Chapter 12

Goats

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Introduction

Veterinary practitioners and pathologists in Australia and New Zealand are very familiar with the common diseases of sheep and cattle, but many do not feel comfortable when dealing with disease problems in goats. This is not surprising considering the relatively small size of the goat industries in both countries and the infrequency with which we are invited to provide advice on goat health and production. Because of a lack of reliable sources of information on goat diseases there is a tendency to extrapolate from our knowledge of sheep or cattle, but this is often inappropriate. Not only do goats have different susceptibility to many of the common diseases of sheep and cattle, they are susceptible to certain diseases that are seldom if ever diagnosed in these species.

The pathology of goat diseases cannot be covered in a single presentation. Instead, I will present an overview of selected clinical syndromes, indicating similarities and differences between goats and other ruminants. Much of the information presented here is based on my experience over the last 20 years as both a goat farmer and a pathologist with a special interest in goat diseases. My farming experience has largely been with fibre and meat goats and that will be my emphasis in this paper. For convenience, the disease syndromes will be considered under different age groups.

Diseases of kids from birth to weaning

Neonatal mortality

Dystocia is less common in goats than in either sheep or cattle, although breed differences exist in all species. Impatient owners sometimes cause problems by trying to assist a doe without ensuring that the head is in the birth canal before pulling on the forelimbs. The diagnosis is based on gross lesions (discussed elsewhere in this seminar).

Newborn kids, especially Angoras, are much more susceptible to **exposure** than lambs because their birth coat does not possess the same insulating properties and they have less subcutaneous fat. This may be a major cause of kid loss on some properties. The danger period is the first hour or so after birth. If the weather is cold and wet or windy, kids born in an exposed site may die of exposure before they have a chance to stand and feed. Such kids are often mistaken for abortions or stillbirths. Small kids (<2.0kg) have particularly low viability unless they

are born in a sheltered environment. Gross lesions are unremarkable although there will be partial aeration of the lungs, represented by few to many pale pink lung lobules contrasting with dark purple areas of atelectasis. Death usually occurs too rapidly for there to be any depletion of fat reserves. There may be some evidence of suckling, depending how soon after birth the kid became chilled.

Mismothering is relatively common, especially with multiple births. Some kids may stand and wander away from the birth site before they have bonded adequately with the doe. The risk is increased if kidding occurs in hilly paddocks. The gross changes in mismothered kids are similar to those in lambs. Footpads are worn and there is depletion and serous atrophy of body fat reserves. Adipose tissue in coronary grooves is usually reddened and gelatinous. The same changes, but in a slightly older age group, may be seen in kids that die of **starvation** due to inadequate milk production by the dam.

Iodine deficiency goitre has been responsible for major losses of newborn kids on properties where there have been no problems in newborn lambs or calves, suggesting that goats have a higher requirement for this trace mineral. Affected kids are either born dead or die soon after birth. Abortion does not appear to be a feature. The thyroid glands are often markedly enlarged and some deaths may be due to tracheal compression or to dystocia. In severe cases the hair coat is poorly developed, the tongue may be protruding and there may be an overshot lower jaw, presumably due to the pressure of the enlarged glands. Many kids with moderately severe goitre are able to survive with treatment. Grossly the thyroid glands are bilaterally enlarged and dark red. Subclinical iodine deficiency may contribute to the increased neonatal losses through exposure on some properties. Supplementation of pregnant does with iodine should be routine.

Congenital white muscle disease due to **intra-uterine selenium deficiency** may be underestimated as a cause of neonatal mortality on some properties, as the lesions are more subtle than those of WMD in older kids (see below).

Enteric diseases

Rotavirus and ***E coli*** are common causes of diarrhoea during the first week of life, especially in kids deprived of colostrum. Lesions and diagnosis are the same as for calves and lambs.

Cryptosporidium sp is common during the first 3-4 weeks of life, especially in artificially-reared and late kids. Infection is often concurrent with rotavirus and/or *E coli.* Gross lesions are not specific but at necropsy, affected kids usually have distended, gas filled loops of large intestine with a small amount of yellow/white content. The diagnosis is best confirmed by microscopic examination of small intestine from freshly dead kids. Cryptosporidia are not species specific and may infect other ruminants in addition to human beings.

