

Risk of high pathogenicity avian influenza to the Australian pig industry: rapid risk assessment

Prepared by Ausvet Pty Ltd for the Department of Agriculture, Fisheries and Forestry

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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 October 2024, H5N1 clade 2.3.4.4b high pathogenicity avian influenza (HPAI) ([Appendix A](#)) was detected in 2 non-commercial pigs in the United States (US) (APHIS 2024f). The pigs were sampled as part of the response to an HPAI outbreak in poultry (specifically, chickens and ducks) on the same farm. Three other pigs on the premises tested negative (APHIS 2024c). The source of the infection was determined to be co-mingling of the pigs with infected poultry and sharing of water sources, housing and equipment (APHIS 2024c). Although the pigs remained healthy, they were humanely culled as part of the investigation.

The genotype detected in these pigs, D1.2, is not the same as the genotypes currently impacting dairy cattle in the US (i.e. genotypes B3.13 and D1.1). Genotype D1.2 HPAI was circulating in both wild birds and poultry in the area at the time of the outbreak (APHIS 2024f), although poultry were implicated as the source of infection.

As of October 2025, Australia remains free of clade 2.3.4.4b HPAI. However, the sustained transmission of clade 2.3.4.4b HPAI in dairy cattle in the US since 2024 has heightened concerns about the potential risk of an epizootic to not only the Australian dairy industry, but also to other livestock industries. For example – detections of clade 2.3.4.4b HPAI in the US have also been confirmed in several other livestock species, including in goats (genotype B3.6) and alpacas (genotype B3.13) (APHIS 2024f, 2024d; AVMA 2024; APHIS 2024e). Importantly, there was no evidence of onwards spread of HPAI from these pig, goat or alpaca infections. A formal qualitative risk assessment has been conducted to assess the risk of HPAI to Australian dairy cattle. Here, we synthesise the latest scientific evidence on HPAI in pigs to inform an evidence-based rapid risk assessment (RRA) for the Australian pig industry.

In regards to public health impacts, pigs raise additional concerns around their potential role as ‘mixing vessels’ for the emergence of novel IAVs (Public Health Agency of Canada 2024). It was beyond the scope of this work to evaluate the public health consequences of the emergence of novel IAVs. A public health risk assessment is recommended to further explore the risk to human health if clade 2.3.4.4b HPAI was to enter and spread within the Australian pig industry.

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) of HPAI to the Australian pig 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 pig 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 pigs in Australia, what is the likelihood of spread within and between commercial piggeries in the next year?
- c) Consequence assessment: What are the consequences of clade 2.3.4.4b HPAI infection in the Australian pig industry (excluding public health consequences)?

Overall assessment

Overall, assuming clade 2.3.4.4b HPAI was present in Australia, the risk to the Australian pig industry was assessed as low, with moderate 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 pig in Australia was assessed as low, with low to moderate uncertainty.

Spillovers of novel influenza A virus (IAV) genotypes into pigs appear to be rare, despite high levels of circulation of HPAI in global wild bird and poultry populations in recent years.

Two natural infections with clade 2.3.4.4b (genotype D1.2) HPAI have been detected in pigs. These were non-commercial animals that shared an environment with an infected poultry flock. Three other pigs on the premises were exposed but did not become infected.

Seroconversion to clade 2.3.4.4b HPAI (indicating past exposure to the virus) was detected in pigs on several occasions between 2016 and 2021. These detections were made in pigs co-housed with infected poultry (1 farm in Italy and 1 in France) and in 3 wild boar from southern Germany. Specifically, exposure to H5N1 and H5N8 subtypes of clade 2.3.4.4b HPAI were detected. However, infectious virus was not recovered in these investigations.

To date, no further reports of clade 2.3.4.4b HPAI infections have been reported in pigs in the US or elsewhere despite widespread disease reported in wild birds, dairy cattle and poultry. However, active surveillance in pigs appears to be limited to research studies and case reports.

