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Food for contagion: synthesis and future directions for studying host–parasite responses to resource shifts in anthropogenic environments

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Human-provided resource subsidies for wildlife are diverse, common and have profound consequences for wildlife–pathogen interactions, as demonstrated by papers in this themed issue spanning empirical, theoretical and management perspectives from a range of study systems. Contributions cut across scales of organization, from the within-host dynamics of immune function, to population-level impacts on parasite transmission, to landscape- and regional-scale patterns of infection. In this concluding paper, we identify common threads and key findings from author contributions, including the consequences of resource subsidies for (i) host immunity; (ii) animal aggregation and contact rates; (iii) host movement and landscape-level infection patterns; and (iv) interspecific contacts and cross-species transmission. Exciting avenues for future work include studies that integrate mechanistic modelling and empirical approaches to better explore cross-scale processes, and experimental manipulations of food resources to quantify host and pathogen responses. Work is also needed to examine evolutionary responses to provisioning, and ask how diet-altered changes to the host microbiome influence infection processes. Given the massive public health and conservation implications of anthropogenic resource shifts, we end by underscoring the need for practical recommendations to manage supplemental feeding practices, limit human–wildlife conflicts over shared food resources and reduce cross-species transmission risks, including to humans.

This article is part of the theme issue 'Anthropogenic resource subsidies and host–parasite dynamics in wildlife'.

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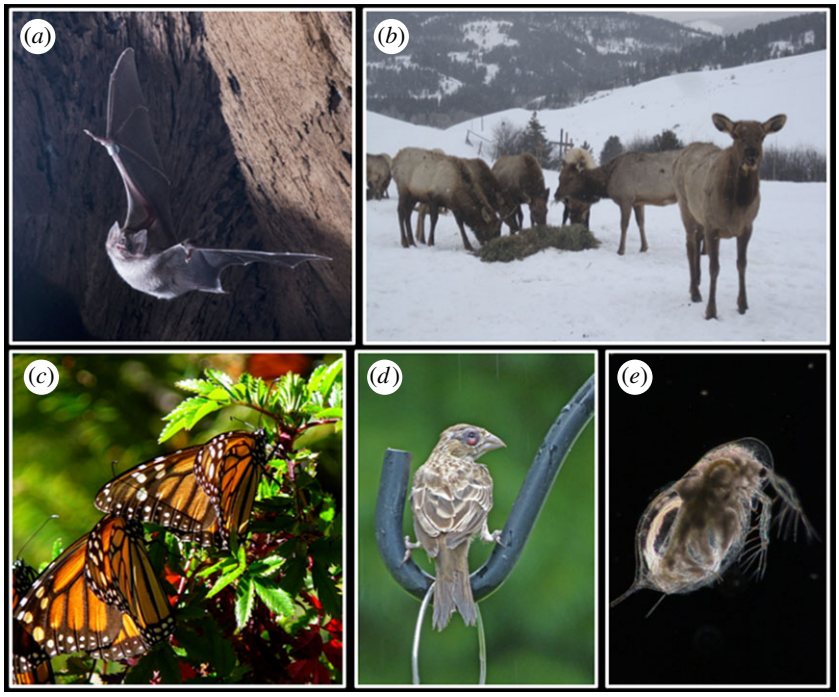

Figure 1. Taxonomic breadth of hosts provisioned by humans covered by studies in this theme issue: (*a*) common vampire bat (*Desmodus rotundus*) in Belize (Brock Fenton), (*b*) elk (*Cervus elaphus*) in the Greater Yellowstone Ecosystem (Paul Cross), (*c*) monarch butterflies (*Danaus plexippus*) in Mexico (Natalie Tarpein), (*d*) house finch (*Haemorhous mexicanus*) infected with *Mycoplasma gallisepticum* in North America (Bob Vuxinic), and (*e*) *Daphnia dentifera* infected with a fungal pathogen (*Metschnikowia bicuspidata*) (Tad Dallas [9]).

1. Introduction

Human feeding of wildlife is pervasive and can occur through both intentional (bird feeders, tourist sites; [1,2]) and unintentional routes (landfills, agricultural crops; [3,4]). In response, animal populations can shift movement behaviours or geographic ranges, experience higher densities and contact rates and show changes in demographic rates and interactions with other species. The population- and community-ecological consequences of supplemental feeding in wildlife have rarely been explored and could be far-reaching, particularly for infectious disease dynamics [5–8].

Papers in this issue directly examine the interactions between anthropogenic resource subsidy and infectious disease dynamics in wildlife using diverse approaches that include mechanistic models, observational field studies and experiments, analysis of citizen science data, and synthetic reviews. Empirical studies presented here examine diverse and engaging empirical systems, ranging from birds at backyard feeders, to bats in urban and agricultural environments, to elk in Yellowstone National Park (figure 1). Despite differences in the biology of distinct systems and environmental contexts, papers in this theme issue point to common questions, patterns and challenges for future work. Our goals in writing this synthesis are to identify these common threads and outline several immediate priorities for future research on the links between human resource subsidies and wildlife disease.

