Tech. Rep. Aust. Mus. Online
Number 38, pp. 3–6, 2023
https://doi.org/10.3853/j.1835-4211.38.2023.1831

Technical Reports of the Australian Museum Online
a peer-reviewed open-access journal
published by the Australian Museum, Sydney
communicating knowledge derived from our collections
ISSN 1835-4211 (online)

An Overview of Koala Retrovirus Epidemiology in Australia

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Abstract. Koala retrovirus (KoRV) epidemiology varies across koala (*Phascolarctos cinereus*) populations with distinct differences in viral prevalence, sequence diversity, and disease impact. Curiously the more genetically restricted southern populations are less impacted by KoRV with the virus not endogenized in its replication competent form in these animals. These southern animals do, however, have replication defective recKoRV variants in their genomes indicating historical exposure to KoRV and recKoRV. Whether southern animals are inherently resistant to KoRV infection and endogenization is not clear. It is also not clear whether the current regional epidemiological patterns will persist or whether exposure to animals with infectious KoRV or cross-breeding between different genetic populations will change the KoRV prevalence with time.

Introduction

Both koala (*Phascolarctos cinereus*) genetics and koala retrovirus (KoRV) prevalence vary regionally across Australia, with a stark demarcation between a more genetically diverse "northern" group (New South Wales and Queensland) and a genetically restricted "southern" group (Victoria and South Australia). These groups of animals also display markedly different disease profiles, with putatively KoRV-related disease syndromes at a much lower rate in the southern animals. All northern animals ever studied have endogenous KoRV-A alongside varying prevalence of other KoRV genotypes that do not appear to be endogenous. Endogenous KoRV loci are shared amongst closely related individuals but are not fixed across the species. Northern koalas also have multiple copies of defective KoRV variants in their genomes, where the central portion of the KoRV genome has been replaced by another koala retro-element termed Phascolarctid endogenous retroelement (PhER). These are known as recKoRVs and are also not fixed.

The southern animals were re-established from off-shore island colonies after localized extinction in the 1920's with a marked genetic bottleneck evident. Southern animals display varying KoRV prevalence without endogenous KoRV loci. Those animals that are KoRV positive tend to have lower viral loads than their northern counterparts. It was previously thought that many of these animals were KoRV free; however, recent work has demonstrated that many (perhaps all) animals that test negative for the KoRV *pol* gene PCR (the most used diagnostic for all KoRV variants) have recKoRV variants within their genomes. These are distinct from the recKoRV variants in the northern animals with an additional indel of unidentified DNA between the KoRV *gag* and PhER sequences. It is not clear at this stage what the significance of this is for potential to cause disease. It is possible that the presence of these variants inhibits infectious KoRV (as happens with defective endogenous retroviruses in other species). It is also possible that southern animals are simply not born tolerized to KoRV-A (or other variants) and are better able to control virus replication via their

Keywords: koala retrovirus, KoRV, Phascolarctid endogenous retroelement
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Submitted: 5 January 2021 **Accepted**: 5 May 2023 **Published**: 21 June 2023 (online only)
Publisher: The Australian Museum, Sydney, Australia (a statutory authority of, and principally funded by, the NSW State Government)
Citation: Tarlinton, Rachael E. 2023. An overview of koala retrovirus epidemiology in Australia. In *Proceedings of the Second Koala Retrovirus Workshop*, ed. D. E. Alquezar-Planas, D. P. Higgins, C. L. Singleton, and A. D. Greenwood. *Technical Reports of the Australian Museum Online* 38: 3–6. https://doi.org/10.3853/j.1835-4211.38.2023.1831