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SHORT COMMUNICATION

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Are dogs not susceptible to retroviral infections?

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Abstract

Retroviruses have been proven to cause infections and diseases in a series of mammalian hosts but not in dogs. Then, this letter discussed the dog susceptibility to retrovirus infection, encompassing arguments to understand why dogs may have not been infected by retroviruses thus far. The potential resistance of retrovirus in dogs enables this provocative short communication to discuss this question, looking at some evolutive aspects. The lineage of canids has shown, throughout its evolutionary history, a smaller accumulation of retroviruses in canid genomes, classified as endogenous retroviruses. In this context, the genomes of canids seem to offer obstacles, which have been evolutionarily conserved, in the face of retroviral infection.

Keywords Retrovirus; dogs, Endogenous retrovirus

Main text

Retroviruses are pathogens that exhibit remarkable success as intracellular agents and are found infecting a myriad of animal species (Greger 2007). During retroviral infection, their RNA-based genome is reverse transcribed and incorporated into the host genome, utilizing the invaded cellular machinery to replicate and infect other cells (Nisole and Saïb 2004). Occasionally, the retrovirus can become integrated into the host's germline cells. If these cells give rise to offspring, the viral genetic material becomes inherited and passed on to future generations, resulting in an endogenous retrovirus (ERV). ERVs serve as a record of retroviruses that infect a host, integrating into their germline and being passed down to offspring (Lee et al. 2013).

While some retroviruses cause asymptomatic infections, others are markedly pathogenic. Certain domestic species, such as cats (*Felis catus*) and rodents (*Mus musculus*), are significantly affected by retroviral infections (Kurth and Bannert 2010). Retroviruses such as feline immunodeficiency virus (FIV) and feline leukemia virus (FeLV) have a global impact on the health of domestic cats by causing immunosuppression (Hartmann 2012). FIV and FeLV are primarily transmitted through contact with saliva and secretions, typically through bites and scratches from infected animals. FeLV is highly pathogenic and can cause various health issues in domestic cats. These include malignancies, bone marrow suppression syndromes leading to anemia, and an increased susceptibility to secondary infectious diseases. The suppressive effects of FeLV on the immune system contribute to these detrimental effects (Hartmann 2011).

In addition to cats, retroviral infections affect various mammalian species. Cattle, equines, ovines, murines, and koalas are frequently infected by retroviruses, which can lead to immunodeficiency and oncogenesis.

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Although there is a large population of dogs, only a few cases of retrovirus infection have been reported, which is an intriguing finding. Interestingly, cultured dog cells are susceptible to retroviral infection, including retroviruses such as the endogenous feline retrovirus RD114 (Riggs et al. 1974). However, in vivo infection of dogs has not achieved the same success (Narushima et al. 2011). Even in experimental settings, retrovirus-based vectors have failed to infect dogs, despite their demonstrated susceptibility in vitro (Schuening et al. 1988).

Based on a master species list obtained from the International Committee on Taxonomy of Viruses—ICTV (https://ictv.global/msl), the current viruses and their taxonomy were grouped in a spreadsheet. Table 1 presents the existing retroviruses and their respective hosts according to the virus–host database (www.genome.jp/virushostdb). This study identified a range of retroviruses from bovines, equids, felines, glires, humans, koalas, murines, ovines, primates, and monkeys. Surprisingly, the ICTV spreadsheet did not list any retroviruses specific to canids, whether they were domestic or wild. Certain species of retroviruses that infect mammals, such as prairie dogs (North American rodents) (Butler et al. 2020), were not described on the ICTV list. However, the absence of recorded canine retroviruses can be attributed to the lack of retrovirus detection in both domestic and wild canids. This is further supported by the Gypsy Database (gydb.org), a comprehensive retrovirus database encompassing a wide range of species (Llorens et al. 2011), which also indicated the absence of retroviruses infecting canids.

Despite the surprising scarcity of retrovirus cases in canids, sporadic detection of retroviral particles has occurred, leading to immunological findings. Modiano et al. (Modiano et al. 1995) detected a putative retrovirus in a dog that presented intense immunosuppression. The affected dog showed lymphocyte proliferative dysfunction, releasing lower levels of IL-2, and marked depletion of cellular components from lymphoid tissues. Similar to other retroviruses, such as HIV-1, canine retroviral particles have been found to infect cells of the mononuclear lineage in a latent state (Modiano et al. 1995).

