Respiratory diseases induced in small ruminants by viruses and mycoplasma*

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Summary: In this paper the primary virus and mycoplasma invaders of the respiratory tract of sheep and goats are considered, including those which do not produce clinically apparent disease.

Among acute viral infections, the widespread occurrence of parainfluenza 3 virus and the established findings that it allows other more sinister organisms to invade the lower respiratory tract would indicate that it is a virus of considerable economic importance. Little is known about the significance of infections caused by adenoviruses and reoviruses. Peste des petits ruminants (PPR), caused by a rinderpest-like virus, is of considerable economic importance in West Africa. The herpesvirus of infectious bovine rhinotracheitis (IBR) is capable of infecting goats and sheep. Infections caused by pox viruses and the border disease virus are not primarily respiratory diseases.

Sheep pulmonary adenomatosis, maedi-visna and retrovirus infection in goats are the main virus infections associated with progressive pneumonias of small ruminants.

At present six identified species of mycoplasma have been recovered from the respiratory tracts of animals. Though one agent

INTRODUCTION

Respiratory problems are common in all species and many agents have now been recovered from the respiratory tracts of animals. Though one agent

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may be the primary invader, most respiratory infections are complicated by the presence of secondary or opportunist organisms. Indeed these secondary organisms may be those which cause the most severe lesions or result in a fatal outcome.

In this paper the primary virus and mycoplasma invaders of the respiratory tract of sheep and goats are considered. Some may be capable of infecting without producing clinically apparent disease. The term «disease» has been loosely interpreted therefore and information on agents has been included even if their pathogenicity for the respiratory tract is limited.

I. — ACUTE VIRAL INFECTIONS

A. PARAINFLUENZA 3

There is no doubt that infection with the virus of parainfluenza type 3 (PI 3) is one of the most prevalent in the world.

The first isolation of this Paramyxovirus from sheep was made by Hore (1966). Since then evidence of infection in sheep, by the isolation of virus or the presence of antibodies, has been recorded in many countries and it is probable that the virus is present in most, if not all countries.

The percentage of sheep showing antibodies varies according to each survey and the country in which the survey has been carried out. In general a high percentage of sheep show antibodies to this virus, for example over 70% of sheep sera contain antibody in the U.S.A. (Fischman, 1967), France (Faye, Charton and Le Layec, 1967), Turkey (Erhan and Martin, 1969), Australia (St. George, 1971) and South Africa (Erasmus, Boshoff and Pieterse, 1967). Clearly infection with PI 3 is both frequent and widespread in sheep.


The clinical signs of infection with PI 3 probably go undetected in the field in most cases though signs of a mild infection, mainly of the upper respiratory tract, may occasionally be seen (Hore et al., 1968; Belak and Palfi, 1974a). Clinical signs following experimental infection are variable but combined intratracheal and intranasal inoculation can produce transient pyrexia, nasal discharge, coughing and other respiratory signs (Hore and Stevenson, 1969).

The relatively mild respiratory disease produced by experimental infection of specific pathogen free (SPF) lambs with PI 3 is markedly altered when Pasteurella haemolytica of biotype A is given by aerosol, about seven days after the initial viral infection (Sharp et al., 1978; Davies et al., 1977). Combined infections of this type will result in a high percentage of lambs developing moderate to severe pneumatic lesions in which high counts of pasteu-
relia can be found. Pasteurella haemolytica without a previous PI 3 infection does not generally produce a high percentage of severe disease when given in an aerosol.

It seems probable that infection in the field situation with PI 3 is one way in which biotype A Pasteurella haemolytica can infect the lungs of sheep to produce outbreaks of respiratory disease (Gilmour, 1978).

Immunity to PI 3 virus is not absolute so that nasal shedding of virus may follow infection even in sheep with moderate levels of circulating antibodies.

2. Pathology.

The apical lobes are most frequently affected. Usually small red, linear areas of consolidation are present. On cross-section the consolidated lesions are distributed extensively and follow smaller bronchi and bronchioles. Pseudo-epithelialization of alveoli, hyperplasia of bronchiolar epithelium, infiltration of alveolar septa and acidophilic cytoplasmic inclusions in bronchiole and alveolar epithelial cells are evident microscopically (Hore and Stevenson, 1969).

3. Diagnosis.

Serum neutralization (SN), haemagglutination-inhibition (HI) and ELISA tests are used to determine evidence of previous infection. Generally a four-fold rise in HI titre is taken as indicating recent infection but such a rise does not always occur despite evidence of infection confirmed by recovery of virus in nasal secretions. Virus can only be isolated from lungs for up to 7-10 days following infection.

