OPA (JAAGSIEKTE): AETIOLOGY, PREVALENCE, CLINICAL FINDINGS, DIAGNOSIS, AND CONTROL IN IRELAND

Seamus Fagan MVB, Veterinary Research Officer, DAFM Regional Veterinary Laboratories, Athlone, reviews the aetiology and pathogenesis of ovine pulmonary adenocarcinoma (OPA), describes clinical and post-mortem findings, and provides guidance on diagnosis, prevention, and flock-level control. Particular emphasis is placed on the epidemiological situation in Ireland and the diagnostic challenges, including the role of transthoracic ultrasound scanning alongside the gold standard of post-mortem confirmation

OPA, also known as Jaagsiekte or ovine pulmonary adenomatosis, is a contagious, progressive, and invariably fatal neoplastic lung disease of sheep caused by the Jaagsiekte sheep retrovirus (JSRV). Classified as one of the five 'iceberg diseases' of sheep, OPA is characterised by a prolonged subclinical phase during which infected animals shed virus and transmit infection to flock-mates long before clinical signs appear. The disease is endemic in the Republic of Ireland and the UK, causing significant economic losses through mortality, premature culling, and reduced productivity.

'Iceberg disease'

OPA is a disease of considerable concern to sheep farmers and veterinary practitioners throughout Ireland and the UK. First described in South Africa in the late 19th century, the disease has since been recognised in most sheep-rearing countries worldwide, with notable exceptions such as Australia and New Zealand[1,2].

The term "Jaagsiekte" is derived from Afrikaans, meaning "driving sickness," referring to the respiratory distress affected animals display when driven or gathered[3]. The disease is caused by the Jaagsiekte sheep retrovirus (JSRV), an exogenous betaretrovirus that induces malignant transformation of the secretory epithelial cells of the lung[4]. The resulting adenocarcinoma is invariably fatal, with no current treatment or licensed vaccine[5].

OPA is classified among the five ovine 'iceberg diseases'—alongside Maedi-Visna, Johne's disease, caseous lymphadenitis, and border disease—because clinically affected animals represent only the visible 'tip' of a larger problem within the flock[6]. The long incubation period and lack of a reliable commercial ante-mortem diagnostic test mean OPA can become deeply established before it is recognised. This review provides veterinary practitioners in Ireland and the UK with a comprehensive account of the disease and guidance on its management.

Aetiology and Pathogenesis

The Causative Agent

OPA is caused by JSRV, a betaretrovirus genetically distinct from the ovine lentiviruses responsible for Maedi-Visna[4]. The ovine genome contains approximately 20 copies of endogenous betaretroviruses (enJSRVs) closely related to the pathogenic exogenous JSRV[4]. Their expression is thought to induce immunological tolerance to exogenous JSRV, explaining why infected sheep fail to mount a detectable humoral immune response[7]. This has profound implications for diagnosis, as serological testing is not possible.

A defining feature of JSRV is that its envelope (Env) protein functions as a direct oncogene[4]. Following infection, the Env protein activates intracellular signalling pathways—including PI3K/Akt and Ras/MAPK—which drive the uncontrolled proliferation of type II pneumocytes and Club (Clara) cells in the distal airways[4,8]. This virally-driven neoplastic transformation causes the characteristic lung tumours.

Transmission

The primary route of JSRV transmission is via aerosol inhalation of infectious respiratory droplets[5]. The virus is present in large quantities in the lung fluid and respiratory secretions of affected animals, with sheep with advanced tumours being the greatest source of infection[9]. However, subclinically infected sheep may also shed JSRV and contribute to transmission[9].

Additionally, JSRV can be transmitted from ewe to lamb via colostrum and milk[9]. Young lambs are highly susceptible, and studies show they can be infected within weeks of birth via this route[1]. Conditions promoting close contact—indoor housing, trough feeding, and high stocking density—greatly facilitate viral spread[9], which is particularly relevant for winter housing in Irish and UK sheep farming.

