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Population ecology

Landscape-level toxicant exposure mediates infection impacts on wildlife populations

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Anthropogenic landscape modification such as urbanization can expose wildlife to toxicants, with profound behavioural and health effects. Toxicant exposure can alter the local transmission of wildlife diseases by reducing survival or altering immune defence. However, predicting the impacts of pathogens on wildlife across their ranges is complicated by heterogeneity in toxicant exposure across the landscape, especially if toxicants alter wildlife movement from toxicant-contaminated to uncontaminated habitats. We developed a mechanistic model to explore how toxicant effects on host health and movement propensity influence range-wide pathogen transmission, and zoonotic exposure risk, as an increasing fraction of the landscape is toxicant-contaminated. When toxicant-contaminated habitat is scarce on the landscape, costs to movement and survival from toxicant exposure can trap infected animals in contaminated habitat and reduce landscape-level transmission. Increasing the proportion of contaminated habitat causes host population declines from combined effects of toxicants and infection. The onset of host declines precedes an increase in the density of infected hosts in contaminated habitat and thus may serve as an early warning of increasing potential for zoonotic spillover in urbanizing landscapes. These results highlight how sublethal effects of toxicants can determine pathogen impacts on wildlife populations that may not manifest until landscape contamination is widespread.

1. Introduction

Agricultural intensification and urbanization can expose wildlife to toxicants such as pesticides, persistent organic pollutants and heavy metals. In addition to direct effects on wildlife health [1], toxicant exposure can influence fitness by modifying susceptibility to, and impacts of, pathogen infection and by altering movement capacity (table 1). Since human-modified habitats can increase toxicant loads [14] and pathogen transmission in wildlife [15], understanding linkages between animal movement, infection and toxicant exposure has important implications for wildlife health and zoonotic risk in urbanizing landscapes.

Exposure to toxicants can amplify or counteract negative effects of infection on individual hosts [4,9,16,17], with implications for population-level transmission. Toxicants can promote transmission through immunocompromise which increases infection susceptibility [18–23] or shedding of infectious stages [4]. Alternatively, toxicants can interfere with transmission by killing parasite stages [5] or upregulating host immunity [7,24]. Past theory has explored how environmental stressors such as toxicants can influence local transmission dynamics under assumptions that all individuals in the population are toxicant-exposed

Table 1. Examples of toxicant effects on infection susceptibility, impacts of pathogen infection and movement capacity in wildlife.

outcome	possible mechanism	example	ref.
↑ infection	toxicant causes host immune suppression	green frog (*Rana clamitans)* tadpoles exposed to pesticides experienced greater encystment by trematode cercariae	[2]
		vampire bats (*Desmodus rotundus*) with higher total mercury concentrations had weaker *Escherichia coli* killing ability and impaired innate immunity	[3]
	toxicant stimulates greater production of parasite infectious stages	snails (*Potamopyrgus antipodarum*) shed more cercariae when exposed to the herbicide glyphosate	[4]
↓ infection	toxicant depresses production of parasite stages	pesticides applied to agar cultures of the amphibian fungus *Batrachochytrium dendrobatidis* inhibited zoospore and zoosporangia production	[5]
	toxicant decreases the likelihood of infectious contacts between individuals	round goby (*Neogobius melanostomus*) exposed to treated wastewater effluent had higher tissue concentrations of pharmaceuticals and displayed reduced aggression	[6]
	toxicant upregulates host immune function, or harms parasite	mallard ducks (*Anas platyrhynchos*) exposed to lead had lower richness and infection intensity of helminths	[7]
↓ survival	synergistic effects of toxicant and parasite	juvenile roundhead galaxias (*Galaxias anomalus*) exhibited no changes in survival when exposed to a trematode parasite or glyphosate singly, but reduced survival when exposed to both	[4]
	energy expenditure metabolizes stored toxicant	Mexican free-tailed bats (*Tadarida brasiliensis*) experienced organochlorine pesticide poisoning after being subjected to simulated migratory flight	[8]
↑ survival	pathogen impedes metabolization of toxicant	zebrafish (*Danio rerio*) infected with a bacterial pathogen and exposed to a high dose of phenanthrene (a polycyclic aromatic hydrocarbon) had higher survival than uninfected fish exposed to the same phenanthrene dose	[9]
↓ movement	toxicant impairs flight performance	lower flight height and movement rate in golden eagles (*Aquila chrysaetos*) exposed to lead	[10]
	toxicant causes physical deformity	amphibians closer to agricultural areas or lawns have been shown to have higher risk of limb malformations, likely due to pesticide exposure	[11,12]
	toxicant decreases host energy reserves	migrating white-crowned sparrows (*Zonotrichia leucophrys*) experimentally dosed with a neonicotinoid insecticide at a stopover site exhibited reduced feeding, rapidly lost body fat, and needed extra time before they were ready to continue migrating	[13]

