Table of contents

Non-productive disorders

Pulmonary form of lentivirosis (SRLV)

- Lentiviral infections in small ruminants are widespread in most countries of the world and have four main clinical forms: respiratory (interstitial pneumonia), nervous (encephalitis), mastitic (interstitial mastitis) and articular (proliferative arthritis) (see chapters 8, 12, 14). The respiratory clinical form is by far the most prevalentin sheep, and it is generally seen in adult animals (≥2 years old).
- Animals with pulmonary lentivirosis show dyspnoea and tachypnea, weakness, loss of weight and delay in walking with the flock (see chapter 19). If the case is uncomplicated, no cough, nasal discharge or fever is observed. However, multiple concurrent diseases such as OPA, ORC or gangrenous pneumonia can be present in sheep, thus complicating the clinical diagnosis of the disease.
- At postmortem, lungs show increased size, volume and weight and a general greyish discolouration with a myriad of grey dots in the pleural surface. Mediastinal lymph nodes are increased in size, surpassing the limit of the diaphragmatic lobes.

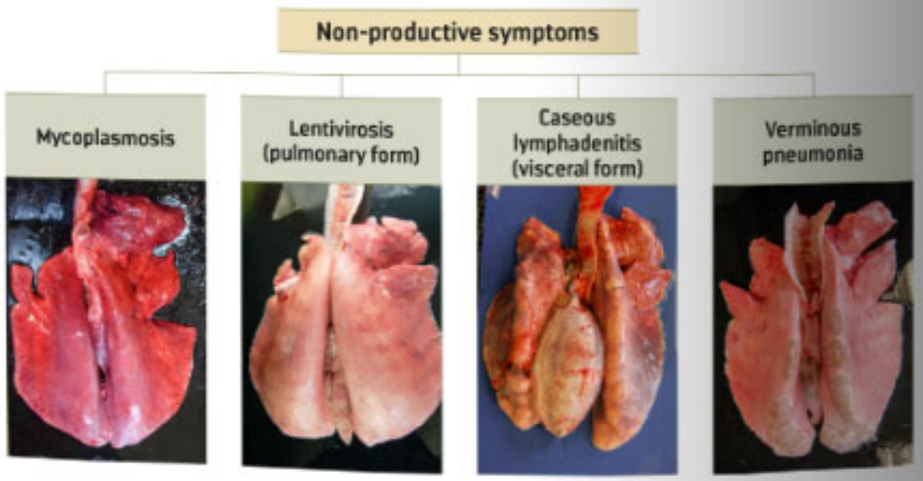
Visceral form of caseous lymphadenitis (CLA)

- Caseous lymphadenitis (CLA) is caused by *Corynebacterium pseudetuberculosis* and is common in sheep and distributed worldwide.
- CLA can present as either the superficial or the visceral form, affecting internal lymph nodes and organs and mostly observed in adults (see chapter 19). In the visceral form, most of the affected animals had lesions in the respiratory system (lung parenchyma and mediastinal lymph nodes). For that reason, this disease is also included in the differential diagnosis of lower respiratory tract diseases.
- Respiratory clinical signs associated with CLA are often not readily recognised because only dyspnoea is detected without audible sounds of the thoracic cavity.

Tuberculosis (TB)

- Tuberculosis (TB) in sheep is caused by *Mycobacterium caprae*, *M. bovis* or occasionally other *Mycobacterium* from *M. tuberculosis* complex. It is an uncommon disease in sheep but it can affect this species in TB endemic areas in the absence of adequate health plans.
- Respiratory clinical signs in the late stage of disease (e.g. deep and productive chronic cough, dyspnoea, and abnormal lung sounds) and severe emaciation are common.

Figure 3.5. Non-productive symptoms



- Pulmonary granulomatous lesions (more frequent; see chapter 19) or generalised process, with miliary lesions or large nodules granulomas in several intern organs may be present.

Verminous pneumonia

- The main pulmonary parasites of sheep are *Dictyocaulus filaria*, *Protostrongylus rufescens* and *Muellerius capillaris*.
- Usually, infections are asymptomatic, but depending on the severity, age and immunological status of the animal, clinical signs range from intermittent cough and tachypnea to persistent cough with respiratory distress. *Dyctiocaulus filaria* producing the most severe clinical signs.
- Verminous pneumonia caused by small lungworms is found in the caudal and dorsal lung lobes, and lesions are usually rounded and tend to converge. *Muellerius capillaris* is a tiny strongylid that causes small reddish or greyish nodules scattered over the whole lung surface. Adult forms of *Dyctiocaulus filaria* are found in the bronchial lumen causing local inflammation and obstruction.
- Chronic forms can lead to wasting condition in affected animals (see Chapter 19).

Diagnosis

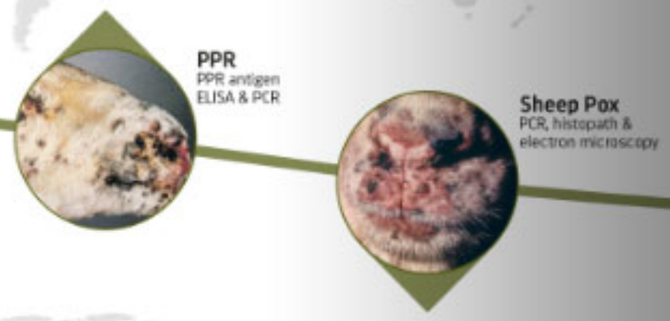
In productive disorders with moist cough and adventitious sounds, the wheelbarrow test must be carried out to confirm or rule out OPA (classic form). In addition, the exhaled air after coughing has to be smelled to detect necrotising processes associated with gangrenous pneumonia. If both diseases are ruled out, the most likely cause of the disease will be ORC. In the case of non-productive disorders, the symptoms are pretty inapparent, and the clinical diagnosis is challenging. Ultrasonography is an advantageous diagnostic technique that can be easily applied in farm conditions.

