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Assessing the direct and indirect effects of food provisioning and nutrient enrichment on wildlife infectious disease dynamics

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Anthropogenic resource supplementation can shape wildlife disease directly by altering the traits and densities of hosts and parasites or indirectly by stimulating prey, competitor or predator species. We first assess the direct epidemiological consequences of supplementation, highlighting the similarities and differences between food provisioning and two widespread forms of nutrient input: agricultural fertilization and aquatic nutrient enrichment. We then review an aquatic disease system and a general model to assess whether predator and competitor species can enhance or overturn the direct effects of enrichment. All forms of supplementation can directly affect epidemics by increasing host population size or altering parasite production within hosts, but food provisioning is most likely to aggregate hosts and increase parasite transmission. However, if predators or competitors increase in response to supplementation, they could alter resource-fuelled outbreaks in focal hosts. We recommend identifying the traits of hosts, parasites or interacting species that best predict epidemiological responses to supplementation and evaluating the relative importance of these direct and indirect mechanisms. Theory and experiments should examine the timing of behavioural, physiological and demographic changes for realistic, variable scenarios of supplementation. A more integrative view of resource supplementation and wildlife disease could yield broadly applicable disease management strategies.

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

1. Introduction

Infectious diseases of wildlife and humans are emerging and resurging globally, affecting biodiversity conservation, agricultural production and human health [1,2]. These increases in infectious disease have co-occurred with anthropogenic alterations to the environment, spanning local to global scales. For example, forest fragmentation, agrochemical pollution and altered temperature variability caused by global climate change are influencing the distribution and spread of vector-borne and wildlife diseases [3–5]. These concurrent increases in anthropogenic modifications to the environment suggest that human activities can have far-reaching effects on infectious disease dynamics in natural and managed host populations.

Human supplementation of resources is a pervasive and potentially strong driver of disease dynamics in wildlife [6,7]. Intentionally or unintentionally, humans often increase resource availability for wild organisms in almost every ecological context [8]. For instance, humans intentionally provide resources by establishing bird feeders, planting and fertilizing agricultural or horticultural fields, and directly feeding wildlife. Many of these activities unintentionally supplement non-target organisms. For example, mammals regularly forage at bird

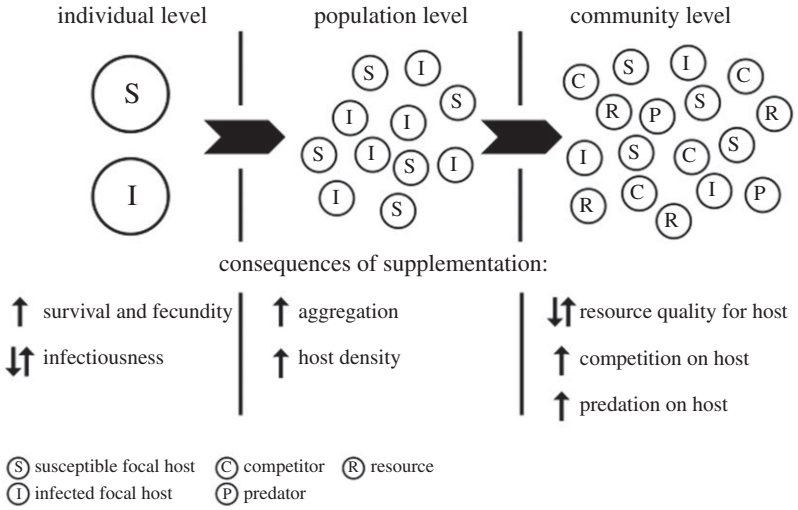

Figure 1. Resource supplementation can alter infectious disease dynamics in wildlife populations through a variety of direct and indirect mechanisms. At the individual level, supplementation can directly increase the survival and fecundity of hosts, and it can alter their production of parasites if infected, thereby influencing infectiousness. At the population level, supplementation can directly increase host density (via increases in survival and fecundity) and aggregation, potentially altering contact and transmission. At the community level, supplementation can indirectly alter disease dynamics by changing the nutritional quality of prey species, as well as attracting or stimulating the densities of competitors and predators of hosts and/or parasites. The relative importance of these direct and indirect mechanisms will likely depend on the duration of supplementation, initial densities of community members, parasite transmission mode and the traits of other species in the community.

