



If left undiagnosed, respiratory disease in sheep can lead to significant losses.

Picture, Hamish Newton

Respiratory disease in sheep

1. Differential diagnosis and epidemiology

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RESPIRATORY disease is commonly encountered in sheep flocks, affecting groups or individuals. It often involves a combination of infectious causes as well as predisposing management factors, potentially leading to significant losses. However, cases of respiratory disease represent only 5-6 per cent of sheep submissions received by the Veterinary Laboratories Agency – a figure that is likely to be lower than the actual prevalence. In many instances, a presumptive diagnosis will be made following clinical and on-farm postmortem examinations. This article discusses the causes of respiratory disease in sheep and describes the diagnostic procedures that can be performed, with a checklist of potential pathogens for veterinary surgeons in the field. An article in the next issue will describe methods for its control and treatment.

LOWER RESPIRATORY TRACT DISEASE

Acute bacterial and viral pneumonia can affect any age of animal, but most commonly occurs in lambs aged three to 12 months after acquired maternal antibody titres have waned. Cases of chronic pneumonia usually present with illthrift and varying degrees of respiratory compromise, with respiratory distress usually initially evident during exercise.

The infectious agents described below have been identified in cases of acute pneumonia, although sub-acute and chronic disease can also occur.

BACTERIAL PNEUMONIA

Mannheimia haemolytica

Mannheimia haemolytica is a major cause of bacterial pneumonia, either as a primary or secondary infection. In the majority of combined infections it is presumed that the other respiratory pathogens involved disrupt the lung's protective mechanisms and predispose the animals to a secondary *M haemolytica* pneumonia. The source of infection is usually carrier adults, although the organism can survive in the environment, on grass or bedding, and in water.

Outbreaks can occur at any time of the year and in any age group, but most cases affect lambs less than 12 months old, although ewes at lambing are also susceptible. *M haemolytica* infections are generally most prevalent during the spring and summer, particularly between May and July, and more specifically in store and fattening lambs in the autumn and winter.

Factors predisposing lambs to outbreaks of *M haemolytica* pneumonia include:

- Housing. Cases often occur three to six weeks after animals are housed, and can last for two weeks;
- Moving lambs from poor pasture to richer aftermath pasture in late summer. A move back to poorer pasture often resolves the outbreak;
- Extreme weather conditions or changes;
- Concurrent infections involving other respiratory pathogens and tickborne fever;
- Stress due to handling.

Clinical signs vary. Mild cases may involve a cough and oculonasal discharge, whereas in more severe outbreaks some animals may be found dead and those that are affected and still alive often have a high temperature in the region of 40.4 to 42°C, hyperpnoea/dyspnoea, loud respiratory sounds, serous nasal and ocular discharges, and froth emanating from the mouth. The clinical course is usually 12 hours to death without treatment, but some animals can survive for as long as three days. Chronic cases can occur following initial recovery, resulting in persistent ill health and poor growth rates. Lambs up to 12 weeks of age will often have a septicaemia with lung involvement.

In outbreaks involving lambs, the morbidity can be up to 40 per cent, with a mortality between 2 and 20 per cent.

Mycoplasma species

Mycoplasmas have a worldwide distribution. Infection principally occurs in housed or densely stocked store lambs, particularly following two months of housing. Outbreaks can take place following the mixing of

market-bought lambs, particularly in the autumn and winter, and also during periods of high temperatures during the summer. Although acute disease has been seen in young lambs, a chronic infection is often found in older lambs and adults.