The aetiological diagnosis can be made in living animals by bronchoalveolar lavage or after necropsy with an aseptic sampling of affected lung tissue and mediastinal lymph nodes. Histological and immunohistochemical diagnoses may also be confirmatory. The serological test can be used to detect sheep infected with lentivirus in order to develop control plans. Periodical coprological tests are the most helpful tool to detect parasites causing verminous pneumonia.

Table IV. Diagnostic techniques

	ORC	OPA	ASPIRATION PN.	PARASITIC PN.	LENTIVIROSIS	CLA
Clinical signs	Easy	Easy in advanced cases	Moderate	Difficult	Challenging	Difficult
Thermography	No	No	No	No	No	No
Ultrasonography	Yes	Yes	Yes	Yes	Yes	Yes
X Ray	Yes	Yes	Yes	Yes	Yes	Yes
CT Scan	Yes	Yes	Yes	Yes	Yes	Yes
Aetiology	*Mannheimia haemolytica Pasteurella multocida Biberstenia trealosis Mycoplasma* sp.	ISRV	*Trueperella pyogenes* and others	*Dictyocaulus filaria Protostrongylus rufescens Muellerius capillaris*	SRLV	*Corynebacterium pseudotuberculosis*
Serology	No	No	No	No	Yes	No
Coprology	No	No	No	Yes	No	No

Viral diseases in high to low mortality outbreaks



PPR
PPR antigen
ELISA & PCR

Sheep Pox
PCR, histopath &
electron microscopy

Worldwide distribution of capripoxviruses from 2000 to 2010

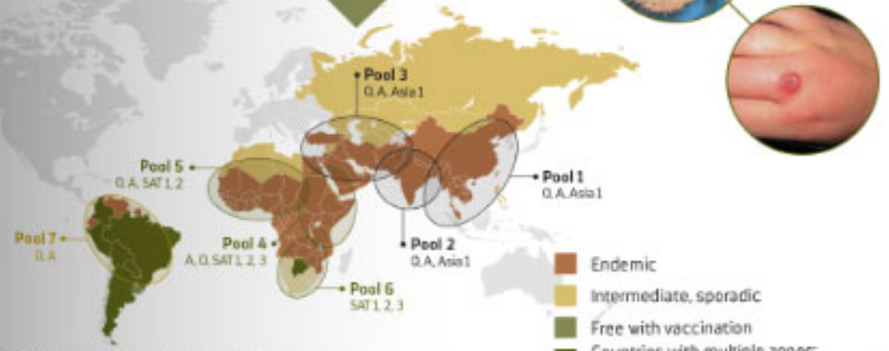
- Lumpy skin disease now in SE Asia
- Sheep pox and goat pox
- Sheep pox/goat pox and lumpy skin
- Not present or no information

Scaled transmission risk suitability of bluetongue virus for sheep, as the primary host at risk, worldwide.
The scale ranges from a low risk, 0, to a high risk, 5.

Bluetongue
Bluetongue antigen
ELISA & PCR

FMD antigen
ELISA & PCR

ORF
PCR, histopath &
electron microscopy



There are 7 "FMD Pools" reflecting the different circulating virus populations globally in the the OIE/FAO-led global FMD control strategy, although increasing trade in livestock & their products between countries means that viruses are increasingly shared between some pools (e.g. Pool 2 to Pool 1).

- Endemic
- Intermediate, sporadic
- Free with vaccination
- Countries with multiple zones: FMD-free, free with vaccination or not free
- Free, Virus present in game parks
- Free

Trauma / injuries

It can be inflicted by predators (e.g. dogs or wild animals), road traffic accidents and also humans. In the case of predator attack, it should be checked for puncture wounds and other tooth marks with bruising signs, as well as haemorrhage into tissues adjacent to injury sites (figure 1.4). It is important to check if the injury was inflicted before death because predation can also happen once an animal has died if the body is not removed immediately. Road traffic accidents and injuries caused by humans may cause recognisable external damage, subcutaneous and internal haemorrhages, fractures, as well as rupture of internal organs.

Figure 1.4. Typical findings due to predator attack.

Sudden death in neonatal lambs

Newborn lambs have great susceptibility to hypothermia, mainly related to adverse weather conditions, low birth weight and hypoglycaemia due to lack of colostrum intake. Also, the period in which clinical signs of illness can be detected is normally short. Common causes of sudden death are described below.

Congenital malformations (see chapters 4, 8 and 10)

It may be lethal and caused by different agents such as congenital goitre by iodine deficiency. Toxic plants can generate arthrogryposis, micrognathia, palatoschisis, hypoplasia or unilateral or bilateral incisor bone aplasia, kyphosis, scoliosis, torticollis, distinct degrees of corneal opacity and/or microphthalmia, ocular dermoids, acephalia, bicephaly, hydranencephaly, tongue hypoplasia, meningocele and syringocele. Viruses such as bluetongue (BTV), Schmallenberg, Akabane and border disease can cause arthrogryposis, hydrocephalus, cerebral cysts and retinal dysplasia.

Neonatal infections

Lambs with lesions such as arthritis, cystitis, enteritis (e.g. lamb dysentery or colibacillosis; see chapter 5), meningitis, omphalophlebitis, pericarditis, pneumonia (see chapter 3), and urachitis.

Starvation / hypothermia

Newborn lambs that walked but did not feed. PME shows: dehydration, little fat reserve, empty gastrointestinal tract, absence of meconium elimination, dark liver with increased consistency, bladder filled with urine, and enlarged and haemorrhagic adrenals. It is also important to check the ewe milk supply and its maternal ability.

Predation

PME shows haemorrhage or other traumatic injuries in the skin, subcutaneous tissue, muscles and bones.

Trauma

At PME can be observed haemorrhage and suffusions in the subcutaneous tissue, fractured ribs, liver, lung and brain haemorrhage with clots in the abdominal and/or thoracic cavity (figure 1.5).

