1 The impact of resource provisioning on the epidemiological responses of different 2 parasites

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8 9 Abstract

- 10 1. Events such as anthropogenic activities and periodic tree masting can alter resource
11 provisioning in the environment, directly affecting animals, and potentially impacting the
12 spread of infectious diseases in wildlife. The effect of these supplemental resources on
13 infectious diseases can manifest through different pathways, affecting host susceptibility,
14 transmission and host demography.
- 15 2. To date however, empirical research has tended to examine these different pathways in
16 isolation, for example by quantifying the effects of provisioning on host behaviour in the wild
17 or changes in immune responses in controlled laboratory studies. Further, while theory has
18 investigated the interactions between these pathways, thus far this work has focussed on a
19 narrow subset of pathogen types, typically directly-transmitted microparasites. Given the
20 diverse ways that provisioning can affect host susceptibility, contact patterns or host
21 demography, we may expect the epidemiological consequences of provisioning to depend on
22 key aspects of parasite life-history, such as the duration of infection and transmission mode.
- 23 3. We developed a suite of generic epidemiological models to compare how resource
24 provisioning alters responses for different parasites that vary in their biology (micro- and
25 macro-parasite), transmission mode (direct, environmental, and vector transmitted) and
26 duration of infection (acute, latent, and chronic). Next, we parameterised these different

27 parasite types using data from the diverse parasite community of wild wood mice as a case
28 study.

29 4. We show there are common epidemiological responses to host resource provisioning across
30 all parasite types examined. In particular, the response to provisioning could be driven in
31 opposite directions, depending on which host pathways (contact rate, susceptibility or host
32 demography) are most altered by the addition of resources to the environment. Broadly, these
33 responses were qualitatively consistent across all parasite types, emphasising the importance
34 of identifying general trade-offs between provisioning-altered parameters.

35 5. Despite the qualitative consistency in responses to provisioning across parasite types, we
36 found notable quantitative differences between parasites, suggesting specific epidemiological
37 outcomes could strongly depend on parasite type, infection duration and permanency of
38 recovery, and whether the parasite is directly, environmentally, or vector transmitted. These
39 analyses therefore highlight the importance of knowing key specific aspects of host-parasite
40 biology, such as host contact behaviours, parasite interactions with the host immune system,
41 and how resource availability shapes host demographics, in order to understand and predict
42 epidemiological responses to provisioning for any specific host-parasite system.

43
44 **Keywords:** host–parasite interactions, supplemental feeding, disease ecology, compartmental models,
45 *Apodemus sylvaticus*

47 **Introduction**

48 The availability of food resources is a key environmental factor that impacts the demography and
49 behaviour of animals, but also has the ability to impact host-parasite interactions (Bradley & Altizer,
50 2007; Robb, McDonald, Chamberlain, & Bearhop, 2008). Natural events, such as periodic tree
51 masting, and human activities such as agricultural expansion or the purposeful feeding of wildlife, can
52 alter host-parasite interactions through several distinct pathways, acting across multiple ecological
53 scales (Jensen, 1982; Oro, Genovart, Tavecchia, Fowler, & Martínez-Abraín, 2013). At the individual
54 scale, food supplementation (or ‘provisioning’) can improve host body condition and immune

55 responses, which can result in lower susceptibility to infection, shorter-lasting infections, and better
 56 health outcomes for the host (Becker, Streicker, & Altizer, 2015; Cypher & Frost, 1999). For
 57 example, a study on lace monitors foraging on urban waste suggested that body condition improved as
 58 a result of provisioning, and this improvement was associated with a lower intensity of blood-borne
 59 parasites compared to unprovisioned individuals (Jessop, Smissen, Scheelings, & Dempster, 2012). At
 60 the between-host scale, hosts can aggregate around resources, thereby increasing contact rates and
 61 transmission (Boutin, 1990), as seen by the aggregation of European greenfinches around
 62 supplemental bird feeders, which increased the transmission of *Trichomonas gallinae* (Lawson et al.,
 63 2012). Finally, at the host population scale, parasite prevalence and transmission potential (as
 64 measured by the basic reproductive number; R_0) can increase through host demographic responses to
 65 provisioning, through the addition of new susceptible individuals to the population via new births or
 66 immigration, which can potentially increase the likelihood of parasite persistence (Boutin, 1990;
 67 Krebs et al., 1995; Nagy & Holmes, 2005). Therefore, resource provisioning can affect parasite
 68 dynamics through separate pathways involving changes in host susceptibility, behaviour and/or
 69 demography. Together, these changes can lead to either net increases or decreases in infection risk,
 70 and impact the epidemiological outcomes in different directions, depending on the balance of how
 71 each pathway is impacted by provisioning.

