Ecological and Evolutionary Perspectives on Transmissible Viral Tumours in Wild Species

A. Garcês, I. Pires and F. Silva*

Abstract

An oncogenic virus is a virus that has the potential to cause cancer. These viruses can induce the malignant transformation of cells by various mechanisms, such as integrating their genetic material into the host cell's genome or by expressing viral proteins that interfere with normal cellular processes. Understanding and monitoring oncoviruses in wildlife is essential for wildlife conservation efforts. This review aims to provide an overview of oncoviruses of veterinary importance in wild animals and their ecological and evolutionary perspectives on transmissibility. These viruses can contribute to declines in population numbers and alter the dynamics of ecosystems. Conservation biologists and veterinarians work to study and manage these diseases to mitigate their impact on wildlife populations.

Key words: *oncogenic; virus; tumours; wildlife*

Introduction

An oncogenic virus is a virus that has the potential to cause cancer (Mui et al., 2017). The first animal oncovirus discovered was the "Rous sarcoma virus (RSV)", a retrovirus identified by American virologist Peyton Rous in 1911. Peyton Rous made this groundbreaking discovery while working with sarcomas (cancers of connective tissues) in chickens (Rous, 1910). Several viruses are known to be oncogenic and classified into two main groups: DNA viruses and RNA viruses (Tempera and Lieberman, 2021). Oncogenic viruses can be divided into direct and indirect oncogenic viruses (Elkhalifa et al., 2023). Direct oncogenic viruses contribute directly to neoplastic cellular transformation, while indirect oncogenic viruses cause chronic inflammation that can lead to tumour development (Elkhalifa et al., 2023).

These viruses can induce malignant cell transformation by various mechanisms. It is important to note that while

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these viruses can increase the risk of cancer, not every animal infected with an oncogenic virus will develop cancer, and even if it develops, it can take years before the first signs appear. Other factors, including the individual's immune response and genetic predisposition, also play a role in cancer development (Xie et al., 2004; Tempera and Lieberman, 2021).

Cancer has been recorded in almost every group of animals, and there is significant variability in the nature and incidence of tumours among species. This disease is a significant factor in morbidity and mortality among various wildlife species. Wildlife cancer detection can be complex; in most cases, it goes largely undetected (McAloose and Newton, 2009). Difficulty in accessing animals (dead or alive), whether due to environmental obstacles (e.g. oceans, deserts, jungles), species-specific adaptations (e.g. deep dive), decomposition, predation or scavenging, or a lack of resources in the field, both human and financial, hinder cancer detection in these populations (Acevedo-Whitehouse and Duffus, 2009; McAloose and Newton, 2009). However, wildlife health monitoring has been increasing in recent years, providing new epidemiological and alarming information concerning the presence and impact of cancer in wildlife populations (McAloose and Newton, 2009).

Oncoviruses are not limited to human populations or domestic animals; they can also affect various wildlife species. In the wild, oncoviruses have been identified in different animals, and these infections can contribute to developing cancers in those species (Parisi et al., 2023). Wildlife oncoviruses can be a significant concern for conservation efforts, as they may impact the health and survival of vulnerable populations (McAloose and Newton, 2009; Mui et al., 2017; Frias-De-Diego et al., 2019). Cancer has become a threat to already endangered populations such as the Western Barred Bandicoot (*Perameles bougainville*) or Loggerhead Turtle (*Caretta caretta*) (McAloose and Newton, 2009). This review gives an overview of the most common oncoviruses of veterinary importance in wild animals and their ecological and evolutionary perspectives on transmissibility.

Oncoviruses in wildlife

Several viruses have been identified as oncoviruses (e.g. Papillomaviridae, Herpesviridae) and are associated with various types of animal cancers (Mui et al., 2017). Here, the authors describe some examples of oncoviruses from different virus families:

Papillomaviridae

Papillomaviruses in wildlife

Papillomaviruses (PV) are small, unenveloped DNA viruses known to produce lesions on the skin and mucous membranes (fibropapillomas and papillomas) of various species, including humans (Araldi et al., 2017; Frias-De-Diego et al., 2019; Rector and Van Ranst, 2013). The PV family has 26 genera, 62 species and 69 known animal PVs with high genetic variability (Table 2). Each papillomavirus species tends to be specific to a particular host species or a closely related group (Humans, 2007; Rector and Van Ranst, 2013). PV has been reported in several wild animals such as *Camelus dromedarius, Alces alces, Capreolus capreolus, Lynx rufus, Phocoena phocoena, Macaca fascicularis, Trichechus manatus latirostris* or *Morelia spilota spilota* (Table 2) (Rector and Van Ranst, 2013). Skin warts are the most common sign of infection with PV. They are usually benign tumours in the skin, but can be malignant when they

develop in the genital tract. PV replicates exclusively in keratinocytes (Lambertsen et al., 1987; Araldi et al., 2017;). The most common macroscopic manifestation of PV infections is the development of warts or papillomas that are raised, rough, and well-demarcated growths on the skin or mucous membranes. They may be small, discrete papules or larger, cauliflower-like structures with multiple finger-like projections and white rough or verrucous texture (Vengušt and Žele, 2012). The colour may be flesh-coloured, pink, brown, or hyperpigmented, depending on the skin pigmentation and inflammation (Garcês et al., 2020). The lesions can be solitary or multiple, and common growth sites include the hands, feet, face, genital, perianal regions, and mucous membranes (De Guise et al., 1994; Rector and Van Ranst, 2013; Frias-De-Diego et al., 2019). They may ulcerate and bleed. Histopathological characteristics can vary depending on the type of papillomavirus, the host species, and the specific tissue involved. It is possible to observe hyperplasia, koilocytosis, acanthosis, papillomatosis, parakeratosis, dysplasia, mitotic figures and inflammatory response (Figure 1).

Transmission of PV requires close cutaneous or mucosal contact with microtraumas in the skin or mucosal surface (Rector and Van Ranst, 2013). Transmission from infected females to susceptible calves through skin contact during suckling or from males to females during breeding is also possible (De Guise et al., 1994; Humans, 2007; Rector and Van Ranst, 2013). Studies also show that the virus can be transmitted by flies and ticks (Araldi et al., 2017).

Herpesviridae

Urogenital carcinoma in wild California sea lions

Around 18–23% of adult animals in wild California Sea Lion (*Zalophus californianus*) populations examined *post-mortem* over the past 40 years displayed urogenital carcinomas (Gulland et al., 2020). The aetiology of this malignant neoplasia remains poorly understood, but researchers believe it is multifactorial, with pollution (organochlorines and polychlorinated biphenyls), genotype and infection with Otarine herpesvirus-1 (OtHV-1) as possible agents (King et al., 2002; Gulland et al., 2020). Figure 2 represents the dis-

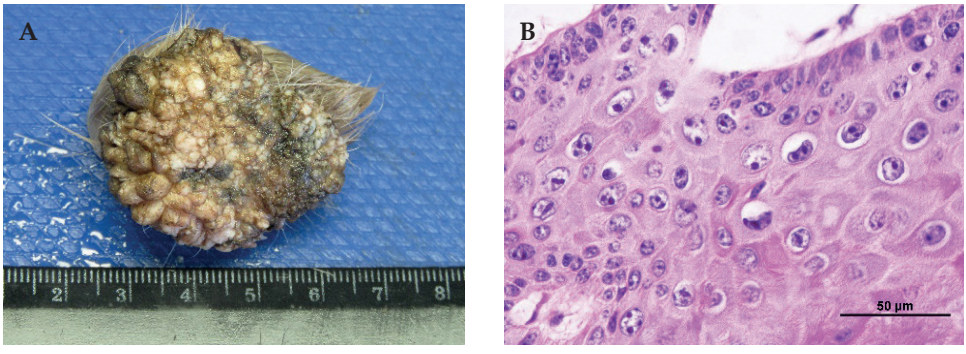

Figure 1. A – Skin fibropapilloma (4x3x3 cm) in Red Deer (*Cervus elaphus*). B - The presence of koilocytes in the spinal stratum and images suggest intranuclear basophilic inclusions in a skin fibropapilloma in Red Deer (*Cervus elaphus*) (Photo Andreia Garcês/Isabel Pires).