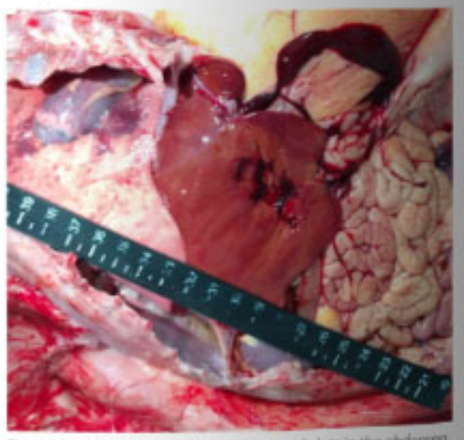


Figure 1.5. Liver rupture with haemorrhage and clots in the abdomen.

Sudden death in growing-fattening lambs

In this category, it is important to differentiate whether it is dealing with an individual or collective condition. Many deaths in this age group are often related to feeding and weather changes, which trigger outbreaks of sudden deaths.

Individual cases

Red gut or intestinal torsion

It is a catastrophic intestinal accident when the root of the mesentery occludes due too secondary to abnormal positioning of the small and/or large intestines. PME shows a bloated abdomen and rapid deterioration of the carcass. Dark red to black intestines, markedly gas-filled, and presence of serosanguinous fluid in the abdominal cavity (figure 1.6). This condition is mainly seen in animals grazing lush, highly digestible pastures and also in artificially fed or creep-fed lambs.

Bloat

It may involve distention of the abomasum and/or cecum, or when developed, the rumen, usually in artificially fed lambs. PME shows lungs and liver compressed and dorsally displaced. Most commonly, the lungs are slight to moderately congested, while the liver and heart are pale. Abomasal rupture can also be found in cases of abomasal bloat. In older weaned lambs, the pressure of the distended rumen causes the caudal oesophagus to look pale in comparison to the cervical oesophagus, which generally presents congested at the thoracic inlet (figure 1.7).

Figure 1.6. Intestinal torsion. Dark red to black segment of small intestine filled with gas.

White muscle disease (see chapter 14)

A disorder caused by selenium/vitamin E deficiency. The onset of clinical signs can occur from birth to several weeks later. Heart failure due to muscle damage can lead to sudden death in young lambs. PME normally shows visible white streaks or patches in the skeletal or cardiac muscle. Diagnosis can be established by histological examination, as well as the measurement of selenium and glutathione peroxidase levels. Although banned in Europe and some countries in the world, **ionophore poisoning** (e.g. monensin) can occasionally occur as the result of a miscalculation in inclusion rate when added to creep feed or flock diet and resembled nutritional myopathy, even though in the PME is also seen hydroperitoneum, hydrothorax and lung oedema.

Collective cases

Clostridial infections

Diseases caused by clostridia in lambs and adult sheep are summarised in table III (see chapter 5 and 6).

Peracute ovine respiratory complex (ORC) (see chapter 2)

Normally, lambs are often "found dead" at 3-4 weeks of age due to a drop in maternal antibodies and stress factors. PME shows signs of septicaemia such as petechiae in the heart, liver and spleen, as well as swelling and hyperaemia of cervical and thoracic lymph nodes. Also, subcutaneous haemorrhages over the throat area, lung congestion and oedema with froth in the airways may be seen.

Figure 1.7. Bloat. Pale caudal oesophagus in comparison to the congested cervical oesophagus at the thoracic inlet. Image courtesy of Dr. Rick Last, Vetdiagnostix.

Table IV. Chemical values of aqueous and vitreous humours in sheep

	AQUEOUS HUMOUR		VITREOUS HUMOUR	
	Reference range (mmol/l)	Recommended sampling time	Reference range (mmol/l)	Recommended sampling time
Calcium	<1	Immediately following death	<1	< 48 hours
Magnesium	< 0.33	< 24 hours	< 0.65	< 48 hours
Urea	> 30	None given	> 6.6	< 36 hours
Beta-hydroxybutyrate	> 2.5	None given	-	-
Nitrate	Presence	< 60 hours	Presence	< 60 hours

Figure 1.15A. Aqueous humour collection. Insert the needle through the limbus.

Figure 1.15B. Aqueous humour collection. Angle towards the cornea keeping the point in front of the lens. Once the tip is free in the aqueous humour, push the vacutainer onto the needle and aspirate 0.5-1 ml of fluid.

Sudden death in rams

When managed in groups and particularly at the beginning of the breeding season, the main causes of sudden death in rams are related to trauma caused by fighting that lead to neck fracture and spinal cord injuries, especially when a new animal is introduced into the flock with adult males (figure 1.16). Clostridial infections (see chapter 5 and 6), mainly histotoxic ones, may also occur due to wounds from fighting. In housed animals, mainly in elite ram-breeding flocks, metabolic metabolic disorders such as ruminal acidosis, enterotoxaemia due to grain overload and obstructive urolithiasis (see chapter 7) should be investigated.

Figure 1.16A. Buck showing acute quadriplegia due to cervical trauma.

Figure 1.16B. Haematoma in the spinal cord.

Sudden death in any adult category

Infections considered exotic or restricted to certain regions

Bluetongue (see chapter 4)

BTV outcome of infection ranges from inapparent to fatal, with the severity of disease dependent on factors related to agent, host, the environment and concomitant stress factors. In peracute cases death can occur. PME normally shows congestion, oedema, haemorrhages of lungs with severe alveolar oedema and the bronchial tree filled with froth; thoracic cavity and pericardial and pleural effusion; hypertrophy of lymph nodes and splenomegaly.

Rift valley disease (RFV)

RVF can be confused with many viral diseases. However, the following clinical signs are characteristic of this disease: a sudden onset of abortions at all stages of pregnancy; an acute febrile disease with high fatality rates in young animals; liver lesions (congestion and necrosis); associated with presence of high mosquito populations and/or flooding of grasslands and; may be related with an influenza-like disease in humans. PME shows generalised lymphadenopathy, petechial and ecchymotic haemorrhages throughout the carcass, and often haemorrhagic gastroenteritis.

