50% of the adults and exposes the remainder to high skin temperatures. Summer skin temperatures in shorn sheep are often in the range 45-55°C. *Wool cover* affects lice survival through effects on skin temperature. Less cover allows greater penetration of solar radiation and greater variation in skin temperatures. The longer the staple the better for lice. Due to temperature and shearing effects there is a marked *seasonal variation* in numbers. Few lice are found after shearing, numbers remain low over summer, then increase from autumn through winter until the next shearing. Increases in numbers can be interrupted by soaking thunderstorms.

**Lifecycle**

All stages of the lifecycle occur on the sheep. Adult females lay 2 eggs every 3 days. These eggs are attached to wool fibre 6-12 mm from the skin and hatch in 10 days (Figure 15.4) Eggs attached to fibre removed from the host, do not hatch. After hatching, the nymphs pass through 3 stages lasting 7, 5 and 9 days respectively. The pre-oviposition period in the female is 3-4 days. The minimum time required to complete the lifecycle is 34 days. Under good conditions it takes 4-5 months for a light infestation (0.3 lice per sq. cm) to develop into a heavy infestation (30 lice per sq. cm). As lice can only survive for 1-2 days off the host, transmission of the parasite is by direct contact between sheep.

*Figure 5.3 Life cycle of Bovicola ovis. Source: Joshua (2001).*
Eradication and control

_B. ovis_ is relatively easy to eradicate from farms, and this is regularly achieved, but this has never been achieved on a state scale. In theory (and practice) eradication can be achieved by a _single effective treatment_ to all sheep, as lice do not survive well off the host. Control is based solely upon chemical treatment of all animals with _effective chemicals_ for sheep to be completely free of lice after treatments with these chemicals. In terms of effectiveness, off shears treatments (backline, dip or spray) are more effective than short wool treatments (dip or spray), which in turn are more effective than long wool treatments (dip or jet). This reliance on chemical treatments raises issues of chemical residues and resistance. Withholding periods to shearing must be strictly observed. Short wool treatments are not only more effective, but they use less chemical and allow longer for it to break down prior to shearing so are preferable from a residue management point of view. Prevention of re-infestation is a major aspect of control. This requires good boundary fencing and care when purchasing new stock.

Readings

The following readings are provided on CD.


Activities

Multi-Choice Questions

Submit answers via WebCT

Useful Web Links

Available on WebCT

Assignment Questions

Choose ONE question from ONE of the topics as your assignment. Short answer questions appear on WebCT. Submit your answer via WebCT

Summary

Summary Slides are available on CD

Diseases of sheep in any form, apart from posing a welfare issue, affect the ability of the animal to convert costly resources, mostly feed, into saleable product. As well they may downgrade the value of that product, and thereby affect profit.

Disease syndromes affecting sheep in Australia have been identified over the time that sheep have been managed. These have changed in nature, incidence and severity as farming environments and sheep systems have changed.

This module is not a veterinary compendium on sheep diseases; useful references are given for such. Rather, some of the more significant and costly diseases likely to be encountered are discussed, including a number with little effect on the living animal but which reduce profit by downgrading carcases. These are obviously more important to enterprises with a significant component of income from specialist meat production.
Useful Web Links


References


‘Veterinary Pathologist, Animal Health Laboratories, Western Australia.