Coccidiosis is common and important as a cause of diarrhoea, and even death of kids, usually from around 1-4 months of age, but sometimes in older kids. Several species of *Eimeria* infect goats, but they are species specific and will not cause problems in lambs or calves. Late kids are usually the most severely affected as the level of environmental contamination is massively amplified by earlier kids. A diagnosis based on demonstration of oocysts in faeces is not reliable. Postmortem examination provides the most useful information. Some species of coccidia in goats (and lambs) produce grossly visible, 1-2 mm diameter, pale nodules in the mucosa. These consist of hyperplastic epithelial cells packed with coccidial stages. They are often found incidentally in small numbers, but if present in large numbers they are likely to be significant, especially if the intestinal content is watery. Mucosal scrapings and/or histology provide more precise information as there may be many additional coccidial stages in the mucosa between nodules. Always consider the possibility of concurrent nematode parasitism and/or yersiniosis in a kid with lesions of coccidiosis.

There are occasional reports of **giardiasis** in dairy goat kids, but its significance is uncertain.

Nematode parasitism can affect kids during this period but is usually more common after weaning and will be discussed later.

Tapeworm infestations (*Monezia expansa*) are common in kids, but do not appear to be clinically significant.

Sudden death

White muscle disease (WMD) caused by deficiency of selenium (or sometimes vitamin E) has caused major losses of kids on some properties in New Zealand. There is convincing evidence that goats are more susceptible to selenium deficiency than either sheep or cattle, at least with regard to the occurrence of WMD. Although it may be congenital, it more often affects well-grown 4-6 week old kids. Affected kids are often just found dead. Lesions are consistently present in the myocardium (especially right ventricle), but may also involve skeletal muscles, including the tongue.

Gross lesions include clear fluid (plus fibrin clots) in body cavities, congested, oedematous lungs, enlarged congested liver (if subacute) and chalky white/yellow lesions in the myocardium. Lesions are usually less prominent in kids with congenital WMD and such cases may be misdiagnosed as stillbirths or exposure.

Rapid diagnosis and treatment with Se and/or vitamin E will reduce further losses but some kids will eventually die of congestive heart failure due to residual cardiac lesions. The disease can be easily prevented by annual pasture Se applications or supplementation of pregnant does, although there have been occasional outbreaks in New Zealand where vitamin E rather than selenium deficiency appeared to be involved.

Clostridial diseases should always be considered in cases of sudden death in young ruminants but their prevalence in goats is not clear. Some authors have claimed that **enterotoxaemia** caused by the epsilon toxin of *Clostridium perfringens* type D is one of the most important diseases of goats, but its occurrence in this species is poorly documented. Some useful experimental work has been conducted over the last 10 years but much of the published information on the natural disease is anecdotal. In my experience in New Zealand, enterotoxaemia is an uncommon disease of goats grazing pasture, although it clearly does occur. Even in zero-grazing dairy goat operations in New Zealand, the disease appears to be rare.

The diagnosis of enterotoxaemia is never easy and the results of experimental studies suggest that the gross lesions in goats may differ from those in lambs. Necrotising colitis with pseudomembrane formation was a feature of the disease in experimentally infected goat kids, but this is not recognised as a lesion of enterotoxaemia in lambs. I have not seen this lesion in the few natural cases of enterotoxaemia that I have seen in goats in New Zealand. The demonstration of glycosuria adds support to a diagnosis of enterotoxaemia but is not specific and may occur in association with other diseases. For example, sheep with listeriosis or polioencephalomalacia will sometimes have glycosuria. The presence of epsilon toxin in ileal contents is widely used to confirm enterotoxaemia but the limitations of such tests should be recognised. Demonstration of perivascular proteinaceous oedema on histological examination of the brain probably remains the most definitive lesion of enterotoxaemia in both sheep and goats, even though there are suggestions that this is less common in kids than in lambs.

