HAL open science

On the need for integrating cancer into the One Health perspective

Antoine M Dujon, Joel S. Brown, Delphine Destoumieux-Garzon, Marion Vittecoq, Rodrigo Hamede, Aurélie Tasiemski, Justine Boutry, Sophie Tissot, Catherine Alix-Panabières, Pascal Pujol, et al.

To cite this version:

Antoine M Dujon, Joel S. Brown, Delphine Destoumieux-Garzon, Marion Vittecoq, Rodrigo Hamede, et al.. On the need for integrating cancer into the One Health perspective. Evolutionary Applications, 2021, 14 (11), pp.2571-2575. 10.1111/eva.13303 . hal-03372780

HAL Id: hal-03372780

https://hal.science/hal-03372780v1

Submitted on 11 Oct 2021

HAL is a multi-disciplinary open access archive for the deposit and dissemination of scientific research documents, whether they are published or not. The documents may come from teaching and research institutions in France or abroad, or from public or private research centers.

L'archive ouverte pluridisciplinaire **HAL**, est destinée au dépôt et à la diffusion de documents scientifiques de niveau recherche, publiés ou non, émanant des établissements d'enseignement et de recherche français ou étrangers, des laboratoires publics ou privés.

Distributed under a Creative Commons Attribution 4.0 International License

Check for updates

Received: 3 September 2021 | Revised: 10 September 2021 | Accepted: 12 September 2021

DOI: 10.1111/eva.13303

PERSPECTIVE

Evolutionary Applications Open Access WILEY

On the need for integrating cancer into the One Health perspective

Summary

Recent pandemics have highlighted the urgency to connect disciplines studying animal, human, and environment health, that is, the "One Health" concept. The One Health approach takes a holistic view of health, but it has largely focused on zoonotic diseases while not addressing oncogenic processes. We argue that cancers should be an additional key focus in the One Health approach based on three factors that add to the well-documented impact of humans on the natural environment and its implications on cancer emergence. First, human activities are oncogenic to other animals, exacerbating the dynamics of oncogenesis, causing immunosuppressive disorders in wildlife with effects on host–pathogen interactions, and eventually facilitating pathogen spillovers. Second, the emergence of transmissible cancers in animal species (including humans) has the potential to accelerate biodiversity loss across ecosystems and to become pandemic. It is crucial to understand why, how, and when transmissible cancers emerge and spread. Third, translating knowledge of tumor suppressor mechanisms found across the Animal Kingdom to human health offers novel insights into cancer prevention and treatment strategies.

1 | INTRODUCTION

The "One World–One Health" concept, initiated in 2004, recognizes the close connections between health of humans and that of other animals and our shared environment. The importance of this concept is evidenced by the accelerated emergence or re-emergence of zoonoses (e.g., Ebola, current COVID-19 pandemic) over the last decades (Peyre et al., 2021). Although the One Health approach proposes a holistic view of health, it has, and still remains, largely focused on zoonotic and vector-borne diseases. Reasons behind this bias are multiple, ranging from the fact that historically it has originated from a collaboration between human and veterinary medicines, and also because zoonotic diseases undoubtedly represent major health and economic threats to human societies. The scope has broadened with the inclusion of other fields, such as antimicrobial resistance (WHO, 2015), (eco)toxicology, and health in urban environments (Destoumieux-Garzón et al., 2018), but not cancer.

The impact of human activities on the natural environment and their implications on cancer emergence in humans have been largely documented (e.g., anthropogenic pollution, occupation, food, and lifestyle; Aktipis & Nesse, 2013) (Briffa et al., 2020; Erren et al., 2015; Turner et al., 2020). These effects and their consequences could increase in the context of climate change (Hiatt & Beyeler, 2020). Beyond this acknowledged link between the environment and cancer, we discuss here three reasons for a broader inclusion of cancer into the One Health concept.