Situation in Ireland and UK

Prevalence and distribution

In the Republic of Ireland, OPA is a notifiable disease[13]. An abattoir survey, by Lee *et al.* in 2017, of 1,911 adult sheep lungs from 18 counties confirmed OPA in 0.5 per cent of animals and detected JSRV in 1.6 per cent, indicating that many infected animals do not develop gross tumours within their commercial lifespan[7]. JSRV-positive sheep were identified in seven counties—Donegal, Kerry, Kilkenny, Offaly, Tipperary, Waterford, and Wicklow—demonstrating a diverse distribution[7].

OPA is endemic throughout the UK, with confirmed cases in England, Scotland, Wales, and Northern Ireland[11]. In Scotland, approximately 10 per cent of flocks are estimated to be JSRV-positive[11].

Economic impact

The economic burden of OPA is substantial. When first introduced into a naïve flock, losses can be as high as 20 per cent within the first few years, while endemically infected flocks typically see annual mortality rates of one per cent to five per cent[9]. Beyond mortality, significant losses arise

Figure 1: Tumour cells produce excessive fluid that may discharge from the nostrils when the sheep's head is lowered.

Figure 2: Tumours appear as firm, grey or light purple, sharply demarcated nodules, predominantly in the ventral portions of the lung lobes.

from premature culling, reduced cull value, lower lamb birth weights, and secondary bacterial pneumonias frequently precipitated by OPA[6,15].

Clinical findings

Signalment and clinical course

Clinical OPA is most common in adult sheep aged three to four years, reflecting the long incubation period[5]. Occasionally, it is seen in animals under one year, particularly lambs born to infected dams[5]. Once clinical signs develop, the disease is progressive and fatal, with death occurring within days to several months[5].

Clinical signs

Key signs include:

- **Progressive weight loss:** Affected sheep become emaciated despite maintaining a normal appetite[5].
- **Respiratory distress:** Progressive tachypnoea and dyspnoea, initially apparent only on exercise[5]. Affected sheep characteristically lag behind the flock.
- **Lung fluid production:** Tumour cells produce excessive fluid that may discharge from the nostrils when the sheep's head is lowered (see Figure 1)[9]. This is pathognomonic for OPA, though absent in approximately one-third of cases ("atypical" form)[13].
- **Secondary bacterial pneumonia:** OPA lesions compromise pulmonary defences, predisposing sheep to *Mannheimia haemolytica* infection[1]. Many sheep present acutely and die from pasteurellosis, with OPA only identified post-mortem.

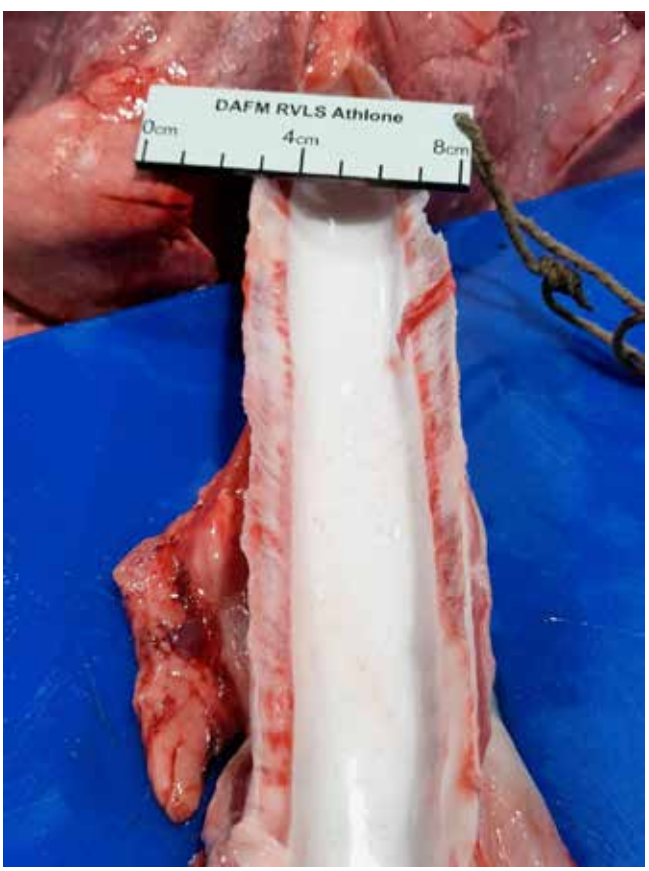

Figure 3: Copious frothy fluid is often present in the airways.