feeders, on crops and on human garbage [9,10]. Similarly, agricultural runoff and nutrient deposition arising from wasted or lost nutrients from farming or industrial practices unintentionally fuel primary production in inland and coastal waters, which can lead to eutrophication (extremely high nutrient loading, primary productivity and decomposition, which can degrade aquatic food webs, cause non-infectious diseases such as hypoxia and create anoxic dead zones; [11]). While a single provisioning event can be highly localized, resource provisioning can occur on extremely large scales because of the broad spatial extent or popularity of provisioning as well as nutrient cycling and transport within and across ecosystems [8,12]. Moreover, the effects of large-scale resource provisioning, especially aquatic eutrophication, can persist long after anthropogenic resource inputs have ceased [13].

Resource supplementation by humans can drive disease dynamics in wildlife by altering the traits and densities of hosts and parasites (figure 1; [6,14]). The combined effects of these simultaneous changes can be difficult to predict *a priori*, in part because they may occur along a continuum of time scales. For example, provisioning can cause relatively rapid changes in animal foraging behaviour and movement patterns that can lead to host aggregation and thus increased contact rates as a result of resource provisioning [10]. After these resources are consumed, they can drive substantial increases or decreases in parasite production and virulence through physiological responses of hosts and parasites [15–17]. At longer time scales, supplementation can increase host survival and fecundity, boosting host population sizes [18], which could also promote host contacts and parasite transmission [19,20].

Supplementation can also alter wildlife disease dynamics by affecting the traits or densities of species that may indirectly influence outbreaks, such as prey, predators and competitors of hosts (figure 1). For example, phosphorus enrichment increases the quality of algae for aquatic consumers (a trait). When infected *Daphnia* consume phosphorus-rich algae, they produce four times as many spores of the virulent bacterium *Pasteuria* than hosts that consume phosphorus-limited algae [21]. Additionally, supplementation could increase the density of predators and competitors of hosts that could alter epidemics by competing for resources or consuming hosts and parasites [22]. Thus, the response of other species to supplementation could enhance or oppose the direct effects of supplementation on wildlife disease in a focal host.

Recent reviews and meta-analyses of the effects of resource supplementation on disease dynamics have focused separately on food provisioning to charismatic vertebrate wildlife [6,7], aquatic nutrient enrichment [23] or agricultural fertilization [24]. This compartmentalized approach risks ignoring general insights for wildlife disease in anthropogenically enriched landscapes, and it limits collaboration across disciplines. All of these types of supplementation are extremely widespread. For example, residents of the USA spent more than $3 billion on food for wildlife in 2001 [25]. Agricultural fertilization impacts ecosystems globally, as approximately 190 million metric tons of fertilizer are applied worldwide each year [26]. Similarly, more than 90% of rivers exhibit elevated nitrogen and phosphorus levels in most regions of the USA, and the annual economic costs of eutrophication in the USA exceed $2 billion [27]. Here, we leverage the fact that resource supplementation is a near ubiquitous aspect of the Anthropocene to identify the unifying and unique effects of supplementation on disease across ecological contexts. Thus, we define resource supplementation broadly to include direct human provisioning of food items as well as enrichment of limiting nutrient resources (i.e. agricultural fertilization and aquatic nutrient input). We note that resource supplementation can increase non-infectious disease in humans and wildlife. For example, eutrophication can cause hypoxia among aquatic organisms, increases in CO_2 and nutrient inputs can increase pollen production and therefore human allergies, and elevated nutrients in drinking water can decrease oxygen carrying capacity of haemoglobin in babies, leading to death (blue baby syndrome) [23]. However, in this review we focus on the effects of resource supplementation on infectious disease. Specifically, we

compare the consequences for wildlife infectious disease across types of supplementation, review an aquatic disease system and a general model to illustrate how food web interactions such as predation and competition can alter or overturn these direct effects, and identify research gaps for advancing integrative research on resource availability and the ecology of infectious disease.