The taxonomic breadth of hosts and pathogens affected by resource provisioning, and the range of food sources examined here, underscore how pervasive this phenomenon has become. Given that responses of several pathogens studied here are accompanied by elevated risks of cross-species transmission to humans, livestock or vulnerable wildlife populations, studies that provide a mechanistic understanding are sorely needed to predict future responses to feeding by humans. The inevitability that human populations will continue to expand, alter habitats globally and encroach on wildlife, means that animal use of resources provided by humans will only increase, lending a sense of urgency to understanding the impacts for wildlife, domestic animal and human health [10].

2. Key findings and common threads across diverse systems and approaches

(a) Host immunity shows complex responses to resource provisioning

Because mounting and maintaining immune defences require energy and nutrients [11,12], access to anthropogenic food subsidies could increase the immune function of wildlife, especially during times or in habitats where natural food sources are scarce or limited [13]. Under the common assumption that provisioning leads to better-defended hosts, pathogen transmission should decrease owing to lower infection probability or faster recovery times [14,15], but such effects might be offset by other processes like aggregation around food that increase pathogen transmission [16]. Hite & Cressler [17] used a nested mechanistic models to show that even if resources decrease host susceptibility to infection, an increase in host densities in response to resource subsidies can override this effect and produce a higher total transmission rate.

Empirical studies in this issue showed that the relationship between provisioning and immunity can depend on the type of defence, quality of resources, and host and pathogen taxonomy, leading to divergent outcomes among study systems (reviewed in Strandin *et al*. [18]). This finding is consistent with past work on domesticated animals showing that

different components of host immunity respond differently to resource subsidies, in part because of the variable costs of different immune process, and also because key macro- and micronutrients can lead to immune system biases [19,20]. In natural systems, Becker *et al*. [21] found that abundant livestock as food for vampire bats predicts stronger innate immunity relative to adaptive immunity. Heightened innate immunity in the bats was further associated with a lower probability of infection by *Bartonella* and haemoplasmas. Importantly, individual dietary history itself did not strongly predict variation in bat immune profiles, suggesting that broader habitat-level factors associated with livestock rearing could underlie parasite exposure and host immunity. In other cases, such as elk supplemented at winter feedgrounds (Cotterill *et al*. [22] and urban flying foxes [23], researchers hypothesized decreased immunocompetence with food provisioning, owing to elevated stress hormones stemming from high host densities and due to coinfections that impair immune response. Immune activity can also be compromised if human-provided food is contaminated with toxins or drugs. As a case in point, Spanish imperial eagles supplemented for conservation purposes with domestic rabbits (that had been treated with antibiotics and antiparasitic drugs) showed decreased complement activity owing to the presence of pharmaceuticals (especially fluoroquinolones) in their food [24]. Similarly, vampire bats that fed more consistently on domestic animals in agricultural habitats had higher concentrations of mercury that were associated with weaker bacterial killing ability of plasma [25].

It is important to note that evidence for nutritional condition altering wildlife immune defences is limited to a relatively small number of hosts, and studies of macro- and micro-nutrient influences on immunity are needed to more critically evaluate this assumption. Genome-wide RNA sequencing could help researchers focus on particular defence mechanisms by quantifying immune gene expression between provisioned and unprovisioned groups, and those with or without known infections [26,27]. In future work, phylogenetically informed meta-analysis could help quantify the importance of food quantity, quality, and host and pathogen traits [28] for immune defence and infection outcomes across wildlife systems.

(b) Behavioural changes in foraging and contact can alter local transmission processes

Several studies in this theme issue demonstrate how resource provisioning can alter key behaviours that underlie pathogen transmission, including foraging behaviour, aggregation and contacts between species [16,29,30]. Crowding of individuals around supplemental resources can lead to higher host densities and contact rates, and thus increase density-dependent transmission, as illustrated previously through theoretical models [14]. Moyers *et al*. [31] designed an experiment to test how feeder density influenced contact rates and exposure to the bacterium *Mycoplasma gallisepticum* in captive house finches. Their work showed that higher bird feeder density in enclosures caused the rapid spread of clinical infections, whereas lower feeder density reduced pathogen spread, possibly due in part to the presence of sub-clinical and potentially immunizing exposures. Importantly, further work is needed to examine how individual-level host heterogeneity in the use of supplemental resources contributes to population-level infection dynamics. For example, can subsets of hosts that aggregate around resources act as super-spreaders, or might host heterogeneity limit the population-level spread of disease?

