

Review



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Urban environment and cancer in wildlife: available evidence and future research avenues

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While it is generally known that the risk of several cancers in humans is higher in urban areas compared with rural areas, cancer is often deemed a problem of human societies with modern lifestyles. At the same time, more and more wild animals are affected by urbanization processes and are faced with the need to adapt or acclimate to urban conditions. These include, among other things, increased exposure to an assortment of pollutants (e.g. chemicals, light and noise), novel types of food and new infections. According to the abundant literature available for humans, all of these factors are associated with an increased probability of developing cancerous neoplasias; however, the link between the urban environment and cancer in wildlife has not been discussed in the scientific literature. Here, we describe the available evidence linking environmental changes resulting from urbanization to cancer-related physiological changes in wild animals. We identify the knowledge gaps in this field and suggest future research avenues, with the ultimate aim of understanding how our modern lifestyle affects cancer prevalence in urbanizing wild populations. In addition, we consider the possibilities of using urban wild animal populations as models to study the association between environmental factors and cancer epidemics in humans, as well as to understand the evolution of cancer and defence mechanisms against it.

1. Introduction

The impact of urbanization (defined as the process by which humans form dense settlements constructed of buildings, roads and supporting infrastructure [1]) on the diversity, ecology and health of wild animals has been a focus of the studies in the field of ecology for the last few decades. These studies have led to an understanding that cities are functional ecosystems and experience the same biological processes as wild ecosystems, including evolution [2]. The accumulation of knowledge in the field of urban ecology has built a solid foundation for a new burgeoning field of study: urban evolutionary biology, which aims to understand how urbanization influences genetic changes within populations [3]. Rapidly expanding urban areas act as alternative selection pressures, to which some species are able to adapt via allele frequency variations and potentially mutations [1]. At the same time, other species rarely seen in cities historically are currently colonizing developed areas by becoming more tolerant of living near humans [4]. The causes of this change in tolerance and the genetic basis of adaptive evolution of the urban environment are not known. Selective pressures acting on wild animals living within city borders or near urban

development include an increased exposure to high human disturbance [5], elevated noise levels [6], increased chemical pollution [7], artificial light at night [8], novel food sources [9,10] and changes in infection patterns [11]. The effects of these factors on human health and wellbeing are relatively well studied (e.g. [12,13]), but we still know very little about the health, life-history strategies and causes of mortality of wild animals living in the cities, or about the mechanisms through which wild animals adapt to urban conditions.

One adverse health effect of urban environments that has received a lot of attention in humans is cancer, which has become one of the leading causes of human mortality. This is mostly due to characteristics of our modern lifestyle, including recent changes in diet, alcohol consumption and smoking, and increased exposure to a mixture of pollutants [14–17]. In addition, an increasing proportion of cancer deaths may be ascribed to the decrease in mortality due to other factors like accidents, hunger or infectious diseases, resulting in an increasing proportion of human populations reaching old age [18].

Wild animal populations can be compared to prehistoric human populations, in which fossil data indicate a low prevalence of cancer [19]. It is clear that the characteristics of a modern lifestyle and the urbanizing environment have brought along a change in cancer prevalence in humans, but so far little attention has been given to similar changes in wild animals. It has only recently been proposed that human activities might increase the cancer rate in wild populations [20,21]. In this article, we identify characteristics of the urban environment that have been associated with cancer in humans, and review the literature on the known health effects of these factors on wild animals, thereby describing the available direct and indirect evidence linking environmental changes resulting from urbanization to cancer-related physiological changes in wild animals. We also discuss the possibilities of changed mortality patterns in urban wild animals, including reduced predation pressures, increased resource availability and changes in host–parasite dynamics, which—as in humans—could lead to a larger proportion of populations reaching old age and accordingly potentially developing cancer. By identifying the knowledge gaps in this field, we suggest future research avenues, with the ultimate aim of understanding the magnitude of how humans' modern lifestyle affects cancer prevalence in urbanizing wild populations as well as the possibilities of using urban wild animal populations as models to study the association between environmental factors and cancer epidemics in humans.