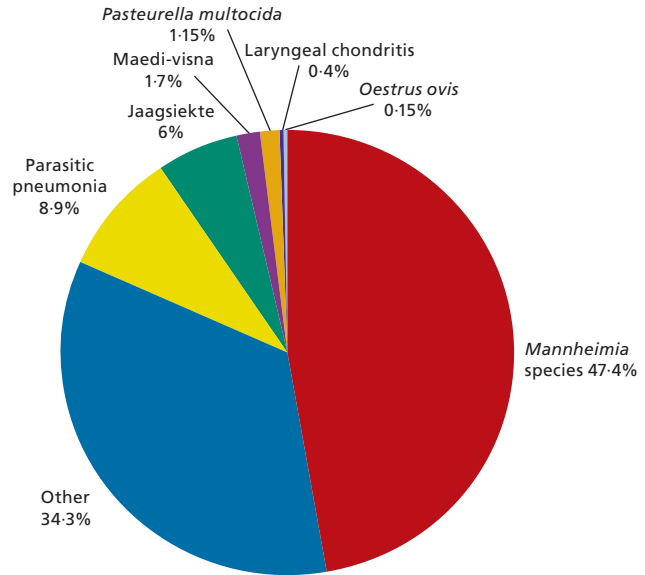
Mycoplasma pneumoniae in sheep is usually caused by *Mycoplasma ovipneumoniae*, which results in an interstitial bronchopneumonia that is often subclinical. However, when clinical signs do occur they include a chronic, persistent, soft cough (which can lead to rectal prolapse), an ocular discharge and a mucopurulent nasal discharge, with signs potentially lasting for six months or more. Cases are usually afebrile, although acute cases can be febrile, anorexic and listless with respiratory distress; in many animals, more severe clinical signs are due to a secondary *M haemolytica* infection.

Clinical signs can be seen in lambs from five weeks of age. Although variable numbers of animals will initially show signs, most of the group may be affected after several weeks. In general, the disease frequently goes undiagnosed and is rarely fatal; however, growth rates may be slowed.

M ovipneumoniae is the most frequently isolated organism from cases of chronic enzootic pneumonia complex in a mixed infection with other respiratory pathogens such as parainfluenza virus type 3 (PI3) and adenovirus. It is thought that the organism may predispose the lower respiratory tract to other infections. It has also been isolated from cases of mastitis and keratoconjunctivitis, although its role in these diseases is unknown.

Mycoplasma arginini may contribute to disease when in mixed infections, but is not necessarily a primary pathogen. *Mycoplasma bovis* has also occasionally been isolated from sheep respiratory tracts.

Exotic mycoplasmas should always be considered in sheep with respiratory problems, as respiratory disease can feature in outbreaks of contagious agalactia (a notifiable disease), together with keratoconjunctivitis, polyarthritis and mastitis.



Average annual diagnoses of respiratory disease in sheep in England and Wales, from 2002 to 2005, based on VIDA data. 'Other' includes diagnoses of parainfluenza virus type 3, *Mycoplasma ovipneumoniae* and lung abscesses, as well as occasional diagnoses such as mycotic pneumonia and *Histophilus somni* pneumonia

Bibersteinia trehalosi

Bibersteinia (formerly *Pasteurella*) *trehalosi* is usually associated with systemic pasteurellosis; however, serotypes T3 and T4 have been isolated from pneumonic lungs in ewes and lambs, although serotype T3 appears to be frequently associated with pneumonic lesions.

Pasteurella multocida

Pasteurella multocida can cause pneumonia in sheep, with outbreaks occurring in lambs, sometimes concurrently with septicaemia.

Bordetella parapertussis

Bordetella parapertussis infection in lambs can produce a mild pyrexia, mild bronchopneumonia and tracheo-bronchitis, with rapid clearance of the organism from

Differential diagnoses of respiratory disease in sheep

Lower respiratory tract disease

BACTERIAL PNEUMONIA

- *Mannheimia haemolytica*
- *Mycoplasma* species
 - *Mycoplasma ovipneumoniae*
 - *Mycoplasma arginini*
 - *Mycoplasma bovis*
- *Bibersteinia trehalosi*
- *Pasteurella multocida*
- *Bordetella parapertussis*
- *Histophilus somni*
- *Chlamydophila abortus/Chlamydophila pecorum*
- *Streptococcus zooepidemicus*
- *Erysipelothrix rhusiopathiae*
- Opportunistic bacteria
 - *Escherichia coli*
 - *Streptococcus* species
 - *Klebsiella* species
 - *Pseudomonas* species
 - *Neisseria* species/*Neisseria cuniculi*

VIRAL PNEUMONIA

- Parainfluenza virus type 3
- Ovine adenovirus
- Respiratory syncytial virus
- Reovirus types 1, 2 and 3
- Herpesviruses
 - Bovine herpesvirus types 1 and 5
 - Ovine herpesvirus types 1 and 2
 - Caprine herpesvirus type 1