Cotterill *et al*. [22] reviewed the implications of intentional winter feeding of elk (to limit encounters with cattle) in the western USA. Feed grounds have facilitated brucellosis transmission among elk by elevating local density and contact rates [32] and, more speculatively, by decreasing immune function. Feeding has now created a policy conundrum: high infection prevalence in elk leads to greater motivation to separate elk and cattle, which leads to continued winter feeding and further infection risk. While numerous papers in this theme issue advance a mechanistic understanding of the links between disease and provisioning, disentangling the roles of aggregation and subsequent contact rates, versus changes in immune functions, for driving pathogen transmission will require further work.

Resource provisioning often causes changes in diet and foraging behaviours, especially among urbanized wildlife populations that subsist on supplemental food. Murray *et al*. [33] showed that white ibises shifting from natural wetlands to urban parks in Florida, where they commonly forage on provisioned food, have lower ectoparasite burdens. To explain this pattern, the authors hypothesize that easier food access might allow birds to spend less time foraging and more time preening to remove parasites. In urban and coastal Queensland, the Australian white ibis experienced explosive population growth in the 1990s due to provisioning from open landfills [34,35]. The abundance of anthropogenic food waste and deliberate feeding in urban parks led to a shift from coastal nesting and foraging to suburban and urban foraging, bringing ibis into greater contact with each other, and with chickens on poultry farms and people in recreational areas [34]. Increased population density and interaction among ibis and with domesticated animals and people could also increase the risk of intra- and interspecies pathogen transmission. Understanding the mechanistic links between shifts in behaviour and disease risk could be strengthened by future studies that simultaneously measure specific behaviours (at the individual level) and changes in infection (at individual and population levels). For some food-provisioned populations, efforts to limit contact rates during high-risk intervals (e.g. by ending feed dates earlier in the season for elk, or spacing out bird feeders at lower density) or preserve particular behaviours (e.g. such as preening or other anti-parasite behaviours) could prove important for managing infection risk in wildlife.

(c) Behavioural changes in host movement can influence landscape-level disease processes

Provisioning can cause changes to host movements and infection patterns at large spatial scales. As reviewed by Satterfield *et al*. [36], anthropogenic food subsidies can decrease migratory movements and concentrate hosts into resource-subsidized regions, where greater host aggregation, year-round parasite accumulation and longer residency times could increase exposure to pathogens [6,28]. The authors note that shifts towards more sedentary behaviour in response to resource provisioning have occurred for multiple migratory and nomadic species, in some cases associated with resulting increases in infection risk [37–40]. For

example, satellite telemetry studies of *Pteropus medius*, the reservoir for Nipah virus in Bangladesh, suggest that this species is much more sedentary than its relative, *P. vampyrus* in Malaysia, which could be due, in part, to anthropogenic food resources (Epstein *et al.*, *unpublished*) [41]. Date palm sap, harvested by humans in Bangladesh, is exploited by frugivorous bats throughout the winter months and is the primary route of Nipah virus spillover from bats to people [42,43]. Alternatively, animals that stop migrating might be exposed to a lower diversity of parasites across their migratory range, and more limited host movements could reduce the spatial spread of pathogens [44,45]. A theoretical model [46] explored these questions for a partially migratory host affected by a vector-borne pathogen. The model showed that when provisioning increased the survival of resident hosts during the non-breeding season, both infection prevalence and the fraction of the population that is non-migratory increased. Because greater proportions of residents permit the sustained transmission of pathogens, this behavioural shift could be especially costly to remaining migrants that travel through areas with infected residents; resource provisioning could therefore threaten the persistence of migratory behaviour.

For some highly mobile hosts, resource provisioning will alter daily foraging movements and habitat use. In Australia, naturally nomadic fruit bats have shifted into urban areas where they feed on native and exotic flowering and fruiting trees planted by humans [38,47]. Paez *et al*. [48] applied optimal foraging theory to explore how urban bat colonies alter their foraging strategies in response to decreasing native habitat and seasonal food availability. Their work predicts that residency in urban patches will increase as native foraging habitats become more isolated, and during periods of overall food scarcity. Longer residency in urban centres could set the stage for less frequent but larger viral outbreaks in bats, resulting in higher exposure to humans and domesticated animals [47,49].

(d) Changes to interspecific interactions can cause cross-species transmission and pathogen emergence

