CSIRO PUBLISHING

SPECIAL ISSUE | REVIEW
https://doi.org/10.1071/WR22118

WILDLIFE RESEARCH

A review of viral and parasitic infections in wild deer in Australia with relevance to livestock and human health

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Handling Editor:
Graham Nugent

ABSTRACT

Wild animals harbour a diverse range of pathogens. In Europe and North America, cervids (Family Cervidae) can act as reservoirs for viral, prion, bacterial, and parasitic infections. Wild deer often inhabit agricultural land, therefore representing a biosecurity risk due to their potential ability to transmit diseases to livestock. Multiple studies have investigated the infection status of wild deer in Australia, mostly during the 1970s and 1980s, and deer populations have increased greatly in abundance and distribution since then. Those studies provide an important baseline for the pathogens carried by wild deer in Australia but are limited by small sample size, the small number of deer species studied, and the disease detection methods used. Recent investigations using ELISA (Enzyme-Linked Immunosorbent Assay), PCR-based assays, and next-generation sequencing have substantially increased our understanding of viral and parasitic infections in Australian deer. These studies indicate that deer may act as reservoirs for pathogens such as *Pestivirus*, *Neospora caninum* and *Entamoeba bovis*. The use of next-generation sequencing has led to the discovery of novel viruses such as Picobirnavirus and a novel species of the genus *Bopivirus*, both of which pose transmission risks for domestic animals. Recent research confirms that wild deer could be a future source of viral and parasitic infections for domestic livestock and other wildlife species.

Keywords: chital deer, fallow deer, genetics, infectious disease, invasive species, pest control, rusa deer, sambar deer, wildlife diseases.

Introduction

Multiple infectious diseases, including avian influenza virus, African swine fever virus and coronaviruses have emerged or re-emerged in recent decades, raising questions about their pathogenesis and epidemiology (Rhyan and Spraker 2010; Barroso *et al.* 2021; McClymont *et al.* 2022). Wildlife populations are the most significant source of emergent infectious diseases that impact human health, biodiversity, and agriculture (Morner *et al.* 2002). As a result, pathogen transmission between wildlife and livestock is a global concern (Rhyan and Spraker 2010). Of particular importance in wildlife health are pathogens that do not exhibit host specificity or can infect host species across different taxa. In this context, it is estimated that 77% of pathogens detected in livestock can infect multiple wild and domestic species (Woolhouse *et al.* 2001), and can therefore be acquired by other species. Wild animals typically carry more pathogens than are present in domestic animals (Walker *et al.* 2017), and some of these pathogens do not need to persist for an extended period within a wildlife population for transmission to livestock to occur (Morgan *et al.* 2006).

Due to altered landscapes or the introduction of non-native species, changes in wildlife density and habitat use lead to new interfaces between livestock and wildlife, potentially exacerbating processes that promote pathogen transmission (Rhyan and Spraker 2010; Miller *et al.* 2013). Because cervids (Family Cervidae) are ruminants and closely related to economically important livestock species such as cattle, sheep, and goats, it is not surprising that they share many pathogens, including several of major agricultural significance. Here, we focus on the role of wild deer as wildlife hosts of viral and parasitic

Received: 30 June 2022
Accepted: 28 April 2023
Published: 11 July 2023

Cite this:
Huaman JL *et al.* (2023)
Wildlife Research, **50**(8–9), 593–602.
doi:10.1071/WR22118

pathogens in Australia. The role of wild deer as a source of infection for livestock has been widely studied in Europe and North America, where wildlife–livestock interactions have increased (Böhm *et al.* 2007; Conner *et al.* 2008; Martin *et al.* 2011; Ruiz-Fons *et al.* 2014).