4. Immunity and control.

PI 3 infection is usually considered to be relatively mild and unnecessary to control. However the complications which may follow invasion by other organisms has led to an increasing interest in PI 3 vaccines. Live attenuated vaccines and subunit vaccines are likely to become available for sheep.

Experimentally live PI 3 virus administered intranasally will stimulate the production of specific IgA antibody into nasal secretions (Smith et al., 1975).

5. Economic significance.

The widespread occurrence of this virus and the established findings that it allows other more sinister organisms to invade the lower respiratory tract would indicate that it is a virus of considerable economic importance.

B. OTHER PARAMYXOVIRUSES AND ORTHOMYXOVIRUSES

There is no confirmed evidence that either of the Paramyxoviruses, parainfluenza 1 and 2, is an important pathogen of sheep. Because of the
heterologous reactions which may occur between members of this group some doubt exists as to the value which can be placed on surveys which suggest that antibody to these viruses is present in sheep.

Sheep do not appear to be important reservoirs of influenza virus, though evidence of infection has been reported from Hungary (Romvary et al., 1962). Experimental infection of sheep with influenza A did not result in clinical illness (McQueen and Davenport, 1963).

C. ADENOVIRUS

All serotypes of mammalian adenovirus share a common group-specific antigen which can be detected by complement-fixation (CF) and gel-diffusion (GD) tests. These tests have been used to show that adenovirus infection occurs in sheep. The first isolation of adenovirus from sheep was reported in 1969 by McFerran et al.

Surveys carried out in a number of countries have shown a variable percentage of sheep with antibodies: Iran: 14% (Ashfar, 1969), Greece: 31% (Pashaleri-Papadopoulou, 1968), but less than 1% in Britain (Darbyshire and Pereira, 1964) and the Republic of Ireland (Timoney, 1971).

Five serotypes of sheep adenovirus which can be distinguished by SN tests, have been isolated, though several isolates are as yet untyped. A survey in Scotland has shown the following percentage of sheep with antibodies to 4 types: type 1: 20.8%, type 2: 40.4%, type 3: 67.8% and type 4: 71.1% (Sharp, 1977). The high percentage of infection found in this survey contrasts with the low prevalence in the earlier British surveys. The use of different test methods (viz. GD and SN) is probably the major reason for the differing results.


Little is known about adenovirus infections in sheep. Adenoviruses have been isolated from apparently healthy sheep (Bauer et al., 1975) and from lambs with respiratory signs (Sharp, 1977). It is not clear what natural infection with adenovirus may produce and experimental infection has generally not resulted in notable clinical disease, though the virus may replicate and produce lesions. In experiments described by Sharp et al. (1976) virus was recovered following infection (p.i.) from nasal swabs (days 1-8 p.i.) and rectal swabs (days 3-9 p.i.). Even when experimental adenovirus infection is followed by an aerosol of Pasteurella haemolytica biotype A, no serious disease results (Sharp, 1977; Davies et al., 1981).

2. Pathology.

Ovine adenovirus type 4 has been shown to be capable of causing pulmonary oedema and mild hepatic lesions (Sharp et al., 1976; Rushton and
Sharp, 1977). Histology of the lungs showed accumulation of fluid in perivascular and peribronchiole areas. In the liver focal necrosis was evident and basophilic inclusion bodies were present in hepatocytes and lymphatic endothelial cells (Rushton and Sharp, 1977).

Virus has been recovered up to 14 days p.i. from tissues in the respiratory and enteric tracts of lambs infected experimentally, which developed areas of atelectasis and in some consolidation of the lungs. Microscopically there was proliferative bronchiolitis and bronchopneumonia with cytomegaly and karyomegaly of epithelial cells. In some nuclei basophilic inclusions were seen containing crystalline arrays of virus particles (Davies et al., 1981).

3. Diagnosis.

Serum antibody can be determined by SN tests and some strains of virus will agglutinate red blood cells of certain species. Rising titres are indicative of recent infection but the isolation of virus is confirmatory of infection.

4. Immunity and control.

Colostral antibody levels decline progressively and lambs are free from such antibody after 3-5 months. New infections can then result in active immunity and at the same time a concomitant increase in pneumonia has been noted in the lambs in such flocks suggesting rapid spread of infection through a susceptible population (Davies et al., 1980).

Vaccination of pregnant ewes has been suggested to provide passive immunity for lambs during the first few weeks of life (Palfi et al., 1980). Successful vaccination of lambs using U.V. inactivated virus adsorbed onto aluminium hydroxide has been reported but the immune response was significantly reduced by the presence of colostral antibody (Palfi et al., 1980).

5. Economic significance.

At present the importance of adenovirus infections in sheep is not known. The work of Davies et al. (1980) suggests that adenovirus causes varying degrees of pneumonia and could therefore result in economically significant loss of productivity.