In early reports of enterotoxaemia in goats there is mention of a "chronic form" characterised by intermittent diarrhoea and wasting. This seems to have been based on anecdotal observations and there is no convincing evidence to support the existence of such a syndrome. It is clear that more information on the disease in goats is required before we can make useful recommendations on prevention.

Tetanus occurs sporadically in goats, usually associated with injuries caused by penetrating wounds, dehorning, castration or ear tagging. The disease is best diagnosed clinically as there are no useful gross or microscopic lesions, other than a potential site at which proliferation of *Cl. tetani* would have occurred.

A **braxy-like syndrome** characterised by necrotising abomasitis in association with *Cl. septicum* has been recognised in goats in Australia and New Zealand. There is diffuse or patchy reddening of the abomasum, necrosis of the mucosa and marked submucosal oedema.

Abomasal bloat is a sporadic problem of artificially reared kids, usually from 4-12 weeks of age and is most common in greedy kids that engorge on milk. Fermentation of milk in the abomasum results in excessive gas formation and bloat. Affected kids are usually found dead with a

markedly distended abdomen. The abomasum may rupture terminally or soon after death, in which case milky fluid and lumps of clotted milk will be present throughout the peritoneal cavity.

Volvulus is another common but sporadic cause of acute death in kids (and older goats). Most cases occur in artificially reared kids. As in abomasal bloat, affected kids have a distended abdomen and may just be found dead. At necropsy, the involved segment of bowel is dark red and distended. Almost any part of the GI tract from the abomasum to the colon may be involved. The mesenteric root should be palpated to identify the twist before removing the GI tract.

Herpesviral enteritis has been associated with outbreaks of abdominal pain, weakness and death in young kids around 1- 2 weeks of age. In one outbreak in a dairy goat flock in New Zealand, it was responsible for the deaths of approximately 80 of 120 kids. At necropsy, affected kids have ulcerative lesions in the gastrointestinal tract, particularly caecum and colon. Eosinophilic, intranuclear inclusion bodies can be detected histologically in intestinal epithelial cells at sites of ulceration. The presence of herpes virus can be confirmed by electron microscopy and/or virus isolation.

Nervous diseases

The encephalitic form of **caprine arthritis/encephalitis** (CAE) occurs in this age group and may be confused clinically with "**swayback**" caused by copper deficiency. The disease is more likely to occur in dairy goats than fibre or meat goats due to pooling of colostrum and increased risk of spreading infection amongst dairy goats. Clinically, affected kids show hind limb ataxia progressing over a few days to recumbency and more severe nervous signs, depending on the regions of brain and spinal cord involved. Gross lesions may be visible in the spinal cord or brain (particularly the cerebellar white matter) as swollen pink or brownish areas of malacia. The lesions are usually unilateral and may be quite localised. Extensive sectioning of the brain and spinal cord is therefore required in some cases before lesions are detected. The diagnosis can be confirmed by histology of the CNS and/or viral culture.

Vertebral osteomyelitis ("spinal abscess") leading to vertebral collapse and damage to the spinal cord is a relatively common cause of sudden onset hind limb paralysis in young kids, usually a few months of age. Bacteria such as *E coli, Streptococcus sp, Fusibacterium necrophorum,* etc may localise in the bone during bacteraemia, usually in the neonatal period. The infection gradually destroys the bone until a pathological fracture occurs. A similar clinical syndrome may follow trauma to the spinal column.

Skin diseases

Dermatophilosis is a common skin disease of fibre goats in New Zealand, presumably due to the combination of wet weather and the lack

of oil in the fleece of goats. Because of the drier conditions experienced in most areas of Australia the disease is less likely to be a significant problem in this country. Lesions in goats occur most commonly on the nose, lips and ears. They appear as small, elevated scabs surrounding a group of hairs, which often stand erect. In this early stage the lesions can best be detected by palpation, but as they progress large crusts develop and hair-loss occurs. Removal of the crusts reveals a moist, reddened epidermis. A syndrome resembling strawberry footrot of sheep has also been recognised in goats with dermatophilosis, and is manifest as a severe crusty dermatitis of distal limbs. Angora goats sometimes develop extensive lesions of dermatophilosis over large areas of their body during long periods of wet weather. This is most likely to occur in goats that spend much of their time in recumbency on wet ground due to footrot.