An additional study case detected a retrovirus in a dog with leukemia (Safran et al. 1992). The affected dog exhibited significant symptoms, including marked anemia, thrombocytopenia, and an elevated white blood cell count, characterized by a high number of lymphoblasts. The retrovirus was isolated from mononuclear cells obtained from the blood, demonstrating cross reactivity with both exogenous and endogenous retroviruses within the retrovirus family, including representative lentiviruses and oncoviruses (Safran et al. 1992). Establishing a causal relationship between these potential retroviruses and specific disease conditions in dogs has proven to be challenging. It is worth noting that in humans, a series of cancer cases has been linked to high levels of ERV protein expression (Attermann et al. 2018). Therefore, while certain instances of retrovirus particle detection in dogs have been reported in the scientific literature, it is essential to consider the possibility of released ERV particles in these cases.

In addition, viral particles, later identified as retroviruses, were discovered in a lineage of lymphoid cells obtained from a dog diagnosed with cutaneous T-cell lymphoma. These cells were phenotypically characterized as $CD3^+/CD4^-/CD8^+$ (Ghernati et al. 1999). The characteristics of the identified viral particles suggested that these retroviruses could be classified as mammalian type-C retroviruses, similar to FeLV. Interestingly, no potential endogenous retroviral particle was released.

The expression of endogenous retroviral particles has been linked to the functioning of the immune system, which is capable of providing protection to the host against invading exogenous retroviruses. Endogenous viral-origin particles can function to induce an antiviral state that mimics the effects of an exogenous infection through recognition by pattern recognition receptors (PRRs), triggering the innate immune response (Russ and Iordanskiy 2023). Concomitantly, the expression of endogenous retroviral proteins seems to play a role in initiating adaptive immunity against invading exogenous viruses. This is achieved through the regulation of interferon release and the activation of CD4 T cells (Hong et al. 2023). Given these favorable conditions, there is a possibility that canids could benefit from the expression of their endogenous retroviruses. A study conducted by Tarlinton and colleagues demonstrated the presence of ERV transcripts in various healthy tissues and cell lines from dogs (Tarlinton et al. 2013).

The clinical and pathological characteristics of immunological, neoplastic, and degenerative diseases in dogs are often similar to those observed in other species. However, unlike several species where retroviruses have been identified as the causative agents of such diseases, dogs have not been found to be affected by retroviruses at present (Wong-Staal and Gallo 1985). The selective pressure that has shaped the development of most domestic dog breeds (*Canis lupus familiaris*) over the past two hundred years (Plassais et al. 2019) can result in reduced genetic diversity within a population, which can have beneficial effects (Marsden et al. 2016), including potential antiretroviral capabilities. However, in addition to domestic dogs, retroviral infection in wild canids has not been identified. An evolutionary analysis demonstrated that the potential to resist retrovirus infection may have originated prior to the speciation event of domestic dogs

Table 1 Retroviruses and hosts

Host		Retrovirus
	Banteng	Jembrana disease virus
	Bovine	Bovine foamy virus
		Bovine immunodeficiency virus
		Bovine leukemia virus
	Feline (domestic cat)	Feline foamy virus
		Feline leukemia virus
		Feline immunodeficiency virus
	Feline (wild)	Feline foamy virus
		Puma lentivirus
	Horse	Equine foamy virus
		Equine infectious anemia virus
	Human	Human immunodeficiency virus
		Human T-lymphotropic virus
	Koala	Koala retrovirus
	Monkey	Cynomolgus macaque simian foamy virus
		Grivet simian foamy virus
		Japanese macaque simian foamy virus
		Langur virus
		Mason-Pfizer monkey virus
		Rhesus macaque simian foamy virus
		Spider monkey simian foamy virus
		Squirrel monkey retrovirus
		Squirrel monkey simian foamy virus
		Taiwanese macaque simian foamy virus
		Western chimpanzee simian foamy virus
		White-tufted-ear marmoset simian foamy virus
		Woolly monkey sarcoma virus
		Yellow-breasted capuchin simian foamy virus
	Murine	Mouse mammary tumor virus
		Finkel-Biskis-Jinkins murine sarcoma virus
		Harvey murine sarcoma virus
		Moloney murine sarcoma virus
		Murine leukemia virus
		Mouse mammary tumor virus
	Primate	Brown greater galago prosimian foamy virus
		Bornean orangutan simian foamy virus
		Central chimpanzee simian foamy virus
		Gibbon ape leukemia virus
		Primate T-lymphotropic virus
		Simian immunodeficiency virus
		Western chimpanzee simian foamy virus
		Western lowland gorilla simian foamy virus
	Goat	Caprine arthritis encephalitis virus
	Sheep	Caprine arthritis encephalitis virus
		Jaagsiekte sheep retrovirus
		Visna-maedi virus