Post-mortem findings

Post-mortem examination remains the definitive confirmation. Grossly, affected lungs are enlarged, heavy, and fail to collapse[9]. Tumours appear as firm, grey or light purple, sharply demarcated nodules, predominantly in the ventral portions of the lung lobes (see Figure 2)[5]. Copious frothy fluid is often present in the airways (see Figure 3)[1]. Histologically, OPA is characterised by the proliferation of type II pneumocytes and club cells (see Figure 4)[1]. Immunohistochemistry (IHC) using antibodies against the JSRV Env protein provides definitive confirmation[11].

Diagnosis

Ante-mortem diagnosis, especially in the subclinical phase, remains a major hurdle. No commercially available, cost-

Country/Region	Estimated OPA Prevalence	Key Data Source
Republic of Ireland (adult sheep at slaughter)	0.5% (OPA); 1.6% (JSRV+ve)	Lee *et al.*, 2017[7]
UK (cull adult sheep at slaughter)	0.9%	Cousens *et al.*, 2015[11]
UK (fallen stock, Northern England)	5.6%	Lovatt & Strugnell, 2013[11]
Scotland (estimated flock-level)	~10% of flocks	Lewis *et al.*, 2011[11]

Table 1: Estimated OPA prevalence.

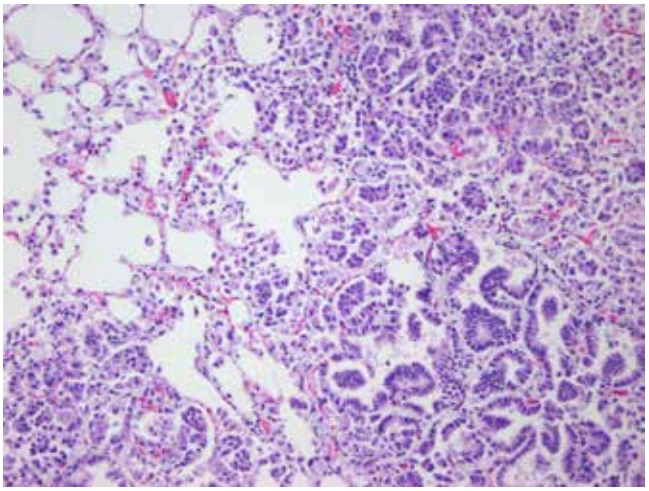

Figure 4: **Histologically, OPA is characterised by the proliferation of type II pneumocytes and club cells. Immunohistochemistry (IHC) using antibodies against the JSRV envelope protein provides definitive confirmation.**

effective test reliably identifies JSRV-infected sheep before gross tumours develop.

Diagnostic tools

- **Serological testing:** ELISAs are not available because JSRV does not elicit a detectable humoral immune response[7].
- **PCR:** PCR can detect JSRV proviral DNA or RNA, but sensitivity on blood samples is very low (11 per cent)[7]. PCR on bronchoalveolar lavage (BAL) fluid is more sensitive (89 per cent) but requires sedation and is not suitable for large-scale screening[7].
- **Transthoracic ultrasound scanning (TTUS):** A practical tool for ante-mortem evaluation in a flock setting[17]. Using a 5-6.5MHz transducer, the surgeon examines both sides of the chest wall[17]. OPA lesions appear as an abrupt loss of the hyperechoic pleural line, replaced by a hypoechoic area[17].

TTUS limitations

- Cannot detect deep-seated or small (<1-2cm) tumours[17].
- A negative scan does not guarantee freedom from JSRV infection.
- False positives can occur from lung abscesses or lungworm lesions[18].
- Requires significant operator expertise.