2. Urban nutrition and cancer

In humans, cancer is related to dietary choices and to changes in diet over our evolutionary history [22]. The major changes that have taken place in our diet concern glycaemic load, fatty acid and macronutrient composition, micronutrient density, acid–base balance, sodium–potassium ratio and fibre content [23]. An example of a population suffering increased cancer prevalence as a result of diet change is the Inuit population, where malignant diseases, including cancers, were thought to be virtually non-existent at the end of the nineteenth century but have become increasingly frequent

during the twentieth century [24]. Wild animals that are in contact with humans live in a disturbed, resource-rich environment, and these environmental properties favour the emergence and proliferation of profiteering/cheating cells, namely, carcinogenesis [22]. Wild animals in urban environments routinely eat anthropogenic food items (e.g. bread, processed foods and sugar-rich foods) that they did not previously eat [10], through supplementary feeding (reviewed by [25]) and/or unintentional food provisioning. At the global level, regions with the highest human densities and per capita food losses are most affected by those anthropogenic subsidies, which have shaped the architecture of many ecosystems [26].

In some cases, supplementary feeding could aid in the maintenance of body condition, especially in wintering animals (reviewed in [27], but see also [28] for no positive effects). By reducing deaths caused by famine, human food can increase the survival of wild animals (e.g. [29]), with the proportion of individuals reaching older age and therefore (like human populations) being more vulnerable to developing cancer. Alternatively, in some instances, supplementary feeding could also increase the ability of an animal to suppress tumour growth due to better body condition. So far, however, there is no evidence for this latter possibility. Despite the increasing popularity of wildlife feeding, the literature on health effects of these practices is sparse and site- or species-specific [30], mainly concentrating on food quantity rather than food quality [10] and general fitness effects rather than specific physiological pathways. Inappropriate nutrition (e.g. high levels of processed fat, sub-optimal levels of protein, vitamins, antioxidants and other essential nutrients) can lead to depletion of fat reserves, poor body condition and decrease in innate and acquired immune responses in wildlife (reviewed by Birnie-Gauvin *et al.* [10] and Becker *et al.* [31]). We can expect the possible link between poor nutrition and cancer to be mediated at least partly by lowered immunity, which results from poor-quality anthropogenic food. In addition, a review of the nutritional effects of supplementary food on wildlife demonstrated the negative effects of provisioning on protein or micronutrient deficiencies [9], which have been suggested to increase cancer risk in humans [32].

In humans, obesity is one of the most important known causes of cancer, and about 10% of all cancer deaths among non-smokers are related to obesity [33]. The underlying mechanisms can be related to changes in metabolic and physiological pathways involved in oncogenesis, including hormone concentrations, growth factors, inflammatory cytokines and oxidative stress [22,33]. The link between anthropogenic food, obesity and cancer is so far virtually unexplored in wild animals, although obesity has been acknowledged as a problem resulting from wildlife feeding [34,35]. We suggest that tourist-fed small mammals (e.g. squirrels in urban parks) are a good place to start looking for links between anthropogenic food, obesity and cancer in wildlife.

3. Infections, urban habitat alterations and cancer

The urban environment can break down existing host–parasite relationships, thereby allowing hosts to 'escape'

their natural parasite communities [36]. However, increased population densities and contact between different species in urban areas can create opportunities for increased disease transmission and act as a proliferation source of novel diseases [11]. Infectious agents have been increasingly recognized as causes of cancer; they are presently accepted as aetiological agents for about 20% of human cancer [37]. Candidate pathogens have been correlated with most of the remaining 80% of human cancers, but their causal role has not yet been determined. Known human tumour viruses have very different genomes and life cycles, and represent a number of virus families [38], indicating that oncogenicity could be a characteristic of a wide range of viruses. While it is known that urbanization can increase the prevalence of viral infections in wild animals (e.g. [39]), the studies on virus prevalence in wildlife in the context of urbanization have so far mainly focused on potential zoonotic diseases, and data on potentially oncogenic viruses in wild animals are largely missing.