LUNG ABSCESSSES

- *Arcanobacterium pyogenes*
- *Staphylococcus aureus*
- *Fusobacterium necrophorum*
- *Actinobacillus lignieresii*
- Tuberculosis
- *Corynebacterium pseudotuberculosis*

CHRONIC VIRAL RESPIRATORY DISEASE

- Maedi-visna
- Ovine pulmonary adenocarcinoma (jaagsiekte)

PARASITIC PNEUMONIA

- Lungworm
 - *Dictyocaulus filaria*
 - *Protostrongylus rufescens*
 - *Muellerius capillaris*

OTHER CAUSES

- Inhalational pneumonia
- Mycotic pneumonia
 - *Aspergillus* species

Upper respiratory tract disease

- Laryngeal chondritis
- Nasal myiasis
- Nasal foreign bodies
- Nasal tumours
 - Adenopapillomas or nasal polyps
 - Squamous cell carcinomas
 - Adenomas
 - Adenocarcinomas
- *Salmonella arizonae* rhinitis

Investigating respiratory disease in sheep

- Clinical history
- Clinical signs
- Postmortem examination – gross pathology, lung bacterial culture, fluorescent antibody test (FAT) on lung tissue or tissue or bronchiole swabs, histopathology of lung tissue
- Bronchoalveolar lavage – bacterial culture, FAT, jaagsiekte PCR
- Single serology – maedi-visna, jaagsiekte PCR
- Paired serology – parainfluenza virus type 3 and *Mycoplasma ovipneumoniae*
- Faecal examination – isolation of lungworm larvae (using the Baermann technique)



Baermann apparatus set up to examine faecal samples for lungworm larvae. A sieve and some gauze are positioned in the funnel, onto which the faeces and some water are placed and left for 24 to 48 hours. The larvae migrate through the gauze and settle in the neck of the funnel. The water is then drained out of the tube at the bottom and the sediment examined microscopically for the presence of first-stage larvae

Bronchoalveolar lavage technique

- Restrain the sheep in a standing position with its neck slightly extended. Clip a 50 x 70 mm area 50 mm distal to the larynx. Disinfect the area with 4 per cent chlorhexidine gluconate followed by 70 per cent alcohol
- Infiltrate the area with lidocaine using a 21 gauge needle
- Identify an area between two tracheal rings by palpation and insert a 12 gauge, 52 x 2.7 mm intravenous catheter at an angle of 45° to the skin. On entering the

tracheal lumen, retract the needle and advance the catheter

- Determine the required length of a 1.7 (5 French gauge) x 570 mm polyethylene extension male to female Luer lock suction catheter by measuring from the entry point on the trachea to a point behind the shoulder. On average, 40 to 45 cm of tubing is required for an adult sheep
- Introduce the tube through the catheter and advance it down the trachea until

resistance is encountered. At this point, the animal usually coughs

- Steadily infuse 30 ml of sterile isotonic saline through the tube using a 60 ml syringe
- Withdraw the fluid after five seconds, by gently moving the intratracheal tube backwards and forwards to aid recovery. Remove the tube in one movement. On average, 10 ml of fluid is recovered

From Sheehan and others (2005)

