Risk of high pathogenicity avian influenza to Australian beef cattle: 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 assessment was conducted based on information available up to 6 March 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 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 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, followed by ongoing transmission among dairy cows. Subsequently, 2 more spillovers into dairy cattle were detected in January and February 2025, respectively (APHIS 2025a, 2025b; CIDRAP 2025; APHIS 2025d; AZDA 2025). Genotype D1.1 was identified in both of those events; however, virus sequences from each event are genetically distinct, indicating 2 separate spillovers (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 event was attributed to a single dairy farm (AZDA 2025). Investigations are ongoing to fully characterise these new spillovers. Critically, the definitive spillover host and transmission pathway into dairy cattle are not known for any of the spillover events. While wild birds are generally implicated, both genotypes have also been 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 transmission of these genotypes within mammal species other than cattle.

As of 6 March 2025, the ongoing HPAI outbreak in dairy cattle has now affected over 978 dairy farms across 17 states (APHIS 2024i). Detections of clade 2.3.4.4b HPAI in the US have also been confirmed in other livestock, including in pigs (genotype D1.2), goats (genotype B3.6) and alpacas (genotype B3.13) (APHIS 2024f, 2024k; AVMA 2024; APHIS 2024h).

While this assessment was conducted based on information available up to 6 March 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 has heightened concerns about the potential risk of an epizootic to not only the Australian dairy industry, but also to other livestock 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 cattle to inform an evidence-based rapid risk assessment (RRA) for the Australian beef cattle industries.

Risk questions

This RRA addresses these risk questions:

1) Assuming clade 2.3.4.4b HPAI was present in Australia, what is the risk (likelihood and consequences) to the Australian commercial beef cattle industry?

- 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 commercial beef animal in Australia in the next year?
- b) Establishment and spread assessment: If clade 2.3.4.4b HPAI were to infect 1 or more commercial beef animals in Australia, what is the likelihood of spread within and between commercial beef production premises in the next year?
- c) Consequence assessment: What are the consequences of clade 2.3.4.4b HPAI infection in the Australian beef cattle industry?

Overall assessment

Overall, assuming clade 2.3.4.4b HPAI was present in Australia, the risk to commercial beef cattle industry was assessed as negligible, with 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 commercial beef animal in Australia was assessed as low, with low to moderate uncertainty.

Despite high levels of HPAI circulation in wild bird and poultry populations globally, spillovers of IAVs into cattle remain rare, even in environments with high cattle exposure (e.g. agricultural operations with significant bird presence). Current evidence points to 3 spillovers of clade 2.3.4.4b HPAI into dairy cattle, which have so far been restricted to the US.

Spillovers of clade 2.3.4.4b HPAI have not been detected in dairy cattle populations outside the US where active surveillance is being conducted. For example – Canada, Germany, Pakistan and the United Kingdom have conducted targeted surveillance for HPAI in dairy cattle with no positive detections.

There have been no reports of clade 2.3.4.4b HPAI infections in US beef cattle despite the widespread outbreak in dairy cattle, although active surveillance is not being conducted.

Establishment and spread assessment

The likelihood of clade 2.3.4.4b HPAI establishing and spreading within and between commercial beef production premises was assessed as negligible, with low uncertainty.

Sporadic IAV infections (with various HxNx subtypes and genotypes) have been recognised in cattle for decades, without documented mammal-to-mammal transmission.

There are several barriers that avian-adapted IAVs must overcome to transmit efficiently amongst mammalian hosts. Generally, multiple infection events in a new host species are required for mammalian-adaptive genetic mutations to emerge and establish (become fixed) in a virus population. Dairy cattle are an exception because 'avian-type' receptors (α -2,3-linked sialic acids) are abundantly expressed in the mammary gland. Therefore, minimal adaptation is required for avian-adapted viruses to spread between lactating dairy cows.

The mammary gland is the main site of virus replication in dairy cattle. While non-lactating cattle can be infected with clade 2.3.4.4b HPAI, the level of virus in non-lactating cattle is much lower than in lactating dairy cows.

Within-herd and between-herd transmission of IAVs in US dairy cattle is thought to be primarily through exposure to unpasteurised (raw) milk (i.e. direct contact with either clinically infected or subclinical animals or contact with milk during milking or via contaminated fomites). Adult beef cattle generally have limited exposure to milk from other animals.

Non-milk-related transmission routes appear to be of little epidemiological relevance in cattle (but cannot be definitively ruled out). For example – no onward transmission was observed in experimental infections of non-lactating cattle with clade 2.3.4.4b viruses.

Consequence assessment

The consequences of clade 2.3.4.4b infection in the Australian beef cattle industry were assessed as minor, with high uncertainty.

Experimental infection studies have demonstrated that clinical disease following clade 2.3.4.4b infection in non-lactating cattle is mild and short-lived (at least with those genotypes investigated). Morbidity in infected dairy herds is generally between 3 and 20% (primarily restricted to lactating cows), although anecdotal reports from California suggest that up to 50 to 60% of some herds can be clinically affected.