First, the interplay/interactions between cancer and infectious diseases make arduous to separate these communicable and non-communicable diseases, and their combination could pose higher risk for human and animal health. For instance, cancers can cause immunosuppression 2020); cancers can also be induced by pathogens (Ewald & Swain Ewald, 2015; Jacqueline et al., 2017). However, little attention has been paid to the role of human-induced cancers in infectious diseases' emergence and spread. Human activities elevate cancer incidences for wildlife species (via chemical pollution, changes in diet, and reductions in genetic diversity; Baines et al., 2021; Giraudeau et al., 2018; Sepp et al., 2019; Ujvari et al., 2018). As in humans, cancer burdens generate a range of immunosuppressive disorders in livestock, and likely in wildlife (Pollock & Roth, 1989; Vittecoq et al., 2013). Animals with cancer, human or otherwise, may be more susceptible, contagious, and debilitated by infectious diseases. Therefore, the evolutionary mismatch between enhanced human-induced cancer risks and maladapted cancer defense levels in animals will indirectly amplify pathogen dynamics in wildlife, in both natural and human-dominated ecosystems. In undisturbed natural environments, a fraction of pathogens is generally maintained by the presence of immunocompromised individuals, a stable equilibrium, that has been reached over evolutionary time (Hassell et al., 2021). By accelerating cancer incidence and the dynamics of oncogenesis,

This is an open access article under the terms of the Creative Commons Attribution License, which permits use, distribution and reproduction in any medium, provided the original work is properly cited.
© 2021 The Authors. *Evolutionary Applications* published by John Wiley & Sons Ltd

anthropogenic activities can disrupt these equilibria. These altered dynamics are complex and not merely correlated with pollution levels, but also driven by, for example, the different susceptibilities of exposed species to carcinogenesis. For instance, a benthic lifestyle within fish communities contributes to elevated cancer incidence through chronic exposure to contaminated sediment and/or consumption of contaminated invertebrates (Black & Baumann, 1991; Martineau et al., 2002). The consequences of these disturbances are difficult to predict given the cascade of indirect effects that may subsequently occur. For example, the impact of higher cancer incidences and carcinogens will depend on the species most affected by cancer (e.g., predators or prey species) and the resulting immunosuppressive disorders (see, for instance, Perret et al., 2020). The relationship between an animal's immunosuppression and cancer burden (e.g., linear, exponential, negative binomial, and with or without critical thresholds) may also vary with species and/or with the organ in which malignancies develop. Clearly, the roles of pollution-induced oncogenesis and immunosuppression in boosting pathogen communities in wildlife and their spread to humans are poorly understood at the moment. Given that parasitism and disease spread may also be limited in ecological communities via mechanisms such as the dilution effect (Civitello et al., 2015), pollution-induced oncogenesis could amplify these transfers if it concomitantly promotes a decline in biodiversity.

A second reason for elevating the importance of cancers in the One Health approach concerns cancers that can themselves become transmissible. There are currently nine known transmissible cancer lineages (one in dogs, two in Tasmanian devil populations, and six in marine bivalves; Dujon, Bramwell, et al., 2020; Dujon, Gatenby, et al., 2020), some of which threaten the survival of animal populations (and species) and are detrimental to ecosystem stability (see, for instance, Hamede et al., 2020). Indubitably, this number is a gross underestimate due to poor monitoring of cancer in wildlife (Dujon, Bramwell, et al., 2020; Dujon, Gatenby, et al., 2020; Dujon, Ujvari, et al., 2020; Ujvari et al., 2016). The conditions for the emergence of transmissible cancers, while poorly understood, require a perfect storm of sequential steps that is reminiscent of a parasitic lifestyle. First, the tumor must shed a large number of cells that are able to survive outside of or on the surface of the original host. Those cells, when in contact with a new host, must then evade immune recognition (particularly challenging in vertebrates) and successfully proliferate in the correct tissue of the new host (Ujvari et al., 2017). Environmental stress, lack of genetic diversity within the host population, and other infectious diseases may weaken the host's immune system enabling transmission and establishment of cancer cells from one individual to another (Dujon, Bramwell, et al., 2020; Dujon, Gatenby, et al., 2020; Dujon, Ujvari, et al., 2020). Human activities may also disperse certain transmissible cancers across the globe, giving previously isolated outbreaks pandemic potentials. Already, a single transmissible cancer lineage has been observed in multiple marine mussel species across multiple oceans, suggesting dispersal by aquaculture and/or shipping (Bramwell et al., 2021; Skazina et al., 2021; Yonemitsu et al., 2019). A full understanding of the ecological impacts of transmissible cancers on ecosystems and biodiversity will require integrating tools from landscape ecology, conservation biology, and epidemiology to identify wildlife species at risk of contagious malignant cell lines, detect their emergence, anticipate their spread, and evaluate consequences to natural and human-dominated ecosystems (Bramwell et al., 2021; Dujon et al., 2020; Dujon, Gatenby, et al., 2020; Dujon, Ujvari, et al., 2020). A worst-case scenario might involve the emergence of a communicable cancer in humans or in livestock, yielding environmental and human/animal health crises similar to classical epidemics and epizootics.