tribution of OtHV-1 worldwide. Primary tumours occur in the cervix, vagina, penis, or prepuce and aggressively metastasise, resulting in death (Venn-Watson et al., 2012; Deming et al., 2021) vagina, penis, or prepuce and aggressively metastasize resulting in death. This cancer has been strongly associated with a sexually transmitted herpesvirus, otarine herpesvirus 1 (OtHV1). The presence of thickened and dysplastic mucosal epithelium characterises the histological lesions. Distinct cytoplasmic borders, polygonal to round cells with moderate amounts of cytoplasm, squamous cell differentiation/keratinisation are present and, in some females, glandular differentiation. Intranuclear inclusion bodies were identified in primary neoplasia and metastatic lesions (abdominal and pelvic lymph nodes, kidney, urinary bladder, liver, lungs and spleen) (Browning et al., 2015). Cytokeratin positivity confirms tumour epithelial ontogeny (Newman and Smith, 2006a; Deming et al., 2018;).

Pollution has already been associated with other tumours, such as the gastric papilloma in the St Lawrence River's Beluga (*Delphinapterus leucas*) (De Guise et al., 1994). Studies showed that blubber from sea lions with urogenital carcinoma had significantly higher levels of polychlorinated biphenyls and DDTs than blubber from healthy sea lions without carcinoma (Newman and Smith, 2006a; Deming et al., 2018).

OtHV1 is a gammaherpesvirus in the genus *Rhadinovirushas*. Electron microscopy, immunohistochemistry, and polymerase chain reaction (PCR) studies have shown the presence of herpesvirus in affected animals (Buckles et al., 2006; Newman and Smith, 2006b). OtH-1 is more prevalent in adult tissue than in juveniles. The virus has been associated with sexual transmission, and Deming et al. (2021) identified that non-cancer OtHV1-positive cases had significantly lower viral loads in their cervix compared to the cervix of animals with urogenital carcinoma (Deming et al., 2021). Vertical transmission during pregnancy or birth has been suggested to introduce OtHV1 to new individuals. Infected mothers may pass the virus to their offspring, contributing to the persistence of the virus in the population (Buckles et al., 2007). Two potential oncogenes were identified in OtHV-1, the viral B-cell lymphoma 2 gene (vBCL2) and viral Fas-associated death-like interleukin-1 beta-converting enzyme-inhibitory protein (vFLIP). Both genes are of host origin and are observed in other gamma-herpesviruses that induce cancer in humans and animals by preventing apoptosis during cell division.

Genetic predisposition to cancer also is possible, as these carcinomas are more common in relatively inbred individuals (Acevedo-Whitehouse et al., 2003). OtHV1, like other herpesvirus, often exhibits latency, where the virus remains dormant within the host cells. Stressors, such as changes in the environment, competition, or compromised immune function, can trigger the reactivation of the virus, leading to shedding and potential transmission (Venn-Watson et al., 2012; Deming et al., 2021).

Retroviridae

Koala retrovirus

The koala (*Phascolarctos cinereus*) population is affected by a virus-denominated koala retrovirus (KoRV) (Figure 3). KoRV belongs to the family Retroviridae, genus *Gammaretrovirus*, and the virions contain a positive-sense single-stranded RNA genome of 8,431 nucleotides in length (Xu and Eiden, 2015). This virus is relatively recent and has become endogenous and fixed in the genome of every koala cell, and it is transmitted vertically

Figure 2. Green the distribution of OtHV1 in California sea lion (*Z. californianus*) (Author: Andreia Garcês).

(Zheng et al., 2020). KoRV has been divided into three major clades and nine subtypes (KoRV-A to KoRV-M) based on sequence differences in the receptor binding domain of the envelope protein gene. The virus has shown high homology with the Gibbon Ape Leukaemia Virus (GALV) (Xu and Eiden, 2015).

Most koalas found positive for KoRV do not have clinical signs of the disease. KoRV-G, KoRV-H, and KoRV-I subtypes have been identified in diseased wild Australian koalas (Stephenson et al., 2021). This virus is associated with the development of lymphoid neoplasia, and in northern koalas (lymphoma and leukaemia), some exogenous subtypes have been associated with more severe chlamydiosis (Stoye, 2006).