Impacts on beef cattle production at an industry level are anticipated to be minor, since mortality in dairy cattle (at least with genotype B3.13) has been low on average (less than 2%). Anecdotally, herd-level mortality rates of up to 20% in some Californian dairy herds have been reported in the news media, but this has not been confirmed by official sources. These mortality rates were also reported during extreme heat waves.

Significant economic impacts to the dairy industry are due to decreased milk production, mortality and early herd removal. These impacts are primarily related to clinically affected lactating cows. Milk is not a major commodity for the beef cattle industry.

Importantly, the impacts of HPAI infection on long-term liveweight gain, fertility and other metrics relevant to beef cattle production have not been investigated.

Public health consequences are negligible, since humans are unlikely to be exposed to milk from beef cattle. Current evidence suggests that infected dairy workers acquired infection through exposure to raw milk or through close contact with secretions from clinically affected animals.

The potential trade impacts of clade 2.3.4.4b HPAI infection in the Australian beef industry 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 beef cattle. Australia relies heavily on access to premium export markets. Trade impacts are more likely to affect live cattle movements than animal products.

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

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.

Recommendations

Key recommendations include:

- 1) To reduce the likelihood of entry into beef cattle:
 - a) Where possible, limit direct contact with wild birds, poultry and peri-domestic wildlife (e.g. rats, foxes, feral cats).
 - b) Where possible, prevent or limit access of wild birds, poultry and peri-domestic wildlife to livestock feed, feed storage, water sources, bedding material, facilities and equipment.
 - c) Avoid cattle access to poultry by-products (e.g. poultry litter or manure used as fertiliser).
 - d) Avoid co-mingling cattle and poultry.
 - e) Avoid sharing (unclean) equipment or vehicles with poultry (and dairy) farms.
- 2) To reduce the likelihood of transmission within the industry:
 - a) Maintain good farm biosecurity (e.g. minimise animal movements, pro-actively manage movement of people, equipment and vehicles).
 - b) Enhance passive surveillance (e.g. monitor for sick livestock, wild birds or wildlife; consider HPAI as a differential diagnosis for unexplained illness).
 - c) Jurisdictions and the Commonwealth should establish testing protocols for HPAI in non-avian species to facilitate testing of suspect cases.
 - d) 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. This would need to be assessed within the specific context of an incursion. To the best of our knowledge, the US is not currently conducting active surveillance in beef cattle (APHIS 2024e).
- 3) To reduce impacts:
 - Response strategies in non-avian species should be considered now (i.e. in peacetime) and clearly communicated so that industries can better understand the likely impacts of potential control measures.
 - b) Consider the use of personal protective equipment (e.g. gloves, apron, respiratory protection and eye protection) in certain circumstances (such as managing sick animals) to reduce the risk of human infection.

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Introduction

This assessment was conducted based on information available up to 6 March 2025.

Clade 2.3.4.4b HPAI in dairy cattle

In 2020, a novel clade 2.3.4.4b high pathogenicity avian influenza (HPAI) virus of the H5N1 subtype (Appendix A) emerged that caused 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 been reported in 25 mammalian families and 87 species as of 14 February 2025 (Peacock et al. 2024; 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).

Influenza A viruses (IAVs) have been recognised to cause sporadic infections in cattle for many decades (Gunning and Pritchard 1997; Sreenivasan et al. 2019; Lopez and Woods 1984; Mostafa et al. 2024). However, there was no robust evidence for sustained transmission between cattle prior to 2024. Then 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 Texas, New Mexico and Kansas (Caserta et al. 2024; Oguzie et al. 2024). 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. Virus sequences also contained genetic changes suggestive of mammalian adaptation (Dholakia et al. 2025).

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, 2025b; CIDRAP 2025; APHIS 2025d; AZDA 2025). Virus sequences from each event are genetically distinct, indicating 2 separate spillovers (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 event was attributed to a single dairy (AZDA 2025). Investigations are ongoing to fully characterise these new spillovers. Importantly, the definitive spillover hosts and transmission pathways into dairy cattle remain unknown. While wild birds are often implicated (e.g. 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 alternative entry pathways are possible. Importantly, there is no evidence for transmission of these genotypes in mammalian species other than cattle.

In dairy cattle, IAV infection is localised to the mammary tissue and the virus is shed in extremely high concentrations in milk, with peak titres exceeding 10^9 50% tissue culture infectious doses (TCID₅₀, a measure of the amount of virus) per ml of milk (Mitchell et al. 1954, 1953; Halwe et al. 2024; Caserta et al. 2024). The infectious dose for cattle is not known, but experimental studies have

used doses of 10^6 TCID₅₀ oronasally or 10^4 – 10^6 TCID₅₀ via the intramammary route (i.e. theoretically 1 μ l or less of milk) (Halwe et al. 2024; Zhou et al. 2024; Baker et al. 2024). Clade 2.3.4.4b HPAI virus in raw (unpasteurised) milk has been shown to remain infectious for at least 1 hour on stainless steel and rubber at 22°C, and for at least 7 days at 4°C (Le Sage et al. 2024; Kaiser et al. 2024). Therefore, 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).