Finally, a third reason to consider cancer in the One Health approach lies in the scientific insights provided by comparative oncology focusing on the diverse tumor suppression mechanisms that have evolved across the animal kingdom (Nunney et al., 2015; Schiffman & Breen, 2015). Cancers appeared during the transition from unicellular to metazoan life, approximately one billion years ago (Domazet-Lošo & Tautz, 2010). Cancers occur in all branches of multicellular life, and they have evolved diverse adaptations to prevent cancer initiation, suppress malignant progression, and alleviate negative fitness consequences of tumor burdens (Aktipis et al., 2015). For instance, one might expect large/long-lived animals to have more cancers than smaller/shorter-lived ones, given that the former have many more cell divisions that potential give rise to cancer-initiating mutations, but such a relationship across species is not observed, due to the various mechanisms of cancer resistance that have evolved in large and long-lived species (i.e., Peto's Paradox, Tollis et al., 2017). Even in smaller species, under particular ecological conditions, selection can favor high levels of cancer resistance, as observed in naked-mole rats and bats (Lambert & Portfors, 2017; Seluanov et al., 2018; Tian et al., 2013). Scientists are increasingly focusing on the ways natural selection has protected animal species from cancer (Boutry et al., 2020). This work is deciphering the underlying tissue, cellular, and genomic mechanisms in cancer-resistant/tolerant species (Abegglen et al., 2015; Keane et al., 2015; Sulak et al., 2016). Another particularly interesting direction of comparative oncology investigates the local evolution of adaptations in species or populations that have been living in environments with excesses of naturally occurring carcinogens (Hourdez et al., 2021; Vittecoq et al., 2018). To what extent do these populations show adaptations to prevent the emergence of cancer versus adaptations to mitigate the negative effects of cancer burdens? To what extent do these populations' anti-cancer adaptations increase or decrease susceptibility to infectious diseases? Of particular interest would be adaptations that simultaneously reduce cancer and infections. Studying species and ecological communities chronically exposed to carcinogens, with a particular focus on host–pathogen dynamics, could shed light on the possible impacts and repercussions of human-induced cancers within wildlife species (Vittecoq et al., 2018). From a medical perspective, the adaptations of long-lived mammals and other species for preventing and suppressing cancer should inspire nature-based solutions to cancer, such as novel cancer treatment strategies, by mimicking the processes allowing these organisms to prevent or limit malignant progression despite high levels of mutagenic substances.