2. Epidemiological consequences of supplementation across contexts

(a) Host aggregation and parasite transmission

Resource supplementation can increase parasite transmission among wildlife because it can increase contact among hosts. Resources provided by humans are often clustered in space and time. When animals exploit these resources, they aggregate, causing local densities to increase dramatically near provisioned resources. High levels of host aggregation can facilitate parasite transmission by increasing infectious contacts among hosts or by concentrating environmental stages of parasites [28,29]. Animals aggregate around feeders, garbage sites and other direct provisioning locations. For example, banded mongoose troops in Botswana transmit a novel agent of tuberculosis as they aggregate to scavenge in garbage sites and human faeces [30]. Aggregation around food waste can also occur in aquatic systems. Fishermen may clean and release large quantities of fish carcasses from ships at water access points or docks, attracting high densities of scavenging birds, fish, mammals and invertebrates [31,32]. Moreover, direct food provisioning by humans to charismatic vertebrates uniquely aggregates humans with wildlife hosts and may disproportionately promote cross-species transmission, which is particularly worrisome because most emerging human parasites are zoonotic in origin [2,6]. Nutrient inputs to agricultural and aquatic systems are less likely to aggregate hosts within habitats (e.g. lakes or fields) than direct provisioning, due to uniform application or mixing processes, respectively. However, variation in resource input rates among agricultural fields or water bodies may attract dispersing hosts at landscape scales.

The epidemiological consequences of host aggregation at provisioned resources are driven by behavioural responses to resource availability in the environment. This has two functional consequences for epidemiological dynamics. First, the effects of provisioning on aggregation and transmission should occur rapidly, because changes in behaviour often occur faster than those in physiology or demography. Second, the effects of provisioning on aggregation should most strongly depend on the number, orientation and characteristics of provisioning sites, rather than any increases in resource consumption by individual hosts [6]. Therefore, managing the spatial and temporal distribution of provisioned resources could provide rapid, powerful control for some parasites of wildlife, but it may require large-scale collaboration among wildlife managers, wildlife associations, other organizations and private citizens.

(b) Host demography and density-dependent contact

Resource supplementation could also increase parasite transmission over longer time scales by increasing host population density. Resource supplementation can increase rates of reproduction and survival for many terrestrial and aquatic hosts [18,33]. If population densities are not regulated by other factors (e.g. predators), then increased reproduction and survival should increase host population densities. If contact rates increase with host density, as is assumed in the density-dependent transmission model, then high host densities will increase contact rates and promote parasite transmission [20]. For example, there is strong theoretical and empirical support for increased measles transmission and persistence in dense human populations [34,35]. However, weak, nonlinear or absent relationships between host population size and transmission rates (or prevalence) are common in wildlife populations because host–parasite contact rates can saturate or even decline at high host densities [19,36–38]. These relationships may arise for several reasons. First, parasites may be transmitted with frequency dependence (i.e. independently of density), especially if they are transmitted sexually or in other social encounters. For example, mating encounters, which can transmit *Mycobacterium bovis* among opossums, did not decrease following experimental population reduction, although transmission was not measured [39]. Alternatively, as population size increases, individuals may disperse more widely and population density (abundance per area or volume) may remain constant if the spatial extent of the population increases with abundance. Given the longer time scale needed and the weak relationships between wildlife population sizes and contact rates, increased fecundity and survival may be relatively less important for wildlife disease compared with other mechanisms, such as aggregation, especially if the duration of supplementation is brief relative to host generation time. However, if supplementation increases the survival of infected hosts (e.g. [40]), and therefore the average infectious period, then increased survival could be relatively important, even when transmission does not strongly depend on density. An important nonlinearity for these demographic effects can arise in aquatic systems: extreme nutrient enrichment can trigger anoxic conditions, which can greatly reduce host survival, potentially reducing host densities and the average infectious period and thereby decreasing infectious disease spread [23,41].

In fertilized plant-disease systems, an additional mechanism links increases in host density to parasite transmission. High densities of plant biomass can moderate temperature, increase humidity and decrease the penetration of UV and visible light, which can increase the survival of fungal parasites in the environment and on leaf surfaces [42]. Increased environmental survival of parasites and elevated contact rates with hosts can greatly increase transmission, because they jointly raise the proportion of parasites successfully contacting hosts before death [43]. Indeed, altering the distance between susceptible plants can effectively control disease caused by fungal pathogens of crops [44]. In contrast to fungi, viral plant parasites and their arthropod vectors are often inhibited in cooler, wetter conditions [42]. Thus, traits of parasites and vectors may explain variation in their responses to microclimatic variation driven by resource supplementation to primary producers.