6. Infection of sheep with bovine adenovirus.

The isolation of a type 2 bovine adenovirus from sheep has been reported (Belak and Palfi, 1974b). This virus was isolated from lambs with enteritis and pneumonia, some of which died. Inoculation of the virus into lambs produced clinical respiratory disease and enteritis, similar to the disease seen in the original outbreak (Belak et al., 1975).

7. Adenovirus infections of goats.

Two serologically different adenoviruses have been isolated from goats with « peste des petits ruminants » in Nigeria. These isolates are not related
to known ovine or bovine adenoviruses, and should probably be considered as two caprine serotypes of adenovirus. Both isolates were considered to be unassociated with the disease (Gibbs et al., 1977).

D. REOVIRUS

Reoviruses have been isolated from several species of domestic animals but their relationship to disease is doubtful.

Antibodies to reovirus have been found in sheep sera. In one small survey 96% of sheep sera had HI antibodies to reovirus type 3 and 15% to types 1 and 2 (Stanley et al., 1964). SN antibodies to type 3 were shown to be IgG by Pringle and Cartwright (1969) who concluded that reovirus infection was widespread in sheep in Scotland. Antibodies to reovirus type 3 were present in 46% sheep sera in Northern Ireland but no antibodies to the other types were found (McFerran et al., 1973).

Type 3 reovirus was first reported as isolated from sheep in 1973 (McFerran et al., 1973), type 1 in 1974 (Belak and Palfi, 1974c) and type 2 in 1976 (Snodgrass et al., 1976).


Type 1 was recovered from lambs with respiratory and enteric disease (Belak and Palfi, 1974c). When the virus was inoculated intratracheally and intranasally into lambs, fever, enteritis and respiratory signs were evident (Belak and Palfi, 1974d). In contrast no disease or lesions were produced following the inoculation of type 3 into lambs, though the virus replicated and produced a serological reaction (McFerran et al., 1974).

2. Pathology.

The lesions described following experimental infection with type 1 were mainly diffuse interstitial pneumonia and alveolar collapse (Belak and Palfi, 1974d).

3. Control and economic significance.

There is no substantial evidence that reovirus produces a sufficiently serious infection in sheep to require control measures or that it is of economic importance.

E. RESPIRATORY SYNCYTIAL VIRUS (RSV)

The first reported evidence of RSV infection in sheep came from Canada where Berthiaume et al. (1973) recorded the presence of complement-fixing antibodies in 81% of 31 sheep. Smith et al. (1975) also found SN antibodies
in sheep in U.S.A. No antibodies to RSV were present in an intensive flock in Britain where respiratory disease was a constant problem (Sharp, 1977).

RSV is a difficult virus to isolate but to date no report exists of its isolation from natural infections in sheep.

Lehmkuhl and Cutlip (1979) inoculated a bovine strain of RSV into lambs and produced a transient, mild disease with fever and serous nasal discharge. Post-mortem examination showed mild interstitial pneumonia. From 2/5 lambs virus was recovered from nasal or tracheal secretions or lung tissue.

**RSV in goats.**

A caprine RSV has been isolated from pygmy goats in America during an outbreak of severe respiratory disease (Lehmkuhl and Smith, 1980). The clinical signs observed in the goats during the outbreak included severe coughing, oculo-nasal discharge, corneal opacity and fever. The virus, which was isolated from the ocular and nasal secretions, was closely related antigenically but considered to be distinct from bovine RSV.

**F. ENTEROVIRUS**

Of the viruses classified within the 3 genera of the *Picornaviridae* viz. Enterovirus, Rhinovirus and Calicivirus only enteroviruses have been isolated from sheep (McFerran *et al.*, 1969; Snodgrass and Sharp, personal communication). At least one isolate differs from known bovine enteroviruses. Though sheep enteroviruses have been isolated from lambs with diarrhoea no disease has been attributed to these viruses following experimental inoculation. No serological surveys on the prevalence of the picornaviruses in sheep appear to have been published.

**G. PESTE DES PETITS RUMINANTS VIRUS (SYNONYM—KATA)**

Peste des petits ruminants (PPR) is a rinderpest-like virus disease affecting sheep and goats which is recognised in West Africa.

Although not primarily a respiratory disease respiratory signs can occur during the acute phase of infection. Bronchopneumonia is a frequent complication which may be associated with secondary infection by *Pasteurella* (Rowlands *et al.*, 1969). Histology of the lung shows a giant cell pneumonia and occasionally eosinophilic inclusion bodies in the epithelial cells of the respiratory tract.

The virus of PPR has been isolated in lamb kidney cells and was shown to be distinct but related to the virus of rinderpest (Taylor and Abegunde, 1979).