METHODS OF DIAGNOSIS

	Method	Comments
Viral pneumonia		
Parainfluenza virus type 3	FAT on BAL fluid or lung tissue during the first six days of infection ELISA Immunohistochemistry on fixed lung	Paired serology confirms recent infection
Ovine adenovirus	AGIDT	Seroconversion takes four to five weeks. Test not currently available
Respiratory syncytial virus	FAT on lung tissue or BAL fluid Immunohistochemistry on fixed lung tissue	
Maedi-visna	AGIDT or ELISA Postmortem examination and lung histopathology	Lungs are two to four times the normal weight of 300 to 500 g
Ovine pulmonary adenocarcinoma	'Wheelbarrow' test Postmortem examination and lung histopathology Jaagsiekte retrovirus PCR	Lifting the hindlimbs produces profuse fluid from the nostrils Detects virus in peripheral blood leucocytes in infected sheep. Sensitivity is highest in sheep with actual lesions. Also detects virus in tissues and respiratory tract exudate
Bacterial pneumonia		
<i>Pasteurella</i> species	Bacterial culture of lung tissue Histopathology	Lesions contain oat-shaped cells
<i>Mycoplasma</i> species	Mycoplasma culture of lung tissue PCR/denaturing gradient gel electrophoresis on tissue or tissue swab ELISA	Needs transport medium Antibody titres peak 41 days postinfection and last for 16 weeks. Paired serology indicates recent exposure
<i>Bordetella</i> species	Bacterial culture	Sampling from the airways is necessary for reliable isolation
Other bacteria	Bacterial culture of lung tissue or BAL fluid	
Tuberculosis	Positive tuberculin test Isolation from lesions or secretions	
Parasites		
Lungworm	Presumptive mild respiratory disease in lambs during late summer and early autumn Baermann technique Postmortem examination	Identification of first-stage larvae Identification of adult worms in the trachea/bronchioles. <i>Muellerius capillaris</i> can be seen by pressing the lung nodule between two slides
Nasal myiasis	Frenzied behaviour of sheep Chronic nasal discharge Postmortem examination	Larvae can be seen in the sinuses
FAT Fluorescent antibody test, BAL Bronchoalveolar lavage, AGIDT Agar gel immunodiffusion test		

the lower respiratory tract. The bacterium may potentially initiate more severe respiratory disease by damaging the tracheal epithelium, thus predisposing lambs to a secondary *M haemolytica* infection.

Histophilus somni

Haemophilus somnus, *Histophilus ovis* and *Haemophilus agni* have now been reclassified as a single family, *Histophilus somni*, the ovine strain of which is also able to infect cattle and goats. However, the bovine strain appears unable to infect sheep.

There are infrequent reports of the involvement of *H somni* in incidents of respiratory disease in sheep, but it has been associated with outbreaks of septicaemia and lung lesions in lambs. *H somni* (*H ovis*) was confirmed as the cause of pneumonia, as well as epididymo-orchitis, meningoencephalitis, septicaemia, mastitis and metritis in sheep in an Australian study (Philbey and others 1991). The organism has also been isolated from sheep with fibrinous pleuritis, bronchopneumonia, necrotising vasculitis and grossly visible lung abscesses (Rahaley 1978, Poonacha and Donahue 1984).

VIRAL PNEUMONIA

Parainfluenza virus type 3

PI3 occurs worldwide and is considered to be a common virus based on serological prevalence studies carried out in various countries. Ovine PI3 is distinct from both bovine and human PI3.

Most clinical cases occur in lambs when protective maternal antibodies wane, although adults can sometimes be affected. PI3 infection causes a mild, undifferentiated interstitial pneumonia. Clinical signs are usually mild or inapparent and most cases are afebrile, although occasionally animals may have pyrexia (40 to 41°C). Some animals may have a copious serous nasal discharge and an ocular discharge. Severe clinical disease is seen following high viral loads in experimental infections or when there is a concurrent infection, particularly with *M haemolytica*. Lung lesions usually persist for two months following infection.

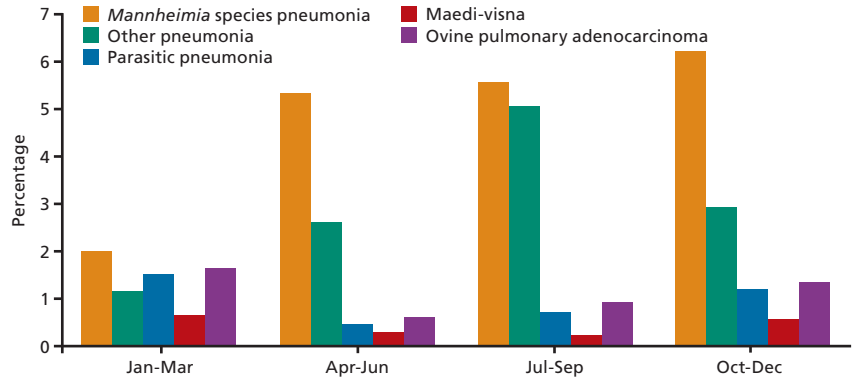
Ovine adenovirus

Sheep seroprevalence studies have revealed that ovine adenovirus is common in many countries. The virus has, in most cases, been isolated from apparently healthy lambs, but has also been isolated from animals with severe pneumonia and enteritis. Clinical signs seen following the experimental infection of lambs include pyrexia, anorexia, nasal discharge, sneezing and pneumonia, with the most severe lesions found in the terminal airways. Central nervous signs, hepatic necrosis, lymphangitis and necrotising enteritis have also all been seen with adenovirus infections. In most cases, the virus can be isolated from the nasal secretions for up to 10 days after infection.