The initial site of virus entry remains unknown but is possibly through the respiratory or oral route with low-level replication in the upper respiratory tract and subsequent systemic spread to the mammary glands (Caserta et al. 2024). Direct infection of the mammary gland through the teat orifice and cisternae (with or without systemic dissemination) has also been proposed (Caserta et al. 2024). Intramammary inoculation via the teat canal (in lactating cattle) and oronasal infection with aerosolised virus (in non-lactating cattle) have been demonstrated experimentally (Halwe et al. 2024; Zhou et al. 2024; Baker et al. 2024). Additionally, calves can be infected by ingesting raw milk from infected cows (Davila et al. 2025).

Within-farm transmission of HPAI appears to be primarily through contact with milk at milking (Halwe et al. 2024; Zhou et al. 2024). Scientific studies have stated that respiratory transmission is not likely to be epidemiologically relevant, although it cannot be completely ruled out (Halwe et al. 2024). For example – infectious virus has been recovered from nasal swabs of experimentally infected non-lactating cattle; however, virus levels were very low compared to those found in milk (10¹–10³ TCID₅₀ per ml, i.e. 1 million times lower) (Halwe et al. 2024). Viral RNA has been detected in other 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.

Epidemiological analyses of the US dairy outbreak suggested that between-farm spread of HPAI is primarily driven by the movement of infected lactating cows, including subclinical animals (APHIS 2024g, 2024j; AVMA 2025). The movement of equipment and other fomites contaminated with milk (particularly milking equipment) is a proposed risk factor (APHIS 2024g). Personnel, visitors, and potentially other animals contaminated with milk may also act as fomites (CEZD 2024). However, there is currently no evidence currently to suggest that wild birds are driving spread between farms (APHIS 2024g; AVMA 2025).

Current national-level control measures in the US have focused on (APHIS 2024j):

- mandatory pre-movement testing of lactating cattle being transported interstate
- enhancing surveillance through awareness-raising and provision of free laboratory testing
- enhancing on-farm biosecurity through the development of guidelines for producers
- a voluntary dairy herd status program utilising bulk milk testing to declare unaffected herds
- national monthly testing of milk silos at dairy processing facilities to identify where disease is present, although not all states are participating.

Additionally, field trials to evaluate H5N1 vaccine candidates in dairy cows are currently underway (APHIS 2024j). Some states have implemented their own mandatory testing programs. For example – from July 2024 <u>Colorado</u> has required weekly sampling of bulk-tank milk from all dairy farms. Since June 2024 <u>lowa</u> has required testing of dairy herds within a 20-km radius of HPAI-infected poultry farms.

The ongoing HPAI outbreak in dairy cattle has now affected over 978 dairy farms across 17 states (APHIS 2024i). While herd-level mortality is widely reported to be low on average (less than 2%), decreased milk production can persist for at least 1-2 months (Caserta et al. 2024; Rodriguez et al. 2024; Baker et al. 2024; APHIS 2024g). Anecdotally, herd mortality rates of up to 20% in some Californian dairy herds has been reported in the news media, but this has not been confirmed by official sources (Douglas 18 October 2024; Rust 4 October 2024). These mortalities may have been exacerbated by extreme heat events (greater than 35°C) and it is unclear what proportion are attributable directly to HPAI infection compared to other diseases secondary to heat stress (Douglas 18 October 2024; Rust 4 October 2024). One case report from an affected dairy in Ohio reported a herd-level mortality of 1.4% and a case fatality rate of 6.8% (the percentage of clinically affected animals that died or were euthanised) (Peña-Mosca et al. 2025). Serological investigations on the same farm indicated a large proportion (83.7%) of subclinical infections, meaning that the infection fatality rate (the percentage of infected animals that died or were euthanised) was much lower than the case fatality rate. The risk of clinical disease in non-lactating cows was negligible (~0.1%) (Peña-Mosca et al. 2025). The long-term impacts of HPAI infection on dairy cattle productivity are not yet known. The American Association of Bovine Practitioners (AABP) has estimated the economic impact of HPAI for dairy cattle to be USD \$100 to \$200 per infected cow (Larkin 2024). The case study in Ohio reported economic losses due to decreased milk production, mortality and early herd removal of \$950 per clinically affected cow (Peña-Mosca et al. 2025). Some export markets have been affected in response to the outbreak; for example – Canada now requires strengthened import controls on dairy cattle from the US (AVMA 2025). The economic impacts of the outbreak are not yet clear, however, following the detection in dairy cows, USD \$200 million was allocated to the federal response (Larkin 2024).

The outbreak also presents a risk to human health—41 of the 70 human HPAI cases in the US have been attributed to exposure to infected dairy herds (CDC 2025). In addition, clade 2.3.4.4b HPAI has been detected in several other livestock species in the US, including in pigs (genotype D1.2), goats (genotype B3.6) and alpacas (genotype B3.13) (APHIS 2024f, 2024h; AVMA 2024; APHIS 2024k). This wholly unexpected outbreak has heightened concerns about the potential risk of an HPAI epizootic to not only the Australian dairy industry, but also to other livestock 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 cattle to conduct an evidence-based rapid risk assessment (RRA) for the Australian beef cattle industries.