Cross-species pathogen transmission requires several ecological, epidemiological and behavioural factors to align [50]. Importantly, anthropogenic provisioning can influence multiple components of this alignment by (*i*) changing host community composition, (*ii*) altering infection dynamics within populations of reservoir hosts, and (*iii*) affecting contact rates between host species. First, because the responses of host species to novel resources in human-altered landscapes can range from disappearance to explosive population growth, provisioning can dramatically alter host community composition and patterns of pathogen transmission [51–53]. As an example of these changes, large-scale monocultures in Brazil and Panama altered rodent communities and increased human exposures to rodent species infected with hantavirus [54,55]. At the largest spatial scales, provisioning could expand host geographic ranges, creating novel opportunities for cross-species transmission where hosts previously did not co-occur [56]. Second, changes to infection dynamics within primary host species (see above sections) can have knock-on effects that amplify or dampen the probability of transmission given interspecific contacts [16]. Third, even if host community composition and disease dynamics in reservoir species remain unchanged, provisioning can facilitate cross-species transmission by altering the frequency and nature of inter-species contacts. For example, bats foraging on mango trees planted near pig farms, or bats drinking palm sap as it runs down tree trunks into collecting vessels, created new routes of Nipah virus transmission from bats to pigs and humans, respectively [43,57]. The common practice of allowing domestic animals to feed on dropped or bitten fruit, that may have been contaminated by bats, also increases the risk of pathogen transmission [58,59]. In Bangladesh, 26 common fruits grown and eaten by people are known to be eaten by frugivorous bats, and eating dropped fruit with animal bite marks regularly occurs (Epstein *et al*., unpublished.) Similar processes could influence pathogen transmission among wildlife when resources promote multi-species aggregations of previously ecologically isolated species [60,61]. Importantly, these mechanisms of resource-driven changes in cross-species transmission might act synergistically. As discussed by Becker *et al*. [21], livestock both stimulates vampire bat population growth and, by its own presence, expands opportunities for cross-species transmission of rabies virus and potentially other pathogens.

Altered dynamics of cross-species transmission are among the most visible and alarming responses to resource provisioning because they can directly impact human health, agriculture, or the conservation of vulnerable wildlife populations. For example, livestock-driven increases in vampire bat rabies have made this disease one of the three most important zoonoses in Latin America and a significant barrier to the advancement of agrarian communities [62,63]. Similarly, the resource-driven rise of Hendra virus cases in humans and horses in Australia created economic and social challenges, ranging from the rising need for veterinary vaccines to protect horses, to conservation challenges as bat persecution is promoted for disease control [47,64]. In Asia, the transmission of zoonoses from provisioned non-human primates to people impacts tourism [65]. Importantly, provisioned landscapes can provide opportunities for spillover infections from humans (or livestock) to wildlife, and potential spillback into humans. For example, in parts of Africa, baboons commonly frequent human settlements and obtain food from houses or waste sites. Parasitological surveys showed baboons near these settlements can harbour parasitic worms and protozoa that commonly infect humans, although further diagnostic work is needed to determine whether the primate isolates match parasite genotypes recovered from nearby humans [66,67]. Better quantifying the contexts under which provisioning mediates cross-species transmission could provide an epidemiological lever to promote more responsible management of anthropogenic food subsidies for wildlife.

3. Critical priorities for future work

(a) Taxonomic biases in studies of provisioning and infection

Work included in this Theme Issue reflects the taxonomic breadth of hosts and parasites studied in the context of resource provisioning, and also highlights taxonomic gaps to be addressed in future work. The empirical studies

presented here focus primarily on mammals (e.g. bats, ungulates) and birds (e.g. passerines, wading birds), with less representation from invertebrates (e.g. monarch butterflies, *Daphnia*). Studies here also focused heavily on microparasites, particularly bacteria and viruses, transmitted through direct and non-close contact (e.g. faecal–oral routes), although ectoparasites are also represented. More generally, throughout the literature, studies of provisioning and host–parasite interactions are biased towards these taxa (reviewed in [16,28]). For example, a recent meta-analysis of over 300 host-parasite interactions was dominated by studies of microparasites transmitted by close and non-close contact, and of helminths transmitted through non-close contact and intermediate hosts [28]. Vector-borne diseases, and protozoan and fungal parasites are generally poorly represented, highlighting a priority for future studies, particularly in light of expanding vector distributions under climate change and the role of fungal parasites in wildlife population declines [68–71]. Past studies of food provisioning and wildlife disease are also heavily biased towards mammals and birds, with much less work on invertebrates and other ectotherms. Civitello *et al*. [72] highlight how nutrient inputs into aquatic ecosystems (as a form of anthropogenic subsidy) can have similar effects on host-parasite interactions as food subsidy to wildlife (by increasing host density and altering parasite production within hosts). This observation stresses the need for greater inclusion of amphibians, reptiles, fish, and invertebrates in studies of provisioning and disease.

(b) Modelling studies to link effects of provisioning across biological scales

Resources can affect within-host processes relevant to pathogen colonization, between-host transmission at the population level, and landscape-level processes such as host dispersal. Mathematical models provide powerful tools for linking infection dynamics across scales of organization and for informing the conditions under which provisioning can increase or decrease infection. For example, theory to date has shown that when resources strongly enhance host defences, this can limit pathogen transmission that otherwise would increase from resource-induced increases in host density [14,73]. If host defences are unchanged or weakened by human-provided resources, increased exposure to pathogens resulting from elevated host densities and behavioural changes are likely to increase pathogen invasion and prevalence [16]. A separate body of theory used metapopulation models to examine how the distribution of resource-rich habitats, and their impact on colonization and extinction, affects host–pathogen dynamics. This work shows that increasing the frequency of provisioning across the landscape increases pathogen establishment and spread; yet nonlinear relationships between infection prevalence and the relative abundance of provisioned habitats can emerge if provisioning and infection influence host movement decisions and dispersal success [74,75]. Despite these recent advances, a need remains for mathematical models that more explicitly link processes across individual, population, and landscape scales.