In all well-studied examples of infection-induced oncogenesis in humans and wildlife, infectious agents probably act jointly with non-infectious environmental factors, such as those discussed in the other sections of this article. Infectious agents typically abrogate the major barriers to cancer, and non-infectious agents further compromise these barriers by generating mutations, altering host defences and stimulating cell proliferation [40]. Pollutants may contribute to infection-induced oncogenesis by causing mutations or through immune suppression. Sea turtle fibropapillomatosis, for example, is caused by an alpha herpes virus and is more prevalent in areas subject to pollution from human activities [41], and levels of polychlorinated biphenyls are elevated in the blubber of genital carcinomas of sea lions induced by a gamma herpes virus [42]. Another example is increased retroviral (feline immunodeficiency virus) infections in feral cats in urban settings with high host densities, which is associated with increased risk of cancer in domestic cats [43,44]. These infection-associated tumours emphasize the need to consider infectious causation when the tumours are linked to immunosuppressive pollutants, or more generally with human activities.

Another mechanism that links urban habitat alteration, infections and cancer is habitat fragmentation and changes in connectivity between populations. Urbanization often results in reduced population sizes or greater isolation (reviewed by [1]). While this may facilitate infection transmission among urban populations, it may also facilitate the escape of uninfected individuals from populations that overcome with infection. Restriction of gene flow between populations due to barriers such as roads and buildings can lead to lower genetic diversity (i.e. [45]). In addition to the clear reciprocal link between genetic diversity and vulnerability to pathogens, accumulating evidence supports an association between reduced genetic diversity, inbreeding and cancer [46].

4. Urban chemical pollution and cancer

Urban pollution can act as a mutagen, increasing mutation rates in the germline or within somatic tissues [1]. For example, proximity both to cities and to steel mills increased germline mutation rate in herring gulls [47] and air filtration

reduced heritable mutation rates in laboratory mice housed outdoors near major highways and steel mills [48]. This process can accelerate adaptation to urban environment. For example, a recent study demonstrated the independent evolution of tolerance to polychlorinated biphenyls (PCBs) in four Atlantic killifish populations in urban estuaries [49]. At the same time, mutations in DNA are considered the proximate cause of cancer [50]. Environmental pollutants are known to cause cancer in humans, and evidence that similar pathways are also affecting the health of wild animals has been accumulating. Classical examples include the effects of water pollution with polycyclic aromatic hydrocarbons (PAHs), PCBs and dichlorodiphenyltrichloroethanes on cancer epidemics in several fish species [51], as well as mammals [42,52]. However, surprisingly, most of the numerous pollutants found in urban environments are unexplored in this context.

One of the possible research directions to pursue would be to study the mixture of pollutants found in the air of cities. This pollution comes predominantly from local vehicular traffic in urban areas with emission of gases, particles, volatile organic compounds and PAHs, many of which are considered as carcinogens. An increased risk of lung cancer associated with exposure to outdoor air pollutants has been consistently found in several studies on humans [53]. Other agents present in air pollution have been shown to be associated with mammary carcinomas in rodents (i.e. benzene, kerosene, toluene and xylenes [54,55]) and human breast cancer (i.e. nitrogen dioxide, benzene and PAHs [56–58]). At the mechanistic level, this relationship between carcinogenesis and air pollution is due to an increase of chromosome aberrations and micronuclei in lymphocytes [59,60], changes in the expression of genes involved in DNA damage and repair, epigenetic effects (DNA methylation), inflammation, telomere shortening, immune response and oxidative stress [61].