Establishment and spread assessment

The likelihood of clade 2.3.4.4b HPAI establishing and spreading within and between piggeries in Australia was assessed as low, with moderate uncertainty.

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 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. In contrast, other mammalian species, including pigs, require further adaptations to transmit effectively.

Experimentally, only clade 2.3.4.4b viruses isolated from mammals have been able to transmit between pigs (i.e. not viruses sampled directly from birds). Therefore, spillover from peri-domestic wildlife (e.g. rats, cats, foxes) may be of more concern than spillover from wild birds. For example – this may occur if pigs scavenge infected carcasses.

Consequence assessment

The consequences of clade 2.3.4.4b HPAI infection in the Australian pig industry were assessed as minor, with moderate uncertainty.

Experimental infection studies suggest that clinical disease following clade 2.3.4.4b infection in pigs is inapparent or mild and short-lived (at least with those genotypes investigated). However, the spillover risk could be underestimated because infections may go unrecognised, particularly as no active surveillance is occurring. For endemic swine IAVs, virus shedding typically ceases by 7 to 10 days post infection (WOAH 2023). There is no robust evidence for a true carrier state for IAVs in any species (MacLachlan et al. 2017).

The potential trade impacts of clade 2.3.4.4b HPAI infection in the Australian pig industry are difficult to predict, adding 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 pigs. Trade impacts may be more likely to affect live animal movements than animal products.

Pigs raise additional concerns around their potential role as ‘mixing vessels’ for the emergence of novel IAVs (Public Health Agency of Canada 2024). For example – the 2009 pandemic H1N1 virus was a reassortant of avian-, human- and classical swine-origin IAV lineages (Smith et al. 2009). It was beyond the scope of this work to evaluate the public health consequences of the emergence of novel IAVs in this rapid risk appraisal. A detailed public health risk assessment is warranted to further explore the risk to human health if clade 2.3.4.4b HPAI was to enter and spread within the Australian pig industry.

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 pig populations:
 - 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 feeding unpasteurised (raw) milk and colostrum, poultry carcasses and poultry by-products (e.g. poultry litter, offal) to pigs (and other animals).
 - d) Avoid co-mingling pigs 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 general 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 for non-avian species, to facilitate testing of suspect cases.
 - d) Surveillance of enzootic IAVs in Australian pig populations may better inform the likelihood of IAV reassortment in swine, which could lead to sustained pig-to-pig transmission.
 - e) An evidence-based active surveillance strategy should be considered 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.
- 3) To reduce impacts:
- a) 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.

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Introduction

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

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).

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). 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, 2025c; CIDRAP 2025; APHIS 2025b; AZDA 2025). Virus sequences from each event are genetically distinct, indicating 2 separate spillovers of genotype D1.1 (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 2025c). 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. Additionally, other clade 2.3.4.4b HPAI genotypes have been detected in livestock species in the US, including in pigs (genotype D1.2), goats (genotype B3.6) and alpacas (genotype B3.13) (APHIS 2024f, 2024d; AVMA 2024; APHIS 2024e). Importantly, there is no evidence for transmission of these genotypes in mammalian species other than cattle.

Detections of clade 2.3.4.4b HPAI were reported in 2 pigs in the US in October 2024 (APHIS 2024f). These were non-commercial pigs that were sampled as part of the response to an HPAI outbreak in poultry (chickens and ducks) on the same farm. Three other pigs on the premises tested negative (APHIS 2024c). The source of the infection was determined to be co-mingling of the pigs with infected poultry and sharing of water sources, housing and equipment (APHIS 2024c). The route of entry of the virus is assumed to be ingestion—a webinar from the Swine Health Information Center

reports that 1 pig on the property was seen eating a dead duck (SHIC 2024). Although the pigs remained healthy, they were humanely culled as part of the investigation.

The genotype detected in these pigs, D1.2, is not the same as the genotypes currently impacting dairy cattle in the United States (US) (i.e. genotypes B3.13 and D1.1). Genotype D1.2 HPAI was circulating in both wild birds and poultry in the area at the time of the outbreak (APHIS 2024f), although poultry were implicated as the source of infection in this case.