[25]. Further work is crucially needed to examine the consequences of heterogeneity in toxicant exposure (e.g. arising from spatial structure).

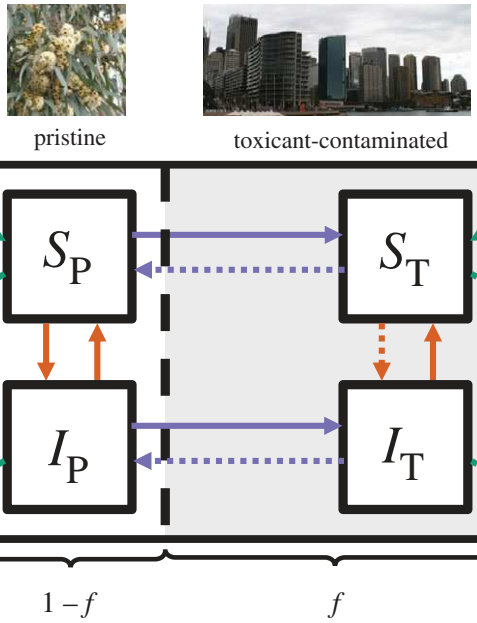
Assessing wildlife disease risk in contaminated landscapes depends on how toxicant exposure influences animal movement. Toxicants can reduce movement capacity directly by causing physical deformities [11,12] or indirectly by interfering with memory and navigation [26,27]. If contaminated habitats attract wildlife [28,29] but impair subsequent movement, these habitats could act as ecological traps [13]. Alternatively, if toxicant impacts are primarily experienced by infected animals, movement of infected animals into contaminated habitats could reduce population-level impacts of virulent pathogens through mechanisms similar to migratory culling [30].

Here, we develop a mathematical model of wildlife infection dynamics in toxicant-contaminated landscapes. We explore how host population size, infection prevalence and potential for zoonotic spillover depend on (i) the proportion of the landscape contaminated and (ii) the effects of toxicant exposure on infection, movement and survival. We interpret our findings in the context of urbanizing landscapes, where we expect more human–wildlife interactions and higher toxicant levels.

2. Methods

In our model, the landscape is divided into 'toxicant-contaminated' and 'pristine' habitat; the fraction of the landscape that is contaminated is denoted by f (figure 1). We consider toxicant-contaminated habitat to be any human-altered habitat where wildlife encounter pesticides, heavy metals or other pollutants. We use differential equations to track the population dynamics of animals by their infection status (susceptible, S, or infected, I) and current habitat (indicated by subscripts T for toxicant-contaminated and P for pristine). Animals in toxicant-contaminated habitat potentially incur costs to survival and movement, and increased or decreased transmission risk. We assume transmission is density-dependent, and costs of toxicant exposure are only incurred while animals remain in toxicant-contaminated habitat. Our model is motivated by a hypothetical flying fox host species infected with a virus. Previous work has documented adverse health effects of toxicants on flying foxes and other bat species