Prevention and control

Prevention

Prevention relies entirely on biosecurity, as there is no vaccine or treatment. The purchase of subclinically infected replacement stock is the greatest risk factor[9].

- **Closed flocks:** The most effective strategy. If not practical, stock should be purchased from flocks with a documented health history[9].
- **Quarantine:** A minimum of four to six weeks for all incoming stock, with any respiratory illness investigated via post-mortem[9]. This only partially mitigates the risk because of the long incubation period of the disease.
- **Boundary biosecurity:** Double fencing or thick hedgerows to prevent nose-to-nose contact with neighbouring flocks[9].

Control and Eradication

Once OPA is confirmed, the goal is to reduce transmission and remove infected animals.

- **Management interventions:** Minimise housing duration, avoid trough feeding (use snacker feeders), and maintain age segregation to prevent mixing older shedders with susceptible lambs[11].
- **Ultrasound screening:** A supplementary tool to identify pre-clinical tumours, allowing for earlier culling[12,17]. It must be accompanied by strict culling of suspect animals and their progeny[9].
- **Flock depopulation:** For high-prevalence or pedigree flocks, complete depopulation followed by thorough disinfection and restocking from OPA-free sources is the only guaranteed eradication method[15].

Future Directions

Research is actively progressing in several areas:

- **Improved diagnostics:** Development of sensitive, commercially viable tests for early JSRV infection, such as improved PCR assays or blood biomarkers[16].
- **Vaccine development:** Understanding the molecular mechanisms of JSRV-induced oncogenesis is providing new targets for vaccine design[4].
- **Pilot projects:** Cadres of veterinary practitioners with validated expertise in OPA control are being developed, providing models for industry-wide engagement[16].

Conclusions

Ovine pulmonary adenomatosis is a serious, endemic disease of sheep in Ireland and the UK. Its insidious nature and the lack of a serological test or vaccine make it challenging for ovine practice. Veterinary practitioners play a central role in educating farmers on biosecurity and management. While ultrasound scanning assists in identifying pre-clinical cases, it is supplementary to the gold standard of post-mortem confirmation. Vigilance, early culling, and strict management remain the most effective weapons against this incurable disease.

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READER QUESTIONS AND ANSWERS

1. WHICH OF THE FOLLOWING IS THE CAUSATIVE AGENT OF OVINE PULMONARY ADENOMATOSIS (OPA)?

A. Maedi-Visna virus (MVV)
B. Jaagsiekte sheep retrovirus (JSRV)
C. *Mannheimia haemolytica*
D. Ovine lentivirus

2. WHAT IS A UNIQUE ONCOGENIC FEATURE OF THE JAAGSIEKTE SHEEP RETROVIRUS (JSRV)?

A. It induces a strong humoral immune response
B. Its envelope (Env) protein functions as a direct oncogene
C. It primarily targets lymphoid tissue
D. It is easily propagated in standard cell culture systems

3. WHICH CLINICAL FINDING IS CONSIDERED PATHOGNOMONIC FOR CLASSICAL OPA WHEN THE SHEEP'S HEAD IS LOWERED?

A. High fever and acute coughing
B. Gradual weight loss despite a good appetite
C. Discharge of clear, frothy pulmonary fluid from the nostrils
D. Swelling of the submandibular lymph nodes

4. WHY IS SEROLOGICAL TESTING (E.G., ELISA) NOT CURRENTLY POSSIBLE FOR DIAGNOSING OPA?

A. The virus does not circulate in the blood
B. Infected sheep fail to mount a detectable humoral immune response due to immunological tolerance
C. Antibodies only appear in the very final stages of the disease
D. The available tests are too expensive for commercial use

5. WHAT ARE THE PRIMARY LIMITATIONS OF TRANSTHORACIC ULTRASOUND SCANNING (TTUS) IN OPA CONTROL? (SELECT ALL THAT APPLY)

A. It cannot detect tumours deep within the lung parenchyma
B. It cannot identify early viral infection before tumour development
C. It requires a high degree of operator experience
D. It is only effective in sheep under one year of age

ANSWERS: 1B; 2B; 3C; 4B; 5A,B, AND C.