Prior to these detections in the US, several studies have detected clade 2.3.4.4b HPAI-specific antibodies in pigs in Europe, indicating past exposure to the virus (Hervé et al. 2021; Schüle et al. 2021; Rosone et al. 2023). In 2016 in France, antibodies to H5N8 clade 2.3.4.4b HPAI were detected in 1 backyard pig following close contact with HPAI-infected domestic poultry (Hervé et al. 2021). No clinical signs were reported. In 2017, 3 of 265 wild boar sera collected in southern Germany had antibodies to H5N8 clade 2.3.4.4b HPAI (Schüle et al. 2021). In Italy in 2021, antibodies to H5N1 clade 2.3.4.4b HPAI were detected in multiple apparently healthy pigs reared on an HPAI-affected free-range poultry farm (Rosone et al. 2023). As viral RNA was not detected, the virus genotypes could not be determined.

In dairy cattle, IAV infection is localised to the mammary tissue and the virus is shed in extremely high concentrations in milk (peak titres of greater than 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). This is because 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). 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).

Scientific studies have stated that respiratory transmission is not likely to be epidemiologically relevant between cattle, 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 (10^1 – 10^3 TCID₅₀ per ml, i.e. 1 million times lower) (Halwe et al. 2024). Although the infectious dose for cattle is not known, 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). These are much higher than the amount detected in nasal swabs. The minimum infectious dose of clade 2.3.4.4b HPAI for pigs is not known. Experimental studies have used doses of 10^5 – 10^9 TCID₅₀ (orally, intranasally and intratracheally) (Arruda et al. 2024; Graaf et al. 2023; Kwon et al. 2024). This could be as little as 0.01–1 ml of milk.

While both α -2,6- and α -2,3-linked sialic acids are highly expressed in the bovine mammary gland (Kristensen et al. 2024), pigs (and humans) express predominantly α -2,6-linked sialic acids in the upper respiratory tract (Nelson and Worobey 2018). Avian-adapted IAVs must evolve specific genetic adaptations to be able to bind these so-called “mammalian” receptors. Additional adaptations, such as the E627K mutation in the polymerase basic 2 (PB2) protein, are also required for efficient replication and transmission in pigs (Arruda et al. 2024). Therefore, while pigs can be infected with

avian-adapted IAVs (Graaf et al. 2023; Arruda et al. 2024; Kwon et al. 2024), the biology of infection is very different to that seen in lactating dairy cows.

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. A formal qualitative risk assessment has been conducted to assess the risk to Australian dairy cattle. Here, we synthesise the latest scientific evidence on HPAI in pigs to inform an evidence-based rapid risk assessment (RRA) for the Australian pig industry.

The Australian pig industry

The Australian commercial pig herd numbers approximately 2.5 million pigs across 4,506 registered pig production sites as of 1 July 2024 (APL n.d.). The pork industry contributed around \$6 billion to the Australian economy in 2023–24, supporting over 34,000 full-time equivalent jobs (APL n.d.). Pork is the second most consumed meat in Australia and all fresh pork is domestically produced, making the domestic pork industry a vital part of Australia's food supply chain (APL 2024:18). Key production areas include Queensland, Victoria, New South Wales and South Australia (APL 2024:18), and there is also significant production in south-west Western Australia.

Pork production operations in Australia can broadly be classified as: 1) large commercial enterprises that are often vertically integrated; 2) private commercial enterprises, often associated with other commercial primary production; and (3) hobby or opportunistic producers (Cutler and Holyoake 2008). Commercial operations can broadly be divided into indoor system farming, free-range farming, and outdoor system farming (APL n.d.). Around 90% of commercial pigs in Australia are housed indoors, with strict biosecurity and limited contact with the external environment. Approximately 5% of Australian sows are free-range, where they are entirely housed outdoors with kennels or huts for shelter. The remainder (~4%) are 'outdoor bred, raised indoors on straw'. In this system, breeding pigs live in open spaces with free access to paddocks during their adult life but have bedded indoor shelter. Piglets are moved to large barns with deep-litter bedding (frequently straw or rice hulls) after weaning, remaining in these barns until sale.