Bandicoot papillomatosis carcinomatosis virus

The Western Barred Bandicoot (*Perameles bougainville)* population has been affected by cutaneous and mucocutaneous papillomatosis and carcinomatosis syndrome in both captive and wild individuals. Bandicoot papillomatosis carcinomatosis virus type 1 (BPCV1) is the cause of this disease (Figure 3). BPCVs are double-stranded DNA viruses and are founding members of a novel virus family. Histological, ultrastructural, and immunohistochemical analyses have shown the involvement of papillomavirus (PV) or polyomavirus (PyV) in the pathogenesis of this disease (Bennett et al., 2008b) tentatively named bandicoot papillomatosis carcinomatosis virus type 2 (BPCV2. A case of BPCV-2 infection in a Southern Brown Bandicoot has been detected (Bennett et al., 2008b).

Clinically, the lesions appear as irregular thickenings and masses in the skin of the digits, body, pouch, mucocutaneous junctions of the lips, and conjunctiva (Woolford et al., 2008). Lesions can lead to difficulties in vision, locomotion, and ability to feed, depending on the anatomic location of the lesions. The lesions may also become abraded, ulcerated, and secondarily infected, leading to sometimes fatal complications. Metastases have been observed in lymph nodes, lungs, and the liver (Bennett et al., 2008a; Woolford et al., 2007, 2008).

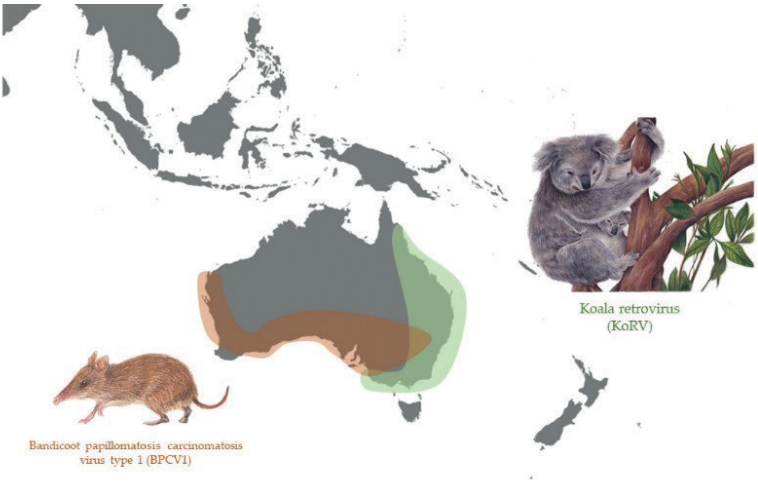

Figure 3. Distribution of koala retrovirus (KoRV) and bandicoot papillomatosis carcinomatosis virus type 1 (BPCV1) (Author: Andreia Garcês).

BPCVs appear to be species-specific. Transmission between individuals can occur through direct contact (e.g., mating, territorial fighting, and foraging behaviour) or vertically (from parents to offspring). While less common, transmission through fomites (inanimate objects or surfaces) contaminated with the virus is possible (Woolford et al., 2007; Bennett et al., 2008a).

Polyomaviridae

Polyomavirus in raccoons

Raccoon polyomavirus (RacPyV) is one of the most recently discovered members of the Polyomaviridae family (Figure 4). RacPyV was found 2010 in North American free-ranging raccoons (Dela Cruz et al., 2013). Polyomaviruses have a circular double-stranded DNA genome. The genomic structure of RacPyV includes genes that encode viral proteins, and its genome is organised into early and late regions (Giannitti et al., 2014; Lu et al., 2017).

RacPyV causes asymptomatic or mild infections (respiratory and gastrointestinal), but in some cases, they have been associated with the development of tumours. This virus has been associated with malignant peripheral nerve sheath tumours and undifferentiated sarcomas (Giannitti et al., 2014). Metastases have only been reported in a single case (Pesavento et al., 2018). Studies suggest that the target cell of virus transformation is the multipotent neuroglial stem cells of the brain's subventricular zone (Church et al., 2016; Pesavento et al., 2018).

The precise mode of transmission remains unknown. It is thought to be transmitted through the oral-faecal route and possibly through respiratory secretions (Giannitti et al., 2014; Church et al., 2016).

Table 2 lists oncogenic viruses present in wildlife, their host, type of tumour and virus strain.

Ecological and Evolutionary Perspectives

From an ecological perspective, transmissible oncovirus can significantly impact the population dynamics of an affected species.

Figure 4. Distribution of Raccoon polyomavirus (RacPyV) (Author: Andreia Garcês).

Table 2. Oncogenic viruses are present in wildlife.