The virus can potentially be transmitted between cattle, goats and sheep.

Respiratory syncytial virus

Respiratory syncytial virus (RSV) has been detected in sheep with rhinitis, and experimental virus inoculation has produced mild pneumonia. A seroprevalence of 84.5 per cent has been reported in the USA (Lehmkuhl and others 1985), and seropositive ewes have been identified in Northern Ireland (Adair and others 1984). There are



Seasonal trends in respiratory disease diagnoses in sheep, from 2002 to 2005, based on diagnosable submissions to the Veterinary Laboratories Agency

two ovine RSV isolates, which are antigenically distinct from bovine and caprine RSV isolates.

Bovine RSV infection in sheep produces severe pathological changes, but usually without apparent associated clinical signs.

PARASITIC PNEUMONIA

Lungworm

Potentially pathogenic lungworms that can be found in sheep include:

- *Dictyocaulus filaria*;
- *Protostrongylus rufescens*;
- *Muellerius capillaris*.

When seen, clinical signs are usually due to *D filaria* infections and occur most commonly in young sheep during the autumn and winter, although cases can be seen between June and November – a warm, wet summer enhances the survival of the larvae.

The spring hatching of first-stage larvae from ewe and yearling faeces or overwintering third-stage larvae on the pasture provide a source of infective *Dictyocaulus* species larvae. During the grazing season, at least three generations of parasites can be present, with adult worms developing four weeks after infection. Most lambs are infected in their first year, resulting in a light, prolonged infection. Strong immunity develops following exposure so there are usually few worms in older sheep, unless they are suffering from a concurrent immunosuppressing condition.

Clinical signs include coughing, increased respiratory rate, bilateral nasal discharge, reduced appetite



Adult *Dictyocaulus filaria* worms (lungworms) found in a lamb's trachea during postmortem examination. Picture, Sian Mitchell

and weight loss. Deaths are uncommon unless there is a secondary bacterial infection.

The incidence of *Protostrongylus* and *Muellerius* species depends on the distribution of their mollusc intermediate hosts. Large numbers of *P rufescens* worms can result in respiratory distress and weight loss, and *M capillaris* can occasionally cause mild respiratory signs. Mixed worm infections can also occur.

The more frequent routine usage of anthelmintics in sheep may contribute to the apparent reduced clinical significance of lungworm infections when compared with cattle.

Hydatid cysts

The intermediate stages of the tapeworm *Echinococcus granulosus* occur as 'hydatid cysts', which are fluid-filled cysts of up to 20 cm in diameter that often contain numerous worm scolices. In sheep, approximately 70 per cent of hydatid cysts are found in the lungs; these lesions are then partially embedded in the lung tissue, although usually with very little local reaction and no associated clinical signs.

OTHER POTENTIAL PATHOGENS

The following pathogens have all been found in cases of acute respiratory disease in sheep, but their prevalence in natural infections is uncertain:

- Reovirus types 1, 2 and 3;
- Bovine herpesvirus types 1 and 5;
- Ovine herpesvirus types 1 and 2;
- Caprine herpesvirus type 1;
- *Chlamydomydia abortus/Chlamydomydia pecorum*;
- *Streptococcus zooepidemicus*;
- *Erysipelothrix rhusiopathiae*;
- Opportunistic bacteria following primary airway or lung damage from other pathogens: *Escherichia coli*, *Streptococcus* species, *Klebsiella* species, *Pseudomonas* species and *Neisseria* (formerly *Branhamella*) species/*Neisseria cuniculi*;
- Fungi: *Aspergillus* species, *Mortierella wolfii*.