Heartwater disease

It can cause sudden death with lymphadenopathy and generalised haemorrhages throughout the carcass. There is no hepatitis, and usually presents a obvious excessive fluid in the serous cavities. Neurological signs can also be observed. Brain smears can be prepared to make a definitive diagnosis.

Sheep pox (see chapter 4 and 15)

Peracute cases may show few clinical signs before sudden death. PME includes signs consistent with septicaemia, as well as hyperaemic lungs with typical pox "gunshot" lesions (figure 1.17).

Peste des petits ruminants, PPR (see chapter 4)

It is a severe, fast-spreading disease of mainly domestic small ruminants. It is characterised by the sudden onset of depression, fever, discharges from the eyes and nose, sores in the mouth, disturbed breathing and cough, foul-smelling diarrhoea and death. PME necrotic lesions in the mouth and nose, congestion of the ileocaecal valve, engorgement and blackening of the folds in the caecum, colon and rectum "zebra striping" lesions (figure 1.18), enlarged spleen, oedematous lymph nodes and bronchopneumonia.

Figure 1.17 Sheep pox infection. Typical "gunshot" lesions in the lungs.
Image courtesy of Dr. G.B. Manjunatha Reddy, ICAR-NIVEDI.

Figure 1.18. Peste des petits ruminants. Engorgement and blackening of the rectal folds, "zebra striping" lesion.
Image courtesy of Dr. G.B. Manjunatha Reddy, ICAR-NIVEDI.

Figure 4.1. Meconium staining the skin due to dystocia.

Figure 4.3. Holoprosencephaly.

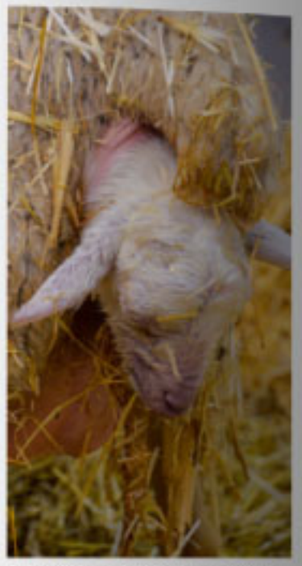


Figure 4.5. Facial swelling due to dystocia.

Figure 4.2. Palatoschisis.

Figure 4.4. Arhinencephaly and ciclopy.

with multiple roles in development and disease following Shh gene expression in the developing embryo.

Craniofacial oedema

Massive accumulation of serous fluid in the subcutaneous facial connective tissue of foetuses is most commonly associated with dystocia (figure 4.5). Alternatively, the presence of extensive oedema that involves the entire body is suggestive of congenital anasarca.

Inherited disorders

In addition to the congenital CFDs described above, there is an abundance of mutations in sheep, causing a range of inborn errors of metabolism of all body systems, with some involving oral structures. Brachygnathia, cardiomegaly and renal hypoplasia syndrome (BCRHS) is a recessively inherited disorder in Australian Poll Merino sheep, with affected lambs stillborn and displaying dwarfism with various other congenital defects. A mutation in the obscurin-like cytoskeletal adaptor 1 (OBSL1) was identified and enabled improved breeding management of the affected flock, plus provided an ovine model for human 3M syndrome-2 that also results in altered stature.

Figure 4.6. Sheep photosensitisation.

Porphyrins are a class of organic compounds characterised by four pyrrole nuclei connected in a ring structure. When combined with iron, porphyrins form haem, which is a component of haemoglobin, cytochromes, catalases and peroxidases, all constituents of many compounds with vital roles in biological systems. The biosynthesis of porphyrins involves a six-step process, starting with aminolaevulinic acid (ALA) and ending with protoporphyrin. Mutations of the uroporphyrinogen decarboxylase (UROD) gene have been shown to cause porphyria cutanea tarda (PCT) in humans and have described in a flock of German Blackface sheep with a single point mutation (C→T) in UROD. Affected animals display the typical signs of porphyria include grey-metal mucosae, photosensitivity and porphyrinuria.

Oral disorders in lambs post-weaning through adulthood

Broken mouth

This is a colloquial term used widely throughout sheep industries to describe the very common oral lesions occurring from the excessive wear and loss of incisor teeth in ageing sheep (figure 4.7 to 4.10). It is associated with trauma from prehension of harsh feed and thistles, plus the grinding of dirt when sheep need to dig for plant roots, especially in droughts.

Figure 4.7 "Broken Mouth" excessive teeth wear.

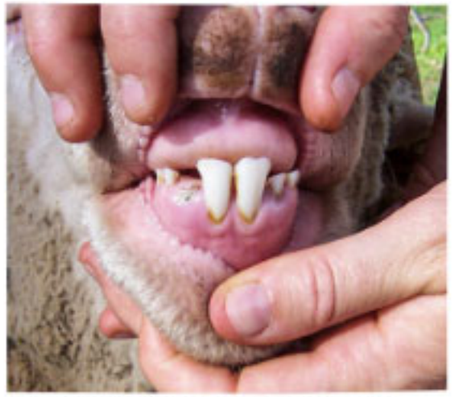


Figure 4.8. Transition from deciduous to permanent incisors.

Figure 4.9. Grass seed in oral mucosa.

Figure 4.10. Excessive upper premolar from loss of lower tooth.

Chapter 5:
Digestive disorders in lambs

Luis Miguel Ferrer. University of Zaragoza (Spain). PhD, Dipl. ECSRHM.
Marta Ruiz de Arcaute. University of Zaragoza (Spain). PhD.

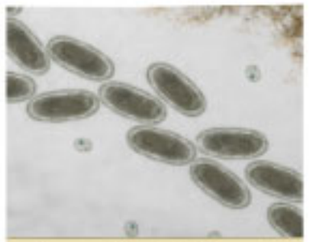
Digestive disorders are the leading cause of death in lambs in the first three weeks of life and, jointly with the ovine respiratory complex, the most critical diseases up to the time of weaning.