(c) Host energetic condition and parasite production

Parasites steal energy and nutrients from their hosts to support their own growth and reproduction. Thus, starving hosts may limit parasite growth. Supplementation could alleviate this resource limitation and increase parasite growth

rate, infectiousness and virulence. However, resource supplementation often increases host reserves of nutrients, such as protein, carotenoids and vitamins, which are needed to mount energetically expensive immune defences against parasites [45–47]. The ultimate effect of supplementation on within-host parasite production depends on the balance of these bottom–up (resource limitation) and top–down (host defence) processes, as well the energetic costs of other stresses imposed by nutrient enrichment, especially toxic components of provisioned food items or hypoxia [7,23,41]. Indeed, positive, negative and unimodal relationships between resource consumption by hosts and parasite proliferation have been reported across animal, plant and microbial host taxa across ecological contexts [16,24].

A key challenge moving forward is to link traits of hosts or parasites with patterns relating resource consumption to parasite production. For example, increased food consumption often reduces parasite loads in vertebrates, but it more frequently increases parasite loads for invertebrate and bacterial hosts [16]. These differences could be attributed to host immune traits, such as the presence or strength of adaptive immune responses, but specific mechanisms have not yet been identified [16]. Physiological studies that manipulate host and parasite access to resources independently or sequentially could identify mechanisms driving this variation. For example, resource supplementation increases frog resistance and tolerance to skin-penetrating nematodes during the earliest stages of infection, such as skin penetration. However, once the parasites also have access to the supplemented resources when they begin feeding within the host, resource supplementation increases parasite establishment in the gut more than it increases host resistance [48]. Thus, resource supply can have differential effects on host immunity and parasite growth throughout the infection sequence. More generally, this study demonstrates that parasite trophic strategy (consumption of host tissues versus consumed food within host guts) and timing could explain variation in resource-dependent infection dynamics.

Trophic strategy also explains variation in resource-dependent infection dynamics for fungal parasites of plants. In many plant–fungi systems, nitrogen fertilization increases disease severity, although some plant and fungal taxa are strong exceptions to this broad pattern [24]. On average, biotrophic fungal parasites, which acquire resources from living tissue, cause increasing disease with nitrogen fertilization. In contrast, nitrogen fertilization causes weak/no increase in disease caused by necrotrophic fungal parasites, which kill tissue and then assimilate resources [24]. Biotrophic fungi may benefit from increased availability of nitrogen within living host tissue, while it may become more difficult for necrotrophs to kill nutrient-rich host tissue [49].

Host taxa and parasite trophic strategy can explain why some infections worsen with resource supplementation and others do not. Variation in resource-dependent infection dynamics should be important because the direction of this effect could either enhance or oppose the effects of increased host aggregation or population size on infectious contact rates. For example, enrichment with limiting nutrients simultaneously increases the reproduction and infectiousness of individual hosts in several aquatic systems, and these joint increases drive larger epidemics in host populations [15,50]. When supplementation causes opposing effects on contact rates and parasite production, epidemiological models can help resolve the net effects [6,51]. Recent meta-analyses of fertilization and plant disease suggest that potentially opposing effects of fertilization on contact rates, parasite production and disease severity may limit the utility of nutrient manipulation as a disease control strategy at large scales [24].

3. Indirect effects and interactions: the importance of community context

(a) Evidence for indirect effects of supplementation

Recent reviews, meta-analyses and theoretical models of food supplementation on wildlife disease have largely focused on the direct epidemiological effects of provisioning, outlined above [6,7]. However, natural host–parasite systems do not exist in isolation. Instead, they are embedded in food webs; hosts compete with other species for prey resources and are consumed by predators. Therefore, supplementation-mediated shifts in the traits and densities of other community members can drive disease dynamics. These community interactions could enhance or overturn the direct epidemiological effects on hosts and parasites. For example, nutrient enrichment increases the elemental quality (a trait) of plant resources for herbivore hosts, which can increase or decrease the virulence and infectiousness of their parasites [52]. Similarly, increased phosphorus additions to marshes from sugarcane agriculture in central America have led to shifts in primary producer density from microphytic- (cyanobacteria mats) to macrophytic-dominated (predominantly cattail) plant communities, indirectly shifting dominance of the mosquito community to a species that more efficiently vectors malaria [53]. Increased resources might also affect shifts in host communities in 'pace of life' and promote faster-living organisms that are more susceptible or infectious because they rapidly exploit resources and invest relatively less in defence mechanisms than slower-paced species [54–56].