The major differential diagnosis for dermatophilosis is contagious ecthyma but the two diseases can be readily differentiated histologically. Impression smears prepared from scabs may also assist by allowing identification of the branching filamentous organisms typical of *Dermatophilus congolensis*.

Contagious ecthyma (scabby mouth, orf) is a common pox viral disease of lambs throughout the world and may also affect goats, particularly young kids. The lesions start as small papules and progress to vesicles, pustules and thick crusty scabs elevated 2-4 mm above the skin surface. As in lambs, the lesions frequently occur at the commissures of the mouth but proliferative oral lesions, on the tongue and gums, are more common in kids than in lambs. The gross lesions may be confused with those of dermatophilosis but are generally more proliferative and have a different distribution. Confirmation of the diagnosis by histological examination or electron microscopy is advisable before recommending prevention by vaccination.

Diseases of weaned kids

Gastrointestinal parasitism

Unlike sheep and cattle, adult goats do not develop a strong immunity to gastrointestinal parasites. As a result, many goat farmers have relied heavily on anthelmintics for parasite control and resistance to the three major drench families has become widespread. The problem is of particular significance on small properties where goats are the predominant or only species. Such properties may become massively contaminated with infective larvae and goats of all ages suffer reduced growth or productivity. The stress of clinical or subclinical parasitism may also predispose to other disease problems, such as yersiniosis or pneumonia. Although gastrointestinal parasitism can occur in goats of any age it is usually of greatest significance in young growing kids in the post-weaning period.

The most important nematodes in goats are usually *Haemonchus contortus, Ostertagia (Teladorsagia) circumcincta*, and *Trichostrongylus* spp. Mixed infestations are common but under some circumstances one or other of these nematodes may predominate. The clinical signs and pathology will vary accordingly. Anaemia is a feature of haemonchosis and may cause acute death in goats of any age. At necropsy, the carcass is pale and large numbers of *Haemonchus contortus* can usually be seen grossly in the abomasum. Pinpoint haemorrhages may be scattered over the abomasal mucosa.

Larvae of *Ostertagia circumcincta* burrow into the abomasal mucosa and induce focal hyperplasia of mucus neck cells. These foci appear grossly as 1-2 mm pale nodules in the abomasal mucosa and are commonly present in small numbers as an incidental finding at necropsy. In severe infestations these nodules may become confluent, creating an irregularly thickened mucosa, likened to Morocco leather, but this lesion is more typical of the disease in cattle caused by *O. ostertagi*.

Trichostrongylus colubriformis can be an important intestinal parasite in goats, causing severe weight loss, diarrhoea and death. Gross lesions are not specific but may include depletion and serous atrophy of body fat reserves, variable quantities of fluid in body cavities, subcutaneous and mesenteric oedema and skeletal muscle atrophy. The serosal surface of the duodenum may be reddened in very heavy, acute infestations with *T. colubriformis*.

The intestine is usually filled with green, foul smelling, fluid and mesenteric lymph nodes are enlarged and moist. When gastrointestinal parasitism is suspected the contents of the entire tract should be collected for a worm count. This is the most precise method of determining the significance of a worm burden, unless the animal was drenched with an anthelmintic within a few days of death. If the majority of nematodes are of one species the possibility of anthelmintic resistance should be suspected.

Lungworm

Lesions caused by *Muellerius capillaris* infestation are common incidental findings in the lungs of goats at necropsy. The lesions consist of firm, slightly raised, grey nodules primarily in the dorsal diaphragmatic lobes. In some cases there are multiple large nodules (up to 1.5 cm diameter), which may coalesce to involve extensive areas of pulmonary parenchyma.

Muellerius capillaris has been incriminated as a cause of diffuse interstitial pneumonia in goats, resulting in chronic progressive respiratory embarrassment and eventual death. However its role in such lesions is difficult to assess, even when degenerate larvae are found histologically throughout the lung, and the possibility of concurrent *Mycoplasma* or retroviral infections must be considered.