Predisposing risk factors

- Mother's feeding
- Colostrum intake
- Mother's age
- Mother's illnesses
- Failures in the health plan
- Facilities (overcrowding, lack of hygiene, etc.)
- Climate
- Workforce
- Artificial lactation

Factors depending on the animal

- Behaviour and vitality of the newborn "will to live"
- Birth weight



Determining risk factors

Digestive viruses (rotavirus)

Bacteria

- *E. coli*
- *Clostridium* sp.
- *Salmonella* sp.
- *Campylobacter* sp.

Parasites

- *Cryptosporidium parvum*
- Coccidia
- Giardias
- Other digestive parasites

Figure 5.1. Lambs diarrhoeal syndrome is a multifactorial and pluricausal disease that can cause significant economic losses.

Figure 7.1. Main causes of abdominal pain in sheep

Intestinal disorders

- Colic/diarrhoea/tenesmus
- Obstruction
- Volvulus
- Coccidiosis
- Salmonellosis
- Acute indigestion

(see chapters 5 and 6)

Gastric compartments

- Dilations
- Traumatic reticuloperitonitis
- Ruminal acidosis/alkalosis
- Bloat (frothy/free-gas)
- Compression
- Ulcers
- Volvulus

(see chapters 5, 6 and 7)

Peritoneum

- Septicaemia/azotaemia
- Ascites
- Uroperitoneum
- Aseptic peritonitis
- Traumatic peritonitis
- Hernias
- Omphalitis

(see chapter 7)

Female reproductive system

- Atony/contractions
- Advanced pregnancy
- Dystocia
- Uterine rupture

(see chapter 10)

Urogenital system

- Anuria/strangury
- Uroliths
- Fibrin and/or clots
- Malformations
- Vesiculitis

(see chapter 7)

Figure 7.2. Information obtained by inspection of the posterior view of the abdomen, divided into quadrants

Left dorsal quadrant

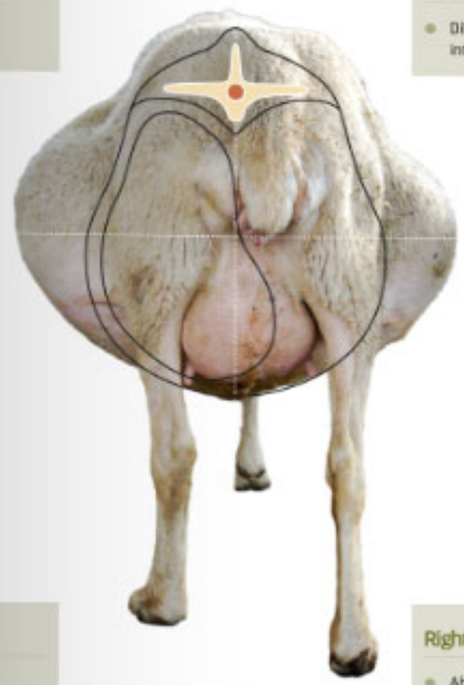
- Free-gas bloat
- Frothy bloat

Right and left dorsal quadrant

- Pneumoperitoneum
- Severe bloat/meteorism

Right dorsal quadrant

- Dilatation of the cecum or other intestinal portions



Left ventral quadrant

- Frothy bloat
- Acute ruminal acidosis
- Indigestion
- Ruminal impaction

Right and left ventral quadrant

- Ascites (symmetrical)
- Bladder rupture
- Indigestion/advanced pregnancy
- Hydroamnion/hydroallantois

In lambs:

- Omphalitis
- Omphalophlebitis

Right ventral quadrant

- Abomasal impaction
- Advanced pregnancy
- Hydroallantois

On either side

- Hernias and muscle tearing and separation
- Abscesses
- Others

Wesselsbron virus

Wesselsbron virus is a mosquito-borne RNA virus within the genus *Flavivirus* in the family *Flaviviridae*. The virus is endemic throughout the African continent and is able to infect a wide range of domesticated animals like sheep, goats, cattle, camels and horses, and humans. Sheep seem to be the most susceptible host, and disease manifests with comparable clinical signs as with Rift Valley fever. Infection in adult sheep and older lambs can remain asymptomatic or manifests with fever, while in newborn lambs, morbidity and mortality rate are high. Infection of pregnant ewes leads to abortion, stillbirths, and the birth of lambs with congenital malformations. In humans cases, infection is associated with fever, headache, myalgia and arthralgia.

Parasites

Toxoplasma gondii

Toxoplasma gondii is a protozoan parasite with a sexual cycle in cats and other felidae, which results in the production of oocysts (figure 11.8). An asexual cycle occurs in a wide range of animal species, including sheep and humans. If a susceptible pregnant ewe ingests feed or water contaminated with sporulated oocysts, the infection may establish in the gravid uterus. Infection in early gestation may result in death and resorption of the foetus. Late-term infections may result in normal but infected and immune lambs. *T. gondii* abortion usually results following infection in mid-gestation, and stillborn and weakly lambs are born, some of which may show neurological signs. Delivery of mummified foetuses of different stages of pregnancy (figure 11.9), and numerous small white foci of necrosis of 1-3 mm in diameter on placental cotyledons while the intercotyledonary allanto-chorion appears normal are typical features of infection with *Toxoplasma gondii* (figures 11.10).

Toxoplasmosis is considered one of the most significant food-borne zoonoses worldwide. All three developmental forms of the parasite, the tachyzoite, the bradyzoite and the oocyst-containing sporozoites are infectious both to the intermediate host and final hosts. *T. gondii* can pose a serious risk to the unborn child if the mother is infected for the first time while pregnant. Immunocompromised people are also at risk.

Neospora caninum

Neospora caninum is an infectious cause of abortion in cattle. The agent is closely related to *Toxoplasma gondii*, and dogs and some other canines have been identified as final hosts. Cattle become infected after ingesting *N. caninum* oocysts. Persistently infected cattle can cause foetal infections in successive pregnancies.