(*a*) model schematic



(*b*) model equations

$$\frac{dS_P}{dt} = \left(b_0 - \frac{b_1(S_P+I_P)}{1-f}\right)(S_P+I_P) - mS_P - \beta_P S_P I_P + \gamma I_P - \sigma f S_P + \sigma(1-c_\sigma)(1-f)S_T$$

$$\frac{dI_P}{dt} = \beta_P S_P I_P - \gamma I_P - (m+\mu)I_P - \sigma f I_P + \sigma(1-c_\sigma)(1-f)I_T$$

$$\frac{dS_T}{dt} = \left(b_0 - \frac{b_1(S_T+I_T)}{f}\right)(S_T+I_T) - \frac{m}{1-c_m}S_T - \beta_T S_T I_T + \gamma I_T + \sigma f S_P - \sigma(1-c_\sigma)(1-f)S_T$$

$$\frac{dI_T}{dt} = \beta_T S_T I_T - \gamma I_T - \frac{m+\mu}{1-\alpha c_m} I_T + \sigma f I_P - \sigma(1-c_\sigma)(1-f)I_T$$

(*c*) model parameters

process	parameter	definition	units	value
demography	m	natural mortality rate	year^{-1}	0.1
	b_0	maximum *per capita* birth rate	host^{-1} year^{-1}	0.4
	b_1	density-dependent per capita birth rate	year^{-1}	$(b_0-m)/50000$ = 6e – 6
	c_m	cost of toxicants to survival		0.2
infection	β_P	transmission rate in pristine habitat	host^{-1} year^{-1}	0.006
	β_T	transmission rate in toxicant-contaminated habitat	host^{-1} year^{-1}	0.0015, 0.006, 0.0105
	γ	recovery rate	year^{-1}	36.5
	μ	disease-induced mortality rate	year^{-1}	0.25
	α	synergistic effect of infection and toxicants on survival		2
movement	f	fraction of the landscape that is toxicant-contaminated		0.01–0.99
	σ	*per capita* dispersal rate	year^{-1}	–log 0.1
	c_σ	cost of toxicants to dispersal		0.2, 0.8

Figure 1. (*a*) Schematic of the compartmental model. Squares represent host population according to infection status (susceptible or infected) and habitat (pristine or toxicant-contaminated). The parameter f represents the fraction of toxicant-contaminated landscape. Horizontal arrows (purple) represent movement between pristine and toxicant-contaminated habitats, vertical arrows (orange) represent transitions between susceptible and infected classes, and diagonal arrows (green) represent demographic processes. Dotted arrows represent processes affected by toxicants. (*b*) Differential equations of the model, colour-coded to represent movement, infection and demographic processes as in (*a*). (*c*) Model parameters with definitions, units and default values used in model simulations.

(electronic supplementary material, table S1). Flying foxes feed on fruiting and flowering plant species in natural, urban and agricultural landscapes [31], where they face exposure to pesticides and heavy metals [32]. These bats are reservoirs of pathogens that can be transmitted to domestic animals and humans, notably Hendra and Nipah viruses [33]. The model structure, equations and parameter values are detailed in figure 1 and the electronic supplementary material.