The commercial pig industry maintains high biosecurity standards, which help to protect pigs from unwanted disease and mitigate disruptions to pork supply (APL n.d.). The National Farm Biosecurity Manual for Pork Production outlines the minimum biosecurity requirements that producers should implement on farm. In contrast to commercial operations, studies have shown that small-scale and backyard pig producers tend to engage in higher risk animal management practices, such as trading at saleyards and undertaking minimal on-farm biosecurity (Schembri et al. 2015). However, there is considerable variation in biosecurity practices between operations. Of particular note, a study of 104 Australian pig producers identified that most backyard producers also kept domestic birds on the premises and 10 to 20% of producers (regardless of whether backyard, small-scale or large-scale) fed dairy products to pigs (Schembri et al. 2015). It was not stated whether pigs specifically co-mingled with domestic poultry, or whether domestic birds were kept separately. Moreover, 15.5% of backyard and small-scale producers were located within a 5 km radius of larger commercial pig farms.

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) of HPAI to the Australian pig industry, excluding public health consequences?
 - 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 pig 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 pigs in Australia, what is the likelihood of spread within and between commercial piggeries in the next year?
 - c) Consequence assessment: What are the consequences of clade 2.3.4.4b HPAI infection in the Australian pig industry?

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.

This 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, individual entry and exposure pathways were not evaluated.

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 pig or pigs.
- Establishment and spread: Onward transmission of clade 2.3.4.4b HPAI virus in pigs, leading to sustained spread within and 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	<i>High</i>	Low	Moderate	Moderate	High
	<i>Moderate</i>	Low	Low	Moderate	Moderate
	<i>Low</i>	Negligible	Low	Low	Moderate
	<i>Negligible</i>	Negligible	Negligible	Low	Low
		<i>Negligible</i>	<i>Low</i>	<i>Moderate</i>	<i>High</i>
		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. An evaluation of public health consequences is out of scope for this assessment.

Table 2 Qualitative consequence categories used in this assessment

Qualitative category	Definition	Examples
Negligible	Insignificant negative consequences on industry productivity or animal health.	Minor production losses, low number of localised infections. No threat to food security or the economy.
Minor	Marginal negative consequences on industry productivity or animal health.	Production losses restricted to a small area (regional level or below).
Moderate	Significant negative consequences on industry productivity or animal health.	Significant production losses across several regions.
Severe	Substantial negative consequences on industry productivity or animal 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	<i>High</i>	Negligible risk	Low risk	Moderate risk	High risk
	<i>Moderate</i>	Negligible risk	Low risk	Moderate risk	High risk
	<i>Low</i>	Negligible risk	Low risk	Low risk	Moderate risk
	<i>Negligible</i>	Negligible risk	Negligible risk	Negligible risk	Negligible risk
		<i>Negligible</i>	<i>Minor</i>	<i>Moderate</i>	<i>Severe</i>
		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 pigs could make contact with an infected wild bird (or other infected species) and the probability of this is equal across all of Australia.
- 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 explore these variables.
- Human health impacts were not considered in the consequence assessment.

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 pig in Australia in the next year?

Likelihood: Low

2.1.1 Rationale

IAVs are known to infect pigs, so the likelihood is not negligible. [H1N1, H1N2 and H3N2 IAVs](#) have been detected sporadically in pigs in Australia. For clade 2.3.4.4b HPAI specifically, both natural and experimental infections have been documented in pigs overseas. However, despite the high level of circulation of clade 2.3.4.4b HPAI in wild birds and poultry in Europe and North America since 2020, reports of natural infections are rare.