In this issue, Hite & Cressler [17] contribute a cross-scale approach by developing a mechanistic framework coupling within-host processes (through improved immune defence and increased pathogen replication in response to resources) and between-host processes (through transmission and resource-mediated population growth rates). Their model explores the consequences of resource acquisition for parasite virulence evolution and its potential to stabilize resource-driven cycles in host population dynamics. The authors demonstrate that linking within-host and population-level processes can produce cyclic host population dynamics and associated within-host cycles of high and low parasite replication, an emergent phenomenon that does not occur when within-host processes are ignored. In other work, Civitello *et al*. [72] demonstrate that incorporating trophic complexity (by considering predators and competitors of provisioned hosts) can reverse predictions about resource-mediated increases in pathogen prevalence. Resource subsidies increase pathogen prevalence when only hosts are present, but competitors and predators can lower infection prevalence (in some cases causing pathogen extinction) when resources are abundant. These studies highlight the importance of considering processes at scales above and below the population level in predicting resource subsidy effects on pathogen transmission dynamics. An additional key insight from theoretical work is that empirical studies must be long enough relative to the duration of infection to capture stable or cyclic responses of population and infection dynamics under provisioning. Promising future avenues include investigating how resources affect coinfection (e.g. in shaping immune-mediated competitive interactions between micro- and macroparasites); the responses of parasites with complex transmission modes (e.g. vector-borne and trophically transmitted parasites); and relationships for multi-host pathogens where host species that differ in competence might respond differently to provisioned resources (e.g. in population density or susceptibility to infection) [10].

Future theoretical models that are paired closely with detailed empirical work could be especially fruitful in understanding the dynamical outcomes of provisioning. Such work could couple local and landscape-level effects of resources on well studied host–pathogen interactions. Given that theory to date on provisioning and infection has focused separately on population and metapopulation scales, one area that is crucially needed involves models that explicitly link local dynamics (e.g. resource effects on individual hosts or contact rates) to regional movements of the host and pathogen that also depend on resource distributions (figure 2). From an applied perspective, such models could also allow researchers to predict the outcomes of different habitat management scenarios that might alter resources in ways that lower infection risks [14,74,76].

(c) Experimental manipulations of food resources to quantify responses of hosts and pathogens

Research manipulating food resources is noticeably rare among the growing body of literature developing around the effects of anthropogenic food subsidies on host–parasite dynamics. Indeed, this theme issue reflects this disparity between observational and experimental approaches, with only a single study [31] among the latter. A handful of studies published elsewhere have experimentally manipulated food; for example, work by Wright & Gompper [77] showed that clumped food resources increased the transmission of endoparasites in raccoons, suggesting a possible behavioural mechanism for changes in prevalence. Wilcoxen *et al*. [78]

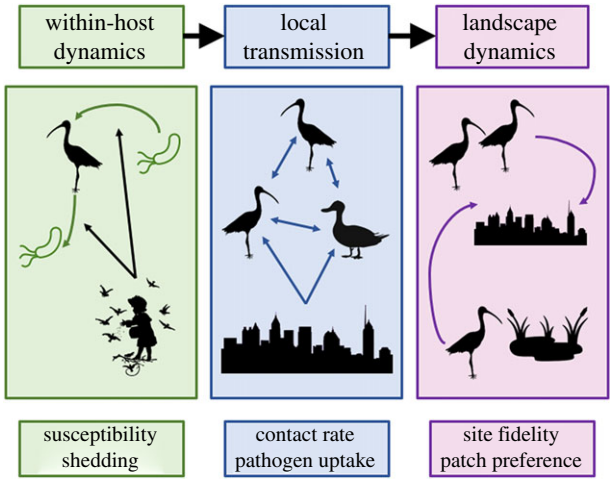

Figure 2. Interactions between human-provided food and pathogen dynamics can occur at multiple scales of organization, as illustrated by American white ibis (*Eudocimus albus*) and environmentally transmitted enteric pathogens. Anthropogenic food subsidies in urban habitats could influence within-host dynamics (e.g. individual susceptibility and intensity of pathogen shedding, on left), local transmission processes (e.g. intra- and interspecific contact rates, uptake of pathogen from the environment, in centre) and landscape dynamics (e.g. host movement between natural and provisioned habitats, site fidelity, on right). Combined modelling and empirical work is needed to quantify the importance of processes operating within scales, and to predict how processes at one scale affect dynamics at larger scales of organization. (Online version in colour.)

and Galbraith *et al.* [79] both manipulated the presence or absence of bird feeders and found effects of feeder presence on health-associated traits such as body condition, as well as effects on the prevalence of diverse parasites and pathogens. Responses to feeder presence in Galbraith *et al.* [79] were parasite- and host-specific. Although experimental in nature, field studies such as these still have difficulty establishing definite causation (e.g. in contrast, see [80]). For example, in some systems, diseased animals could be more strongly motivated to seek out supplemental food resources, leading to patterns of higher infection prevalence at supplemented sites that could also be interpreted as a positive effect of resources on pathogen transmission [81].