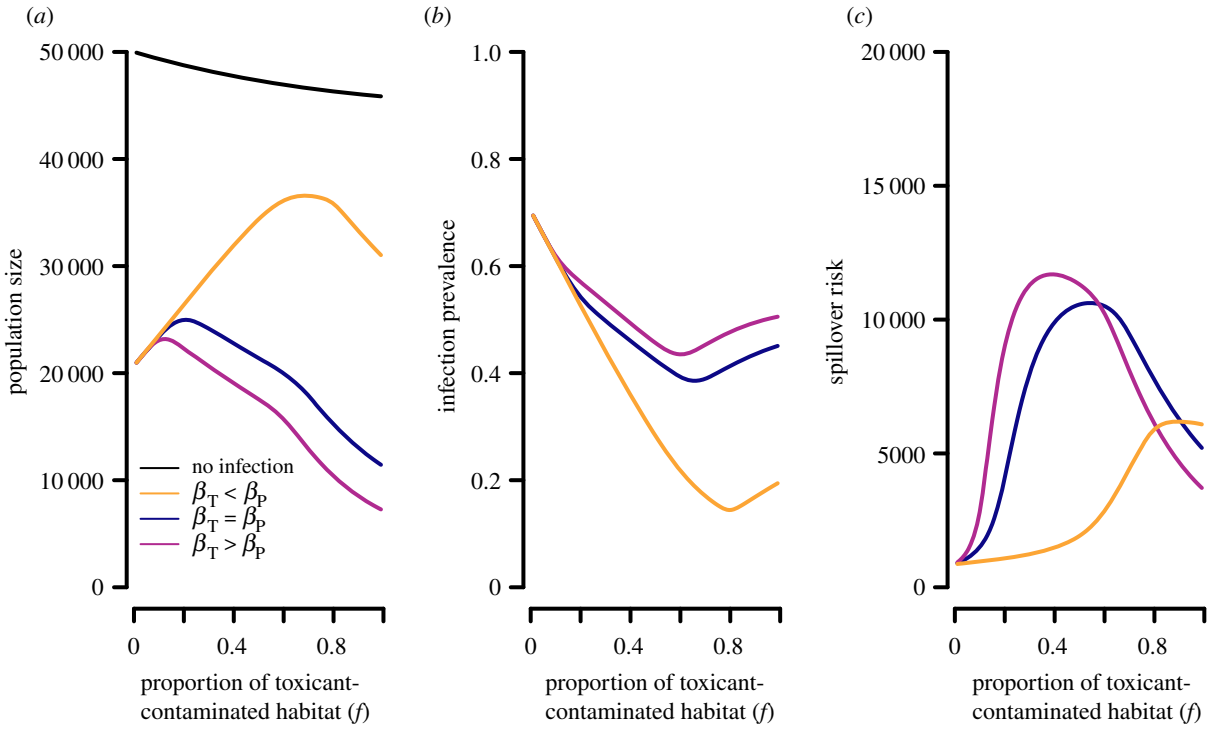


Figure 2. Equilibrium population size (*a*), infection prevalence (*b*) and spillover risk (the density of infected animals in toxicant-contaminated habitat) (*c*) plotted as a function of the proportion of toxicant-contaminated habitat. In all panels, the dispersal cost from toxicant-contaminated habitat is relatively low ($c_\sigma = 0.2$). Transmission is constant in pristine habitat ($\beta_P = 0.006$) and varies in toxicant-contaminated habitat ($\beta_T = 0.0015$, 0.006 and 0.0105; orange, blue and purple lines, respectively). Population size in the absence of infection is shown for comparison in (*a*) (black line). Other parameter values are provided in figure 1.

We quantified the landscape-level effects of toxicants on host population viability and infection risk by the equilibrium host population size, $N^* = S_P + I_P + S_T + I_T$, and equilibrium infection prevalence, $p^* = (I_P + I_T)/N^*$, respectively. We quantified the potential for zoonotic spillover (henceforth 'spillover risk') as the number of infected animals in toxicant-contaminated habitat divided by this habitat's frequency on the landscape, $\rho = I_{T^*}/f$. We focused on the density of infected animals in toxicant-contaminated habitat because we expect more frequent human–wildlife encounters in this human-modified habitat. Specifically, we expect that most areas with high human activity will be toxicant-contaminated, and that these areas also attract wildlife owing to food subsidies provided by crops and ornamental plantings. Our equation reflects a higher human encounter risk when infected animals are concentrated into small amounts of toxicant-contaminated habitat, relative to if the same number of animals were distributed across more widespread toxicant-contaminated habitat.

To explore how wildlife population and infection dynamics are affected by increasing landscape contamination, we varied *f* from 1 to 99%, representing the transition from a totally pristine landscape to a totally contaminated one. For each value of *f*, we recorded population size, infection prevalence, and spillover risk after simulations reached equilibrium (i.e. after 50 years). We initiated each model run with 50 000 hosts, 100 of which were infected; hosts were initially distributed between toxicant-contaminated and pristine habitats according to their relative frequencies on the landscape. For our default parameterization, we assumed costs of toxicant exposure on host survival were higher for infected than uninfected individuals, and that toxicants reduced dispersal from toxicant-contaminated habitat for all hosts.