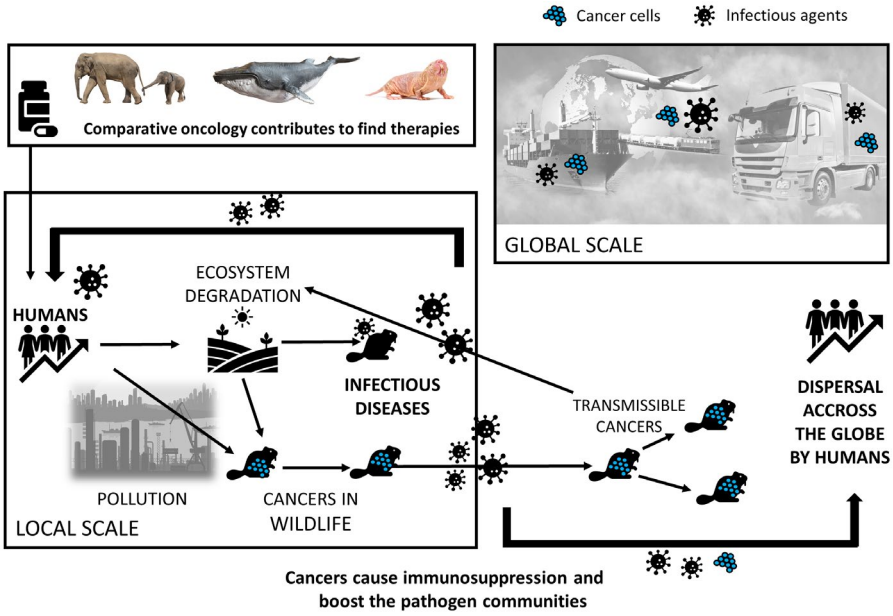

FIGURE 1 Cancer in the One Health perspective: comparative oncology, interplay between human activities, novel cancer risks experienced by wildlife species, and infectious disease dynamics

Thus, comparative oncology offers key insights into cancer epidemiology, prevention, and novel therapies for humans.

In conclusion, humans, domestic animals, and wildlife species are on the front lines of environmental changes and exposure to toxic hazards from anthropogenic activities. While it is well recognized that these processes directly affect immunity and hence pathogen dynamics (e.g., Dittmar et al., 2014; Tracy et al., 2020), much less is known concerning the indirect links resulting from interactions involving non-communicable diseases, such as cancer. The direct and indirect effects do not necessarily yield the same outcomes. Cancer-causing or cancer-associated processes are undoubtedly an under-appreciated health consequence of modern anthropogenic changes, and they cannot be considered in isolation from other biological players in ecosystems, especially pathogens. Ecological impacts, sooner or later, are usually followed by evolutionary responses, leading to novel equilibria in ecosystems. However, we currently ignore how the novel equilibria will be achieved and shaped in an increasingly stressful world. A particular question remaining to be answered is how pollution and consequent oncogenic processes will impact infectious diseases and thus human health particularly before reaching a novel equilibrium. Exploring the interplay between human activities, novel cancer risks experienced by wildlife species, cancer defense evolution, biotic interactions, and infectious disease dynamics, notably those transmissible to humans, should become a stronger topic within the One Health perspective (Figure 1). Because one single method or model cannot thoroughly describe these complex dynamics, more than ever researchers must engage in greater exchanges and collaborations involving different disciplines, such as field and experimental ecology, epidemiology, eco-toxicology, immunology, evolutionary biology, oncology, and pharmacy.

DATA AVAILABILITY STATEMENT

Data sharing is not applicable to this article as no new data were created or analyzed in this study.

KEYWORDS

cancer, comparative oncology, infectious diseases, One Health, Peto's paradox

FUNDING INFORMATION

This work is supported by the MAVA Foundation, an ANR TRANSCAN (ANR-18-CE35-0009), an ARC Linkage (LP170101105), Deakin SEBE_RGS_2019, a CNRS International Associated Laboratory Grant, and the French Laboratory of Excellence project "TULIP" (ANR-10-LABX-41; ANR-11-IDEX-0002-02).

Antoine M. Dujon[1,2,3]
Joel S. Brown[4]
Delphine Destoumieux-Garzón[5]
Marion Vittecoq[1,2,6]
Rodrigo Hamede[7]
Aurélie Tasiemski[8]
Justine Boutry[1,2]
Sophie Tissot[1,2]
Catherine Alix-Panabieres[1,2,9]
Pascal Pujol[1,2,10]
François Renaud[1,2]
Frédéric Simard[2]
Benjamin Roche[1,2]
Beata Ujvari[3]
Frédéric Thomas[1,2]

[1]CREEC/CANECEV (CREES), Montpellier, France
[2]MIVEGEC, Université de Montpellier, CNRS, IRD, Montpellier, France
[3]School of Life and Environmental Sciences, Centre for Integrative Ecology, Deakin University, Waurn Ponds, Vic., Australia
[4]Department of Integrated Mathematical Oncology, Moffitt Cancer Center, Tampa, Florida, USA
[5]IHPE, Université de Montpellier, CNRS, Ifremer, Université de Perpignan Via Domitia, Montpellier, France
[6]Tour du Valat, Research Institute for the Conservation of Mediterranean Wetlands, Arles, France
[7]School of Natural Sciences, University of Tasmania, Hobart, Tas., Australia
[8]Univ. Lille, CNRS, Inserm, CHU Lille, Institut Pasteur de Lille, U1019-UMR9017-CIIL-Centre d'Infection et d'Immunité de Lille, Lille, France
[9]Laboratory of Rare Human Circulating Cells (LCCRH), University Medical Centre of Montpellier, Montpellier, France
[10]Oncogenetic Department, University Medical Centre of Montpellier, Montpellier, France