Australian beef cattle industries

Australia has one of the largest and most efficient beef cattle industries in the world, contributing significantly to the national economy. The Australian beef herd comprised almost 28 million head of cattle in June 2023, distributed across over 53,000 agricultures businesses (MLA 2024). In 2023, approximately 2.2 million tonnes carcase weight of beef and veal was produced, with industry

turnover of the beef cattle farming sector valued at \$25.3 billion. Australia was the world's second largest beef and bovine meat exporter in 2023, accounting for almost 3% of global beef production (MLA 2024).

Production systems include southern pasture-based and northern extensive rangeland-based cowcalf systems, as well as feedlot systems, where animals are backgrounded on pasture and finished in feedlots (Greenwood et al. 2018). The northern system is characterised by vast properties with minimal intervention and low stocking densities, due to a reliance on relatively low quality pasture that is dependent on monsoonal rainfall (Greenwood et al. 2018). These enterprises typically utilise *Bos indicus* breeds, due to their heat tolerance, tick resistance and hardiness. Production is mostly focused on the live export market.

Southern systems are generally more winter rainfall-dominant and use native and improved pastures and forages, which enable higher stocking densities (Greenwood et al. 2018). These systems may be set-stocked or use rotational grazing. *Bos taurus*, especially British breeds such as Angus and Hereford, are typical (Greenwood et al. 2018). Breeding is generally closely managed, with calving in autumn or spring depending on feed availability and weaning at 4 to 9 months of age (Greenwood et al. 2018). Production is geared towards cattle for the domestic market and for beef exports (Greenwood et al. 2018). Dairy or dairy-cross cattle for slaughter are also an important component of the southern Australian beef industry (Greenwood et al. 2018).

Around a third of Australian cattle are transitioned to a feedlot for finishing (MLA 2025). Feedlots are characterised by higher stocking densities and rapid turnover of the population, with cattle typically spending 50 to 120 days on feed (MLA 2025). In 2022–23, there were approximately 350 feedlot businesses, with 2.8 million grain-fed cattle turned off in 2023–24 (MLA 2024).

Risk questions

This RRA addresses these risk questions and sub-questions:

- 1) Assuming clade 2.3.4.4b HPAI was present in Australia, what is the risk (likelihood and consequences) to the Australian commercial beef cattle industry?
 - a) Entry assessment: What is the likelihood of clade 2.3.4.4b HPAI spilling over into at least 1 commercial beef animal in Australia in the next year?
 - b) Establishment and spread assessment: If clade 2.3.4.4b HPAI were to infect 1 or more commercial beef animals in Australia, what is the likelihood of spread within and between commercial beef production premises in the next year?
 - c) Consequence assessment: What are the consequences of clade 2.3.4.4b HPAI infection in the Australian beef cattle industry?

1 Methods

We previously (in 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.

Our assessment is based on 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 a beef animal or animals.
- Establishment and spread: Onward transmission of clade 2.3.4.4b HPAI virus in beef cattle, 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

of spread	High	Low	Moderate	Moderate	High
od c	Moderate	Low	Low	Moderate	Moderate
Likelihood of establishment and s	Low	Negligible	Low	Low	Moderate
establ	Negligible	Negligible	Negligible	Low	Low
	·	Negligible	Low	Moderate	High
			Likelihoo	d 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

	High	Negligible risk	Low risk	Moderate risk	High risk
pooqi	Moderate	Negligible risk	Low risk	Moderate risk	High risk
Likelihood	Low	Negligible risk	Low risk	Low risk	Moderate risk
	Negligible	Negligible risk	Negligible risk	Negligible risk	Negligible risk
	·	Negligible	Minor	Moderate	Severe
			Consequenc	es	

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 May 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 beef cattle could make contact with an infected wild bird (or other infected species) and the probability of this is equal across all of Australia.
- That risk was equal across different industry sectors (e.g. extensive systems, pasture-based systems, feedlots, lifestyle farms) and cattle breeds (e.g. Bos taurus vs Bos indicus). A more

comprehensive formal risk assessment would be required to analyse how risk may vary across different subpopulations.

- 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 reasonable biosecurity is practiced by commercial herds, such as not feeding cattle raw milk or poultry carcasses or by-products.
- That the biology (including pathogenesis, virulence and transmissibility) of any virus entering Australia is not substantially different to the genotype B3.13 virus.
- That the pathogenesis of HPAI virus infection is not substantially different between beef cattle
 and dairy cattle. Critically, the risk will vary depending on whether an incursion strain is avianadapted 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 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 commercial beef animal in Australia in the next year?

