EFFECTS OF URBAN LANDSCAPES ON WILDLIFE BEHAVIOR, HEALTH, AND DISEASE: AUSTRALIAN FLYING FOXES AS A CASE STUDY

by

CECILIA ANNE SÁNCHEZ

(Under the Direction of Sonia Altizer)

ABSTRACT

Many wildlife species have established semi-permanent or year-round populations in urban landscapes. Animals in cities can often access predictable and abundant resources, but at the same time might experience lowered diet quality, exposure to toxicants and stressors, and greater pathogen transmission. The health of urban wildlife is relevant to humans, especially given risks of zoonotic pathogen transmission. The aim of this dissertation is to investigate how urban landscape features, including altered resources and exposure to toxicants, can change wildlife behavior, health, and infectious disease. I first synthesized the literature to quantify the extent to which urbanization affects four metrics of wildlife health; this meta-analysis demonstrated an overall small but significant negative effect of urbanization on wildlife health, driven by higher toxicant loads and greater parasitism by parasites transmitted through close contact. I next examined the individual and environmental predictors of foraging movements of wild flying foxes in a recently-established urban population in Adelaide, South Australia. This work showed that flying foxes were significantly more likely to forage at sites more

intensively used by humans, and that flying foxes in better body condition flew shorter distances each night, visited fewer foraging sites, and had smaller foraging areas. Using data from flying foxes captured in Adelaide and seven other locations across Australia, I next examined individual and environmental predictors of metal concentrations in bat fur, and associations between metals and bat parasitism. This study demonstrated that flying foxes captured at sites surrounded by greater human modification had higher metal concentrations in fur, and provided evidence for positive and negative relationships between ectoparasite abundance and metal concentrations. Lastly, I developed a mechanistic model of host-parasite dynamics to understand the interactive consequences of pathogens and toxicants on infection dynamics and population size of wildlife in an urbanizing landscape. Results suggested the extent of contaminated habitat across the landscape could enhance or reduce impacts of infection on host populations. Collectively, this dissertation provides evidence for multiple sublethal effects of urban landscapes for wildlife, and suggests important gaps for future work on the movement and survival consequences of toxicant exposure in wildlife.

INDEX WORDS: Australia; Body condition; Contaminants; Ecotoxicology; Fruit bat; GPS; Movement; Parasite; *Pteropus*, Urbanization

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TABLE OF CONTENTS

Page

LIST OF TABLES

Page

LIST OF FIGURES

Page

CHAPTER 1

INTRODUCTION AND LITERATURE REVIEW

We live in an increasingly urban world, with nearly 70% of the world's human population expected to live in urban areas by 2050 [1, 2]. Animal biodiversity typically declines with increasing urbanization because many species are excluded from urban habitats [3], but some wildlife can thrive. Both unintentionally and deliberately, humans provide resources that wildlife can use, such as plentiful and predictable food in the form of garbage or backyard feeders [4, 5], and shelter for roosting or reproduction [6, 7]. Urban areas can also serve as refuges from predators for small mammals and birds [8, 9]. From foxes in Switzerland [10] to otters in Singapore [11], many wildlife species are establishing semi-permanent or year-round populations in human-dominated landscapes [12-14]. For example, raccoons can flourish in urban areas due to ample food and higher survival [15].

However, advantages of urban living can come hand in hand with risks; as one example, concentrated resources can intensify intra- and interspecific competition [16, 17]. Urban wildlife also face exposure to toxicants such as pesticides, air pollutants, and polychlorinated biphenyls (PCBs) [18-20]; indeed, pollution has been implicated as a major driver of wildlife defaunation, especially in aquatic animals [21]. Anthropogenic stressors like noise and light pollution can also adversely affect health [22, 23]; for instance, tree frogs exposed to traffic noise had higher stress levels and reduced immune

function [24]. Other urban features such as cars and wind turbines can cause direct mortality [25, 26].

Importantly, urban landscapes can alter infectious disease dynamics for wildlife [27]. Wildlife populations may experience higher disease prevalence due to high host density or pathogen transmission from domestic animals [28]. Toxicants including heavy metals and PCBs can also reduce immunity, contributing to increased susceptibility to infectious disease [29]. For example, kestrels exposed to volatile organic compounds in air displayed reductions in delayed-type hypersensitivity response, a measure of cell-mediated immunity [30]. In turn, disease in urban wildlife can impact human health, because wildlife may transmit pathogens to humans [31, 32]. Wild boars have become more common in Berlin in recent decades as the animals seek anthropogenic food; the boars can carry *Leptospira* spp. and have been linked to at least one human leptospirosis case [33, 34].

My dissertation research investigated how urban landscape characteristics, including altered resources and exposure to toxicants, can affect wildlife behavior, health, and infectious disease. Four main goals underpinned this work: ***(1)*** analyze previous work to quantify the extent to which urbanization affects wildlife health metrics (Chapter 2), ***(2)*** assess the foraging behavior of wild flying foxes in a recently-established urban population in South Australia (Chapter 3), ***(3)*** compare metal concentrations in Australian flying foxes captured across a gradient of urbanization (Chapter 4), and ***(4)*** develop a mechanistic model to explore dual effects of toxicants and infection on the population and disease dynamics of a wildlife population in an urbanizing landscape (Chapter 5).

In Goal 1, I explored the net effects of urbanization on the health of wildlife populations (Chapter 2). Drawing upon data from more than 100 published studies, I co-led a phylogenetic meta-analysis to compare four health metrics (body condition, stress, disease, and toxicant loads) in urban and non-urban wildlife populations. This work is the first to quantify generalizable relationships between wildlife health and urbanization. The findings of this chapter suggested that overall, urbanization is harmful to wildlife health, but that results depended on the health metric and animal taxonomic group studied. A key finding was that urban wildlife populations had higher toxicant loads than non-urban populations across all taxonomic groups studied. This chapter also identified geographic areas and taxonomic groups that have received little research attention to date, and thus might be priorities for future investigation.

Chapters 3-5 of my dissertation focused on Australian flying foxes (*Pteropus* spp.). These bats are highly mobile and can respond flexibly to spatiotemporal changes in the availability of flowering and fruiting resources. In Australia, flying foxes increasingly reside in urban areas, owing to loss of natural habitat and planting of fruiting and flowering trees in cities and suburbs [35-37]. Though flying foxes play an important ecosystem role as pollinators and seed dispersers [38], human attitudes towards the bats can be negative, in part because flying foxes can transmit harmful pathogens to other animals and humans. In Australia, flying foxes can carry Hendra virus, a pathogen that "spills over" from bats to horses, and occasionally from horses to humans; infection does not appear harmful to flying foxes, but has high case fatality rates in horses and humans [39]. Poor nutrition has been implicated as a factor in Hendra virus infection in flying foxes [40, 41], and given that urban and agricultural resources used by bats might be less

nutritious than native vegetation [42], it is important to understand how flying foxes use urban landscapes and how these landscapes can affect their health.

In Chapter 3, I described a multi-year study of the foraging movements of grey-headed flying foxes (*P. poliocephalus*) in a recently-established urban population in Adelaide, South Australia. I used global positioning system technology to track movements of *P. poliocephalus* during winter and summer periods, and examined relationships between foraging metrics and individual and environmental predictors (as part of Goal 2). This work revealed that body condition was a key predictor of movement and that Adelaide flying foxes foraged mostly in human-dominated habitats, possibly owing to the close proximity of resources in these areas.

Given that the meta-analysis in Chapter 2 demonstrated differences in toxicant loads between urban and non-urban wildlife populations, in Goal 3 I used data from flying foxes captured in Adelaide and seven other sites across Australia to test if human land use surrounding flying capture sites predicted exposure to metals (Chapter 4). I analyzed fur samples from three flying fox species for 13 metal concentrations, and also assessed several measures of parasitism. A key finding in this chapter was that bats captured at sites with greater human impact had overall higher metal concentrations in fur. I found evidence of positive and negative relationships between ectoparasite abundance and metal concentrations, suggesting multiple causal mechanisms.

In Chapter 5, I extended my work on metal exposure in flying foxes by developing a mechanistic model to explore how toxicants and infectious disease could interact to affect a wildlife population (Goal 4). Model results suggested that toxicants that have little effect on population size in the absence of infection can severely depress

population size in the presence of infection when the majority of landscape is contaminated by toxicants. Impacts on population size were more severe when toxicants had a high cost to dispersal.

Collectively, this dissertation provides evidence for multiple sublethal health effects of urban living for wildlife that otherwise appear to acclimate to urban habitats. Across diverse animal taxa and within the focal study species of flying foxes, I found support for higher toxicant loads in urban wildlife. My work demonstrates the importance of considering multiple aspects of health, including toxicants, parasitism, body condition, and movement, when assessing urban impacts on wildlife. Model exploration shows that multiple stressors operating together can substantially lower population viability, intensify animal disease risks, and potentially increase human exposure to zoonotic pathogens transmitted by wildlife.

CHAPTER 2

CITY SICKER? A META-ANALYSIS OF WILDLIFE HEALTH AND URBANIZATION[1]

[1] Murray MH*, Sánchez CA*, Becker DJ, Byers KA, Worley-Tonks KEL, Craft ME. Accepted by *Frontiers in Ecology and the Environment*. * = equal first authorship. Reprinted here with permission of the publisher.

ABSTRACT

Urban development can alter resource availability, land use, and community composition, which, in turn, influences wildlife health. Generalizable relationships between wildlife health and urbanization have yet to be quantified and could vary across different measures of health and among species. We present a phylogenetic meta-analysis of 516 comparisons of the toxicant loads, parasitism, body condition, or stress of urban and non-urban wildlife populations reported in 106 studies spanning 81 species in 30 countries. We found a small but significant negative relationship between urbanization and wildlife health, driven by considerably higher toxicant loads and greater parasite abundance, greater parasite diversity, and/or greater likelihood of infection by parasites transmitted through close contact. Invertebrates and amphibians were particularly affected, with urban populations having higher toxicant loads and greater physiological stress than their non-urban counterparts. We also found strong geographic and taxonomic bias in research effort, highlighting future research needs. Our results suggest that some types of health risks are more pronounced for wildlife in urban areas, which could have important implications for conservation.

IN A NUTSHELL:

- We examined the relationships between urbanization and four aspects of wildlife health: exposure to toxic substances, parasite infection, body condition, and stress
- Our analysis of multiple studies found that, overall, urbanization is harmful to wildlife health

- Urban wildlife species are exposed to more toxic substances and are at greater risk of direct transmission of parasites as compared with non-urban wildlife
- Only a small number of urban wildlife studies focus on amphibians, reptiles, or invertebrates, or in locations outside of Europe and North America
- Future research should focus on less represented wildlife species and locations, should measure several aspects of health, and aim to identify the consequences to wildlife health of exposure to toxic substances

INTRODUCTION

Urban areas are rapidly expanding worldwide, and this growth has widespread consequences for wildlife. Urban wildlife species must cope with different conditions than their counterparts in non-urban areas; these include altered resource availability, warmer temperatures, habitat fragmentation, and pollution (Figure 2.1; [1]). As compared with non-urban areas, cities are associated with increased population densities of wildlife species [43]; greater frequency and intensity of human disturbance [44]; and altered community assemblages including humans and invasive, introduced, and domestic species [45]. These differences affect wildlife physiology, behavior, and health [46].

Wildlife in cities can suffer ill effects from exposure to toxicants (eg pesticides, heavy metals, persistent organic pollutants); for example, fish exposed to municipal and industrial wastewater in China had poorer body condition [47]. Toxicants can also increase susceptibility to infection [48]. Human-induced landscape changes, such as habitat fragmentation and patchy food distribution, can promote animal aggregation by limiting dispersal or attracting animals to shared food sources. This aggregation may increase the spread of parasites transmitted through close contact [49]; parasite deposition on soil, water, or artificial feeders [50]; and stress through inter- and intraspecific competition [51]. Urban populations can also exhibit greater chronic stress due to disturbances associated with urban development [52].

Yet some species can thrive in urban areas. Reliable food in urban habitats can improve body condition [53], and some urban populations exhibit lower baseline stress levels than rural conspecifics, in part due to higher resource availability [54]. Changes in behavior and community composition associated with urbanization can also lead to lower

parasite prevalence. For instance, urban carnivores like red foxes (*Vulpes vulpes*) can have smaller numbers of endoparasites, such as the tapeworm *Echinococcus multilocularis*, due to a switch in diet from intermediate rodent hosts to anthropogenic food [55]. Ectoparasites such as ticks can also be less prevalent among urban wildlife, potentially due to changes in habitat and exposure [56].

Because urbanization can generate positive and negative health effects that may be host-, parasite-, or region-specific, predicting the overall effect of urbanization on wildlife health is challenging. Although a growing body of literature has documented the changes in wildlife health that occur with urbanization [32], analyses of the overall effects of urbanization on wildlife health and how these differ across taxonomic groups and health metrics are lacking. Differences in scale between studies may also obscure patterns; for example, some mechanisms act at local scales (resource availability), whereas others extend beyond city limits (noise and light pollution). Understanding net effects across studies could facilitate predictions about where conservation concerns could arise and where management of wildlife or habitats will be needed.

To address this knowledge gap, we performed a phylogenetic meta-analysis (ie accounting for phylogenetic relationships; [57]) of 516 wildlife health records from 106 studies comparing health metrics between urban and non-urban populations. We chose four metrics to broadly represent health: two direct health outcomes (body condition and parasitism) and two physiological changes linked to health consequences (stress and toxicant loads in tissues). Because we were interested both in the overall effect of urbanization on wildlife health and in drivers of variability, we considered how host and parasite traits, study location and methodology, and degree of anthropogenic

development influence observed outcomes. Because we anticipated bias toward studies demonstrating poorer health outcomes in more urban areas, we also analyzed publication bias. Finally, we identified future research directions and potential effects of urbanization on biodiversity and conservation.

META-ANALYSIS

We identified 7541 published studies on urban wildlife health using a systematic search ([58]; Appendix A Figure S2.1). Of these, 106 met our previously defined inclusion criteria by being field studies of sufficient sample size ($n > 4$) that compared body condition, physiological stress levels, parasitism, or tissue toxicant concentrations between urban and non-urban populations of the same wildlife species. We considered including immune function but ultimately did not do so due to variations in methodology among the papers.

For each comparison of the same measure of health between urban and non-urban populations of the same species, which we defined as an individual record, we extracted and documented the host species, health metric assessed (body condition, stress, parasitism, or toxicants), and study location (study coordinates if provided, or centroid of a named location). We also extracted test statistics (odds ratios, R^2, χ^2, F), directionality of the association between urbanization and health, P value, and sample size. We converted test statistics into the correlation-based r as our standardized effect size [59]. If statistics were not reported, we calculated odds ratios, Cohen's d, or used the P value and sample size to obtain r [60]. We assigned negative values to r when health was lower for urban wildlife (poorer body condition, greater parasitism, higher baseline stress, lower

induced stress response, higher toxicant concentrations; Table 2.1) and converted *r* into Fisher's *Z* (*Zr*) as a normalizing transformation using the R package *metafor* [61, 62]. Studies not reporting sample size or effect direction and studies pooling multiple species were excluded. For descriptive purposes, we defined effect sizes as significantly different from zero if their back-transformed 95% confidence intervals (CIs) did not cross zero.

Health metric methodology

We further divided health metrics based on original study methodology (Table 2.1). We classified whether *body condition* was measured using qualitative scores, raw quantitative measures, or size-adjusted quantitative measures [63]. We categorized *stress* measures as glucocorticoid concentrations, heterophil-to-lymphocyte ratios (baseline or in response to a stressor), or other measures (eg oxidative damage, blood glucose). We recorded whether the *parasitism* measure was infection status (binary variable), infection intensity (parasite load), or parasite richness (number of parasite species). Finally, we grouped *toxicants* into metals (eg cadmium, lead, mercury) or non-metals (eg pesticides, polychlorinated biphenyls).

Wildlife and parasite traits

We classified wildlife species into five *taxonomic groups*: herpetofauna (amphibians and reptiles), birds, fish, invertebrates, and mammals. We delineated whether a species' life history is primarily terrestrial or aquatic using the primary literature or Animal Diversity Web (http://animaldiversity.org). For parasites, we recorded parasite type as microparasites (bacteria, viruses, fungi, and protozoa) or macroparasites (helminths and

ectoparasites); we created two categories because of low group sample sizes. We used the Global Mammal Parasite Database to classify parasite transmission route as close contact (transmitted directly from one individual to another), non-close contact (eg environmental contamination), vector transmission (eg insect vectors), intermediate hosts (eg consuming infected prey), or via multiple routes (each as a binary covariate; [64]).

Spatial analysis

We estimated urban development surrounding study sites using global terrestrial human footprint maps (GHF) in QGIS [65, 66]. The GHF dataset combines population density and anthropogenic development into a standardized score (0–50), with scores >10 indicating built environments. We extracted GHF values in raster cells surrounding urban and non-urban study sites, at either the study coordinates or the centroid of a named location. We calculated the average GHF value within 1-km and 10-km buffers to measure urbanization at the local scale and account for landscape context surrounding the site. If a study was performed along an urbanization gradient, we used the GHF values at the most and least urban sites; if a study had multiple urban and non-urban replicates, we used the average GHF values. Using these scores, we calculated the mean GHF score across the urban and non-urban sites (mean urbanization) and subtracted the GHF score of the most urbanized sites from the least urbanized sites (difference in urbanization) for each study. The GHF score is available for 1993 and 2009; we used the GHF value closest to the study year. We subtracted the average score at study sites in 1993 from the score in 2009 as a measure of change in GHF over time (change in urbanization). We recorded gross domestic product (GDP) and average GHF score of the study country to

account for differences in environmental policies in high- versus low-income countries. Using QGIS, we also measured the inter-site distance between urban and non-urban study sites to test whether health differences were stronger with greater distance between study populations.

Statistical analysis

We used a hierarchical random-effects model (REM) accounting for phylogenetic dependence of individual species relatedness and multiple records within each study to estimate the size and strength of the overall relationship between urbanization and wildlife health [60]. To first identify the primary predictors of effect size across our full dataset (n = 516 records), we fit a set of mixed-effect models (MEMs) considering taxonomic group, health metric, species life history, and all two-way interactions.

Given the results of this analysis (see below), we stratified our data by health metric. We used an MEM to test whether effect size differed among the health metrics (toxicant concentrations, n = 189; body condition, n = 60; parasitism, n = 194; stress, n = 73). We fit sets of MEMs separately to each health metric dataset to assess whether urbanization–wildlife-health relationships vary by wildlife species traits (taxonomic group, life history), parasite traits (parasite type, transmission route), health metric methodology, and study country metrics (mean country GHF, log GDP).

In a third analysis applied to data where site location was provided (n = 302 records, 81% of studies), we fit a set of MEMs with metrics of urbanization intensity (mean urbanization and difference in urbanization across sites, change in urbanization between time periods) and inter-site distance as moderators of effect size. We also

included interaction terms between these urbanization metrics and taxonomic group and health metric. All models in these three analyses included the same random effects (each record was nested within its associated study and similarity between closely related species was accounted for by structuring species within a phylogenetic correlation matrix) and included weighting by sampling variance using the *metafor* package.

We tested for evidence of publication bias, which includes preferential publication of significant over non-significant results or studies with a small effect size [67]. We generated funnel plots of effect sizes against standard errors to visualize potential bias for the full dataset and each health metric subset; low bias is expected when effects with high precision remain close to the mean and effects with low precision are spread symmetrically from the mean [68]. For each of the funnel plots, we tested for asymmetry using rank correlation tests [69]. We then used the trim-and-fill method with an R0+ estimator, which estimates the number of missing records based on the spread of effect sizes relative to the overall mean, to test whether the number of records missing due to publication bias differed from zero [70]. We adjusted *P* values from these two tests with the Benjamini and Hochberg correction to adjust for multiple comparisons [71]. Statistical analyses are explained in greater detail in Appendix A.

RESULTS

Dataset description

Our dataset included 516 records from 106 published studies quantifying wildlife health (DRYAD repository: https://doi.org/10.5061/dryad.b74d971). Studies were conducted in 30 countries on all six continents containing cities (Figure 2.2), with more than one-third

of studies being conducted in the US (n = 38, 36%). GHF scores varied across urban sites (minimum GHF = 0.7, maximum GHF = 24.9). The parasitism and toxicant datasets each comprised about one-third of all records (37.6% and 36.6%, respectively), while the stress and body condition datasets were less well represented (14.1% and 11.6%, respectively) (Figure 2.3). The predominant health metrics used differed by taxonomic group (χ^2 = 274.49, P < 0.001), with parasitism dominated by mammals (72%), stress and body condition by birds (64% and 45%, respectively), and toxicants by fish (37%), birds (29%), and mammals (27%) (Figure 2.3). Our search identified no fish/parasitism records, mammal/stress records, or invertebrate/body condition records.

Research effort for toxicants and parasitism showed greater growth over time than for body condition and stress (χ^2 = 21.46, P < 0.001; Appendix A Figure S2.2; Table S2.1), and research effort for birds, fish, and mammals showed greater growth over time than for herpetofauna and invertebrates (χ^2 = 24.98, P < 0.001; Appendix A Figure S2.2; Table S2.1).

Relationships between urbanization and wildlife health

Across all records, 60% (n = 311) reported a negative relationship between urbanization and health (r < 0), 37% (n = 190) reported a positive relationship (r > 0), and 3% (n = 15) reported true null effects (r = 0). The toxicant dataset was dominated by records reporting negative relationships, while the other health datasets had high variation in effect direction (Figure 2.3). Within records reporting a negative urbanization–health relationship, the proportions of significant (ie 95% CIs per effect size do not cross zero) and non-significant records were approximately equal (48% significant, 52% non-

significant). Within records reporting a positive relationship, a larger proportion was non-significant (46% significant, 54% non-significant).

Our REM identified significant heterogeneity in effect sizes across the entire dataset (I^2 = 98.06, Q_{515} = 12644, P < 0.001), and an overall small but significant negative correlation between urbanization and wildlife health (r = –0.16, z = 2.09, P = 0.04). Comparison among alternative models showed that the interaction between taxonomic group and health metric best predicted effect size (w_i = 1, R^2 = 0.29, Appendix A Table S2.2; Q_{16} = 71.43, P < 0.001; Figure 2.3). Adjusting for multiple comparisons, this model showed strong negative relationships between urbanization and health for toxicants in herpetofauna (r = –0.82, 95% CI = –0.94 to –0.53), toxicants in birds (r = –0.36, 95% CI = –0.55 to –0.14), toxicants in invertebrates (r = –0.92, 95% CI = –0.97 to –0.80), and stress in invertebrates (r = –0.88, 95% CI = –0.96 to –0.71).

Moderators of effect size per health metric

Because health metrics had greater predictive power than taxonomic group (Appendix A Table S2.2), we stratified our data by health metric for more detailed model comparisons. Within this analysis, parasite transmission route and animal taxonomic group were the top predictors of how urbanization correlates with health (Appendix A Table S2.2). For the parasitism dataset, the most parsimonious MEM contained whether parasites were transmitted through close contact or another route (ΔAICc = 0.80, w_i = 0.11, Q_1 = 11.9, P < 0.001). Effect sizes were most negative for parasites spread through close contact (β = –0.3, z = 3.45, P < 0.001, R^2 = 0.25; Figure 2.4).

For toxicant and stress datasets, both top MEMs mirrored our first set of analyses, with the best model containing taxonomic group (w_i = 0.68 and w_i = 0.78, respectively); no other models were competitive (Appendix A Table S2.2). For the toxicant dataset, this MEM explained 21% of effect size variation, and predicted effect size to be most negative for herpetofauna and invertebrates (Q_4 = 9.22, P = 0.06; Figure 2.4). For the stress dataset, this MEM explained 55% of variation and predicted the most negative correlations for invertebrates (Q_3 = 35.68, P < 0.001; Figure 2.4). No covariates were more competitive than an intercept-only model for the body condition dataset (Appendix A Table S2.2). A MEM with life history suggested terrestrial wildlife show slightly more positive body condition relationships with urbanization than aquatic wildlife (ΔAICc = 1.81, w_i = 0.11), but this was not statistically significant (Q_1 = 1.27, P = 0.26; Figure 2.4).

Does intensity of urbanization predict effect size?

The mean urbanization score (GHF) within 1 km per study and its interactions with health metrics explained the most variation in effect size for studies providing study locations (1 km: w_i = 0.62, R^2 = 37%; Q_7 = 35.86, P < 0.0001; Appendix A Table S2.2). Post-hoc analysis showed this association was only significant for parasitism; more urban regions showed more positive effect sizes for parasitism (β = 0.05, P < 0.001) (Appendix A Figure S2.3). A model with inter-site distance and its interaction with health metrics received marginal support (ΔAICc = 4.05, w_i = 0.08, R^2 = 0.29; Q_7 = 21.86, P < 0.01). As distance between contrasting sites increased, effect size became more negative for toxicant outcomes (β = –0.02), whereas all other health metrics showed non-significant positive relationships with distance (Appendix A Figure S2.3).

Publication bias

We found evidence of publication bias depending on health metric (Appendix A Figure S2.4). Funnel plots suggested bias in effect size reporting, which rank correlation tests confirmed for the full dataset (toward negative correlations: $z = –4.79$, $P < 0.001$) and body condition dataset (toward positive correlations: $z = 3.89$, $P < 0.001$). We did not find significant publication bias for toxicant, parasitism, or stress effect sizes (toxicants: $z = 1.71$, $P = 0.12$; parasitism: $z = –1.62$, $P = 0.12$; stress: $z = –1.54$, $P = 0.12$). Trim-and-fill analyses suggested the number of missing studies did not differ from zero for most datasets; for stress data, this analysis suggested that six (± 6) effect sizes greater than the mean were missing ($P = 0.04$).

DISCUSSION

Does urbanization pose health risks for wildlife?

Identifying contexts in which urbanization influences wildlife health is critical for understanding urban adaptation, human–wildlife conflict, and biodiversity conservation in cities. Our meta-analysis suggests an overall negative relationship between urbanization and wildlife health, mainly driven by considerably higher toxicant loads and greater parasite abundance, greater parasite diversity, and/or greater likelihood of infection by parasites transmitted through close contact. We found no significant difference in body condition and stress levels with urbanization. For all health metrics, the direction and magnitude of effect sizes varied greatly by taxonomic group. Our findings highlight the complexity of urbanization's effects on wildlife health.

Across all wildlife taxa, toxicant loads were significantly higher in urban animals than in their non-urban conspecifics. Although it is not surprising that urban wildlife species are subject to greater exposure to heavy metals, organic compounds, and pesticides associated with industrial and anthropogenic features, such as roads and managed lawns [72], our results demonstrate that this exposure results in uptake into wildlife tissue. For instance, urban predators like bobcats (*Lynx rufus*) can be exposed to anticoagulant rodenticides from consuming contaminated prey [73]. Toxic metals like cadmium, lead, and mercury can bioaccumulate in tissues through food consumption and are more abundant in urban populations, as seen in common blackbirds (*Turdus merula*; [74]) and common perch (*Perca fluviatilis*; [75]). Although toxicant exposure can have downstream effects on wildlife health, including abnormal development, reproduction, and immune function [48, 76], the biological relevance of relatively higher toxicant concentrations is less clear, especially across species.

We observed a higher likelihood of infection by parasites transmitted through close contact, along with greater parasite abundance and diversity, in urban as compared to non-urban populations, perhaps because some urban-adapted hosts live at higher local densities due to abundant and patchily distributed food resources. For instance, urban raccoon populations can reach high densities, potentially promoting rabies transmission [77]. Conversely, parasite transmission by routes other than close contact (ie transmitted via vectors, trophic transmission, or environmental contamination) was lower in urban areas, perhaps due to shifts in habitat availability or host community structure. For instance, the prevalence of *Campylobacter* spp in house crows (*Corvus splendens*) in Tanzania was higher in rural villages, where infections are more common in poultry than

in urban villages [78]. Predicting any changes in parasitism with urbanization could therefore depend on parasite life history.

Our data showed no consistent differences in wildlife body condition with urbanization. Increased access to resources could buffer populations from negative effects of urbanization. For example, white-footed tamarins (*Saguinus leucopus*; [53]) in urban areas had higher size-adjusted mass than their rural counterparts but also had higher cholesterol levels, presumably from food provisioning with cholesterol-rich anthropogenic food. In contrast, rufous-collared sparrows (*Zonotrichia capensis*) had lower body mass in urban areas, possibly due to higher intraspecific competition [51]. Given that no model performed better than the null, there appear to be contrasting effects of urbanization on wildlife body condition.

As with body condition, we did not find significant differences in stress levels between urban and non-urban populations. Cities may not present additional stressors beyond those experienced in rural settings; alternatively, variation in stress outcomes could reflect difficulty in interpreting stress responses across methods (eg heterophil-to-lymphocyte ratios or glucocorticoid concentrations) or sampling times (eg time of day, reproductive season). Furthermore, chronically stressed individuals may not show increased stress measures, which complicates interpretation of results [79]. For instance, urban ornate tree lizards (*Urosaurus ornatus*) had lower stress responses relative to their non-urban counterparts [54], suggesting chronic stress.

Despite an overall negative relationship between urbanization and wildlife health, we also found ample support for positive health effects. Positive (albeit non-significant) effects were found for four health metric–taxonomic group pairs: body condition and

mammals, parasitism and mammals, parasitism and birds, and stress and herpetofauna. Given the bias toward negative correlations in the dataset, the benefits of urbanization for wildlife deserve careful consideration.

Future directions

Our results cannot tease apart the causal mechanisms for observed relationships between urbanization and health, but they do suggest several ways in which future studies could adopt more mechanistic approaches. First, we recommend that studies examine multiple health metrics simultaneously; <20% of studies in our analysis did so. This would identify mechanisms by which toxicant exposure impairs health, such as through altered immune function, gene expression, or organ function. Second, studies of urban wildlife health should quantify urbanization using landscape metrics relevant to the focal wildlife population [80]. For instance, the degree of urbanization can be classified according to land cover, human population density, or a combination of several metrics [81]. Providing details of study location coordinates and how urbanization was quantified will also facilitate cross-study comparisons in towns and cities of different sizes. Finally, researchers should consider how species traits (eg generalist versus specialist diet, social system, life span) could influence health outcomes. Previous studies have attempted to predict traits associated with urban adaptation [82], which could aid in predicting health outcomes in urban areas.

Echoing previous work [83], we found that most published urban wildlife research has been conducted in cities in North America and Europe (Figure 2.2). However, many rapidly urbanizing areas are in low- and middle-income countries near

global biodiversity hotspots [84], where relationships between wildlife health and urbanization could vary with climatic, ecological, and socioeconomic differences. Research in South America, Africa, Asia, Australia, and countries with intense urban development (eg India; Figure 2.2) would improve global inferences. Although the studies in our meta-analysis were conducted at sites that ranged widely in their degree of human development (ie human footprint values), it remains unclear whether urban wildlife studies to date reflect an unbiased representation of global urbanization intensity, and how other types of land use (eg agriculture) affect wildlife health relative to urbanization. Furthermore, we recognize the potential for spatial error and change over time when using point locations to estimate local human footprint from a global dataset [85]. When we accounted for the degree of urban development, we found that habitats with greater urban development were associated with lower parasitism (with all transmission modes combined due to smaller sample size), suggesting that the transmission of some types of parasites may be interrupted in highly urban areas.

Finally, our meta-analysis revealed taxonomic biases for each health metric; for example, most toxicant studies focused on fish, whereas most parasitism studies focused on mammals. These patterns could be driven by their relevance to human health; aquatic systems receive wastewater outputs from human activity, and most urban wild mammals share parasites with domestic animals and people [86]. Our results may be biased in terms of wildlife species representation; species that experience severe health threats in cities may be rare and therefore less present to be sampled. Even in populations that have persisted in urban settings, negative effects could be masked; for instance, stress responses might dull in response to sustained threats [87]. When assessing impacts of

urbanization, researchers should attempt to sample a broad suite of species and consider whether any species lack representation because they are rare or are excluded from urban areas.

Implications for conservation and policy

Urban living appears to pose several health threats for wildlife, especially through increased exposure to toxicants like heavy metals and pesticides. Invertebrates and herpetofauna seem especially vulnerable to toxicant exposure in urban areas, which has implications for conservation. For example, many amphibian and reptile populations are already in decline due to fungal diseases, such as chytridiomycosis [88] and snake fungal disease [89]. Observational studies have linked greater loads of heavy metals and pesticides with increased susceptibility to infection in toads and frogs [90], highlighting the threat posed by higher toxicant loads to urban wildlife.

