Risk of high pathogenicity avian influenza to Australian small ruminant industries: rapid risk assessment

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Acknowledgement of Country

We acknowledge the continuous connection of First Nations Traditional Owners and Custodians to the lands, seas and waters of Australia. We recognise their care for and cultivation of Country. We pay respect to Elders past and present, and recognise their knowledge and contribution to the productivity, innovation and sustainability of Australia’s agriculture, fisheries and forestry industries.

Key findings

This is a rapidly evolving area and new information is being released regularly. This analysis was conducted based on information available up to 3 June 2025.

Summary of the event

In early 2024, a syndrome of non-specific systemic illness, milk abnormalities and an abrupt drop in milk production was observed in lactating dairy cattle in the United States (US) in Texas, New Mexico and Kansas (Caserta et al. 2024; Oguzie et al. 2024). Clade 2.3.4.4b high pathogenicity avian influenza (HPAI) of the H5N1 subtype (see Appendix A 'Influenza virus nomenclature') was identified as the cause of the syndrome, specifically a single genotype, B3.13 (Oguzie et al. 2024). Phylogenetic and epidemiological evidence suggested a single spillover event into cattle at that time, followed by ongoing transmission among dairy cows. Subsequently, 2 more spillovers into dairy cattle were detected in January and February 2025, respectively (APHIS 2025a, 2025d; CIDRAP 2025; APHIS 2025b; AZDA 2025). Genotype D1.1 was identified in both those events; however, virus sequences from each event were genetically distinct, indicating 2 separate spillovers (APHIS 2025b). Both D1.1 spillovers were detected through testing of milk from processing plant silos. Three of 11 silo samples collected in Nevada tested positive and trace-back revealed that 2 herds were infected with this D1.1 genotype (APHIS 2025d). The Arizona D1.1 spillover was attributed to a single dairy farm (AZDA 2025). Critically, the definitive spillover hosts and transmission pathways into dairy cattle were not determined for any of the spillover events. While wild birds are generally implicated, both genotypes were also confirmed in poultry and other mammals (including cats, peri-domestic wildlife (e.g. raccoons, rodents) and humans), raising the possibility of alternative entry pathways. Importantly, there is no evidence for sustained transmission of these genotypes within mammal species other than cattle. To date, the US remains the only country where sustained transmission of clade 2.3.4.4b HPAI in dairy cattle has occurred.

The ongoing HPAI outbreak in dairy cattle has now affected at least 1,072 dairy farms across 17 states (APHIS 2024f). Small ruminant infections have also been reported. For example – in March 2024, clade 2.3.4.4b HPAI genotype B3.6 was detected in neonatal goat kids on a farm following an outbreak in backyard poultry (APHIS 2024c). Adult goats on the same premises tested negative. Chickens and ducks on the premises were found to be infected with the same genotype as the kids and shared the same pasture and water source. Then in March 2025, a lactating ewe tested positive for clade 2.3.4.4b HPAI in the United Kingdom (UK) on a premises experiencing an outbreak in captive birds (DEFRA and APHA 2025; Schnirring 2025; HAIRS 2025). Detections of clade 2.3.4.4b HPAI in the US have also been confirmed in other livestock, including in pigs (genotype D1.2) and alpacas (genotype B3.13) (APHIS 2024g, 2024c, 2024d), highlighting the potential for infection in species beyond dairy cattle.

While this assessment was conducted based on information available up to 3 June 2025, as of October 2025, Australia remains free of clade 2.3.4.4b HPAI.

The recent outbreak of clade 2.3.4.4b HPAI in dairy cows in the US, along with the detections in both goats in the US and a ewe in the UK, has heightened concerns about the potential risk to Australia's small ruminant industries. A formal qualitative risk assessment is currently being conducted to assess the risk to Australian dairy cattle. Here, we synthesise the latest scientific evidence on HPAI in small ruminants to inform an evidence-based rapid risk assessment for the Australian small ruminant industries. For the purposes of this assessment, the small ruminant industries comprise sheep and goat industries.

Risk questions

This rapid risk assessment addresses these risk questions:

1) *Assuming clade 2.3.4.4b HPAI was present in Australia*, what is the risk (likelihood and consequences) to Australian small ruminant industries?
    a) Entry assessment: Assuming clade 2.3.4.4b HPAI was present in Australia, what is the likelihood of clade 2.3.4.4b HPAI spilling over into at least 1 small ruminant in Australia in the year following incursion?
    b) Establishment and spread assessment: If clade 2.3.4.4b HPAI were to infect 1 or more small ruminants in Australia, what is the likelihood of spread within and between small ruminant production premises in the year following incursion?
    c) Consequence assessment: What are the consequences of clade 2.3.4.4b HPAI infection in Australian small ruminants?

Overall assessment

Overall, assuming clade 2.3.4.4b HPAI was present in Australia, the risk to Australian small ruminant industries was assessed as negligible, with very high uncertainty.

Key findings supporting this assessment include:

Entry assessment

The likelihood of clade 2.3.4.4b HPAI spilling over into at least 1 small ruminant in Australia was assessed as low, with moderate uncertainty.

Despite high levels of HPAI circulation in wild bird and poultry populations globally, spillovers into small ruminants are detected rarely. One infection (based on detection of viral RNA) has been reported from a sheep in the UK and 5 infections were reported in newborn goat kids in the US.

These spillover events were associated with infected backyard poultry flocks, with contaminated environment and water sources implicated as the potential sources of infection. Other factors likely contributed to the susceptibility of the goat kids to infection, as 5 others also died on the premises but tested negative for HPAI. This could suggest that host compromise or poor husbandry may have weakened immunity, increasing susceptibility to infection.

Critically, limited surveillance is being done for HPAI infection in small ruminants. Therefore, infection rates may be underestimated. One serological study conducted in Pakistan in 2023 found that 23.9% of goat and 31.0% of sheep samples were positive for antibodies against clade 2.3.4.4b HPAI, indicating prior exposure (Wong et al. 2024). If accurate, this suggests that infections in small ruminants may be subclinical, as no history of disease was reported in that study.

One of 85 sheep grazing pastures in Norway where sick and dead wild birds were present was found to have antibodies against clade 2.3.4.4b HPAI (Tønnessen and Paulson 2025). All sheep remained healthy.

Establishment and spread assessment

The likelihood of clade 2.3.4.4b HPAI establishing and spreading within and between small ruminant production premises was assessed as negligible, with high uncertainty.

No other adult sheep on the infected premises were infected following the detection in the UK. The lambs of the infected ewe tested negative for viral RNA, although 1 lamb returned a weakly positive serological reaction on an unaccredited test, indicating either exposure to the virus or a false positive result. No adult goats on the infected premises in the US tested positive.

Of the various samples collected, including nasal swabs, only the milk of the infected ewe tested positive (at relatively high cycle threshold values) and infectious virus was not recovered, noting that sampling only occurred late in infection. Virus levels in the samples collected from the goat kids were not reported.

In dairy cattle, exposure to milk from infected cows is thought to be the primary mode of transmission. This is because ‘avian-type’ receptors (α-2,3-linked sialic acids) are abundantly expressed in the bovine mammary gland. Therefore, minimal adaptation is required for avian-origin viruses to infect the bovine udder, and virus can replicate to very high levels, facilitating spread in milk. Although studies are limited, preliminary investigations indicate that mammary tissues from small ruminants also express these ‘avian-type’ receptors. Animals within the sheep and goat dairy sectors would therefore likely be at a higher risk of exposure. However, non-dairy small ruminants would not be exposed to milk from other small ruminants.

Although limited data are available, Australian sheep and goat dairy industries appear to have limited connectedness. That is, animals (especially lactating ewes or does), vehicles and equipment are not frequently moved between premises (Zalcman and Cowled 2017).

Critically, no experimental infection studies with clade 2.3.4.4b HPAI have been conducted in small ruminants. Therefore, the dynamics of virus shedding in small ruminants, over time and in different tissues, secretions and excretions, are not known. Routes of infection in sheep (e.g. respiratory, ingestion, intramammary) are not known. This adds significant uncertainty to our assessment.

To transmit efficiently amongst mammalian hosts via non-milk associated routes, avian-adapted influenza A viruses must overcome several barriers. Generally, multiple infection events in a new host species are required for mammalian-adaptive genetic mutations to emerge and establish in a virus population. So far, sustained mammal-to-mammal transmission has only been documented in limited cases: fur farms in Europe, in wild marine mammals in South America, and in dairy cows in the US.