Correspondence
Frederic Thomas, CREEC/CANECEV (CREES), Montpellier, France.
Email: frederic.thomas2@ird.fr

Brown, Destoumieux-Garzon, Ujvari and Thomas equal contribution.

ORCID

Antoine M. Dujon https://orcid.org/0000-0002-1579-9156
Delphine Destoumieux-Garzón https://orcid.org/0000-0002-8793-9138
Marion Vittecoq https://orcid.org/0000-0002-8930-6051
Catherine Alix-Panabieres https://orcid.org/0000-0002-6401-2903
Beata Ujvari https://orcid.org/0000-0003-2391-2988
Frédéric Thomas https://orcid.org/0000-0003-2238-1978

REFERENCES

Abegglen, L. M., Caulin, A. F., Chan, A., Lee, K., Robinson, R., Campbell, M. S., Kiso, W. K., Schmitt, D. L., Waddell, P. J., Bhaskara, S., Jensen, S. T., Maley, C. C., & Schiffman, J. D. (2015). Potential mechanisms for cancer resistance in elephants and comparative cellular response to DNA damage in humans. *JAMA – Journal of the American Medical Association*, *314*, 1850. https://doi.org/10.1001/jama.2015.13134

Aktipis, C. A., Boddy, A. M., Jansen, G., Hibner, U., Hochberg, M. E., Maley, C. C., & Wilkinson, G. S. (2015). Cancer across the tree of life: Cooperation and cheating in multicellularity. *Philosophical Transactions of the Royal Society B: Biological Sciences*, *370*(1673), 20140219. https://doi.org/10.1098/rstb.2014.0219

Aktipis, C. A., & Nesse, R. M. (2013). Evolutionary foundations for cancer biology. *Evolutionary Applications*, *6*, 144–159. https://doi.org/10.1111/eva.12034

Baines, C., Lerebours, A., Thomas, F., Fort, J., Kreitsberg, R., Gentes, S., Meitern, R., Saks, L., Ujvari, B., Giraudeau, M., & Sepp, T. (2021). Linking pollution and cancer in aquatic environments: A review. *Environment International*, *149*, 106391. https://doi.org/10.1016/j.envint.2021.106391

Black, J. J., & Baumann, P. C. (1991). Carcinogens and cancers in freshwater fishes. *Environmental Health Perspectives*, *90*, 27–33. https://doi.org/10.1289/ehp.90-1519473

Boutry, J., Dujon, A. M., Gerard, A.-L., Tissot, S., Macdonald, N., Schultz, A., Biro, P. A., Beckmann, C., Hamede, R., Hamilton, D. G., Giraudeau, M., Ujvari, B., & Thomas, F. (2020). Ecological and evolutionary consequences of anticancer adaptations. *Iscience*, *23*(11), 101716. https://doi.org/10.1016/j.isci.2020.101716

Bramwell, G., Schultz, A. G., Sherman, C. D. H., Giraudeau, M., Thomas, F., Ujvari, B., & Dujon, A. M. (2021). A review of the potential effects of climate change on disseminated neoplasia with an emphasis on efficient detection in marine bivalve populations. *Science of the Total Environment*, *775*, 145134. https://doi.org/10.1016/j.scitotenv.2021.145134

Briffa, J., Sinagra, E., & Blundell, R. (2020). Heavy metal pollution in the environment and their toxicological effects on humans. *Heliyon*, *6*, e04691. https://doi.org/10.1016/j.heliyon.2020.e04691

Civitello, D. J., Cohen, J., Fatima, H., Halstead, N. T., Liriano, J., McMahon, T. A., Ortega, C. N., Sauer, E. L., Sehgal, T., Young, S., & Rohr, J. R. (2015). Biodiversity inhibits parasites: Broad evidence for the dilution effect. *Proceedings of the National Academy of Sciences*, *112*(28), 8667–8671. https://doi.org/10.1073/pnas.1506279112