Deer are not native to Australia and were first introduced in the early 19th century from Europe and Asia (Bentley 1998; Hall and Gill 2005). Six species – chital (*Axis axis*), fallow (*Dama dama*), hog (*Axis porcinus*), red (*Cervus elaphus*), rusa (*Cervus timorensis*), and sambar (*Cervus unicolor*) deer – have established self-sustaining wild herds in Australian habitats (Bentley 1998; Moriarty 2004*a*). Wild deer are present in all Australian states and territories, but are most widespread and abundant in Tasmania, Victoria, New South Wales, and the Australian Capital Territory (Forsyth *et al.* 2015; Davis *et al.* 2016; Forsyth *et al.* 2016; Crittle and Millynn 2020). For approximate current and potential distributions of the six species of wild deer in Australia, see fig. 2 in Davis *et al.* (2016). Wild deer populations continue to increase in size and distribution in mainland Australia and Tasmania (Moriarty 2004*a*; Davis *et al.* 2016; Cunningham *et al.* 2022). Wild deer are particularly widespread and abundant in south-eastern Australia, where population densities of up to 39 per km^2 occur in agricultural landscapes (Bengsen *et al.* 2022). Bioclimatic modelling, which matches animal species' requirements to suitable environments, suggests that deer currently occupy a small proportion of their potential distribution in Australia (Moriarty 2004*a*; Davis *et al.* 2016).

Australia is currently free of many animal pathogens detected elsewhere in the world, such as those causing Foot and Mouth Disease or Lumpy Skin Disease, and exotic diseases remain a major threat to Australia's livestock industry as well as to human and wildlife health. Therefore, ongoing monitoring to determine the presence of pathogens in wildlife is crucial to identifying potential reservoirs of infectious diseases and preventing future disease outbreaks. Wild deer populations currently pose a disease risk (Fig. 1), and further increases in size and distribution could increase that risk. However, data about the infection status of Australian wild deer populations are sparse, with only six studies reported before 2014, all of them identified in localised surveys conducted 41–56 years ago (Munday 1966; Munday 1972; Presidente and Westbury 1979; Slee and Presidente 1981; English 1982; McKenzie *et al.* 1985). More recently, however, at least 11 further surveys have been reported, many of them our own work (Davies 2014; Koehler *et al.* 2016; Panozzo 2018; Huaman *et al.* 2020, 2021*a*, 2021*b*, 2021*c*, 2022*a*, 2022*b*; Jenkins *et al.* 2020; Lamb *et al.* 2021).

Here, we provide an updated assessment of the prevalence of both viral and parasitic infections in wild deer in Australia. We conducted this assessment by searching the PubMed database using the keywords 'Deer', 'Australia' and 'Disease'; 'Deer', 'Australia' and 'Pathogen', or 'Deer', 'Australia', and 'Parasite' (in the title and abstract) and inspecting the references in the publications identified in this literature search.

Fig. 1. A farm paddock in Victoria, Australia. Note the presence of deer scats in the foreground and domestic animals (horses) in the background.

Our search was conducted on 02 December 2022. We also highlight how advanced genomic techniques have increased our understanding of wild deer diseases. In this present review we aimed to: (1) review current knowledge of deer infection in Australia; (2) examine how this compares to deer overseas; and (3) assess the implications for wild and domestic animal populations and humans.

Viral diseases in deer

Viral infections of deer have been examined across multiple decades in Australia. All six deer species have been assessed for various viral infections; however, most data are available for fallow deer. The first study of diseases in Australia's wild deer was published in 1966 (Munday 1966) and assessed fallow deer in Tasmania for the presence of Bovine Herpesvirus (BoHV-1) and *Pestivirus*. BoHV-1 causes two clinical diseases in cattle: infectious bovine rhinotracheitis and infectious pustular vulvovaginitis. The prevalence of antibodies to BoHV-1 in adult cattle in Australia is typically 25–40%, with the virus occurring across the continent but being particularly associated with feedlots (Gu and Kirkland 2008). No BoHV-1 was detected in the survey of Munday (1966), or in other surveys of small areas and a more recent study across large areas of eastern Australia (Table 1). These findings suggest that Australian deer are not carriers of BoHV-1, despite the moderate prevalence levels in livestock and the extensive sharing of pastoral habitats by cattle and wild deer across Australia (Gu and Kirkland 2008). Multiple deer species have been experimentally infected with BoHV-1 (Chow and Davis 1964; Mollema *et al.* 2005). Bovine Herpesvirus has also been detected serologically in deer in Poland (<5% prevalence) and Hungary (12–47% prevalence via PCR detection and subsequent sequencing) (Kalman and Egyed 2005; Fabisiak *et al.* 2018). All studies involving Australian deer were

Table 1. Viral infections investigated in wild deer in Australia.