Yersiniosis

Yersiniosis is now recognised as a significant disease of young goats in New Zealand. It occurs most frequently in late autumn or winter and is usually triggered by stress. Inadequate nutrition in combination with winter shearing, cold wet weather and lack of shelter has been responsible for major losses on some properties, particularly in feral goats. The disease may also occur soon after prolonged transport or may be associated with concurrent disease (e.g. coccidiosis or gastrointestinal parasitism).

Affected goats may die acutely after a short period of foul-smelling diarrhoea, or may develop a more prolonged syndrome characterised by diarrhoea, dehydration and weight loss. In live animals, diagnosis depends on culture of the organism from the faeces but positive results should be interpreted with caution as various *Yersinia* species can be cultured from the faeces of clinically normal animals. *Y. enterocolitica* is the most common cause of caprine yersiniosis although some cases are associated with *Y. paratuberculosis*.

Gross lesions include mild reddening of the small intestines, fluid-filled intestinal contents and enlarged mesenteric lymph nodes, but are non-specific. The histological lesions are sufficiently characteristic to allow confirmation of the diagnosis.

Polioencephalomalacia

Thiamine responsive polioencephalomalacia (PEM) is common in goats from a few months of age to early adulthood. Clinical signs include blindness (without ocular lesions), recumbency, opisthotonos, nystagmus and extensor rigidity. Death usually occurs after one to several days. Rapid response to thiamine administration is considered to be a useful diagnostic aid, but such treatment is only likely to be successful if administered early in the disease process.

The gross lesions of PEM are confined to the brain. Cerebral gyri are often flattened due to swelling of the brain and there may be herniation of the medulla and posterior cerebellum into the foramen magnum. The cerebral grey matter, particularly in occipital and parietal regions, is discoloured yellow and on cut surface is swollen softened and sometimes partly separated from the underlying white matter. The cerebral lesions are bilaterally symmetrical and have a segmental distribution with a discrete margin between normal and abnormal grey matter. Similar yellow malacic foci may be present in the caudal colliculi, thalamus and caudate nuclei. Necrotic nervous tissue fluoresces under UV light in a dark room and this can be a useful technique in cases where the gross lesions are equivocal. The diagnosis can usually be confirmed by histological examination of fixed brain.

Listeriosis

The encephalitic form of listeriosis is more common in goats than in either sheep or cattle. It occurs primarily in adults during the winter months, but is discussed here for convenience. The disease is usually sporadic, although outbreaks have followed silage feeding in New Zealand. Clinical signs include depression, increased salivation, facial paralysis, nystagmus, head tilt and circling (in the direction of the tilt) progressing to recumbency

Gross lesions are generally unremarkable although the meninges may be slightly cloudy and small foci of malacia may be present in the brain stem or cerebellar peduncles. Definitive diagnosis requires histological examination of the brain and/or culture of the organism *Listeria monocytogenes*. The lesions are largely confined to the brain stem and spinal cord and consist of non-suppurative meningoencephalitis and multifocal suppurative encephalitis with microabscess formation and variable necrosis. In some cases of caprine listeriosis the lesions are restricted to the spinal cord, resulting in paresis or hind limb paralysis.

"Water belly"

The accumulation of oedema fluid in the subcutis of the intermandibular region ("bottle jaw") and along the ventral thorax and abdomen is a well-recognised consequence of diseases causing either severe hypoalbuminaemia or right-sided heart failure. The first consideration when presented with a young goat with such a lesion is therefore likely to be gastrointestinal parasitism but in Angora goats of South African and/or Texan origin there is a syndrome commonly referred to as "water belly" that appears to be related to stress rather than to hypoalbuminaemia or heart disease.