Consequence assessment

The consequences of clade 2.3.4.4b infection in Australian small ruminants were assessed as minor, with very high uncertainty.

Animal health and welfare impacts in small ruminants are not known. The single infection in a sheep was associated with localised mastitis, but no other clinical signs (e.g. no respiratory signs). The infections in goat kids were associated with neurological signs; however, a clear link to HPAI was not established, as 5 others also died on the premises but tested negative.

The Australian sheep and goat dairy industries are relatively small (AHA 2022). The economic impact of mastitis in Australian small ruminant dairy industries has not been examined. The severity of mastitis caused by clade 2.3.4.4b HPAI in small ruminants, and how this compares with the disease in cattle, is based on a single infection in a non-commercial lactating ewe.

Impacts on meat and fibre production are anticipated to be minor, based on limited mortality in other artiodactyl species (e.g. non-lactating cattle, pigs, alpacas). This is speculative and thus highly uncertain. The impacts of HPAI infection on liveweight gain, reproduction, wool quality and other metrics relevant to small ruminant meat and fibre production have not been investigated.

The potential trade impacts of clade 2.3.4.4b HPAI infection in Australian small ruminants are difficult to predict, adding considerable uncertainty to the assessment. It is possible that trading partners may impose restrictions or additional testing requirements if clade 2.3.4.4b HPAI was detected in Australian small ruminants. To our knowledge, there have been no reported impacts on sheep or goat trade following the UK and US isolated detections.

Response measures may result in significant industry disruption and socio-economic impacts, if implemented. Specific response policies for livestock in the event of an outbreak are being considered by governments. This adds further uncertainty to the assessment.

Public health consequences are negligible, since humans are unlikely to be exposed to raw milk from small ruminants in most circumstances. There are no reliable statistics on how many Australians consume (or are exposed to) raw sheep or goat milk but we assume that consumption is small-scale. The sale of raw sheep’s milk for human consumption is illegal in Australia, although some states allow the production and sale of raw goat's milk for human consumption. The number of sheep and goat dairy workers is assumed to be very small, as these are niche industries. Current evidence suggests that infected dairy (cattle) workers acquired infection through exposure to raw milk or through close contact with secretions from clinically affected animals. Certain activities (e.g. drenching of sick animals) may increase the risk of zoonotic transmission.

Importantly, with the continued evolution of clade 2.3.4.4b viruses and the emergence of novel genotypes, the biological properties (such as pathogenesis, virulence and transmissibility) of these viruses may change over time, which may change the results of this risk assessment.

Recommendations

Key recommendations include:

1) To reduce the likelihood of entry into small ruminants:
    a) Current spillovers of clade 2.3.4.4b HPAI into small ruminants have most likely been via infected poultry or direct contact with large numbers of sick and dead wild birds. Therefore, avoid co-mingling small ruminants and poultry and limit contact with potentially contaminated environments.
    b) Where possible, prevent (or limit) access of wild birds, poultry and peri-domestic wildlife to small ruminant feed, pasture, feed storage, water sources, bedding materials and facilities.
    c) Avoid sharing (unclean) equipment or vehicles with poultry (and dairy cattle) farms.
2) To reduce the likelihood of transmission between premises:
    a) Minimise unnecessary animal movements and keep detailed movement records, particularly if moving animals between small ruminant dairy premises.
    b) Maintain good farm biosecurity (e.g. pro-actively manage movement of people, equipment and vehicles).
    c) Enhance general surveillance (e.g. monitor for sick livestock, wild birds or wildlife; consider HPAI as a differential diagnosis for unexplained illness or mastitis in small ruminants).
    d) Jurisdictions and the Commonwealth should establish testing protocols for non-avian species for HPAI, to facilitate testing of suspect cases.
    e) Consideration should be given as to whether an evidence-based active surveillance strategy is required following detection of a clade 2.3.4.4b HPAI spillover event in small ruminants. This would need to be assessed within the specific context of an incursion.
3) To reduce impacts:
    a) Response strategies in non-avian species should be considered now (i.e. in peacetime) and clearly communicated so that industry can better understand the likely impacts of potential control measures.
    b) Consider the use of personal protective equipment (such as gloves, apron, respiratory protection and eye protection) in certain circumstances (e.g. during milking in small ruminant dairies or when in close contact with secretions from sick animals like during drenching) to reduce the risk of human infection.
    c) Do not consume raw milk, colostrum or raw milk products from small ruminants.

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Introduction

This assessment was conducted based on information available up to 3 June 2025.

In 2020, a novel clade 2.3.4.4b high pathogenicity avian influenza (HPAI) virus of the H5N1 subtype (see Appendix A 'Influenza virus nomenclature') emerged that led to unprecedented transmission and disease in wild bird populations in all continents except Oceania (Fusaro et al. 2024; Wille et al. 2024). While infections in mammals have previously occurred with other HPAI viruses, mammals seem to be unusually susceptible to clade 2.3.4.4b HPAI—infections have now been reported in 25 mammalian families and 92 species (FAO 2025). Sustained mammal-to-mammal transmission has now been documented in several settings, including on fur farms in Europe, in wild marine mammals in South America, and in dairy cows in the United States (US) (Peacock et al. 2024).

In early 2024, a syndrome of milk abnormalities and non-specific systemic illness accompanied by an abrupt drop in milk production was observed in lactating dairy cattle in the US in Texas, New Mexico and Kansas (Caserta et al. 2024; Oguzie et al. 2024). Unexpectedly, clade 2.3.4.4b HPAI of the H5N1 subtype was subsequently identified as the cause of the syndrome (Oguzie et al. 2024). Phylogenetic analyses revealed that all viral sequences taken from dairy cows were closely related and fell into a single monophyletic lineage (called genotype B3.13)—that is, they were all descended from a single common ancestor (Worobey et al. 2024; Caserta et al. 2024). This implies a single spillover event into cattle with ongoing transmission between dairy cows.

Subsequently, 2 more spillovers of clade 2.3.4.4b HPAI, both of genotype D1.1, were detected in dairy cattle, the first in Nevada in January 2025 and the second in Arizona in February 2025 (APHIS 2025a, 2025d; CIDRAP 2025; APHIS 2025b; AZDA 2025). Virus sequences from each event were genetically distinct, indicating 2 separate spillovers of genotype D1.1 into dairy cattle (APHIS 2025b). Both events were detected through testing of milk from processing plant silos. Three of 11 silo samples collected in Nevada tested positive and trace-back revealed that 2 herds were infected with this D1.1 genotype (APHIS 2025d). The Arizona D1.1 spillover was attributed to a single dairy (AZDA 2025). Importantly, the definitive spillover hosts and transmission pathways into dairy cattle remain unknown. While wild birds are often implicated (e.g. through feed contaminated with wild bird faeces (Burrough et al. 2024)), this is speculative. Both genotypes B3.13 and D1.1 have also been confirmed in poultry and other mammals (including humans), suggesting that alternative entry pathways are possible.

Clade 2.3.4.4b HPAI can also infect other livestock species, including small ruminants. In March 2024, 5 goat kids in the US tested positive following unusual deaths of 10 newly kidded goats on the property where a backyard poultry flock had been depopulated due to HPAI (AVMA 2024). Sequencing of the virus revealed the same strain in both goat kids and poultry (genotype B3.6), which is different to the genotypes so far detected in dairy cattle (genotypes B3.13 and D1.1) (APHIS 2024c). The goats and poultry had access to the same environment, including shared pasture and water sources. The goat kids developed neurological signs, including incoordination and seizures. However, other concurrent disease processes were thought to contribute to this clinical presentation, since HPAI was only identified in 5 of the 10 kids displaying neurological signs (APHIS 2024c). Adult goats from the same farm tested negative for HPAI (AVMA 2024).

To date, there has been 1 confirmed case of clade 2.3.4.4b HPAI in sheep, with a single lactating ewe testing positive in the United Kingdom (UK) in mid-March 2025. This ewe had clinical signs of mastitis but was otherwise healthy (i.e. there was no evidence of respiratory disease) (DEFRA and APHA 2025). The case was identified during routine surveillance of co-located livestock on a premises where HPAI had been confirmed in captive birds. Another 19 sheep on the property tested negative (HAIRS 2025).