So far, only a handful of studies have been published on the relationship between air pollution and cancer incidence in captive animals, and no studies have, to the best of our knowledge, ever studied this topic in wild populations. In captive mice, for example, an increase in the incidence of lung adenoma and tumour multiplicity of urethane-induced adenomas was associated with traffic-related air pollution [62]. As an indirect link between air pollution and oncogenic processes in wild populations, exposure to volatile organic compounds is correlated with an upregulation of intracellular antioxidants (i.e. glutathione), suggesting an increased production of reactive oxygen species, a factor known to influence cancer development [63]. Future studies should thus take advantage of new technologies available to measure exposure to air pollution at the individual level [63] to study the dose at which animals are exposed in the wild and the impact of this contamination on cancer incidence.

5. Light and noise pollution in urban environments

In humans, the link between artificial light at night (ALAN) and cancer was first established in female employees working rotating night shifts (reviewed by Chepesiuk [64]) and was recently also confirmed in the context of urbanization [65]. The increased breast cancer risk in female night shift workers

has been postulated to result from the suppression of pineal melatonin production [66]. Melatonin, a hormone present in all vertebrates and also in bacteria, protozoa, plants, fungi and invertebrates, is involved in the regulation of circadian rhythms; it peaks at night and is suppressed by light [67]. In a laboratory experiment, it was shown that even minimal light contamination (0.2 lux) disrupted normal circadian production of melatonin and promoted tumour growth in rats [68]. Direct links between ALAN, melatonin and cancer prevalence have not been established for wild animals so far. However, there are several examples of ALAN–wildlife studies showing changes in the levels of hormones that have been related to cancer in humans (e.g. testosterone in Siberian hamster *Phodopus sungorus* [69]; corticosterone in social voles *Microtus socialis* [70]; melatonin in mouse lemurs *Microcebus murinus* [71] and European blackbirds *Turdus merula* [72]).

Although hormonal effects might be the most important pathway in linking light pollution to cancer prevalence, other possibilities should also be considered. Among them, obesity and metabolic disruption are well-studied consequences of ALAN in humans [73] and should also be considered in wild animals. Light pollution can also affect sleep in wild animals. For example, great tits slept significantly less and woke up earlier when a light-emitting diode was placed in their nest-box [74]. An increase in sleep duration has been postulated as a mechanism that helps to decrease cancer burden, because sleep duration is associated with immune system strength [75]. Because studies on the effects of ALAN on the health of wild animals have so far concentrated largely on hormonal changes, the next steps would be to expand these studies to (i) characterize the specific cancer-related physiological pathways affected by ALAN and to (ii) analyse neoplasia prevalence in animals subjected to ALAN. As the clearest link with ALAN in humans is to breast cancer, more studies on light pollution effects on wild mammals are needed, considering that the main focus of studies on ALAN to wildlife has so far been on birds and insects.

In addition to light pollution, anthropogenic noise pollution is an important environmental stressor that is rapidly gaining attention among biologists and can, among other effects, disrupt the normal sleep–wake cycle of animals [76]. In laboratory rats, noise stress increased plasma levels of stress hormones and oxidative stress [77]. Continued oxidative stress can lead to chronic inflammation, which in turn could exacerbate most chronic diseases including cancer [78]. Studies on humans have cautiously linked noise pollution levels to higher risks of non-Hodgkin lymphoma [79] and an increased risk of oestrogen receptor-negative breast cancer [80]. As expected, nothing is so far known about the effects of noise pollution on cancer prevalence in wild animals. Nevertheless, house sparrow (*Passer domesticus*) nestlings reared under traffic noise had reduced telomere length when compared with their unexposed neighbours, an effect that could be mediated by oxidative stress [81]. Shorter telomeres have been linked to increased vulnerability of several types of cancer (e.g. [82]). In addition, noise exposure increased stress hormone levels and suppressed cellular immunity in tree frogs (*Hyla arborea* [83]), and both of these effects are generally considered to be cancer risk factors (e.g. [84]). Because it is so difficult to disentangle the effects of noise from other anthropogenic stress sources such as traffic pollution, disturbance or light pollution in the field,

experimental studies on the physiological effects of noise pollution on wild animals are needed.