We considered three scenarios for how toxicant exposure influences transmissibility: the pathogen is (i) equally transmissible in both habitats ($\beta_T = \beta_P$); (ii) less transmissible in toxicant-contaminated habitat (e.g. reflecting immune priming, reduced parasite survival in hosts, or reductions in activity/intraspecific contacts resulting from toxicant exposure; $\beta_T < \beta_P$), or (iii) more transmissible in toxicant-contaminated habitat (e.g. reflecting toxicant-induced increases in susceptibility or shedding, or crowding around toxicant-contaminated food subsidies; $\beta_T > \beta_P$). We performed sensitivity analyses by covarying five parameters relating to infection, toxicant exposure and their interaction using Latin Hypercube Sampling (see electronic supplementary material). Our model was solved numerically in R v. 3.6.1 [34] using the *deSolve* package [35].

3. Results

(a) Population and prevalence impacts

In the absence of infection, equilibrium host population size declines monotonically with *f*, the extent of landscape contamination (figure 2*a*). However, the magnitude of the decline is low, even at high values of *f*, reflecting largely sublethal effects of toxicant exposure. In the presence of infection, initially increasing the proportion of toxicant-contaminated habitat reduces overall prevalence (figure 2*b*). At low fractions of toxicant-contaminated habitat, the number of hosts in toxicant-contaminated habitat is too low to sustain transmission, and costs to survival and movement mean that infected animals are less likely to return to pristine habitat. When toxicant-contaminated habitat is sufficiently common, host abundance supports more transmission in toxicant-contaminated habitat than remaining pristine habitat, leading to an increase in infection prevalence (figure 2*b*). As a result of changes in prevalence, host population size initially increases with *f*, then decreases (figure 2*a*). When a high fraction of the habitat is contaminated, the combined costs of infection and toxicants lead to steep host population declines.

When transmission is lower in toxicant-contaminated than pristine habitat ($\beta_T < \beta_P$), toxicant-contaminated habitat acts as a sink for the pathogen under low to moderate landscape contamination; relatively small host population declines occur

only when almost all landscape is contaminated (figure 2*a*, yellow line). Conversely, when transmission is enhanced in toxicant-contaminated habitat ($\beta_T > \beta_P$), the combined effects of toxicants and infection drive more severe host population declines in an increasingly contaminated landscape (figure 2*a*, purple line). Infection prevalence in wholly-contaminated landscapes is always lower than in pristine landscapes, even when the pathogen is more transmissible in toxicant-contaminated habitat, reflecting higher infection-induced mortality in toxicant-exposed hosts (figure 2*b*). A higher cost of toxicants to host movement reduces host population size and prevalence across all landscapes (electronic supplementary material, figure S1A,B).

(b) Spillover risk

Spillover risk is highest at intermediate levels of landscape contamination (figure 2*c*). Peak spillover risk is higher, and occurs at lower proportions of landscape contamination, when the pathogen is more transmissible in toxicant-contaminated habitat (figure 2*c*) and the cost of toxicant exposure to dispersal is higher (electronic supplementary material, figure S1C). Across all scenarios, spillover risk attains its peak at a higher level of landscape contamination than maximum host population size (figure 2 and electronic supplementary material, figure S1).

(c) Sensitivity analyses

Across all parameter combinations, prevalence tended to (i) increase with pathogen transmissibility in toxicant-contaminated habitat and (ii) decrease with increasing costs of toxicant exposure to movement, and costs to survival resulting from infection, toxicant exposure and their synergistic effects (electronic supplementary material, figure S2). The sensitivity of prevalence to each parameter depended on the extent of landscape contamination; prevalence was most sensitive to virulence in pristine habitat and toxicant costs to movement in mostly pristine landscapes ($f = 0.1$), and most sensitive to transmissibility and costs to survival in toxicant-contaminated habitat in mostly contaminated landscapes ($f = 0.9$).