Figure 11.8. Young non-immune cats and other felidae are responsible for the excretion of *Toxoplasma gondii* oocysts that infect sheep and other mammals.

Figure 11.9. Twin abortion with a full-term foetus and a mummified foetus of 3 to 4 months of gestation.

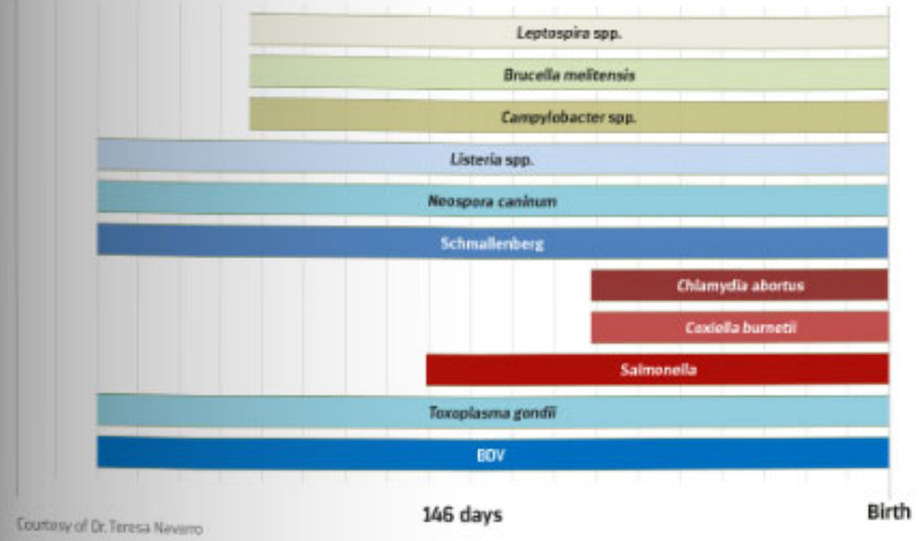
Figure 11.10 A. and 11.10 B. Delivery of mummified foetuses and numerous small white foci of necrosis of 1-3 mm in diameter on placental cotyledons while the intercotyledonary allanto-chorion appears normal, are typical features of an infection with *Toxoplasma gondii*.

Abortions due to *N. caninum* have also occasionally been reported in small ruminants.

Mycotic abortions

Aspergillus fumigatus and other fungal causes sporadically cause mainly late-term abortion in pregnant ewes.

Figure 11.11. **Abortion chronology**



Courtesy of Dr. Teresa Navarro

Livestock farmers should seek maximum profitability although applying sustainable procedures and respecting animal welfare. In order to achieve this profitability, herd productivity must be maximum. High productivity performance depends on the assessment of both male and female fertility, reducing non-productive days and increasing reproduction and production rates. Removing rams that are infertile or present subfertility can substantially increase farm profitability.

Infertility or subfertility may be temporary or permanent. Temporary fertility problems are more common than permanent fertility problems.

Infertility and subfertility may be caused by several factors (figure 13.1).

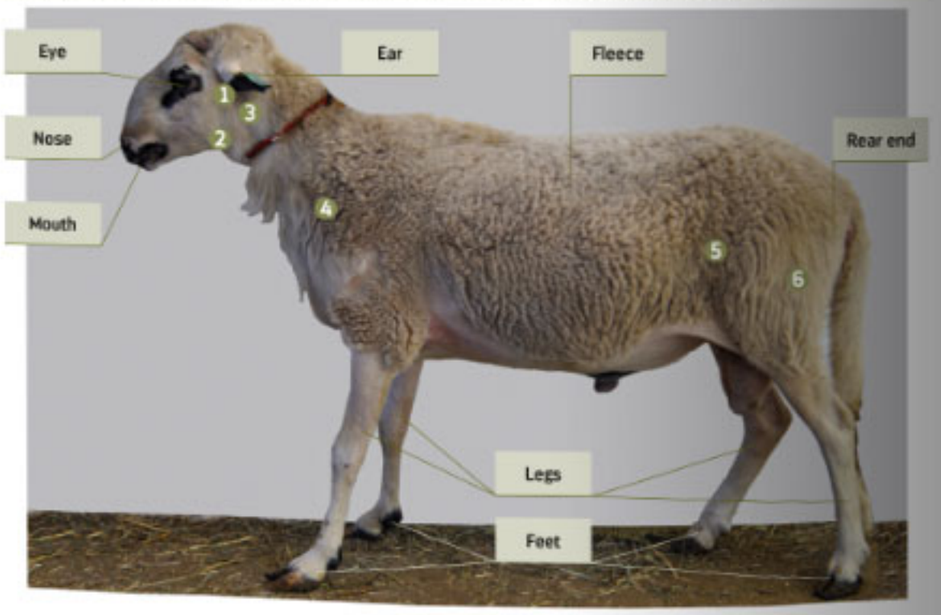
Breeding soundness examinations (BSE) allow to estimate males' reproductive ability, identify fertility problems, and decide a course of treatment. It should be performed at different times in a ram's life (figure 13.2).

A BSE examination includes:

1 A complete clinical exam.
2 A thorough examination of the reproductive tract.
3 A semen assessment.
4 A libido evaluation.

Figure 13.2. Moments when to perform the breeding soundness examination

Figure 13.3. Main body structures to inspect during a general examination of a ram. Body localization of lymph nodes to be felt during a general assessment (green circles)



Clinical exam

Anamnesis is an important part of the evaluation of the reproductive ability of rams and herds. It allows to identify matters and situations of the past and understand the owner's expectations of their rams. When existing data records concerning the herd and rams should be consulted, as the owner's perception is often misleading.

A **physical examination** includes a complete observation of all conditions that might interfere with the ram's breeding ability: body temperature, body condition, structural correctness, lymph nodes, fleece size and wool cover on the testis, skin, and rear end (table I and figure 13.3).