Herpesviruses and *Chlamydomydia abortus* have produced a mild interstitial pneumonia experimentally (Whetstone and Evermann 1988, Scott and others 1990, Navarro and others 2004, Li and others 2005). *Streptococcus zooepidemicus* was found in 10-month-old lambs with a fibrinous pleuritis, pericarditis and pneumonia; co-grazing carrier ponies were suspected to be the source of infection (Stevenson 1974). *Erysipelothrix rhusiopathiae* has been isolated from adult sheep with a severe necrotising alveolitis (Griffiths and others 1991).

Mycotic pneumonia, which is often due to *Aspergillus fumigatus*, is rarely encountered, but can occur as either a primary infection or secondary to disseminated systemic infections, particularly following mammary infections (Pérez and others 1999). It produces lung oedema and greyish-white pyogranulomatous nodules. *M wolfii* has also produced lesions in experimentally infected sheep (Cordes and others 1972).

Immunosuppression may predispose cases to mycotic infection and spoiled feed is often thought to be the likely source.

LUNG ABSCESSSES

Lung abscesses are often caused by secondary opportunistic bacteria such as *Arcanobacterium pyogenes* and *Staphylococcus aureus* following previous lung damage,

for example, after an acute pneumonic incident or secondary to ovine pulmonary adenocarcinoma (jaagsiekte). They may also follow embolic spread from another focus of infection. *S aureus* lung abscesses can occur with tick pyaemia, and *Fusobacterium necrophorum* can produce abscesses in cases of inhalational pneumonia or secondary to pharyngeal abscessation. Similarly, *Actinobacillus lignieresii* may cause lung lesions secondary to oral abscesses. Pyaemia can develop following lung abscessation, sometimes presenting as sudden death.

Tuberculosis (TB) and caseous lymphadenitis (CLA) are both contagious conditions that potentially lead to lung abscesses. Tuberculosis is a rare condition in sheep but has been found in animals aged two years and older that have had close contact with TB-infected cattle. It results in either caseous lung lesions or abscesses containing liquid pus. CLA is caused by the bacterium *Corynebacterium pseudotuberculosis*, which produces abscesses in the superficial lymph nodes or skin, although lung abscesses can also occur. Spread occurs when purulent material discharges from ruptured abscesses and contaminates the environment and fomites (eg, shearing equipment). Lung lesions usually occur secondary to a disseminated haematogenous spread, although they may follow aerosol spread.

CHRONIC RESPIRATORY VIRUSES

Maedi-visna

Maedi-visna virus is a lentivirus – a non-oncogenic retrovirus – causing primarily an interstitial pneumonia, as well as indurative mastitis, arthritis and potentially neurological signs. The first cases in the UK were identified in 1979, but a low annual diagnostic rate suggests the disease is not widespread in the UK, although it is hard to detect infection due to the insidious nature of the virus.

The virus can be transmitted via milk, respiratory secretions and faeces, and survival for up to two weeks outside the body has been suggested. A low level of intrauterine transmission is believed to occur (Cutlip and others 1981), and the virus has also been detected in semen from rams with a concurrent orchitis.

Clinical disease is usually not evident in a flock until at least 50 per cent of ewes are seropositive. Clinical signs are seen in sheep at least two years of age, but most are four to five years old.

There is a protracted period of lesion development, consisting of a slow, replicating, lymphoid hyperplasia, with infected animals never developing a protective immunity to the disease. Many sheep remain asymptomatic carriers, with 25 to 30 per cent developing clinical signs.