(b) A case study and a general epidemiological model for community-mediated indirect effects of supplementation on disease

Here, we review insights from an aquatic invertebrate–fungal disease system and a general theoretical model to highlight how interactions with competitors and predators can alter patterns predicted from the direct epidemiological effects of resource supplementation. Ultimately, a deeper understanding of species interactions should improve predictions of the epidemiological consequences of food supplementation for more charismatic wildlife.

The *Daphnia dentifera–Metschnikowia bicuspidata* disease system illustrates the intersection between resource-dependent epidemiological and food web context. *Daphnia dentifera* is a filter-feeding freshwater zooplankton that consumes suspended algae and particulate material in small, thermally stratified temperate lakes. While foraging on algae, *Daphnia* inadvertently consume free-living spores of the fungus *M. bicuspidata* that are suspended in the water column. Infection is invariably fatal, and upon host death, thousands of spores are released from the decomposing host's corpse into the water column where they may be consumed by new hosts. In the Midwestern USA, epidemics occur annually each autumn [57]. *Daphnia* do not receive direct food subsidies

from humans. However, they exist in lakes that vary in nutrient and algal concentrations and may be impacted by local and large-scale eutrophication processes, such as agricultural runoff and nutrient deposition [58].

Resource availability powerfully shapes disease severity and parasite transmission in this *Daphnia*–fungus system. The rate that *Daphnia* filter water (which determines their rate of exposure to fungal spores) depends on their functional response to food availability. When resource density is high, *Daphnia* filter smaller volumes of water per unit time because they spend more time handling food; therefore, their *per capita* contact rates with fungal spores decline with resource density [59]. Once infected, however, increased resource consumption substantially increases the production of fungal spores (i.e. infectiousness) and virulence of infection [58,60]. Field surveys of annual fungal epidemics have demonstrated that epidemics are larger in lakes with greater resource availability (quantified as algae or nutrients), likely due to increased infectiousness [61]. These larger epidemics cause rapid evolutionary increases of host resistance to infection [61–63]. This relationship between resource quantity and disease echoes broader patterns that causally link high food and water quality to larger fungal epidemics through the same increases in infectiousness [51,64,65].

This resource-fuelled disease system exists within a broader ecological food web (figure 2*a*). Predators and competitors can directly affect *Daphnia*–fungus epidemiology by consuming hosts or parasites [66–72]. However, predators and competitors could also shape fungal epidemics indirectly through their interaction with food resources. Zooplanktonic competitors of *Daphnia* consume algae, indirectly reducing resource availability for *Daphnia*. When predators consume *Daphnia* or other invertebrate filter feeders, they reduce herbivory rates on algae and increase *per capita* resource availability for the invertebrate filter feeders that remain. Here, we assess whether interactions with predators and competitors can shape epidemics across gradients of resource enrichment using a general theoretical model inspired by this *Daphnia*–fungus–food web disease system [73,74]:

$$\frac{dS}{dt} = e_S f_S(S+I)R - d_S S - u f_S Z S - f_P P S, \tag{3.1}$$

$$\frac{dI}{dt} = u f_S S Z - (d_S + v) I - f_P P I, \tag{3.2}$$

$$\frac{dZ}{dt} = \sigma\left(\frac{R}{R+R_h}\right)(d_S+v)I - mZ - [f_S(S+I) + f_C C]Z, \tag{3.3}$$

$$\frac{dR}{dt} = r\left(1-\frac{R}{K}\right)R - (f_S(S+I) + f_C C)R, \tag{3.4}$$

$$\frac{dC}{dt} = e_C f_C R C - d_C C - f_P P C \tag{3.5}$$

and

$$\frac{dP}{dt} = f_P(S+I+C)P - d_P P. \tag{3.6}$$

The model tracks the densities of susceptible hosts (S), infected hosts (I), parasites (Z), resources (R), competitors of the host (C) and predators of hosts and competitors (P) through time. Resources grow logistically with a maximal rate r and carrying capacity K, and are consumed by hosts and competitors. Susceptible hosts increase at a rate determined by conversion efficiency (e_S, births per unit of resource) and foraging rate (f_S) on resources, while they die at a background death rate (d_S). Hosts become infected following a transmission rate defined by f_S and their per-parasite susceptibility to infection (u), and they are consumed following the predator's feeding rate on hosts and competitors (f_P). Infected hosts increase from transmission, but die at an elevated rate due to virulence (v) and consumption by predators. Free-living parasites increase from resource-dependent release from dead infected hosts (with maximum yield σ) and they are lost through consumption by hosts and competitors and background mortality, m. Resistant competitors increase at a rate determined by their conversion efficiency (e_S) and foraging rate (f_C), and they also decrease from background mortality and predation. Finally, predators increase from consumption of hosts and competitors and die with a background death rate (d_P).