Table I. Body feature to be evaluated during a general clinical assessment

FEATURES	DESCRIPTION
Body condition	Body condition should be assessed by feeling the muscle and fat along the backbone between the last rib and the front of the hip bones. The body condition score may be rated on a scale of 1 to 5, with 1 being emaciated and 5 being extremely fat.
Structural correctness	The likely source of the aetiological agent and how it is being spread.
Head	Should be held up. Head shaking, hunching, hiding, or tucking may be a sign of illness or injury. Verify horns growth.
Ears	Verify the ears for discharges or odour. Be aware of the ears' normal position. Any change may indicate distress or illness.
Eyes	They should be bright, clean, alert. Check out for cloudy, watery, dry, swollen, or constantly blinking eyes since they may indicate illness or injury blindness. Verify for entropion and wool blindness.
Nose	The snout should be free of any discharge, fluid, crustiness, or blood. The nose should be soft and not cracked. An excessively runny or blocked nose may be a symptom of nasal bots or an upper respiratory infection. Respiration should be assessed at rest from a distance since mild changes in the respiratory pattern may not be apparent when the ram becomes alerted and demonstrates herd behaviour. The breath should be silent and smooth. An adult ram should have between 12-20 breaths per minute. Sometimes it may be important to compare herd members' respiratory rate as their rate increases when they are hot or active. A breathing-diminished capacity may be a result of lungworms or pneumonia.
Mouth	The mouth should not have any sores, abscesses, or scabs. Gums should not be red. The jaw should not be swollen or enlarged - bottled jaw. The tongue should not be swollen or bloody. Teeth health should be verified. All these problems may decrease food ingestion.
Legs and feet	The rams should be able to put his full weight on his legs and feet. Since rams herd together and they mask mild signs of lameness in the presence of a stranger, normal stance and gait should be assessed at a distance. Look for ease of rising, normal stance, and smoothness of gait. Check out for signs of arthritis, weak pasterns, muscular atrophy, or feet problems. Hooves should be trimmed and free of cracks, swelling, debris, or abscesses. Any of these problems can cause lameness, discomfort, infections, and footrot.
Lymph nodes	The size and firmness of the peripheral lymph nodes should be exam starting with the parotid nodes and moving to the submandibular, retropharyngeal, prescapular, prefemoral, and inguinal nodes. Enlargement of one lymph node may indicate a reaction to a local infection or inflammation, whilst the involvement of several nodes may indicate systemic infection.
Fleece size and wool cover on the testes	The fleece should be relatively uniform as the wool loss may be a sign of parasites or mineral deficiency or been caused by high fever or severe stress. Fleece size impact on ram's reproductive performance depends on environmental conditions. Under hot and high humidity conditions the fleece should be shear to prevent overheating, food intake reduction, libido, and spermatogenesis impairing or attracting infections. Scrotal shearing improves local thermoregulation, which affects sperm production and the quality of stored spermatic cells, and reduces the risk of *Dermatophilus congolensis* infection. If animals are indoors and lying on a deep bed of straw for long periods, spermatogenesis can be compromised.
Skin	To check out the health of the skin, move away the wool. The skin should be bright, flexible, and free of external parasites, abscesses, wounds, blisters, dry patches, or pressure sores.
Rear end	The rear end under the tail should be relatively clean and free of parasites, discharges, excessive accumulations of faecal matter, crusts, and blood. The anus should not be irritated or prolapsed.

White line lesions

White line lesions include conditions known as white line disease, shelly hoof, shelly toe, toe and white line abscess (if pus is present).

- White line lesions are defects in the white line region of the sole on the abaxial solar surface of the hoof in the soft, non-pigmented, white horn between the sole and hoof wall (figure 14.10).
- The defects vary from small discrete lesions to extensive separation of the hoof wall from the underlying insensitive laminae (figure 14.11).
- Impaction of these pockets of separation with soil and bedding may occur.
- White line lesions do not usually cause lameness unless infection and pus formation has occurred in the deeper sensitive tissues, causing toe or white line abscesses (figure 14.12).
- Pus from these abscesses may track up the wall laminae and emerge at the coronary band where hair loss, fluid swelling or a discharging purulent material maybe observed (figure 14.13).

Figure 14.10. White line disease.

Figure 14.11. White line disease with marked separation.

White line lesions may occur in all ages of sheep, they are not always associated with lameness and the cause of the condition is not known.

Septic pedal arthritis

Septic pedal arthritis is sporadic but common cause of severe, non-weight bearing lameness in sheep.

- Animals are severely lame.
- The foot is swollen.
- The interdigital space is widened.
- Purulent discharging sinus maybe present on the abaxial coronary band (figure 14.14).

Bacterial infection of the distal interphalangeal joint is considered to occur as consequence of extension of interdigital infection into the distal interphalangeal joint structures. Pus then tracks abaxially across the distal interphalangeal joint to emerge at abaxial coronary band.

Figure 14.12. White line abscess.

Figure 14.13. White line abscess tracking to the coronary band.

Figure 14.14. Septic pedal arthritis.

Figure 14.15. Traumatic injury to hoof.

Figure 14.16. Soil balling.

Trauma

Damage to the hoof wall or sole will be obvious, however in chronic cases the initiating cause may be difficult to determine due to secondary infection and/or the proliferation of inflamed and granulomatous soft tissue (figure 14.15).

Sharp objects and over-zealous hoof trimming are often culpable for traumatising the hoof wall.

Thorns

- The thorn may be visible protruding from the ventral aspect of the horn capsule or there just be a dark pin-prick mark visible where it has broken off or fallen out.
- Sheep are likely to have significant lameness (10/10) while a thorn is still present in the ventral aspect of the hoof. Lameness may decrease shortly after the thorn drops out but then increase again if infection is present and starts to build up in the hoof capsule to form an abscess.

Pastures that contain plants with sharp thorns are potentially problematic in pre-disposing soft lambs' feet to being punctured.