Urban invertebrates appear especially vulnerable to health risks in urban areas because they exhibited greater increases in toxicant loads and stress levels than non-urban invertebrates and other wildlife taxa. However, we acknowledge that these results are based on a small sample. Increased toxicant loads and chronic stress can suppress immune function, potentially increasing infection risk [48, 76]. For the three bee species in our study, this could have important consequences for colony health [91] and urban pollinator conservation, as urban honeybees can be subject to higher concentrations of pesticides and greater oxidative stress [92]. We found few studies on urban invertebrate and herpetofauna health; we therefore encourage researchers to examine the health impacts of urbanization and traits conferring sensitivity in these taxa. Beyond wildlife

conservation, our results suggest that the risks of toxicant exposure and the transmission of some wildlife parasites may be higher for domestic animals and the public in urban relative to non-urban settings. Future research on urban wildlife health will be critical for maintaining urban biodiversity and public health in our rapidly urbanizing world.

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Table 2.1. Description of health metric methodology used by studies in the meta-analysis and how directions of health effects were assessed

Health metric	Methods of measurement	Direction of association between urbanization and health (health effect)
Toxicants	● Metal concentrations (eg lead, copper, zinc) ● Non-metal concentrations (eg organochlorine pesticides, polychlorinated biphenyls)	Positive for decreased measures; negative for increased measures
Parasitism	● Infection status (ie infected or uninfected) ● Infection intensity (ie number of parasites per infected individual) ● Parasite richness (number of parasite species)	Positive for decreased measures; negative for increased measures
Body condition	● Qualitative scores (eg subjective fat score) ● Raw quantitative measure (eg body mass, length) ● Size-adjusted quantitative measure (eg residuals of mass ~ length regression)	Positive for increased measures; negative for decreased measures
Stress	● Glucocorticoid concentrations (higher levels indicate more stress) ● Heterophil:lymphocyte ratios (baseline values or in response to a stressor) ● Other measure	Positive for decreased baseline measures; negative for decreased induced measures (ie in response to a stressor)

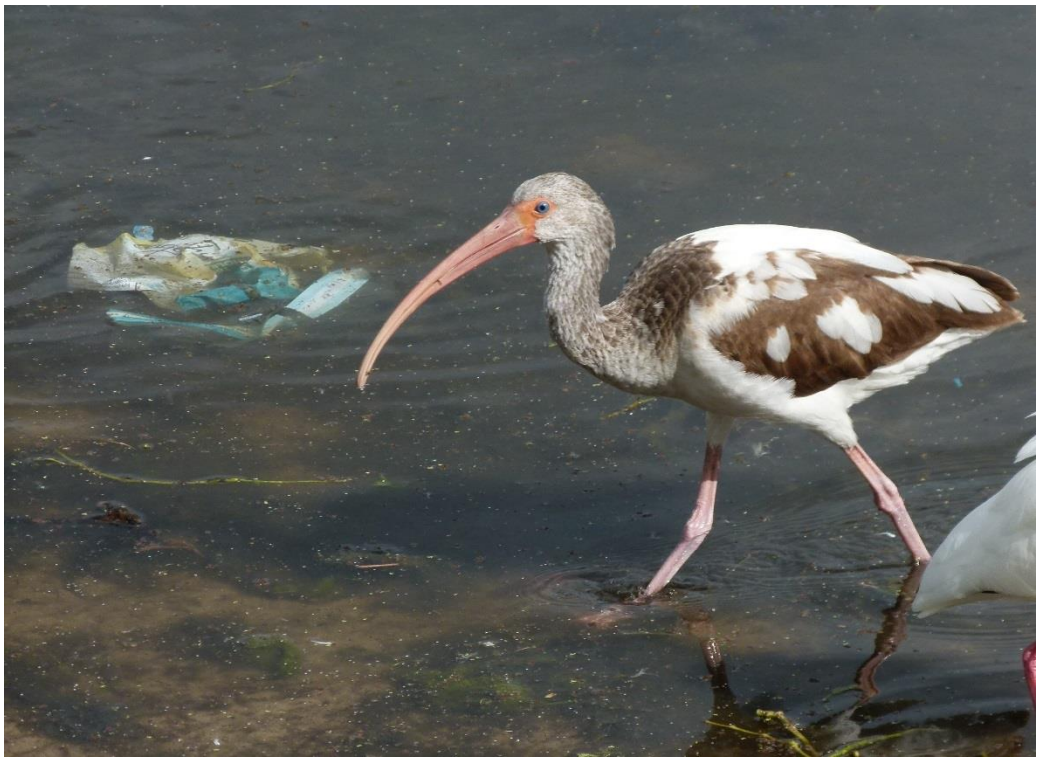

Figure 2.1. Urban wildlife species can be more likely to be exposed to toxicants via foraging in polluted environments; here, an American white ibis (*Eudocimus albus*) forages in an urban pond containing litter.

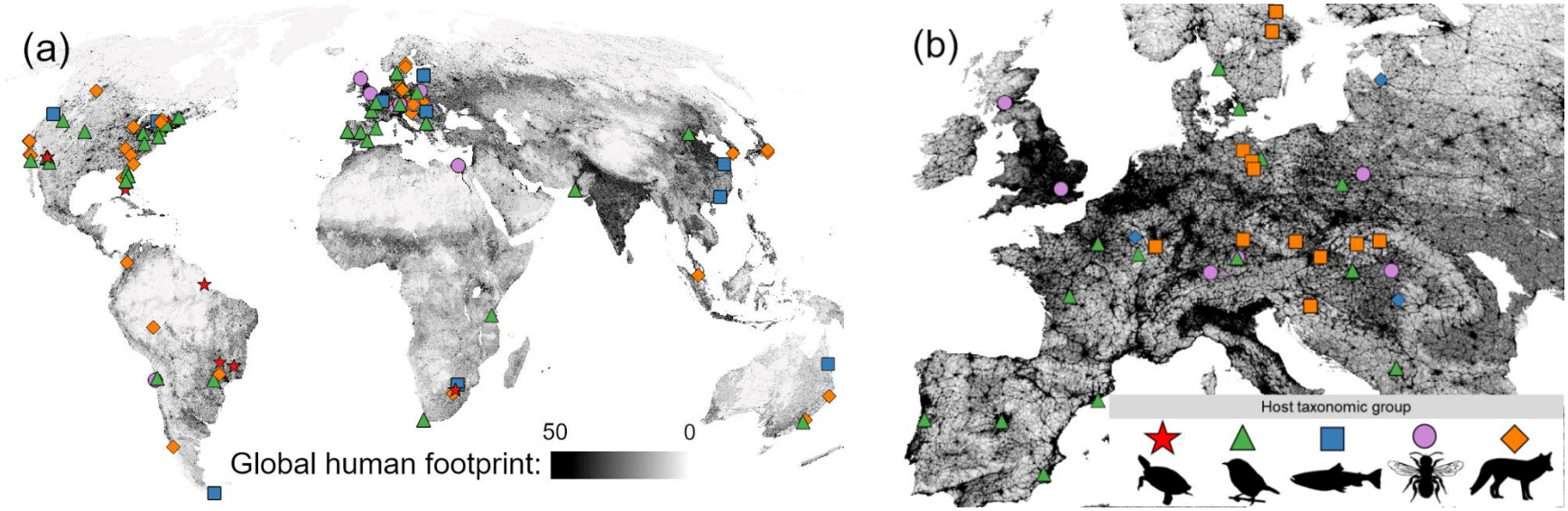

Figure 2.2. The (a) global and (b) European distribution of 106 studies comparing the health of urban and non-urban wildlife populations in 30 countries. For clarity, each study is represented once as the centroid of all within-study locations. Study locations are based on wildlife taxa (herpetofauna = red stars, birds = green triangles, fish = blue squares, invertebrates = purple circles, mammals = orange diamonds). Base map shows the 2009 global terrestrial human footprint map, in which darker areas indicate more urban development.

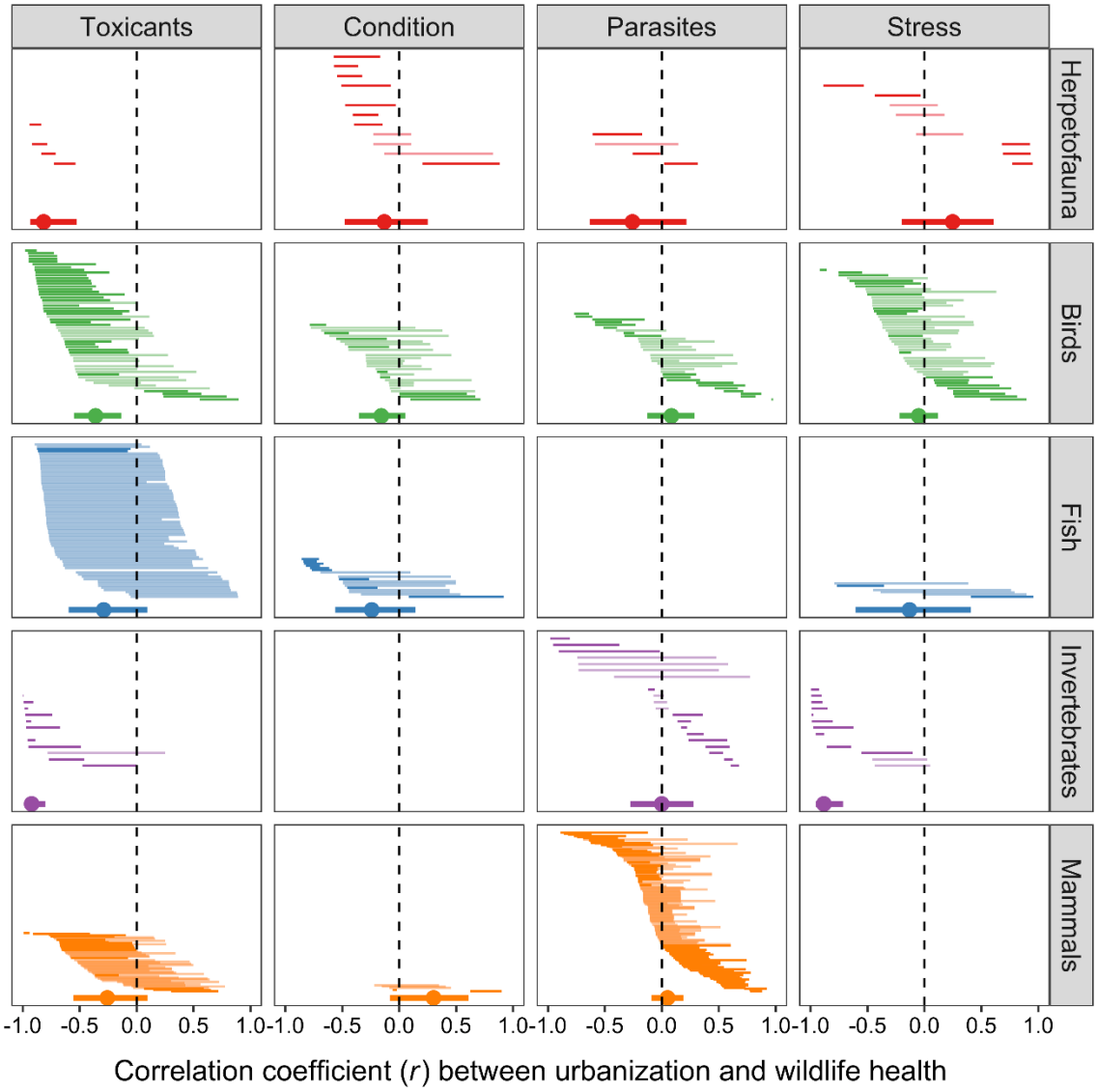

Figure 2.3. Range and grand means from random effects models (REMs) for the correlations between wildlife health and urbanization. The columns represent results stratified by health metric, while the rows represent results stratified by animal taxonomic group. Thin lines represent 95% confidence intervals (CIs) for effect sizes of individual records; thick circles and lines (at the bottom of each panel) represent REM estimates (uncorrected for publication bias). CIs for individual records that cross the dashed line (r = 0, no relationship between health and urbanization) are partially transparent.

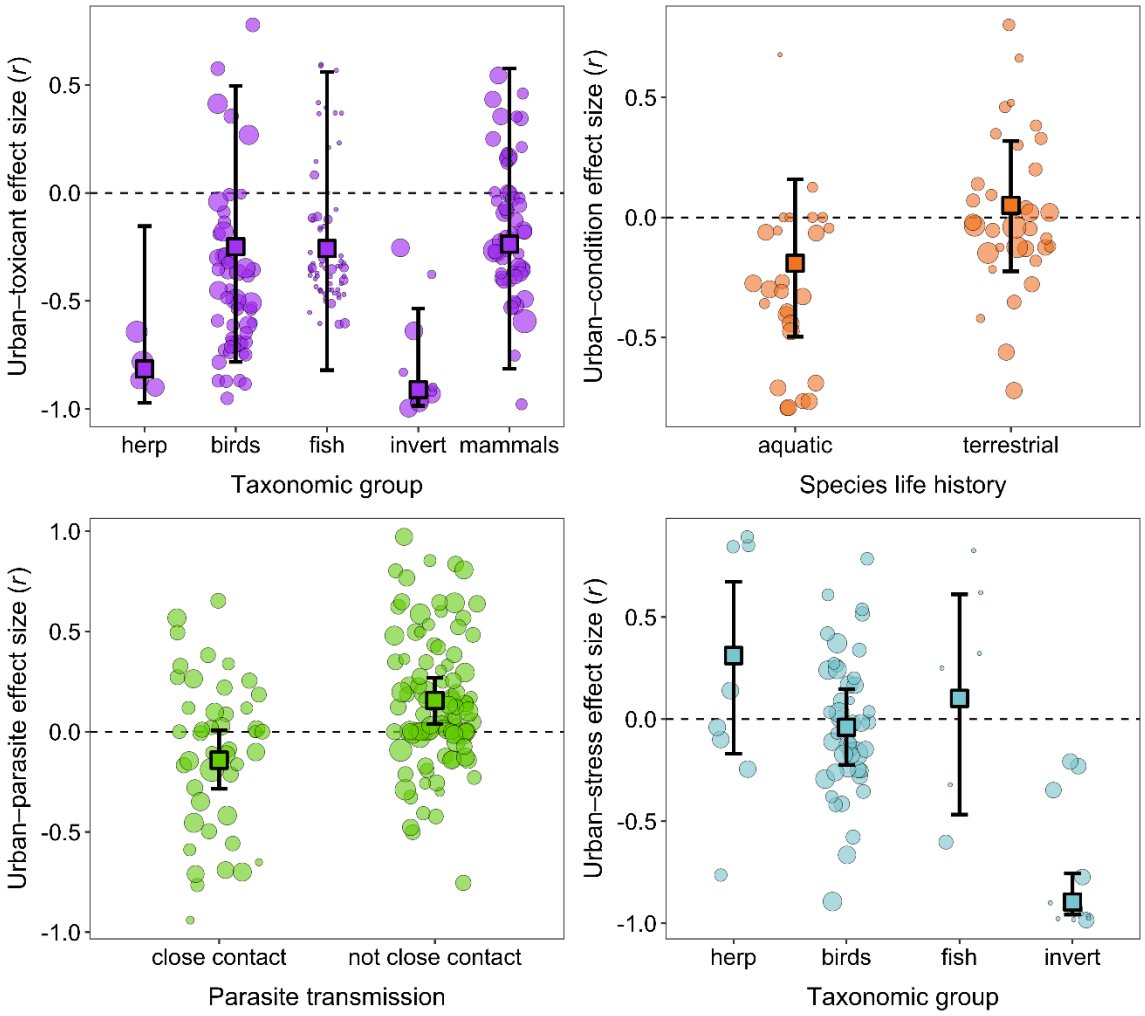

Figure 2.4. Most supported predictors (ΔAICc = 0) of effect size for each health metric dataset (clockwise from top left: toxicants, body condition, stress, parasitism). Squares show the predicted means and 95% CIs from the respective mixed-effects models, and circles show individual records scaled by their sample size. The dashed line shows no relationship between health and urbanization ($r = 0$).

CHAPTER 3

BODY CONDITION PREDICTS GREY-HEADED FLYING FOX (PTEROPUS POLIOCEPHALUS) FORAGING MOVEMENTS IN AN URBAN LANDSCAPE[2]

[2] Sánchez CA*, Reardon TB, O'Leary M, van Weenen J, Altizer S, Boardman WSJ. Submitted to *Movement Ecology*.

ABSTRACT

Background. Food resources are a major driver of animal movement, and human-provided food in urban areas can alter wildlife foraging behavior. In recent decades, pteropodid fruit bats (flying foxes) have settled in urban areas to feed on fruiting and flowering trees planted by humans. Understanding the consequences of this shift towards urban foraging for bat movement and health is important for predicting future bat-human interactions and associated health risks. In this study, we examined the foraging behavior of Australian flying foxes to: 1) characterize bat movements in a newly-established urban population, 2) explore individual and environmental predictors of movement behavior, and 3) analyze the selection of foraging sites and food plants utilized.

Methods. We deployed lightweight GPS loggers to track the movements of 32 grey-headed flying foxes (*Pteropus poliocephalus)* captured in Adelaide, South Australia in 2016-2018. We calculated quantitative metrics including nightly distance traveled, foraging and core area, and number of foraging sites visited. We used regression models to analyze whether these foraging metrics were correlated with body condition, age, sex, daily temperature, and season. We ground-truthed feeding sites to identify plant species visited, and statistically examined the selection of foraging sites in relation to human land use.

Results. Bats in better body condition flew shorter distances each night, visited fewer foraging sites, and had smaller foraging areas. Male bats had longer nightly round-trip distances than females, and younger bats visited more foraging sites per night. Bats foraged more in urban residential and recreational sites than less disturbed sites; however,

we did not quantify nectar or fruit availability at foraging sites and so could not assess preference.

Conclusions. Our work suggests that the urban flying foxes in Adelaide foraged largely in human-dominated habitats, and that bats in better body condition made shorter, more efficient foraging flights. Understanding how animals move across and utilize resources within human-modified habitats is increasingly important for wildlife conservation and managing human-wildlife conflicts.

Keywords: Adelaide; fruit bat; GPS tracking; habitat selection; movement ecology; seasonality; urbanization

BACKGROUND

Many animals move to acquire food resources [93], with movements ranging from long-distance annual migrations [94] to daily travels in local environments [95]. At the individual level, greater foraging success often predicts improved body condition and energy reserves, translating to increased fitness. Both internal and environmental factors determine an animal's foraging movements [96]. For example, home ranges of female striped mice (*Rhabdomys pumilio*) increased with scarcity of food plants, fewer competitors, and larger mouse body mass, among other factors [97], while in European shags (*Phalacrocorax aristotelis*), juvenile birds spent more time foraging than adults, likely to compensate for poorer foraging ability [98].

Urbanization alters the quantity and distribution of resources available to wildlife. Many animal species disappear from cities altogether, but some wildlife can utilize human-provided resources and habitats found in urban areas [4, 5]. In terms of foraging movements, studies that span a diverse range of terrestrial mammals show that animals living in highly human-impacted environments often travel shorter distances in search of food, potentially driven by access to predictable resources [99]. Shorter movement distances in response to human-provided resources can also manifest at larger scales. For example, Eurasian blackcaps (*Sylvia atricapilla*) in Germany and Austria are increasingly wintering in Britain, rather than migrating a longer distance to overwinter in Spain, in response to supplemental feeding of birds in urban gardens [100].

Reduced movement could benefit wildlife by allowing them to allocate more resources towards growth, energy storage, or reproduction [101-103]. For instance, a study of African lesser bushbabies (*Galago moholi*) reported that urban bushbabies spent

less time moving, ate more anthropogenic food, and had higher body mass index values than did rural conspecifics [104]. Yet feeding on urban resources could be costly if those resources provide lower nutrient content than natural food sources, contain toxicants (e.g. pesticides, heavy metals), or stimulate harmful behavior (e.g. aggression in response to high aggregation) [4, 105, 106]. Decreased movement coupled with higher local densities in urban areas could also increase exposure to pathogens transmitted by fecal-oral routes or environmental contact, allowing infectious stages to accumulate in the animals' environment over time [107, 108].

Pteropodid fruit bats (flying foxes) as a group have exhibited changes in movement in response to anthropogenic resources. These highly mobile animals respond flexibly to changes in resource distribution and abundance, and in forested environments, typically show nomadic long-distance movements to track ephemeral flowering and fruiting resources [109, 110]. Flying foxes increasingly reside in urban areas and feed on urban and agricultural resources [35]. In Australia, this behavioral shift has been attributed largely to a combination of native habitat destruction, planting of (largely non-native) fruiting and flowering trees in cities that offer predictable food, and artificial watering [36, 37, 111-113]. Shifts in foraging behavior can affect bat health [35]. Human-provided food used by bats might be less nutritious than native vegetation [42]. Because poor nutrition might increase susceptibility to infection [40, 41], and because flying foxes can transmit viruses to humans, livestock, and pets [114], urban resources could influence the health of humans and other animals. Therefore, work exploring links between urban resources, bat foraging movements, and health is crucially needed [115-117].

We examined the foraging movements of Australian flying foxes in a recently-established urban population using global positioning system (GPS) technology. We collected data from grey-headed flying foxes (GHFF; *Pteropus poliocephalus)* captured in Adelaide, South Australia during two winter and two summer periods. The Adelaide flying fox population formed within the past decade and is located far to the west of the previous known *P. poliocephalus* distribution (Figure 3.1). Our study objectives were to 1) characterize bat movements across the novel Adelaide landscape, 2) explore individual and environmental predictors of movement, and 3) analyze the selection of foraging sites and food plants utilized. Given that flowers and fruits are more abundant and reliable in summer than winter [118], we expected bats to fly shorter distances each night and have smaller foraging areas in summer. We used multiple body condition indices as a proxy for overall health, and predicted that bats caught in the summer would be in better body condition owing to higher resource availability and reduced flight energetics. We expected sex differences in foraging metrics owing to size dimorphism [119] and different energy requirements (e.g. territory defense, lactation), but did not predict a direction for this difference given conflicting results in other studies [115, 120]. Based on previous work on urban flying foxes [111, 121], we expected that Adelaide bats would forage primarily in human-modified habitats. Finally, we expected that animals that forage over shorter distances might show greater body condition, if less energy is expended on movement, or if animals in better condition can better access and defend nearby resources.

METHODS

Study population and location

In winter 2010, ~500 GHFF formed a temporary camp in a suburban backyard in Adelaide, South Australia, growing to ~1200 individuals within a week (T.B. Reardon, personal observation). Adelaide is >600 km northwest of the previously known range of *P. poliocephalus* (Figure 3.1), and their 2010 range expansion might have been driven by a national shortage in their preferred flowering and fruiting plants [122]. Bats appeared to leave Adelaide (i.e. a camp could not be located) soon after the shortage was over but a camp was again observed in Adelaide in early 2011 when ~50 GHFF settled in the Adelaide Botanic Gardens. The population began to grow and after reaching ~400 individuals, was relocated to nearby Botanic Park (34°54'56.7"S, 138°36'24.7"E) by the Department for Environment and Water. Population counts using direct observation (aided by 10 x 42 mm binoculars) began in 2011 (Figure 3.1). The population has grown to more than 20,000 individuals at times, primarily through interstate immigration, with intermittent declines owing to mortality during extreme heat events as well as recent emigration during an apparent food shortage (J. van Weenen, personal observation).

Bat capture, logger attachment, and data collection

We captured 310 total *P. poliocephalus* by mist net in August (winter) and February (summer) periods during 2015-2018 as part of a larger study to characterize the health and behavior of the Adelaide flying fox population (W. Boardman, unpublished data). Bats were captured pre-dawn as they returned to the roost after foraging and placed in individual cotton bags. We transported the bats to the Adelaide Zoo Animal Health

Centre and anesthetized them with inhalant Isoflurane [123]. We recorded each bat's sex, weight to the nearest 1 g with a digital scale, and forearm length to the nearest 1 mm with vernier calipers. Age in years was estimated by a single person (W. Boardman) based on molar wear and coloration. Each bat was assigned a body condition score (BCS; from 1-5 with higher values indicating better condition) based on palpation of pectoral muscles and prominence of the sternum [124].

Following the protocol of de Jong [125], we attached 15g data loggers (e-obs GmbH, Munich, Germany) to 32 GHFF (Aug. 2016: n = 5; Feb. 2017: n = 9; Aug. 2017: n = 7; Feb. 2018: n = 11) to track their movements (Appendix B Figure S3.1). Only male and non-pregnant female bats weighing >600g were considered for logger deployment. The loggers are battery and solar powered and collect GPS, acceleration, altitude, speed, and heading data. Acceleration was recorded every 30 s on three axes throughout the night (~6pm-7am local time). The frequency of GPS fixes was acceleration-informed [126], such that fixes were collected more frequently during flight (every 30 s), and less frequently during rest or minor movement (every 45 min). The roost was visited daily to remotely download the previous night's data using a handheld e-obs base-station. We downloaded 1 – 14 consecutive nights of movement data from each logger (209 nights total).

Identification of foraging plants

To characterize bat diet breadth and preferences, we ground-truthed a subset of locations that we suspected were visited by bats. We visualized movement tracks in Google Earth, identified clusters of GPS fixes as potential foraging sites, and collected flower, leaf,

bark, and/or fruit samples for subsequent identification at the State Herbarium of South Australia. However, we were not able to quantify the food resources available at the time of foraging (e.g. nectar flow, fruit abundance and ripeness).

Calculation of foraging distance and area

All movement and statistical analyses were performed in the R computing environment v 3.4.3 [127]. The dataset was first trimmed so that only the first fix of each GPS burst was retained (bursts are useful for improving estimation of altitude, speed, and heading, which were not needed in this study). We calculated the number of hours each bat was tracked per night and subsequently excluded incomplete nights (< 8 hours of data). We calculated the nightly distance flown by each bat by summing the distance between successive GPS points [128]. When a bat began and ended its flight at the roost, we designated this a round-trip, and calculated the median and maximum round-trip distance for each bat. We excluded non–round-trip nights from further calculations because we believe this represented aberrant behavior (Additional file 2: calculation of foraging distance and area).

We estimated the area traversed by tracked GHFF using minimum convex polygons (MCPs). The foraging and core areas for each bat were calculated respectively as the areas of 95% and 50% MCPs constructed with the *adehabitatHR* package [129]. We restricted calculation of foraging and core areas to bats with at least three round-trips, conservatively assuming that small sample sizes would not accurately estimate area. We also constructed seasonal 95% MCPs for use in habitat selection analyses (described below).

Estimating number of foraging sites

In addition to visually identifying the largest clusters of GPS points in Google Earth for ground-truthing purposes (described above), we used a systematic protocol to identify foraging sites. We first filtered out all high-speed GPS fixes (ground speed >2 m/s), then generated a distance matrix for remaining points [130]. We identified low speed clusters (at least 6 low-speed points within 60m of each other), calculated the centroid of each [131, 132], and excluded clusters within 200m of the roost. We considered all remaining cluster centroids to be foraging sites. We calculated the nightly number of foraging sites used by each bat and the straight-line distance from each foraging site to the roost, and identified the most distant foraging site visited by each bat [133]. For our habitat selection analyses, we repeated this procedure to identify the location of foraging sites for each bat over all nights of its logger deployment, but used a more conservative definition of a foraging site (at least 12 low-speed points) to identify more heavily used sites.

Statistical analyses

We used regression models to assess the importance of individual and environmental predictors in explaining variation in nightly round-trip distance and nightly number of foraging sites. We fit generalized linear mixed models (GLMMs) with maximum likelihood, using a gamma distribution and log link for the distance model, and a Poisson distribution and log link for the number of sites model [134]. For both distance and number of sites, we set bat age, sex, body condition, season, maximum daily temperature, and hours tracked as fixed effects, and batID and date as random effects.

Maximum daily temperature data were downloaded from the Australian Government Bureau of Meteorology (Adelaide Kent Town station, #23090, located ~1.3km SE from the roost). In addition to body condition score, we derived two additional measures of body condition. We calculated weight to forearm ratio (WFR), which is a commonly used metric of condition in bats [135]. We also calculated a scaled mass index (SMI), which controls for covariation in body length and body mass associated with growth [136] (Additional file 2: calculation of scaled mass index). For each outcome variable, the three measures of body condition (WFR, BCS, and SMI) were included individually in three separate candidate models. We used Akaike information criterion corrected for small sample size (AICc) to rank models [137, 138], and refit the best-supported model with restricted maximum likelihood, for which we reported model estimates. We visualized residual plots with the *DHARMa* package [139].

To examine predictors of foraging area and core area, we fit generalized linear models (GLMs) with a gamma distribution and log link; random effects were not included because foraging and core areas were derived per individual across nights. We included age, sex, body condition, season, and the number of nights a bat was tracked as predictors. We again included the three body condition measures individually in three separate models for each outcome variable, and used AICc to rank models.

To examine the selection of foraging sites within the area bats traverse (third-order habitat selection [140]), we compared the land use at foraging sites to that of randomly chosen locations. We examined habitat selection separately by season, as available resources can change seasonally. We designated the centroids of foraging sites (see above) as “used” sites, and generated random “available” sites within the seasonal

95% MCPs (excluding the ocean), such that for each season, there was a 10:1 ratio of available to used sites. Due to logistical constraints, nectar and fruit resources were not quantified at the foraging sites or randomly-generated available sites. We randomly assigned bats to available sites in proportion to the number of foraging sites used by a bat. We obtained a land use raster of the Catchment Scale Land Use of Australia [141], which provides land use classes at three hierarchical scales according to the Australian Land Use and Management (ALUM) Classification (version 8) at a 50m resolution. We used the "Extract Values to Points" tool in ArcGIS 10.4.1 [142] to determine the tertiary (i.e. finest scale) land use of used and available sites. We performed two logistic regressions, one for each season, to model the probability of a site being used or available as a function of the land use at the site and the body condition, sex, and age of the associated bat. To reduce the number of land use classes for the regression, we condensed the tertiary land classes into five categories, similar to the ALUM primary classes: 1) *Natural*, 2) *Agricultural and Plantation Production*, 3) *Residential and Farm Infrastructure* (including urban and rural residential), 4) *Non-residential Intensive Use* (e.g. intensive animal production, industrial, recreation, transportation) and 5) *Water*. As with the movement analyses, we included the three body condition measures in separate models for each season, and used AICc to rank models.

Finally, we examined whether bat body condition differed by season. We constructed three linear models with the body condition measures (WFR, BCS, SMI) as separate outcome variables, and season, sex, and age as predictor variables. WFR was modeled with a gamma distribution (log link), and SMI was modeled with a normal

distribution. Due to the limited range of values, BCS was binned into two groups (≤3 and >3) and modeled with a binomial distribution.

RESULTS

Data loggers were deployed on 14 female and 18 male *P. poliocephalus*; bats weighed between 640g and 1008g, corresponding to a logger burden of <2.5% body weight, well under the recommended 5% threshold for bats [143]. After excluding nights with <8 hours of data and non–round-trip nights, we retained approximately 90% of the data (186/209 nights). Nightly round-trip distance varied widely within and between bats (Appendix B Figure S3.2). The median round-trip distance was 31.93 km, and the largest round-trip distance was 179.34 km (Appendix B Figure S3.3). The most distant foraging site was 40.47 km from the roost (straight line distance). Distance and area metrics for each bat are summarized in Appendix B Table S3.1.

We observed foraging site fidelity in GHFF movement patterns. Some bats made repeated visits to the same foraging site over multiple nights with little deviation in flight path, while others visited a few core foraging sites with occasional long-distance excursions to other sites (Appendix B Figure S3.4). Flying foxes typically visited 2-6 foraging sites per night (Appendix B Table S3.1). During the winter, foraging paths appeared primarily north-south oriented (Figure 3.2A), while summer foraging paths clustered closer to the roost (Figure 3.2B).

Predictors of foraging distance, area, and number of sites visited

Bats in better body condition flew shorter distances each night, visited fewer foraging sites each night, and had smaller foraging areas (Figure 3.3, Table 3.1; Appendix B Table S3.2). Specifically, holding other explanatory variables constant, a one-unit increase in BCS was associated with a 43% decrease (95% Wald confidence interval: 61% – 19% decrease) in nightly round-trip distance, and a 31% decrease (95% CI: 44% – 14% decrease) in the number of nightly foraging sites. Similarly, a one-unit increase in WFR was associated with an 86% decrease (95% CI: 97% – 38% decrease) in foraging area. Models incorporating BCS and WFR were well supported by model selection, but SMI received little to no support (Appendix B Table S3.2).

We also found that males had longer nightly round-trip distances than did females (Table 3.1). Holding other explanatory variables constant, male bats traveled 37% farther in their nightly round-trip distance than did female bats (Figure 3.3). Finally, bat age predicted the number of nightly foraging sites, with younger bats visiting more sites per night. An estimated one-year increase in age was associated with a 6% decrease in the number of nightly foraging sites. We found no significant predictors of core foraging area in our analyses (Table 3.1).