We used this model to determine equilibria of disease prevalence as a function of resource provisioning using the numerical integration function lsoda in the R package deSolve ([75]; code provided in electronic supplementary material). We examined four variants of this model that always contained the resource and disease system (S, I, Z and R state variables): (i) only resources, hosts and parasites ($SIZR$), (ii) $SIZR$ with competitors ($SIZR + C$), (iii) $SIZR$ with predators ($SIZR + P$) and (iv) $SIZR$ with competitors and predators ($SIZR + C + P$). We simulated the eco-epidemiological dynamics of each model across a gradient of nutrient enrichment, which we represented as increases in the population growth rate of algal resources, r. We then plotted equilibrium infection prevalence against resource productivity for each model (figure 2*b*).

Equilibrium infection prevalence can depend strongly on food web structure and eutrophication. When considering only resources, hosts and parasites, there is a minimum productivity to enable parasite invasion, and increases in r monotonically increase equilibrium prevalence (figure 2*b*1). However, the presence of resistant competitors (figure 2*b*2) or predators (figure 2*b*3) can reverse this effect and cause equilibrium prevalence to decrease with increasing eutrophication. Interestingly, multimodal dynamics can occur when there are both competitors and predators present (figure 2*b*4). This general model illustrates that the consequences of resource supplementation for disease can depend on the interplay between hosts, parasites and other species in the food web. Incorporating competition, predation or other community interactions, and other indirect epidemiological consequences of resource supplementation (e.g. toxic foods or hypoxia; [7,23]) could improve models tailored to specific resource management scenarios.

4. Moving forward

(a) Theory

The variety of direct and indirect effects of resource provisioning on wildlife infectious disease and their potential dependence on time-lags and spatial scale highlight the need for quantitative tools for prediction and management. Theoretical studies should generate hypotheses for the relative importance of resource-driven changes in aggregation, demographic changes and parasite production/infectiousness, especially under realistic supplementation scenarios, such as when resources are haphazardly or predictably pulsed, or when they are aggregated at multiple spatial scales. Theory for resource-driven epidemiology should incorporate approaches that can more effectively address the ecological effects of spatial and temporal factors. For example, delay- and integro-differential equations