Pathogen	Sample size	Prevalence (%)	Deer species	Diagnostic method	State or territory	Reference
Akabane virus	396	13.0	Red	VNT	Qld	McKenzie *et al.* (1985)
	ND	ND	Rusa	ND (antibodies)	NSW	Moriarty (2004*b*)
	10	0.0	Fallow	PCR	ACT	Huaman *et al.* (2020)
	60	0.0	Fallow		NSW	
	7	0.0	Rusa			
	43	0.0	Chital		Qld	
	2	0.0	Fallow		Vic.	
	22	0.0	Sambar			
Bovine Ephemeral Fever Virus	432	43.0	Red	VNT	Qld	McKenzie *et al.* (1985)
	ND	ND	Rusa	ND (antibodies)	NSW	Moriarty (2004*b*)
	10	0.0	Fallow	PCR	ACT	Huaman *et al.* (2020)
	60	0.0	Fallow		NSW	
	7	0.0	Rusa			
	43	0.0	Chital		Qld	
	2	0.0	Fallow		Vic.	
	22	0.0	Sambar			
Bopivirus	59	5.0	Fallow	PCR	NSW	Huaman *et al.* (2021*b*)
	3	0.0	Sambar			
	6	1.0	Red			
	12	1.0	Fallow		Vic.	
	11	0.0	Sambar			
	0	0.0	Red			
Bovine herpesvirus	31	0.0	Fallow	VNT	Tas.	Munday (1966, 1972)
	ND	0.0	Fallow, rusa	ND (antibodies)	Vic.	Presidente and Westbury (1979)
	86	0.0	Fallow	VNT	NSW	English (1982)
	405	0.0	Red	VNT	Qld	McKenzie *et al.* (1985)
	34	0.0	Fallow	ELISA	ACT	Huaman *et al.* (2020)
	164	0.0	Fallow		NSW	
	80	0.0	Rusa			
	110	0.0	Chital		Qld	
	2	0.0	Fallow		Vic.	
	42	0.0	Sambar			
Bluetongue	396	13.0	Red	VNT	Qld	McKenzie *et al.* (1985)
Epizoootic Hemorrhagic Disease Virus	432	19.0–50.0	Red	VNT	Qld	McKenzie *et al.* (1985)
	10	0.0	Fallow	PCR	ACT	Huaman *et al.* (2020)
	60	0.0	Fallow		NSW	
	7	0.0	Rusa			
	43	0.0	Chital		Qld	
	2	0.0	Fallow		Vic.	
	22	0.0	Sambar			
Palyam group	432	86.0	Red	VNT	Qld	McKenzie *et al.* (1985)
Parainfluenza-3	10	0.0	Fallow	VNT	Tas.	Munday (1972)
Pestivirus	76	14.5	Fallow	VNT	Tas.	Munday (1966, 1972)

(*Continued on next page*)

Table 1. (*Continued*).

Pathogen	Sample size	Prevalence (%)	Deer species	Diagnostic method	State or territory	Reference
	ND	1.0	Sambar	VNT	Vic.	Slee and Presidente (1981)
	86	1.2	Fallow	VNT	NSW	English (1982)
	405	4.0	Red	VNT	Qld	McKenzie *et al.* (1985)
	34	7.7	Fallow	ELISA	ACT	Huaman *et al.* (2020)
	164	6.1	Fallow		NSW	
	80	2.5	Rusa			
	110	0.0	Chital		Qld	
	2	0.0	Fallow		Vic.	
	42	0.0	Sambar			
Picobirnavirus	60	53.3	Fallow	PCR	NSW	Huaman *et al.* (2021*c*)
	11	36.4	Sambar		Vic.	
Other arboviruses	ND	ND	Fallow, rusa, sambar	VNT	Vic., NSW	English (1982), Presidente and Westbury (1979), Slee and Presidente (1981)

ND, no data; Qld, Queensland; NSW, New South Wales; ACT, Australian Capital Territory; Vic., Victoria; Tas., Tasmania; VNT, Virus neutralisation test; NGS, next-generation sequencing; ELISA, Enzyme-Linked Immunosorbent Assay.

conducted using serological tests, and it may be that this detection method is less sensitive than PCR, but further work is needed to confirm this.

Pestivirus infection is widespread in Australian cattle, causing the clinical disease Bovine Viral Diarrhoea (Reichel 2000; Scharnbock *et al.* 2018). Studies of *Pestivirus* exposure in deer species in Australia have been conducted across small geographical areas, with more recent studies encompassing larger land areas (Table 1). All these studies have found variable but low prevalence. Seropositive fallow deer were first reported in a small study in Tasmania with a prevalence of 14.5% (11/76 deer) (Munday 1966), and a decade later in another small study in NSW with a prevalence of 1.2% (1/86) (English 1982).