Two natural infections in pigs have been identified in the US (APHIS 2024c). In that report, only 2 of 5 pigs on the premises became infected, even when exposed to high levels of environmental contamination due to a concurrent poultry outbreak (APHIS 2024c). In 2016 in France, antibodies to H5N8 clade 2.3.4.4b HPAI (suggesting previous exposure to the virus) were detected in 1 backyard pig from 10 herds tested following close contact with HPAI-infected domestic poultry (Hervé et al. 2021). In 2017, 3 (out of 265) wild boar sera collected in southern Germany had antibodies to H5N8 clade 2.3.4.4b HPAI (Schüle et al. 2021). In Italy in 2021, antibodies to H5N1 clade 2.3.4.4b HPAI were detected in blood collected from pigs reared on an affected free-range poultry farm (Rosone et al. 2023). Notably, viral RNA was not detected in nasal swabs collected from the same herd.

The likelihood of spillover will vary by production type. Novel IAVs can be introduced into pig populations directly from wild birds or poultry (e.g. the Eurasian H1N1 lineage and genotype D1.2 HPAI), through reverse zoonosis from humans (e.g. pandemic H1N1 viruses) or through reassortment between human and swine viruses (for example – H3N2 IAVs) (Long et al. 2019; MacLachlan et al. 2017; Deng et al. 2012; Wong et al. 2018; Ma et al. 2008; APHIS 2024f). The incursion likelihood is thought to be very low in commercial indoor piggeries with high biosecurity where contact with other species (apart from humans) is limited (Arruda et al. 2024). In contrast, the likelihood may be higher for operations where pigs have outdoor access (e.g. free-range and ‘outdoor bred, raised indoors on straw’ commercial operations and most small-scale and backyard herds) (APL n.d.; Schembri et al. 2015). For example – an Australian study found that backyard producers were significantly more likely than large-scale producers to keep domestic poultry (Schembri et al. 2015). A serological study from Nigeria revealed a H5 seropositivity rate in abattoir samples from backyard and free-range pig holdings of 14% (42 out of 300 samples) during a period when clade 2.3.2.1c HPAI was circulating in poultry in the region (Meseko et al. 2018).

The feeding of waste milk from infected dairy cattle is a theoretical spillover pathway (AABP 2024). However to-date, clade 2.3.4.4b HPAI has not been detected in dairy cattle outside the US. Similarly, ingestion of infected poultry carcasses or offal has been implicated in mammalian IAV infections in carnivores, scavengers and humans (Thanawongnuwech et al. 2005; CDC 2024; Plaza et al. 2024; Graziosi et al. 2024). The Northern Territory and Queensland specifically prohibit feeding of material

of bird origin to pigs (Australian Pork Limited 2022). However in other jurisdictions, material of avian origin and milk, milk products or milk by-products of Australian origin are not considered prohibited pig feed (AHA 2019). While these are unlikely to be found in commercial feed, small-scale and backyard producers are known to feed dairy products and unprocessed meat products to pigs (Schembri et al. 2015). There is a lack of data around what proportion of pig producers feed milk products and material of bird origin in Australia, and how this may vary by operation type.

Experimentally, pathology following clade 2.3.4.4b infection is primarily restricted to the lower respiratory tract, with pigs either remaining healthy or showing a short duration of lethargy and fever (1–2 days) (Graaf et al. 2023; Arruda et al. 2024; Kwon et al. 2024). It is possible that such infections would go unnoticed, particularly in individual animals.

Uncertainty: Low to moderate

Because IAV infections may go undetected in pigs, the true frequency of spillover is unknown. Active surveillance for HPAI is not being conducted in pig populations. Additional serological surveillance of pig populations is needed to better understand the true spillover rate.

2.2 Establishment and spread assessment

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

Likelihood: Low

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. In contrast, for other mammalian species, including pigs, further adaptation is required for efficient transmission.