(Fig. 1). Based on Repbase (www.girinst.org), a database of transposable sequences, including ERVs, the domestic dog genome presented a lower number of ERV elements across mammalian genomes. The phylogenetic tree was based on the study conducted by Vogel et al. (Vogel 2005). These results raised the hypothesis that the diminished quantity of ERVs in canids is attributable to the lower efficiency of retroviruses to infect and integrate into canid genomes due to restriction barriers. The endogenization capacity of canid retroviruses appears to be comparatively lower than that in other animal species. Previous studies have indicated that the canid genome contains a relatively lower quantity of endogenous retroviral elements compared to other species, including humans, mice, and opossum (Barrio et al. 2009; Blikstad et al. 2008).

Certain genomes must exhibit heightened vigilance in their antiretroviral defenses. Considering that canid genomes contain approximately five times fewer relatively complete proviruses than human and rodent genomes (Fig. 1), an effective defense seems to be successful. Interactions with defense mechanisms against foreign nucleic acids exert significant selective pressures that influence the evolution of retroviruses. Some retroviral restriction factors have been identified, such as TRIM5alpha, a host protein that ubiquitinates the incoming preintegration complex, leading to its degradation at the proteasome, thereby limiting retroviral infection in various hosts and cell types (Stremlau et al. 2004). This and other retroviral restriction factors pose obstacles that must be overcome by retroviruses. Interestingly, the viral restriction factor TRIM5alpha has been identified in primates, while in other mammals, its presence is either absent or lacks functionality (Sawyer et al. 2007; McEwan et al. 2009). Moreover, a multitude of other viral restriction factors exists. This study encompasses an evolutionary analysis of several viral replication restriction factors, including BST2, MOV10, SERINC1-5, SAMDH1, TRIM11, TRIM19, TRIM21, and TRIM22 (Fig. 2). Maximum likelihood phylogenetic trees were inferred using MEGA software (Kumar et al. 2018), employing the JTT matrix-based model (Jones et al. 1992). (Accession codes are available in the supplementary material). While all phylogenies revealed a consistent evolutionary pattern across species, one noteworthy finding emerged: TRIM22 is present in primates and canids but absent in