Likelihood: Low

2.1.1 Rationale

Spillover of IAVs into cattle is possible but appears to be uncommon. Current phylogenetic and epidemiological evidence points to 3 spillovers of clade 2.3.4.4b HPAI into dairy cattle in the US (Worobey et al. 2024; Caserta et al. 2024; APHIS 2025a). Other clade 2.3.4.4b HPAI genotypes have spilled over into other domestic species. For example—genotype D1.2 in poultry and swine, and genotype B3.6 in poultry and goats (APHIS 2024f, 2024k; CDC 2024). There have been no reports of clade 2.3.4.4b HPAI infections in US beef cattle despite the widespread outbreak in dairy cattle, although active surveillance is not being conducted.

Clade 2.3.4.4b HPAI infection has not been reported in dairy cattle populations outside the US, despite epizootic circulation of clade 2.3.4.4b HPAI for several years and active surveillance in some countries. For example – Canada has tested over 1,000 samples of pasteurised retail milk and almost 1,500 samples of raw (unpasteurised) milk at processing plants since mid-2024 with no positive detections (CFIA 2024; Wallace et al. 2025). No evidence for H5N1 infection was found in dairy cattle or farm workers in Punjab province, Pakistan, in a study conducted from January 2023 to March 2024 (Ahmed et al. 2024). In June 2024, approximately 1,400 bovine serum samples collected from regions in Germany particularly affected by avian HPAI outbreaks had been tested for antibodies to IAV, with no positive detections (Friedrich-Loeffler-Institut 2024). Additionally, around 350 bulk milk tank samples from Germany were tested for viral RNA, with negative results in all cases. From May to June 2024, a cross-sectional survey was conducted across 455 dairy farms in England, Scotland and Wales (Animal and Plant Health Agency 2024). No evidence for clade 2.3.4.4b HPAI infection was found in 508 bulk milk samples from these farms. Importantly, we are not aware of any active surveillance in beef cattle in any country.

There has always been a risk of spillover of IAVs into cattle—this is not a unique feature of genotype B3.13, D1.1 or clade 2.3.4.4b viruses. IAVs shown to infect cattle have included H1N1, clade 2.2 H5N1, a European clade 2.3.4.4b H5N1, H3N2 and other H1 and H3 subtypes. Sporadic IAV infections in cattle have been reported from Japan, the United Kingdom, Hungary and Russia (Saito 1951; Gunning and Pritchard 1997; Lopez and Woods 1984; Sreenivasan et al. 2019; Mostafa et al. 2024; Kalthoff et al. 2008; Halwe et al. 2024). Yet spillovers have been detected very infrequently (that is, every decade or so). The source of these infections was not discussed in the case reports.

Without knowing the spillover host and entry pathways, it is difficult to assess the frequency of spillover relative to exposure. For the current clade 2.3.4.4b HPAI dairy cattle outbreak, speculation has mostly focused on exposure to wild birds (Caserta et al. 2024; CEZD 2024).

For B3.13, prior to detections in cattle, 4 closely related virus sequences were found in wild animals: 2 in waterfowl (in Colorado and Wyoming), 1 in a peregrine falcon in California, and 1 in a skunk in New Mexico (USDA Veterinary Services 2025; Butt et al. 2024; Caserta et al. 2024), suggesting spillover of B3.13 from a wildlife host (probably wild birds but mammals can't be ruled out). However, during the same period there were 269 HPAI detections in commercial and backyard poultry in the US, for which sequencing information is not available (APHIS 2024c). Therefore, spillover from poultry to cattle (either directly or indirectly) must also be considered as a possibility. Of note, a small number of US dairy operations feed poultry meal to cows (Mcdougal 25 June 2024; USDA 2016), which is illegal in Australia (AHA 2023). Poultry litter may also be used as a fertiliser, both in the US and in Australia.

For D1.1, this genotype was the predominant genotype in wild birds in the North American flyways at the time of spillover and has also been identified in mammals, domestic poultry and humans (APHIS 2025d; CDC 2024; USDA Veterinary Services 2025).

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

Uncertainty: Low to moderate

Previous spillovers (into either dairy or beef cattle) without sustained cow-to-cow transmission may have gone undetected, leading to underestimation of the likelihood. However, active surveillance of non-US dairy herds is being conducted in some areas (e.g. Canada, Germany, Pakistan, United Kingdom). Given the lack of clinical signs in non-lactating cattle, it is possible that widespread infection could be unrecognised.

2.2 Establishment and spread assessment

If clade 2.3.4.4b HPAI were to infect 1 or more commercial beef animals in Australia, what is the likelihood of spread within and between commercial beef production premises in the next year?

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 required for mammalian adaptation typically require multiple transmission events in mammals (and sustained selection pressure) (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.

IAVs in cattle localise to the mammary gland, with very high virus levels in milk. 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 2024g; Peacock et al. 2024; Halwe et al. 2024; Zhou et al. 2024). Subclinical animals can shed virus in milk and

therefore also pose a transmission risk (Caserta et al. 2024). Beef cattle (apart from calves) are rarely exposed to raw milk from other cattle. Calves can be infected by ingesting raw milk from infected cows (Davila et al. 2025).