Soil balling / snow balling

- Hard lumps of material are found accumulated in the interdigital space (figure 14.16).
- Excoriation of the skin and pressure on the digits cause pain.
- Interdigital dermatitis may result.

Wet or muddy or snowy conditions can pre-dispose to accumulation of material between the digits.

Interdigital hyperplasia

- Skin folds at the skin horn junction in the interdigital space enlarge and become inflamed.
- Sheep may or may not be lame.

Large interdigital skin folds maybe hereditary. Mechanical trauma and secondary bacterial infection exacerbate inflammation, pain and lameness.

Post-weaning growth retardation

Weaned lambs on pasture

After the peak in milk production, sheep farmers have to decide whether they keep supplementing ewes with concentrates or start feeding the lambs directly, using creep feeding systems. Weaning age depends on several factors. Weaning should be performed in such a way that growth retardation is avoided as much as possible.

After weaning, lambs should be fed grass of the best quality available. The optimal growth of grass is reached when its height is about 8-12 cm. Beyond this length, dying leafs prevent the sun from accessing the underlying plant parts, and this will result in a decline in overall production. Information about the digestibility of forages can be found in table IV. Stocking rate of weaned lambs should be adapted to the amount of grass available. Table VI gives an overview of grass production depending on the height of pasture grass. Consequently, knowing the area the lambs are grazing, the features of the grass, and the requirements, make evaluation of adequacy of the stocking rate possible.

Table VI. **Production of grass in kg DM/hectare, depending on the height of pasture grass (source: "Feeding the Ewe" (AHDB, 2018))**

HEIGHT OF PASTURE GRASS (CM)	KG DM/HECTARE
2	900
3	1,200
4	1,500
6	1,650
8	2,000
9	2150
12	2,500

Weaned lambs kept indoors

Weaned lambs kept indoors are almost always fed roughage and concentrates. If lambs do not grow well under these circumstances, quality and quantity of both should be checked. If feed intake is too low, palatability should be checked. Lambs should have fresh water available, preferably from birth onwards.

In lambs kept indoors, a number of metabolic disorders can occur:

Subacute ruminal acidosis (SARA). Its occurrence is promoted by:

- Excess of readily fermentable carbohydrates.
- Quick intake of concentrates as a consequence of competition for feed and high stocking rate.
- Bad management of feed supply.
- Heat stress.

Lambs with this disorder can show some of the following symptoms:

- Low intake of concentrates.
- Thinner animals than normal.
- Higher morbidity and mortality rates.
- Sometimes diarrhoea or lameness.

Urolithiasis. Its occurrence is promoted by several predisposing factors:

- Incorrect calcium-phosphorus balance in the diet, normally an excess of phosphorus; the calcium-phosphorus ratio should be higher than 2:1.
- Vitamin A deficiency.
- SARA.
- Inadequate water supply in combination with high stocking rate.
- Bad water quality.
- High cation-anion balance in the diet.

In lambs kept indoors, the following environmental conditions may adversely affect animal health, animal welfare, and performance of animals:

- A high stocking rate.
- Bad quality of bedding: bedding should be abundant, dry and clean.
- Bad and insufficient ventilation.
- Social stress: mixing of animals from different origins leads to social stress.
- Heat stress: usually the temperature humidity index is used to measure heat stress.

Trace element deficiencies

Trace elements are essential for health and productivity, but their exact role is not always clear. The most common trace element deficiencies in lambs are those of cobalt, copper and selenium.

Deficiencies of a number of trace elements have been mentioned as cause of inadequate birth weight, however evidence to support this is thin. Cobalt/vitamin B_{12} deficiency during pregnancy may negatively influence feed utilisation, however in several countries where deficiency in growing lambs is a problem in autumn and winter, deficiencies in pregnant ewes are not existent or play a subordinate role.

Cobalt/vitamin B_{12} deficiency is an important cause of ill-thrift in lambs in many countries in the world. Microorganisms in the rumen require dietary cobalt for the synthesis of vitamin B_{12} which acts catalytically in the activity of the two mammalian enzymes, methylmalonyl-CoA mutase and methionine synthase. Insufficient supply of dietary cobalt leads to vitamin B_{12} deficiency, which in lambs is characterised by growth retardation, loss of weight, serous ocular discharge, acute photosensitisation, and reduced immune reactivity (figures 18.4 to 18.7). Growth retardation can start from two months of age onwards.

The main consequences of deficiencies of copper, cobalt, iodine, manganese, zinc and selenium are summarised in table VII.

Figure 18.4. Twin ram lambs: the left ram lamb was cobalt/vitamin B_{12} deficient, the ram lamb on the right hand was cobalt supplemented.

Figure 18.5. Watery discharge from the eyes in the acute phase of cobalt/vitamin B_{12} deficiency.

Figure 18.6. Hepatogenous photosensitisation as a consequence of severe cobalt/vitamin B_{12} deficiency.

Figure 18.7. A lamb recovering from hepatogenous photosensitisation as a consequence of cobalt/vitamin B_{12} deficiency.

Table VII. **An overview of the main clinical signs of trace element deficiencies in lambs and sheep**

TRACE ELEMENT	CONSEQUENCES OF DEFICIENCY
Copper	Loss of wool pigment, steely wool, swayback, abnormal bone formation, anaemia, growth retardation
Cobalt	Ill-thrift, photosensitisation, anorexia, emaciation, listlessness, anaemia
Iodine	Goitre, poor wool growth, hair loss, infertility, late abortion, birth of weak or dead lambs, reduced weight gain
Manganese	Deficiency seldomly in sheep: swollen joints, stiff gait, ataxia. Retarded growth only in situations of extreme deficiency
Zinc	Deficiency seldomly in sheep: wool loss, parakeratosis, growth retardation
Selenium	Usually both selenium and vitamin E deficiency. White muscle disease (most important) and infertility. For body weight loss, extreme deficiency required