Experimentally, infection with avian-adapted clade 2.3.4.4b viruses led to limited viral replication in the upper respiratory tissues (in only 1 of 8 pigs) and no infectious virus could be recovered, suggesting negligible likelihood of onwards transmission (Graaf et al. 2023). In another pig study using mink-adapted clade 2.3.4.4b, infectious virus was isolated at low levels from nasal, oropharyngeal and tissue samples between 1 and 5 days post-challenge (Kwon et al. 2024). However, virus shedding was not high and/or frequent enough for transmission to in-contact pigs. A further study using both avian- and mammalian-derived clade 2.3.4.4b viruses showed that mammalian-derived viruses (genotypes B2.1 and B3.2), but not avian-derived viruses (genotypes B1.1 and B2.1), were detected in the nasal cavity of inoculated pigs and transmitted to some in-contact pigs. Notably, both mammalian-derived viruses (but not the avian-derived B2.1 virus) contained the well-known PB2 E627K adaptation. There is a theoretical potential for mammalian-adapted IAVs to spill over from peri-domestic wildlife (including rodents), particularly in small-scale rearing systems, although this has not been observed or demonstrated. For example – pigs may scavenge accessible carcasses. These mammalian IAVs may be more likely to transmit between pigs.

The levels of virus detected in the naturally infected pigs in the US were very low (APHIS 2024f). While the infectious dose of clade 2.3.4.4b HPAI for pigs is not known, experimental infection studies have used relatively high doses for infection 10^5 – 10^9 TCID₅₀ (Arruda et al. 2024; Graaf et al. 2023; Kwon et al. 2024). Note that milk can contain very high levels of virus, so this ‘dose’ could be present in as little as 1 µl to 1 ml of raw milk from infected cows (Caserta et al. 2024; Halwe et al. 2024).

Although multiple pigs on a single premises were sero-positive for clade 2.3.4.4b HPAI in Italy, the authors do not suggest that there was pig-to-pig transmission (Rosone et al. 2023). Rather, they speculate that pigs were most likely infected through prolonged exposure to high viral loads through fomites, soil and feed contaminated by the infected poultry.

In the event of spillover into pigs, reassortment with enzootic swine-adapted IAVs leading to enhanced transmissibility amongst pigs, and potentially other mammals, is possible (Arruda et al. 2024). Australian pigs were reportedly free of IAV prior to 2009, with the H1N1 2009 pandemic strain introduced as a spillover from humans (Deng et al. 2012). Subsequently, H1N1, H1N2 and H3N2 IAVs have been [detected sporadically](#) in pigs in Australia (Wong et al. 2018). Currently, no routine active surveillance is undertaken for IAVs in Australian pig populations (DAFF 2024).

The risk of spread will vary by production system, due to different biosecurity practices, movement patterns, and other factors. Non-commercial pigs (which may have a higher likelihood of spillover) are arguably less likely to have contact with other pigs and contribute to spread within industry. However, it has been estimated that around 5% of pigs in Australia are sold live at public saleyards (Schembri et al. 2015), which may be an avenue for enhanced disease spread. A more comprehensive formal risk assessment would be required to analyse detailed movement patterns within the Australian pig industry and how these vary by production system. Feral pigs (and other peri-domestic wildlife species) may also play a role in disease spread if mammalian transmission was to become established (Gentle et al. 2022).

Uncertainty: Moderate

It is not known whether clade 2.3.4.4b HPAI can spillover to pigs directly from other mammals, although given the number of cases in scavenger species it is assumed to be possible if an infected carcass was consumed. The minimum infectious dose of clade 2.3.4.4b HPAI for pigs is not yet known.

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.

2.3 Consequence assessment

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

Consequences: Minor

2.3.1 Rationale

The animal health consequences are likely to be minor. In the 2 documented cases of natural infection of pigs with clade 2.3.4.4b HPAI in the US, the pigs remained healthy until they were humanely culled as part of response operations (APHIS 2024c). In domestic pigs with serological evidence of previous exposure to clade 2.3.4.4b HPAI, no clinical signs were reported (Hervé et al. 2021; Rosone et al. 2023). Experimentally, in pigs infected with clade 2.3.4.4b HPAI there is evidence of pathology in the lower respiratory tract and pigs sometimes showing a short duration of lethargy and fever (1–2 days) (Graaf et al. 2023; Arruda et al. 2024; Kwon et al. 2024).