Scientific studies have stated that non-milk-related transmission routes 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 were very low (i.e. 10^1 – 10^3 TCID₅₀ 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 have used doses of 10^6 TCID₅₀ oronasally or 10^4 – 10^6 TCID₅₀ via the intramammary route (Halwe et al. 2024; Zhou et al. 2024; Baker et al. 2024). Viral RNA has been detected in other 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). Further studies are required to investigate the infectivity of these sample types. Rectal swabs and faeces have been consistently negative when tested.

Current genomic and epidemiological evidence do not support that wild or peri-domestic birds are spreading HPAI between cattle herds (APHIS 2024g). However, mechanical vectoring by wild birds cannot be ruled out (e.g. if they were contaminated with raw milk or other infectious material such as poultry litter) (Caserta et al. 2024). Similarly, mechanical carriage between cattle herds by other domestic and peri-domestic animals (and humans) cannot be ruled out, although there is no current evidence to directly support this. A recent report suggested that dairy workers may have spread virus from infected dairy herds to indoor domestic cats (Naraharisetti et al. 2025). However, it is impossible to draw robust conclusions from this study because the dairy workers declined testing, there was no testing of other cats in the households, and other exposure sources were not investigated (e.g. feeding of raw food or raw milk to cats).

Thus, for sustained transmission between beef cattle the virus would need to acquire several mammalian genetic adaptations and beef cattle would need to be exposed to an infectious dose of virus, which is currently thought to be limited to exposure to raw milk. We consider that the likelihood of this is negligible (i.e. extremely unlikely; may only occur in exceptional circumstances).

If onward transmission was possible then response measures, risk mitigation measures (e.g. on-farm biosecurity) and cattle movement patterns will influence the risk and speed of spread.

Uncertainty: Low

The roles of non-lactating cattle and non-milk associated transmission routes in HPAI spread are poorly understood. Relatedly, the minimum infectious dose in cattle is not yet known. If other transmission routes, such as respiratory spread, are found to be epidemiologically relevant, then this may disproportionately affect certain subgroups (e.g. feedlot cattle).

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). The more mammalian infections

that occur, the more opportunities for these adaptations to emerge. However, we do not factor this into our uncertainty assessment because we have assumed that the virus biology does not differ substantially to the genotype B3.13 virus (see Section 1.2).

2.3 Consequence assessment

What are the consequences of clade 2.3.4.4b HPAI infection in the Australian beef cattle industry?

Consequences: Minor

2.3.1 Rationale

The mammary gland is the main site of virus replication, which results in necrosis and destruction of milk secreting epithelial cells likely leading to the systemic clinical signs seen in lactating animals (Caserta et al. 2024; Peña-Mosca et al. 2025). Clinical signs in affected dairy cows include reduced feed consumption, abnormal milk (e.g. thickened, clotted milk with yellow discolouration), gastrointestinal signs (e.g. decreased rumen motility), fever, lethargy and dehydration (APHIS 2024g; Baker et al. 2024; Caserta et al. 2024; Oguzie et al. 2024; Burrough et al. 2024; Rodriguez et al. 2024; El Masry et al. 2024; Halwe et al. 2024). Some farms also reported an association with respiratory signs and abortion (APHIS 2024g; Rodriguez et al. 2024). While clinical signs can be quite severe in lactating dairy cows, animals typically recover within 5 to 17 days, with decreased milk production persisting for 1–2 months (Caserta et al. 2024; Rodriguez et al. 2024; Baker et al. 2024; APHIS 2024g). One case study reported that the risk of clinical disease in non-lactating cows was negligible (~0.1%) (Peña-Mosca et al. 2025).

With sustained cow-to-cow transmission in US dairy cows, morbidity in infected herds is generally between 3 and 20% (Caserta et al. 2024; Oguzie et al. 2024; Burrough et al. 2024). The apparent between-herd prevalence ranged from 2.7% to 58% for affected states, although this continues to be an evolving situation (Rodriguez et al. 2024). Morbidity is likely to be lower in beef cattle where milk-associated transmission will be less important, unless further mammalian adaptation occurs to increase the efficiency of other transmission routes (e.g. respiratory).

Herd-level mortality is typically low (less than 2%), although above-average mortality (2-fold higher) was noted on some farms (Caserta et al. 2024). Anecdotally, mortality up to 20% in some Californian dairy herds has been reported in the news media, but this has not been confirmed by official sources (Douglas 18 October 2024; Rust 4 October 2024). This may have been exacerbated by atypical extreme heat events (greater than 35°C).

If we assume that IAVs in non-lactating cattle may behave similarly to influenza D virus, then the animal health consequences can be considered minor to negligible. However, we note that this is a major assumption and these viruses are separate genera, with potentially very different biology. When considering the consequences of influenza D virus to beef cattle, its significance as a primary pathogen remains unclear (MacLachlan et al. 2017). In endemic areas, influenza D virus is commonly detected in cattle with respiratory disease as part of the bovine respiratory disease complex (MacLachlan et al. 2017). Seroprevalence studies suggest that many cattle may be infected without showing clinical signs.