The economic importance of this disease in West Africa is considerable.
H. HERPESVIRUSES

The herpesvirus of infectious bovine rhinotracheitis (IBR) appears to be capable of infecting goats and sheep.

IBR virus has been isolated from a single lamb « suffering from a respiratory disorder » (Trueblood et al., 1978). Natural infection with IBR virus apparently caused severe respiratory disease and keratitis in 2 goats, from the eyes and nose of which virus was recovered (Mohanty et al., 1972).

Antibody to IBR virus has been found in goats and sheep, particularly the former. Of 561 goats surveyed in Chad, 13% had antibodies to IBR virus (Maurice and Provost, 1970); 12% of goats had antibody in Tanzania whereas sheep were negative (Jesse and Rampton, 1975), and in a survey in Nigeria 11.2% of goats had antibody whereas only 2.9% of sheep showed evidence of infection (Taylor et al., 1977).

A further herpesvirus, apparently distinct from IBR, has been isolated from goat kids with a generalised fatal infection in which lesions were confined to the alimentary tract (Saito et al., 1974).

On intranasal inoculation into adult female goats fever was produced and virus could be recovered from nasal secretions. In kids intranasal inoculation resulted in fever, oculo-nasal discharge and bronchial sounds. This herpesvirus was not pathogenic when inoculated into lambs and calves (Berrios et al., 1975).

I. POXVIRUSES

Although sheep pox and goat pox are not primarily respiratory diseases, lesions can apparently occur in the lungs of severely affected animals. Borrel (1903) described hard, dense, hyaline nodules of varying size in the lungs of sheep with sheep pox. The characteristic sheep pox cells, the « cellules claveleuses » may be found in such lesions as in the skin. Lung lesions occur in many cases of sheep pox and inhalation is thought to be a main route of infection (Davies, 1976).

It is unlikely that sheep pox or goat pox would be recognised by the respiratory signs which may occur but are more likely to be diagnosed by the more obvious skin lesions.

Occasionally, in a badly affected case of contagious pustular dermatitis (« orf ») in sheep bronchopneumonia may occur, but this may be the result of the inhalation of large numbers of bacteria from orf lesions around the nostrils and mouth where these are extensive and severe.

J. BORDER DISEASE VIRUS

The Pestivirus which causes this disease is related antigenically and in other characteristics to the viruses of mucosal disease - virus diarrhoea and
swine fever (hog cholera). Although border disease is not primarily a respiratory disease, four clinico-pathological syndromes are recognised in one of which pneumonitis may occur together with lymphoid infiltrations in a wide range of organs (R.M. Barlow, personal communication).

II. — VIRUS INFECTIONS ASSOCIATED WITH PROGRESSIVE PNEUMONIAS

A. SHEEP PULMONARY ADENOMATOSIS (JAAGSIEKTE)

Sheep pulmonary adenomatosis (SPA) is a contagious tumour of the lungs of sheep. SPA has been reported from over 20 countries and most continents, but not from Australia or New Zealand.

Tumours have been found in many breeds and no clear association with any breed or sex of sheep has been established (Martin et al., 1979). No definite information is available on the prevalence of this disease in sheep in any country, probably because satisfactory diagnostic tests have not been available.

Two viruses have been associated with pulmonary adenomatosis: a herpesvirus (Mackay, 1969 a & b) (sometimes called Herpesvirus ovis or bovid herpesvirus 4) and a retrovirus (Perk et al., 1974), both of which may be involved in the production of the tumours (Martin et al., 1979). The herpesvirus has been isolated from the lung tumour of sheep in several countries: Britain (Mackay, 1969 a & b), Kenya (Malmquist et al., 1972) and South Africa (Verwoerd et al., 1979). The close association of this virus with the tumour is thus established. Its precise role, if any, in the development of the tumour has not been elucidated however. No correlation was found between genome sequences of the herpesvirus and the presence of the tumour (De Villiers and Verwoerd, 1980).

The retrovirus has not been isolated in cell cultures but its presence in tumours has been detected by the determination of an RNA-dependant DNA polymerase (reverse transcriptase enzyme) in virus particles with other characteristics of a retrovirus such as a buoyant density of 1.15-1.20 g/ml and 60-70 S RNA (Perk et al., 1974). It seems probable that this virus is the cause of SPA but experimental proof is awaited.


Clinical disease is seldom seen in lambs, but generally in mature sheep of 2-4 years old. The incubation period is long and experimentally is usually longer than 5 months.

Only when tumours become sufficiently large or extensive to reduce normal lung function do clinical signs develop. Signs usually develop therefore in
an individual sheep which shows respiratory disease but has no fever. Exaggerated respiratory movements, which increase on exercise, are evident. Coughing and fever are not features. On auscultation both inspiratory and expiratory sounds are present sometimes accompanied by very obvious adventitious sounds. Loss of weight is marked. Death is the inevitable outcome, sometimes as a result of intercurrent *Pasteurella* pneumonia.