Most experiments to date manipulate food through experimental supplementation, but future work could reduce access to anthropogenic foods, especially for species for which finding or monitoring unprovisioned populations is difficult. For example, vampire bats in Latin America are most abundant and thus readily sampled near livestock-rich areas [82], and locating unprovisioned rainforest populations is difficult [83]. Moreover, multiple confounding factors, including habitat characteristics and host density, differ between provisioned and unprovisioned groups [21]. In this case, restricting access to livestock, such as through artificial lighting to deter bat feeding [84], might be one way to monitor host and pathogen responses to reduced access to anthropogenic food. For other hosts, limiting access to human foods through fencing, or through campaigns to restrict tourist feeding of wildlife, could generate heterogeneity in resources.

Manipulating food quantity and quality is needed to explore the effects of food nutritional value on multiple measures of host immune defence, the host microbiome (discussed below), and susceptibility to target pathogens. Some experimental provisioning work has examined individual and population-level outcomes in birds and rodents [85–88]. Many of these experiments have been conducted in semi-controlled settings, such as aviaries and field enclosures, reflecting challenges associated with regulating food and disease exposure in free-ranging wildlife, which can disperse over large areas. However, confinement might also impact disease outcomes in unnatural ways, such as by increasing the frequency and intensity of intraspecific transmission opportunities, and inducing stress that often impairs host immunity [18].

Future field experiments might simultaneously control multiple components of provisioning, especially if anthropogenic foods dampen the seasonality or pulsed timing of natural resources, and at the same time make food more spatially aggregated, or change resource quality. These same studies could experimentally reduce infections in some hosts, to separate responses of host behaviour, physiology and fitness from parasite infection itself. Given the pervasiveness of provisioning, many opportunities exist to integrate experiments within current feeding activities, particularly within wildlife management and conservation efforts (e.g. [22]). Moreover, the strong causal inference provided by well-planned and executed experiments (e.g. by manipulating both infection and resources in free-ranging wildlife [85]) necessitates greater emphasis on these approaches to better understand how anthropogenic resources affect host–parasite dynamics.

(d) Understanding consequences of resource subsidies for the evolution of pathogen virulence

By affecting pathogen transmission and within-host processes, resource provisioning could ultimately affect host and pathogen evolution, an idea explored in depth by Hite & Cressler [17]. General theory on virulence evolution predicts that greater opportunities for horizontal pathogen transmission, such as might be created by aggregation around provisioned resources, could favour the evolution of more virulent pathogen strains [89]. As described earlier, Hite & Cressler's paper used a multi-scale model to show that such a result can arise even when provisioning increases host immunity. Empirical work is crucially needed from naturally occurring host–pathogen systems to test the virulence of pathogen strains from provisioned and unprovisioned host populations (e.g. [37]).

Although not examined by papers in this issue, provisioning can, in some cases, allow wildlife to better tolerate infection [16], an idea supported by laboratory studies demonstrating that improved nutrition can prolong the survival of infected animals and increase the duration of pathogen shedding [90,91]. Because host mortality cuts short the infectious period for many pathogens, this can constrain greater within-host replication by pathogens, and hence limit virulence evolution. By contrast, more tolerant hosts could select for more virulent pathogen strains by releasing pathogens from some of the costs of virulence [90]. Thus, although improved condition could reduce disease-induced mortality of provisioned hosts in the short term, provisioning could favour the evolution of higher virulence in the longer term [92]. Evolutionary models and empirical studies that explore the impact of resource subsidies on host tolerance to

infection, within the context of other processes, are needed to identify the conditions under which provisioned populations support pathogen strains of higher virulence.

(e) Seeking how changes to the host microbiome affect larger-scale infection processes

Another important area for future work is understanding how dietary changes associated with provisioning could impact the host microbiome and within-host dynamics [16]. The composition of gut microbial communities can influence the immune system, thereby affecting host susceptibility and pathogen colonization [93]. For example, experimental simplification of microbiota from Cuban tree frog tadpoles increased their susceptibility to invasion by gut helminths as adults [94]. The composition and diversity of the gut microbiome is itself strongly shaped by individual diet [95,96], and thus provisioned wildlife would be expected to differ in both their microbiota and their susceptibility to enteric pathogens. Yet field studies of microbiomes in provisioned hosts are rare; in one example, the gut microbiota of baboons foraging on leftover food in Bedouin settlements mirrored the gut microbiota of people living in the Bedouin communities [97].