A larger study assessing *Pestivirus* prevalence in red deer sourced from 20 localities in eastern Australia in 1985 reported 4% prevalence (McKenzie *et al.* 1985). Recently, a study sourcing five deer species from locations in eastern Australia reported a similar prevalence of 3% (13/432) for *Pestivirus* antibodies (Huaman *et al.* 2020). These findings are consistent with the low seroprevalence of deer species globally for *Pestivirus* (reviewed in Passler *et al.* 2016). In addition, Australian cattle exhibit high seropositive rates (53%) (Scharnbock *et al.* 2018), but deer experimentally infected with *Pestivirus* have a low ability to shed the virus or display clinical symptoms (reviewed in (Passler *et al.* 2016)). Therefore, deer seem to be a spill-over end-host for *Pestivirus* and seldom able to transmit the virus to cattle.

Many significant viral infections of Australian livestock are carried by mosquitoes, termed arboviruses. Because arboviruses are vector-borne viruses, their occurrence is mainly driven by the effects of temperature and rainfall. There are distinct seasonal geographical changes in the incidence of important livestock-associated arboviruses (Geoghegan *et al.* 2014). Four agriculturally significant arboviruses have been detected in Australian deer: Bluetongue virus; Akabane virus; BEFV (Bovine Ephemeral Fever Virus); and EHDV (Epizoootic Hemorrhagic Disease Virus) (Table 1). Most of these observations have come from one large study of wild deer conducted across Queensland in 1985, in which 43% of deer were seropositive for BEFV, 19–50% seropositive for one of five strains of EHDV, and 13% seropositive for Akabane virus (McKenzie *et al.* 1985). Similar results were reported in a small study conducted in NSW in 2004, with rusa deer seropositive for Akabane virus and EHDV. More recently, a large-scale PCR-based screening study of deer blood samples collected from eastern Australia revealed no infection for a range of vector-borne diseases including BEFV, EHDV and Akabane (Huaman *et al.* 2020, 2021*a*). However, considering (1) endemicity of these pathogens in eastern Australia, (2) the presence of suitable vectors, and (3) expected increases in the distribution and abundance of deer (in the absence of substantial control efforts) (Davis *et al.* 2016; Cunningham *et al.* 2022), the possibility that deer species could be a future source of infection of these pathogens cannot be ruled out.

Using next-generation sequencing (NGS), three recent studies identified and characterised novel viruses in deer serum, plasma, and faecal samples (Huaman *et al.* 2021*b*, 2021*c*, 2022*a*). These studies illustrate how novel molecular techniques can change our understanding of viral epidemiology and evolution. Picobirnaviruses (PBVs) were detected in serum and plasma from fallow, rusa, and sambar deer (Huaman *et al.* 2021*c*), with subsequent molecular screening of a range of specimens collected from wild deer and of faecal samples

from farmed cattle. PBVs have been detected in several animal species worldwide, mostly in faecal samples but also in blood and respiratory tract samples (Smits *et al.* 2011; Malik *et al.* 2014). High prevalence of PBVs were recorded in deer and cattle sampled in south-eastern Australia (Huaman *et al.* 2021*c*), with a predominance of genogroup I and simultaneous genogroups I and II detection in these host species. Moreover, the detection of identical sequences in the trachea and nasal swabs of the same animal, with no amplification in the lung, suggests active replication and infection of PBVs in the upper respiratory tract, expanding our knowledge of picobirnavirus tropism.

Genomic and phylogenetic analyses of NGS data revealed the presence of a new member of the genus *Bopivirus*, proposed as '*Bopivirus* C' (Huaman *et al.* 2021*b*), in samples from south-eastern Australia. Further epidemiological investigation showed a prevalence of 8.5% in fallow deer, 16.7% in red deer and 0% in sambar deer and cattle. Phylogenetic and sequence analyses of the positive samples indicated that the same genotype is circulating. To our knowledge, this study reports for the first time a deer-origin bopivirus and the presence of a member of the genus *Bopivirus* in Australia.