Destoumieux-Garzón, D., Mavingui, P., Boetsch, G., Boissier, J., Darriet, F., Duboz, P., Fritsch, C., Giraudoux, P., Le Roux, F., Morand, S.., Paillard, C., Pontier, D., Sueur, C., & Voituron, Y. (2018). The one health concept: 10 Years old and a long road ahead. *Frontiers in Veterinary Science*, *5*, http://dx.doi.org/10.3389/fvets.2018.00014

Dittmar, J., Janssen, H., Kuske, A., Kurtz, J., & Scharsack, J. P. (2014). Heat and immunity: An experimental heat wave alters immune functions in three-spined sticklebacks (*Gasterosteus aculeatus*). *Journal of Animal Ecology*, *83*(4), 744–757. https://doi.org/10.1111/1365-2656.12175

Domazet-Lošo, T., & Tautz, D. (2010). Phylostratigraphic tracking of cancer genes suggests a link to the emergence of multicellularity in metazoa. *BMC Biology*, *8*(6), 1–10. https://doi.org/10.1186/1741-7007-8-66

Dujon, A. M., Bramwell, G., Roche, B., Thomas, F., & Ujvari, B. (2020). Transmissible cancers in mammals and bivalves: How many examples are there? *BioEssays*, *43*, 2000222. https://doi.org/10.1002/bies.202000222

Dujon, A. M., Gatenby, R. A., Bramwell, G., MacDonald, N., Dohrmann, E., Raven, N., Schultz, A., Hamede, R., Gérard, A.-L., Giraudeau, M., Thomas, F., & Ujvari, B. (2020). Transmissible cancers in an evolutionary perspective. *Iscience*, *23*(7), 101269. https://doi.org/10.1016/j.isci.2020.101269

Dujon, A. M., Ujvari, B., & Thomas, F. (2020). Cancer risk landscapes: A framework to study cancer in ecosystems. *Science of the Total Environment*, *763*, 142955.

Erren, T. C., Groß, J. V., Steffany, F., & Meyer-Rochow, V. B. (2015). "Plastic ocean": What about cancer? *Environmental Pollution*, *207*, 436–437. https://doi.org/10.1016/j.envpol.2015.05.025

Ewald, P. W., & Swain Ewald, H. A. (2015). Infection and cancer in multicellular organisms. *Philosophical Transactions of the Royal Society B: Biological Sciences*, *370*(1673), 20140224. https://doi.org/10.1098/rstb.2014.0224

Giraudeau, M., Sepp, T., Ujvari, B., Ewald, P. W., & Thomas, F. (2018). Human activities might influence oncogenic processes in wild animal populations. *Nature Ecology and Evolution*, *2*, 1065–1070. https://doi.org/10.1038/s41559-018-0558-7

Hamede, R., Owen, R., Siddle, H., Peck, S., Jones, M., Dujon, A. M., Giraudeau, M., Roche, B., Ujvari, B., & Thomas, F. (2020). The ecology and evolution of wildlife cancers: Applications for management

and conservation. *Evolutionary Applications*, *13*, 1719–1732. https://doi.org/10.1111/eva.12948

Hassell, J. M., Newbold, T., Dobson, A. P., Linton, Y. M., Franklinos, L. H. V., Zimmerman, D., & Pagenkopp Lohan, K. M. (2021). Towards an ecosystem model of infectious disease. *Nature Ecology and Evolution*, *5*, 907–918. https://doi.org/10.1038/s41559-021-01454-8

Hiatt, R. A., & Beyeler, N. (2020). Cancer and climate change. *Lancet Oncology*, *21*(11), e519–27.