Species	Tumour	Virus Strain	Ref.
Papillomaviridae (DNA viruses)			
Sperm Whale (*Physeter catodon*)	Genital papilloma	n/a	(Lambertsen et al., 1987)
Burmeister's Porpoise (*Phocoena spinipinnis*)		PsPV1	(Van Bressem et al., 2007)
Dusky Dolphin (*Lagenorhynchus obscurus*)		n/a	(Van Bressem et al., 1999, 2000)
Bottlenose Dolphin (*Tursiops truncatus*)	Genital papilloma, lingual papilloma, cutaneous papilloma and fibropapilloma	TtPV2, TtPV1, TtPV3, TtPV4, TtPV5, TtPV6, TtPV7	(Van Bressem et al., 1999; Rehtanz et al., 2006)
Common Bent-wing Bat (*Miniopterus schreibersii*)	Cutaneous papilloma	MscPV1, MscPV1 TT20F	(Rector and Van Ranst, 2013)
Moose (*Alces alces*)		AaPV1 (EEPV)	(Rector and Van Ranst, 2013)
Yak (*Bos grunniens*)		BgPV1	(Rector and Van Ranst, 2013)
Arabian Camel (*Camelus dromedarius*)		CdPV1, CdPV2	(Rector and Van Ranst, 2013)
Roe Deer (*Capreolus capreolus*)		CcaPV1 (RdPV1, CcPV1)	(Rector and Van Ranst, 2013)
White-tailed Deer (*Odocoileus virginianus*)		OvPV1 (DPV)	(Rector and Van Ranst, 2013)
Reindeer (*Ranifer tarandus*)		RtPV1 (RPV)	(Rector and Van Ranst, 2013)
Bobcat (*Lynx rufus*)		LrPV1	(Rector and Van Ranst, 2013)
Asiatic Lion (*Panthera leo persica*)		PlpPV1	(Rector and Van Ranst, 2013)
Racoon (*Procyon lotor*)		PlPV1	(Rector and Van Ranst, 2013)
Cougar (*Puma conclor*)		PCPV1	(Rector and Van Ranst, 2013)

Snow Leopard (*Uncia uncia*)	Cutaneous papilloma	UuPV1	(Rector and Van Ranst, 2013)
Polar Bear (*Ursus maritimus*)		UmPV1	(Rector and Van Ranst, 2013)
California Sea Lion (*Zalophus californianus*)		ZcPV1	(Rector and Van Ranst, 2013)
Red Deer (*Cervus elaphus*)		CePV1, BPV-1	(Garcês et al., 2020; Savini et al., 2016; Scagliarini et al., 2013)
Short-beaked Common Dolphin (*Delphinus delphis*)		DdPV1	(Rector and Van Ranst, 2013)
Harbor Porpoise (*Phocoena phocoena*)		PphPV1, PphPV2, PphPV4	(Rector and Van Ranst, 2013)
Rickett's Big-footed Bat (*Myotis ricketti*)		MrPV1	(Rector and Van Ranst, 2013)
Egyptian Fruit Bat (*Rousettus aegyptiacus*)		RaPVB1	(Rector and Van Ranst, 2013)
Giraffe (*Giraffa camelopardalis*)		GcPV1	(Pesavento et al., 2018; Vanmechelen et al., 2017)
Brush-tailed Bettong (*Bettongia penicillata*)		BpPV1	(Rector and Van Ranst, 2013)
European Hedgehog (*Erinaceus europaeus*)		EePV1 (EHPV)	(Rector and Van Ranst, 2013)
Common Chaffinch (*Fringilla coelebs*)		FcPV1	(Rector and Van Ranst, 2013)
Colobus Monkey (*Colobus guereza*)		CgPV1, CgPV2	(Rector and Van Ranst, 2013)
Cynimolgus Macaque (*Macaca fascicularis*)		MfPV1 to MfPV11	(Rector and Van Ranst, 2013)
Rhesus Macaque (*Macaca mulatta*)		MmPV1 (RhPV1)	(Rector and Van Ranst, 2013)
Bonobo (*Pan paniscus*)		PpPV1	(Rector and Van Ranst, 2013)
Chimpanzee (*Pan trogodytes*)		PtPV1	(Rector and Van Ranst, 2013)
Hamadryas Baboon (*Papio hamadryas*)		Pepv1	(Rector and Van Ranst, 2013)
African Grey Parrot (*Psittacus erithacus*)		PePV1	(Rector and Van Ranst, 2013)
Wood Mouse (*Apodemus sylvaticus*)		AsPV1	(Rector and Van Ranst, 2013)
Porcupine (*Erethizon dorsatum*)		EdPV1	(Rector and Van Ranst, 2013)
Deer Mouse (*Peromyscus manuculatus*)		PmPV1	(Rector and Van Ranst, 2013)
Diamond Python (*Morelia s. spilota*)		MsPV1	(Rector and Van Ranst, 2013)
Florida Manatee (*Trichechus manatus latirostris*)		TmPV1, TmPV2	(Rector and Van Ranst, 2013)
Water Buffalo (*Bubalus arnee*)		BPV-2	(Roperto et al., 2013)
Loggerhead Sea Turtle (*Caretta caretta*)		CcPV1	(Rector and Van Ranst, 2013)
Green Sea Turtle (*Chelonia mydas*)		CmPV1	(Rector and Van Ranst, 2013)
Zebra (*Equus* spp.)		BPV-1	(Rector and Van Ranst, 2013)
Stone Marten (*Martes foina*)		MfoiPV1	(Kuhar et al., 2021)