A valuable and diagnostic clinical feature is the discharge of fluid from the nose which occurs when the head is lowered and the hind legs are raised.

2. Pathology.

Tumours of SPA are confined to the lungs and very occasionally to the associated lymph nodes. Ventral areas of the lungs are most frequently affected with the tumours which are solid, grey or light purple. Lungs are usually heavy and a copious quantity of frothy fluid is present in the respiratory passages. Pleurisy is sometimes present over the tumour area.

On histological section the thin alveolar cells are replaced by columnar or cuboidal cells, and in the bronchioles proliferation may be seen (Markson and Terlecki, 1964).

3. Diagnosis.

No satisfactory confirmed diagnostic tests are recognised. Hyperglobulinaemia does occur (Hod *et al.*, 1977) but such alterations in serum proteins can arise from other causes of cachexia.

4. Immunity and control.

Limited observations suggest that antibodies to the herpesvirus are frequently present in the serum of sheep in Scotland (Martin *et al.*, 1979) and in South Africa 60-70% of sheep have antibodies to the herpesvirus (Verwoerd *et al.*, 1979). At present nothing is known about the prevalence of the retrovirus.

Because of the lack of a suitable means of diagnosing subclinical infection control in a flock is based on the early culling of sheep which show respiratory signs or wasting. In such flocks care should be taken to examine all sheep which are over 2 years old, at frequent intervals.

International movement of sheep can result in the introduction of infection into the sheep flocks of a country. As no diagnostic test is available an accurate history of the flock of origin and of the other flocks in the area should be taken and local veterinary advice sought before agreement to the importation of sheep is given.

5. Economic significance.

When the disease first appears in flocks losses may be heavy but endemic infection is considered to cause an annual loss of around 1-2% (Mackay and Nisbet, 1966).
The importance of this disease is probably low in international economic terms, but importation should be avoided as in susceptible flocks losses may be very high (Palsson, 1976).

B. MAEDI-VISNA

Maedi is a progressive interstitial pneumonia and visna a meningo-leucoencephalitis.

Both diseases are slowly progressive, afebrile diseases of sheep caused by closely related or identical viruses belonging to the family Retroviridae, subfamily Lentivirinae. Antigenically distinct strains have been described (Narayan et al., 1978). The virus nucleoid contains reverse transcriptase.

Maedi-visna virus has now spread to sheep in most European countries and is recognised in several countries in Asia and Africa. In the U.S.A. the disease has been termed (Montana) progressive pneumonia and in the Netherlands, « zwoegersiekte » (De Boer et al., 1978). Maedi is histologically similar to the disease described in France as « la bouhite » (Lucam, 1942).

The prevalence of infection is not known in many countries but infection would appear to be increasing in the last decade or so (De Boer and Houwers, 1979). In the U.S.A. evidence of infection has been found in over 40% of sheep sera from the midwest and northwest states (Cutlip et al., 1977). In another survey, in a western range flock of sheep in the U.S.A., 64% had antibodies (Huffman et al., 1981). In the Netherlands one survey indicated that about 30% of sheep had antibodies (De Boer et al., 1978).


Clinical signs of maedi only appear after a long incubation period which varies with the extent of the exposure and may be up to 5 years (De Boer et al., 1978). An affected sheep shows signs of progressive respiratory embarrassment, loss of weight and discolouration of the fleece. Despite the increasing respiratory signs and weight loss, appetite is maintained throughout the course of the illness which can last for several months.

The nervous form of the disease (visna) is encountered much less frequently than maedi. In visna, fine muscular tremors progress to paresis and eventually paralysis.

Hypochromic anaemia and a lymphocytic leucocytosis have been reported in cases of maedi (Palsson, 1976).

2. Pathology.

In the early stages of infection no lesions may be evident in the lungs. However in more advanced cases of maedi the lungs are generally greatly enlarged and heavy (e.g. 2-4 times normal weight). Such lungs feel inelastic.
and have been likened to a plastic or rubber sponge. Some lungs may be pale whereas others are mottled alternatively grey and brown. Secondary bacterial infections can alter the appearance of affected lungs. The associated lymph nodes are very enlarged.

Microscopically there is thickening of the interalveolar septa resulting from infiltration by mononuclear cells and proliferation of the septal cells. A prominent feature is focal hyperplasia of lymphoid tissue around bronchi and vessels in the lung. Gradually the cell infiltrates are replaced by fibroblasts and collagen fibre accompanied by proliferation of alveolar epithelium and smooth muscle tissue so that there is extensive obliteration of alveoli.