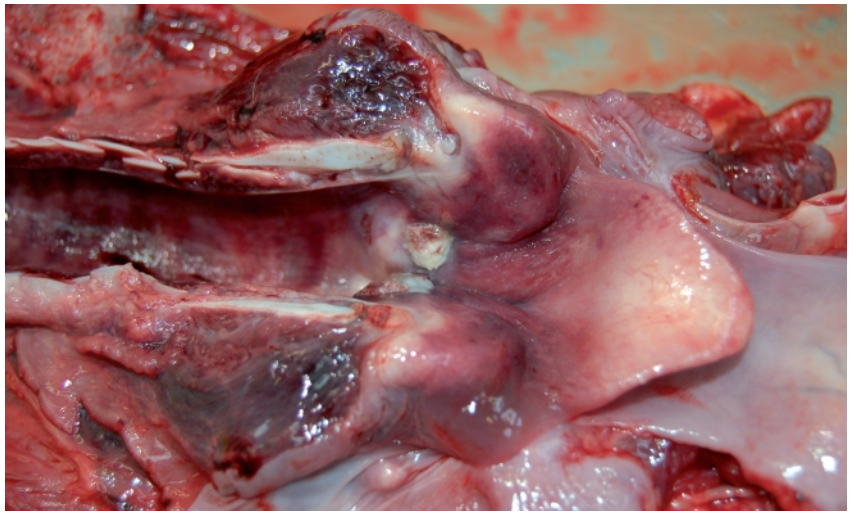
The production of antibodies can occur several months following infection, while some infected animals never develop an antibody response. The mortality in uncomplicated cases is low, with deaths generally occurring due to a secondary bacterial infection, pregnancy toxæmia or harsh climatic conditions. Right ventricular hypertrophy can develop due to the lung lesions.

Cases present as bright and alert, with loss of condition and slow-onset afebrile respiratory distress, which is usually only evident on exertion. Coughing and a nasal discharge are occasionally seen. In advanced cases, the ewes flare their nostrils, extend their necks and breathe through the mouth. Signs progress over a three- to six-month period.

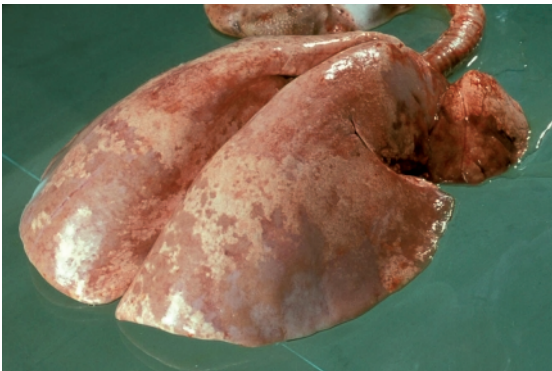
The economic impact in a flock is from earlier culling of ewes and a decrease in lamb weights at weaning.



Profuse quantities of fluid are produced by the neoplastic tissue in sheep affected by ovine pulmonary adenocarcinoma. Fluid draining from the nose, particularly when the hindlimbs are lifted (the 'wheelbarrow' test), allows a presumptive diagnosis. Picture, Hamish Newton



Laryngeal chondritis in a Texel shearling ram that had died soon after developing acute-onset respiratory distress



Postmortem examination is necessary for the diagnosis of ovine pulmonary adenocarcinoma. These lungs from an adult ewe show extensive neoplastic changes in the caudal lung lobes due to jaagsiekte retrovirus infection. The neoplastic tissue has a characteristic grey-purple appearance. Picture, Tim Jones

In extreme conditions, losses of 10 to 20 per cent gross margin per ewe have been reported.

Ovine pulmonary adenocarcinoma

Ovine pulmonary adenocarcinoma is caused by the jaagsiekte retrovirus, which induces bronchioloalveolar adenocarcinoma lung tumours that account for 70 per cent of all sheep tumours.

Horizontal transmission of the virus occurs via aerosol, and contaminated food and water troughs. The possibility of intrauterine infection has been suggested but is thought to occur at a low level, if at all. The disease incubation period is long and clinical signs are usually not seen until sheep are two to four years old. In exceptional cases, tumours have been seen in lambs as young as two months and in sheep as old as 11 years. The incidence of clinical cases in endemic flocks ranges between 2 and 10 per cent annually, with management practices, such as length of housing period and stocking densities, influencing infection rates.

Clinical signs consist of weight loss and a progressive, afebrile respiratory distress, particularly following exertion. Auscultation reveals high-pitched, moist lung sounds due to the accumulation of large amounts of fluid in the lungs that can exude from the nostrils, which is a characteristic finding of the disease. Death usually occurs due to a secondary bacterial pneumonia, although in some cases right ventricular hypertrophy can lead to heart failure.

MISCELLANEOUS DISEASES

Inhalational pneumonia

Inhalational pneumonia frequently affects an individual or a small number of animals. It occurs most often in bottle-reared lambs or following incorrect drenching or dipping, or after the inhalation of a foreign body or inhalation of rumen contents in laterally recumbent animals.