Beluga Whale (*Delphinapterus leucas*)	Gastric papilloma	PV-HMU-1, PV-HMU-2	(Li et al., 2023; Martineau et al., 2002)
Northern Fulmar (*Fulmarus glacialis*)	Mesenchymal neoplasm	FgPV1	(Gaynor et al., 2015)
Tapir (*Tapirus* sp.)	Sarcoid	n/a	(Kidney and Berrocal, 2008)
Herpesviridae (DNA viruses)			
California Sea Lion (*Zalophus californianus*)	Genital carcinoma, lymphoma	OtHV1, OtHV2, OtHV3	(King et al., 2002; Venn-Watson et al., 2012)
South American Fur Seal (*Arctocephalus australis*)	Genital carcinoma	OtHV1	(Dagleish et al., 2013)
Loggerhead (*Caretta caretta*)	Fibropapillomas	ChHV5	(Lu et al., 1992)
Hawksbill (*Eretmochelys imbricata*)	Fibropapillomas	ChHV5	(Lu et al., 1992)
Flatback (*Natator depressus*)	Fibropapillomas	ChHV5	(Lu et al., 1992)
Olive Ridley (*Lepidochelys olivacea*)	Fibropapillomas	ChHV5	(Lu et al., 1992)
Leatherback (*Dermochelys coriacea*)	Fibropapillomas	ChHV5	(Lu et al., 1992)
Kemp's Ridley (*Lepidochelys kempii*)	Fibropapillomas	ChHV5	(Lu et al., 1992)
Leopard Frog (*Rana pipiens*)	Renal carcinoma	LTHV	(Granoff, 1973)
White-fronted Goose (*Anser albifrons*)	Malignant lymphoma	MDV-1	(Murata et al., 2012)
Mountain Gorilla (*Gorilla beringei beringei*)	Lymphoid hyperplasia	GbbLCV-1	(Smiley Evans et al., 2017)
Hepadnaviridae (DNA viruses)			
Woodchuck (*Marmota monax*)	Hepatocellular carcinoma	WHV	(Summers et al., 1978)
Retroviridae (RNA viruses)			
Walleye Fish (*Sander vitreus*)	Dermal sarcoma	WDSV	(Martineau et al., 1992; Rovnak and Quackenbush, 2010)
Attwater's Prairie Chicken (*Tympanuchus cupido attwateri*)	Lymphomas	REVs	(Drew et al., 1998)
Atlantic salmon (*Salmo salar*)	Swim bladder leiomyosarcoma	SSSV	(Paul et al., 2006)
Koala (*Phascolarctos cinereus*)	Lymphoma/ leukaemia, osteochondroma	KoRV-A to KoRV-M	(Williams and Barker, 2000)
Gibbon (*Hylobates lar)*	Lymphosarcoma, lymphoblastic leukaemia, myelogenous leukaemia	GaLV-SEATO, GaLV-Br, GaLV-H, GaLV-X, GaLV-SF	(Williams and Barker, 2000)
Wildcat (*Felis silvestris*)	Leukaemia	FeLV	(Fromont et al., 2000)
Cougar (*Puma concolor*)	Leukaemia	FeLV	(Petch et al., 2022)
Jaguar (*Panthera onca*)	Leukaemia	FeLV	(Silva et al., 2016)
Guigna (*Leopardus guigna*)	Leukaemia	FeLV, FIV	(Mora et al., 2015)