Hourdez, S., Boidin-Wichlacz, C., Jollivet, D., Massol, F., Rayol, M. C., Bruno, R., Zeppilli, D., Thomas, F., Lesven, L., Billon, G., Duperron, S., & Tasiemski, A. (2021). Investigation of Capitella spp. symbionts in the context of varying anthropic pressures: First occurrence of a transient advantageous epibiosis with the giant bacteria Thiomargarita sp. to survive seasonal increases of sulfides in sediments. *Science of the Total Environment*, *798*, 149149. https://doi.org/10.1016/j.scitotenv.2021.149149

Jacqueline, C., Tasiemski, A., Sorci, G., Ujvari, B., Maachi, F., Missé, D., Renaud, F., Ewald, P., Thomas, F., & Roche, B. (2017). Infections and cancer: The "fifty shades of immunity" hypothesis. *BMC Cancer*, *17*, 257. https://doi.org/10.1186/s12885-017-3234-4

Kareva, I. (2020). Immune suppression in pregnancy and cancer: Parallels and insights. *Translational Oncology*, *13*, 100759. https://doi.org/10.1016/j.tranon.2020.100759

Keane, M., Semeiks, J., Webb, A. E., Li, Y. I., Quesada, V., Craig, T., Madsen, L. B., van Dam, S., Brawand, D., Marques, P. I., Michalak, P., Kang, L., Bhak, J., Yim, H.-S., Grishin, N. V., Nielsen, N. H., Heide-Jørgensen, M. P., Oziolor, E. M., Matson, C. W., … de Magalhães, J. P. (2015). Insights into the evolution of longevity from the bowhead whale genome. *Cell Reports*, *10*, 112–122. https://doi.org/10.1016/j.celrep.2014.12.008

Lambert, M. J., & Portfors, C. V. (2017). Adaptive sequence convergence of the tumor suppressor ADAMTS9 between small-bodied mammals displaying exceptional longevity. *Aging*, *9*(2), 573–582. https://doi.org/10.18632/aging.101180

Martineau, D., Lemberger, K., Dallaire, A., Labelle, P., Lipscomb, T. P., Michel, P., & Mikaelian, I. (2002). Cancer in wildlife, a case study: Beluga from the St. Lawrence estuary, Québec, Canada. *Environmental Health Perspectives*, *110*, 285–292. https://doi.org/10.1289/ehp.02110285

Nunney, L., Maley, C. C., Breen, M., Hochberg, M. E., & Schiffman, J. D. (2015). Peto's paradox and the promise of comparative oncology. *Philosophical Transactions of the Royal Society B*, *370*(1673), 20140177. https://doi.org/10.1098/rstb.2014.0177

Perret, C., Gidoin, C., Ujvari, B., Thomas, F., & Roche, B. (2020). Predation shapes the impact of cancer on population dynamics and the evolution of cancer resistance. *Evolutionary Applications*, *13*, 1733–1744.

Peyre, M., Vourc'h, G., Lefrançois, T., Martin-Prevel, Y., Soussana, J.-F., & Roche, B. (2021). PREZODE: Preventing zoonotic disease emergence. *The Lancet*, *397*, 792–793. https://doi.org/10.1016/S0140-6736(21)00265-8

Pollock, R. E. (1989). Cancer-induced immunosuppression: Implications for therapy? In *Seminars in surgical oncology*, vol. *5*(6), (pp. 414–419). John Wiley & Sons, Inc. https://doi.org/10.1002/ssu.2980050607

Schiffman, J. D., & Breen, M. (2015). Comparative oncology: What dogs and other species can teach us about humans with cancer. *Philosophical Transactions of the Royal Society B: Biological Sciences*, *370*, 20140231. https://doi.org/10.1098/rstb.2014.0231

Seluanov, A., Gladyshev, V. N., Vijg, J., & Gorbunova, V. (2018). Mechanisms of cancer resistance in long-lived mammals. *Nature Reviews Cancer*, *18*, 433–441. https://doi.org/10.1038/s41568-018-0004-9

Sepp, T., Ujvari, B., Ewald, P. W., Thomas, F., & Giraudeau, M. (2019). Urban environment and cancer in wildlife: Available evidence and future research avenues. *Proceedings of the Royal Society B: Biological Sciences*, *286*, 20182434.