4. Discussion

Wildlife increasingly find themselves in human-modified landscapes, potentially influencing their exposure to toxicants. We developed a mechanistic model to understand the consequences of landscape-level toxicant exposure on host–pathogen dynamics, through sublethal and synergistic lethal effects of infection and toxicant exposure. We found that the extent of contaminated habitat could intensify or dampen pathogen impacts on host populations. Contaminated habitat acted as a sink for pathogens when most of the landscape was pristine, but typically exacerbated pathogen-related host population declines once the landscape was mostly contaminated. Under scenarios where land conversion increases the amount of contaminated habitat over time, wildlife population declines would be expected to occur prior to maximum spillover risk. The largest population impacts of the pathogen were seen when infection prevalence was lower in more contaminated landscapes, indicating high mortality from the combination of infection and toxicants.

Unexpectedly, we found cases where toxicant-contaminated habitat can benefit wildlife by reducing pathogen transmission. When rare, on the landscape toxicant-contaminated habitat may support too few animals to maintain local transmission of density-dependent pathogens and prevent infected animals from returning to pristine habitat through sublethal costs to movement and elevated mortality. Further, if toxicants reduce host contacts or pathogen transmissibility, then increasing landscape contamination can lead to higher population size compared with an entirely pristine landscape. Moderate toxicant-induced movement costs conveyed some benefits by trapping infected individuals in contaminated habitats; however, when movement costs were too high, the net effect on population size tended to be negative, since contaminated habitats became overcrowded, reducing density-dependent fecundity and increasing toxicant-induced mortality.

Our results suggest wildlife whose movement is severely impaired by toxicants could be most negatively affected by landscape contamination. For example, amphibians closer to agricultural areas or lawns have higher risk of limb malformations, likely due to pesticide exposure [36]. Future work should investigate the degree to which amphibians and other vertebrate species experience toxicant-induced deformities or other movement impairments.

A previous model that explored effects of environmental stressors (e.g. eutrophication, heavy metals) on host infection dynamics found that negative, positive and nonlinear relationships between contaminants and infection were possible, but that increasing environmental stressors generally reduced infection prevalence owing to stress-mediated declines in host density [25]. This model assumed all hosts were exposed to stressors, and that stressors increased infection susceptibility. We similarly found the effect of toxicants on infection prevalence and its population-level impacts to be context-dependent and influenced by the extent of landscape contamination and toxicant-induced costs to movement. By also exploring scenarios in which toxicant-contaminated habitat reduces transmission, we found that increasing a stressor (i.e. ubiquity of toxicants) could reduce prevalence by purging the pathogen, thus counterintuitively increasing host populations.

Future work could incorporate additional biological complexity that could modify our model predictions. We assumed that an animal immediately recovers from ill effects of toxicants upon leaving toxicant-contaminated habitat. Allowing toxicants to accumulate in hosts in toxicant-contaminated habitat, and to decrease gradually when hosts leave, could exacerbate toxicant effects on population and infection dynamics. Similarly, accounting for age-related toxicant exposure (e.g. through placental transfer or lactation [37]) and impacts (through bioaccumulation) could influence infection dynamics by inhibiting maternal immunity and age-dependent virulence. Models could further explore linkages between movement, toxicants and infection: for example, by incorporating infection-dependent movement decisions [38,39], or investigating how movement could act as a stressor triggering negative effects of toxicants [8].

Our work suggests that increasing urbanization, if accompanied by greater levels of toxicants, could cause drastic declines in wildlife populations facing other stressors such as infectious disease. Accounting for effects of toxicants on wildlife movement and infection competence could also be crucial for determining zoonotic spillover risk in human-modified landscapes. We recommend that wildlife managers and public health professionals assess multiple health metrics in a focal species, including toxicant exposure and infection

prevalence, and also consider the degree of contamination in the surrounding landscape.

Data accessibility. R code to reproduce analyses and figures is available at: https://dx.doi.org/10.5061/dryad.8w9ghx3kf [40].

Authors' contributions. C.A.S., S.A. and R.J.H. participated in study design; C.A.S. and R.J.H. performed mathematical analyses; C.A.S. and R.J.H. drafted the article; C.A.S., S.A. and R.J.H. revised the article critically; all authors gave final approval for publication of the maunuscript and agree to be held accountable for the content therein.

Competing interests. We declare we have no competing interests.

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