A nearly complete genome of an endogenous betaretrovirus was characterised for the first time in fallow deer (Huaman *et al.* 2022*a*). Further genomic analysis showed that this provirus, tentatively named cervid endogenous betaretrovirus 1 (CERV β1), has typical betaretroviral genome features (*gag-pro-pol-env*). In addition, CERV β1 pol sequences were detected by PCR in wild populations of all the six non-native deer species in Australia. Phylogenetic analyses suggested that CERV β1 endogenisation occurred between 3.3 and 5 million years ago (Huaman *et al.* 2022*a*). Although this provirus does not appear to constitute a current risk for livestock, these results provide important insights into the evolution of betaretroviruses in cervids.

Parasitic diseases in deer

Unlike viral infections, research into parasitic infections on Australian deer populations was mostly conducted in the last 20 years, the majority of which was reviewed in Cripps *et al.* (2019). The research has focused on detecting gastrointestinal parasites and helminths. However, exposure to *Sarcocystis*, *Entamoeba*, and *Neospora caninum* and four vector-borne parasites (*Babesia*, *Theileria*, *Trypanosoma*, and *Plasmodium*) was recently assessed in PCR-based assays in wild deer from eastern Australia (Table 2).

PCR-based screening of deer serum and blood samples detected no active infection (i.e. where the pathogen replicates) of *Sarcocystis* and a range of vector-borne infections relevant for the livestock industry (Huaman *et al.* 2020, 2021*a*). Despite this non-detection, it remains possible that deer could become infected with these parasites (and act as a source of spill-back infection) in the future, given their endemicity in livestock, the presence of suitable vectors, and the ongoing spread of deer (Davis *et al.* 2016). This survey represents the first large-scale molecular study of its type in Australian deer and provides important baseline information about the infection status of wild deer in eastern Australia.

Following a similar research project detecting *N. caninum* parasites in wild dogs (*Canis familiaris*) (Davidson *et al.* 2022), and given the current gaps in knowledge about the sylvatic life cycle of this parasite, the seroprevalence of *N. caninum* was investigated in wild deer (*unpublished data*). Using a competitive ELISA (cELISA) test, a seroprevalence of 4% was detected in deer serum samples from fallow, red, and sambar deer collected in south-eastern Australia. These results suggest that wild deer contribute to the sylvatic cycle of this parasite in Australia. To our knowledge, this is the first reported detection of *N. caninum* antibodies in Australian wild deer and highlights the usefulness of cELISA for assessing serological assays in wildlife populations.

The intestinal parasite *Entamoeba bovis* has been previously detected in farm and wild ruminants worldwide, but its epidemiology and distribution in wild ruminants remain largely unexplored (Stensvold *et al.* 2010). *E. bovis* has also been detected in Australia, including in feral goats (*Capra hircus*) from Western Australia (Al-Habsi *et al.* 2017) and wild deer from eastern Australia (Huaman *et al.* 2022*b*). The cross-species infection between deer and livestock species was evaluated by estimating the time to a most recent common ancestor (TMCRA) of *E. bovis* sequences of wild deer and cattle origin (Huaman *et al.* 2022*b*). The TMCRA in this study was estimated to be >200 years ago (i.e. before cattle and deer were introduced to Australia), providing no evidence of *E. bovis* transmission between wild deer and cattle in Australia. This finding is surprising, but it is possible that wild deer populations have until recently been at a too-low density to play an important role in the transmission of these parasites.

Panozzo (2018) focused on interspecific transmission of gastro-intestinal parasites between livestock and wild deer (*C. unicolor*, *D. dama* and *A. porcinus*). She showed that the prevalence of parasites typically associated with cattle (e.g. *Monieza spp.*) was significantly higher in deer samples collected close to farmland, suggesting (but not proving) inter-species transmission. This study reported a high number of parasite species from deer samples that are relevant to the livestock industry and established that about a quarter of deer samples were positive for *Fasciola hepatica* and gastro-intestinal parasites, including *O. ostertagi*, *O. leptospicularis*, *Haemonchus spp.*, *O. radiatum*, *Trichostrongylus spp.*, and *C. oncophora*. The high prevalence of *F. hepatica* is consistent with the findings of Jenkins *et al.* (2020) and Lamb *et al.* (2021). Both studies focused on identifying the risk that deer pose to maintaining the infection of *F. hepatica* in livestock and established that approximately 50% of the samples were positive for this important parasite. These results led Jenkins *et al.* (2020) to conclude that deer are likely to represent a

Table 2. Parasitic infections investigated in wild deer in Australia.