The syndrome is very common in young Angora goats during the first week or so after shearing, sometimes affecting up to 15% of the mob. In severe cases there is a massive quantity of clear fluid in the subcutis along the ventral body wall and even extending down into the legs. In mild cases the fluid is only obvious on palpation. Affected animals are usually healthy, continue to eat and the oedema fluid may come and go over a period of a few days before it resolves spontaneously. In some animals the oedema persists for a week or more, leading to thickening of the distal limbs with oedema fluid and early fibrosis. The syndrome also occurs sporadically in association with other forms of stress, such as transport, cold weather or concurrent disease.

Angoras with uncomplicated "water belly" have normal or slightly low serum albumin concentrations, but not low enough to cause oedema through reduced colloid osmotic pressure. The aetiology is unknown, but it may be due to excessive release of aldosterone (in addition to cortisol) from the adrenal gland in response to ACTH. Increased aldosterone would cause sodium retention and hypervolaemia. The slightly low serum

albumin would therefore be due to dilution rather than increased loss or reduced formation.

The challenge for the practitioner is to decide whether the ventral oedema is just stress-related and will resolve spontaneously or whether there is some underlying disease process that requires treatment. Clinical history, clinical examination and laboratory tests can all contribute significantly to the decision-making process.

Diseases of adult goats

Diseases causing weight loss

Starvation, possibly combined with subclinical parasitism, is probably more common in grazing goats than in sheep or cattle. The feed requirements of goats are often underestimated, especially during pregnancy, lactation or after shearing, as they are unable to graze as low as sheep and will lose condition if presented with short pasture (<4cm). Goats grazing such pastures are also likely to be ingesting larger numbers of nematode larvae. A poor condition score in fibre goats (especially Angoras) may be masked by a bulky fleece.

Chronic **gastrointestinal parasitism** should always be considered as a contributing factor, if not the sole cause, of weight loss or poor condition in goats. Confirmation in the live animal should be based on clinical history and faecal egg counts. In goats, as in sheep, faecal egg counts usually reflect the magnitude of the worm burden (providing the infestation has reached patency). Gross lesions in animals that die or are sacrificed include depletion of body fat reserves, fluid-filled intestines and enlarged oedematous lymph nodes (+/- mineralised foci). Serosal lymphatics in the small intestine may be prominent due to blockage of afferent lymphatics by parasitic granulomas. There may be mesenteric and subcutaneous oedema in severe cases due to hypoalbuminaemia. Nematodes may be visible grossly in abomasum, but worm counts (on abomasal and small intestinal contents) are recommended for objective assessment of the worm burden.

Johne's disease has been diagnosed in goats (primarily dairy breeds) on properties both in Australia and New Zealand. In North America and Europe it is common in dairy goat flocks and is considered to be of economic importance. The disease is uncommon in fibre and meat goats, presumably because of their more extensive management. In New Zealand, goats have been shown to be susceptible to both the ovine and bovine strains of *Mycobacterium avium* subsp *paratuberculosis.* The clinical signs in affected goats are similar to those in sheep. Gross lesions are also similar to those in sheep, although caseation and mineralisation of mesenteric lymph nodes is a feature of the disease in goats and must be differentiated from tuberculosis (by culture).

Chronic **fascioliasis** presents as weight loss, anaemia and bottle jaw (in severe cases) and therefore may be mistaken for nematode parasitism or Johne's disease. Goats, like sheep, are highly susceptible to reinfection after initial exposure. Clinical pathology typically reveals anaemia, panhypoproteinaemia (due to blood loss) and moderate elevations in serum GGT activity. Demonstration of fluke eggs in faeces (by sedimentation technique) is necessary for confirmation of a diagnosis in the live animal.

Gross lesions at necropsy include tissue pallor (anaemia) and a firm liver with an irregular, scarred capsular surface, sometimes with evidence of old migratory tracts. Intrahepatic bile ducts are prominent on cut surface due to their thickened, fibrous wall and may be present in their lumen. In acute fascioliasis multiple tortuous haemorrhagic tracts are present throughout the liver.

The lentivirus of CAE has been associated with **chronic progressive pneumonia** and weight loss in adult goats, but the disease has not been reproduced experimentally with the virus and its involvement is uncertain. At necropsy, the lungs fail to collapse and are diffusely firm and pale grey/pink. A similar chronic pneumonia in goats may occur in association with severe *Meullerius capillaris* infestations.