6. Changes in survival and life-history strategies

In humans, increased survival and the consequent increased proportion of the population reaching old age have been suggested to be one of the causes of current cancer epidemics because cancer is an age-related disease [85]. A meta-analysis on birds indicated that the urban environment may enhance survival [86], possibly through increased resource availability or lower predation pressure. Lower rates of predation and resulting higher survival in urban habitats have also been shown in small mammals (e.g. [4]). Age structures of urban wild animal populations have rarely been studied, but there are some data supporting the hypothesis that there are more old animals in urban populations than in rural populations (e.g. [87]). While senescence effects are shown to be common in wild animals [88], cancer demography data are lacking for wild populations, and more research is needed to elucidate if cancer rates are higher in aged wild animals [89]. However, numerous studies in zoo animals (e.g. [90]) have indicated that, as in humans, survival to old age can lead to increased cancer mortality in a wide range of animal species.

While age can be a risk factor for cancer development, increased survival prospects can also lead to changes in life-history strategies and physiological investment patterns, with higher investments in self-maintenance over reproduction [89]. For example, it has been shown that reduced predation alone can substantially slow the rates of physiological ageing in mammals, leading to a ‘slower’ life strategy (delayed reproduction and longer somatic maintenance [91]). A slower-paced life with increased investment in self-maintenance (with a trade-off in lower reproductive investment) has been suggested for birds living in urban habitats [86] (see also [92] for the emergence of a life-history physiology syndrome in urban *Daphnia*). This can result in stronger cancer defence mechanisms in animals in more stable, more resource-rich and less risky habitats, as cities are for some species. Accordingly, comparing urban and rural populations of wild animals could help to identify physiological mechanisms related to tumour suppression. These types of study would hugely benefit if the age of the study subjects was known. We are therefore in urgent need of establishing longitudinal research projects including urban and rural animal populations that would allow us to take into account the age of the animal as well as distinguish the causes of mortality in urban and rural wild animal populations.

7. Conclusion and future directions

Urbanization affects an ever-increasing number of wild animals and their habitats. Our responsibility is to ensure that the development of human societies does not come at the expense of wild animal diversity and health. At the same time, urbanizing wild animal populations could be a promising model system for understanding the evolution of cancer and physiological defences against it, and help to define the factors of the urban environment that have the strongest potential to increase cancer risk. Studying cancer prevalence and defence mechanisms in urban wild animals could therefore lead to a better understanding of how to develop an