Tiger (*Panthera tigris*)	Leukaemia	FeLV	(Tangsugjai et al., 2010, pp. 2004–2005)
Leopard (*Panthera pardus*)	Leukaemia	FeLV	(Tangsugjai et al., 2010, pp. 2004–2005)
Clouded Leopard (*Neofelis nebulosa*)	Leukaemia	FeLV	(Tangsugjai et al., 2010, pp. 2004–2005)
Fishing Cat (*Prionailurus viverrinus*)	Leukaemia	FeLV	(Tangsugjai et al., 2010, pp. 2004–2005)
Leopard Cat (*Felis bengalensis*)	Leukaemia	FeLV	(Tangsugjai et al., 2010, pp. 2004–2005)
Iberian Lynx (*Lynx pardinus*)	Leukaemia	FeLV	(Luaces et al., 2008)
Chinook Salmon (*Oncorhynchus tshawytscha*)	Plasmacytoid leukaemia	WDSV	(Eaton et al., 1993)
Northern Pike (*Esox lucius*)	Esocid lymphosarcoma or lymphoma	n/a	(Coffee et al., 2013)
Muskellunge (*Esox masquinongy*)	Esocid lymphosarcoma or lymphoma	n/a	(Coffee et al., 2013)
Atlantic Salmon (*Salmo salar*)	Epidermal papillomatosis	n/a	(Coffee et al., 2013)
House Mouse (*Mus musculus*)	Leukaemia and lymphoma	MuLVs	(Chattopadhyay et al., 1981)
White Sucker (*Catostomus commersoni*)	Epidermal papillomas	n/a	(Coffee et al., 2013)
Hooknose (*Agonus cataphractus*)	Cutaneous fibroma/ fibrosarcoma	n/a	(Coffee et al., 2013)
Angelfish (*Pterophyllum scalare*)	Fibroma of the lip	OEHV1	(Coffee et al., 2013)
Bicolor Damselfish (*Stegastes partitus*)	Neurofibromatosis	n/a	(Coffee et al., 2013)
Adenoviridae (DNA viruses)			
Polar Bear (*Ursus maritemus*)	Hepatic neoplasia	PBAdV-1	(Gaynor et al., 2015)
Poxviridae (DNA viruses)			
Eurasian Red Squirrel (*Sciurus vulgaris*)	Skin fibromas	SQPV, BerSQPV	(Atkin et al., 2010; Bruemmer et al., 2010)
Eastern Grey Squirrel (*Sciurus carolinensis*)	Skin fibromas	SQPV	(Atkin et al., 2010; Bruemmer et al., 2010)
North American Red Squirrel (*Tamiasciurus hudsonicus*)	Skin fibromas	SQPV	(Himsworth et al., 2009)
Eastern Cottontail Rabbit (*Sylvilagus floridanus*)	Cutaneous fibromas	RFV/SFV	(Barrett and McFadden, 2007)
Eastern and Californian Grey Squirrel (*Sciurus griseus*)	Cutaneous fibromas	SQFV	(Barrett and McFadden, 2007)
European Hare (*Lepus europaeus*), African Hare (*L. capenus*)	Dermal tumours	FIBV	(Barrett and McFadden, 2007)

Tapeti (*Sylvilagus brasiliensis*), Brush Rabbit (*Sylvilagus bachmani*)	Cutaneous fibromas	MYXV	(Barrett and McFadden, 2007)
Polyomaviridae (DNA viruses)			
Racoon (*Procyon lotor*)	Brain neuroglial tumours	RacPyV	(Church et al., 2016; Dela Cruz et al., 2013)
Others			
Western Barred Bandicoots (*Perameles bougainville*)	Cutaneous and mucocutaneous hyperplasias, papillomas	BPCV1	(Woolford et al., 2008)
Southern Brown Bandicoot (*Isoodon obesulus*)	Cutaneous and mucocutaneous hyperplasias, papillomas	BPCV-2	(Bennett et al., 2008b)