Habitat selection

Our automated foraging site classification identified 203 sites (137 summer, 66 winter). Nearly all foraging sites belonged to one of three tertiary land classes: urban residential (100 sites; subclass of *Residential and Farm Infrastructure*), recreation and culture (59 sites; subclass of *Non-residential Intensive Use*), and public services (13 sites; subclass of

Non-residential Intensive Use). Season-specific logistic regressions showed that, controlling for the habitat available in Adelaide (95% MCPs), the probability of a site being used for foraging was significantly predicted by land use (Appendix B Table S3.3). In summer, bats had 21.1 times the odds (95% CI: 5.1 – 87.5) of foraging at *Non-residential Intensive Use* sites and 16.1 times the odds (95% CI: 3.9 – 66.4) of foraging at *Residential and Farm Infrastructure* sites, compared to foraging at *Natural* sites. In winter, bats had 12.3 times the odds (95% CI: 2.8 – 53.4) of foraging at *Non-residential Intensive Use* sites and 7.2 times the odds (95% CI: 1.7 – 30.6) of foraging at *Residential and Farm Infrastructure* sites, compared to foraging at *Natural* sites. Models incorporating BCS, WFR, and SMI were equally supported by model selection; coefficients reported above are for the model with WFR, as this is the simplest condition measure of the three.

Foraging plants

We identified 21 unique species of plants from 150 suspected foraging sites identified in Google Earth (Appendix B Table S3.4). At these sites, we primarily observed plant species in the Myrtaceae family, especially lemon-scented gum (*Corymbia citriodora*), blue gum (*Eucalyptus leucoxylon*), and Mugga ironbark (*E. sideroxylon*); and in the Moraceae family, including common fig (*Ficus carica*) and Moreton Bay fig (*F. macrophylla*). Palm trees (Arecaceae family) were also popular, although these were rarely identified to species owing to difficulty in obtaining physical samples. Most plant species identified at foraging sites were native to Australia (18/21), with about half of these native to South Australia (7/18). Four species documented here were previously

identified as significant food for GHFF in other areas of Australia (having high weighted productivity*reliability scores; [144]. We found good accordance between foraging sites identified with Google Earth versus our automated procedures; 43% of Google Earth sites were within 10 m of an auto-identified site, and 69% were within 50 m.

Body condition predictors

Linear models indicated that when body condition was measured as SMI, bats were in significantly better condition in winter than in summer (Appendix B Table S3.5). When condition was measured as WFR, male bats and older bats had significantly greater body condition (Appendix B Table S3.5). Season did not predict changes in body condition when measured as WFR, and no predictors tested here (sex, age, season) explained changes in body condition when measured as BCS.

DISCUSSION

Our study documents the fine-scale foraging movements of GHFF in a newly-established urban population in South Australia. Flying foxes typically foraged at a small number of sites (six or fewer) located near the roost, with occasional excursions to more distant sites, including the longest one-night round-trip (~180 km) observed in this species to our knowledge. Foraging distances observed here were similar to those reported in previous studies of flying foxes roosting in urban areas [133, 145]; comparisons of area are more difficult to make owing to the variety of methods used in past studies.

Among bats examined here, better body condition predicted shorter foraging distances and the use of fewer foraging sites. Our finding of negative relationships between flying fox body condition and foraging movements aligns with several other studies (Table 3.2). Negative condition–movement relationships could result from several underlying mechanisms. On the one hand, body condition might determine movement behavior; for example, flying foxes can be territorial [38], and bats in better condition might better defend feeding sites near the roost, forcing bats in poorer condition to travel to more distant sites [146]. On the other hand, changes in movement might alter body condition. Conducting long-distance flights, potentially to explore for new resources [147, 148], could deplete bats' energy and decrease body condition. As a third explanation, resource distribution and quality could control both movement and bat body condition. If high-quality resources are present near the roost, this could simultaneously decrease foraging distance and the number of foraging sites visited while boosting body condition.

Past work on flying fox movements further showed that the direction of condition–foraging metric relationships varies among studies, and within a study, can depend on the body condition measure used (Table 3.2). This emphasizes the importance of measuring body condition in multiple ways [63] to improve conclusions drawn regarding relationships between body condition and foraging metrics. In particular, different body condition measures might change on different time scales, and some body condition measures might better reflect overall health and nutrition for a given species. For example, a qualitative measure like BCS that relies on physical examination of an animal might be better able to capture changes in fat or muscle that reflect resource

acquisition over weeks or months. More generally, improved measures to assess bat body condition are needed, as current measures can be subjective (BCS) or difficult to employ in the field (quantitative magnetic resonance analysis [149]).

We found little evidence for seasonal differences in bat foraging movements or associations between movement and daily temperature. Resources planted in Adelaide might act as a buffer against seasonal variation. Previous work reported that in Melbourne, 13 plant species providing food for GHFF grew naturally, but 87 non-native species had been planted after European settlement [112]. The authors proposed that non-native plants provide a continual source of food for flying foxes during times of natural resource scarcity (May – August). Our finding of greater bat condition (when measured as SMI) in winter than summer ran counter to predictions based on resource availability. However, in insectivorous bats, SMI has been proposed to be a less informative predictor of condition than simple body mass [150], so our finding should be interpreted cautiously.

The observations of longer nightly round-trip distances among male flying foxes, and greater foraging site visits among younger bats, might be explained by sex and age differences in roost emergence times and exploratory flights. Previous work examining GHFF emergence timing (i.e. when bats leave the roost at night to forage) showed that males typically left the roost later than females [151]. In this case, bats that emerge later might travel farther to reach non-depleted resources, if high-quality patches in closer proximity to roosts are used by bats that emerge first. In Pacific flying foxes (*P. tonganus*) younger bats engage in longer, exploratory flights [147], which could explain our finding that they visited a larger number of foraging sites. Alternatively, younger bats

may not be adept at identifying productive foraging sites, necessitating them to visit multiple sites to meet their dietary needs. As with body condition, younger bats might be also forced away from foraging sites by territorial bats. An important caveat for results reported here is that age estimation based on visual inspection of tooth wear is difficult, as wear begins early in life for flying foxes [152].

In terms of site selection, bats tracked in our study were more likely to forage at residential and other human-modified sites than in less-disturbed natural environments. In particular, bats foraged primarily in urban residential and recreational sites (e.g. parks), consistent with flying fox studies in other parts of Australia and elsewhere [111, 153, 154]. Because we did not survey resource availability in natural areas, it is difficult to assess whether flying foxes foraged less in these areas due to lack of resources or due to a preference for resources in other habitats. Past work suggested that flying foxes prefer native species that produce abundant nectar, and that there may be a threshold of flowering intensity above which animals seek out these resources [109]. If abundant nectar is not available, flying foxes use urban resources [37]. Unlike urban sites, we found no evidence that agricultural sites were more likely to be used for foraging than natural sites, suggesting that at least in Adelaide, agricultural resources are not disproportionately used by flying foxes. In other parts of Australia and the world, orchard fruit consumption by flying foxes is commonly reported, economically costly, and a source of human-bat conflict and pathogen transmission [155]. If the Adelaide GHFF population continues to grow, or new camps form in South Australia, it is possible that selection of foraging sites could change.

Our work adds to the small but growing body of literature on the movement of flying foxes in urban environments, and is relevant to diverse stakeholders including land managers, orchardists, airport managers, and electric companies [155-158]. In particular, bats comprised the most airstrikes by planes in Australia from 2006-2015 [159]. Our goal of tracking movement patterns that are representative of a recently-formed urban bat camp, which numbers nearly 20,000 individuals at present, was limited by the high cost of data loggers and consequently, the small number of bats monitored here. At the same time, the number of bats tracked here matches or exceeds that of several other papers published on flying fox movements during the past decade (Table 3.2). Our goal was to capture fine-scale foraging data, and we prioritized frequent collection of GPS fixes over a short-term period. Because movement patterns might change during other parts of the year (e.g. mating season, which typically occurs for GHFF in March-April; [38]), longer-term studies that examine flying fox movements throughout the course of a year are needed. Work contrasting movement patterns of flying foxes in urban and non-urban colonies is also necessary.

CONCLUSIONS

We examined the foraging movements of Australian flying foxes in a recently-established, previously undescribed urban population. Our work suggests that in Adelaide, urban flying foxes foraged largely in human-dominated habitats, and that bats in better body condition made shorter, more efficient foraging flights. Understanding how animals move in human-modified habitats is important for wildlife conservation and managing human-wildlife conflicts. Studying the diet, health, and movements of animals

that host zoonotic pathogens is also relevant for human health. Future GPS tracking studies and continuing improvements in technology are likely to uncover intricacies of local and global animal movements.

LIST OF ABBREVIATIONS

AICc: Akaike information criterion corrected

ALUM: Australian Land Use and Management

BCS: body condition score

GHFF: grey-headed flying fox

GLM: generalized linear model

GLMM: generalized linear mixed model

GPS: Global Positioning System

MCP: minimum convex polygon

SMI: scaled mass index

WFR: weight to forearm ratio

DECLARATIONS

Ethics approval and consent to participate: A permit to undertake scientific research was granted by the Government of South Australia Department for Environment and Water (M26371-4). Fieldwork was approved by the University of Adelaide Animal Ethics Committee (S-2015-028A) and the UGA Animal Care and Use Committee (AUP A2015 03-028-Y3-A1).

Consent for publication: Not applicable

Availability of data and materials: If the paper is accepted, movement data will be archived at the Movebank Data Repository.

Competing interests: The authors declare that they have no competing interests.

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Authors' contributions: CAS, SA, and WSJB conceived the ideas and designed methodology; CAS, TBR, MO, JvW, and WSJB collected the data; CAS analyzed the data; CAS led the writing of the manuscript. All authors contributed critically to the drafts and gave final approval for publication.

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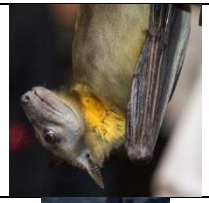
Table 3.1. Model outputs for nightly round-trip distance and number of foraging sites, foraging area, and core area. Three candidate models (using separate measures of body condition) were created for each response variable and ranked by AICc. The best-supported model for each response is reported here; see Table S3.3 for all candidate models. Bolding indicates *P* values < 0.05. BCS: body condition score; WFR: weight to forearm ratio; maxTemp: maximum daily temperature.

Response variable	**Term**	**Estimate**	**SE**	**t or z**	***P***
Nightly round-trip distance (n = 185)	age	-0.04	0.05	-0.84	0.402
	sex(male)	0.31	0.16	2.01	**0.044**
	BCS	-0.57	0.18	-3.09	**0.002**
	season(winter)	0.22	0.30	0.72	0.471
	maxTemp	-0.02	0.02	-0.93	0.352
	hours	-0.07	0.08	-0.85	0.396
Nightly number of foraging sites (n = 185)	age	-0.06	0.03	-2.07	**0.038**
	sex(male)	0.11	0.09	1.19	0.233
	BCS	-0.38	0.11	-3.34	**0.001**
	season(winter)	-0.08	0.18	-0.46	0.647
	maxTemp	-0.01	0.01	-0.46	0.647
	hours	0.03	0.07	0.37	0.710
Foraging area (n = 27)	age	0.01	0.16	0.04	0.972
	sex(male)	0.93	0.51	1.84	0.081
	WFR	-1.98	0.66	-2.98	**0.007**
	season(winter)	-0.31	0.49	-0.63	0.532
	nights	0.17	0.11	1.50	0.149
Core area (n = 27)	age	0.01	0.17	0.08	0.938
	sex(male)	0.74	0.53	1.40	0.177
	WFR	-1.15	0.70	-1.65	0.114
	season(winter)	0.17	0.52	0.33	0.742
	nights	0.03	0.12	0.25	0.803

Table 3.2. Relationships between body condition and foraging metrics (distance, range) in bats in the *Pteropodidae* family. Only studies published within the last ten years are included. Negative relationships between body condition and foraging metrics are shaded. Image credits: *P. dasymallus* by Koolah, *A. jubatus* by de Jong et al. 2013, and *P. lylei* by Malene Thyssen licensed under CC BY-SA 3.0 *E. helvum* by Kayt Jonsson and *P. rufus* by Bernard Dupont licensed under CC BY 2.0. *P. alecto* by Andrew Mercer licensed under CC BY-SA 4.0. *P. poliocephalus* by Michelle Power used with permission. [a] linear model performed using data from the paper. [b] t-test performed using data from the paper. [c] 50% utilization distribution, kernel method. [d] 95% utilization distribution, kernel method. [e] 100% minimum convex polygon. [f] 80% cluster core area

Species and sample size		Condition measure	Foraging metric (unit)	Direction of condition – foraging metric relationship	*P*	Source
	Orii's flying fox (*Pteropus dasymallus inopinatus*); *n* = 19	WFR	Mean daily home range (ha)	Positive	0.76[a]	[154]
	Giant golden-crowned flying fox (*Acerodon* jubatus); *n* = 6	BCS	Mean nightly distance (km)	Negative	0.73[b]	[125]
			Maximum nightly distance (km)	Positive	0.45[b]	
			Mean distance to foraging areas (km)	Positive	0.80[b]	
			Mean number of foraging areas per night	Negative	0.41[b]	
		WFR	Mean nightly distance (km)	Positive	0.73[a]	
			Maximum nightly distance (km)	Positive	0.93[a]	
			Mean distance to foraging areas (km)	Negative	0.20[a]	
			Mean number of foraging areas per night	Negative	0.65[a]	

Species and sample size		Condition measure	Foraging metric (unit)	Direction of condition – foraging metric relationship	*P*	Source
	Straw-coloured fruit bat (*Eidolon helvum*); *n* = 16	WFR	Mean nightly distance (km)	Positive	0.041[a]	[133]
			Maximum distance to foraging sites (km)	Positive	0.078[a]	
			Core area[c] (ha)	Positive	0.066[a]	
			Foraging area[d] (ha)	Positive	0.056[a]	
	Madagascan flying fox (*Pteropus* rufus); *n* = 15	WFR	Home range[e] (ha)	Negative	0.82[a]	[148]
			Foraging area[f] (ha)	Negative	0.71[a]	
	Black flying fox (*Pteropus alecto*); *n* = 11	BCS	Mean nightly distance (km)	Negative	0.047	[115]
			Mean distance to foraging areas (km)	Negative	0.064[b]	
			Mean number of foraging areas per night	Negative	0.26[b]	
		Weight	Mean nightly distance (km)	Positive	0.70[b]	
			Mean distance to foraging areas (km)	Negative	0.55	
			Mean number of foraging areas per night	Positive	0.63	
	Lyle's flying fox (*Pteropus lylei*); n = 13	WFR	Maximum nightly distance (km)	Negative	0.026[b]	[153]
			Core area[c] (sq. km)	Negative	0.25[b]	
			Foraging area[d] (sq. km)	Negative	0.22[b]	
	Grey-headed flying fox (*Pteropus poliocephalus*); *n* = 32	BCS	Nightly round-trip distance	Negative	0.04	This study
			Nightly number of foraging sites	Negative	0.0008	
		WFR	Foraging area	Negative	0.007	

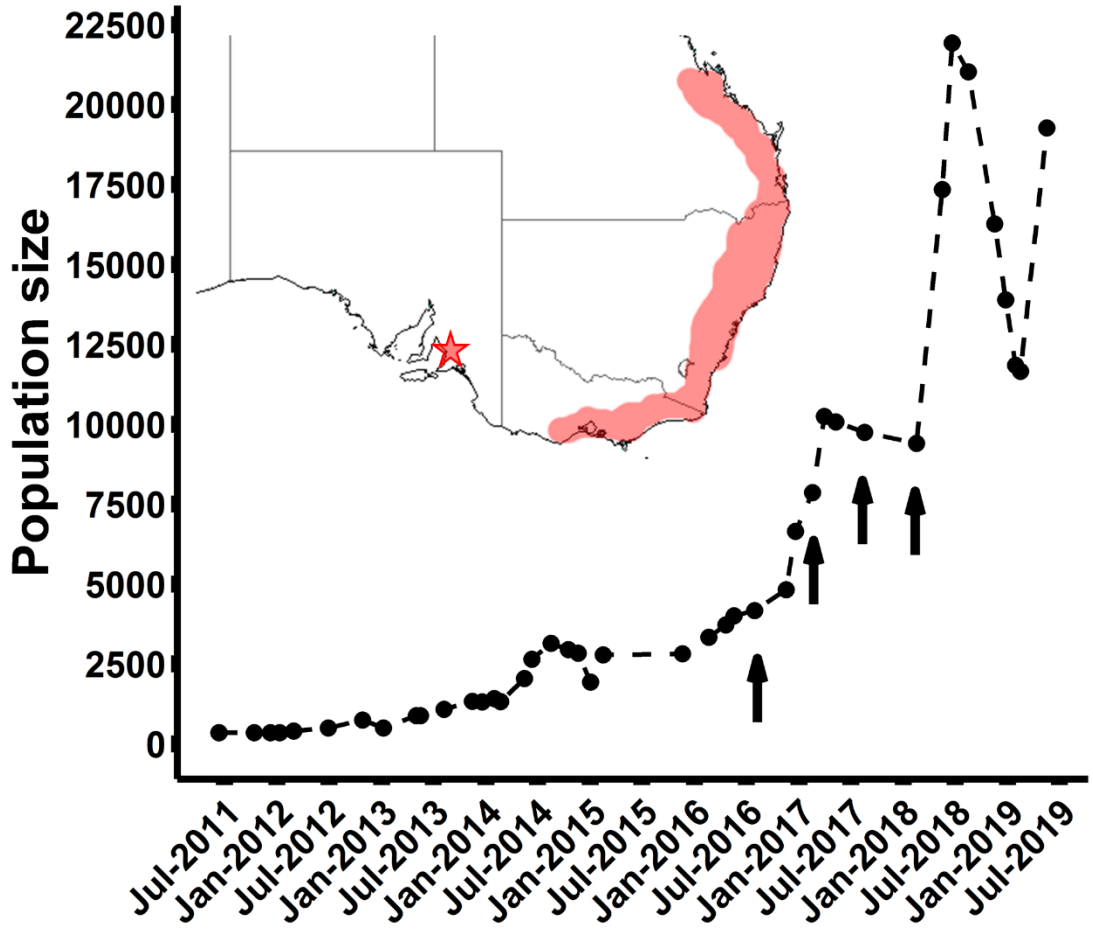


Figure 3.1. Population estimates of the Adelaide grey-headed flying fox camp. Points indicate observer counts of flying foxes. Arrows indicate GPS data collection periods of the current study. The inset shows a partial map of Australia, with the typical range of the grey-headed flying fox shaded in red, and the approximate location of Adelaide indicated by a star.

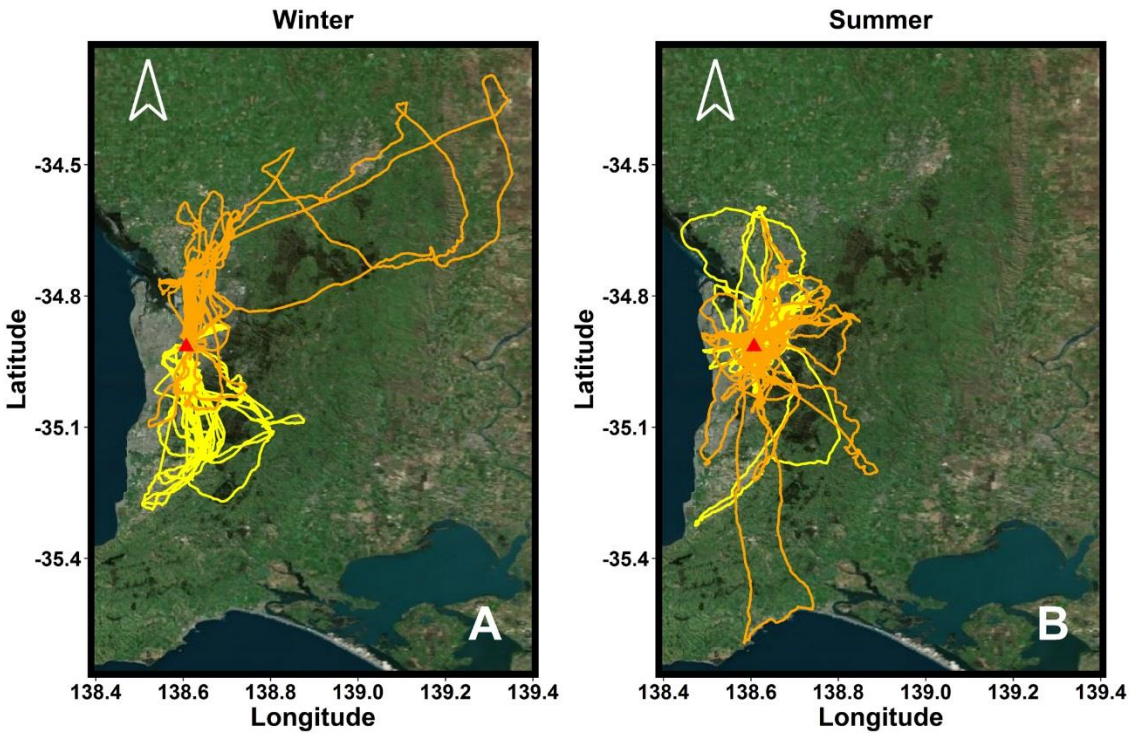


Figure 3.2. GPS tracks of *P. poliocephalus* during **A**) winter 2016 (5 bats, yellow paths) and winter 2017 (7 bats, orange paths) and **B**) summer 2017 (9 bats, yellow paths) and summer 2018 (11 bats, orange paths). The location of the roost is indicated by a red triangle.

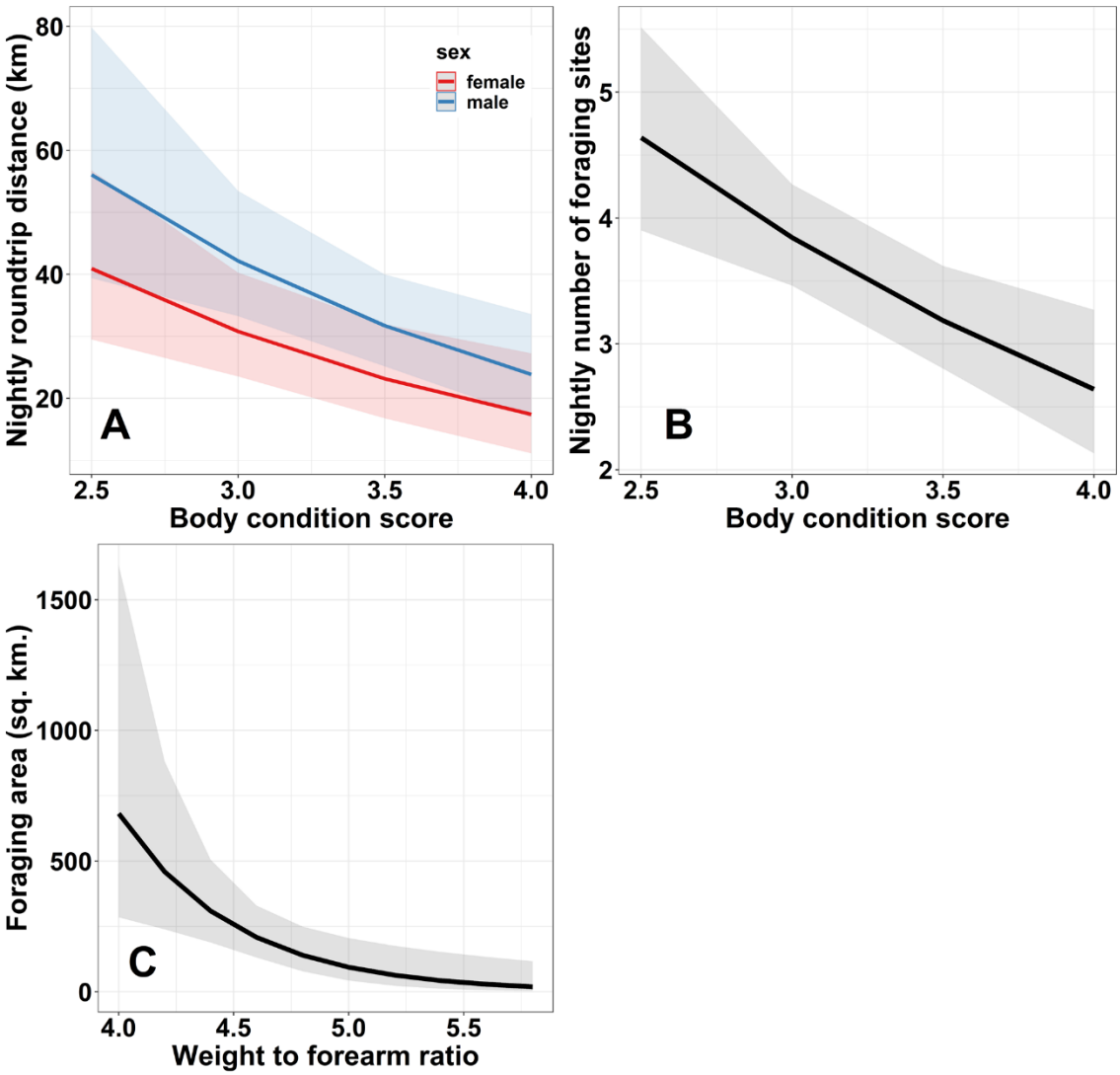


Figure 3.3. Predicted relationships between body condition measures and **A)** nightly round-trip distance (shown for both sexes), **B)** nightly number of foraging sites, and **C)** foraging area (95% MCP). Shaded areas represent 95% confidence intervals. Predictions are generated from GLMMs (distance, foraging sites) and a GLM (foraging area). Discrete predictors are held constant at their proportions.

CHAPTER 4

LAND USE, SEASON, AND PARASITISM PREDICT METAL CONCENTRATIONS IN AUSTRALIAN FLYING FOX FUR[3]

[3] Sánchez CA, Penrose M, Kessler MK, Becker DJ, McKeown A, Hannappel M, Boyd V, Camus MS, Padgett-Stewart T, Lunn T, Peel AJ, Westcott DA, Rainwater TR, Chumchal MM, Cobb GP, Altizer S, Plowright RK, Boardman WSJ. To be submitted to *Environmental Science & Technology*.

ABSTRACT

Urban-living wildlife can be exposed to metal contaminants dispersed into the environment through industrial, domestic, and technological applications. Metal exposure carries lethal and sublethal consequences, depending on dose and frequency of exposure, and the age and condition of animals. In particular, heavy metals such as arsenic, lead, and mercury can damage organs and act as carcinogens. Many species of bats reside and forage in human-modified habitats, and could be exposed to contaminants in air, water and food. Here we quantified 13 metals in fur samples from three flying fox species captured at eight sites across Australia. For a subset of bats, we assessed ectoparasites, blood parasites, and viral infection. We examined relationships between metal concentrations and environmental (land use surrounding capture site, season) and individual predictors (species, sex, age, body condition, parasitism). As expected, bats captured at sites with greater human impact had higher metal loads. At one site, bats had lower metal concentrations in summer than in winter, possibly owing to changes in food availability and foraging. Relationships between ectoparasites and metals were mixed, suggesting multiple causal mechanisms. Because some bats harbor pathogens that can transmit to humans and other species, future research exploring interactions between metal exposure, immunity, and infection is needed to assess consequences for pathogen transmission and bat health.

INTRODUCTION

Wildlife in urban areas face exposure to environmental toxicants (e.g., heavy metals, pesticides, persistent organic pollutants) through contaminated food, water, and air [28]. Landscape maintenance of urban green spaces such as parks and lawns can introduce fertilizers and pesticides into soil and waterways, facilitated by high impervious surface cover [160]. Water can also be contaminated with point source toxicants such as industrial wastewater and oil or chemical spills [161, 162]. Pesticides used to poison nuisance wildlife can reach non-target species via bioaccumulation (i.e., persistence in tissues and organs) and biomagnification (i.e., passing up through the food chain) [163-165]. North American bald eagles provide a striking example of biomagnification: dichlorodiphenyltrichloroethane (DDT) used to control insects during the 1940s-60s subsequently accumulated in fish and eagles, causing thinning of eagle eggshells and reproductive failure [166-168]. Transportation and industrial activities can degrade air quality, sometimes across large distances, such as observed for atmospheric deposition of polychlorinated biphenyls [18, 169, 170]. Even after contaminant-generating sources are removed from an environment, toxicants themselves can persist for months to years, and continue to harm wildlife [171]. One recent meta-analysis found that urban wildlife had significantly higher toxicant loads than non-urban conspecifics across diverse animal taxa [172].

Heavy metals such as mercury and lead, and metalloids such as arsenic, are naturally occurring trace elements that can reach toxicity at relatively low levels of exposure from anthropogenic activities. Some metals such as cobalt, copper, iron, and zinc can serve as micronutrients essential to biochemical and physiological functions, yet

become toxic at higher levels of exposure through their interactions with enzymes involved in metabolism, detoxification, and damage repair. Other metals such as cadmium, lead, and mercury have no known biological function, and represent systemic toxicants that can damage organs and disrupt DNA even at low levels of exposure. Consequences of metal exposure for humans and vertebrate animals include altered foraging and other behaviors resulting from neurological damage, reduced body condition, physical deformities, reduced fecundity, and mortality [173-176]. Some animals exposed to toxicants show lower immune function or reduced behavioral defenses such as grooming [177, 178]. For example, female tree swallows breeding at mercury-contaminated sites had higher mercury concentrations in blood and weaker immune response than birds at non-contaminated sites [76]. Additionally, negative effects of metal exposure can be exacerbated by other stressors (e.g., competition, predation, food limitation, habitat alteration) [179, 180]. Laboratory rats experimentally exposed to both concentrated air pollutants and chronic social stress exhibited elevated levels of inflammation biomarkers [181].

Bats as a group are well-suited to study biological and environmental predictors of metal exposure [182]. Their long lifespans (up to 40 years [183]) permit metal accumulation in organs and tissues over time, and their high mobility and dietary breadth allow them to forage in natural and human-modified habitats. Most studies of exposure to metals and other toxicants in bats have focused on insectivorous species [184-186], likely because there is a clearer exposure route (i.e., uptake through insect prey). Fruit bats (that feed on nectar, pollen, and fruits) are increasingly settling in urban and agricultural areas, where they consume introduced and cultivated plant species [35, 187], which could

expose bats to food resources laden with pesticides or metals [188-190], or polluted waterways [191]. Experimental dosing has suggested fruit bats absorb more lead than do other mammals [192], which might make species in this group especially vulnerable to negative toxicant effects. If toxicants impair immune function in bats, this could increase their exposure to (or slow their recovery from) pathogens. Because some bats host viruses and other pathogens that can be transmitted to domesticated animals and humans, impaired immune function owing to metal exposure could pose public health risks [193]. Some studies have reported high concentrations of toxicants in bats infected with fungal or viral pathogens, but failed to demonstrate a causal association [194, 195].

In this study, we examined the metal exposure of three *Pteropus* fruit bat species (flying foxes) captured at eight sites across Australia between 2015 and 2018. Specifically, we measured the concentrations of 13 metals in fur samples, including mercury, lead, and cadmium, for which low levels of exposure are known to cause toxicity for vertebrates. We tested for relationships between metal concentrations and environmental (land use surrounding bat capture site, season) and individual-level predictors (species, sex, age, body condition). We predicted that metal concentrations would be higher for bats captured in areas with greater human modification (e.g., urbanization, industrialization, agriculture), and in older bats and those with poorer body condition. We also assessed ectoparasite burden, haemosporidian parasite infection status, and viral infection status in a subset of bats, as additional indicators of health. High metal concentrations might predict positive infection status, although the direction of this relationship could vary among parasites. For example, some metals might weaken bat immune defenses or reduce grooming behavior, leading to a positive relationship

between metal concentrations and parasitism. Alternatively, if metals are toxic to parasites, such as might occur if enzymatic pathways in the parasites themselves are disrupted, then exposure could reduce infection for some parasite groups [196].

METHODS

Animal capture and sampling

Three species of pteropodid flying foxes (black flying fox, BFF, *Pteropus alecto*; spectacled flying fox, SFF, *P. conspicillatus*; grey-headed flying fox, GHFF, *P. poliocephalus*) were captured between June 2015 and September 2018 at eight sites across three Australian states (Queensland, New South Wales, and South Australia) (Figure 4.1, Table 4.1). Flying foxes were captured between pre-dawn using mist nets as they returned from nightly foraging, and anesthetized under veterinary supervision with inhalant isoflurane [123]. We recorded each bat's species, sex, weight (nearest g), and forearm length (nearest mm). Body condition was calculated as the ratio of weight to forearm length (WFR). Age in years was estimated for South Australia bats (n = 207); all other bats were assigned to an age class (adult, subadult, or juvenile) based on secondary sexual characteristics [119]. The number of ectoparasitic bat flies (family *Nycteribiidae*) was recorded if present. A fur clipping (~20-80 mg) was taken from the chest or back of bats. In total, we obtained fur samples from 721 flying foxes at four sites in Queensland (Goldsborough, Hervey Bay, Redcliffe, Tolga, Toowoomba), two sites in New South Wales (Tamworth, Woolgoolga) and one site in South Australia (Adelaide; Figure 4.1, Table 4.1). After all samples were collected, flying foxes were allowed to recover from anesthesia and released at the capture site.