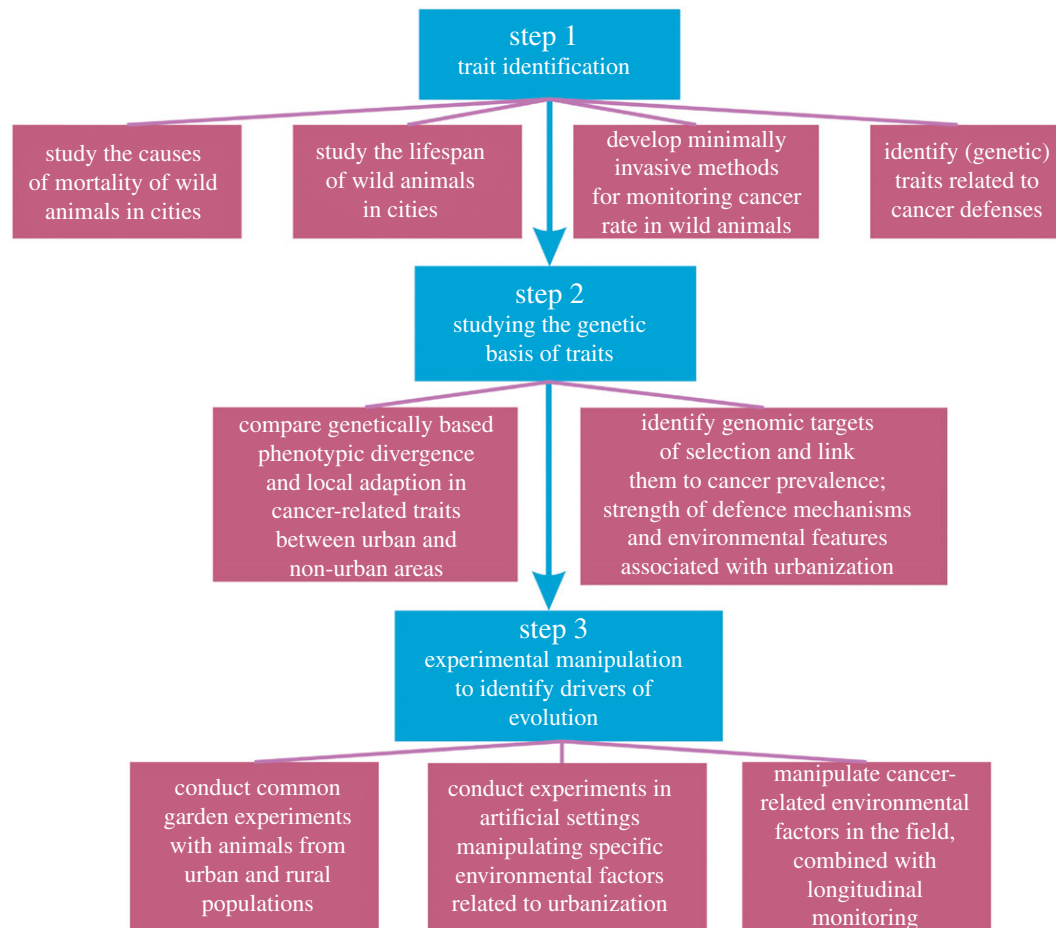


Figure 1. Flow chart of possible experimental designs for studying cancer in wild populations. Steps proposed here are based on the suggestions for studying evolution in urban environments by Donihue & Lambert [2]. (Online version in colour.)

urban environment with minimal negative health effects for both humans and wild animals. At the same time, urban areas could be considered as natural laboratories for studying the evolution of cancer. This is a promising research avenue, considering the notion that the fastest measured rates of evolution are associated with human-altered environments [2].

Species probably vary in their susceptibility to cancer due to variation in tolerance to environmental oncogenic factors [93] and variation in cancer defence mechanisms [94]. Inter-specific variation in cancer risk may depend on life-history characteristics such as body size, growth rate and investment in sexual signal traits, but also on physiological mechanisms such as wound healing or the presence or depth of placenta-tion (reviewed by Harris *et al.* [94]). While the existence of these internal species-specific differences in cancer defence have to be acknowledged, investment in cancer defences still exhibits a considerable amount of plasticity depending on extrinsic factors such as mortality risk and resource predictability [89]. Accordingly, if we want to extrapolate the impact of urban environment on cancer probability from wildlife to humans, we must take these species-specific differences into account. The best way to do that would be to compare cancer prevalence and cancer defences between populations of the same species living in habitats that are more or less affected by urbanization. Considering that cities tend to be more similar to one another than they are to nearby non-urban ecosystems, studying cancer susceptibility and resistance in the context of urbanization would also contribute to understanding of how common convergent

evolution is in these physiological processes across different species, traits and genes (see also [95] for key questions in urban evolutionary ecology).