Infection with IAVs in swine is a Category 4 emergency animal disease (i.e. mainly production loss diseases) in Australia (AHA 2018) and the response to an outbreak will be determined by a risk assessment. No action will be required unless the risk assessment indicates an unacceptable threat to animal or public health (AHA 2018). Response measures following the US swine detections were negligible, although the 5 pigs present on the property were culled as part of the investigation.

The trade impacts of an incursion of clade 2.3.4.4b HPAI into Australian pigs are not known and it is difficult to predict how trading partners would respond. To our knowledge, there were no trade restrictions implemented following the clade 2.3.4.4b HPAI detections in US swine. Likewise, no industry-level socio-economic impacts resulted from the serological detections in Italy, France or Germany. Based on observations of the response to detections in US dairy cattle, trade impacts may be more likely to affect live animal movements compared to meat and animal products. Australia has not exported live swine in the last 10 years.

Uncertainty: Moderate

The animal health and welfare impacts of infection are based on a limited number of experimental studies with relatively small sample sizes and limited cases of natural infection with clade 2.3.4.4b HPAI. The long-term impacts of infection on growth, fertility and other production metrics in pigs are not known.

Potential response measures and any trade implications of clade 2.3.4.4b HPAI detections in Australian pigs would be 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) of HPAI to the Australian pig industry, excluding public health consequences?

Risk: Low

In summary, spillover of clade 2.3.4.4b into pigs is possible (via various pathways), and may be more likely in production systems where pigs have outdoor access. Current evidence suggests that

sustained transmission amongst pigs is unlikely, at least with avian-adapted clade 2.3.4.4b viruses. However, reassortment with enzootic IAVs present in Australian pig populations could lead to rapid acquisition of pig-to-pig transmissibility. This assessment was conducted assuming that clade 2.3.4.4b HPAI was present in wild birds in Australia (i.e. presence of avian-adapted viruses)—if pigs are infected with a mammalian-adapted virus the risk is likely to be higher. Evidence to date suggests that production impacts to the industry are likely to be minimal with avian-adapted viruses.

Uncertainty: Moderate

We considered the uncertainty around the overall risk estimate to be moderate as data on clade 2.3.4.4b HPAI infection in pigs are limited.

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 pig industry, excluding public health consequences. 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. A formal evaluation of the public health consequences was out of scope for this rapid risk assessment, however public health considerations are discussed in the Consequence Assessment and Limitations.

Overall, the likelihood of entry of clade 2.3.4.4b HPAI (defined as the initial spillover, without onward transmission) into Australian pigs was assessed as low, with low to moderate uncertainty. Sporadic spillovers may occur (with both clade 2.3.4.4b and other IAVs) and current evidence suggests that sporadic infections in pigs are likely to go undetected. There is a lack of sero-surveillance data assessing prior exposure to clade 2.3.4.4b viruses in pigs. The entry risk depends 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 pigs. 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 the Australian pig herd was assessed as low, with moderate 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). Reassortment of clade 2.3.4.4b HPAI with enzootic IAVs circulating in Australian pig populations may lead to acquisition of gene segments pre-adapted for pig-to-pig aerosol transmission.

The consequences of clade 2.3.4.4b HPAI incursion into the Australian pig industry were assessed as low, with moderate uncertainty. Animal health impacts are likely to be minor based on currently available information.

Taken together, the risk of clade 2.3.4.4b HPAI to the Australian pig industry was assessed as low, with moderate uncertainty.