Urine was collected from bats captured from four of the eight sites (n = 39; Figure 4.1). We palpated bats' abdomens gently to express urine and collected samples in 1.5 mL screw-cap tubes (Axygen, Union City, CA). Samples were placed on cooler packs in the field and later stored at ~ -20°C. Blood smears were prepared for bats captured from three sites (n = 82; Figure 4.1). Using a 25-gauge needle, blood was drawn from the cephalic vein and a drop was used to make a thin blood smear in the field.

Fieldwork in Queensland was authorized under section 173P of the Nature Conservation Act 1992. Fieldwork in New South Wales was authorized under section 132c of the National Parks & Wildlife Act, 1974 (SL101396). Fieldwork in South Australia was authorized by the Government of South Australia Department of Environment, Water and Natural Resources (M26371-4). Ethical approval was granted by the CSIRO Ecosystem Sciences Animal Ethics Committee (13-02), the University of Adelaide Animal Ethics Committee (S-2015-028), the University of Georgia Animal Ethics Committee (A2015 03-028-R3), and the Griffith University Animal Ethics Committee (ENV/10/16/AEC).

Analyses of biological samples

Fur samples were analyzed at Baylor University for ten metals: cadmium, chromium, cobalt, copper, lead, nickel, selenium; strontium; tin, vanadium, and two metalloids: antimony and arsenic, following previously published methods [197] (see Supplementary Material for details). Briefly, fur samples were individually weighed (~0.05 g) and placed in borosilicate glass tubes (VWR International, Radnor, PA). Batches of 25-30 samples were digested with nitric acid and hydrogen peroxide in a series of heating and cooling

steps, then filtered into acid-rinsed Erlenmeyer flasks (VWR International) and diluted in ultrapure water. Blanks were included for each batch. Human hair standard (Sigma-Aldrich, St. Louis, MO) was used as a standard reference material, with one reference sample included for each bat capture site. Samples were analyzed by inductively coupled plasma mass spectrometry (ICP-MS) using a 7900 ICP-MS (Agilent Technologies, Santa Clara, CA). Metal concentrations are reported in ng/g.

Following metal analyses, remaining fur samples were sent to Texas Christian University and analyzed for total mercury (methylmercury + inorganic mercury; hereafter, mercury) using direct mercury analysis (DMA-80 Direct Mercury Analyzer, Milestone, Shelton, CT), which uses thermal decomposition, gold amalgamation, and atomic absorption spectroscopy [198]. Quality assurance included reference (National Research Council of Canada Institute for National Measurement Standards) and duplicate samples. Reference samples (DORM-4) were analyzed every 10 samples, and the mean recovery percentage for DORM-4 was 103 ± 4.31% (n = 81). Duplicate samples were analyzed every 20 samples, and the mean relative difference percentage was 6.83 ± 7.05% (n = 44). Limited amounts of hair available for analysis and low concentrations resulted in some samples (n = 55) falling below the mercury detection limit (0.1 ng, approximately 0.6 ng/g); these values were subsequently estimated as half of the detection limit (i.e., 0.05 ng) divided by the sample weight. Mercury concentrations are reported in ng/g.

Blood smears (n = 82) were examined at the University of Georgia for blood parasites (order Haemosporida). Smears were stained with Modified Wright's stain and the monolayer of each blood smear was scanned at both 500X and 1000X magnifications

by a board-certified veterinary clinical pathologist (M. Camus). Samples for which one or more infected erythrocyte were detected were scored as positive for blood parasites.

Urine samples (n = 39) were shipped on cooler packs to the Australian Animal Health Laboratory (Geelong, Victoria). Samples were condensed into eight pools and screened for 11 paramyxoviruses (Cedar virus, Geelong paramyxovirus, Grove virus, Hendra virus, Hervey virus, Menangle virus, Nipah virus, Teviot virus, Tioman virus, Yarra Bend paramyxovirus, Yeppoon virus) using a multiplex bead X-Tag assay for nucleic acid detection [199].

Statistical analyses

All statistical analyses were performed in the R computing environment v 3.6.1 [127]. In initial data exploration, six fur samples were found to have extremely low concentrations of all metals except mercury. Because these samples were processed consecutively in the laboratory, we considered it likely that a technical error had occurred and excluded those values from analyses described below. Due to the extreme range in values, metal concentrations were log-transformed for further analyses.

We calculated summary values for metal concentrations for the three flying fox species. We used generalized linear mixed models (GLMMs) to first compare species differences in metal concentrations while controlling for site. For each metal, we used the *lmerTest* package [200] to run a GLMM (gamma distribution, log link) with species as a fixed effect and site as a random effect. Pairwise comparisons of species means were made with the *multcomp* package [201] with a Holm adjustment for multiple comparisons.

To determine whether bats captured at sites with greater human impact had higher metal concentrations, we calculated the average Human Footprint (HFP) score within a 20 km buffer (typical foraging range for flying foxes [202, 203]) around each site. HFP ranges from 0-50, and is a composite measure of human impacts including population density, and the proportion of land area assigned to agriculture, built environments, and transportation. We used the most recent human footprint dataset available (2009) from Venter and colleagues [66] to calculate this score. We next used a principal component analysis (PCA) on all metal concentrations in fur to create a composite index of metal exposure. Metal concentrations were log-transformed, then centered and scaled to have unit variance. Horn's parallel analysis supported retention of the first three principal components [204]. We then used three LMMs [200] (gaussian distribution, identity link) to test whether site HFP explained variation in PC1, PC2, or PC3. We included species and sex as fixed effects, and site as a random effect in each model (n = 402).

For bats captured in Adelaide (n = 202 GHFF), we examined the effect of season, sex, body condition, and age on metal concentrations in fur. We focused on Adelaide because 1) it was the only site at which bats were captured multiple times in separate seasons (two summer and two winter sampling periods), and 2) it allowed us to avoid possible confounding effects of species or site on relationships between metal concentrations and sex, body condition, and age. We used a separate linear model (gaussian distribution, identity link) for each metal, with season, sex, WFR, and estimated age (in years) as predictor variables.

To examine associations between ectoparasites and metal concentrations, we only considered data from flying foxes caught in Queensland (five sites), as <1% (2/254) of

flying foxes from New South Wales and South Australia had ectoparasites. We used the *glmmTMB* package [134] to run a GLMM (Poisson distribution, log link) to model ectoparasite burden (number of ectoparasites per bat) as a function of each metal (n = 157). We included species, sex, WFR, and age class (adults versus non-adults) as additional fixed effects and site as a random effect. We checked for multicollinearity in the ectoparasite models using the *performance* package [205]; if any variance inflation factors (VIFs) were > 10, we sequentially removed predictor variables with the highest VIF until all remaining VIFs were < 10 [206].

Lastly, we examined the probability of blood parasite infection in GHFF and SFF captured at three sites (blood smears were not made for one of the four sites where blood was collected). We used a GLM (binomial distribution, logit link) to model infection status as a function of each metal concentration in fur, species, sex, WFR, and age class (n = 79). Site was not included to avoid perfect separation of data. We checked for multicollinearity in the blood parasite models as described above.

RESULTS

Most fur samples used for metal analysis were from BFF (n = 339; 47.0%) and GHFF (n = 336; 46.6%); the remainder were from SFF (n = 46; 6.4%). GHFF had the largest spatial distribution, with samples from across nearly ten degrees of latitude and six of eight sites (Figure 4.1; Table 4.1). Slightly more than half of samples were from females (n = 395, 54.8%). We prepared blood smears for 82 of the 721 flying foxes (Table 4.1; SFF in Tolga; GHFF in Tamworth and Woolgoolga). Microscopic examination of blood smears revealed that nearly 60% (48/82) of smears contained intraerythrocytic

haemosporidian gametocytes [207]. Pooled urine samples (n = 39; SFF in Goldsborough and Tolga; GHFF in Tamworth and Woolgoolga) were negative for all 11 viruses for which we screened.

Metal concentrations in relation to species and human footprint

Minimum, median, and maximum values for concentrations of 13 metals measured in fur are presented in Appendix C Table S4.1. Among fur samples, copper, tin, and strontium consistently had the highest median concentrations across species, while mercury, cadmium, and antimony had the lowest median concentrations (Figure 4.2). Controlling for site, we found significant differences between species in mean concentrations of six metals (Appendix C Figure S4.1). SFF had significantly higher concentrations of cadmium and cobalt than BFF and GHFF, and significantly higher concentrations of selenium than GHFF. BFF had significantly higher concentrations of chromium and strontium than GHFF, and significantly higher concentrations of vanadium than SFF. There were no significant pairwise differences among bat species for concentrations of antimony, arsenic, copper, lead, mercury, nickel, and tin.

Principal component analysis showed support for three principal components for which adjusted eigenvalues were greater than 1. Specifically, PC1, PC2, and PC3 respectively explained 37.1%, 14.2%, and 11.2% of the variation in metal concentrations in fur. PCA loadings are provided in Table 4.2; we considered loadings with an absolute value >0.258 as significant [208]. PC1 was loaded positively by all metals, with eight metals above this cutoff; we therefore considered PC1 to represent overall metal load. PC2 had significant positive loadings of arsenic and significant negative loadings of

cobalt, strontium, and vanadium. PC3 had significant negative loadings of cadmium, cobalt, mercury, and selenium.

Calculation of human footprint values across sampling locations showed that Adelaide and Redcliffe had the highest human footprint scores (28.0 and 26.9 respectively); Tamworth, Toowoomba, and Hervey Bay had moderate scores (11.7, 11.1, and 10.9 respectively); and Woolgoolga, Tolga, and Goldsborough had the lowest scores (9.3, 5.9, and 5.7 respectively). Analysis of associations between metal composite values (PC1, 2, and 3) and human footprint (LMM analyses) showed a significant positive relationship between metal PC1 score (overall metal load) and human footprint score (β = 0.12, SE = 0.020, p = 0.011; Figure 4.3). A post-hoc comparison of species means showed that SFF had significantly higher metal PC1 scores than GHFF (estimated mean difference = 1.76, SE = 0.50, p = 0.001). There were no significant predictors of metal PC2 score. With respect to metal PC3 score, male bats had significantly higher values than females (β = 0.22, SE = 0.081, p = 0.006), and there were significant differences in PC3 scores between species (BFF-GHFF = 0.97, SE = 0.34, p = 0.005; BFF-SFF = 3.04, SE = 0.72, p = 6.7 e-5; GHFF-SFF = 2.07, SE = 0.68, p = 0.005; Figure 4.3), but not among sites with different human footprint values.

Seasonal and individual predictors of metal concentrations

Linear models demonstrated consistent seasonal differences in metal concentrations from GHFF captured in Adelaide (Table 4.3). Nine metal concentrations in fur (antimony, arsenic, cadmium, chromium, cobalt, copper, lead, strontium, and tin) were significantly lower in summer than in winter; concentrations of mercury and nickel were significantly

higher in summer. There was limited evidence of sex, body condition, and age-associated differences in metal concentrations. Male bats had significantly lower concentrations of cadmium, mercury, and strontium than females. Better body condition (higher WFR) was significantly associated with higher concentrations of mercury and lower concentrations of tin and vanadium. Age was significantly correlated with higher concentrations of mercury and tin. We found no significant predictors of selenium concentrations in fur.

Relationships between metals and parasites

We found significant relationships between ectoparasite burden and four of the 13 metals. In a GLMM, ectoparasite burden was positively correlated with nickel and selenium concentrations and negatively correlated with cobalt and mercury concentrations (Figure 4.4; Appendix C Table S4.2). There was a positive association ($p = 0.051$) between chromium and ectoparasite burden. There were no effects of other metal concentrations (antimony, arsenic, cadmium, copper, lead, strontium, tin, vanadium), species, sex, WFR, and age class on ectoparasite burden. We found no significant relationships between blood parasite infection status and any of the 13 metals, but bats in worse body condition had a greater probability of blood parasite infection (Appendix C Table S4.3). Infection status did not vary by species, sex, or age class.

DISCUSSION

Our analysis of metal concentrations in 721 fur samples from three species of flying foxes captured at eight sites across Australia aligns well with a limited number of prior studies showing low levels of metal exposure among frugivorous bats [185, 209]. In one

phylogenetic comparative study of 29 bat species, frugivores generally had the lowest total mercury concentrations in fur, typically ranging from 10-100 ng/g, compared with concentrations of 1000-34000 ng/g for most insectivores [210]. Similarly, median total mercury concentrations reported here were 18.9, 25.1, and 36.8 ng/g for BFF, GHFF, and SFF respectively. Low mercury concentrations for frugivorous mammals in general has been attributed to low diet connection to aquatic ecosystems, where mercury contamination can reach biologically significant levels. Another study reported mean concentrations of lead in fur for three groups of Australian flying foxes: 1) urban with liver/kidney concentrations of lead higher than toxic cutoffs in domestic animals (20750 ng/g), 2) urban with liver/kidney concentrations below these cutoffs (5820 ng/g), and 3) non-urban (850 ng/g) [211]. Median lead concentrations in our study were 1260, 1640, and 2260 ng/g for BFF, GHFF, and SFF respectively, which are consistent with the two groups of bats that did not have toxic concentrations of lead in their livers or kidneys. Importantly, our study provides a substantial dataset for future comparisons of metal concentrations in flying foxes that are currently experiencing a rapidly changing landscape in Australia.

Controlling for site, we observed significant species-level differences in concentrations of several metals. SFF and BFF typically had higher metal concentrations in fur than GHFF. These differences might be due to dietary differences between flying fox species [38, 203], or might reflect differences in species distribution. In particular, SFF have a limited geographic range (far North Queensland [38]). The tropical rainforest habitat of the SFF might have naturally higher levels of some metals than the sclerophyll (e.g. eucalypts) habitat of the GHFF and BFF captured in this study; alternatively, the

two sites at which we caught SFF could be near an unknown anthropogenic toxicant source.

In support of the idea that human mediated landscape-level changes, including urbanization and industrial activities, affect fruit bat toxicant exposure, we found a significant, positive association between metal PC1 score (i.e., overall metal load) and human footprint values surrounding capture sites. This is consistent with a previous report of higher Pb concentrations in fur, bones, and tissues of Australian flying foxes from urban areas, in which urban bats could have been exposed to greater atmospheric deposition from car and industrial emissions [211]. We also found generally lower concentrations of metals in GHFF captured in Adelaide in summer (February) compared to those captured in winter (August). Although the timing of fur moult might influence metal concentrations [212-214], we found the opposite effect than would be expected (i.e., lower concentrations after moulting), given that moult has been reported to occur in June for GHFF [215]. Instead, these seasonal differences in metal levels might also be linked to land use; specifically, seasonal differences in food availability could drive changes in foraging movements. Blossoming of flowering species can be scarcer during winter [118], leading bats to forage more on reliable urban resources [111], which could expose them to more pollution. While our findings suggest that land use surrounding bat roosts can influence metal concentrations, flying foxes are highly mobile and can move long distances between roosts [110]. Therefore, although we know the sites at which bats were captured, we cannot determine how long a bat has stayed at that site or whether bats from urban roosts are foraging within the urban environment.

In contrast to previous work [211, 214], we collected biological samples from live, outwardly healthy bats, suggesting that the metal concentrations we detected were not sufficient to cause acute toxicity. However, exposure to metals can have sub-lethal effects. Indeed, we found that higher concentrations of chromium and nickel in fur were associated with greater ectoparasite burden. In experimental studies, chromium and nickel have been demonstrated to have immunosuppressive effects in small mammals, sometimes causing increased susceptibility to infection and mortality [216, 217]; it seems plausible that these metals might reduce flying foxes' immune defenses against ectoparasites. Another explanation might be that higher metal concentrations cause lethargy in flying foxes, resulting in lower grooming rates and higher ectoparasite loads. Finally, instead of a direct link between metal concentrations and ectoparasites, particular bat roosts could independently have both higher metal concentrations and ectoparasites.

In contrast, higher concentrations of cobalt and mercury were significantly associated with lower ectoparasite burden. One possible explanation is that these metals could be toxic to the parasites themselves. A study of mallard ducks found that birds with higher lead concentrations had both lower intensity and richness of helminths, which was attributed to direct toxic effects of lead on helminths or upregulation of duck immune function by lead [218]. Alternatively, higher concentrations of cobalt or mercury could stimulate bat activity. For instance, common loon chicks with higher mercury concentrations in blood spent less time riding on their parents' backs and more time preening [219]. A third explanation is that ectoparasites could be acting as sinks for metals; however, this phenomenon is more typically observed with helminths [220]. Future work testing metal concentrations in bat ectoparasites would be valuable.

Bats as a group face many stressors, including toxicant exposure, habitat loss, climate change and extreme weather events, disease, and hunting by humans [25, 182]. Our work shows that even frugivorous bats, which are expected to have fewer dietary ties to toxicants, still face exposure to many metals, and that at least some of these metals carry a physiological cost in the form of greater parasitism. Looking forward, there is a crucial need for research on immune effects of toxicants in bats. For example, a study of vampire bats in Belize linked higher concentrations of total mercury in fur to higher neutrophil counts and weaker bacterial killing ability [221]. In Australia, flying foxes (in particular BFF and SFF) can transmit deadly Hendra virus to horses and then to humans; transmission events typically occur in peri-urban areas, where bats are drawn due to planted food resources [222]. If these urban areas are also where flying foxes face the most toxicant exposure, this could contribute to bat susceptibility to infection and viral shedding, which might increase the potential for spillover infections.

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Table 4.1. Capture site, date captured, species, sex, and age class of flying foxes from which fur samples were analyzed for metal concentrations. BFF: black flying fox, GHFF: grey-headed flying fox, SFF: spectacled flying fox.

State	Site	Capture date	Species			Sex		Age class			Total
			BFF	GHFF	SFF	Female	Male	Adult	Subad.	Juv	
New South Wales	Tamworth*†	July 2015	0	24	0	8	16	10	10	4	**24**
	Woolgoolga*†	July 2015	1	22	0	9	14	19	4	0	**23**
Queensland	Goldsborough*	June 2015	0	0	11	3	8	11	0	0	**11**
	Hervey Bay	July 2018	15	45	0	28	32	39	12	9	**60**
	Redcliffe	May 2018	53	6	0	30	29	38	7	14	**190**
		July 2018	45	26	0	43	28	18	10	43	
		Sept. 2018	60	0	0	27	33	43	7	10	
	Tolga*†	June 2015	0	0	35	19	16	31	1	3	**35**
	Toowoomba	June 2018	46	2	0	28	20	25	5	18	**171**
		July 2018	63	3	0	37	29	35	16	11	
		Sept. 2018	56	1	0	26	31	26	7	24	
South Australia	Adelaide	Aug. 2016	0	49	0	28	21	Not assessed			**207**
		Feb. 2017	0	57	0	40	17				
		Aug. 2017	0	48	0	33	15				
		Feb. 2018	0	53	0	36	17				
Total			**339**	**336**	**46**	**395**	**326**	**295**	**79**	**136**	**721**

*Urine samples collected at this site

†Blood smears collected at this site

Table 4.2. Loadings of 13 metals measured in flying fox fur onto three principal components retained after parallel analysis.

Metal	**PC1**	**PC2**	**PC3**
antimony	0.35	0.10	0.21
arsenic	0.28	0.34	0.11
cadmium	0.21	-0.074	-0.57
chromium	0.35	-0.00088	0.15
cobalt	0.13	-0.55	-0.35
copper	0.38	0.12	0.15
lead	0.33	0.15	-0.25
mercury	0.019	0.20	-0.42
nickel	0.16	-0.23	-0.073
selenium	0.27	0.17	-0.33
strontium	0.21	-0.51	0.17
tin	0.37	0.15	0.11
vanadium	0.28	-0.35	0.23

Table 4.3. Output for linear models to examine the effect of season, sex, age and body condition (WFR: weight to forearm length ratio) on metal concentrations in fur of grey-headed flying foxes in Adelaide, South Australia. *n* = 202 for all metals except selenium (*n* = 200) and mercury (*n* = 195).

Metal	**Term**	**Estimate**	**SE**	**t**	**p**
antimony	season(summer)	-0.38	0.070	-5.45	**1.5 e-7**
	sex(male)	0.086	0.065	1.33	0.19
	WFR	-0.090	0.054	-1.66	0.10
	age	0.016	0.021	0.78	0.44
arsenic	season(summer)	-0.40	0.089	-4.51	**1.1 e-5**
	sex(male)	-0.052	0.083	-0.63	0.53
	WFR	0.088	0.070	1.26	0.21
	age	0.035	0.027	1.32	0.19
cadmium	season(summer)	-0.68	0.079	-8.55	**3.5 e-15**
	sex(male)	-0.17	0.074	-2.27	**0.02**
	WFR	0.062	0.062	1.01	0.31
	age	0.032	0.024	1.33	0.19
chromium	season(summer)	-0.30	0.083	-3.62	**3.8 e-4**
	sex(male)	0.11	0.077	1.45	0.15
	WFR	-0.049	0.065	-0.76	0.45
	age	0.023	0.025	0.91	0.36
cobalt	season(summer)	-0.29	0.084	-3.38	**8.7 e-4**
	sex(male)	-0.089	0.078	-1.14	0.26
	WFR	-0.059	0.066	-0.89	0.37
	age	0.040	0.025	1.56	0.12
copper	season(summer)	-0.33	0.045	-7.19	**1.3 e-11**
	sex(male)	-0.017	0.042	-0.41	0.69
	WFR	-0.045	0.035	-1.28	0.20
	age	-0.0079	0.014	-0.58	0.56
lead	season(summer)	-0.87	0.081	-10.68	**<2 e-16**
	sex(male)	-0.12	0.075	-1.55	0.12
	WFR	-0.049	0.063	-0.77	0.44
	age	0.0068	0.024	0.28	0.78
mercury	season(summer)	0.32	0.077	4.18	**4.5 e-5**
	sex(male)	-0.30	0.071	-4.30	**2.8 e-5**
	WFR	0.42	0.060	6.99	**4.5 e-11**
	age	0.062	0.023	2.66	**0.01**

nickel	season(summer)	0.38	0.18	2.14	**0.03**
	sex(male)	-0.22	0.17	-1.31	0.19
	WFR	0.19	0.14	1.36	0.18
	age	-0.088	0.054	-1.63	0.10
selenium	season(summer)	0.0051	0.078	0.066	0.95
	sex(male)	-0.024	0.071	-0.35	0.73
	WFR	0.071	0.061	1.15	0.25
	age	0.028	0.023	1.21	0.23
strontium	season(summer)	-0.45	0.092	-4.86	**2.3 e-6**
	sex(male)	-0.18	0.085	-2.13	**0.04**
	WFR	0.041	0.072	0.57	0.57
	age	0.029	0.028	1.05	0.29
tin	season(summer)	-0.21	0.043	-4.96	**1.5 e-6**
	sex(male)	-0.035	0.040	-0.89	0.38
	WFR	-0.089	0.033	-2.66	**0.01**
	age	0.032	0.013	2.46	**0.02**
vanadium	season(summer)	-0.11	0.086	-1.26	0.21
	sex(male)	-0.0026	0.080	-0.033	0.97
	WFR	-0.14	0.067	-2.09	**0.04**
	age	0.040	0.026	1.56	0.12

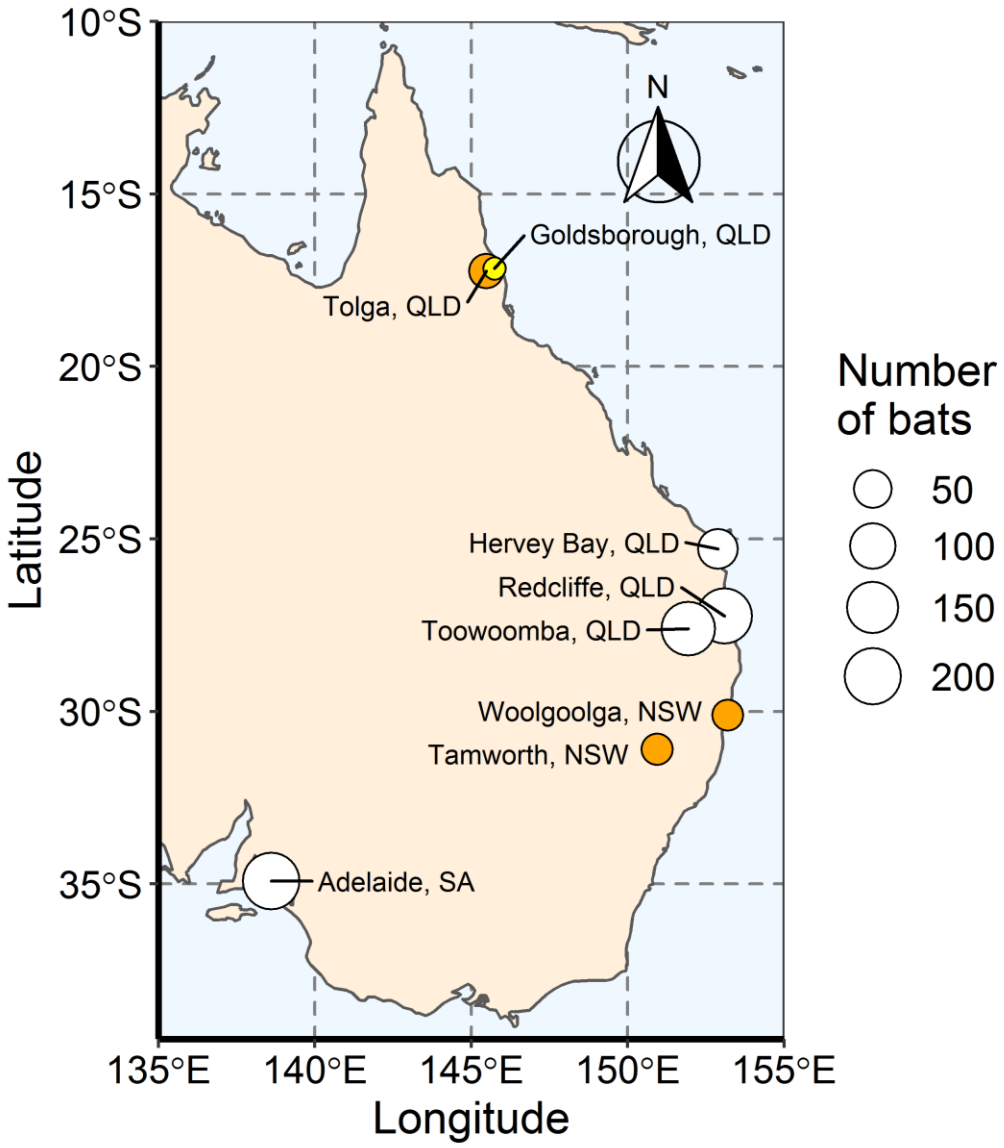

Figure 4.1. Map of eastern Australia showing the eight sites where flying foxes were captured. The area of each bubble corresponds to the number of flying foxes sampled from that site, with further details provided in Table 4.1. Fur samples were collected from flying foxes captured at all sites. Urine and blood smears were collected for sites in orange. Urine was collected for sites in yellow.

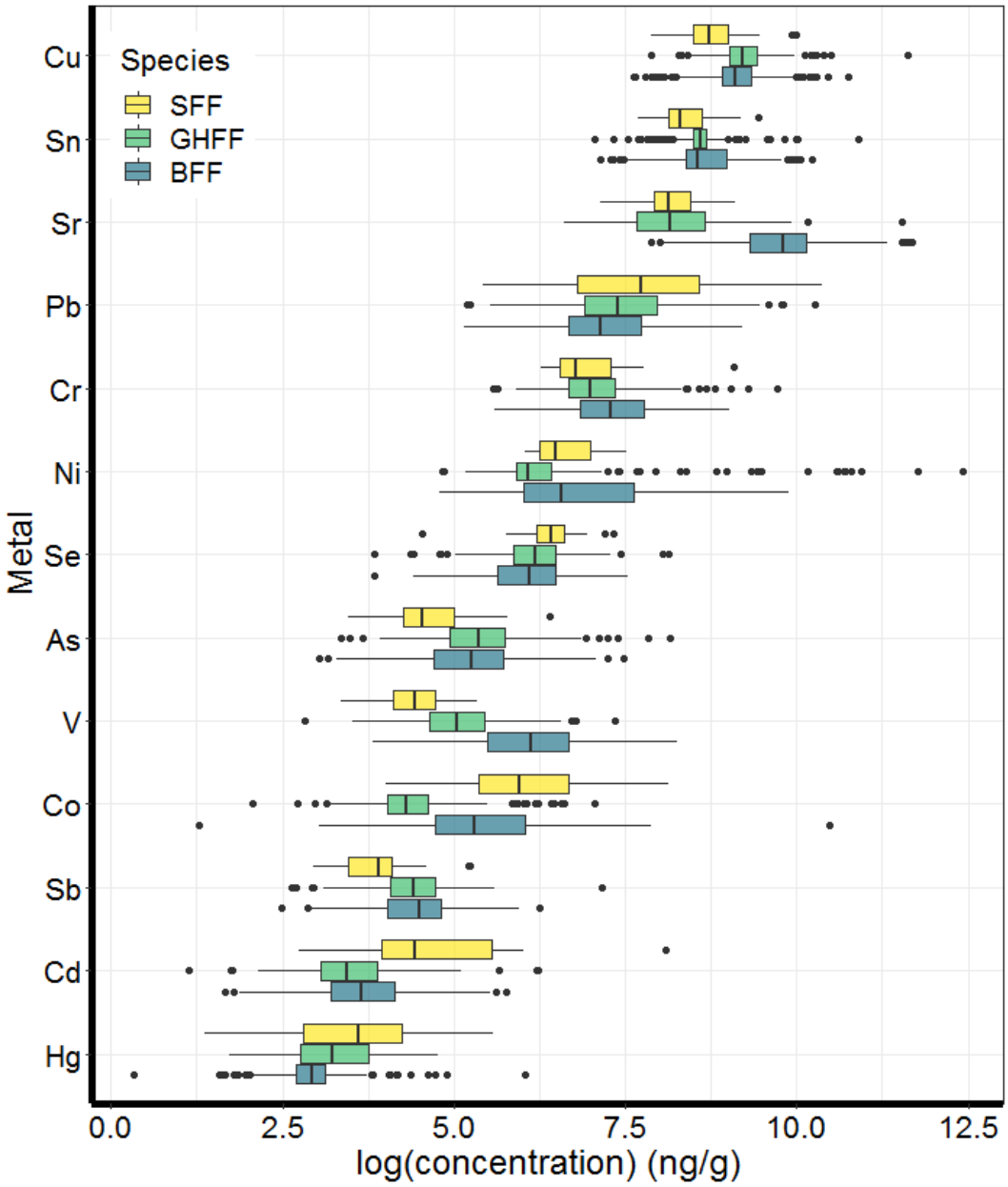

Figure 4.2. Boxplots showing log-transformed concentrations of 13 metals measured in flying fox fur. Sample sizes for each species–metal combination can be found in Appendix C Table S4.1. In each boxplot, the middle line represents the median value, box represents the interquartile range, the whiskers extend to 1.5 times the interquartile range, and any points beyond this range are plotted separately. BFF: black flying fox, GHFF: grey-headed flying fox, SFF: spectacled flying fox

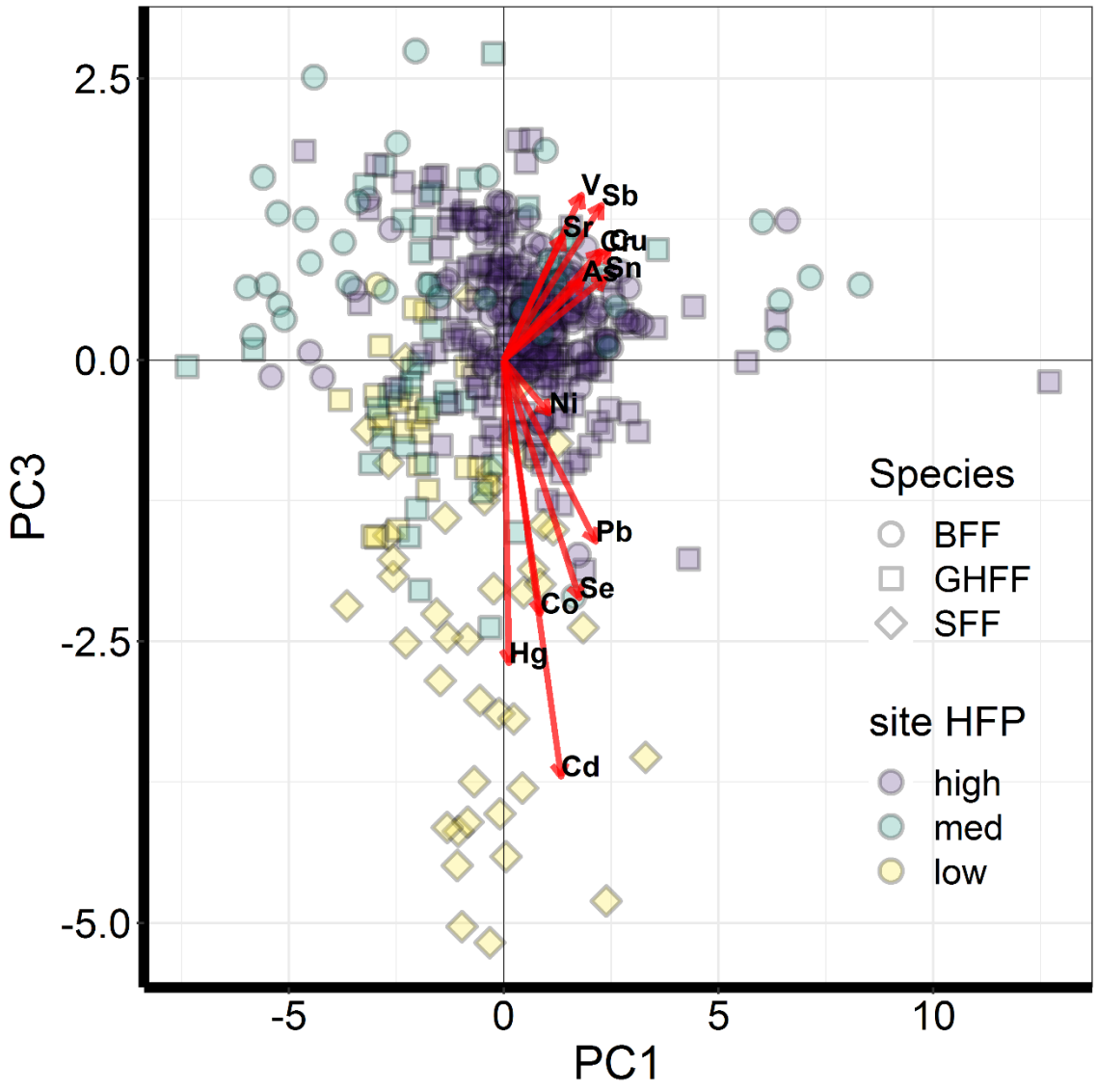

Figure 4.3. Biplot of PC1 versus PC3 with loadings of 13 metals measured in 402 flying fox fur samples. The shape of each point represents species (BFF: black flying fox, GHFF: grey-headed flying fox, SFF: spectacled flying fox). Each point is colored according to the Human Footprint (HFP) score of the capture site. Scores were condensed into three categories: high (Adelaide and Redcliffe), medium (Tamworth, Toowoomba, and Hervey Bay), and low (Woolgoolga, Tolga, and Goldsborough).