Further evidence for small ruminant susceptibility to clade 2.3.4.4b HPAI comes from a serosurvey for influenza A viruses (IAVs) in small ruminants in Pakistan in 2023. This study found that 23.9% of goat and 31.0% of sheep serum samples had antibodies against clade 2.3.4.4b HPAI, indicating prior exposure (Wong et al. 2024). Seropositivity was also detected to other IAVs, including clade 2.3.2.1c H5 HPAI. The paper did not report any history or evidence of clinical disease in sampled animals.

The cells of the bovine udder express specific sugar molecules (α-2,3-linked sialic acids) that avian-adapted IAVs can bind to without having to evolve specific mammalian genetic adaptations (Peacock et al. 2024; Good et al. 2024; Kristensen et al. 2024). Because of this, IAV infection in dairy cattle is mostly localised to the mammary tissue and the virus is shed in extremely high concentrations in milk (Mitchell et al. 1954, 1953; Halwe et al. 2024; Caserta et al. 2024). Virus titres can peak at 10^9 50% tissue culture infectious doses ($TCID_{50}$, a measure of the amount of virus) per ml of milk. Hence, exposure of susceptible individuals to raw milk or colostrum (either through direct contact with infected cows or via exposure to contaminated fomites) is currently thought to be the primary pathway for transmission between cows (Halwe et al. 2024; Zhou et al. 2024). Studies on sialic acid distribution in other livestock species are limited. One recent study examined the expression pattern of different sialic acids (i.e. 'mammalian' vs 'avian') in mammary gland tissue of various livestock species (including sheep and goats) (Nelli et al. 2025). Sheep and goat mammary tissue showed evidence of staining for both α-2,3-linked ('avian') and α-2,6-linked ('mammalian') sialic acids. This suggests that both mammalian- and avian-adapted IAVs have the potential to bind to small ruminant mammary tissue. Importantly, this study assessed sialic distribution via histochemistry, not direct virus binding. Sialic acid distribution in other small ruminant tissues (e.g. the respiratory tract) has not been studied.

The emergence of sustained mammalian transmission in dairy cattle marks an unprecedented shift in HPAI epidemiology. This raises concerns about other livestock species being at risk of similar events, particularly if novel clade 2.3.4.4b HPAI genotypes emerge. A formal qualitative risk assessment is currently being conducted to assess the risk to Australian dairy cattle. Here, we synthesise the latest scientific evidence on HPAI in small ruminants to inform an evidence-based rapid risk assessment for the Australian small ruminant industries.

Australian small ruminant industries

The small ruminant industries in Australia, encompassing sheep and goat production, are a significant part of the country's agricultural landscape, particularly in the rangelands and broadacre farming regions. Both sheep and goat industries comprise meat, fibre and very small dairy sectors.

Sheep are generally managed extensively in pastured-based systems (Sheep Producers Australia n.d.). The majority of the national flock is found in the southern states, with 36.6% in New South Wales, 14.9% in Victoria, 18.8% in southern Western Australia and 14.9% in South Australia (MLA

2024). Tasmania and Queensland represent 3% and 4% of the national flock, respectively (MLA 2024). Commercial sheep production does not occur in the Northern Territory. There are approximately 20,000 Australian agricultural enterprises involved in lamb and sheep production (Sheep Producers Australia n.d.). The majority of enterprises that run sheep are mixed, deriving income from both sheepmeat and wool, with many also farming beef cattle and crops (Sheep Producers Australia n.d.).

The national sheep flock was estimated at 78.8 million in 2023 (MLA 2024). Merinos are the primary breed bred in Australia due to their superior wool production. They are frequently crossed with another breed, usually a meat or dual-purpose breed, to produce high-quality prime lambs, although purebred Merino lamb production remains the dominant mating process for large producers (greater than 5,000 head).

Australia is the world leader for sheepmeat and fibre exports, with the sheepmeat sector valued at $4.5 billion in the FY2022–23 (Sheep Producers Australia n.d.; MLA 2024). As much as 70% of Australian lamb and over 90% of mutton is exported, primarily to China (30.9%), the US (14.9%) and Malaysia (5.7%) (MLA 2024). Wool exports were valued at $3.6 billion in the FY2021–22, with China being the primary market (Australian Wool Innovation 2023). Australia also exports live sheep, primarily to the Middle East (DAFF 2025). In 2024, there were 433,078 live sheep exported from Australia.

The Australian sheep dairy industry is a boutique industry, comprising a small number of milking herds (probably less than 20, although reliable data are not readily available). Common dairy sheep breeds include East Friesians, along with some African breeds (e.g. Awassi) and British breeds. Based on various sheep dairy public websites, flock sizes are small (10s to a few hundred head) and dairy sheep are often raised with other livestock, sometimes including dairy goats and dairy cattle.

Goatmeat production in Australia is mostly centred on semi-wild rangeland goats, with over 90% of production traditionally being rangeland enterprises (GICA n.d.). However, the number of managed goat production systems has increased over recent years (MLA 2023). In addition to rangeland goats, Boer goats are also used for meat production in Australia (GICA n.d.). Production is closely linked to environmental conditions, with NSW producing the largest number of goats (MLA 2023). In 2022, 1.7 million head were slaughtered nationwide.

Australia is one of the largest goatmeat exporters globally, with the US being the primary market (MLA 2023). Goatmeat is also exported to South Korea, Canada, Taiwan and Trinidad and Tobago. Australian goatmeat exports were valued at USD$182.6 million in 2021 (MLA 2023). Malaysia is Australia's largest live goat importer, followed by China (DAFF 2025). In 2024, 14,749 live goats were exported.

The goat fibre sector was established in the 1970s and uses both Cashmere and Angora breeds (AgriFutures Australia 2021). Available data suggest that the Australian mohair industry represents 5% of global production, while the cashmere industry represents 0.5% of global production. Reliable data on the number of producers in Australia (or geographical distribution) are not readily available.

In 2016, there were 68 dairy goat farms registered with jurisdictional dairy registration bodies, not including businesses that were not producing food products (e.g. those solely producing soap)

(Zalcman and Cowled 2017). Dairy goat businesses were distributed across all Australian jurisdictions except for the Northern Territory and the Australian Capital Territory. It was estimated that the Australian national dairy herd consisted of 30,550 milking goats and 46,152 total head, with an average of 678 goats per farm. A survey of producers revealed that the industry was fragmented, with producers having little contact with each other.

Risk questions

1) Assuming clade 2.3.4.4b HPAI was present in Australia, what is the risk (likelihood and consequences) to Australian small ruminant industries?
    a) Entry assessment: Assuming clade 2.3.4.4b HPAI was present in Australia, what is the likelihood of clade 2.3.4.4b HPAI spilling over into at least 1 small ruminant in Australia in the year following incursion?
    b) Establishment and spread assessment: If clade 2.3.4.4b HPAI were to infect 1 or more small ruminants in Australia, what is the likelihood of spread within and between small ruminant production premises in the year following incursion?
    c) Consequence assessment: What are the consequences of clade 2.3.4.4b HPAI infection in Australian small ruminants?

1 Methods

We previously (January 2025) conducted a literature review on the current state of knowledge around HPAI in dairy cattle and other livestock species (Schlosberg et al. 2025). We examined over 265 information sources, including peer-reviewed and preprint journal articles and grey literature such as books, technical reports and conference proceedings. Additionally, we consulted with international influenza virus expert Dr Michelle Wille. Dr Wille is a Senior Research Fellow in Microbiology and Immunology at the Centre for Pathogen Genomics at the University of Melbourne and Honorary Appointment at the World Health Organization Collaborating Centre for Reference and Research on Influenza at the Peter Doherty Institute for Infection and Immunity.

Importantly, this assessment is based on the currently available information. Rapid risk assessments are intended to be iterative and estimates should be revised as new information becomes available (FAO 2021), particularly in a situation that is rapidly evolving.

As the scope of this work was a rapid risk assessment, we did not specifically evaluate individual entry and exposure pathways.