3. Diagnosis.

A diagnosis of maedi-visna virus infection may be made on the presence of serum antibodies (preferably GD or ELISA), the isolation of virus (from white blood cells or tissues), or histopathology of the lung. Frequently two or more methods may confirm the presence of infection, but sometimes only a single method gives a positive result.

4. Immunity and control.

Sheep which become infected generally produce antibodies though there may be a delay of several months before antibodies can be detected. The presence of antibody however does not eliminate the virus which can still be recovered from peripheral leucocytes and other tissues. It is important therefore to emphasise that the presence of antibody indicates that the sheep is infected and may transmit infection to other sheep.

Infection is spread by the respiratory route, particularly where contact is close, but also via the milk of infected ewes. Two methods of control have been advocated (De Boer et al., 1978). One is the rearing in isolation of lambs caught at parturition before they are able to suck or obtain colostrum from their mother. The other method relies on serological testing of flocks at intervals of about 6 months with the elimination from the flocks of any positive sheep. In both methods isolation of the clean sheep from the infected flock is essential. Infection is probably not transmitted to the lambs in utero (De Boer et al., 1979).

Because of improved serological diagnostic techniques and greater awareness of this slow virus disease, the international spread of this infection should be reduced in future.

5. Economic significance.

The slow insidious nature of this disease may be disarming but its economic significance should not be underrated.

When maedi-visna infection has become endemic in flocks, losses have sometimes been remarkably high. In Iceland losses were estimated as 15-30%
annually (Palsson, 1976), and in the Netherlands a continuous annual mortality of 15% was noted on some individual farms (Ressang et al., 1968).

**C. RETROVIRUS INFECTION IN GOATS**

An infectious disease of goats known as leukoencephalomyelitis, caprine arthritis-encephalitis (CAE) syndrome or goat leukoencephalitis-arthritis, has been described (Cork et al., 1974; Clements et al., 1980). CAE has been reported in goats in America (Cork et al., 1974), Australia (O’Sullivan et al., 1978) and in Sweden (Sundquist et al., 1981).

CAE is caused by a retrovirus with a virion-associated reverse transcriptase enzyme and antigenic determinants which cross react with the P 30 component of the virus of maedi-visna. It has been suggested that CAE virus is a variant of the maedi-visna virus (Narayan et al., 1980).

1. **Clinical disease.**

   The clinical syndrome may take 3 forms viz.:
   
   a) progressive paresis leading to paralysis which is mainly seen in young goats,
   
   b) chronic progressive synovitis and periarthritis in older goats,
   
   c) both of these syndromes may be accompanied by subclinical interstitial pneumonia, which may also occur alone (Robinson, 1981).

   The disease affects goat kids within the first few months of life. The disease is afebrile and characterised by progressive ataxia leading to paralysis within a few weeks of onset and there is an accompanying interstitial pneumonia of varying extent. It is believed that goats may become infected *in utero* or immediately after birth (Cork et al., 1974).

2. **Pathology.**

   In addition to the lesions of leukoencephalomyelitis in which there is dense perivascular cuffing with monocytes, infiltration of the parenchyma with macrophages, proliferation of glial cells and demyelination, there is interstitial pneumonia with hyperplasia of peribronchiolar lymphoid tissue.

   A somewhat similar disease to CAE, termed granulomatous encephalitis (GE) has been described in adult goats. In addition to the granulomas in the brain this infection may result in chronic interstitial pneumonia and is believed to be caused also by a retrovirus related to maedi-visna (Robinson, 1981). Although certain points of pathological difference exist between CAE and GE the precise aetiological difference, if any, between these two diseases is uncertain.

3. **Control and economic significance.**

   The economic significance of retrovirus infections in goats is not clear at present. However with the increasing interest in goats and a possible increase
in international trade in goats, the presence of these infections must be borne in mind. Serological tests similar to those used for maedi-visna will detect the presence of antibody.

III. — MYCOPLASMA INFECTIONS

The first isolation of mycoplasma from the respiratory tract of small ruminants was reported from sheep in 1955 (Greig, 1955). Since then the number of isolates has increased, particularly in the last decade. At present 6 identified species of mycoplasma have been recovered from the respiratory tract of sheep and goats, not all of which have been associated with disease.

Confirmed isolations of the following species of mycoplasma from the respiratory tracts of sheep and goats have been reported:

- *M. agalactiae* from goats (Cottew and Lloyd, 1965) and sheep (Arisoy et al., 1967).
- *M. arginini* from goats (Arisoy et al., 1967) and sheep (Barile et al., 1968).
- *M. mycoides* subsp. *capri* (« PG 3 ») from goats (Chu and Beveridge, 1949).
- *M. mycoides* subsp. *mycoides* from goats and rarely from sheep (Al-Aubaidi, 1972).
- *M. ovipneumoniae* from sheep (Mackay et al., 1963) and goats (Harbi, 1977).
- *Acholeplasma laidlawii* from sheep (Krauss and Wandera, 1970).