Reproductive disorders

Herpes viral vulvovaginitis and **balanoposthitis** is relatively common in goats in New Zealand and probably Australia. In one serological survey in New Zealand, the prevalence rate of reactors in feral goats was 9.8% and in dairy goats 1.2%. Outbreaks of vulvovaginitis typically occur following introduction of an infected buck or teaser to a group of naive does. Lesions around and inside the vulva are initially ulcerative and cause considerable discomfort to the doe. They then become encrusted with seropurulent to haemorrhagic exudate before resolving spontaneously over a period of 2-3 weeks. Affected does usually do not conceive during the active phase of the disease but will conceive at the next cycle. The infection becomes latent and may reappear later in life following a period of stress. The virus causes ulcerative lesions in the mucosa of the penis or prepuce of bucks, but these may not be apparent at the time of examination.

Caprine herpes virus has also been incriminated as a cause of abortion in a flock that had experienced an outbreak of vulvovaginitis, but this is an uncommon manifestation of the infection.

Unlike sheep and cattle, most **abortions** in goats are non-infectious. The stress of shearing, inadequate nutrition or inclement weather (or combinations of these) during late pregnancy commonly causes abortion in fibre or meat goats reared in extensive conditions. The losses can be substantial on some properties but early abortions often go undetected and may just present as low kidding percentages. In sheep, nutritional

deprivation in late pregnancy is more likely to result in pregnancy toxaemia, but this is uncommon in goats.
The most common cause of infectious abortion in goats is **toxoplasmosis**. As in sheep, the manifestations of intrauterine infection with *Toxoplasma gondii* vary from mummification of foetuses to the birth of weak kids that die soon after birth. Characteristic foci of necrosis may be visible grossly on cotyledons and occasionally in the liver or lungs of aborted foetuses, but the diagnosis is best confirmed by histological examination of the brain and cotyledons. Lesions may also be detected histologically in the heart, liver and lungs. *Listeria monocytogenes* is also recognised as a cause of infectious abortion in goats, but *Campylobacter spp* is not. *Salmonella Brandenburg* has recently become a major cause of abortion in sheep in the South Island of New Zealand and is also recognised in cattle, but has yet to be reported as a cause of abortion in goats.

As a rule, foetuses that have aborted as a result of maternal stress are relatively fresh while those that have died of infection *in utero* are in a relatively advanced state of autolysis.

There are few significant reproductive diseases reported in bucks. Epididymitis due to *Brucella ovis* infection has been reproduced in goats experimentally but does not appear to be a significant natural pathogen of goats.

Skin diseases

Squamous cell carcinoma occurs in the perineal region, especially around the vulva, of Angora goats and to a lesser extent in other breeds. Angora goats have pale skin and tend to hold their tails in the air, exposing the perineum to sunlight. The tumour is probably more common in Australia than New Zealand and was relatively common in aged Angora does imported from Australia to New Zealand during the mid 1980's. In dairy goats, particularly Saanens, the udder is a predilection site. By the time veterinary opinion is requested the lesions are usually large and ulcerated. In some cases the ulcerated surface will be almost black due to the presence of dried blood and dirt and could be misdiagnosed as a melanoma on gross inspection. The diagnosis can be easily confirmed by histopathology or cytological examination of scrapings from a cleaned surface. Local invasion of squamous cell carcinoma may be extensive but metastasis is uncommon.

Malignant melanoma is common in Angoras and Saanens in Australia and New Zealand, usually originating on the ears, muzzle or perineum and rapidly metastasising to regional lymph nodes and/or lungs. Most are heavily pigmented and appear black grossly. By the time they are detected they have often metastasised, so the prognosis should always be guarded, even if the primary lesion appears to have been completely removed.

Papillomas (warts) are also relatively common and are found most frequently on the udder of dairy goats. Some cases appear to progress to squamous cell carcinoma.