Significant economic impacts to the dairy industry are due to decreased milk production, mortality and early herd removal (Peña-Mosca et al. 2025). These impacts are primarily related to clinically affected lactating dairy cows. Since milk is not a major commodity for the beef cattle industry and mortality is typically low, industry-level impacts of sustained clade 2.3.4.4b HPAI transmission are likely to be minor.

Cattle do not 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 in cattle than for other species. Although cattle are natural hosts of influenza D viruses (a separate genus), the genetic distance between influenza D viruses and IAVs prevents reassortment (Peacock et al. 2024).

Humans are unlikely to be exposed to milk from beef cattle. Current evidence suggests that infected dairy workers acquired infection through exposure to raw milk or through close contact with secretions from clinically affected animals (Morse et al. 2024). Additionally, there appears to be little selective pressure in lactating cattle for the virus to acquire 'mammalian-receptor'" specificity (i.e. α -2,6-linked sialic acid binding), since the bovine mammary gland abundantly expresses α -2,3-linked sialic acids (Peacock et al. 2024; Good et al. 2024).

Response measures, such as movement restrictions and surveillance approaches, may result in significant industry disruption and socio-economic impacts, if implemented. Response arrangements for bovine species in the Australian context are currently being considered and animal health authorities are looking closely to the US response. The response to the US dairy cattle outbreak has involved mandatory pre-movement testing and veterinary certification for lactating dairy cattle, as well as strengthened traceability requirements (APHIS 2024b). These requirements do not apply to non-lactating cattle. Some states have implemented additional restrictions on a state-by-state basis, for example – New York state and Colorado. Depopulation is not being recommended in the US due to the self-limiting nature of the disease in cattle (APHIS 2024a).

The trade impacts of an incursion of clade 2.3.4.4b HPAI into Australian beef cattle are not known and it is difficult to predict how trading partners would respond. Australia relies heavily on access to premium export markets. 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). However, it is possible that trading partners may impose restrictions or additional testing requirements if clade 2.3.4.4b HPAI was detected in Australian beef cattle. Trade impacts are more likely to affected live animal movements compared to meat and animal products. For example – <u>Canada</u> and Mexico implemented enhanced controls for importation of lactating dairy cattle from the US, including enhanced certification prior to export and pre-movement testing (Hunter 29 October 2024). Non-lactating cattle, including beef cattle, were not subject to these enhanced controls. In contrast, Israel requires pre-export testing for all cattle types (Hunter 29 October 2024). Turkey has prohibited importation of all live cattle from the US (Hunter 29 October 2024).

To our knowledge, previous spillovers of IAVs into dairy cattle have not resulted in trade impacts (Saito 1951; Lopez and Woods 1984; Gunning and Pritchard 1997; Gunning et al. 1999; Sreenivasan et al. 2019; Mostafa et al. 2024). The International Dairy Foods Association reports that there are no known disruptions or barriers to US dairy trade and exports, despite the widespread outbreak in US

dairy cattle (International Dairy Foods Association n.d.). We assume that impacts on trade would be more likely if multiple animals are infected and if transmission between cattle is sustained.

There is no indication that clade 2.3.4.4b HPAI poses a risk to beef supply. Testing in the US detected clade 2.3.4.4b HPAI nucleic acid in 1 of 333 cull dairy cow diaphragm muscle samples (APHIS 2025c). Muscle tissues from the same animal corresponding to common retail cuts of meat in that animal were negative. This animal was identified as unwell through the food inspection process and did not enter the food supply chain. No evidence of clade 2.3.4.4b HPAI was detected in samples of retail ground beef in the US (APHIS 2025c). The risk to United Kingdom (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 (Browne et al. 2025; Agriculture and Horticulture Development Board 2024).

Uncertainty: High

Since it has been less than 12 months since the start of the outbreak in dairy cattle, longer-term production impacts following infection and susceptibility to re-infection are not yet clear. The impacts of HPAI infection on beef cattle production metrics (e.g. long-term liveweight gain and fertility) have not been assessed. It is possible that IAV infection may predispose to other secondary infections, which may contribute to impacts in certain populations (e.g. feedlots). For example – IAV infection (although not specifically clade 2.3.4.4b) has been demonstrated experimentally to result in dysfunction of multiple innate immune components (Didierlaurent et al. 2007). Impacts may also be exacerbated by other factors, such as heat stress, poor nutrition, or other infections.

Potential response measures and trade implications of clade 2.3.4.4b HPAI detections in beef cattle are highly uncertain and dependent on the epidemiological context of an outbreak (e.g. number of herds affected, prevalence in other species, prevalence in the importing country).

2.4 Risk estimation

Assuming clade 2.3.4.4b HPAI was present in Australia, what is the risk (likelihood and consequences) to the Australian commercial beef cattle industry?