A. *M. OVIPNEUMONIAE*

*M. ovipneumoniae* is probably a world-wide inhabitant of the respiratory tract of sheep and has been isolated from apparently healthy as well as diseased sheep. The isolation of this mycoplasma has been reported from several countries including Australia (Sullivan et al., 1973), America (St. George and Carmichael, 1975), Britain (Foggie et al., 1976), Germany (Stipkovits and Schimmel, 1977), Iraq (Al-Sultan and Zubaidy, 1978), the Sudan (Harbi et al., 1981), Iceland (Friis et al., 1976) and Hungary (Stipkovits et al., 1975).


*M. ovipneumoniae* is considered to be responsible for chronic respiratory infection which progresses slowly. Infection probably starts in lambs shortly after birth having spread from carrier ewes, from which the mycoplasma can be recovered repeatedly by swabbing the nasal mucosa. The number of infected lambs increases progressively till, under intensive conditions, over
90% of the lambs are infected when 3.5-4 months old (Jones et al., 1979). Poor growth, exercise intolerance and even pneumonia have been ascribed to infection with *M. ovipneumoniae* (St. George et al., 1971).

2. Pathology.

The lungs of naturally-infected lambs are stated to be greyish in appearance with red areas of collapse of varying size (St. George et al., 1971). The main histological features are interstitial thickening due to proliferation of septal cells, accumulation of monocytes in alveoli and lymphoid hyperplasia around bronchioles and vessels (St. George et al., 1971). When lambs are exposed experimentally to infection with *M. ovipneumoniae* a proportion develop these changes (Sullivan et al., 1973; Foggie et al., 1976).

Rarely, however, is natural infection uncomplicated. A frequently associated pathogen is *Pasteurella haemolytica* (Thurley et al., 1977). Dual infection with *M. ovipneumoniae* and *Pasteurella haemolytica* of biotype A has been shown experimentally to produce « proliferative-exudative » lung changes (Gilmour et al., 1979), indistinguishable from the « atypical pneumonia » described by Stamp and Nisbet (1963). This is a common disease of lambs less than 1 year old. Acute disease is followed by a chronic condition in which lung lesions and infection with mycoplasma may persist for 7 months or longer (Gilmour et al., in press).

The economic importance of combined infection in lambs is believed to be considerable. In addition to losses due to death of lambs, infected lambs fail to gain weight as rapidly as non-infected control lambs and require to be fed over a longer period before they achieve a satisfactory weight (Jones et al., in press).

3. *M. ovipneumoniae* in goats.

*M. ovipneumoniae* has been isolated from the respiratory tract of goats in Sudan (Harbi, 1977), America (Livingston and Gauer, 1979) and Australia (Cottew, 1980), but the pathogenicity of this organism for goats is unknown.

B. *M. ARGININI*

*M. arginini* is a common inhabitant of the respiratory tract of sheep but its ability to produce pathological changes, as a single infection, is doubtful (Foggie and Angus, 1972; Jones, 1976). It may possibly act synergistically to alter or exacerbate respiratory infections.

C. CONTAGIOUS CAPRINE PLEUROPNEUMONIA (CCPP)

CCPP is one of the most serious and economically important diseases of goats but fortunately sheep are not affected. CCPP has been reported as occurring in 31 countries (McMartin et al., 1980).
The term CCPP is often applied to any mycoplasma pneumonia of goats and considerable confusion has arisen over the precise mycoplasma causing this infection. Several reviews have been written on this disease and its cause (Swift, 1978; Cottew, 1979a; McMartin et al., 1980).

Three mycoplasmas have been isolated from CCPP-type disease, each of which has been considered to be the aetiological agent. These mycoplasmas are \textit{M. mycoides} subsp. \textit{capri}, \textit{M. mycoides} subsp. \textit{mycoides} and an unclassified mycoplasma strain F 38 isolated by MacOwan and Minnette (1976).

\textit{M. mycoides} subsp. \textit{capri} (type strain PG 3) is considered to be the classical causal agent (Cottew, 1979a). Natural infection with \textit{M. mycoides} subsp. \textit{capri} produces a respiratory disease which is confined to goats although both sheep and goats can be infected experimentally but the disease is not contagious.