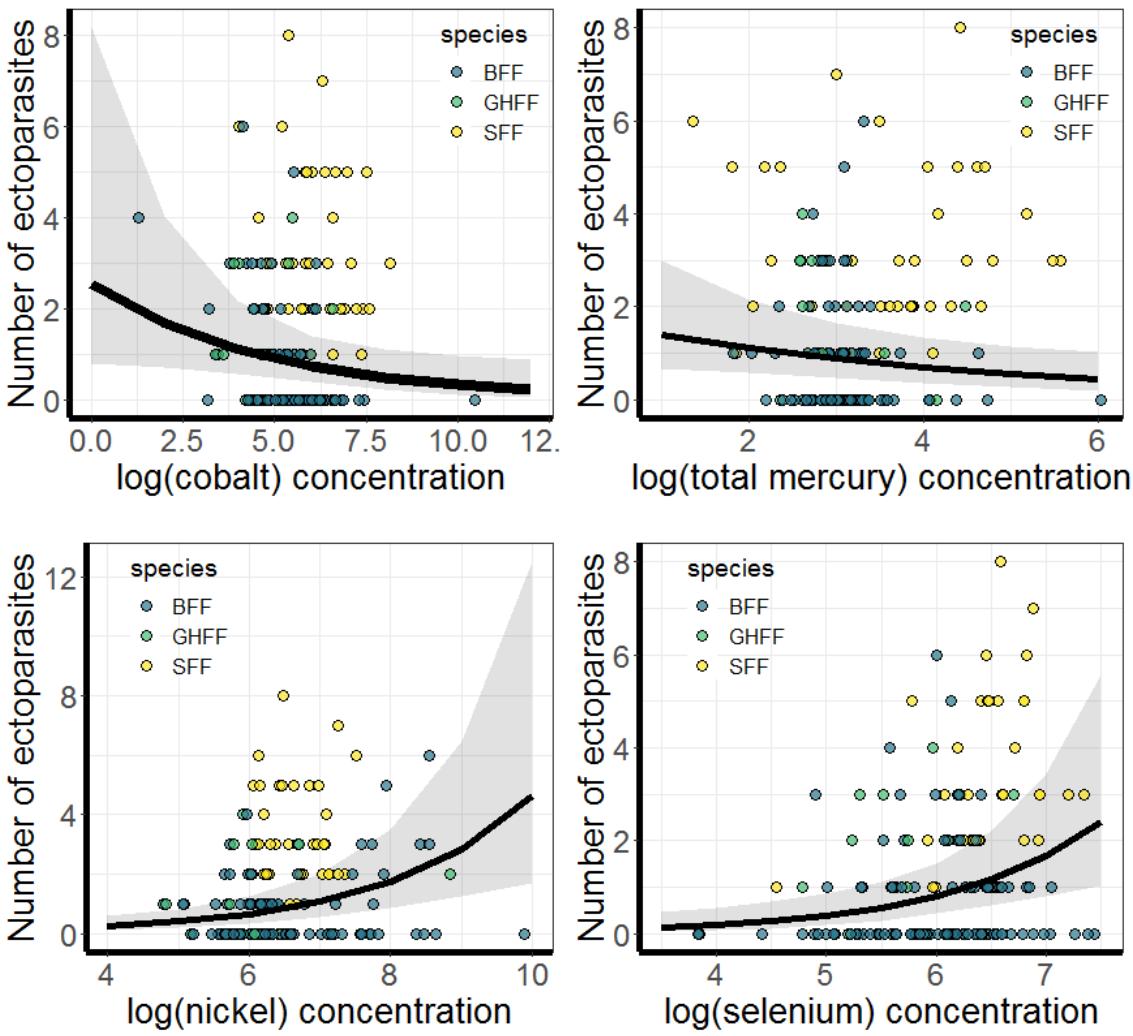

Figure 4.4. Predicted counts of ectoparasites as a function of cobalt, total mercury, nickel, and selenium concentrations in flying fox fur from each respective GLMM. Raw data are overlaid as points. All metal concentrations are log-transformed and in units of ng/g. BFF: black flying fox, GHFF: grey-headed flying fox, SFF: spectacled flying fox.

CHAPTER 5

LANDSCAPE-LEVEL TOXICANT EXPOSURE MEDIATES INFECTION IMPACTS ON WILDLIFE POPULATIONS[4]

[4] Sánchez CA, Altizer S, Hall RJ. To be submitted to *Biology Letters.*

ABSTRACT

Anthropogenic landscape modification such as agricultural intensification and urbanization can expose wildlife populations to toxicants that have profound effects on their health and behavior. In particular, toxicants can have complex interactions with infection dynamics and animal movement. We developed a mechanistic model to understand the interactive consequences of pathogens and toxicants on a wildlife population, by exploring toxicant effects on host movement, survival, and pathogen transmission in a toxicant-contaminated landscape. We found that the proportion of the landscape contaminated by toxicants was a crucial determinant of pathogen impacts. When a small fraction of the landscape was contaminated, costs to movement and survival from toxicant exposure trapped infected animals in contaminated habitats and reduced landscape-level transmission, especially if toxicant exposure reduced infection success. However, these effects were reversed when the majority of the landscape was contaminated. Intermediate levels of landscape contamination minimized infection prevalence and maximized the density of infected hosts in contaminated habitat, a proxy for the risk of pathogen spillover to humans. These results highlight how sublethal effects of toxicants can be crucial determinants of pathogen impacts on wildlife populations that may not manifest until landscape contamination is widespread.

INTRODUCTION

Anthropogenic toxicants such as persistent organic pollutants, heavy metals, and pharmaceutical products are widespread [223-225] and have profound effects on wildlife health and behavior [178, 226-228]. Urban wildlife populations tend to have higher toxicant loads than nonurban populations [172] and may be especially at risk of negative health effects. Toxicant exposure has been linked to increased infection susceptibility due to immune suppression [229], notably in marine mammals [230, 231] but also birds [232], amphibians [233], and fish [234]. For instance, green frog (*Rana clamitans*) tadpoles exposed to pesticides experienced greater encystment by trematode cercariae [235], which was attributed to immunosuppressive effects of the pesticides. Toxicants might also increase infection in a population by inducing greater production of infectious stages, as observed in snails that shed more cercariae when exposed to the herbicide glyphosate [236].

However, toxicants could also reduce infection in a wildlife population, such as by killing or depressing the production of free-living parasite stages (e.g. pesticide effects on the amphibian fungus *Batrachochytrium dendrobatidis* [237]). Toxicants can also reduce aggression [238], decreasing the likelihood of infectious contacts between individuals. Heavy metals can upregulate immune function in some species (e.g. lead in mallards [218], copper in blow flies [239], which could reduce susceptibility to parasites. A mechanism that could reduce transmission of density-dependent pathogens is toxicant-driven declines in host density [240, 241].

Sometimes toxicants and infection act in concert to produce unexpected effects [242]. Their combination may be detrimental to a host, as observed with juvenile

roundhead galaxias (*Galaxias anomalus*) that exhibited no changes in survival when exposed to a trematode parasite or glyphosate singly, but reduced survival when exposed to both [236]. In other cases, toxicants and infection together may benefit a host. For instance, 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 [243], while bumblebees (*Bombus terrestris*) inoculated with a microsporidian parasite and exposed to a pesticide exhibited improved learning [244]. These studies underscore the importance of considering the effects of toxicants and infection on wildlife together, rather than in isolation.

In addition to altering infection outcomes, toxicants have been shown to affect animal movement, both in the laboratory (e.g. decreased and increased activity in fish exposed to heavy metals [245]) and the wild (e.g. lower flight height and movement rate in golden eagles (*Aquila chrysaetos*) exposed to lead [246]). Toxicants can reduce movement directly by causing physical deformities [90, 174] or indirectly (e.g. impaired memory and collision avoidance in insects exposed to neonicotinoid pesticides [247, 248]). If habitats contaminated by toxicants attract wildlife (e.g. agricultural crops treated with pesticides [249]; artificial wetlands constructed to treat wastewater [250]), but then impair subsequent movement, these habitats could act as ecological traps. For example, 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 [173].

Although previous work has explored how toxicants can interact with infection at a local scale [240, 242, 251], it is currently unknown how the ubiquity of toxicant-

contaminated habitats across landscapes used by wildlife influence population viability and infection dynamics at larger scales, especially when toxicants have sublethal effects on infection susceptibility and animal movement. Here, we develop a compartmental model of host-parasite dynamics to study joint effects of toxicants and infection for a wildlife species whose range includes toxicant-contaminated habitat. We further place our model in the context of urbanizing landscapes, where we expect more humans and higher toxicant levels (e.g. due to industrial pollution, transportation emissions, pesticide application). Specifically, we explore how population and infection dynamics respond to varying 1) the amount of toxicant-contaminated landscape and 2) the effects of toxicants on infection, movement, and survival. We examine three outcomes of interest: population size, infection prevalence, and the density of infected animals in toxic habitat as a proxy for the risk of pathogen spillover from animals to humans.

METHODS

Model development and parameters

We categorize animals according to their infection status and location as susceptible (S) or infected (I), with the subscripts T and P denoting occupancy of "toxic" habitat (contaminated by toxicants) or "pristine" habitat (free from toxicants). The parameter f represents the fraction of the overall landscape that is contaminated; thus $1 - f$ represents the fraction of the landscape that is pristine. Animals in toxic habitat potentially incur costs to fecundity, survival, and movement, and increased or decreased transmission risk. A schematic of the model is provided in Figure 5.1.

Demography: Animals are born into the susceptible class at a density-dependent rate. In both habitat types the maximum per capita birth rate is b_0 and the density-dependent term in each type reflects the relative amount of pristine and toxic habitat (b_1 / $(1 - f)$ and b_1 / f respectively). In pristine habitat, susceptible animals experience mortality at rate m. In toxic habitat, susceptible animals experience mortality at rate m / $(1 - c_m)$, where c_m represents the mortality cost imposed by negative effects of toxicants.

Infection: Density-dependent pathogen transmission occurs in pristine and toxic habitat at respective rates β_P and β_T. In pristine habitat, infected animals experience mortality at rate $m + \mu$, where μ is the additional disease-induced mortality. In toxic habitat, infected animals experience mortality at rate $(m + \mu) / (1 - \alpha c_m)$, where α controls the net effect of being infected while in toxic habitat on survival. If α is < 1, being in toxic habitat offsets the negative consequences of infection (e.g. toxicants stimulate immune defense), while if α is > 1, being in toxic habitat amplifies disease-induced mortality (i.e. greater than additive effects of toxicants and disease on survival). In both habitat types, animals recover from infection at rate γ.

Movement: Animals disperse at per capita rate σ in pristine habitat and $\sigma (1 - c_\sigma)$ in toxic habitat, where c_σ is the cost of toxicants on dispersal. The probability that animals switch habitats during dispersal depends on the relative frequencies of each habitat type. If an animal moves between habitats, it maintains its infection status. However, when an animal disperses out of toxic habitat, for simplicity we assume it immediately "recovers" from all ill effects of toxicants. The differential equations describing the model are provided below, and a summary of model parameters and their default values appears in Table 5.1.

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

$$\frac{dI_P}{dt} = \beta_P S_P I_P - (m+\mu)I_P - \gamma I_P - \sigma f I_P + \sigma(1-c_\sigma)(1-f)I_T \quad \text{Eq. 2}$$

$$\frac{dS_T}{dt} = (b_0 - \frac{b_1(S_T+I_T)}{f})(S_T + I_T) - \frac{m}{1-c_m}S_T - \beta_T S_T I_T + \gamma I_T + \sigma f S_P - \sigma(1-c_\sigma)(1-f)S_T \quad \text{Eq. 3}$$

$$\frac{dI_T}{dt} = \beta_T S_T I_T - \frac{(m+\mu)}{1-\alpha c_m}I_T - \gamma I_T + \sigma f I_P - \sigma(1-c_\sigma)(1-f)I_T \quad \text{Eq. 4}$$

Model parameterization and analysis

Our model was motivated by a hypothetical flying fox (fruit bat) host species infected with a virus. These animals feed on fruiting and flowering plant species in natural, urban, and agricultural landscapes [35, 110], where they face exposure to toxicants including pesticides and heavy metals [184, 185]. In natural forested landscapes, flying foxes roost communally in camps and periodically relocate as food sources are depleted [109, 252]. In human-altered habitats where food availability is more stable (and where toxicant exposure is more likely), camps persist longer through time [36]. Additionally, flying foxes are reservoirs of pathogens that can be transmitted to domestic animals and humans, notably Hendra and Nipah viruses [253]. In order for our model to apply more generally to other wildlife species, and given uncertainty on how viruses circulate in bats [254], we model transmission as a simple density-dependent process with no lasting immunity from infection. Further details of model parameterization are provided in Appendix D, describing demographic, dispersal, and infection parameters relevant to flying foxes and their viruses. To explore the generality of our findings to other systems,

extensive sensitivity analyses were performed by co-varying multiple model parameters using Latin Hypercube Sampling [255].

We examined three outcomes of interest in our analyses. The landscape-level effects of toxicants and infection on population viability were quantified by the equilibrium population size, $N^* = S_P + I_P + S_P + I_T$. The net effect of contaminated habitat on landscape-level infection processes was quantified by the equilibrium infection prevalence, $P^* = (I_P + I_T) / N^*$. We defined "spillover risk" as the density of infected animals in toxic habitat, since we assumed this is where humans and wildlife are most likely to co-occur. Spillover risk was quantified as the total number of infected animals in toxic habitat divided by the habitat's relative frequency, $\rho = I_T^* / f$, and thus represents the total number of infected animals if 100% of the landscape was contaminated.

For each landscape contamination scenario, we initiated the model with 50,000 hosts, 100 of which were infected with a virus; hosts were distributed between toxic and pristine habitats according to the relative proportion of those habitats in the landscape. We imposed a low cost of toxicants on survival, and a moderate synergistic effect of infection and toxicants on survival. We explored six cases in which we co-varied the pathogen transmission rate in toxic habitat relative to pristine habitat, and the cost of toxicants on dispersal. The three scenarios for transmission were: the pathogen is less transmissible in toxic habitat ($\beta_T < \beta_P$); no difference in transmission between habitat types ($\beta_T = \beta_P$); and the pathogen is more transmissible in toxic habitat ($\beta_T > \beta_P$). For these three scenarios the cost of toxicants for host dispersal was either low ($c_\sigma = 0.2$) or high ($c_\sigma = 0.8$). All other model parameters were held constant across simulations (Table 5.1).

To explore how wildlife population and infection dynamics are affected by increasing contamination on the landscape, we varied the fraction of the landscape that is contaminated, 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 the population size, infection prevalence, and spillover risk after 50 years (simulations revealed that 50 years was sufficient for populations and infection to reach an equilibrium). All model analyses were performed in R version 3.6.1 [127], and we used the *deSolve* package [256] to solve the system of differential equations.

We also explored the sensitivity of infection prevalence to parameter variation using Latin hypercube sampling. We varied five parameters related to toxicants, infection, and movement: transmission in toxic habitat (β_T), infection-induced mortality (μ), the cost of toxicants to movement (c_σ), the cost of toxicants to survival (c_m), and the net effect of being infected while in toxic habitat on survival (α); parameter ranges are provided in Appendix D Table S5.1. Using the *lhs* package [257], we generated 5000 samples from a Latin hypercube design in which parameters were distributed uniformly and retained samples for which $\alpha < 1/c_m$. To derive partial rank correlation coefficients (PRCCs) between parameters and equilibrium infection prevalence we used the *sensitivity* package [258]. We performed sensitivity analyses for three values of f representing low, intermediate, and high levels of toxic habitat in the landscape.

RESULTS

Scenario 1: Toxicants have a small movement cost

We visualized population size, infection prevalence, and spillover risk as a function of *f*, the fraction of the landscape contaminated by toxicants (Figures 5.2 and 5.3). In the absence of infection, host population size declines monotonically with the extent of landscape contamination (Figure 5.3A). However, the magnitude of the decline is low, even at high values of *f*, indicating largely sub-lethal effects of toxicant exposure in the absence of infection. In the presence of infection, population size initially increases with *f* at low levels of landscape contamination, then decreases (Figure 5.2). The initial increase is driven mainly by an increase in susceptible hosts in toxic habitat, reflecting reduced transmission at low host density and higher mortality of infected individuals there. Further, since toxicant exposure reduces dispersal rates, infected hosts are less likely to return to pristine habitat to transmit infection. As *f* increases and pristine habitat shrinks, density-dependent transmission and host population size in pristine habitat declines. In toxic habitat, reduced host dispersal capacity causes overcrowding, which reduces reproduction through density-dependent effects, and increases both transmission and mortality from combined effects of toxicants and infection. Together these result in overall population declines. When transmission risk is lower in toxic than pristine habitat, toxic habitat acts as a sink for the pathogen (Figure 5.3A, dotted line); host population size increases with *f* even when more than 50% of the landscape is contaminated, and relatively small population declines occur only when almost all of the landscape is contaminated. Conversely, when transmission is enhanced in toxic habitat (Figure 5.3A,

dashed line), the combined effects of toxicants and infection drive more severe population declines in an increasingly contaminated landscape.

Increasing the proportion of toxic habitat also has non-linear effects on infection prevalence (Figure 5.3B), with overall prevalence decreasing with *f* until more than half of the landscape is contaminated, and then increasing. This reflects the relatively large reduction in transmission in pristine habitat as the landscape becomes contaminated, which is outpaced by an increase in transmission in toxic habitat once it is the commonest habitat type. Prevalence drops further, and over a larger range of *f*, when transmission is lower in toxic habitat, while prevalence remains relatively high when habitat contamination increases transmission.

Spillover risk (i.e. the density of infected individuals in toxic habitat) has a hump-shaped relationship with landscape contamination (Figure 5.3C). When transmission is the same in each habitat type, spillover risk is highest when approximately 50% of the landscape is contaminated. Spillover risk is much lower, and maximized when most habitat is contaminated, when toxicants reduce transmission. Conversely, when toxicants increase transmission, peak spillover risk is higher and occurs at a lower value of *f* (i.e. before most of the landscape is contaminated). Across all scenarios, as an increasing fraction of the landscape becomes contaminated, population size is maximized first (i.e. at a lower value of *f*), followed by the peak in spillover risk, followed by the minimum in overall infection prevalence.

Scenario 2: Toxicants have a large movement cost

Increasing the cost of contamination on dispersal results in more severe impacts on population size across all transmission scenarios (Figure 5.3D). When transmission in toxic habitat is greater than or equal to transmission in pristine habitat, the population size decreases monotonically with increasing landscape contamination (Figure 5.3D). When transmission is lower in toxic habitat, population size initially decreases at very low *f*, but then increases with increasing *f*. However, infection still strongly regulates the population to less than 60% of the disease-free population size. Compared to when the cost to dispersal is low, infection prevalence drops more rapidly to a lower minimum (Figure 5.3E), and spillover risk increases more rapidly to a higher maximum (Figure 5.3F); minimum prevalence and maximum spillover risk both increase with the transmission rate in toxic habitat.

Sensitivity analyses

Increases in disease-induced mortality, costs of toxicants on survival, and combined effects of toxicants and infection on mortality had a strong negative effect on infection prevalence for all three levels of landscape contamination ($f = 0.1$, $f = 0.5$, and $f = 0.9$; Appendix D Figure S5.1). In contrast, increasing pathogen transmission in toxic habitat had a positive effect on prevalence. Increasing the cost of toxicants on movement had a negative effect on prevalence at low and intermediate levels of landscape contamination and no effect on prevalence at a high level of landscape contamination. Upper observed prevalence values from Latin Hypercube Sampling were ~0.8.

DISCUSSION

Many wild species are found in increasingly human-modified landscapes, potentially influencing their exposure to toxicants. We developed a mechanistic model to understand the consequences of toxicant exposure on host-pathogen dynamics, through sub-lethal effects of toxicants on host movement and behavioral or immunological effects on transmission risk, as well as synergistic lethal effects of infection and toxicant exposure. While toxicants had little negative effect on host population size in the absence of infection, we found that the extent of contaminated habitat across the landscape could enhance or reduce impacts of infection on host populations. Contaminated habitat can potentially act as a sink for pathogens when most habitat is pristine, but typically exacerbated pathogen-related declines once the majority of the landscape was contaminated. In landscapes that became increasingly contaminated over time, we consistently found that population declines preceded the 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 combined effects of infection and toxicants.

Unexpectedly, we found cases in which toxicants can benefit a wildlife population. Population size can increase when the landscape changes from being totally pristine to having a small fraction of toxic habitat. When rare, toxic habitat may reduce transmission of density-dependent pathogens, prevent infected animals from returning to pristine habitat if toxicants reduce movement, and purge infected individuals from the population through elevated mortality. Further, if toxicants lower transmission of a virulent pathogen, then increasing landscape contamination can lead to higher maximum

population size and a net increase in population compared to a pristine landscape. Moderate toxicant-induced movement costs conveyed some benefits to host populations 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 become overcrowded, reducing density-dependent fecundity and increasing toxicant-induced mortality of uninfected and infected individuals.

Our results suggest that animal species 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 been shown to have higher risk of limb malformations, likely due to pesticide exposure [259]. In one study, deformity rates of newly-metamorphosed toads reached nearly 50% at some sites, though it was unclear whether deformity was due to pesticides, heavy metals, parasite infection, or a combination of factors [90]. Future work could examine the impact of malformations on dispersal ability in amphibians, and investigate the degree to which other vertebrate species experience toxicant-induced deformities [260] or other impairments to movement.

A previous model that explored effects of environmental stressors (e.g. eutrophication, heavy metals) on host disease dynamics found that negative, positive, and non-linear relationships between stress and disease were possible, but that increasing stress generally reduced disease due to stress-mediated declines in host density [240]. This model assumed that contamination affected the entire environment, and that stress increased susceptibility to infection [240]. By incorporating toxic and pristine habitats in our model, and creating scenarios in which transmission could be lower in toxic habitat,

we found that host population size could increase while infection prevalence still decreased.

Our model made several simplifying assumptions that may not hold in other systems. For example, we assumed that an animal immediately recovers from all ill effects of toxicants if it moves out of toxic habitat. However, some toxicants can accumulate in the body (e.g. heavy metals like chromium and nickel [261]), which could potentially reduce or negate any beneficial effects of reduced pathogen exposure. Model extensions could allow toxicant concentrations to accumulate in hosts as they stay longer in toxic habitat, and for toxicants to decrease gradually when hosts leave. Adding different host age and sex classes could also allow inclusion of maternal transfer of toxicants (e.g. through placental transfer or lactation [262]). Additional work could also consider how movement could act as a stressor, revealing negative effects of toxicants. For instance, severe energy expenditure can mobilize toxicants stored in body fat, as observed in Mexican free-tailed bats (*Tadarida brasiliensis*) that experienced pesticide poisoning after being subjected to simulated migratory flight [263].

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. Only very low landscape contamination was beneficial for population size, as toxic habitat offered a refuge from infection. When considering population viability, wildlife managers should seek to 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.

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Table 5.1. Model parameters with definitions, units, and values used to produce figures.

Process	Parameter	Definition	Units	Value
Demography	m	Natural death rate	1/year	0.1
	b_0	Maximum per capita birth rate	1/host/year	0.4
	b_1	Density-dependent per capita birth rate	1/host/year	$(b_0-m)/50000$
	c_m	Cost of toxicants to survival		0.2
Infection	β_P	Transmission rate in pristine habitat		0.006
	β_T	Transmission rate in toxic habitat		0.0015, 0.006, 0.0105
	γ	Recovery rate	1/year	36.5
	μ	Disease-induced mortality	1/year	0.25
	α	Effect of infection and toxicants on survival		2
Movement	f	Fraction of the overall landscape that is contaminated		0.01-0.99
	σ	Per capita dispersal rate	1/movement/year	-log(0.1)
	c_σ	Cost of toxicants to dispersal		0.2, 0.8

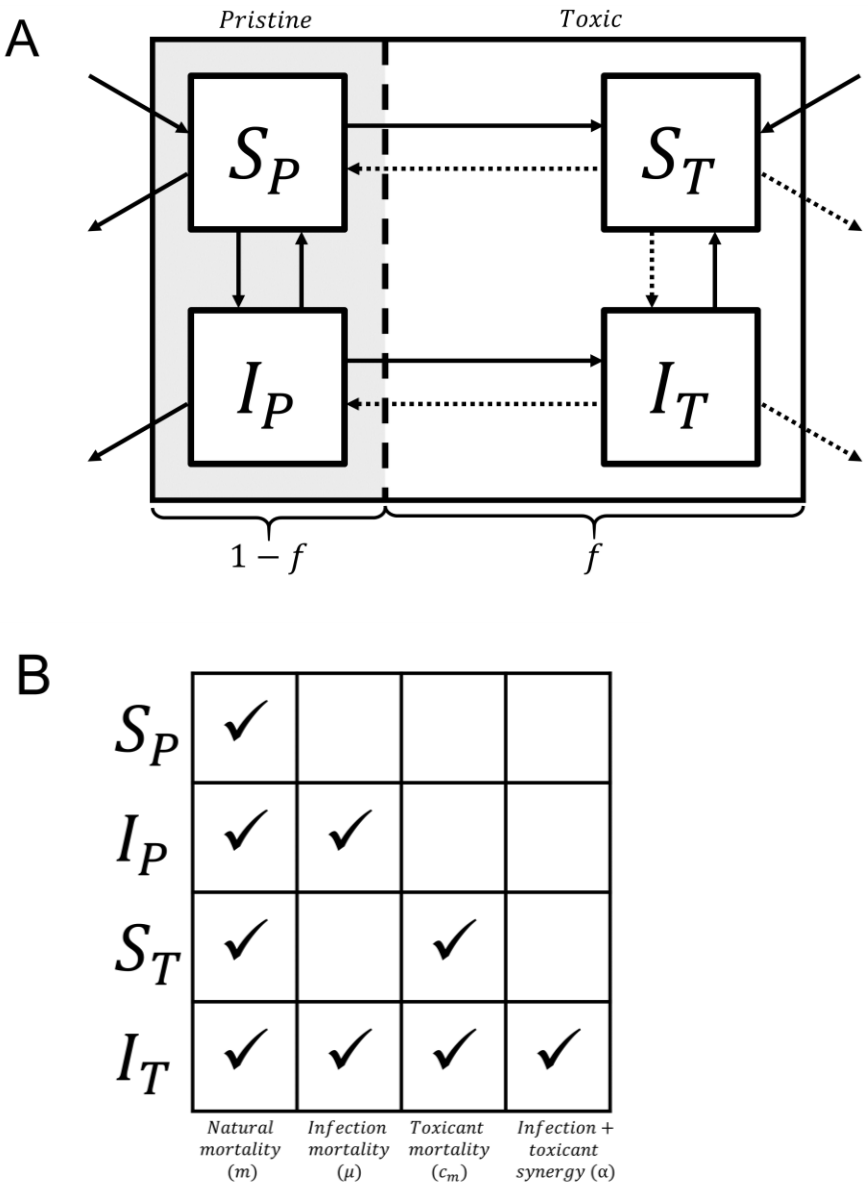

Figure 5.1. Schematic of the toxicant-infection model. **A.** Boxes represent the number of Susceptible (S) or infected (I) individuals in pristine (P) or toxic (T) habitat. The parameter *f* represents the fraction of the overall landscape that is contaminated by toxicants; thus 1 – *f* represents the fraction of the landscape that is pristine. Horizontal arrows represent movement between pristine and toxic habitats, vertical arrows represent transitions between the susceptible and infected classes (i.e. infection and recovery), and

diagonal arrows represent demographic processes (i.e. births and deaths). Dotted lines represent processes that could be affected by toxicants (movement out of toxic habitat, pathogen transmission in toxic habitat, and deaths in toxic habitat). **B.** Figure showing differential mortality based on infection status (susceptible or infected) and habitat type (pristine or toxic). In pristine habitat, susceptible animals experience only natural mortality, while infected animals experience natural and disease-induced mortality. In toxic habitat, susceptible animals experience natural mortality and mortality imposed by toxicants. Infected animals experience natural mortality, disease-induced mortality, and toxicant-induced mortality. Being infected while in toxic habitat can also produce greater than additive effects on mortality.

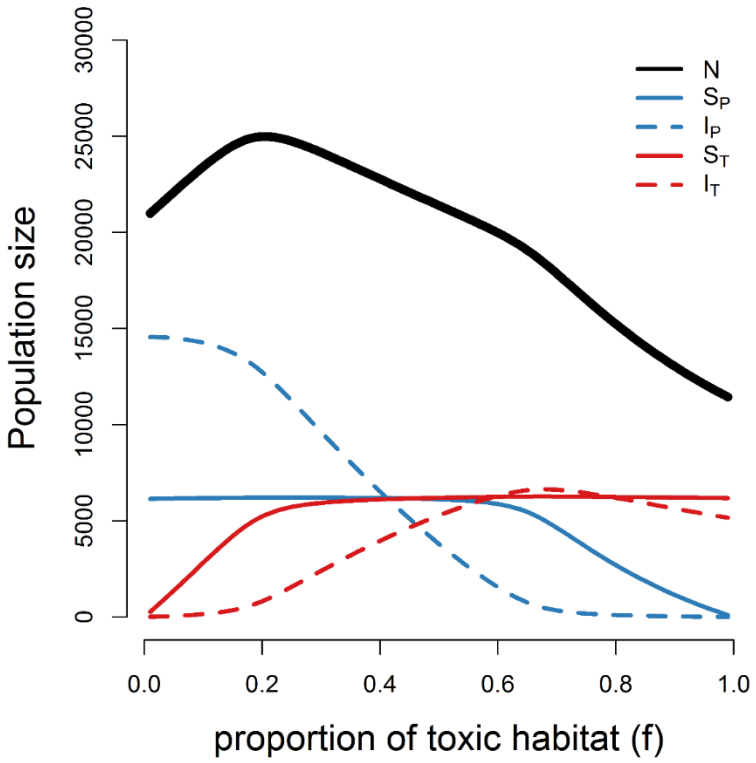

Figure 5.2. Equilibrium population sizes as a function of the proportion of toxic habitat in a population infected by a virulent pathogen. The overall population size across the landscape is denoted by the thick black line. Susceptible and infected host population sizes are denoted by solid and dashed lines, respectively, with the line color indicating the population size in pristine (blue) and toxic (red) habitats. Here, the transmission rate is assumed equal across habitat types ($\beta_T = \beta_P = 0.006$) and the cost to dispersal from toxic habitat is relatively low ($c_\sigma = 0.2$); other parameter values are provided in Table 5.1.