UPPER RESPIRATORY TRACT DISEASE

Upper respiratory tract infections usually present with a unilateral or bilateral serous or mucopurulent nasal discharge, coughing and sneezing. The viral causes of pneumonia discussed earlier can present with primarily upper respiratory tract signs.

The following diseases specifically affect the upper respiratory tract and are encountered sporadically in sheep flocks.

LARYNGEAL CHONDRITIS

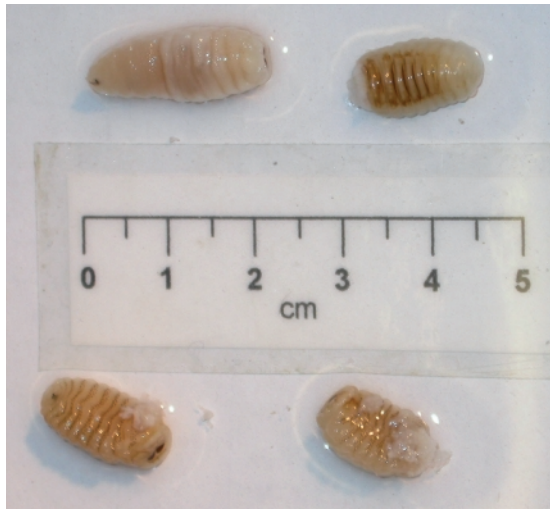
Laryngeal chondritis is an acute obstructive upper respiratory condition resulting from chronic suppurative lesions in the arytenoid cartilages of the larynx. The cause is uncertain but there appears to be a genetic predisposition in short-necked breeds such as Texels and Southdowns, although any breed can be affected. Other suggested causes include drenching injuries, rough handling, dry, dusty feed or grass awns. There is a higher incidence in rams than ewes.

Bacteria isolated from outbreaks include *A pyogenes* and mixed environmental bacteria, including *E coli* and *F necrophorum*.

Cases present with severe dyspnoea and laryngeal stridor, which can result in inhalational pneumonia. The condition is fatal if not treated promptly.

NASAL MYIASIS

Nasal myiasis, caused by *Oestrus ovis* larvae, produces a chronic rhinitis and sinusitis due to mucosal irritation by larvae located in the frontal, maxillary and nasal sinuses. First-stage larvae are deposited by adult flies on sheep nostrils between June and October, and the larvae then live and develop in the sinuses for a two- to 10-month period. First-stage larvae overwinter in the



***Oestrus ovis* larvae, an incidental finding in the sinuses of an adult ewe during postmortem examination**

sinuses and complete their development in May and June of the following spring.

Up to 80 per cent of a flock may be affected and the main associated problem is sheep annoyance. The animals tend to stamp their feet, sneeze and press their noses to the ground. Breathing can be difficult due to blocked and inflamed nostrils and a mucopurulent nasal discharge. If seen, clinical signs are usually observed in older ewes and often in late summer. Occasionally, the aberrant loca-

tion of larvae or secondary bacterial infections can cause an inflammatory reaction that interrupts feeding. This can lead to illthrift or, in some cases, a cerebral bacterial infection, pneumonia and occasionally death.

NASAL FOREIGN BODIES

Problems due to foreign bodies frequently involve inhaled plant material, such as grass seeds, resulting in a unilateral nasal discharge, which may become mucopurulent and even malodorous due to secondary bacterial infection.

NASAL TUMOURS

Nasal tumours, such as adenopapillomas or nasal polyps, squamous cell carcinomas, adenomas or adenocarcinomas occur sporadically in sheep in the UK. Clinical signs include a seromucous nasal discharge, which may be unilateral and can become mucopurulent due to secondary bacterial infection. Increased respiratory noise and distortion of the maxillary bones may also occur. Contagious nasal adenocarcinomas or enzootic nasal tumours are caused by a virus related to the jaagsiekte retrovirus. This disease occurs in other countries but has not yet been identified in the UK.

SALMONELLA ARIZONAE RHINITIS

A mild proliferative rhinitis was diagnosed in seven-month-old lambs, with *Salmonella arizonae* confirmed as the causal pathogen (Brogden and others 1994).

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