1.1 Definitions

We use these definitions in this assessment:

- Entry: The initial incursion or spillover of clade 2.3.4.4b HPAI virus into 1 or more small ruminants.
- Establishment and spread: Onward transmission of clade 2.3.4.4b HPAI virus in small ruminants, leading to sustained spread between premises (under the baseline assumption of no control measures) and an outbreak in the industry.
- Likelihood: The estimated probability or chance that the event will occur (FAO et al. 2020). The qualitative likelihood categories used in this assessment are defined in Table 1.

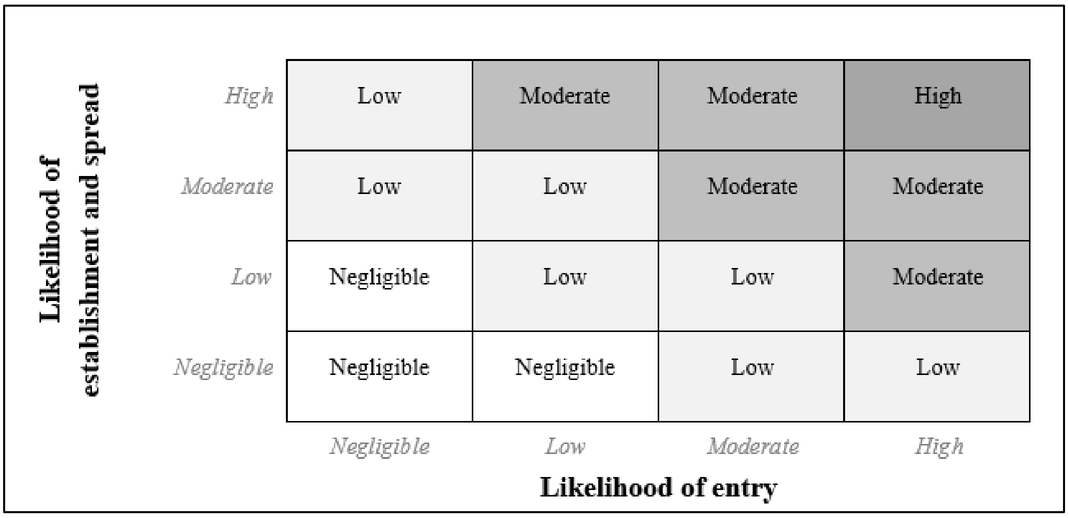
Table 1 Qualitative likelihood categories used in this assessment

Qualitative category	Definition
Negligible	Extremely unlikely; may only occur in exceptional circumstances
Low	Unlikely; may occur, but not in the majority of cases
Moderate	Likely; may occur in the majority of cases
High	Very likely; expected to occur frequently

From (FAO 2021)

To estimate the combined likelihood of entry and establishment and spread, we followed the methodology used by Wildlife Health Australia in their clade 2.3.4.4b HPAI incursion risk assessment for Australia (WHA 2023) (Figure 1).

Figure 1 Matrix for combining likelihood of entry and establishment and spread



Likelihood of establishment and spread

High	Low	Moderate	Moderate	High
Moderate	Low	Low	Moderate	Moderate
Low	Negligible	Low	Low	Moderate
Negligible	Negligible	Negligible	Low	Low
	Negligible	*Low*	*Moderate*	*High*

Likelihood of entry

From (WHA 2023)

Consequences: The level or severity of impacts or outcomes if the event occurs (FAO et al. 2020). The qualitative consequence categories used in this assessment are defined in Table 2.

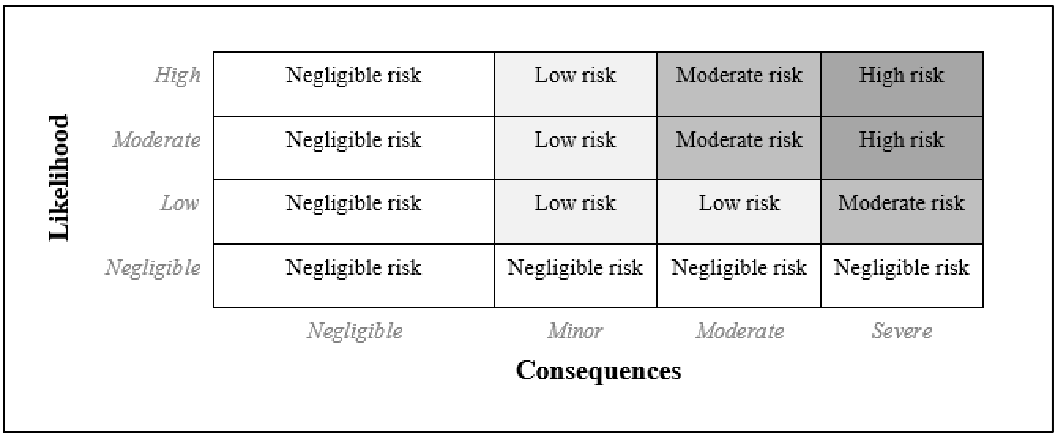
Table 2 Qualitative consequence categories used in this assessment

Qualitative category	Definition	Examples
Negligible	Insignificant negative consequences on industry productivity or animal or human health.	Minor production losses, low number of localised infections. No threat to food security or the economy.
Minor	Marginal negative consequences industry productivity or animal or human health.	Production losses restricted to a small area (regional level or below).
Moderate	Significant negative consequences on industry productivity or animal or human health.	Significant production losses across several regions.
Severe	Substantial negative consequences on industry productivity or animal or human health.	Significant production losses at the national level.

Modified from (FAO et al. 2020)

Risk: The overall risk of the event, considering both the likelihood and consequences (FAO et al. 2020). The risk estimation matrix used in this assessment is given in Figure 2.

Figure 2 Risk estimation matrix



Likelihood				
High	Negligible risk	Low risk	Moderate risk	High risk
Moderate	Negligible risk	Low risk	Moderate risk	High risk
Low	Negligible risk	Low risk	Low risk	Moderate risk
Negligible	Negligible risk	Negligible risk	Negligible risk	Negligible risk
	Negligible	Minor	Moderate	Severe

Consequences

Modified from (WHA 2023)

Uncertainty: The level of confidence we have in our estimate, given the availability of information about a parameter (Vose 2000). The qualitative uncertainty categories used in this assessment are defined in Table 3.

Table 3 Qualitative uncertainty categories used in this assessment

Qualitative category	Definition
Very low	Reliable data and information are available in sufficient quantity; results strongly anchored in empiric data or concrete information
Low	Reliable data and information available but may be limited in quantity, or be variable; results based on expert consensus
Moderate	Some gaps in availability or reliability of data and information, or conflicting data; results based on limited consensus
High	Limited data or reliable information available; results based on educated guess
Very high	Lack of data or reliable information; results based on crude speculation only

From (FAO et al. 2020)

1.2 Assumptions

We made these assumptions when conducting our assessment:

- That clade 2.3.4.4b HPAI is present in wild birds in Australia. This is a hypothetical assumption for the purpose of this assessment—as of October 2025, Australia remains free of clade 2.3.4.4b HPAI. Without this assumption, we would also need to consider the incursion risk and risk of establishment within Australia. This has already been comprehensively assessed by Wildlife Health Australia (WHA 2023).
- That the incidence of clade 2.3.4.4b HPAI infection in wild birds is moderate to high and geographically homogeneous across Australia. That is, Australian small ruminants could make contact with an infected wild bird and the probability of this is equal across all of Australia.
- That clade 2.3.4.4b HPAI is only present in wild birds. If infection was additionally present in poultry and/or other mammal populations then risk would likely be higher.

- That risk of entry is equal across different small ruminant industry sectors (e.g. pasture-based vs rangeland systems, lifestyle farms, dairies) and small ruminant types (e.g. sheep vs goats, different breeds). A more comprehensive formal risk assessment would be required to analyse how risk may vary across different subpopulations.
- That reasonable biosecurity is practiced by commercial herds, such as not exposing small ruminants to poultry carcasses or by-products, as stipulated by the Australian ruminant feed ban. We also assume that small ruminants are not fed raw milk from dairy cattle.
- That no specific control measures are in place to mitigate spread between premises at the time of an incursion (i.e. animal, people, vehicle and equipment movements continue as normal).
- That the biology (including pathogenesis, virulence and transmissibility) of any virus entering Australia is not substantially different to previously evaluated clade 2.3.4.4b HPAI viruses. Critically, the risk will vary depending on whether an incursion strain is avian-adapted or mammalian-adapted (although we assume that viruses circulating in wild birds are avian-adapted), and potentially according to genotype and other host and environmental factors. A comprehensive risk assessment would be required to further explore these variables.