Lice are very common in goats, especially fibre breeds, and are becoming increasingly difficult to control. Since heavy infestations reduce the quality of mohair and cashmere, control of lice is important. Many synthetic pyrethroid-based pour-on formulations developed for sheep have been used in goats but now appear to be less effective in controlling lice, probably due to the development of resistance. Also, goats do not have as much oil in their fleece as sheep and the spread and persistence of the insecticide is therefore less. Some goat farmers in New Zealand are now using plunge or spray dips for lice control, although good results are currently being achieved with the insect growth regulators ("Zapp", "Magnum").

The biting lice *Damalinia caprae* and *D. limbata* and the sucking louse *Linognathus stenopsis* are the most common species found on goats in New Zealand and Australia. Although *D. ovis* has been shown to infect goats experimentally, establishment and persistence of infection in the field is unlikely. Lice in goats and sheep should therefore be considered species specific.

Demodectic, **sarcoptic** and **chorioptic mange** are reported in goats, but are of limited importance in New Zealand. As in other species, *Demodex caprae* invades hair follicles, but the skin nodules may be quite large and filled with material resembling pus. A smear of the contents will reveal large numbers of mites. *Sarcoptes scabei* var. *caprae* cause intense pruritis as they burrow through the epidermis. *Chorioptes bovis* infects the scrotum and feet of goats but is seldom clinically significant.

Ticks are a problem in goats in some areas of New Zealand, and may cause significant blood loss in heavy infestations. Hide damage also occurs.

Fly strike is less common in goats than in sheep but does occur. Footrot lesions often become struck during the summer, as does the skin beneath the urine-stained fibre around the pizzle of Angora bucks.

Summary of differences in disease susceptibility between goats and sheep

Diseases to which goats are less susceptible than sheep

- **Enterotoxaemia**

Very common in lambs but rare in goats in New Zealand (and probably overseas). The disease in goats may differ slightly from that in sheep and needs further work.

- **Brucellosis**

Brucella ovis is not recognised as a cause of epididymitis in bucks. Nor does it appear to cause any disease problems in does.

- **Campylobacteriosis**

Although there are occasional overseas reports of abortions caused by Campylobacter spp in goats, these are of questionable validity. There is no doubt that C. fetus subsp. fetus is an important cause of abortion in sheep.

- **Salmonellosis**

Goats appear to be relatively resistant to enteric forms of salmonellosis, although there are occasional reports of infection following stress (e.g. transport).

- **Facial Eczema (sporidesmin toxicity)**

Goats are approximately 4 times more resistant than sheep to direct dosing with sporidesmin. Furthermore, the grazing habits of goats make them less likely to be ingesting large numbers of spores from pasture. As a result, clinical facial eczema in goats is uncommon.

- **Pregnancy Toxaemia**

Unlike sheep, goats subjected to nutritional deprivation in late pregnancy are likely to abort rather than develop pregnancy toxaemia.

- **Fly Strike**

Goats are susceptible to fly strike but it is much less common than in sheep and tends to involve specific sites, such as feet and pizzle.

- **Vaginal prolapse (bearing)**

A very common and annoying problem in sheep in New Zealand but rarely, if ever, seen in goats.

Diseases to which goats are more susceptible than sheep

- **Footrot/footscald**

Probably the most significant disease problem of goats in New Zealand (especially Angoras and Boer goats) and a significant impediment to their widespread acceptance by commercial farmers.

- **Yersiniosis**

A common stress-related disease of goats in winter, sometimes causing significant losses. Is seldom a problem in well-fed goats in good condition.

- **Coccidiosis**

Kids appear to be much more susceptible to the effects of enteric coccidiosis than lambs. Infection is often combined with yersiniosis and/or nematodiasis.

- **GI parasitism**

Goats do not develop the same age-related immunity to internal parasites as occurs in sheep and cattle. Furthermore, they tend to metabolise anthelmintics more rapidly than sheep. As a result, anthelmintic-resistant nematodes are more common in goats than in sheep.

- **Trace element deficiencies**

There is good circumstantial evidence that goats are more susceptible to deficiencies of selenium, iodine, copper and possibly cobalt than either sheep or cattle.

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