Pathogen (genera or species)	Sample size	Prevalence (%)	Deer species	Diagnostic method	State or territory	Reference
Babesia, *Plasmodium*, *Theileria*, *Trypanosoma*, *Sarcosytis*	31	0.0	Fallow	PCR	ACT	Huaman *et al.* (2021*a*)
	105	0.0	Fallow		NSW	
	63	0.0	Rusa			
	2	0.0	Fallow		Vic.	
	38	0.0	Sambar			
	4	0.0	Chital		Qld	
Babesia bovis	105	0.0	Chital	IFAT	Qld	
Cryptosporidium	700-950	1.0–9.0	Fallow, red, sambar	PCR	Vic.	Cinque *et al.* (2008), Koehler *et al.* (2016), Nolan *et al.* (2013)
	137	0.7	ND	PCR	NSW	Ng *et al.* (2011)
Entamoeba bovis	48	72.9	Fallow	PCR	NSW	Huaman *et al.* (2022*b*)
	12	100.0			Vic.	
	11	100.0	Sambar			
Fasciola hepatica	15	1.0	Red	M.E	Qld	McKenzie *et al.* (1985)
	17	53.0	Fallow	M.E	NSW	Jenkins *et al.* (2020)
	1	0.0	Fallow	M.E	ACT	
	1	0.0	Sambar	M.E	Vic.	
	79	45.0	Fallow	M.E	NSW	Lamb *et al.* (2021)
	ND	15.0	Hog	M.E	Vic.	Game Management Authority (2006)
Giardia	137	21.2	ND	PCR	NSW	Ng *et al.* (2011)
	950	0.0–14.0	Fallow, red, and sambar	PCR	Vic.	Koehler *et al.* (2016), Nolan *et al.* (2013)
Neospora caninum	76	1.3	Fallow	ELISA	NSW	Huaman *et al.* (unpubl. data)
	6	0.0	Red			
	3	0.0	Sambar			
	21	0.0	Fallow		Vic.	
	8	0.0	Red			
	75	8.0	Sambar			
Toxoplasma	74	0.0	Fallow	IFAT	Tas.	Munday (1972)
Other gastrointestinal helminths	22	2.0–18.0	Red	M.E	Qld	McKenzie *et al.* (1985)
	ND	ND	Fallow, rusa	M.EI	NSW	Moriarty (2004*b*), Munday (1972), Mylrea *et al.* (1991)
	312	15.0-60.0	Fallow, sambar, hog	M.E, PCR, NGS	Vic.	Davies (2014), Panozzo (2018)

ND, no data; Qld, Queensland; NSW, New South Wales; ACT, Australian Capital Territory; Vic., Victoria; Tas., Tasmania; VNT, Virus neutralisation test; IFAT, Immunofluorescence antibody test; M.E, microscopic examination; NGS, next-generation sequencing.

major source of environmental contamination. The presence of these parasites reduces livestock growth rates, which delays animals reaching slaughter weight (Roeber *et al.* 2013; Mazeri *et al.* 2017).

Current and future directions

This review suggests a low risk of transmission from wild deer to livestock in Australia. However, in Europe and North America wild deer were considered an important source of infection for livestock, and several publications focused on their local consequences and implications (Böhm *et al.* 2007; Conner *et al.* 2008; Martin *et al.* 2011; Ruiz-Fons *et al.* 2014). This discrepancy likely reflects differences between Australia and Europe/North America in climate, farming practices, and potential wildlife hosts. Changes in weather patterns in Australia due to climate change can alter the distributions, movements, and dispersal rates of invertebrate vectors (Bryan *et al.* 1996). Climate change could also

influence the distributions and abundances of wildlife hosts. Thus, climate patterns can significantly impact pathogen transmission and interactions between wildlife hosts, vectors, and humans. Indeed, climate change is increasingly recognised as playing a major role in the emergence of vector-borne diseases in humans and wildlife (Fouque and Reeder 2019; Rocklov and Dubrow 2020). Therefore, long-term changes, especially warming temperatures, could significantly alter the distribution and prevalence of vector-borne diseases in wildlife populations (Fouque and Reeder 2019).