2 Results

2.1 Entry assessment

Assuming clade 2.3.4.4b HPAI was present in Australia, what is the likelihood of clade 2.3.4.4b HPAI spilling over into at least 1 small ruminant in Australia in the year following incursion?

Likelihood: Low

2.1.1 Rationale

Spillover of IAVs into small ruminants is possible but appears to be uncommon. There have been 6 documented cases of clade 2.3.4.4b HPAI in small ruminants; 5 very young goat kids on a single premises in the US and 1 lactating ewe in the UK (AVMA 2024; APHIS 2024c; DEFRA and APHA 2025). This is despite widespread circulation of clade 2.3.4.4b HPAI in wild bird populations across Europe, Asia and North America for several years. Other small ruminants on those affected premises tested negative (although not all were tested). It is unclear how much active surveillance is being done in small ruminants globally. Notably, risk-based HPAI surveillance in livestock on HPAI-affected poultry premises has been in place in the UK since 2024 (HAIRS 2025). There are no reports of routine testing of sheep and/or goat milk for clade 2.3.4.4b HPAI.

A serosurvey conducted in Pakistan in 2023 suggested that clade 2.3.4.4b HPAI infection in sheep and goats may be underappreciated, with 31.0% of sheep and 23.9% of goat samples testing positive for antibodies against clade 2.3.4.4b HPAI, suggesting prior exposure (Wong et al. 2024).

In May 2025, researchers at the Norwegian Veterinary Institute reported that at least 1 of 85 sheep that grazed pasture during a major HPAI outbreak had antibodies against clade 2.3.4.4b HPAI virus (Tønnessen and Paulson 2025). The flock had direct contact with sick and dead wild birds while grazing. All sheep remained healthy.

Other IAVs can also infect small ruminants; thus, the risk is not restricted to clade 2.3.4.4b HPAI. For example– a serosurvey from Qatar in 2019–20 found that 8% of sampled sheep were seropositive for IAV subtype H1N1, while none of the sampled goats were seropositive (Zedan et al. 2025). A 1954 study inoculated IAV directly into the udder of a lactating goat; virus could be detected in milk for 8 days post-infection, suggesting bona fide infection and replication in the udder (Mitchell et al. 1954).

The current hypothesis for infection of the lactating ewe in the UK is that virus was introduced directly into the teat, possibly via suckling lambs or when the keeper was stripping the udder or stimulating the lamb to suckle, resulting in a localised mastitis (HAIRS 2025). It was also suggested that there may be a small possibility of contamination of the udder if the sheep lies down on bedding or flooring contaminated with bird faeces (HAIRS 2025). It is not known how the newborn goats became infected (e.g. respiratory, ingestion, contact or other), although the environment was thought to be very heavily contaminated (APHIS 2024c). No experimental infection studies with clade 2.3.4.4b HPAI have been conducted in sheep or goats.

With the lack of active surveillance in small ruminants, it is difficult to assess the frequency of spillover relative to exposure (and therefore the likelihood). The 6 confirmed clade 2.3.4.4b HPAI infections in small ruminants would suggest spillover from poultry, as all cases occurred on non-commercial properties with infected poultry and poor biosecurity measures (HAIRS 2025; AVMA 2024). However, transmission from wild birds to both poultry and livestock concurrently in these cases cannot be excluded. The proportion of Australian sheep and goats that may share an environment with domestic poultry is not known. Given that most small ruminants in Australia are extensively reared (e.g. pasture-based or rangeland), contact with wild birds is expected to be relatively high.

Biosecurity and awareness of HPAI infection in non-poultry species may be lower amongst smallholder and non-commercial producers compared to commercial sheepmeat and wool producers. For example– there may be higher rates of small ruminants being housed with, or in close proximity to, domestic poultry and waterfowl on smaller premises. However, this has not been explicitly studied in Australia.

A Belgian risk assessment evaluated the likelihood of clade 2.3.4.4b HPAI exposure in goats in Belgium as ‘very low’, although that was based on a low infection pressure in wild birds at the time of the assessment (RAG-V-EZ 2024).

A Canadian risk assessment stated that ‘it is reasonable to assume that livestock with direct contact with infected birds and/or their contaminated environments (such as food and water sources) may become infected; however, the extent of these infections that may have already occurred or that may occur in the future is unknown’ (Public Health Agency of Canada 2024).

Thus, spillovers may occur but, given the likely number of small ruminant exposures (considering the incidence of HPAI in wild birds and poultry globally), spillovers are infrequent.

Uncertainty: Moderate

Previous spillovers into small ruminant populations without sustained animal-to-animal transmission may have gone undetected, leading to underestimation of the likelihood. This is compounded by a lack of active surveillance of small ruminant populations in high-risk countries. Given the lack of knowledge regarding clinical signs in small ruminants, it is possible that widespread infection could be unrecognised. There have been no experimental infection studies investigating pathogenesis of clade 2.3.4.4b HPAI in these species. The infectious dose for sheep and goats is not known; nor are the potential infection pathways (e.g. oral, respiratory, other).

2.2 Establishment and spread assessment

If clade 2.3.4.4b HPAI were to infect 1 or more small ruminants in Australia, what is the likelihood of spread within and between small ruminant production premises in the year following incursion?

Likelihood: Negligible

2.2.1 Rationale

Most spillovers of IAVs from birds to mammals result in dead-end infections. This is because the necessary genetic changes for efficient mammalian infection typically require multiple transmission events in mammals (and sustained selection pressure) to emerge (Arruda et al. 2024). Dairy cattle are an exception because ‘avian-type’ receptors (α-2,3-linked sialic acids) are abundantly expressed in the mammary gland (Peacock et al. 2024; Good et al. 2024). Therefore, minimal adaptation is required for avian-adapted HPAI viruses to spread between lactating dairy cows via the milk.

It is unknown whether sheep or goats have these ‘avian-type’ receptors in their mammary glands (or in other tissues) (HAIRS 2025). However, 1 recent study identified expression of both α-2,3-linked (‘avian’) and α-2,6-linked (‘mammalian’) sialic acids in sheep and goat mammary tissue, similar to lactating dairy cattle, suggesting that both mammalian and avian IAVs have the potential to bind to the small ruminant udder (Nelli et al. 2025). Intriguingly, the sialic acid distribution in sheep, goat and beef cattle mammary tissue more closely resembled that of the lactating dairy cow, compared to non-lactating dairy cow tissue. It is not known whether the sheep, goat and beef animal used in the study were lactating at the time of tissue collection. Importantly, that study only assessed sialic distribution via histochemistry, not direct virus binding. However, preliminary unpublished findings from the UK also suggest similarities between the bovine and sheep udder in terms of sialic acids (HAIRS 2025).

Cells of the bovine udder support very high levels of virus replication, leading to very high virus levels in milk (e.g. as little as 1 µl of milk may be infectious). This is true of both clade 2.3.4.4b viruses and ancestral IAVs (Mitchell et al. 1953; Caserta et al. 2024; Halwe et al. 2024; Ríos Carrasco et al. 2024). As such, exposure of susceptible individuals to raw milk or colostrum, either through direct contact with infected cows or via exposure to contaminated fomites, is currently thought to be the primary pathway for transmission between cows (APHIS 2024e; Peacock et al. 2024; Halwe et al. 2024; Zhou et al. 2024). Subclinical dairy cows can also shed virus in milk and therefore pose a transmission risk (Caserta et al. 2024). As no experimental infection studies have been done in small ruminants, the dynamics of clade 2.3.4.4b HPAI replication in these species is not known. Relatively low levels of viral RNA were detected in 2 milk samples from the infected sheep, with no infectious virus able to be isolated (HAIRS 2025). However, the animal may have been infected for up to 2 weeks before being sampled (HAIRS 2025). In dairy cattle, virus concentrations in milk peak at 2 days post-infection and infectious virus was isolated up to 8 days post-infection (Halwe et al. 2024). In a 1954 study, infectious IAV was recovered from the milk of a lactating goat following intramammary inoculation, with virus levels peaking on day 5 post-infection (Mitchell et al. 1954).