3.1 Limitations

The risk assessment was subject to the following limitations:

- Data on clade 2.3.4.4b HPAI in pigs are limited to a few experimental studies with relatively small sample sizes or are based on 2 natural infections documented in the US and limited serological studies in Europe. Therefore, the full scale of infection and long-term impacts are not yet understood.
- There is a lack of data around what proportion of pig producers feed milk products and material of bird origin in Australia, and how this may vary by operation type.
- Without knowing the definitive spillover host or incursion pathway, it is difficult to make targeted recommendations to mitigate entry risk.
- Surveillance for IAVs in Australian pig populations is limited so it is challenging to objectively assess the risk due to reassortment of swine viruses.
- Existing response strategies for IAV in pigs are not necessarily applicable for clade 2.3.4.4b HPAI 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 pigs.
- This rapid risk appraisal does not evaluate the potential public health consequences of infection establishing in the Australian pig industry. If a pig was co-infected with both clade 2.3.4.4b HPAI and an enzootic swine-adapted IAV, it is possible that reassortment could generate novel HPAI genotypes with enhanced transmissibility between pigs and potentially other mammal species including humans. For example – the 2009 pandemic H1N1 virus was a reassortant of avian-, human- and classical swine-origin IAV lineages (Smith et al. 2009). A detailed public health risk assessment is warranted to further explore the risk to human health if clade 2.3.4.4b HPAI was to enter and spread in Australia.
- 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 pigs. Therefore, many of the recommendations focus on general enhancements to biosecurity and have been extrapolated from recommendations to dairy producers and to small ruminant (sheep and goat) and camelid stakeholders (APHIS 2024a; AABP 2024; APHIS 2024d).

3.2.1 Reduce the likelihood of entry into pig populations

- 1) Avoid co-mingling pigs and poultry (APHIS 2024b).
- 2) Avoid sharing (unclean) equipment or vehicles with poultry (and dairy) farms.
- 3) Avoid feeding raw milk and colostrum to pigs (and other animals) (APHIS 2024b).
- 4) Avoid feeding poultry carcasses and by-products (e.g. poultry litter, offal) to pigs (and other animals).
- 5) Minimise access to other bird and animal carcasses (in free-range pigs or pigs housed outdoors).
- 6) Visitors from poultry and dairy farms should be subject to similar visitor entry protocols as those from other piggery premises (e.g. stand-down period, site-specific clothing).
- 7) Humans with influenza-like illness or conjunctivitis should not have contact with pigs.
- 8) 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, avoid co-mingling livestock species).
- 9) Monitor for and report any unexpected mortality or behaviours in wild birds or domestic or wild animals.
- 10) Where possible, prevent (or limit) wild bird access to feed, feed storage, water sources, bedding materials and facilities.
- 11) Manage standing water bodies. For example – limit wild bird access if possible, minimise pig access if not required as a water source, improve drainage if not required (AABP 2024).

3.2.2 Reduce the likelihood of transmission within the industry

- 12) Surveillance for enzootic IAVs in Australian pig populations may better inform assessment of the risk due to reassortment in swine, which could lead to sustained pig-to-pig transmission.
- 13) Minimise unnecessary animal movements and keep detailed movement records. This may be particularly relevant to certain sub-populations (e.g. stud and show pigs).
- 14) Only move healthy animals and isolate new arrivals for at least 21 days upon arrival (based on recommendations in dairy cattle) (AABP 2024). Some resources state 30 days.
- 15) Monitor animals for signs of illness and isolate sick animals. Consider HPAI as a differential diagnosis for non-specific illness in pigs.
- 16) 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 general surveillance (Sergeant et al. 2022).

- 17) Avoid moving (unclean) equipment or vehicles between premises.
- 18) Use dedicated routes for vehicles that do come onto the premises (and avoid direct contact with animals).
- 19) Limit non-essential visitors, maintain a visitor logbook and establish visitor entry protocols (e.g. site-specific clothing).
- 20) 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.
- 21) 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 surveillance strategy and cost-benefit analysis.

3.2.3 Reduce impacts

- 22) 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 detection, investigation and reporting of outbreaks) (Gates et al. 2021).

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, HPAI and LPAI respectively), 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
PB2	polymerase basic 2
RRA	rapid risk assessment
TCID ₅₀	50% tissue culture infectious doses

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