Comparative work on the microbiome between provisioned and wild populations is necessary to establish how specific dietary differences influence gut microbial composition and diversity. For example, shifts from protein- to carbohydrate-rich diets in urban-foraging wildlife such as white ibis [33] could have especially pronounced effects on microbiomes, and, in turn, pathogen invasion. In one rare case study, shifts toward grain-based diets may have disrupted the microbiota of Canada geese and facilitated *Clostridium perfringens* colonization [98]. From another perspective, foraging on anthropogenic resources in urban and agricultural environments could also expose species such as vampire bats and flying foxes [21,48] to contaminants (e.g. pesticides and antibiotics) that alter microbial community composition [99]. When possible, manipulative experiments are needed to examine causal relationships between different components of provisioned diets and the microbiome. Moreover, relationships among microbiome diversity, microbiome composition and susceptibility to pathogen challenge in the context of provisioning must be elucidated to understand how changing microbiota influences host susceptibility to infection. Finally, data linking diet, microbial diversity and immunity could be used to parameterize mathematical models to holistically explore how provisioning influences infection dynamics.

4. Implications of provisioning for conservation and human health

(a) The importance of understanding human motivations for feeding wildlife

The pervasiveness and popularity of intentional wildlife provisioning (e.g. [100]) suggests that humans have strong underlying motivations for this activity, particularly in the case of backyard bird feeding, on which people spend $4.5 billion annually in the USA alone [101]. Although bird feeding is the most prevalent form of intentional provisioning, a clear picture of the disease risks this activity imposes on wildlife and humans remains elusive [81]. The intentional feeding of charismatic mammals is common and probably alters disease risk as well. For example, provisioning of wild primates is prevalent within the context of Hindu and Buddhist culture, and has been enhanced with increasing tourism [102]. Motivations for feeding wildlife are complex and may vary regionally [103,104], but numerous studies have shown a key impetus of the psychological benefits of direct human–wildlife interaction [105], including a sense of pleasure or relaxation, feelings of usefulness and an increased connection to nature [106–109]. In fact, the vast majority of people surveyed about their willingness to interact with wild primates were aware of the potential disease risks associated with this interaction, and yet more than half still responded that they would touch wild primates if given the opportunity [110]. Welfare motivations are also commonly cited by those who provision wildlife [108], including a desire to help wildlife or 'assist them through hard times' [107,109]. Indeed, provisioning tends to be strongest in seasons when natural food is perceived to be limited [107], suggesting a strong role of welfare motivations.

Cox & Gaston [100] suggest that positive reactions from wildlife, as well as psychological benefits to humans, strongly motivate people to offer supplemental foods, although more empirical evidence is needed. For example, humans that receive significant positive benefits from feeding (increased well-being or reduced stress) are probably more likely to continue provisioning. On the other hand, Cox & Gaston [100] also propose that the negative consequences of supplemental feeding, such as disease transmission among wildlife [111], or human health risks, often do not feed back to dampen provisioning behaviour because these effects are rarely apparent to the public [107]. The recent trend toward reduced feeding of (non-bird) wildlife in the USA [100] suggests that active campaigns against feeding of mammals are beginning to influence human behaviour. Thus, by tapping into the welfare motivations for feeding wildlife, changes in human behaviour are possible. Success in changing behaviour might be more even more likely when campaigns directly target the negative effects on humans, such as in cases of human–wildlife conflict and pathogen spillover.

To the extent possible, intentional supplemental feeding should be managed to maximize benefits to both humans and wildlife. For example, the recently documented association between higher levels of afternoon bird abundance and reductions in the severity of depression, anxiety and stress in humans led the authors to propose the active use of supplemental feeding to create 'optimal' bird abundance levels for human health [112]. For many bird species, supplemental feeding decreases starvation risk [113] and can improve breeding success [114]. Yet, feeding has also been associated with changes in community structure [115], range expansion [116], and, as this issue illustrates, pathogen transmission. Unfortunately, it seems unlikely that optimal levels of feeding for humans and wildlife will coincide. Thus, given the species- and habitat-specific effects of supplemental feeding [115,117], determining the ideal levels of provisioning for most wildlife will be challenging. In cases where clear negative effects of resource provisioning on wildlife are documented, educational campaigns would ideally leverage welfare-driven motivations for feeding by creating negative feedback loops on human behaviour [100]. Overall,