If infectious clade 2.3.4.4b HPAI is present in the milk of small ruminants, exposure rates to contaminated milk are likely to be low in most settings. Small ruminants (apart from lambs and kids) are rarely exposed to raw milk from other small ruminants. While calves have been proven to become infected from the ingestion of contaminated raw milk (Davila et al. 2025), it is unclear whether lambs or kids can be infected by this pathway. The lambs of the infected ewe in the UK tested negative for viral RNA, although 1 lamb returned a weakly positive serological reaction on an unaccredited test (HAIRS 2025). Within-herd transmission would presumably be more likely on small ruminant dairies, where contamination could occur through shared milking equipment or during the milking process, as for dairy cattle, if sheep or goat milk is indeed infectious.

Movement of infected animals (either clinical or subclinically or preclinically infected), with subsequent within-herd spread through milk-associated routes, appears to be the major pathway for between-herd spread in the US dairy cattle industry (Campbell et al. 2025). As there is a small ruminant dairy sector in Australia, albeit small-scale, it is worth noting these potential transmission pathways. In Australia, most sheep milk producers process their own milk on-site, limiting the need for raw milk transport (AHA 2022). Goat dairies either process milk on-site, self-deliver to a manufacturer twice a week, or have a specified contractor for transport (AHA 2022). Transmission between goat dairies would seem unlikely, given the fragmented nature of the industry, with little contact between premises (Zalcman and Cowled 2017). Contact networks amongst Australian sheep dairies have not been investigated. Data on the frequency and pattern of live animal movements between small ruminant dairies may be available through the National Livestock Identification Scheme database.

Studies in cattle have stated that non-milk-related transmission routes (e.g. respiratory aerosol and/or droplet transmission, shedding in urine) appear to be of little epidemiological relevance, however, cannot be definitively ruled out (Halwe et al. 2024). For example– nasal shedding of infectious genotype B3.13 HPAI virus was observed from non-lactating cattle for up to 7 days following experimental intranasal infection (Halwe et al. 2024; Kalthoff et al. 2008). However, no transmission to sentinel animals was seen. Virus levels in the upper respiratory tract of infected cattle were very low (i.e. 10^1–10^3 $TCID_{50}$ per ml), 1 million times lower compared to those found in milk (Halwe et al. 2024). The infectious dose for cattle is not known, but experimental studies used doses of 10^6 $TCID_{50}$ oronasally or 10^4–10^6 $TCID_{50}$ via the intramammary route (Halwe et al. 2024; Zhou et al. 2024; Baker et al. 2024). Viral RNA has been detected in other cattle samples at low levels, such as urine, ocular swabs, whole blood and serum, although infectious virus has never been recovered and results vary between studies (Caserta et al. 2024; Halwe et al. 2024; Baker et al. 2024; Zhou et al. 2024; Davila et al. 2025). Rectal swabs and faeces have been consistently negative when tested.

Data for small ruminants are limited. Brain and tissue samples from the 5 infected goat kids were reported to be positive, although further details (e.g. tissue types, virus levels, infectious virus vs viral RNA) were not provided (AVMA 2024). In the single sheep infection, viral RNA was detected only in 2 milk samples; nasal swabs, rectal swabs, blood and over 50 tissues samples from organs at post-mortem were negative for viral RNA (HAIRS 2025). However, it is likely that this was a late-stage infection, with the ewe developing mastitis around the 3 March 2025 and sampling occurring on the 10 March, 17 March and 19 March. There was no evidence of transmission to other sheep on the premises in the UK event or to adult goats on the infected premises in the US event.

Thus, for sustained transmission between small ruminants the virus would either need to localise to mammary tissue, as in dairy cattle, and be spread through milk-associated routes (presumably more likely within the small ruminant dairy industries), or the virus would need to acquire several mammalian genetic adaptations to be transmitted efficiently via non-milk associated routes. We consider the likelihood of this to be negligible (i.e. extremely unlikely; may only occur in exceptional circumstances).

Uncertainty: High

The likelihood assessment is complicated by a lack of understanding of the relevant transmission routes in small ruminants and the movement patterns in Australian small ruminant dairy populations. The minimum infectious dose in small ruminants is not yet known. It is not known which secretions and/or excretions from small ruminants are infectious (if any), and how long infectivity can persist in these samples. If other transmission routes such as respiratory spread are found to be epidemiologically relevant, then this may disproportionately affect intensively housed subgroups (e.g. while penned up prior to shearing) compared to, for example– rangeland goats. If milk-associated transmission is relevant in small ruminants, then the dairy industries are likely to be disproportionately affected, although between-herd contacts appear to be negligible (Zalcman and Cowled 2017).

It remains uncertain whether clade 2.3.4.4b HPAI viruses will acquire the necessary adaptations for sustained transmission among mammals, though similar mutations have emerged independently in fur farms, marine mammals, and dairy cattle (Peacock et al. 2024).

2.3 Consequence assessment

What are the consequences of clade 2.3.4.4b HPAI infection in Australian small ruminants?

Consequences: Minor

2.3.1 Rationale

There is a lack of information available regarding the pathogenesis of clade 2.3.4.4b HPAI in small ruminants, making it difficult to assess the health and welfare impacts of infection. In lactating cattle, the mammary gland is the main site of virus replication, which results in necrosis and destruction of milk secreting epithelial cells contributing to the systemic clinical signs seen in lactating animals (Caserta et al. 2024; Peña-Mosca et al. 2025). The recent case of infection in a lactating ewe with mastitis suggests similarities to lactating cattle (noting the sample size of 1) (HAIRS 2025). As the clinical impacts in affected dairy cattle can be quite pronounced, although self-limiting, the health and welfare impacts in lactating sheep could likewise be assumed to be moderate. However, impacts in the meat and fibre sectors are expected to be minimal, unless further mammalian adaptation occurs to increase the efficiency of other transmission routes (e.g. respiratory).

The HPAI-infected goat kids in the US displayed no clinical signs at birth but developed neurological signs between 5 and 9 days of age (APHIS 2024c). Neurological signs are frequently observed in some other HPAI-affected mammals, typically members of the order Carnivora (Peacock et al. 2024). It is not known whether the neurological signs shown by these goats were caused by the HPAI infection, since an additional 5 goat kids showing neurological signs were negative for HPAI (APHIS 2024c). The risk factors contributing to severe disease following clade 2.3.4.4b HPAI infection in small ruminants are not yet known.

Economic impacts for US dairy cattle producers are significant and arise due to decreased milk production, mortality and early herd removal of clinically affected lactating dairy cows (Peña-Mosca et al. 2025). Thus, economic impacts of clade 2.3.4.4b HPAI in small ruminants are anticipated to disproportionately affect small ruminant dairy producers. Transmission would presumably be less efficient in the small ruminant dairy industries compared to the dairy cattle industry, due to the lower density of herds and less frequent movements (e.g. animals, people, vehicles, equipment) between herds. As the Australian small ruminant dairy industries are relatively small, the national-level production impacts of sustained clade 2.3.4.4b HPAI transmission are likely to be minor (APHIS 2024e; Rodriguez et al. 2024). However, the long-term impacts on infection on relevant production metrics (e.g. fertility, liveweight gain) are not known.

Greater economic impacts to industry may arise if export markets are affected. The trade impacts of an incursion of clade 2.3.4.4b HPAI into Australian small ruminants are not known and it is difficult to predict how trading partners would respond. To our knowledge, there have been no reported impacts on sheep or goat trade following the UK and US isolated detections. However, those detections were in non-commercial animals and export of live animals for slaughter and fattening has been banned in the UK since 2024. WOAH has stated that, based on currently available information, restrictions to the international trade of healthy cattle and their products are not recommended unless justified by an import risk analysis (WOAH 2024); current evidence indicates that small ruminants would pose less of a trade risk than dairy cattle.