The spread of the tumour can lead to population decline or altered age structures within populations. It may induce changes in the behaviour of affected individuals, for example, alterations in mating performances or social interactions (Hahn, 2002). Habitat structure, host density, and interactions between infected and uninfected individuals affect disease transmission (Mui et al., 2017; Tempera and Lieberman, 2021). These alterations have already been observed in species such as the Eurasian Red Squirrel (*Sciurus vulgaris*) (Shuttleworth et al., 2022) or Western Barred Bandicoots (*Perameles bougainville*) (Woolford et al., 2007), whose populations are in decline due to the high prevalence of tumours caused by a virus.

Another example is the FeLV infection in wild felids, some already very threatened species, due to contact with domestic cats. Outbreaks in these populations could have disastrous consequences. Studies suggest that changes in the environment, like the introduction of artificial feeders, may increase the contact among lynxes and between lynxes and other species, raising the risk of FeLV transmission (Luaces et al., 2008; Tangsugjai et al., 2010). This finding implies that conservation plans should carefully consider any interventions that might inadvertently increase disease risks.

From an evolutionary perspective, the coevolutionary dynamics between the host and the transmissible tumour are of interest since they raise questions about how the host's immune system responds to the cancer cells and how they evolve to evade the immune system. The genetic diversity within host populations also may influence susceptibility to transmissible tumours, therefore, understanding the genetic basis of resistance or susceptibility can provide insights into the evolutionary processes at play (Mui et al., 2017; Tempera and Lieberman, 2021).

Transmissible tumours can have profound evolutionary consequences. If the disease leads to the decline or extinction of a population, it can impact the genetic diversity of the species and, consequently, its evolutionary potential. The cancer cells themselves may undergo adaptive changes to enhance their transmission. Understanding the genetic mechanisms that facilitate transmission is essential to comprehend the evolutionary dynamics of these diseases (Pesavento et al., 2018; Tempera and Lieberman, 2021).

Comprehensive disease control programmes that encompass prevention and eradication are necessary. However, implementing such programmes is often challenging due to limited data, high costs, and logistical difficulties. The study by López et al. (2009) on managing an FeLV outbreak in Iberian lynxes is a relevant example. The plan aimed to conduct serological surveys, isolate infected lynxes, vaccinate uninfected ones, and control feral and domestic cat populations. The outbreak was successfully contained within eight months, and over 80% of the lynx population was vaccinated.

It is urgent to focus on assessing pathogen risks, prioritising surveillance and control strategies, and understanding the role of domestic reservoirs and environmental factors in disease outbreaks. Proactive planning and preparedness are crucial to protect endangered species from potentially devastating disease outbreaks (López, 2009).

Conclusions

It is important to note that the relationship between viruses and animal cancers can vary. In some cases, the association is well-established, while in others, the role of the viruses in cancer development is still poorly studied. Additionally, just as in humans, the development of cancer in animals is likely influenced by a combination of factors, including genetics, the immune system, and environmental factors.

Research on oncoviruses in animals is essential for understanding the mechanisms of cancer development and developing strategies for prevention and treatment. Veterinary medicine and wildlife conservation efforts often focus on monitoring and managing the impact of oncoviruses on animal health.

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Ekološke i evolucijske perspektive na prijenosne virusne tumore u divljih životinja

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Onkogeni virus je virus koji potencijalno može prouzročiti rak. Ti virusi mogu izazvati malignu transformaciju stanica različitim mehanizmima, poput integriranja njihovog genetskog materijala u genom stanice domaćina ili ekspresijom virusnih proteina koji ometaju normalne stanične procese. Razumijevanje i nadziranje onkovirusa u divljih životinja od osnovne je važnosti u naporima za očuvanje divljih životinja. Ovaj pregled ima za cilj pružiti prikaz onkovirusa od veterinarske važnosti u divljih životinja i njihove ekološke i evolucijske perspektive na prijenosne virusne tumore. Ovi virusi mogu doprinijeti smanjenju populacije i izmijeniti dinamiku ekosustava. Konzervacijski biolozi i veterinari rade na proučavanju i upravljanju ovim bolestima kako bi se ublažio njihov utjecaj na populacije divljih životinja.

Ključne riječi: *onkogeno, virus, tumori, divlje životinje*