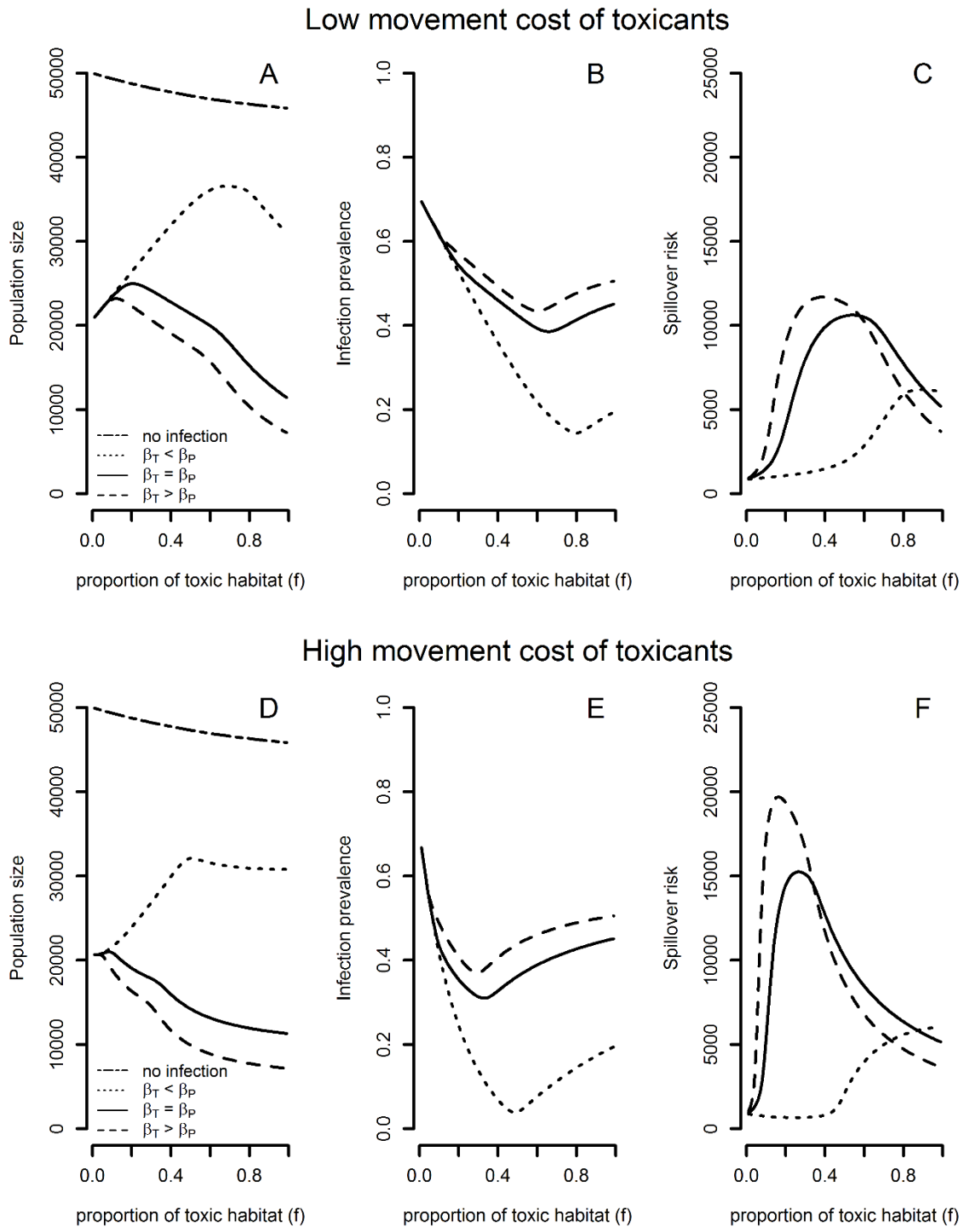

Figure 5.3. Population size, infection prevalence, and spillover risk (the density of infected animals in toxic habitat) plotted as a function of the proportion of toxic habitat in the landscape. Rows indicate scenarios where the cost to dispersal from toxic habitat is

(A-C) low (c_σ = 0.2) or (D-F) high (c_σ = 0.8). Pathogen transmission in pristine habitat is constant (β_P = 0.006). Line style indicates the three scenarios for transmission rate in toxic habitat: less than, equal to, or greater than β_P (β_T = 0.0015, 0.006, and 0.0105; dotted, solid, and dashed lines, respectively). Population size in the absence of infection is also shown for comparison in panels A and D (dashed-dotted line). Other parameter values are provided in Table 5.1.

CHAPTER 6

CONCLUSIONS

The overarching aim of this dissertation was to examine how urban landscape features, including altered resources and exposure to toxicants, affect wildlife behavior, health, and infectious disease. I first used meta-analytic techniques to quantify the impacts of urbanization on four wildlife health metrics (Chapter 2). I next tracked the foraging movements of urban flying foxes in Adelaide, South Australia and identified key predictors of their movement (Chapter 3). I then quantified metal concentrations in fur of flying foxes captured at eight locations across Australia and assessed relationships between metals and human land use, as well as flying fox parasitism (Chapter 4). Finally, I built a mechanistic model to explore interactive effects of toxicants and infectious disease on population size and disease dynamics of wildlife in an increasingly contaminated landscape (Chapter 5).

A main result of Chapter 2 was that comparisons of toxicant loads between urban and non-urban wildlife populations were heavily dominated by negative health relationships (i.e. greater toxicant concentrations in urban wildlife). However, our dataset had geographic and species biases, demonstrating gaps for future research. For instance, we identified few studies conducted outside of North America and Europe, and no health comparisons of urban and non-urban bat populations, even though bats make up approximately 20% of all mammal species [264]. In Chapter 4, I assessed metal concentrations in flying foxes captured at sites ranging in human impact, as measured by

human footprint scores. The results of this work supported the pattern found in Chapter 2, as flying foxes captured in sites surrounded by higher human impact had greater overall metal loads.

Fur samples were collected from live, outwardly healthy flying foxes, suggesting that the metal concentrations we measured were not sufficient to cause acute poisoning in the animals. Yet results of Chapter 5 show that in the presence of an infectious pathogen, sublethal effects of toxicants on movement and survival can cause a substantial decrease in wildlife population numbers. This is especially relevant to Australian flying foxes, as these animals host a variety of pathogens, among them Hendra virus and other paramyxoviruses [265]. I found that spectacled and black flying foxes typically had higher metal concentrations in fur than grey-headed flying foxes (Chapter 4); spectacled and black flying foxes are also considered primary reservoirs for Hendra virus [266, 267] suggesting that these species may be especially at risk of interactive toxicant—infection effects.

Metal exposure might also increase susceptibility of flying foxes to other stressors such as extreme heat events [268], cyclones [269], or food shortages [252]. Future work could compare metal concentrations, for example, in fur of flying foxes that survive or die during a heat stress event to test whether metals are a risk factor. Food shortages might be even more likely to reveal negative effects of metals or other toxicants on survival, as toxicants could be mobilized from storage in the body as resources are depleted. Determining the clinical effects of toxicants on flying fox health is an important topic for future research.

Though Chapter 2 demonstrated an overall negative effect of urbanization on wildlife health, it also provided ample support for health benefits, with nearly 40% (190/516) of records in the dataset reporting a positive urbanization–health relationship. Results from this work hinted that urban mammals may have better body condition than their non-urban counterparts. My finding that Adelaide flying foxes that foraged closer to the roost and at fewer sites were in better body condition (Chapter 3) suggests that one way urban areas improve wildlife body condition is by providing reliable food resources nearby, thereby decreasing energy expenditure. Though there has been some research comparing nutritional values of agricultural versus native fruits [42], future work to assess nutritional content of urban resources used by flying foxes would be valuable, as would more nuanced measures of body condition for flying foxes. Extending to other species, body condition was the least studied health metric in the meta-analysis dataset (12% of records; Chapter 2), revealing a need for more research on how body condition is affected by urbanization. Improved methods to assess body condition for wildlife would be an important first step to understand how condition varies in urban and non-urban populations [63].

In Chapter 5, we assumed that toxicant exposure and pathogen transmission occur within the same habitat; for example, animals drawn to a constructed wetland used to treat wastewater could be exposed to heavy metals and be infected by conspecifics. However, for central-place foragers such as flying foxes, which roost at one site during the day but forage at several sites during the night, toxicant exposure could occur separately from infection (e.g. exposure to pesticides at feeding sites, but infection at the roost where bats are aggregated). Future work could explore modeling frameworks that

explicitly compare infection dynamics for species that roost communally and disperse to forage across contaminated and uncontaminated habitats. In general, it would be useful to investigate the routes by which different species uptake different toxicants.

In the meta-analysis of Chapter 2, urban populations of birds and mammals exhibited a (statistically non-significant) pattern of having less parasitism than non-urban populations. Similarly, results of Chapter 5 showed that increasing landscape contamination caused a decline in infection prevalence over low to intermediate levels of contamination. However, at high landscape contamination, infection prevalence began to rise again. The studies included in our meta-analysis dataset ranged in urbanization, as measured by human footprint scores (0.7 to 24.9), but did not represent the upper limits of urbanization (maximum human footprint value of 50). Future work comparing parasitism measures of urban and non-urban wildlife might reveal non-linear or positive relationships between urbanization and infection if wildlife are captured at sites encompassing a larger urbanization gradient.

As more wildlife are acclimating to urban areas, it is essential to understand how their foraging resources, behavior, and health are changing. Seeing urban wildlife can excite and inspire humans, but can also cause conflict. Depending on the species, we may want to attract some animals to urban areas by providing resources that can sustain healthy populations, yet exclude others by making urban habitat less attractive or providing resources in natural areas. Flying foxes are one example of a group that may increasingly require new management strategies to keep populations healthy while reducing public health risk; this dissertation provides new data that could help guide management decisions.

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APPENDIX A

CHAPTER 2 SUPPLEMENTAL INFORMATION

Supplemental methods

Systematic search details

We identified published studies on urban wildlife health through Web of Science and CAB Abstracts using the following search strings: (*urban* OR city OR cities OR town) AND (health OR disease OR pathogen OR infect* OR *parasite OR bacteria* OR virus OR fung* OR ecto* OR helminth* OR condition OR survival OR stress OR tox*) AND (wild*), with restrictions on research area. The Web of Science search was restricted by language (ENGLISH), citation index (SCI-EXPANDED), and research area (ECOLOGY OR EVOLUTIONARY BIOLOGY OR ENVIRONMENTAL SCIENCES OR VETERINARY SCIENCES OR BIODIVERSITY CONSERVATION OR ZOOLOGY OR PARASITOLOGY OR PUBLIC ENVIRONMENTAL OCCUPATIONAL HEALTH OR INFECTIOUS DISEASES OR MICROBIOLOGY OR TOXICOLOGY OR PATHOLOGY OR MYCOLOGY OR IMMUNOLOGY OR HEMATOLOGY OR ENVIRONMENTAL STUDIES OR ENTOMOLOGY OR BIOTECHNOLOGY APPLIED MICROBIOLOGY OR GASTROENTEROLOGY HEPATOLOGY OR VIROLOGY OR ONCOLOGY OR BIOLOGY OR ORNITHOLOGY OR ENDOCRINOLOGY METABOLISM OR URBAN STUDIES). The CAB Abstracts search was restricted by language (English), publication type (academic journals), and research domain (ecology and environmental sciences).

Statistical analysis

To assess differences in research effort over time, we tabulated the number of records per year and by taxonomic group and health metric. We fit two generalized linear models

with Poisson errors, one with the interaction between year and taxonomic group and another with the interaction between year and health metric.

To examine relationships between urbanization and wildlife health, we used a hierarchical phylogenetic meta-analysis framework with study and species as random effects [270]. We nested observation within study to account for pseudoreplication, as 72% of studies had multiple effect sizes. To account for phylogenetic dependence, the covariance structure of the species random effect used the correlation matrix of an animal phylogeny obtained from the Open Tree of Life with the *rotl* and *ape* packages [271-273]. All models had the same random effects fit with rma.mv in the *metafor* package [62].

We first used a random effects model (REM) to estimate the overall relationship between urbanization and wildlife health (ie the mean effect size) across our full dataset (n = 516). To then identify the primary predictors of effect size, we fit a set of mixed-effect models (MEMs) that considered animal taxonomic group, health metric, life history (aquatic versus terrestrial), and their two-way interactions. We excluded the MEM with an interaction between life history and taxonomic group because we lacked complete data for all combinations of the levels of these variables). From each model, we derived a pseudo R^2 using the variance components [274]. We used maximum likelihood (ML) to compare models with AICc [137], and refit MEMs with REML to derive R^2. We considered MEMs with ΔAICc ≤ 2 to be competitive, and visualized top MEMs by back-transforming Zr into r. For the REM, we used the REML-estimated variance components to quantify I^2 as a measure of heterogeneity.

Given the results of this analysis (health metric had more explanatory power than animal taxonomic group), we stratified our data by each health metric dataset to test support for animal, parasite, and environmental traits as mechanisms that underlie relationships between urbanization and wildlife health. That is, we fit four sets of MEMs, with a separate model set for each health metric dataset. Six predictor variables were common to the global MEM for each dataset: animal taxonomic group, life history, mean country human footprint (average global human footprint score of the country in which a study was located), country gross domestic product (GDP; log-transformed), the interaction between mean country human footprint and life history, and the interaction between country GDP and life history. In addition to these six variables, each global MEM for the four datasets included dataset-specific predictor variables. The global MEM for the body condition dataset *(n* = 60) included how condition was quantified (raw measure of mass or size, mass adjusted for length, or qualitative scores) as a predictor variable. The global MEM for the parasitism dataset (*n* = 194) included the following predictor variables: parasite type (microparasite, macroparasite), parasite measure (whether infection status was measured as a binary variable, infection intensity, or parasite richness), if the parasite is transmitted via close contact, non-close contact, vectors, or an intermediate stage (four separate binary variables), if the parasite is transmitted by one versus multiple transmission routes (binary), the interaction between parasite type and mean country human footprint, the interaction between parasite type and country GDP, the interactions between parasite type and the five transmission route variables, and all two-way interactions among the transmission route variables. The global MEM for the toxicant dataset (*n* = 189) included toxicant type (whether toxicants

were metals or non-metals (eg pesticides)), the interaction between toxicant type and life history, the interaction between toxicant type and mean country human footprint, and the interaction between toxicant type and country GDP as predictor variables. The global MEM for the stress dataset (n = 73) included stress measure (whether stress metrics were based on glucocorticoid levels (eg hair cortisol), leukocyte profiles (eg HL ratios), or other measures (eg oxidative damage, blood glucose)), the interaction between stress measure and life history, the interaction between stress measure and mean country human footprint, and the interaction between stress measure and country GDP as predictor variables. We excluded any MEMs containing an interaction between two predictor variables if we lacked data for any combination of the levels of these variables). We generated candidate sets of all possible MEMs per dataset with the *MuMIn* package [138], limiting each candidate MEM to four predictors to ensure that the number of models considered did not exceed the sample size for each dataset [137]. We again used AICc to compare candidate models within each dataset.

To understand the influence of urban development, we examined the subset of data for which site location was provided (n = 302), allowing us to calculate the average GHF score across the most and least urban sites within a study (the *mean urbanization*), the quantitative difference in GHF scores between the most and least urban sites in a study (the *difference in urbanization*), and the change in study site GHF scores from 1993 and 2009 (the *change in urbanization*). We compared a set of MEMs that included either the mean urbanization, the difference in urbanization, the change in urbanization (each calculated at 1-km and 10-km buffers; six models), or the distance between the most and

least urban sites. We also considered each of these seven urbanization metrics in an interaction with health metric and with animal taxonomic group.

Table S2.1. ANOVA table from generalized linear models (Poisson errors) assessing research effort over time and according to health metric and taxonomic group

	χ^2	*p*
Counts ~ year*health		
Year	353.55	< 0.001
Health	127.71	< 0.001
Year:health	21.46	< 0.001
Counts ~ year*taxonomy		
Year	353.55	< 0.001
Taxonomy	216.79	< 0.001
Year:taxonomy	24.98	< 0.001

Table S2.2. Ranking of mixed-effects models (MEMs) predicting effect size for the relationship between urbanization and wildlife health for the full dataset, for each health metric dataset, and for studies where we obtained quantitative data on urbanization between extreme sites; models are ranked by ΔAICc with the number of parameters (*k*), pseudo R^2 and Akaike weights (w_i)

MEMs fit to full dataset (*n* = 516)	***k***	***R²***	**ΔAICc**	***wᵢ***
~ health metric + taxonomic group + health metric:taxonomic group + 1	17	0.29	0	1
~ life history + health metric + 1	5	0.14	12.73	0
~ health metric + 1	4	0.12	14.94	0
~ health metric + taxonomic group + 1	8	0.15	15.51	0
~ life history + health metric + life history:health metric + 1	8	0.11	15.67	0
~ life history + 1	2	0.05	20.66	0
~ 1	1	0	25.31	0
~ taxonomic group + 1	5	0.04	26	0
MEMs fit to toxicant dataset (*n* = 189)	***k***	***R²***	**ΔAICc**	***wᵢ***
~ taxonomic group + 1	5	0.21	0	0.68
~ life history + 1	2	0.1	5.07	0.05
~ 1	1	0	6.2	0.03
~ life history + GDP + 1	3	0.08	7.12	0.02
~ life history + toxicant type + 1	3	0.07	7.17	0.02
~ life history + Country human footprint + 1	3	0.08	7.18	0.02
~ GDP + toxicant type + GDP:toxicant type + 1	4	0.05	7.49	0.02
~ life history + toxicant type + life history:toxicant type + 1	4	0.09	7.57	0.02
~ Country human footprint + GDP + toxicant type + GDP:toxicant type + 1	5	0.13	7.76	0.01
~ life history + GDP + life history:GDP + 1	4	0.08	7.93	0.01
~ life history + GDP + toxicant type + GDP:toxicant type + 1	5	0.1	8.04	0.01
~ toxicant type + 1	2	0	8.1	0.01
~ GDP + 1	2	0	8.15	0.01
~ Country human footprint + 1	2	0	8.31	0.01
~ life history + Country human footprint + GDP + 1	4	0.03	9.11	0.01

~ life history + GDP + toxicant type + 1	4	0.05	9.16	0.01
~ life history + Country human footprint + toxicant type + 1	4	0.07	9.32	0.01
~ life history + Country human footprint + life history:Country human footprint + 1	4	0.06	9.33	0.01
~ life history + GDP + toxicant type + life history:GDP + 1	5	0.05	9.34	0.01
~ life history + GDP + toxicant type + life history:toxicant type + 1	5	0.06	9.7	0.01
~ life history + Country human footprint + toxicant type + life history:toxicant type + 1	5	0.07	9.71	0.01
~ GDP + toxicant type + 1	3	0	9.85	0
~ life history + Country human footprint + GDP + life history:GDP + 1	5	0.04	10	0
~ Country human footprint + toxicant type + 1	3	0	10.12	0
~ Country human footprint + GDP + 1	3	0	10.22	0
~ life history + Country human footprint + GDP + toxicant type + 1	5	0.03	11.26	0
~ life history + Country human footprint + GDP + life history:Country human footprint + 1	5	0.02	11.29	0
~ life history + Country human footprint + toxicant type + Country human footprint:toxicant type + 1	5	0.07	11.39	0
~ life history + Country human footprint + toxicant type + life history:Country human footprint + 1	5	0.07	11.49	0
~ Country human footprint + GDP + toxicant type + 1	4	0	12	0
~ Country human footprint + toxicant type + Country human footprint:toxicant type + 1	4	0	12.27	0
~ Country human footprint + GDP + toxicant type + Country human footprint:toxicant type + 1	5	0	14.17	0
MEMs fit to body condition dataset (*n* = 60)	***k***	***R²***	**ΔAICc**	***wᵢ***
~ 1	1	0	0	0.27
~ life history + 1	2	0	1.81	0.11
~ Country human footprint + 1	2	0	1.84	0.11
~ life history + GDP + life history:GDP + 1	4	0	2.04	0.1
~ GDP + 1	2	0	2.37	0.08
~ condition + 1	3	0.05	2.9	0.06
~ life history + Country human footprint + 1	3	0	3.32	0.05
~ taxonomic group + 1	4	0.03	4.09	0.03
~ life history + GDP + 1	3	0	4.28	0.03
~ Country human footprint + GDP + 1	3	0	4.37	0.03
~ life history + Country human footprint + GDP + life history:GDP + 1	5	0	4.46	0.03
~ life history + Country human footprint + life history:Country human footprint + 1	4	0	4.64	0.03
~ condition + Country human footprint + 1	4	0.03	5.2	0.02
~ condition + GDP + 1	4	0.01	5.29	0.02

~ life history + Country human footprint + GDP + 1	4	0	5.97	0.01
~ life history + Country human footprint + GDP + life history:Country human footprint + 1	5	0	7.16	0.01
~ condition + Country human footprint + GDP + 1	5	0	7.8	0.01
MEMs fit to parasite dataset (*n* = 194)	***k***	***R²***	**ΔAICc**	***wᵢ***
~ close + Country human footprint + 1	3	0.25	0	0.16
~ close + 1	2	0.25	0.8	0.11
~ close + Country human footprint + nonclose + 1	4	0.25	2.11	0.05
~ close + Country human footprint + GDP + 1	4	0.23	2.22	0.05
~ life history + close + Country human footprint + 1	4	0.22	2.23	0.05
~ life history + close + Country human footprint + life history:Country human footprint + 1	4	0.22	2.23	0.05
~ close + Country human footprint + nonclose + close:nonclose + 1	5	0.23	2.71	0.04
~ close + GDP + 1	3	0.23	2.91	0.04
~ life history + close + 1	3	0.22	2.94	0.04
~ close + nonclose + 1	3	0.24	3	0.03
~ close + Country human footprint + parasite measure + 1	5	0.22	3.51	0.03
~ close + parasite measure + 1	4	0.21	4.25	0.02
~ close + nonclose + close:nonclose + 1	4	0.21	4.32	0.02
~ close + Country human footprint + GDP + nonclose + 1	5	0.23	4.37	0.02
~ life history + close + Country human footprint + GDP + 1	5	0.2	4.49	0.02
~ life history + close + GDP + 1	4	0.19	5.12	0.01
~ life history + close + GDP + life history:GDP + 1	4	0.19	5.12	0.01
~ close + GDP + nonclose + 1	4	0.22	5.14	0.01
~ Country human footprint + parasite type + Country human footprint:parasite type + 1	4	0.13	5.2	0.01
~ Country human footprint + parasite type + 1	3	0.15	5.29	0.01
~ close + Country human footprint + nonclose + parasite measure + 1	6	0.21	5.78	0.01
~ life history + close + Country human footprint + parasite measure + 1	6	0.19	5.8	0.01
~ Country human footprint + vector + 1	3	0.01	5.8	0.01
~ close + Country human footprint + GDP + parasite measure + 1	6	0.19	5.81	0.01
~ Country human footprint + 1	2	0.06	5.82	0.01
~ Country human footprint + GDP + parasite type + GDP:parasite type + 1	5	0.07	6.3	0.01
~ life history + close + parasite measure + 1	5	0.18	6.42	0.01
~ close + GDP + nonclose + close:nonclose + 1	5	0.19	6.43	0.01

~ close + GDP + parasite measure + 1	5	0.19	6.44	0.01
~ close + nonclose + parasite measure + 1	5	0.2	6.5	0.01
~ vector + 1	2	0	6.52	0.01
~ parasite type + 1	2	0.09	7.18	0
~ Country human footprint + nonclose + parasite type + Country human footprint:parasite type + 1	5	0.11	7.18	0
~ Country human footprint + mroute + parasite type + Country human footprint:parasite type + 1	5	0.13	7.19	0
~ Country human footprint + mroute + 1	3	0.08	7.23	0
~ 1	1	0	7.24	0
~ Country human footprint + GDP + parasite type + Country human footprint:parasite type + 1	5	0.1	7.3	0
~ Country human footprint + mroute + parasite type + 1	4	0.15	7.36	0
~ Country human footprint + nonclose + parasite type + 1	4	0.14	7.45	0
~ Country human footprint + GDP + parasite type + 1	4	0.13	7.47	0
~ GDP + parasite type + GDP:parasite type + 1	4	0.01	7.6	0
~ Country human footprint + parasite type + parasite measure + Country human footprint:parasite type + 1	6	0.12	7.63	0
~ Country human footprint + nonclose + 1	3	0.05	7.72	0
~ Country human footprint + parasite type + parasite measure + 1	5	0.14	7.86	0
~ close + nonclose + parasite measure + close:nonclose + 1	6	0.17	7.98	0
~ Country human footprint + GDP + 1	3	0.03	7.98	0
~ Country human footprint + GDP + vector + 1	4	0	7.99	0
~ mroute + 1	2	0.06	8	0
~ life history + Country human footprint + 1	3	0.02	8.01	0
~ life history + Country human footprint + life history:Country human footprint + 1	3	0.02	8.01	0
~ Country human footprint + intermediate + 1	3	0.05	8.01	0
~ Country human footprint + parasite measure + 1	4	0.05	8.13	0
~ Country human footprint + parasite measure + vector + 1	5	0	8.4	0
~ nonclose + 1	2	0	8.66	0
~ life history + close + GDP + parasite measure + 1	6	0.15	8.69	0
~ GDP + vector + 1	3	0	8.7	0
~ mroute + parasite type + 1	3	0.12	8.71	0
~ close + GDP + nonclose + parasite measure + 1	6	0.17	8.73	0
~ GDP + nonclose + parasite type + GDP:parasite type + 1	5	0	8.8	0
~ nonclose + parasite type + 1	3	0.09	8.94	0

~	GDP + mroute + parasite type + GDP:parasite type + 1	5	0.03	8.99	0
~	parasite measure + vector + 1	4	0	9.07	0
~	GDP + parasite type + 1	3	0.06	9.34	0
~	life history + 1	2	0	9.36	0
~	Country human footprint + mroute + parasite type + mroute:parasite type + 1	5	0.16	9.37	0
~	GDP + 1	2	0	9.38	0
~	intermediate + 1	2	0	9.38	0
~	Country human footprint + GDP + mroute + 1	4	0.05	9.43	0
~	parasite measure + 1	3	0	9.5	0
~	Country human footprint + GDP + mroute + parasite type + 1	5	0.12	9.58	0
~	Country human footprint + mroute + parasite measure + 1	5	0.07	9.58	0
~	Country human footprint + GDP + nonclose + parasite type + 1	5	0.11	9.67	0
~	parasite type + parasite measure + 1	4	0.07	9.67	0
~	GDP + parasite type + parasite measure + GDP:parasite type + 1	6	0	9.68	0
~	Country human footprint + nonclose + parasite type + nonclose:parasite type + 1	5	0.12	9.71	0
~	Country human footprint + nonclose + parasite measure + 1	5	0.04	9.88	0
~	Country human footprint + intermediate + nonclose + 1	4	0.03	9.91	0
~	Country human footprint + GDP + nonclose + 1	4	0.02	9.92	0
~	Country human footprint + nonclose + parasite type + parasite measure + 1	6	0.13	9.96	0
~	Country human footprint + mroute + parasite type + parasite measure + 1	6	0.14	9.98	0
~	Country human footprint + GDP + parasite type + parasite measure + 1	6	0.11	10.07	0
~	GDP + mroute + 1	3	0.03	10.17	0
~	life history + Country human footprint + GDP + 1	4	0	10.2	0
~	life history + Country human footprint + GDP + life history:Country human footprint + 1	4	0	10.2	0
~	life history + Country human footprint + GDP + life history:GDP + 1	4	0	10.2	0
~	Country human footprint + intermediate + GDP + 1	4	0.02	10.2	0
~	mroute + parasite measure + 1	4	0.05	10.3	0
~	Country human footprint + GDP + parasite measure + 1	5	0.02	10.32	0
~	life history + Country human footprint + parasite measure + 1	5	0.02	10.37	0
~	life history + Country human footprint + parasite measure + life history:Country human footprint + 1	5	0.02	10.37	0
~	Country human footprint + intermediate + parasite measure + 1	5	0.04	10.39	0
~	mroute + parasite type + mroute:parasite type + 1	4	0.13	10.51	0

~	intermediate + nonclose + 1	3	0	10.63	0
~	Country human footprint + GDP + parasite measure + vector + 1	6	0	10.63	0
~	nonclose + parasite measure + 1	4	0	10.66	0
~	GDP + nonclose + 1	3	0	10.83	0
~	GDP + mroute + parasite type + 1	4	0.08	10.92	0
~	nonclose + parasite type + nonclose:parasite type + 1	4	0.09	11.09	0
~	GDP + nonclose + parasite type + 1	4	0.06	11.14	0
~	mroute + parasite type + parasite measure + 1	5	0.1	11.24	0
~	nonclose + parasite type + parasite measure + 1	5	0.07	11.26	0
~	GDP + parasite measure + vector + 1	5	0	11.33	0
~	life history + GDP + 1	3	0	11.54	0
~	life history + GDP + life history:GDP + 1	3	0	11.54	0
~	intermediate + GDP + 1	3	0	11.55	0
~	life history + parasite measure + 1	4	0	11.61	0
~	intermediate + parasite measure + 1	4	0	11.7	0
~	GDP + parasite measure + 1	4	0	11.72	0
~	Country human footprint + intermediate + nonclose + intermediate:nonclose + 1	5	0.04	11.73	0
~	Country human footprint + GDP + mroute + parasite measure + 1	6	0.05	11.82	0
~	GDP + parasite type + parasite measure + 1	5	0.04	11.92	0
~	Country human footprint + intermediate + nonclose + parasite measure + 1	6	0.02	12.09	0
~	Country human footprint + GDP + nonclose + parasite measure + 1	6	0.01	12.11	0
~	Country human footprint + intermediate + GDP + nonclose + 1	5	0	12.14	0
~	GDP + mroute + parasite measure + 1	5	0.02	12.56	0
~	life history + Country human footprint + GDP + parasite measure + 1	6	0	12.56	0
~	intermediate + nonclose + intermediate:nonclose + 1	4	0	12.58	0
~	intermediate + nonclose + parasite measure + 1	5	0	12.61	0
~	Country human footprint + intermediate + GDP + parasite measure + 1	6	0.01	12.62	0
~	GDP + mroute + parasite type + mroute:parasite type + 1	5	0.1	12.74	0
~	intermediate + GDP + nonclose + 1	4	0	12.85	0
~	GDP + nonclose + parasite measure + 1	5	0	12.92	0
~	mroute + parasite type + parasite measure + mroute:parasite type + 1	6	0.11	13.2	0
~	taxonomic group + 1	4	0	13.2	0

~ GDP + nonclose + parasite type + nonclose:parasite type + 1	5	0.05	13.33	0
~ GDP + mroute + parasite type + parasite measure + 1	6	0.06	13.54	0
~ nonclose + parasite type + parasite measure + nonclose:parasite type + 1	6	0.06	13.54	0
~ GDP + nonclose + parasite type + parasite measure + 1	6	0.04	13.55	0
~ life history + GDP + parasite measure + 1	5	0	13.87	0
~ life history + GDP + parasite measure + life history:GDP + 1	5	0	13.87	0
~ intermediate + GDP + parasite measure + 1	5	0	13.96	0
~ intermediate + nonclose + parasite measure + intermediate:nonclose + 1	6	0	14.77	0
~ intermediate + GDP + nonclose + intermediate:nonclose + 1	5	0	14.83	0
~ intermediate + GDP + nonclose + parasite measure + 1	6	0	14.91	0
~ taxonomic group + parasite measure + 1	6	0	15.45	0
MEMs fit to stress dataset (*n* = 73)	***k***	***R^2***	**ΔAICc**	***w_i***
~ taxonomic group + 1	4	0.55	0	0.78
~ life history + GDP + 1	3	0.05	5.67	0.05
~ GDP + 1	2	0.14	6.99	0.02
~ life history + 1	2	0	7.3	0.02
~ life history + Country human footprint + GDP + life history:Country human footprint + 1	5	0.07	7.38	0.02
~ life history + Country human footprint + GDP + 1	4	0.06	7.48	0.02
~ life history + GDP + life history:GDP + 1	4	0	7.94	0.01
~ 1	1	0	8.12	0.01
~ GDP + stress measure + 1	3	0.16	8.71	0.01
~ Country human footprint + GDP + 1	3	0.16	8.84	0.01
~ life history + Country human footprint + life history:Country human footprint + 1	4	0	9.42	0.01
~ life history + Country human footprint + 1	3	0	9.67	0.01
~ life history + Country human footprint + GDP + life history:GDP + 1	5	0	9.86	0.01
~ stress measure + 1	2	0	10.38	0
~ Country human footprint + 1	2	0	10.41	0
~ Country human footprint + GDP + stress measure + 1	4	0.17	10.69	0
~ GDP + stress measure + GDP:stress measure + 1	4	0.18	10.77	0
~ Country human footprint + GDP + stress measure + Country human footprint:stress measure + 1	5	0.15	10.97	0
~ Country human footprint + stress measure + 1	3	0	12.76	0
~ Country human footprint + GDP + stress measure + GDP:stress measure + 1	5	0.17	13.09	0

~ Country human footprint + stress measure + Country human footprint:stress measure + 1	4	0	15.18	0
Quantitative urban (*n* = 302)	***k***	***R²***	**ΔAICc**	***wᵢ***
~ mean urbanization_1000*health metric	8	0.37	0	0.62
~ mean urbanization_10000*health metric	8	0.35	1.51	0.29
~ inter-site distance*health metric	8	0.29	4.05	0.08
~ mean urbanization_1000	2	0.09	10.63	0
~ difference in urbanization_10000*health metric	8	0.2	11.62	0
~ change in urbanization_10000*health metric	8	0.2	11.99	0
~ change in urbanization_1000*health metric	8	0.21	12.18	0
~ difference in urbanization_1000*health metric	8	0.19	12.29	0
~ mean urbanization_10000	2	0.07	12.46	0
~ 1	1	0	15.51	0
~ mean urbanization_1000*taxonomic group	10	0.15	16.11	0
~ difference in urbanization_1000*taxonomic group	10	0.14	17.29	0
~ difference in urbanization_10000	2	0	17.33	0
~ change in urbanization_10000	2	0	17.51	0
~ difference in urbanization_1000	2	0	17.53	0
~ inter-site distance	2	0	17.57	0
~ change in urbanization_1000	2	0	17.58	0
~ inter-site distance*taxonomic group	10	0.12	18.2	0
~ mean urbanization_10000*taxonomic group	10	0.11	18.68	0
~ difference in urbanization_10000*taxonomic group	10	0.1	19.52	0
~ change in urbanization_10000*taxonomic group	10	0.03	22.9	0
~ change in urbanization_1000*taxonomic group	10	0	24.67	0

Notes: Explanation of predictor variables:

Health metric methodology

Health metric: aspect of health (body condition, parasitism, stress, or toxicants) that was compared between urban and non-urban wildlife populations.