There is no indication that clade 2.3.4.4b HPAI poses a risk to small ruminant commodity supply. The probability of exposure via meat or offal from infected sheep in the UK was assessed as very low for cooked meat and low if raw meat of offal is consumed, with high uncertainty (HAIRS 2025). The US Department of Agriculture has conducted 3 safety studies around clade 2.3.4.4b HPAI in beef and concluded that the meat supply is safe (APHIS 2025c). The risk to UK consumers from HPAI in US beef products was assessed as negligible and the UK has continued to import fresh and frozen beef from the US throughout 2024 despite the outbreak in US dairy cattle (Browne et al. 2025; Agriculture and Horticulture Development Board 2024). The Government of Canada has stated that HPAI is not a food safety concern when safe food handling, preparation and good hand hygiene are practiced. However, if infectious clade 2.3.4.4b HPAI is found to be present in sheep and/or goat milk, the risk from raw milk or raw milk cheeses to consumers would need to be evaluated. To date, no confirmed cases of human disease due to consumption of clade 2.3.4.4b HPAI-contaminated raw milk or dairy products (from cows or other species) have been reported. However, this remains a theoretical concern and the US Centers for Disease Control and Prevention continues to recommend the consumption of only pasteurised milk and dairy products made from pasteurised milk. While the sale of raw sheep's milk for human consumption is generally illegal in Australia, some states (such as Queensland, New South Wales and South Australia) allow the production and sale of raw goat's milk for human consumption, subject to specific requirements and an advisory statement that the milk is unpasteurised. Raw milk cheeses are permitted to be sold in Australia under strict conditions and approvals. Raw milk is sometimes sold as bath milk or cosmetic milk, although these must be labelled as not for human consumption and are often treated with a bittering agent. The risk of human infection from such products is unknown at this time. Food standard pasteurisation is effective at inactivating clade 2.3.4.4b HPAI in cow's milk and dairy products (Suarez et al. 2025; Caceres et al. 2024; Nooruzzaman et al. 2024; Kwon, Gebhardt, et al. 2024; Spackman et al. 2024; Alkie et al. 2025; Cui et al. 2024; Schafers et al. 2025).

Current evidence suggests that infected dairy (cattle) workers in the US acquired infection through exposure to raw milk (i.e. splashing onto mucous membranes or inhaling contaminated droplets) or through close contact with secretions from clinically affected animals (Morse et al. 2024). Humans may be exposed to raw milk from dairy small ruminants but are unlikely to be exposed to raw milk from meat or fibre small ruminants.

Small ruminants are not known to harbour enzootic IAVs, in contrast to some other species like pigs and humans (MacLachlan et al. 2017). Therefore, the risk of emergence of novel reassortant viruses with pandemic potential is lower than for species such as pigs. Analysis of the haemagglutinin from the US goat HPAI cases did not identify any binding to 'mammalian'-type sialic acids and high fusion pH was retained, suggesting that the virus isolated from goats was unlikely to efficiently transmit between mammals (Yang et al. 2025). Analysis of the publicly available goat viral sequences did not identify key amino acid changes known to be important for mammalian adaptation (e.g. those listed in HAIRS 2025). Due to the low virus levels in the sheep's milk, sequencing to date of that virus has been unsuccessful (HAIRS 2025).

The operational and logistical impacts of a clade 2.3.4.4b HPAI outbreak in small ruminants in Australia are likely to be minor, though this is contingent on the specific response measures chosen and how they are implemented. Following the US clade 2.3.4.4b HPAI detections in goat kids, all species on the premises were quarantined and an epidemiological investigation was conducted. Poultry at the premises were already quarantined from the previous HPAI detection. Samples were collected from the dead goat kids and affected poultry. Non-lethal sampling was conducted on adult goats on the premises. Minnesota's quarantine requirements for HPAI in livestock include movement restrictions on all cattle, poultry, other exposed livestock and susceptible animals (e.g. cats), manure, waste milk and livestock carcasses from the infected premises for at least 30 days. Following the UK clade 2.3.4.4b HPAI detection in a sheep, the affected animal was culled (HAIRS 2025). Additional serological sampling was conducted on the remaining sheep and lambs on the premises. No other specific response measures were reported. In Australia, possible response measures, such as movement restrictions and surveillance approaches, may result in significant industry disruption and socio-economic impacts, if implemented. For example– movement restrictions applied to all susceptible species on a premises for at least 30 days may be severely disruptive to mixed farming operations. The impact of control measures would depend on how many premises are impacted. Depopulation of livestock has not been a feature of responses overseas, and the Australian government is closely monitoring and learning lessons from the management of clade 2.3.4.4b HPAI in the US, Canada and the UK.

Uncertainty: Very high

Potential response measures and trade implications of clade 2.3.4.4b HPAI detections in small ruminants are dependent on the epidemiological context of an outbreak (e.g. number of herds affected, prevalence in other species, prevalence in the importing country). The impacts of infection in small ruminants are unclear; for example – a relationship between infection and neurological signs in the goat kids cannot be completely ruled out and the case of mastitis in the lactating ewe suggests that impacts may be moderate in lactating animals or dairy operations. The long-term consequences of infection for meat and fibre production are not known.

2.4 Risk estimation

Assuming clade 2.3.4.4b HPAI was present in Australia, what is the risk (likelihood and consequences) to Australian small ruminant industries?

Risk: Negligible

2.4.1 Rationale

Taken together, spillovers of clade 2.3.4.4b HPAI into small ruminants can occur but current evidence suggests that these are infrequent relative to exposure (i.e. unlikely but not negligible). Sustained transmission between small ruminants would appear to be unlikely, although this is based on a lack of spread between small ruminants on 2 affected premises. It is not known whether infected lactating small ruminants can transmit infection to suckling lambs or kids, or to other adult small ruminants. While viral RNA was found in the milk from the infected ewe (but not in any other samples), infectious virus was not recovered, although this may have been due to delayed sampling. Viral RNA was detected in multiple tissues from newborn goats, although other factors may have contributed to increased susceptibility in these animals. The assessment may change if non-milk associated transmission routes are shown to be important in small ruminants.

If sustained transmission were to occur in small ruminants, industry-level production impacts are likely to be mild, although the long-term effects of infection on small ruminant production metrics are not yet known. Impacts are more likely to be felt in the sheep and goat dairy industries. Response measures (if implemented) and trade restrictions may result in socio-economic impacts to industries. However, based on the response to the US dairy cattle outbreak, the impacts on trade of dairy cattle and dairy products have been minor, despite the outbreak affecting over 1,072 herds across 17 states.

Uncertainty: Very high

We considered the uncertainty around the overall risk estimate to be very high, based on the very high level of uncertainty for the consequence assessment.

3 Discussion and conclusions

The scope of this work was to conduct a rapid risk assessment for the risk of clade 2.3.4.4b HPAI to Australian small ruminant industries. Rapid risk assessments are conducted over a limited time frame and result in a qualitative assessment of the risk of an event (FAO 2021). They are less comprehensive than a formal risk assessment, which may take months to complete, depending on the methodologies chosen and the challenges faced in gathering data (FAO 2021). A formal qualitative or quantitative risk analysis may be required if more nuanced insights are required (WOAH 2010), such as exploring how risks vary between different sectors of the population (e.g. production systems, breeds). A full assessment of individual entry and exposure pathways was beyond the scope of this analysis.

Overall, the likelihood of entry of clade 2.3.4.4b HPAI (defined as the initial spillover, without onward transmission) into Australian small ruminants was assessed as low, with moderate uncertainty. The likelihood of entry will depend on the geographical distribution and incidence of clade 2.3.4.4b infection in potential spillover host populations, which in turn drives the exposure rate between infected individuals and small ruminants. For the purposes of this assessment, we assumed that the incidence of clade 2.3.4.4b HPAI infection in wild birds was moderate to high and geographically homogeneous across Australia. If infection was additionally present in poultry and/or other mammal populations then risk would likely be higher. A more comprehensive formal risk assessment would be required to analyse specific scenarios of interest.

The likelihood of establishment and spread of clade 2.3.4.4b HPAI in Australian small ruminants was assessed as negligible, with high uncertainty. Exposure to infected milk has been the primary within-herd transmission pathway implicated in cattle. If the biology of infection in small ruminants is similar to cattle, exposure may also occur in the small ruminant dairy sector but is unlikely in the meat and fibre sectors. Importantly, the goat dairy industry is considerably more fragmented than the dairy cattle industry (Zalcman and Cowled 2017), although reliable data are lacking on the frequency of movements (of animals, people, vehicles and equipment) amongst sheep and goat dairy premises. If non-milk related routes are important for transmission between small ruminants, then the likelihood of establishment and spread will be higher.