Risk: Negligible

2.4.1 Rationale

Taken together, spillovers of clade 2.3.4.4b HPAI into cattle do occur but current evidence suggests that these are infrequent relative to exposure (i.e. unlikely but not negligible). It is likely that a lactating infected beef animal may transmit infection to suckling calves. However, sustained transmission between adult cows, or from an infected calf to an adult cow, would appear to be highly unlikely given the lack of exposure of adult cattle to milk and milking machinery (particularly between different premises). If sustained transmission were to occur in beef cattle, industry-level production impacts are likely to be mild, although the long-term effects of infection on beef cattle production metrics are not yet known. Response measures (if implemented) and trade restrictions may result in considerable socio-economic impacts to industry. However, based on the response to the US dairy cattle outbreak, the impacts on trade of dairy cattle and dairy products have been minor.

Uncertainty: High

We considered the uncertainty around the overall risk estimate to also be high, based on the 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 the Australian beef cattle industry. 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 then 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 types). 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 the Australian commercial beef herd was assessed as low, with low to 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 cattle. 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 also widespread 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 beef cattle was assessed as negligible, with low uncertainty. 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). Beef cattle are unlikely to be exposed to milk from other infected cows, the major transmission pathway implicated between cattle.

The consequences of clade 2.3.4.4b HPAI incursion into the Australian commercial beef herd were assessed as minor, with 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 beef cattle are not yet known.

Taken together, the risk of clade 2.3.4.4b HPAI to the Australian commercial beef cattle industry was assessed as negligible, with high uncertainty. The uncertainty in the overall assessment was primarily driven by uncertainty around the impacts of potential response measures and trade implications.

3.1 Limitations

The risk assessment was subject to the following limitations:

- Data are limited to a few experimental studies with relatively small sample sizes or are based on field experience since the start of the US dairy outbreak (less than 12 months). Therefore, the long-term impacts of infection are not yet understood.
- There are no data on breed differences (e.g. dairy vs meat/fibre animals, Boer vs rangeland goats). This is important information in the Australian context.
- The role of non-milk based transmission pathways in cattle is unclear. If respiratory transmission
 or ingestion is identified as relevant to within-herd and between-herd disease spread, the risk to
 non-lactating cattle may be increased.
- Without knowing the definitive spillover host or incursion pathway, it is difficult to make targeted recommendations to mitigate entry risk.
- We did not consider non-commercial beef cattle in this assessment. Biosecurity and awareness
 of HPAI infection in non-poultry species may be lower amongst smallholder producers and pet
 owners. For example there may be higher rates of noncompliance with the ruminant feed ban.
- Response strategies for clade 2.3.4.4b HPAI in cattle 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 beef cattle.
- 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. extensive systems, pasture-based systems, feedlots, lifestyle farms) or geographical
 regions.
- No studies have investigated variability in clade 2.3.4.4b HPAI impacts (e.g. pathogenesis or transmissibility) between cattle breeds or species (e.g. Bos taurus vs Bos indicus). This is important information in the Australian context.
- 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

3.2.1 Reduce the likelihood of entry into beef cattle populations

- 1) Where possible, prevent (or limit) wild bird access to feed, feed storage, water sources, bedding materials and facilities.
- 2) Manage standing water bodies. For example limit wild bird access if possible, minimise cattle access if not required as a water source, improve drainage if not required (AABP 2024).
- 3) In beef calf-rearing operations, avoid feeding raw milk or colostrum sourced from dairy cattle.
- 4) Avoid feeding raw milk to adult beef cattle (as well as other animals) (APHIS 2024d).
- 5) Avoid cattle access to poultry by-products (e.g. poultry litter used as fertiliser).
- 6) Avoid co-mingling cattle and poultry (APHIS 2024d).
- 7) Avoid sharing (unclean) equipment or vehicles with poultry (and dairy) farms.
- 8) Visitors from poultry and dairy farms should be subject to similar visitor entry protocols as those from other beef cattle premises (e.g. stand-down period, site-specific clothing).
- 9) Humans with influenza-like illness or conjunctivitis should not have contact with cattle.
- 10) 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).
- 11) Monitor for and report any unexpected mortality or behaviours in wild birds or domestic or wild animals.

3.2.2 Reduce the likelihood of transmission within the industry

- 12) Minimise unnecessary animal movements and keep detailed movement records.
- 13) Only move healthy animals and isolate new arrivals for at least 21 days upon arrival (AABP 2024). Some resources state 30 days.
- 14) 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 cattle.
- 15) 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).
- 16) Avoid moving (unclean) equipment or vehicles between premises.
- 17) Use dedicated routes for vehicles that do come onto the premises (and avoid direct contact with animals).
- 18) Limit non-essential visitors, maintain a visitor logbook and establish visitor entry protocols (e.g. site-specific clothing).
- 19) 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.

20) In the event of an incursion, sero-surveillance 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

- 21) 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 passive surveillance systems (i.e. for rapid investigation and reporting of outbreaks) (Gates et al. 2021).
- 22) Consider the use of personal protective equipment (such as gloves, apron, respiratory protection and eye protection) in certain circumstances (e.g. close contact with secretions from sick animals like during drenching).
- 23) Do not consume raw milk.
- 24) 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).

- 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
AABP	American Association of Bovine Practitioners
HPAI	high pathogenicity avian influenza
IAV	influenza A virus
RRA	rapid risk assessment
TCID ₅₀	50% tissue culture infectious doses

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