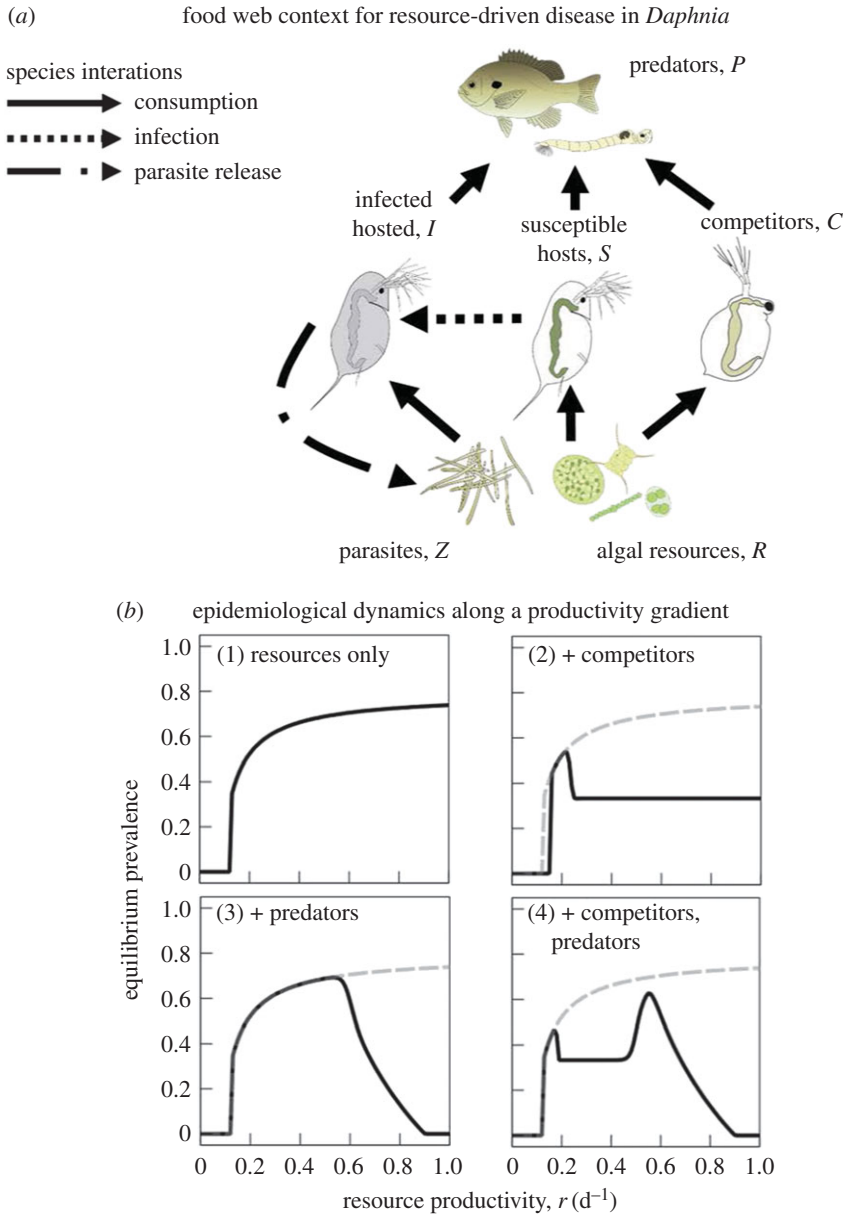

Figure 2. A case study of resource-fuelled epidemics in a community context. (*a*) *Daphnia* (susceptible, S and infected, *I*) filter water to consume resources, *R*, and inadvertently consume fungal spores, *Z*, suspended in the water. Infected hosts can release over 100 000 spores upon death. Competing invertebrates, *C*, remove parasites and algae, but strongly resist infection. Predators, *P*, consume hosts and competitors. Resources, predation and competition shape epidemic size in field studies, experiments and models. (*b*) A general model incorporating these interactions illustrates the intersection between resources, competition and predation for disease. (1) Increased resource input (represented as algal productivity) stimulates epidemics in the absence of predators and competitors by increasing parasite production within hosts (replicated with dashed grey lines in 2–4). (2) Competitors can reverse this effect by consuming parasites and resources. (3) Predators can also overturn the initial increases in parasite spread by consuming hosts. (4) Jointly, competition and predation can create complex multimodal relationships between productivity and disease. In this case, competition initially reduces disease in focal hosts. As productivity increases, predation initially stimulates disease by reducing competitor density, which indirectly increases resource availability. Ultimately, as in (3), predators reach high densities at high productivity and can eliminate disease from focal hosts. Parameter values: r = varied, $K = 250$, $e_S = 0.5$, $f_S = 0.01$, $d_S = d_C = 0.05$, $u = 10^{-4}$, $v = 0.05$, $\sigma = 10^5$, $R_h = 20$, $m = 0.2$, $e_C = 0.5$, $f_C = 0.0075$, $f_P = 0.0002$, $d_P = 0.01$. (Online version in colour.)

allow for traits to depend on fixed time-lags or the recent history of resource consumption, respectively [76,77]. These approaches could reveal the importance of the timing of behavioural, physiological and demographic changes for realistic, temporally variable scenarios of supplementation. Alternatively, individual based models (IBMs) could address issues of spatial scale by incorporating movement patterns, species interactions or contact/social behaviours onto realistic landscapes. For example, an IBM built from bioenergetic theory accurately captures the dynamics of experimental *Daphnia* populations responding to pulsed resources [78], and this model could be extended to incorporate parasitism [60]. Regardless of modelling framework, the endpoints examined should depend on the management goals. For example, if the focal host is the intermediate host or vector for a human parasite, then the density of infected hosts might be a much better index of human risk of exposure than infection prevalence. Alternatively, if species conservation is the primary concern, then host survival or population size might be the most important endpoints. In conservation contexts, it may be especially important to resolve the effects of resource supplementation on tolerance and resistance to infection [48], because highly tolerant hosts may exhibit high levels of infection with minimal harm to individuals and populations.