Climate change may pose a similar risk when considering the distribution of deer, and the potential for the spread of wild deer to new parts of Australia could result in new epidemiological challenges. The social behaviour of hosts is also likely to be an important factor influencing the epidemiology of some pathogens. Each of the six deer species in Australia congregates at preferred feeding areas, increasing the probability of disease spread among individuals. Male deer may further contribute to the risk of disease transmission through contact with multiple females during the breeding season.

Advances in high-throughput sequencing and bioinformatics have greatly increased our understanding and capacity to identify novel microorganisms. For example, in the last decade, a novel virus (Schmallenberg virus – SBV) was described in European cattle, constituting an emerging threat to the livestock industry in Europe and worldwide (Hoffmann *et al.* 2012; Endalew *et al.* 2019). Since its description in 2011, high seroprevalence of SBV has been detected in wild deer in Europe, indicating that deer could act as a source of this virus for livestock (Mouchantat *et al.* 2015; Garcia-Bocanegra *et al.* 2017; Jimenez-Ruiz *et al.* 2021). As mentioned above, next-generation sequencing was instrumental in identifying a novel *Bopivirus* in fallow and rusa deer from south-eastern Australia (Huaman *et al.* 2021*b*). The authors hypothesised that this virus, like other enteric picornaviruses, is transmitted via the faecal–oral route. However, further work is required to determine its distribution in other deer species (e.g. chital, rusa and hog deer), and livestock species other than cattle, to increase our understanding of the potential for cross-transmission between wild deer and livestock.

As discussed above, deer are infected by and susceptible to many diseases, some of which are zoonotic, meaning that they can also affect humans. Increasing the focus on wildlife disease surveillance to detect emerging infectious diseases and integrate wildlife and environmental health into One Health policies (Cunningham *et al.* 2017) is crucial to prepare Australia to better recognise and manage the adverse impacts of zoonotic diseases. Indeed, high wild deer densities (e.g. 39 per km^2; Bengsen *et al.* 2022) may cause concern for human health via the transmission of infectious agents through direct contact, the consumption of venison, or contamination of the environment (particularly water) with faeces or urine.

Several zoonotic diseases occur in wild deer in Australia, including Q fever, leptospirosis, fasciolosis, cryptosporidiosis, and giardiasis. Recently, attention has turned to the potential role of deer as reservoirs of the SARS-CoV-2 virus, which causes COVID-19 in humans. There was high seroprevalence of SARS-CoV-2 in free-ranging white-tailed deer (*Odocoileus virginianus*) in the USA, revealing high susceptibility to infection of this deer species and active deer-to-deer transmission (Murphy and Ly 2021; Palmer *et al.* 2021; Hale *et al.* 2022). Moreover, the identification of SARS-CoV-2 variants suggests human-to-deer infections and that wild deer can sustain transmission (Hale *et al.* 2022). In contrast, no evidence of SARS-CoV-2 infection was found in common European deer species (Holding *et al.* 2022; Moreira-Soto *et al.* 2022). These studies indicate that deer should be considered when identifying potential reservoirs and intermediate hosts of emerging zoonotic diseases.

Finally, we recommend developing a passive surveillance system through the Australian deer hunter community. Deer hunters harvest large numbers of deer (Moloney *et al.* 2022). Training in identifying, recording macroscopic lesions, and collecting specimens could be provided to hunters as part of the licensing process, and hunters could help monitor the presence and distribution of infectious diseases. The investigations conducted in the last 5 years provide vital baseline data for future research. Monitoring changes in the disease status would offer a more comprehensive view of the dynamics of infectious diseases, which is important if the risks of those disease to humans, livestock, and wildlife are to be minimised. These findings extended our knowledge of known and novel viruses and parasites associated with Australian deer.

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Data availability. This manuscript reviewed published literature; as such, no new data were generated in this study.

Conflicts of interest. David Forsyth and Anthony Pople were guest Associate Editors for this special issue. Despite this relationship, they did not at any stage have editor-level access to this manuscript while in peer review, as is the standard practice when handling manuscripts submitted by an editor to this journal. *Wildlife Research* encourages its editors to publish in the journal, and they are kept separate from their manuscripts' decision-making process. The authors have no further conflicts of interest to declare.

Declaration of funding. This study was funded by the Centre for Invasive Species Solutions (PO1-L-002). The funding body had no role in the preparation or review of the manuscript.

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