Condition: whether an animal's body condition was measured using a qualitative (eg fat score), raw quantitative (eg body length), or adjusted quantitative (eg mass/length) metric.

Parasite measure: infection status as a binary variable, infection intensity, or parasite richness.
Stress measure: whether animal stress was measured based on glucocorticoid levels (eg hair cortisol), leukocyte profiles (eg HL ratios), or other measures (eg oxidative damage, blood glucose).
Toxicant type: whether a toxicant was a metal or non-metal (eg pesticide).

Wildlife traits
Taxonomic group: whether the wildlife species belonged to herpetofauna (amphibian or reptile), birds, fish, invertebrates, or mammals.
Life history: whether most of a species' life history is spent in an aquatic or terrestrial environment.

Parasite traits
Close: parasite transmitted through close contact as defined by the Global Mammal Parasite Database (GMPD; 0/1)
Intermediate: parasite transmitted through trophic dynamics as defined by the GMPD (0/1);
Nonclose: parasite transmitted through non-close contact as defined by the GMPD (0/1);
Vector: parasite transmitted via a vector as defined by the GMPD (0/1);
Mroute: parasite transmitted via a single GMPD transmission route or more than one route (0/1);
Parasite type: microparasite (bacterium, fungus, protozoan, virus) or macroparasite (ectoparasite, helminth).

Metrics of urbanization
Country human footprint: average Global Human Footprint (GHF) score of the country in which a study was located.
GDP: gross domestic product (GDP) of the country in which a study was located (log-transformed).
Inter-site distance: the distance between the most and least urban sites in a study (quarter-root transformed).
Change in urbanization: the difference in GHF scores between 1993 and 2009 within a study (with 1-km or 10-km buffer around each set of coordinates).
Difference in urbanization: the difference in GHF scores between the most and least urban sites within a study (with 1-km or 10-km buffer around each set of coordinates).
Mean urbanization: the average GHF score across the most and least urban sites within a study (with 1-km or 10-km buffer around each set of site coordinates).

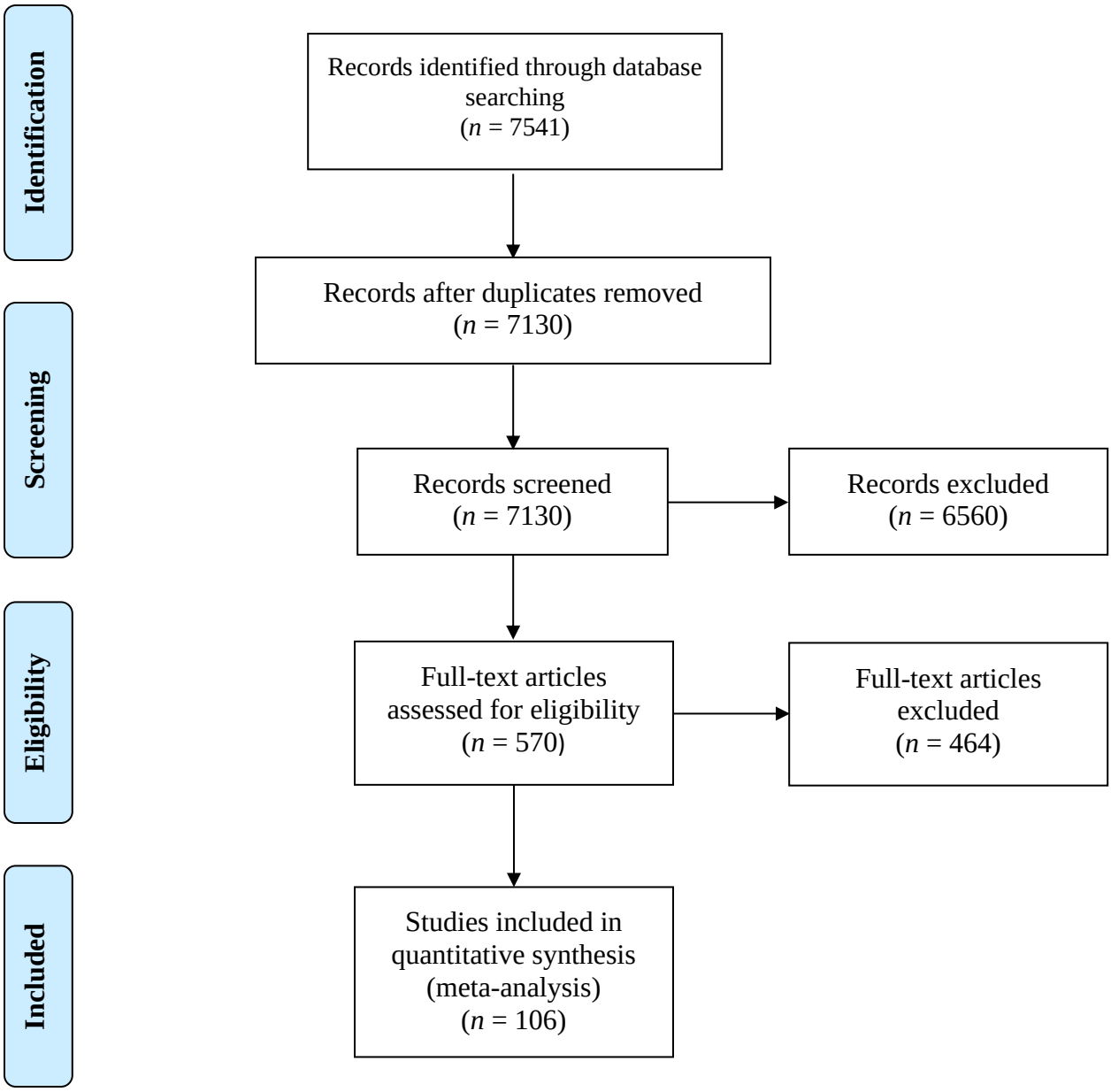

Figure S2.1. PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) diagram showing the procedure for article exclusion from the meta-analysis based on titles, abstracts, and full text. Articles were included if they compared the body condition, stress, toxicant loads, or the prevalence, intensity of infection, or diversity of parasites or pathogens across urban and non-urban populations of the same wildlife species.

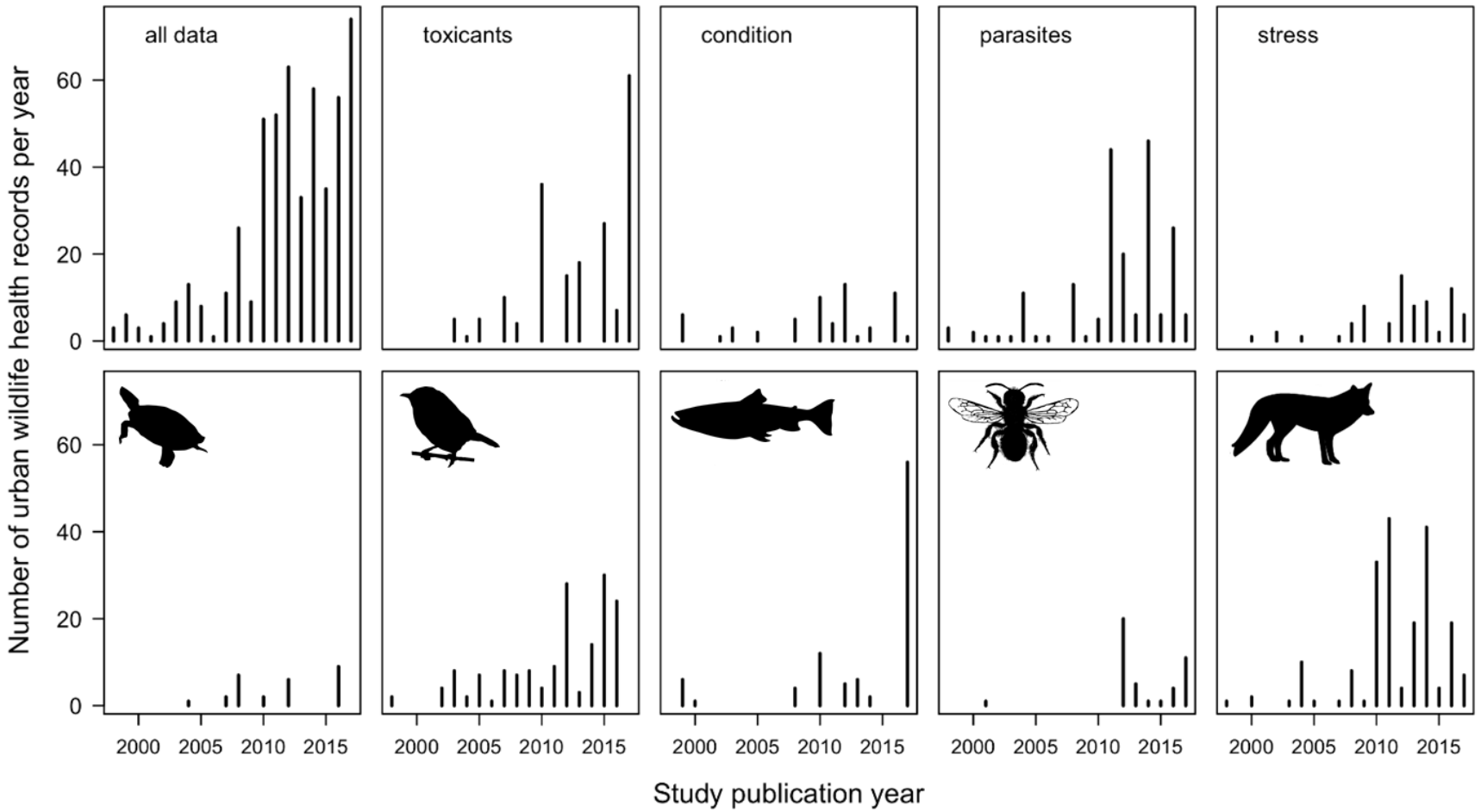

Figure S2.2. Change over time in the number of individual records for urban wildlife health by health metric (top row) and taxonomic group (bottom row, left to right: herpetofauna, birds, fish, invertebrates, and mammals).

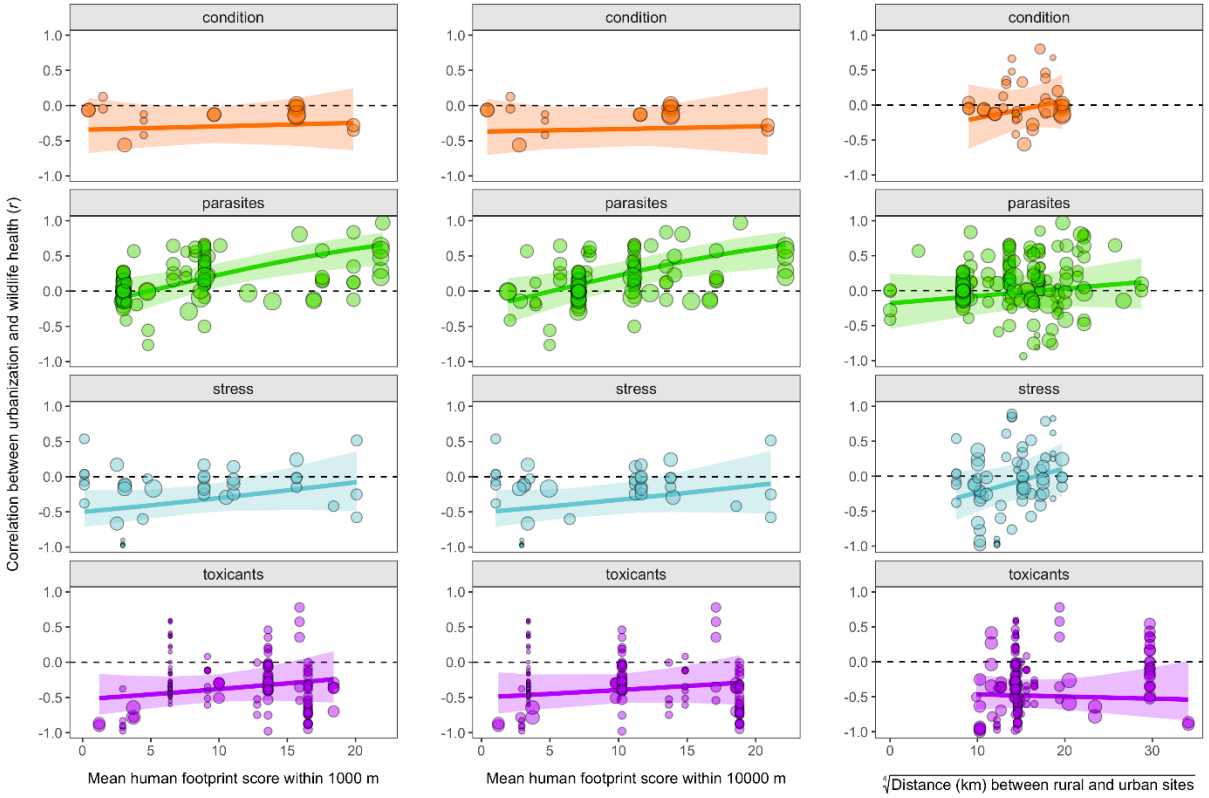

Figure S2.3. Most competitive mixed-effect models for how the mean urbanization footprint per study and distance between the most rural and urban wildlife populations predict effect size. The filled polygons and colored lines show the means and 95% confidence interval for the interaction between health metric and inter-site distance. Circles show individual records scaled by their sample size, and the dashed line shows no relationship between health and urbanization.

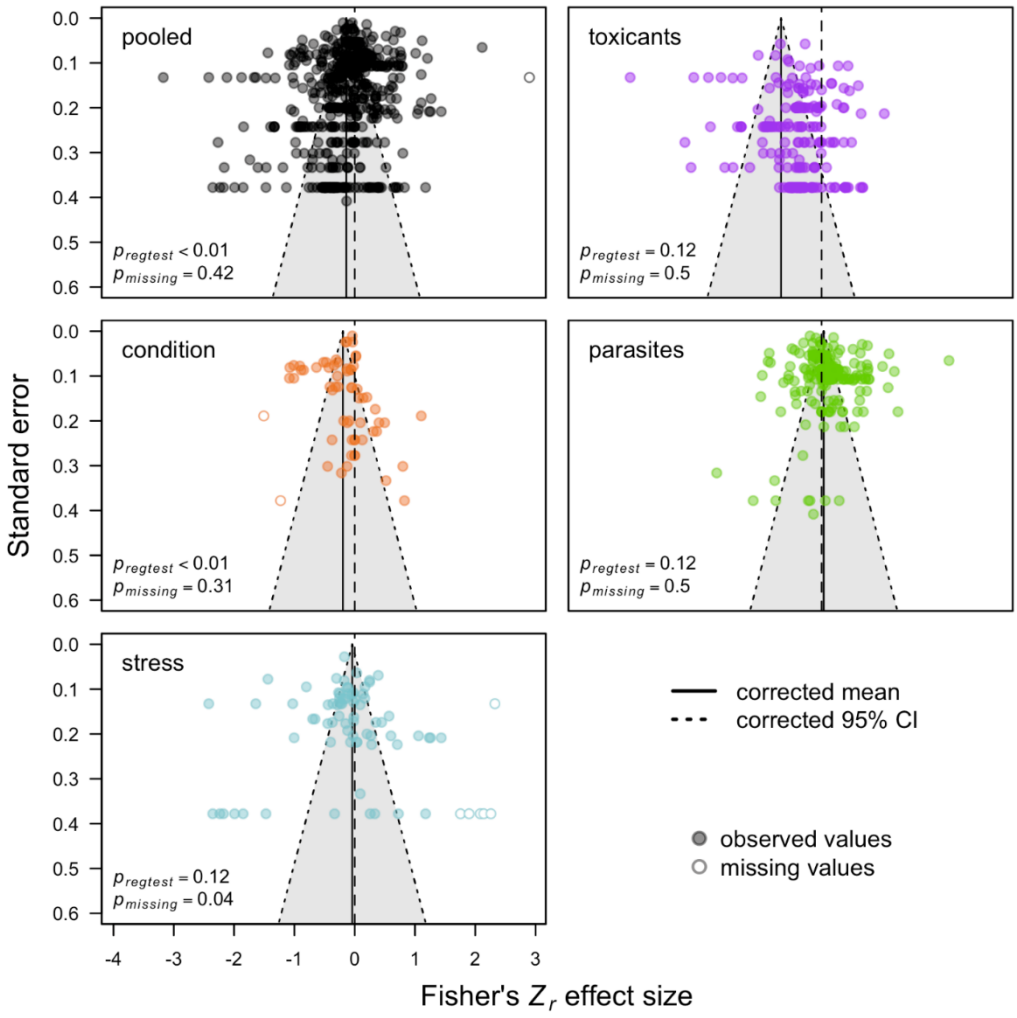

Figure S2.4. Funnel plots illustrating the relationship between effect size and standard error (shaded circles) and the effect of correcting funnel plot asymmetry (white circles) with trim-and-fill analysis on the estimated true correlation between urbanization and health outcomes (solid line). The central dashed line shows where $Z_r = 0$ (no effect), while the solid line shows the estimated mean effect size after adjusting for potential publication bias. p_{regtest} is the *P* value for the association between effect size and standard error; p_{missing} is the *P* value for the null hypothesis that no records are missing due to publication bias.

APPENDIX B

CHAPTER 3 SUPPLEMENTAL INFORMATION

Supplemental methods

Calculation of scaled mass index (SMI)

We calculated SMI [136] as one of three measures of flying fox body condition. SMI is calculated as $\widehat{M}_i = M_i \left[\frac{L_0}{L_i}\right]^{b_{SMA}}$ where:

$\widehat{M}_i$ = scaled index for individual *i*,

M_i = body mass of individual *i*

L_i = linear body measurement of individual *i* (here, forearm length)

L_0 = a chosen linear body measurement within the range of *L*

b_{SMA} = a scaling exponent calculated from an SMA/RMA (standardized or reduced major axis) regression of mass on length measurements from the study population

We used the median forearm length of all Adelaide flying foxes captured from 2015-2018 as our value of L_0 (n = 306; 4 of the 310 individuals captured during this time period were missing a forearm length measurement). This produced a L_0 value of 161.05 mm.

Before calculating b_{SMA}, we first plotted body weight (g) by forearm length (mm) (again from all Adelaide flying foxes captured from 2015-2018) to identify potential outliers [136]. Three bats had noticeably shorter forearm lengths (<140 mm). In a boxplot of forearm length, these three points extended beyond the range of the lower whisker (Q1 – 1.5 IQR). We therefore chose to exclude them when calculating b_{SMA}. We next conducted a likelihood ratio test using the sma function in the *smatr* package [275] to determine whether the slopes of an SMA regression of log-transformed body weight (g) on log-transformed forearm length (mm) were significantly different for males and females. We failed to reject the null hypothesis that the slopes were equal (likelihood

ratio statistic: 0.51, df = 1, P = 0.48), indicating that it was appropriate to use one b_{SMA} value for males and females. Bundling male and female measurements produced a b_{SMA} value of 4.65 (95% CI: 4.36 – 4.96).

Calculation of foraging distance and area

All movement and statistical analyses were performed in the R computing environment v 3.4.3 [127]. The dataset was first trimmed so that only the first fix of each GPS burst was retained (bursts are useful for improving estimation of altitude, speed, and heading, which were not needed in this study). We calculated the number of hours each bat was tracked per night and subsequently excluded incomplete nights (< 8 hours of data). We calculated the nightly distance flown by each bat by summing the great circle distance between successive GPS points using the spDists function in the *sp* package [128]. *Nightly roundtrip distance* (from the roost to foraging sites and back) flown by each bat was calculated by summing the great circle distance If the first or last GPS point of a night's track was >100m from the roost's center (e.g. due to battery depletion or movement outside the GPS collection window), the distance between the roost and the starting or ending point was added to the roundtrip distance for that night. If both the last GPS point of one night and the first point of the next night were >100m from the roost, this was a potential indication that the bat did not return to the roost during the day. Following examination of the GPS tracks in Google Earth, we found that on four occasions, bats did not to return to the roost in the morning and instead spent the day elsewhere; after a second night, they returned to the roost. Three of these days away from the roost occurred on the same date, which was also the first day following logger

deployment on those bats. We therefore chose to exclude non–round-trip nights from further calculations because we believe this represented aberrant behavior. The *maximum foraging distance* was calculated as the straight-line distance from the roost to the furthest foraging site identified by our automated procedure [133]. Summary statistics (*median roundtrip distance*, *maximum roundtrip distance*) were calculated for each bat.

We estimated the area traversed by tracked GHFF using minimum convex polygons (MCPs). The foraging and core areas for each bat were calculated respectively as the areas of 95% and 50% MCPs constructed with the *adehabitatHR* package [129]. We restricted calculation of foraging and core areas to bats with at least three round-trips, conservatively assuming that small sample sizes would not accurately estimate area. We also constructed seasonal 95% MCPs (i.e. one winter MCP for all bats tracked in Aug. 2016 and Aug. 2017, one summer MCP for all bats tracked in Feb. 2017 and Feb. 2018) for use in habitat selection analyses.

Table S3.1. Summary of distance and area metrics calculated for each bat. Shaded rows indicate capture in winter (August) and white rows indicate capture in summer (February). Nights of data were counted when at least 8 hours of data were collected and the bat made a round-trip from the roost and back.

Bat ID	Capture date	Sex	Est. age (yr)	Weight (g)	Forearm length (mm)	WFR	BCS	SMI	Nights of data	Median nightly roundtrip distance (km)	Max. nightly roundtrip distance (km)	Max. foraging distance (km)	Avg. nightly number foraging sites	50% MCP (km^2)	95% MCP (km^2)
106	8/8/2016	F	<2	673	160	4.21	3	693.77	7	20.51	115.06	40.47	3.0	113.53	277.12
110	8/8/2016	M	<2	657	160	4.11	3.5	687.2	6	74.70	109.10	40.46	2.8	183.93	557.53
115	8/9/2016	M	6	883	163	5.42	4	834.97	8	15.58	20.63	6.31	2.1	2.03	6.92
121	8/9/2016	M	4-5	909	166	5.48	4	789.7	10	16.18	80.42	25.84	2.3	94.96	171.72
132	8/9/2016	M	6-8	1008	173	5.83	4	720.83	10	27.12	29.04	9.76	1.9	5.82	17.64
199	2/21/2017	F	4	716	161	4.45	2.5	727.47	5	47.00	55.33	15.78	4.2	12.12	33.89
200	2/21/2017	F	3	688	169	4.07	3	557.6	10	13.35	108.38	21.38	4.7	37.14	501.98
201	2/21/2017	M	3-4	768	164	4.68	3	705.87	9	48.77	60.52	15.77	3.9	78.73	244.51
203	2/22/2017	M	4	808	167	4.84	3	680.74	7	23.33	42.49	10.14	4.7	3.56	24.20
204	2/22/2017	M	3-4	744	164	4.53	2.5	678.03	6	30.92	40.05	10.30	5.2	18.49	41.60
205	2/22/2017	F	4-5	761	168	4.53	2.5	618.46	6	54.11	87.34	30.66	4.0	74.94	229.87
206*	2/22/2017	F	3-4	718	161	4.46	2.5	716.96	2	34.24	37.80	11.45	3.5	--	--
207	2/22/2017	F	6-7	743	170	4.37	2.5	585.84	9	17.17	87.59	34.75	3.1	41.53	238.49
208	2/22/2017	F	3	690	172	4.01	2.5	515.18	6	30.75	143.30	17.15	4.5	85.38	770.30
209	8/11/2017	F	3-4	701	158	4.44	3.5	777.51	3	52.68	83.26	35.85	4.3	53.16	131.20
210	8/11/2017	M	3-4	670	158	4.24	3	732.26	6	50.48	66.43	26.10	4.0	41.80	135.71
211*	8/11/2017	F	5-6	640	151	4.24	3	863.43	2	28.61	37.71	15.10	2.5	--	--
215	8/11/2017	F	4	668	162	4.12	3	649.99	3	18.29	59.39	16.77	6.3	13.38	102.91
217	8/11/2017	M	3-4	666	159	4.19	3.5	698.65	3	21.50	89.42	34.65	6.0	107.77	176.62
218	8/12/2017	M	3-4	718	161	4.46	3.5	729.51	4	70.93	89.63	32.28	4.5	35.50	210.54
226	8/12/2017	M	6-8	696	157	4.43	3	783.45	6	67.47	102.81	21.98	5.2	158.52	428.76

264	2/11/2018	M	3-4	734	167	4.40	3.5	620.12	5	35.04	50.79	14.33	2.6	44.15	71.08
265	2/11/2018	M	4	791	161	4.91	3	794.43	7	28.74	80.25	31.46	4.7	35.71	221.14
269*	2/11/2018	F	5	732	160	4.58	3.5	761.2	0	--	--	--	--	--	--
277*	2/11/2018	M	6	793	161	4.93	4	796.44	2	19.61	29.35	7.33	4.0	--	--
287	2/11/2018	F	5	772	168	4.60	4	632.62	5	13.44	16.50	3.69	3.2	1.45	5.36
289	2/11/2018	M	5	701	170	4.12	2.5	552.72	10	36.37	88.89	31.35	5.2	50.33	526.66
291	2/11/2018	M	7	796	168	4.74	3.5	650.49	11	16.34	110.51	31.84	2.1	45.60	800.91
298	2/13/2018	M	5	750	164	4.57	3.5	689.33	6	42.55	179.35	25.13	3.5	364.04	1596.60
302	2/13/2018	M	5	762	168	4.54	3	626.16	7	39.58	76.25	17.49	4.3	39.51	361.65
306	2/13/2018	F	8+	700	163	4.29	3	661.92	4	58.06	59.90	23.00	3.3	64.85	90.38
310*	2/21/2018	F	NA	800	168	4.62	4	657.38	1	45.70	45.70	15.48	6.0	--	--

*Core area and foraging area were not calculated for bats with less than 3 nights of data.

Table S3.2. Summary of model outputs for nightly round-trip distance, nightly number of foraging sites, foraging area, and core area. Three candidate models (using separate measures of body condition) were created for each response variable and ranked by ΔAICc. P values ≤ 0.05 are in bold. BCS: body condition score; WFR: weight to forearm ratio; SMI: scaled mass index; maxTemp: maximum daily temperature; hours: nightly hours of tracking data collected; nights: number of nights that a bat was tracked

Response variable	**Model**	**ΔAICc**	**Term**	**Estimate**	**SE**	**t or z**	**P**
Nightly roundtrip distance (*n* = 185)	~ age + sex + BCS + season + maxTemp + hours	0	age	-0.04	0.05	-0.84	0.40
			sex(male)	0.31	0.16	2.01	**0.04**
			BCS	-0.57	0.18	-3.09	**0.002**
			season(winter)	0.22	0.30	0.72	0.47
			maxTemp	-0.02	0.02	-0.93	0.32
			hours	-0.07	0.08	-0.85	0.40
	~ age + sex + WFR + season + maxTemp + hours	2.91	age	-0.001	0.06	-0.02	0.98
			sex(male)	0.35	0.17	2.02	**0.04**
			WFR	-0.55	0.22	-2.47	**0.01**
			season(winter)	0.12	0.30	0.40	0.69
			maxTemp	-0.01	0.02	-0.63	0.53
			hours	-0.07	0.08	-0.77	0.44
	~ age + sex + SMI + season + maxTemp + hours	9.38	age	-0.07	0.06	-1.19	0.23
			sex(male)	0.18	0.18	1.00	0.32
			SMI	-0.0004	0.001	-0.29	0.77
			season(winter)	0.03	0.33	0.08	0.93
			maxTemp	-0.01	0.02	-0.69	0.49
			hours	-0.07	0.09	-0.78	0.43

Nightly number of foraging sites (*n* = 185)	~ age + sex + BCS + season + maxTemp + hours	0	age	-0.06	0.03	-2.07	**0.04**
			sex(male)	0.11	0.09	1.19	0.23
			BCS	-0.38	0.11	-3.34	**0.0008**
			season(winter)	-0.08	0.18	-0.46	0.65
			maxTemp	-0.01	0.01	-0.46	0.65
			hours	0.03	0.07	0.37	0.71
	~ age + sex + WFR + season + maxTemp + hours	2.22	age	-0.03	0.04	-0.95	0.34
			sex(male)	0.14	0.11	1.34	0.18
			WFR	-0.40	0.14	-2.88	**0.004**
			season(winter)	-0.18	0.18	-1.00	0.32
			maxTemp	-0.004	0.01	-0.33	0.74
			hours	0.04	0.07	0.55	0.58
	~ age + sex + SMI + season + maxTemp + hours	11.07	age	-0.08	0.04	-2.08	**0.04**
			sex(male)	0.03	0.12	0.23	0.82
			SMI	-0.0004	0.0008	-0.46	0.65
			season(winter)	-0.20	0.20	-0.97	0.33
			maxTemp	-0.005	0.01	-0.39	0.70
			hours	0.02	0.07	0.28	0.78
Foraging area (*n* = 27)	~ age + sex + WFR + season + nights	0	age	0.01	0.16	0.04	0.97
			sex(male)	0.93	0.51	1.84	0.08
			WFR	-1.98	0.66	-2.98	**0.007**
			season(winter)	-0.31	0.49	-0.63	0.53
			nights	0.17	0.11	1.50	0.15
	~ age + sex + SMI + season + nights	6.68	age	-0.04	0.15	-0.26	0.80
			sex(male)	0.84	0.52	1.64	0.12
			SMI	-0.006	0.004	-1.55	0.14
			season(winter)	-0.05	0.56	-0.08	0.93
			nights	0.03	0.11	0.26	0.80

	~ age + sex + BCS + season + nights	8.52	age	-0.12	0.15	-0.79	0.44
			sex(male)	0.68	0.50	1.35	0.19
			BCS	-0.41	0.55	-0.75	0.46
			season(winter)	-0.52	0.54	-0.97	0.35
			nights	0.07	0.11	0.62	0.54
Core area (*n* = 27)	~ age + sex + WFR + season + nights	0	age	0.01	0.17	0.08	0.94
			sex(male)	0.74	0.53	1.40	0.18
			WFR	-1.15	0.70	-1.65	0.11
			season(winter)	0.17	0.52	0.33	0.74
			nights	0.03	0.12	0.25	0.80
	~ age + sex + SMI + season + nights	2.99	age	-0.04	0.15	-0.26	0.80
			sex(male)	0.58	0.52	1.13	0.27
			SMI	-0.002	0.004	-0.42	0.68
			season(winter)	0.15	0.56	0.27	0.79
			nights	-0.07	0.11	-0.63	0.53
	~ age + sex + BCS + season + nights	3.07	age	-0.06	0.15	-0.39	0.70
			sex(male)	0.57	0.50	1.15	0.26
			BCS	-0.16	0.54	-0.29	0.77
			season(winter)	0.04	0.53	0.07	0.95
			nights	-0.06	0.10	-0.60	0.56

Table S3.3. Results of two logistic regressions to model the probability of a site being used or available as a function of the land use at the site and bat age, sex, and body condition. Values are reported for β (beta) coefficient, SE (standard error), OR (odds ratio) and 95% CI (Wald confidence interval). 95% CIs that do not cross 1 are considered significant and marked in bold.

