

Cancer is a ubiquitous disease of mammals, study concludes

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Cancer is often considered as a human disease. While it is true that cancer seriously affects humans, other animals also experience this disease. Not just pets, but wild animals too.

Nonetheless, the what extent to which [animals](#) are exposed to cancers and how often their health is affected by it has been little known so far.

This is not a surprise, as in [wild animals](#), any serious illness will likely result in the untraceable death of the animal due to starvation or predation. Moreover, cancer is an age-associated [disease](#), where old individuals are more prone to develop the disease.

Consequently, the [risk](#) of cancer in wild animals, where age is rarely known is difficult to estimate. Therefore, in order to explore how often do animals face cancer, this research focused on zoo animals, where individuals are followed throughout their lives.

Using data on 191 species and 110,148 individual mammals, this research now demonstrates that cancer is a ubiquitous disease of mammals and it can emerge anywhere along the mammalian phylogeny.

The research also highlights that cancer risk is not uniformly distributed along the phylogeny of mammals. For instance, Carnivorans are particularly prone to be affected by cancer (e.g. over 25% of Clouded leopards, Bat-eared foxes and Red wolfs die of cancer), while ungulates appear consistently highly resistant to this disease.

This research also tried to explore whether diet differences explain the observed phylogenetic pattern in cancer risk. The results indicate that consuming animals, especially mammalian prey, indeed increases cancer risk across mammals. The authors argue that high cancer risk in carnivorous mammals might be related to their low microbiome diversity, limited physical exercise under human care, oncogenic viral infections or to other physiological aspects of carnivorous mammals.

The study also addresses a fascinating evolutionary question. Tumors are diseases of mutational origin, and mutations usually arise during cell division. Consequently, animals with larger bodies and longer lifespans that have undergone more cell division are likely to have a higher risk of developing tumors.

This theory has been supported by several studies in humans, for example, where greater body size (height) is associated with a higher risk of cancer. However, these correlations do not seem to hold across species, as an elephant and a mouse have a similar likelihood of developing cancer, even though their lifespans and body sizes differs by orders of magnitude. The discrepancy in the effect of body size and life expectancy on cancer risk is known as the Peto's paradox.

This research now provides proof that cancer risk is largely independent of body mass and life expectancy across mammals, providing thus unequivocal evidence for the validity of Peto's paradox. This result provides support for the claim that evolution has fashioned more and more efficient tumor suppressor mechanisms along the evolution of extended longevities and/or larger body sizes.

"Overall our work highlights that cancer might represent a serious and significant threat to [animal welfare](#), that need considerable scientific attention, especially in the context of recent environmental changes caused by humans," says co-author Fernando Colchero, University of Southern Denmark.

"Moreover, a better understanding of [cancer risk](#) and resistance in various animal species can provide major advancements in the quest for natural anticancer defenses and could revolutionize cancer medicine. For instance, studying species that are highly resistant to cancer, can provide basic information for the development of bio-mimetic natural cancer treatments, which, contrary to most [cancer](#) treatments are non-toxic to the host."

The study, "Cancer risk across mammals," is published in *Nature*.

More information: Orsolya Vincze, Cancer risk across mammals, *Nature* (2021). [DOI: 10.1038/s41586-021-04224-5](https://doi.org/10.1038/s41586-021-04224-5).

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