Skazina, M., Odintsova, N., Maiorova, M., Ivanova, A., Väinölä, R., & Strelkov, P. (2021). First description of a widespread *Mytilus trossulus*-derived bivalve transmissible cancer lineage in *M. trossulus* itself. *Scientific Reports*, *11*(1), 5809. https://doi.org/10.1038/s41598-021-85098-5

Sulak, M., Fong, L., Mika, K., Chigurupati, S., Yon, L., Mongan, N. P., Emes, R. D., & Lynch, V. J. (2016). TP53 copy number expansion is associated with the evolution of increased body size and an enhanced DNA damage response in elephants. *elife*, *5*, e11994. https://doi.org/10.7554/eLife.11994

Tian, X., Azpurua, J., Hine, C., Vaidya, A., Myakishev-Rempel, M., Ablaeva, J., & Seluanov, A. (2013). High-molecular-mass hyaluronan mediates the cancer resistance of the naked mole rat. *Nature*, *499*(7458), 346–349.

Tollis, M., Boddy, A. M., & Maley, C. C. (2017). Peto's Paradox: How has evolution solved the problem of cancer prevention. *BMC Biology*, *15*(1), 1–5. https://doi.org/10.1186/s12915-017-0401-7

Tracy, A. M., Weil, E., & Harvell, C. D. (2020). Warming and pollutants interact to modulate octocoral immunity and shape disease outcomes. *Ecological Applications*, *30*(2), e02024. https://doi.org/10.1002/eap.2024

Turner, M. C., Andersen, Z. J., Baccarelli, A., Diver, W. R., Gapstur, S. M., Pope, C. A., Prada, D., Samet, J., Thurston, G., & Cohen, A. (2020). Outdoor air pollution and cancer: An overview of the current evidence and public health recommendations. *CA: A Cancer Journal for Clinicians*, *70*(6), 460–479. https://doi.org/10.3322/caac.21632

Ujvari, B., Gatenby, R. A., & Thomas, F. (2016). Transmissible cancers, are they more common than thought? *Evolutionary Applications*, *9*(5), 633–634. https://doi.org/10.1111/eva.12372

Ujvari, B., Gatenby, R., & Thomas, F. (2017). Transmissible cancer: The evolution of interindividual metastasis. In B. Ujvari, B. Roche, & F. Thomas (Eds.), *Ecology and evolution of cancer* (pp. 167–179). Elsevier.

Ujvari, B., Klaassen, M., Raven, N., Russell, T., Vittecoq, M., Hamede, R., Thomas, F., & Madsen, T. (2018). Genetic diversity, inbreeding and cancer. *Proceedings of the Royal Society B: Biological Sciences*, *285*, 20172589.

Vittecoq, M., Giraudeau, M., Sepp, T., Marcogliese, D. J., Klaassen, M., Renaud, F., Ujvari, B., & Thomas, F. (2018). Turning natural adaptations to oncogenic factors into an ally in the war against cancer. *Evolutionary Applications*, *11*, 836–844. https://doi.org/10.1111/eva.12608

Vittecoq, M., Roche, B., Daoust, S. P., Ducasse, H., Missé, D., Abadie, J., Labrut, S., Renaud, F., Gauthier-Clerc, M., & Thomas, F. (2013). Cancer: A missing link in ecosystem functioning? *Trends in Ecology and Evolution*, *28*(11), 628–635. https://doi.org/10.1016/j.tree.2013.07.005

WHO (2015). *Global action plan on antimicrobial resistance* (pp. 1–19). World Health Organization (WHO).

Yonemitsu, M. A., Giersch, R. M., Polo-Prieto, M., Hammel, M., Simon, A., Cremonte, F., Avilés, F. T., Merino-Véliz, N., Burioli, E. A. V., Muttray, A. F., Sherry, J., Reinisch, C., Baldwin, S. A., Goff, S. P., Houssin, M., Arriagada, G., Vázquez, N., Bierne, N., & Metzger, M. J. (2019). A single clonal lineage of transmissible cancer identified in two marine mussel species in South America and Europe. *eLife*, *8*, http://dx.doi.org/10.7554/elife.47788

How to cite this article: Dujon, A., Brown, J. S., Destoumieux-Garzon, D., Vittecoq, M., Hamede, R., Tasiemski, A., Boutry, J., Tissot, S., Alix-Panabieres, C., Pujol, P., Renaud, F., Simard, F., Roche, B., Ujvari, B., & Thomas, F. (2021). On the need for integrating cancer into the One Health perspective. *Evolutionary Applications*, 00, 1–5. https://doi.org/10.1111/eva.13303