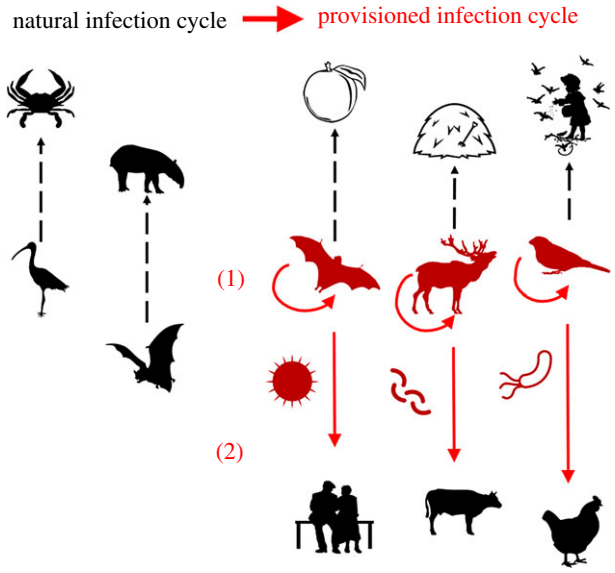

Figure 3. Possible effects of provisioning on amplifying pathogen spillover risks by (1) increasing pathogen transmission and shedding from reservoir hosts (e.g. through increased aggregation, susceptibility and shedding intensity) and (2) increasing opportunities for contact between humans and domestic animals and either reservoir hosts or pathogen in the environment. Silhouettes and arrows display case studies from this theme issue where provisioning had little effect or decreased infection relative to more natural environments (black; white ibis, vampire bats) and where provisioning amplified infection cycles (red; flying foxes, elk, house finches) and could potentially increase the risks of cross-species transmission.

effective management of intentional provisioning will require significantly more data than are currently available on both human motivations for feeding, effects of feeding on wildlife and potential feedback loops between wildlife effects and human behaviour. Given the enormous and potentially growing scale of human supplementation of wildlife [101], developing effective management tools is both timely and critical.

(b) Recommendations for limiting disease risks associated with human–wildlife contacts

The proximity with wildlife afforded by resource subsidies in urban and agricultural landscapes brings humans and domestic animals into contact with wildlife pathogens, and wildlife into contact with human pathogens (figure 3). Some of the most readily observed examples include growing populations of urban mesocarnivores (e.g. foxes, raccoons and skunks) that can attack humans and domestic animals when infected with rabies [118]. Non-human primates can also become aggressive following habituation to human-provided food, leading to the transmission of zoonotic viruses in some cases [119], and exposing primates to respiratory infections from human researchers and tourists in other scenarios [120]. Wildlife professionals might be exposed to zoonotic pathogens when translocating non-human primates in response to human–wildlife conflict [121]. Even when interspecific contacts between wildlife and humans are rare, pathogens can transfer between humans and wildlife by environmental routes or through arthropod vectors. Examples include a rise in human infections with the soil-borne tapeworm *Echinococcus multilocularis*, attributed to provisioned urban red foxes in Europe [122]. Human and animal Nipah virus infections have occurred through the consumption of food contaminated by bat excreta [123,124], and greater human exposures to hantavirus through environmental infectious stages followed the growth of rodent populations that exploit agricultural crops [54,55]. Zooanthroponoses (pathogens transmitted from humans to other animals) are less appreciated, but affect wildlife globally [125,126]. The preponderance of environmentally and vector-transmitted pathogens at the human–wildlife interface raises important challenges to recognize links with resource provisioning. Epidemiological investigations that identify agents of disease must be followed with ecological studies to identify natural hosts and the ecological context that enables cross-species transmission [127]. Fortunately, rapid and powerful DNA/RNA sequencing technologies [128], together with increasingly sophisticated tools for inferring pathogen transmission between species [129] offer currently under-used opportunities to improve scientific understanding of the changing patterns of pathogen transmission in provisioned environments.

Under some circumstances, ecological interventions that build on a mechanistic understanding of host and pathogen biology can prevent cross-species transmission. Most notably, preventing wildlife access to unintentionally provisioned resources, or creating a barrier between provisioned resources and domesticated animals (e.g. planting orchards away from livestock enclosures to reduce the risk of Nipah spillover on farms in Malaysia), can restrict opportunities for overlap between host species and function as a barrier to pathogen spillover [50]. As one key example, blocking the foodborne transmission of Nipah virus from pteropid fruit bats to humans using a bamboo skirt placed at the top of date palm sap collection pots restricts bat access to this shared food resource, and could reduce the risk of Nipah virus exposure in humans [130,131]. This case study highlights not only how basic ecological data on the foraging behaviour of reservoir hosts can aid in the design of interventions, but also how insights from social science and the application of locally available practices can produce economically affordable management tools [132]. Such 'ecological interventions' may also be cheaper and more effective than antibiotics or vaccines that are mobilized after cross-species exposures occur. Other intervention strategies can promote sanitary best practices to prevent the build-up on infectious stages on feeders (e.g. washing backyard bird feeders), encouraging the dispersal of feed in smaller units over larger areas to reduce aggregation and lower contact rates (e.g. with management-based feeding [133]), and educating the public about disease risks posed by well-intentioned but harmful feeding activities [134,135]. Given that resource provisioning is ultimately derived from human actions, perceptions and policies, the integration of ecological, sociological and management perspectives will be a key lever by which infectious disease risks can be minimized for the well-being of humans, domesticated animals and wildlife.

Data accessibility. This article has no additional data.

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