(b) Experiments

Experiments are needed to determine if different types of anthropogenic resource supplementation have predictable effects on parasite transmission due to their clustering, duration and magnitude. For instance, because of the likely greater clustering of directly supplemented food resources in terrestrial and certain near-shore habitats (e.g. ports; [10,32]), resource supplementation in these systems might disproportionately increase host aggregation and contact rates, and thus parasite transmission more so, on average, than nutrient enrichment in aquatic systems or fertilization in more uniform agricultural settings. However, these hypotheses assume that hosts can track heterogeneous resource supplements, which can only occur if the hosts are mobile. Thus, this hypothesized difference in the effects of resource supplements on parasite transmission across habitat types should depend on host mobility relative to the spatial scale of supplementation. Given that plants are generally less mobile than animals, one might also expect that the effects of resource supplementation on parasite transmission will be a product of an interaction between habitat (e.g. aquatic versus terrestrial) and host type (plant versus animal).

These hypotheses assume that contact rates are a major driver of parasite transmission. However, we emphasized in this review that many factors other than contact rates could facilitate transmission, such as host densities, host immunity and natural enemies of hosts or parasites. The relative importance of these various mechanisms when resources are supplemented also remains an open question. We suspect that contact rates might be important because of how quickly they can respond to resource supplementation if hosts are mobile and thus, they could rapidly affect transmission. By contrast, physiological changes will have more delayed effects and demographic changes will be even more delayed. We hypothesize that variation in the spatial orientation of provisioning sites and the duration of supplementation should modulate the relative importance of aggregation versus physiological and demographic mechanisms for disease dynamics. This highlights the need to consider temporal lags and spatial scales to adequately detect the influences of resources on disease dynamics in experiments and field surveys. These hypotheses, however, will also clearly depend on the type of parasite transmission. Parasites with complex life cycles might not respond as strongly or quickly to heterogeneous supplementation as those with direct life cycles because they will be less likely influenced by behavioural aggregation that can inflate contact rates. Likewise, parasites with frequency-dependent transmission might not respond to heterogeneous resources as strongly as those with density-dependent transmission because host aggregation around resources should be less likely to increase rates of, e.g. sexual contacts. Studies that integrate animal movement with physiological and demographic responses in provisioned landscapes are needed to assess the relative importance of these mechanisms in promoting transmission across spatial and temporal scales.

Another key challenge moving forward involves identifying the traits of hosts, parasites or interacting species that best predict epidemiological responses to supplementation [79]. Meta-analyses suggest that parasite trophic strategy or the taxonomic status of hosts or parasites influences whether supplementation increases or decreases disease severity and the production of parasites [16,24]. However, direct experimental tests of these hypotheses remain rare. Physiological studies that manipulate host and parasite access to resources independently or sequentially, track nutrients that might limit immune defences or parasite growth, and measure the activation and efficacy of host immune defences are needed to identify the functional traits of hosts, parasites or resources that modulate resource–infection relationships (e.g. [48,80]). Such studies would be most informative in systems with multiple host or parasite species (e.g. [81]). Similarly, community ecology studies could evaluate multiple predator and competitor species to identify the traits that predict whether they will promote or inhibit disease in focal hosts along resource or productivity gradients [43,82].

5. Conclusion

Human supplementation of resources for wildlife is extremely pervasive. Traditional approaches to resource supplementation–disease interactions have been compartmentalized, focusing on particular types of hosts, parasites, habitats, scales and endpoints. However, a more unified view of resource supplementation effects on infectious diseases could allow disease biologists to identify common mechanisms, processes and spatio-temporal scales driving epidemiological outcomes across these diverse host–parasite systems and supplementation scenarios. We recommend the development and experimental testing of theory that explicitly incorporates our growing mechanistic understanding of the direct epidemiological effects of supplementation, the potential for indirect effects with coexisting species, such as predators and competitors, and the spatio-temporal scale of ecologically realistic supplementation scenarios. While ecologically relevant differences may exist among some types of habitats and resource supplementation regimes, the relative importance of putatively unique and common consequences of resource supplementation remain unknown. An integrative assessment of the direct and indirect effects of resource supplementation on wildlife disease could ultimately yield new broadly applicable or precisely targeted disease management and conservation strategies.

Data accessibility. This article has no additional data.

Competing interests. We declare we have no competing interests.

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