Spillovers from poultry and/or wild birds are likely to involve avian-adapted strains. Generally, multiple transmission events in a new host species are required for mammalian-adaptive changes to 1) emerge stochastically (i.e. randomly) through error-prone replication, and 2) be selected for within the virus population. However, the evolutionary barrier to some mammalian adaptations appears to be relatively low, since these mutations have emerged rapidly and repeatedly in mammals (Peacock et al. 2024).

The consequences of clade 2.3.4.4b HPAI incursion into the Australian small ruminants were assessed as minor, with very high uncertainty. Production impacts are likely to be negligible in most contexts. However, the trade restrictions and response measures that may be implemented in response to detections in Australian small ruminants are not yet known.

Taken together, the risk of clade 2.3.4.4b HPAI to Australian small ruminant industries was assessed as negligible, with very high uncertainty. The uncertainty in the overall assessment was driven by uncertainty around the impacts of potential response measures and trade implications, as well as the significant lack of information available regarding the pathogenesis in small ruminants. We have assumed for this assessment that the virus biology does not differ substantially to current clade 2.3.4.4b HPAI viruses (see Section 1.2); however, the continued evolution of clade 2.3.4.4b HPAI and the potential for acquisition of mammalian-adaptive mutations introduces significant uncertainty regarding its broader risk to mammalian species, beyond just small ruminants.

3.1 Limitations

The risk assessment was subject to the following limitations:

- Data are limited to 6 known clade 2.3.4.4b HPAI infections in small ruminants: 1 in a lactating sheep and 5 in newborn goat kids with concurrent disease processes. Therefore, the health, welfare and long-term impacts of infection in small ruminants are not yet understood.
- There are no data on breed differences (e.g. dairy vs meat or fibre animals, Boer vs rangeland goats). This is important information in the Australian context.
- The role of non-milk associated transmission in small ruminants is unclear. If respiratory transmission or ingestion is identified as relevant to within-herd and between-herd disease spread in small ruminants, the risk may be increased.
- We assumed that the risk of spillover was equal across all of Australia. A more formal assessment would need to be conducted to look at how risk differs across different industry sectors (e.g. pasture-based systems, unmanaged rangeland systems, lifestyle farms, small ruminant dairies) or across geographical regions.
- Response strategies for clade 2.3.4.4b HPAI in small ruminants have not been developed so the industry-level impacts of response measures are not clear.
- It is difficult to predict how trading partners may respond to detections of clade 2.3.4.4b HPAI in Australian small ruminants.
- The choice of framework used may influence the risk estimate, for example – depending on the likelihood and consequence categories and definitions used. For this assessment, we followed previous methodology used by the Food and Agriculture Organization of the United Nations and Wildlife Health Australia (see Section 1). Our definitions may differ from those used by other organisations for specific purposes, such as the World Trade Organization.

3.2 Recommendations

There have only been limited cases of natural infection with clade 2.3.4.4b HPAI in small ruminants. Therefore, many of the recommendations focus on general enhancements to biosecurity and have been extrapolated from recommendations to dairy producers (APHIS 2024b; AABP 2024; APHIS 2024c).

3.2.1 Reduce the likelihood of entry into small ruminants

1) Current spillovers into small ruminants have most likely been via infected poultry or direct contact with large numbers of sick and dead wild birds. Therefore, avoid co-mingling small ruminants and poultry and limit contact with potentially contaminated environments, especially at lambing and kidding.
2) Where possible, prevent (or limit) wild bird and poultry access to small ruminant feed, pasture, feed storage, water sources, bedding materials and facilities.
3) Avoid sharing (unclean) equipment or vehicles with poultry (and dairy cattle) farms.
4) Avoid small ruminant access to poultry by-products (e.g. poultry litter used as fertiliser).
5) Visitors from poultry and dairy farms should be subject to enhanced visitor entry protocols (e.g. stand-down period, site-specific clothing).
6) Humans with influenza-like illness or conjunctivitis should not have contact with small ruminants.
7) Monitor for and report any unexpected mortality or behaviours in wild birds or domestic or wild animals.
8) Manage standing water bodies. For example – limit wild bird access if possible, minimise small ruminant access if not required as a water source, improve drainage if not required.
9) Where possible, prevent (or limit) peri-domestic mammal access to feed, feed storage, water sources, bedding materials and facilities (e.g. rodent control, manage farm cats, feral animal control, avoid co-mingling livestock species).

3.2.2 Reduce the likelihood of transmission within the industry

10) Minimise unnecessary animal movements and keep detailed movement records, particularly between small ruminant dairy premises.
11) Only move healthy animals and isolate new arrivals for at least 21 days upon arrival (AABP 2024). Some resources state 30 days.
12) Monitor animals for signs of illness and isolate sick animals. Consider HPAI as a differential diagnosis for non-specific illness, reduced milk production and mastitis in small ruminants.
13) Raising awareness of HPAI amongst producers, particularly around disease recognition and the importance of notification and investigation of clinical cases, will help to increase the sensitivity of passive surveillance (Sergeant et al. 2022).
14) Avoid moving (unclean) equipment or vehicles between premises, particularly between small ruminant dairies.

15) Use dedicated routes for vehicles that do come onto the premises (and avoid direct contact with animals).

16) Limit non-essential visitors, maintain a visitor logbook and establish visitor entry protocols (e.g. site-specific clothing).

17) Jurisdictions and the Commonwealth should establish testing protocols for non-avian species for HPAI, to facilitate testing of suspect cases. Producers must be informed on how to access testing.

18) In the event of an incursion, serosurveillance to understand the prevalence and geographical distribution of infection may be warranted in specific circumstances. However, this should be guided by a more detailed evidence-based active surveillance strategy and cost-benefit analysis.

3.2.3 Reduce impacts

19) Response strategies in non-avian species should be considered now (i.e. in peacetime) and clearly communicated so that industry can better understand the likely impacts of potential control measures. Empowerment of farmers and producer cooperation is critical to optimising both active and general surveillance systems (i.e. for rapid investigation and reporting of outbreaks) (Gates et al. 2021).

20) Consider the use of personal protective equipment (such as gloves, apron, respiratory protection and eye protection) in certain circumstances (e.g. during milking in small ruminant dairies or when in close contact with secretions from sick animals like during drenching).

21) Do not consume raw milk, colostrum or raw milk products from small ruminants. Food standard pasteurisation is effective at inactivating clade 2.3.4.4b HPAI in milk and dairy products.

22) In the event of an incursion, viral genome sequencing should be used to monitor for genetic changes that may indicate mammalian adaptation.

Appendix A: Influenza virus nomenclature

There are 4 types (genera) of influenza viruses: A, B, C and D (MacLachlan et al. 2017).

1) Influenza A viruses (IAVs) infect birds and some mammals, as well as causing seasonal flu in humans.
2) Influenza B viruses also cause seasonal flu in humans and can infect certain mammal species, but not birds.
3) Influenza C viruses infect humans and pigs.
4) Influenza D viruses infect pigs and cattle.

Within the IAVs, viruses are frequently grouped by either 1) their pathogenicity in domestic poultry (i.e. high and low pathogenicity avian influenza), or 2) based on the key surface proteins of the virus, haemagglutinin (H) and neuraminidase (N). There are currently 18 recognised H types and 9 recognised N types (Sreenivasan et al. 2019). While all H subtypes can exist as LPAI viruses, only H5 and H7 can become HPAI viruses (MacLachlan et al. 2017).

Within a given IAV subtype (e.g. H5), there can be many different lineages or clades (e.g. clade 2)—that is, not all H5s are the same. Over time, as these lineages continue to transmit and evolve, these clade names can be made more specific (e.g. clade 2.3.4.4b). Importantly, these lineage or clade names only refer to the H genetic segment. Because influenza viruses are segmented viruses, as well as mixing the H and N genetic segments they can also mix the other 6 segments. This mixing in IAVs is referred to as reassortment and ‘mixed’ viruses are referred to as reassortants.

An IAV *genotype* refers to the full gene constellation of all 8 genetic segments. That is, clade 2.3.4.4b represents many different gene constellations, all with the same clade 2.3.4.4b H segment. While many biological properties of IAVs depend primarily on the H gene segment (e.g. receptor binding, antibody and vaccine evasion), biological properties can also vary between genotypes due to variation in the other genetic segments.

Glossary

Term	Definition
HPAI	high pathogenicity avian influenza
IAV	influenza A virus
$TCID_{50}$	50% tissue culture infectious doses

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