By acknowledging the diversity of cancer aetiologies, there is the possibility of detecting the ecological conditions where anthropogenic impacts on the environment should increase or decrease cancer prevalence. While most urban environmental factors (pollution, low-quality food and infections) should increase cancer prevalence, some characteristics of the urban environment can be considered cancer suppressive. For example, urbanization can affect oncogenic pathogens more than their hosts, leading to fewer cancers caused by infection. Similarly, increased resource availability can lead to better body condition and immune defences. Urban environmental factors can act as selection pressures that may cause new mutations or act on standing genetic variation within populations, leading to both higher cancer probability through DNA mutations and to higher probability for genome-based cancer defence mechanisms to arise [1].

Given that advancing age is indisputably the most significant risk factor for cancer, a higher prevalence of cancer (or oncogenic processes) is expected in the prey population under such conditions. In predator–prey relationships, different ecosystem consequences are expected depending on which protagonist—the prey or the predator—is the most affected by human-induced oncogenic processes. Because these issues, in turn, differentially affect the frequency of genes involved in cancer resistance, numerous and complex reciprocal feedbacks are expected [75]. Thus, while

urbanization and other anthropogenic changes in the environment are expected to increase the frequency/severity of oncogenic processes in wildlife species [20], there are currently no simple answers to the questions about how this will influence biodiversity and ecosystem functioning in urban habitats.

It has been suggested that urban settings unintentionally provide experimental macrocosms for studying the ability of organisms to adapt to rapid changes in their habitats due to intense human land use ('the urban Petri dish' [2]). For testing evolutionary hypotheses in urban settings, a three-tiered programme has been suggested, including (i) identification of traits that vary with ecological context, (ii) studying the genetic basis of those traits and (iii) experimental manipulation to directly identify drivers of those trait differences [2].

Accordingly, the first steps should be comparing traits related to cancer prevalence and cancer defences between urban and rural populations (figure 1). As a first step, we need a better understanding of age structures and causes of death in urban wild animal populations compared to their rural counterparts. As a second step, minimally invasive methods for assessing cancer prevalence in wild populations need to be developed. And third, we need methods for assessing the investment in cancer defences, both on the level of immune system functioning and gene expression. Since the link between pollution in aquatic environments and cancer in wildlife has been convincingly established, a good starting point would be ponds and canals in city parks in highly urbanized areas, which are important habitats for fish and a wide variety of wild and semi-domesticated birds.

The second step would be to study differences in genes related to tumorigenesis or tumour suppression between wild animals from urban and rural habitats. As an example, a study in the flounder (*Platichthys flesus*) found higher polymorphism of the known tumour suppressor gene p53 in populations living in highly contaminated versus reference estuaries [96]. As a third step, experimental evolutionary approaches using urban environmental characteristics (i.e. the use of laboratory or controlled field manipulations to investigate evolutionary processes) are needed, because they may not only intensify the selection of already known

suppressive mechanisms, but could also lead to the discovery of novel tumour suppressor mechanisms [93]. Both field and experimental evolutionary studies have demonstrated that organisms exposed to environmental oncogenic factors can—sometimes rapidly—evolve specific adaptations to cope with pollutants and their adverse effects on fitness [49]. It is suggested that the fastest rates of evolution globally take place in human-impacted habitats [97], and there is strong evidence of adaptive evolution in urban systems (reviewed by Donihue & Lambert [2]). From an applied perspective, Vittecoq *et al.* [93] suggested that studying these species could inspire novel cancer treatments by mimicking the processes allowing these organisms.

Although this area now commands the attention of a variety of researchers, a broad predictive framework is lacking, mainly because the links between urbanization, oncogenic processes and biodiversity are complex. One single method or model cannot thoroughly reveal how organisms challenged by an urban context resist cancer progression, or how ecosystems will react to an increase in cancer prevalence in resident species. A focused interdisciplinary research effort combining the work of urban ecologists, cancer biologists, animal physiologists and geneticists will be rewarded with an understanding of how modern lifestyles affect cancer prevalence in urbanizing wild populations and how animals cope with this selection pressure, possibly allowing us to use urban wild animal populations as models to study the association between environmental factors and cancer epidemics in humans.

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