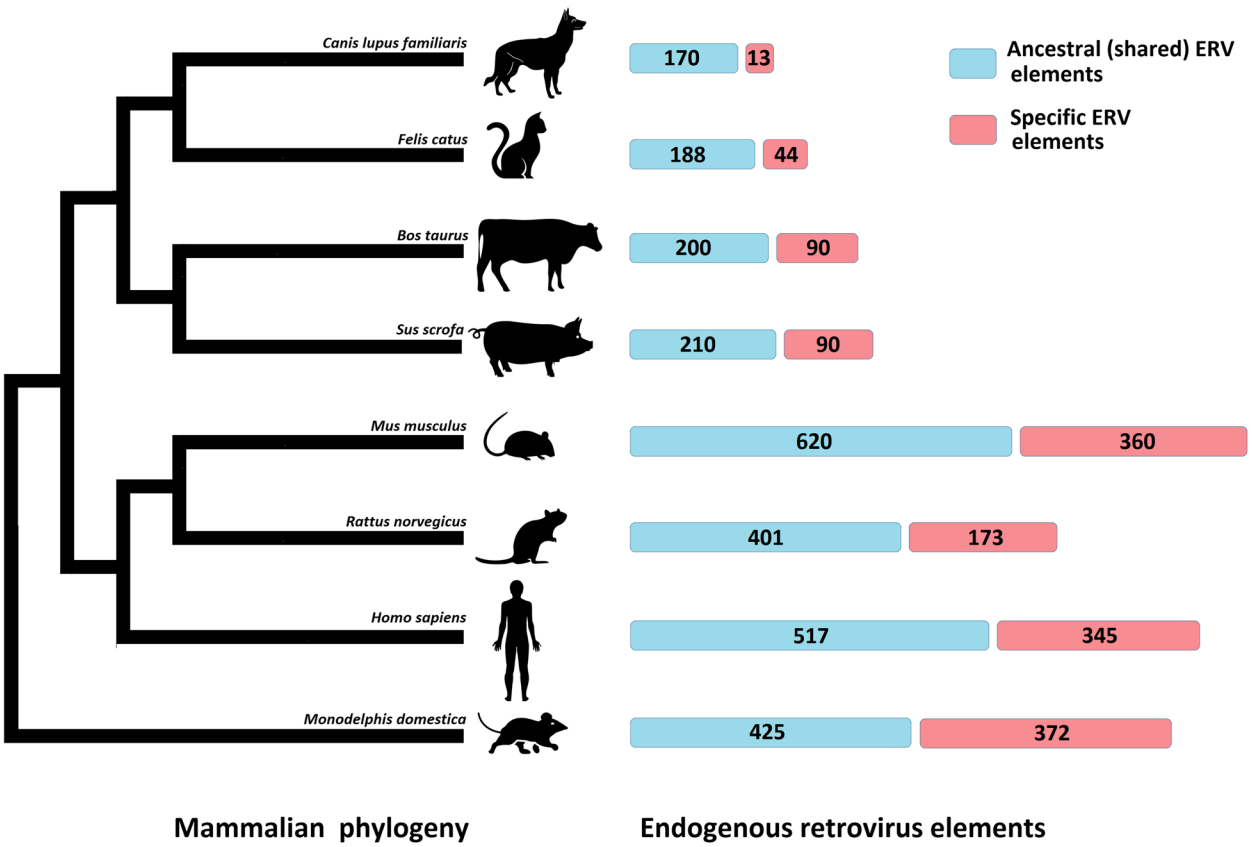

Fig. 1 Evolutionary perspective of endogenous retroviruses (ERVs) in mammals. The phylogenetic tree was constructed based on the study by Vogel et al. (Vogel 2005). The numbers of ERV elements were obtained from the Repbase database (www.girinst.org)