Glossary of terms

<table>
<thead>
<tr>
<th>Acute disease</th>
<th>Disease with rapid onset, resolution or death in a matter of hours or days</th>
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<tbody>
<tr>
<td>Anaemia</td>
<td>Abnormally low numbers of red blood cells (erythrocytes) or haemoglobin concentration in the blood</td>
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<tr>
<td>Anorexia</td>
<td>Lack of loss of appetite for food. &quot;Inappetence&quot; is used interchangeably</td>
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<tr>
<td>Anthelmintic</td>
<td>Chemical administered to host animals to kill or suppress “worms” (ie roundworms, tapeworms, flukes)</td>
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<tr>
<td>Antibiotic</td>
<td>Chemical, based on natural compounds, administered to host animals to kill or suppress bacteria</td>
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<tr>
<td>Chronic disease</td>
<td>Disease that persists for some time, generally more than a week, but up to years</td>
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<tr>
<td>Clinical disease</td>
<td>Disease that can be readily ascertained from abnormalities detected by the five senses</td>
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<tr>
<td>Clinical signs</td>
<td>The observable signs of disease. Roughly equivalent to the term “symptoms” used in human medicine</td>
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<tr>
<td>Term</td>
<td>Definition</td>
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<td>-------------------------------</td>
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<tr>
<td>Diagnosis</td>
<td>The process that enables the specific disease or disease syndrome affecting an animal or group of animals to be determined. An important part of this process is the differential diagnosis, in which the signs observed are compared with those of a range of possible disease syndromes followed by a process of elimination.</td>
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<tr>
<td>DrenchPlan</td>
<td>The recommended sheep worm management program for central and southern NSW.</td>
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<tr>
<td>Epidemiology</td>
<td>The study of the frequency, distribution and determinants of disease in animal populations. Clinical medicine is the study and practice of disease control in individual animals or small groups of animals, while epidemiology is the study and practice of disease control in populations.</td>
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<tr>
<td>Export Slaughter Interval (ESI)</td>
<td>The minimum suggested time interval between the last treatment with a product and slaughter for export.</td>
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<tr>
<td>Fever</td>
<td>Abnormally high body temperature. Often due to generalised infection or toxemia.</td>
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<tr>
<td>Helminth</td>
<td>Parasitic worm. Roundworm, tapeworm or fluke.</td>
</tr>
<tr>
<td>Helminthiasis</td>
<td>Disease caused by helminths. Also called helmintosis.</td>
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<tr>
<td>Hypobiosis</td>
<td>Arrested development. Lifecycle pauses at some stage, usually in the host, until conditions become favourable for the lifecycle to resume.</td>
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<tr>
<td>Hypoproteinaemia</td>
<td>Deficiency of protein in the blood. Commonly associated with gastrointestinal helminthiasis.</td>
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<tr>
<td>Immunity</td>
<td>Resistance to disease due to the specific action of the immune system. Different from the broader term resistance to disease which may involve both immune and non-immune defences.</td>
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<tr>
<td>Inflammation</td>
<td>Protective response of the immune system to tissue injury or invasion. The cardinal signs of inflammation are redness, heat, swelling and pain.</td>
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<tr>
<td>Meat Withholding Period (Meat WHP)</td>
<td>The minimum period of time which must elapse between the last treatment of an animal with a product and slaughter for human consumption in Australia.</td>
</tr>
<tr>
<td>Pathogenesis</td>
<td>The mechanisms by which disease is induced in the host animal.</td>
</tr>
<tr>
<td>Pathogenicity</td>
<td>The severity of the disease caused by the particular organism. Also known as virulence.</td>
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<tr>
<td>Resistance</td>
<td>Ability of an animal to resist colonisation by a disease organism (eg. worms).</td>
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<tr>
<td>Sub-clinical disease</td>
<td>Disease processes that are not readily detectable with the five senses at a clinical examination. Often the most economically-important form of disease.</td>
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<tr>
<td>Systemic disease</td>
<td>Disease which is generalised throughout the body. Generally spread via the bloodstream.</td>
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<tr>
<td>Vaccination</td>
<td>Controlled exposure of animals to antigen or antibody to prevent disease. Based upon activating or enlisting the immune system to combat disease.</td>
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<tr>
<td>Voluntary feed intake</td>
<td>The feed intake of animals with unrestricted access to feed.</td>
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<tr>
<td>Wool Withholding Period (Wool WHP)</td>
<td>The amount of time required to elapse between treatments with insecticides and shearing under Australian law.</td>
</tr>
<tr>
<td>Worm egg count (WEC)</td>
<td>The number of worm eggs per unit of host faeces usually. Normally in eggs per gram of faeces (epg). Widely known as faecal egg count (FEC), but worm egg count is the more correct term.</td>
</tr>
<tr>
<td>WormKill</td>
<td>The recommended sheep worm management program for the north-western, summer rainfall region of NSW.</td>
</tr>
</tbody>
</table>