\textit{M. mycoides} subsp. \textit{mycoides} isolates from goats with pleuropneumonia (e.g. Perreau, 1971, 1979; Bar-Moshe and Rappaport, 1979) comprise two morphological forms, namely small and large colony types (Cottew, 1979b). The small colony types include the classical agent of contagious bovine pleuropneumonia and are experimentally pathogenic for cattle, sheep and goats. The large colony forms appear to be pathogenic only for goats and sheep (MacOwan, 1976; Rosendal, 1981). One strain (F 30) of this organism, not only produced pleuropneumonia in sheep and goats following intratracheal/endobronchial inoculation, but was also shown to cause oedema at the site of subcutaneous inoculation and as the condition was not transmissible by contact, MacOwan (1976) considered that this disease was not classical CCPP.

McMartin et al. (1980) argue that only mycoplasma F 38 is capable of causing classical CCPP. These authors emphasise that strain F 38 can produce disease with the three essential features noted in the original description (Hutcheon, 1881, quoted by McMartin et al., 1980). These three points were:

1. the disease was readily contagious to susceptible goats,
2. sheep and cattle were not affected, and
3. local oedematous reactions did not occur in goats following subcutaneous inoculation.

On the basis of these findings McMartin et al. (1980) consider that only mycoplasma F 38 is capable of causing classical CCPP. This view is supported by the recent isolations of an F 38-type mycoplasma from goats with CCPP in the Sudan (Harbi et al., 1981).

A fourth mycoplasma \textit{M. capricolum} ("pp goat") is pathogenic for goats and for sheep but pneumonia is not a feature of the disease. This organism mainly causes septicaemia and polyarthritis.

After an incubation period of 2-28 days (generally 8-10 days) symptoms of CCPP develop rapidly. Affected goats show fever and acute respiratory distress. Breathing becomes difficult and the affected goat stands coughing, with its mouth open, tongue protruding, drooling saliva and bleating in a distressed way. The course may be short with death occurring within 2 days but frequently the illness continues for 3-4 weeks (Hutyra et al., 1938). Morbidity is generally about 100% and about 50-90% of affected goats may die.

2. Pathology.

Lesions of CCPP are confined to the thorax. In the early stages lungs may show yellow nodules surrounded by zones of congestion but as the disease progresses these coalesce to include large areas of lung. Sero­fibrinous pleurisy develops over the affected area which may involve a whole lobe or even the entire lung. Thickening of interlobular tissue has been described in some cases but McMartin et al. (1980) quote the original description by Hutcheon in 1881 who maintained that no thickening of the interlobular septa occurs. The bronchial and mediastinal lymph nodes are markedly swollen but other organs in the body are normal.

3. Immunity and control.

Classical CCPP is highly contagious and spreads rapidly to other goats but not to sheep and cattle (McMartin et al., 1980). A combination of slaughter, isolation and vaccination has been used to control the disease. Streptomycin given on the third day of fever has improved recovery and such goats are completely immune (Rurangirwa et al., 1981). Tylosin and oxy­tracycline may also be valuable therapeutically. Sonicated F 38 mycoplasma in complete Freund’s adjuvant stimulated HI antibodies and protected goats against in-contact challenge infection (Rurangirwa et al., 1981).

4. Economic importance.

CCPP is of major economic importance where goats form an important sector of the livestock of any area or country.

D. CHLAMYDIAL INFECTIONS

Chlamydia are neither true viruses nor bacteria and have therefore not been considered in this paper. They have however never been shown to be important pathogens of the respiratory tract of sheep in Britain.

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CONSIDERING THAT

more research is required into the syndrome of respiratory diseases of sheep and goats;

it is recognised that certain viruses, for example parainfluenza 3, adenovirus, reovirus and maedi-visna virus may produce clinical disease per se. However, in certain countries or under certain conditions, additional microorganisms, for example bacteria or mycoplasma may be necessary for clinical disease to be produced;

one well-recognised and important disease, contagious caprine pleuropneumonia, which apparently is confined to Africa, is caused by a still unnamed mycoplasma-type organism (F 38);

in general, the cause and epidemiology of much respiratory disease are still largely unknown,

accordingly

THE COMMITTEE

RESOLVES TO RECOMMEND THAT

1. Research efforts should be directed to clarify the aetiology and epidemiology of respiratory disease, aimed at achieving more satisfactory control measures.

2. Veterinary authorities in all countries should be aware of the danger of importing such infections through normal international traffic in live sheep and goats.

3. « Slow virus disease » in particular presents a problem in detection due to the protracted nature of the disease process and the difficulty of determining the presence of infection by serological means. Thus any positive serological result in a flock of sheep should be accepted as indicating the presence of infection in that specific flock.

4. The availability of simple diagnostic methods would facilitate the dissemination of information on the presence of respiratory viruses and mycoplasma and the application of appropriate measures of control.

(Adopted by the International Committee of the O.I.E. on 29 May 1982.)
REFERENCES

I. — Acute Viral Infections


II. — Progressive Pneumonias


### III. Mycoplasmas