Summer

Variable		**β**	**SE**	**OR**	**95% CI**
Land use	Natural	Reference	--	--	--
	Agricultural and plantation production	0.44	0.87	1.56	0.28 – 8.58
	Non-residential intensive use	3.05	0.73	**21.12**	**5.10 – 87.52**
	Residential and farm infrastructure	2.78	0.72	**16.13**	**3.92 – 66.39**
	Water	1.29	1.01	3.62	0.50 – 26.23
Age		-0.02	0.07	0.98	0.85 – 1.13
Sex	Female	Reference	--	--	--
	Male	-0.04	0.23	0.96	0.61 – 1.50
WFR		0.23	0.41	1.25	0.56 – 2.82

Winter

Variable		**β**	**SE**	**OR**	**95% CI**
Land use	Natural	Reference	--	--	--
	Agricultural and plantation production	-0.04	0.92	0.97	0.16 – 5.89
	Non-residential intensive use	2.51	0.75	**12.29**	**2.83 – 53.39**
	Residential and farm infrastructure	1.98	0.74	**7.22**	**1.70 – 30.61**
	Water	1.73	1.03	5.64	0.75 – 42.68
Age		-0.01	0.09	0.99	0.83 – 1.18
Sex	Female	Reference	--	--	--
	Male	0.06	0.34	1.06	0.55 – 2.06
WFR		-0.19	0.27	0.83	0.49 – 1.39

Table S3.4. Plant species identified by ground-truthing suspected grey-headed flying fox foraging sites in Adelaide, South Australia, 2016-2018. Only plants identified to species level are included. Gray shading indicates that a species is considered a significant food plant for grey-headed flying foxes [144].

Family	**Plant species**	**Common name**	**Native to Australia**	**Native to South Australia**
Fabaceae	*Acacia pendula*	Weeping myall	Yes	Yes
Loranthaceae	*Amyema miquelii*	Box mistletoe	Yes	Yes
Myrtaceae	*Corymbia citriodora*	Lemon-scented gum	Yes	No
	C. maculata	Spotted gum	Yes	No
	Eucalyptus aff. eremophila	Sand mallee	Yes	No
	E. aff. robusta	Swamp mahogany	Yes	No
	E. camaldulensis	Red river gum	Yes	Yes
	E. fasciculosa	Pink gum	Yes	Yes
	E. globulus	Tasmanian bluegum	Yes	No
	E. leucoxylon	Blue gum	Yes	Yes
	E. megacornuta	Warty yate	Yes	No
	E. microcarpa	Grey box	Yes	Yes
	E. occidentalis	Flat topped yate	Yes	No
	E. sideroxylon	Mugga ironbark	Yes	No
	E. stricklandii	Strickland's gum	Yes	No
	E. viminalis	Ribbon gum	Yes	Yes
Moraceae	*Ficus carica*	Common fig	No	No
	F. macrophylla	Moreton Bay fig	Yes	No
	F. platypoda	Small-leaved Moreton Bay fig	Yes	No
Oleaceae	*Olea europaea*	Olive	No	No
Arecaceae	*Phoenix canariensis*	Canary Island date palm	No	No

Table S3.5. Results of linear models to explore predictors of flying fox body condition. WFR was modeled with a gamma distribution (log link) and SMI was modeled with a normal distribution. Due to the limited range of values, BCS was binned into two groups (≤3 and >3) and modeled with a binary distribution. P values ≤ 0.05 are in bold.

Model	Term	Estimate	SE	t	P
WFR ~ season + sex + age	season(winter)	-0.02	0.03	0.55	0.585
	sex(male)	0.07	0.03	2.45	**0.02**
	age	0.02	0.01	2.60	**0.02**
BCS ~ season + sex + age	season(winter)	1.11	0.83	1.34	0.18
	sex(male)	1.33	0.84	1.59	0.11
	age	0.18	0.28	0.66	0.51
SMI ~ season + sex + age	season(winter)	86.39	27.31	3.16	**0.004**
	sex(male)	21.08	26.84	0.79	0.44
	age	12.07	8.94	1.35	0.19

WFR: weight to forearm ratio. BCS: body condition score. SMI: scaled mass index

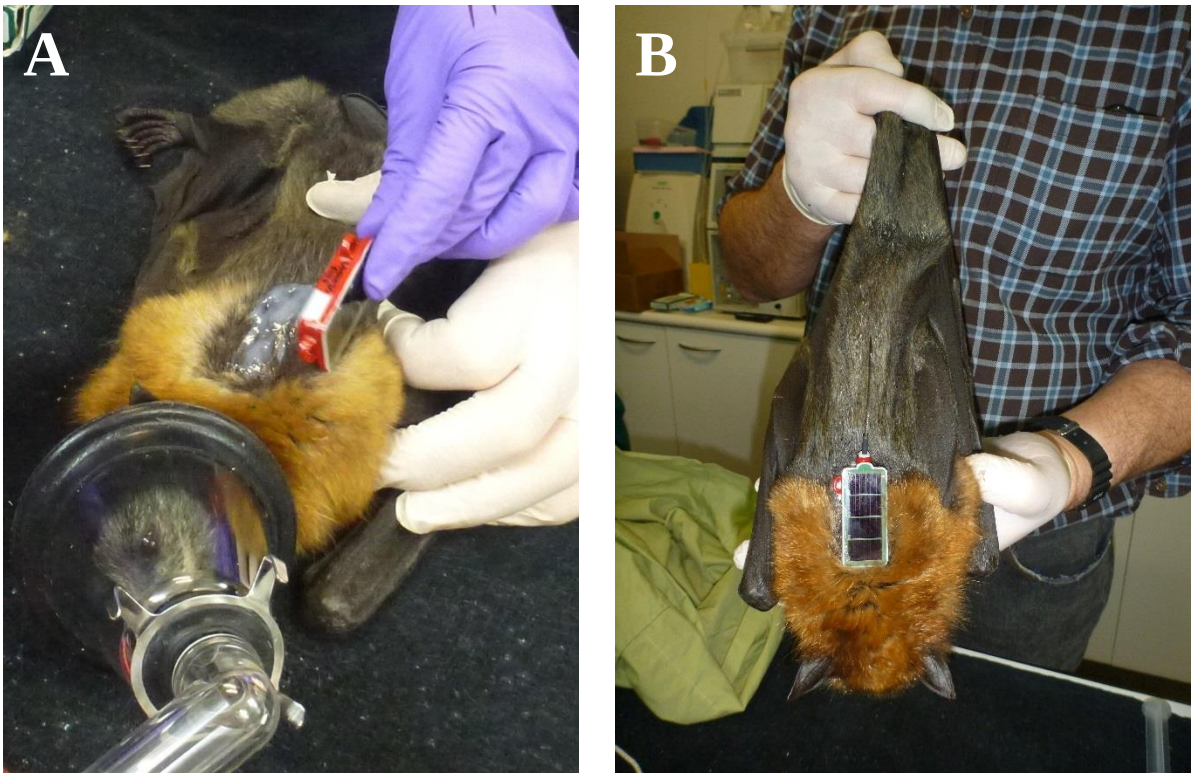

Figure S3.1. Logger attachment process. **A**) Glue is applied to the back of the bat where fur has been clipped. **B**) Logger in place on the bat following attachment.

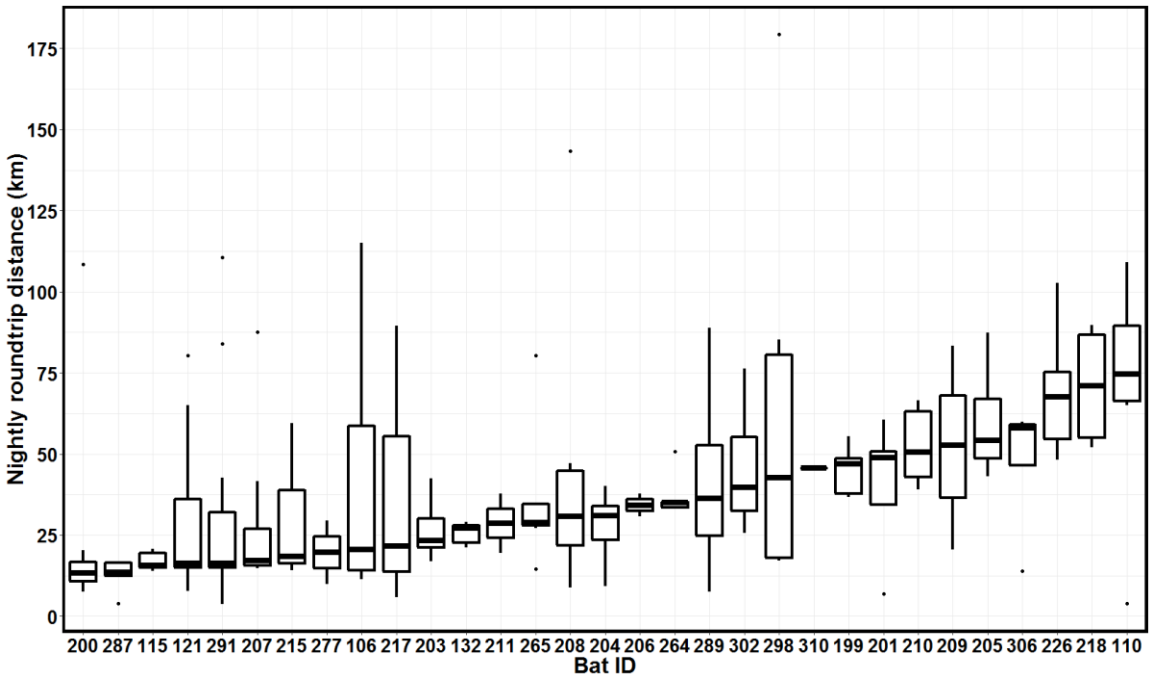

Figure S3.2. Boxplot showing the variation in nightly roundtrip distance (distance from the roost to all foraging sites and back in km) within and between bats. The median roundtrip distance was 31.93 km (1st quartile: 16.98; 3rd quartile: 52.78).

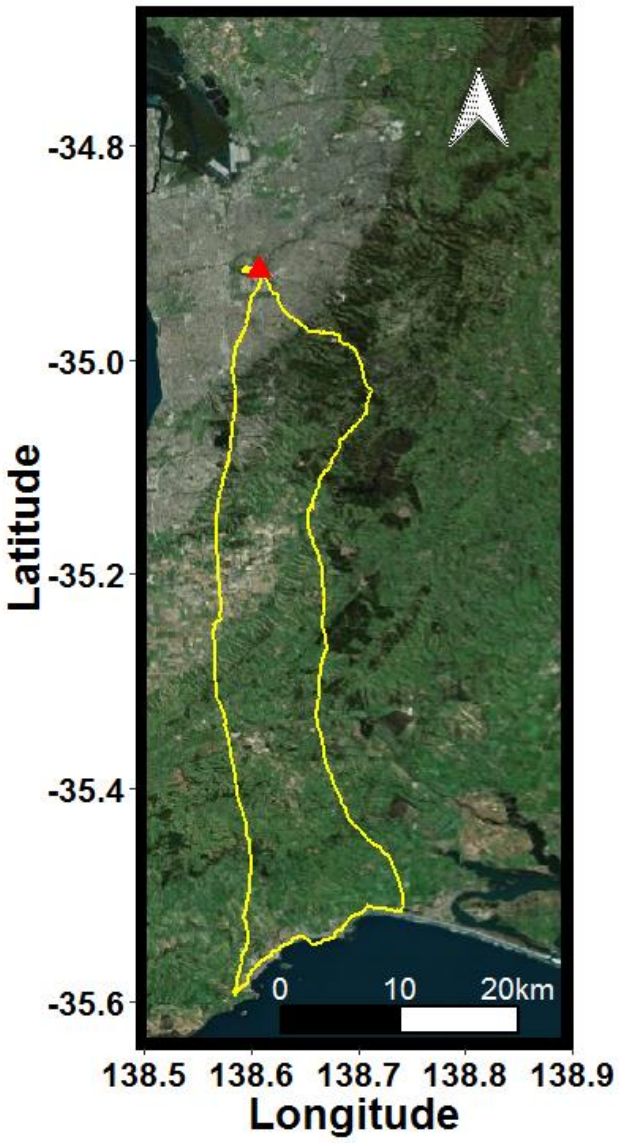

Figure S3.3. Path flown by bat 298 the night of 2/14/2018, traveling in a clockwise direction. The total distance was 179.35km. The location of the roost is marked with a red triangle.

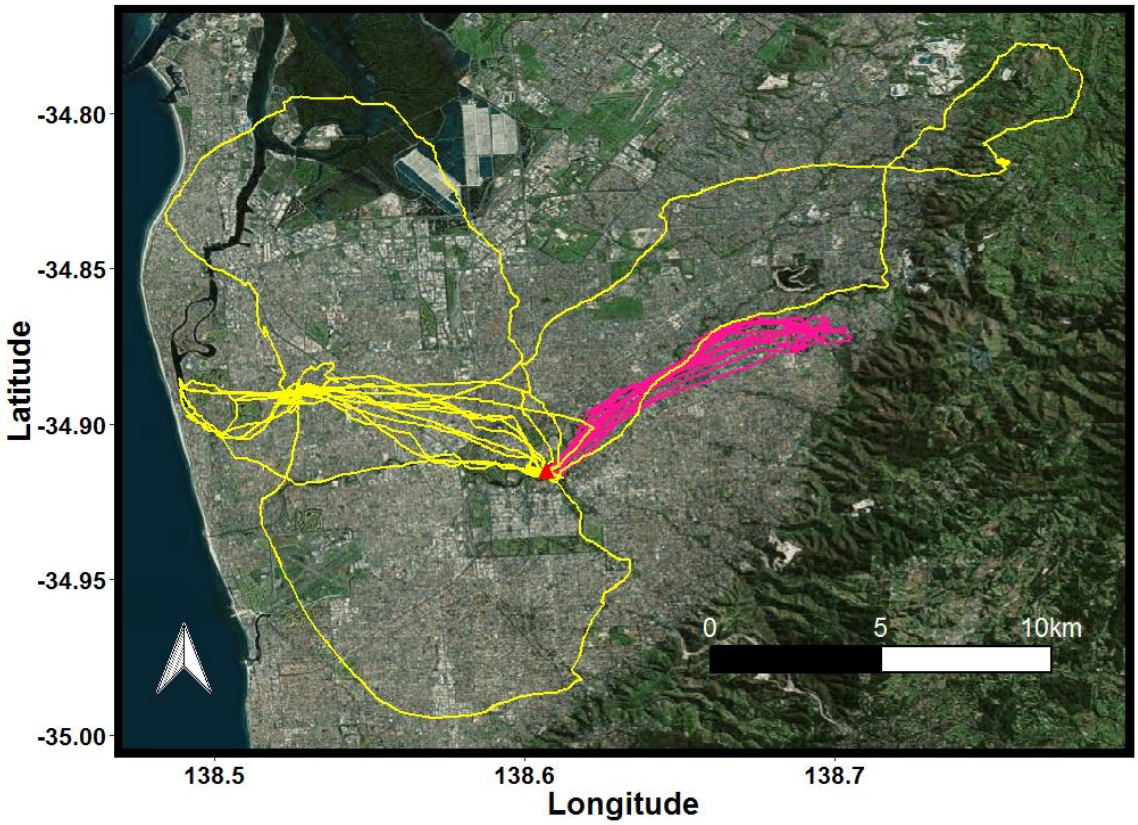

Figure S3.4. Examples of repeated visits to foraging sites over several nights (Bat 132, 10 nights, pink) and visits to core foraging sites with occasional long-distance excursions (Bat 302, 7 nights, yellow). The location of the roost is indicated by a red triangle.

APPENDIX C

CHAPTER 4 SUPPLEMENTAL INFORMATION

Supplemental methods

Fur sample digestion and analysis by ICP-MS

At Baylor University, fur samples were analyzed for the presence of ten metals (cadmium, chromium, cobalt, copper, lead, nickel, selenium, strontium, tin, vanadium) and two metalloids (antimony and arsenic; hereafter referred to as metals).

Fur digestions were performed in batches of 25 to 30 samples. Blanks were included for each batch. Human hair standard (Sigma-Aldrich, St. Louis, MO) was used as a standard reference material (SRM); one SRM sample was included for each bat capture site. To digest a fur sample, approximately 0.05g of fur was weighed to the nearest 0.001g and transferred to a borosilicate glass tube (VWR International, Radnor, PA). Next, 0.25 mL of genpure water and 2.5 mL of 1:1 HNO_3 (Fisher Scientific, Waltham, MA) were added. Samples in a batch were heated for 15 minutes at 95°C $\pm$ 5°C, then left to cool. Next, 1mL of concentrated HNO_3 was added to each sample, and the samples were heated for 30 minutes at the same temperature. After cooling, 0.25mL of genpure water and 0.75mL of 30% H_2O_2 (Fisher Scientific) was added to each sample, and the samples was heated for 60 mins at the same temperature. Samples were then filtered into acid-rinsed Erlenmeyer flasks (VWR International). Before filtration, filters were dampened with genpure water. Samples weighing close to 0.05g were filtered into 25mL flasks, samples below the desired weight were filtered into 10mL flasks, and blanks and SRMs were filtered into 20mL flasks. After pouring each sample, each tube was rinsed with genpure water and the contents were poured through the filter. After the filters drained, a small amount of genpure water was applied around the filter to filter any

sample left on the sides. Samples were then diluted. Samples in the 25mL flasks were diluted to 25mL and the samples in 10mL flasks were diluted to 10mL.

Metal concentrations in fur were determined using an Agilent 7900 ICP-MS. Metal standards were ordered from Sigma-Aldrich. Standards were mixed and diluted to 10ppm. The ICP-MS metal mix internal standard (10ppm standard, Agilent Technologies, Santa Clara, CA). A concentration curve was generated using calibration standards ranging in concentrations from 0.01ppb to 1000ppb. Calibrations were only accepted if all the desired metal responses had a linear distribution with an R^2-value of greater than 0.995. Calibration blanks were included. The standard at the midpoint of the calibration curve was used as a Continuing Calibration Check (CCC) for quality control. The CCC was analyzed every 20 samples to ensure instrument stability. Internal standards were used to monitor percent recovery. If responses fell below 85% or rose above 120% recovery, standards and outlier samples were rerun. Fur weights were used in combination with the dilution volume to determine a dilution factor (dilution factor = dilution volume / weight) for each sample.

Table S4.1. Sample size and minimum, median, and maximum concentrations of metals measured in flying fox fur, separated by species. Concentrations are reported to a maximum of three significant figures.

Sample	Analyte	Unit	Black flying fox				Grey-headed flying fox				Spectacled flying fox			
			n	*min*	*median*	*max*	*n*	*min*	*median*	*max*	*n*	*min*	*median*	*max*
Fur	antimony	ng/g	162	12.1	90.4	516	277	14.1	83.0	1290	45	19.3	49.3	189
	arsenic	ng/g	162	20.7	190	1780	277	28.4	212	3460	45	32.1	93.7	596
	cadmium	ng/g	162	5.27	38.6	321	277	3.10	31.2	510	45	15.4	84.0	3300
	chromium	ng/g	162	269	1450	8320	277	261	1080	16700	45	530	885	8860
	cobalt	ng/g	162	3.60	202	35300	277	7.84	74.3	1170	45	55.6	387	3420
	copper	ng/g	162	2030	9000	46900	277	2620	9960	111000	45	2660	6130	22000
	lead	ng/g	162	172	1260	9960	277	179	1640	28900	45	228	2260	32300
	(total) mercury	ng/g	274	1.38	18.9	416	314	5.67	25.1	119	43	3.91	36.8	262
	nickel	ng/g	162	120	718	19700	277	125	440	247000	45	421	658	1840
	selenium[1]	ng/g	162	46.5	450	1880	275	46.3	484	3380	45	94.5	610	1540
	strontium	ng/g	162	2650	17900	118000	277	743	3510	103000	45	1250	3390	8960
	tin	ng/g	162	1270	5170	27600	277	1150	5420	53900	45	2180	4020	12600
	vanadium	ng/g	162	46.0	460	3860	277	16.9	154	1570	45	28.6	84.7	210

[1]Two additional selenium concentrations were below detection level.

Table S4.2. Model results for GLMM (Poisson distribution, log link) of ectoparasite burden as a function of log-transformed concentrations of 13 metals measured in flying fox fur (*n* = 157; bats from Queensland sites only). Capture site was included as a random effect. WFR: weight to forearm ratio. Black flying fox is the reference level for species and adult is the reference level for age class.

Model term	**Estimate**	**SE**	**z**	**p**
antimony	-0.12	0.20	-0.56	0.57
arsenic	0.049	0.16	0.30	0.77
cadmium	-0.14	0.19	-0.77	0.44
chromium	0.27	0.14	1.95	0.05
cobalt	-0.21	0.096	-2.15	**0.03**
copper	-0.38	0.27	-1.42	0.16
lead	0.042	0.19	0.23	0.82
(total) mercury	-0.23	0.11	-2.19	**0.03**
nickel	0.49	0.12	4.13	**3.7e-5**
selenium	0.73	0.21	3.42	**6.2e-4**
strontium	-0.44	0.23	-1.91	0.06
tin	-0.077	0.30	-0.26	0.80
vanadium	0.023	0.25	0.093	0.93
sex(male)	-0.11	0.21	-0.52	0.60
WFR	-0.21	0.22	-0.96	0.34
species(GHFF)	0.32	0.67	0.48	0.63
species(SFF)	1.06	0.77	1.38	0.17
age class (juvenile/subadult)	-0.13	0.38	-0.35	0.73

Table S4.3. Model results for GLM (binomial distribution, logit link) of blood parasite infection as a function of log-transformed concentrations of 13 metals measured in fur (*n* = 79 from Tolga, Tamworth, and Woolgoolga). Grey-headed flying fox is the reference level for species.

Model term	**Estimate**	**SE**	**z**	**p**
antimony	0.90	0.75	1.20	0.23
arsenic	-0.90	0.59	-1.52	0.13
cadmium	0.29	0.61	0.47	0.64
chromium	0.57	1.07	0.54	0.59
cobalt	-0.30	0.43	-0.69	0.49
copper	-1.28	1.18	-1.09	0.28
lead	-0.96	0.58	-1.64	0.10
total mercury	0.53	0.52	1.03	0.30
nickel	-1.04	1.51	-0.69	0.49
selenium	0.81	0.98	0.83	0.41
strontium	2.47	1.32	1.87	0.06
tin	0.97	2.07	0.47	0.64
vanadium	0.53	1.03	0.51	0.61
sex(male)	0.34	0.76	0.45	0.65
WFR	-2.92	1.21	-2.42	**0.02**
species(SFF)	-0.41	1.23	-0.34	0.74
age class (juvenile/ subadult)	-1.29	1.68	-0.77	0.44

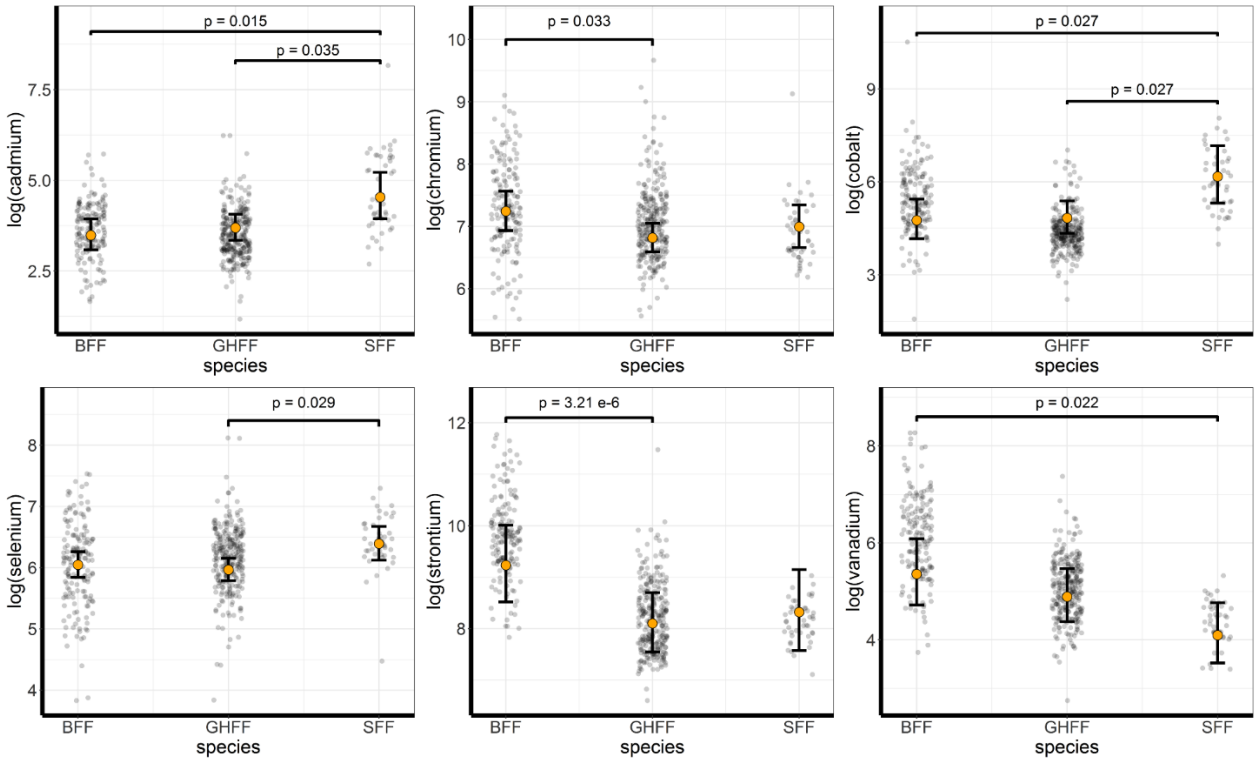

Figure S4.1. Plots showing significant species differences in log-transformed concentrations of metals measured in fur. A GLMM (gamma distribution, log link) was used for each metal, with species as a fixed effect and site as a random effect. Predicted means and 95% confidence intervals are depicted by an orange dot and black bars, while jittered raw data are plotted as gray points. Species pairwise comparisons were performed with a Holm adjustment for multiple comparisons. Plots are not displayed for those metals with no significant species differences (antimony, arsenic, copper, lead, mercury, nickel, tin).

APPENDIX D

CHAPTER 5 SUPPLEMENTAL INFORMATION

Supplemental methods

Details of model parameterization

Demography and movement: The density-independent birth rate, b_0, was estimated by taking the maximum annual number of offspring raised by a flying fox [36], and equating this to the expected number of births in one year, $e^{b_0*(1\ year)}$. The mortality rate, m, was obtained from [276], corresponding to a 10-year lifespan. The density-dependent component of the birth rate, b_1, was fixed so that the disease-free carrying capacity in a toxicant-free landscape is 50,000, i.e. $b_1 = (b_0 - m)/50000$. Some flying foxes move between roosts frequently (every 1-2 weeks) while others can spend months at a site [110]; here, the baseline dispersal rate, σ, was set assuming that the probability of remaining in the same location for one year, $e^{-\sigma*(1\ year)}$, = 0.1. The recovery rate, γ, was estimated as the reciprocal of an expected infection duration of 0.027 years (i.e. 10 days), based on a mid-range estimate of duration of viremia [254].

Table S5.1. Ranges for parameters varied in the Latin hypercube sampling procedure

Parameter	***Definition***	***Range***
β_T	Transmission rate in toxic habitat	$\beta_P \pm 0.75*\beta_P$
μ	Disease-induced mortality	0 – 1
c_σ	Toxicant-imposed movement cost	0.05 – 0.95
c_m	Toxicant-imposed survival cost	0.05 – 0.95
α	Net effect on mortality of being infected while in toxic habitat	0 – 19.9

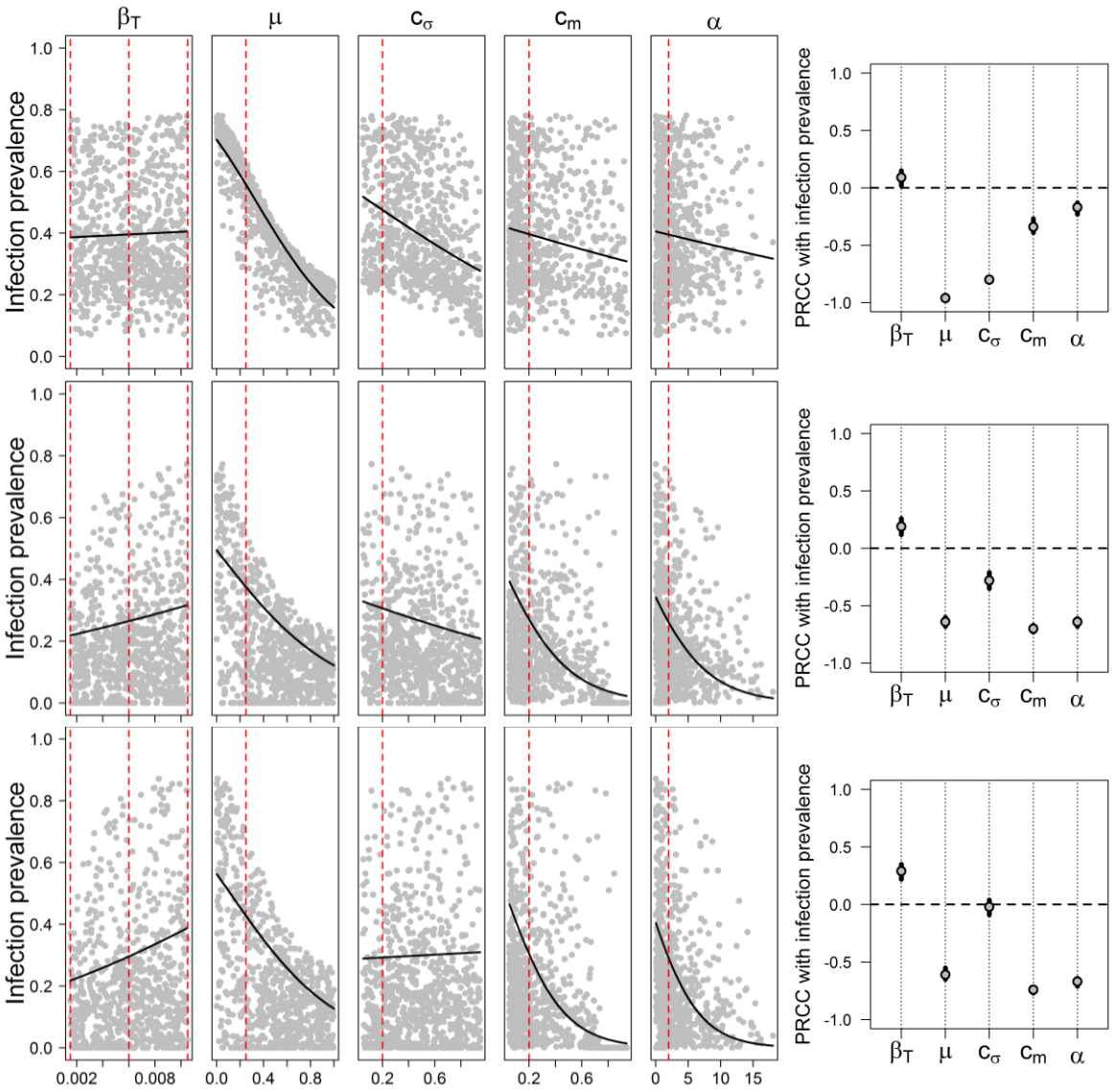

Figure S5.1. Results of sensitivity analyses. Left column: Predicted equilibrium (50 years) infection prevalence with varying parameter values (see Table S5.1) using Latin Hypercube Sampling to sample the parameter space. Right column: Partial Rank Correlation Coefficient (PRCC) sensitivity analysis. PRCC values indicate the strength and direction of association between model parameters and infection prevalence. Results are provided for three values of landscape contamination (top row: $f = 0.1$, middle row: $f = 0.5$, bottom row: $f = 0.9$).