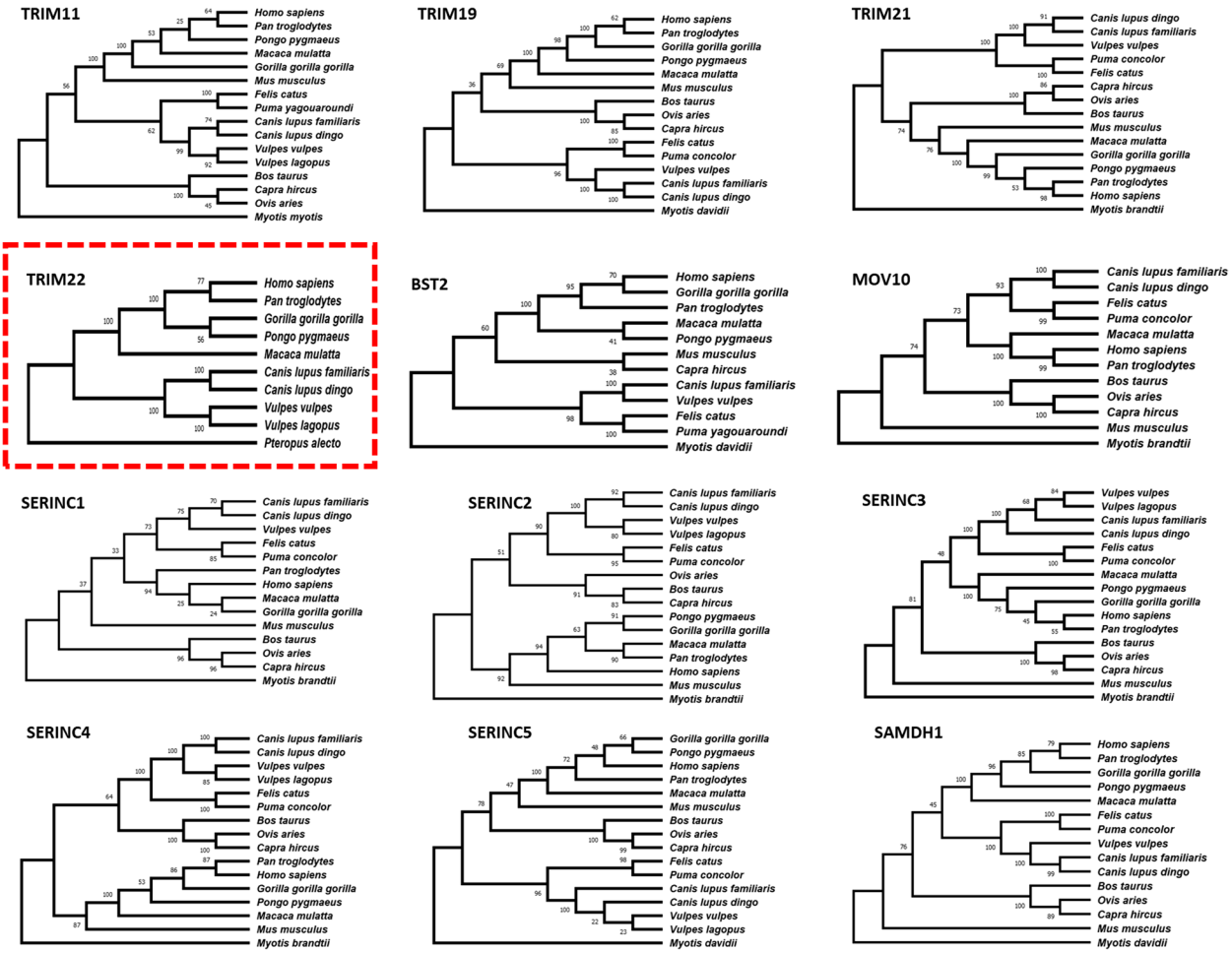

Fig. 2 Evolutionary analysis of viral restriction factors. Bats were adopted as the outgroup. Maximum likelihood phylogenetic trees were inferred using MEGA software, employing the JTT matrix-based model. Sequences were obtained from the NCBI protein database (https://www.ncbi.nlm.nih.gov/protein/). Accession numbers are available in the supplementary data

other mammals. TRIM22 plays a significant role in ubiquitinating retroviral proteins, leading to their degradation and thereby inhibiting the replication and assembly of new viral particles. Furthermore, TRIM22 possesses the capability to suppress the transcription of retroviruses, such as HIV-1 (Pagani et al. 2021).

It is indeed intriguing to acknowledge the resistance of canids to retrovirus infection, especially when considering the successful spillover of retroviruses into other mammals such as felines. These animals share habitats, coexist within the same landscapes, and even hunt the same prey. Consequently, they are likely to have encountered retroviruses carried by other species' bodily fluids on numerous occasions. However, attempts by retroviruses to infect dogs have been unsuccessful. The ability of dogs to resist retroviruses must be based not on a single factor alone but on a combination of characteristics that collectively make it challenging for retroviruses to successfully infect these hosts. In this brief study, we highlighted certain factors that could contribute to dogs' resistance against retroviruses. However, numerous aspects warrant consideration—epigenetic programming, the ability to express elements that hinder the establishment of retroviruses, or alterations in genetic patterns that encode receptors unfavorable for retroviral infection—such as the deletion of 32 amino acids from the CCR5 coreceptors used by HIV-1 (O'Brien and Moore 2000).

Dogs have been used as models for developing therapies and understanding molecular pathophysiology due to their clinical and genetic similarity to rare human diseases. The increasing interest in the genetics and genomics of dogs has paved the way for the development of new models for studying human diseases. However, the disparity in the number of ERV elements between canid and human genomes raises the possibility of an imbalanced

model. Humans possess approximately five times more ERVs than dogs (Fig. 1), and since ERVs can act as potent sources of genetic and transcriptional regulation disruption, this disparity may impact the comparative analysis (Buttler and Chuong 2022). Regulatory activities derived from ERVs have been associated with inflammatory diseases, cancer, and autoimmunity (Buttler and Chuong 2022; Singh 2007; Kassiotis 2014). When considering the adoption of dogs as a model to study these human diseases, it is important to take into account the potential lack of influence of ERVs. The decreased number of ERVs in canid genomes compared to humans may impact the regulatory landscape and should be considered in the analysis.

Despite the extensive search for retroviral particles among canids, specifically domestic dogs, there are few reports in the literature, most of the time showing negative outcomes. Taking these studies together, retroviral particles were not identified in dogs, as observed in other mammals, such as cats and humans. Thus, virological, immunological and genetic studies should be performed to assess why dogs have almost not been infected by retroviruses, in opposition to what happens with primates, felines and other mammals. An investigation able to detect retroviruses in different species of canids, such as wolves, foxes or other wild species, could elucidate whether the resistance of canids to exogenous retroviruses is restricted to domestic dogs or is broader. Finally, it seems that dogs and wild canids may have effectively protected their genomes from retroviral integration, resulting in resistance against exogenous retroviral particles and, consequently, a decreased number of endogenous retrovirus integrations.

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s44149-023-00097-5.

Additional file 1.

Code availability

Not applicable.

Authors' contributions

JC and LRL equally contributed to the conception of the study, evolutionary inferences, result analysis, and paper writing. LRL created the figures and tables. JC participated in the study coordination. JHC contributed to data curation, result analysis, and also reviewed the draft paper.

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Availability of data and materials

The data used to compare endogenous retrovirus elements were obtained from Repbase (www.girinst.org), a database of transposable sequences.

Declarations

Ethics approval and consent to participate

This research included secondary biological data with no possibility of individual identification. The Research Ethics Committee was consulted, and it was determined that this study did not require ethical approval.

Competing interests

The authors state that there are no conflicts of interest to declare.

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