72
 73 Given the many ways in which provisioning can affect infectious disease epidemiology, mechanistic
 74 models can be invaluable tools to explore how various provisioning-altered parameters relating to
 75 host demography, contact behaviour, and host immune defence interact to affect the dynamics of
 76 parasite infection and spread. In particular, previous theoretical studies demonstrated that a range of
 77 parasite responses (i.e. changes in prevalence and basic reproductive number, R_0) could arise from
 78 coupling functional responses of host birth and death, host susceptibility and contact rate parameters
 79 to the level of resource provisioning (Becker & Hall, 2014). For instance, increases in both host
 80 density and contact rates due to resource provisioning boost R_0 (McCallum, Barlow, & Hone, 2001),
 81 when host immunity is not directly affected. However, if a host with access to improved nutritional
 82 resources was better able to protect itself from infection due a strengthened immune response, then

83 the parasite R_0 is significantly reduced. Additionally, parasite extinction could even occur at
 84 intermediate provisioning levels, with invasion (R_0) and persistence only occurring at low or high
 85 provisioning levels (Becker et al., 2015). Thus, by showing that parasite persistence changes along a
 86 gradient of resource provisioning, depending on the underlying effects of resources on the host.
 87 Therefore, modelling approaches are valuable tools for understanding the opposing patterns in disease
 88 outcomes observed in nature.

89

90 To date, however, theory has focussed on a narrow subset of parasite types, typically directly-
 91 transmitted microparasites that fit the classic ‘Susceptible-Infected’ (SI) or ‘Susceptible-Infected-
 92 Recovered’ (SIR) frameworks. Given the diverse ways that provisioning can affect proximate factors
 93 relating host physiology/immunology, contact patterns or host demography, we may expect the
 94 ultimate epidemiological consequences of provisioning to depend on key aspects of parasite life-
 95 history, such as the duration of infection, parasite type and transmission mode. For instance,
 96 environmentally transmitted or vector-borne parasites could exhibit different responses to
 97 provisioning-induced changes in host population size and contact structure, compared to directly-
 98 transmitted parasites. Furthermore, how macroparasites, rather than microparasites, are impacted by
 99 provisioning of the host remains poorly understood, as impacts on burden-dependent processes
 100 (Anderson & May, 1978; Dobson & Hudson, 1992) or altered environments for parasite reproduction
 101 and survival (Seppälä, Liljeroos, Karvonen, & Jokela, 2008) may introduce additional responses not
 102 seen for microparasites. To understand how different parasite types may vary in their overall
 103 responses to host resource provisioning, we constructed a series of five epidemiological models,
 104 which represent a range of parasites that vary in their biology (micro- and macroparasite),
 105 transmission mode (direct, environmental, and vector transmitted) and duration of infection (acute,
 106 latent, and chronic). Next, we parameterised these different parasite types using data from the diverse
 107 parasite community of wild wood mice (*Apodemus sylvaticus*) as a case study. We demonstrate that
 108 there are some generalities in how different parasites respond to resource provisioning of the host that
 109 are consistent across parasite types and transmission modes. However, we also found notable
 110 differences in the magnitude of these responses by the different parasites. These analyses therefore

highlight the importance of identifying specific aspects of host and parasite biology, to properly infer the effects of provisioning for any specific host-parasite system.

Methods

Model construction

We constructed five compartmental models, each describing a different, but common parasite transmission structure: (i) macroparasite, (ii) microparasite SIR (susceptible-infected-recovered), (iii) environmentally transmitted microparasite SIS (susceptible-infected), (iv) microparasite SAL (susceptible-active-latent), and (v) vector-borne microparasite SIS (Figure 1). The specific details of each model are given in the Supplementary Material, but they all followed standard structures well-known from the epidemiological literature (e.g., (Anderson & May, 1978, 1981, 1991) Importantly, all five models contained parameters that incorporated the effects of increased resource provisioning of the host on the three key, separate pathways: (1) host demography (through long-term changes to the host's carrying capacity, K), (2) contact behaviour (α), and (3) immune defence (through changes to host susceptibility, δ). In particular we assumed the host's carrying capacity (K) and contact rate (α) would increase with resource provision to the host, while host susceptibility (δ) would decrease as hosts had more access to supplemental nutrition (as in Becker & Hall (2014) and Becker, Streicker & Altizer (2015)).

Figure 1. Schematic diagram of the macroparasite and microparasite models. Boxes represent populations in each system. For microparasite models, hosts were classified by their infection status, therefore S , I , R , A and L represented susceptible, infected, recovered, acute and latent populations, respectively. For the vector-borne parasite, the subscripts V and H denote classes relating to the vector and host populations respectively. For the macroparasite model, only one host population (H) was considered, P represents the size of the adult parasite population and L represents the size of the environmental pool of infective parasite larvae. Arrows represent populations transitions and dashed arrows represent the mechanism through susceptible population gets infected.

To capture the effects of resource provisioning on both host contact rate and susceptibility, we followed the approach developed by Becker & Hall (2014), where provisioning is represented by the parameter ρ . Here $\rho = 0$ represents no supplemental resource provisioning (hence, all parameters are at baseline values), while increasing values of ρ correspond to increasing levels of resource provisioning, for example due to human-provided food sources, land-use change increasing access to novel resources, or periodic access to resources, such as through tree masting. As in Becker & Hall (2014), parameter responses to provisioning were assumed to have a monotonic and saturating

behaviour that depends on the parameter's sensitivity to provisioning, represented by θ_x , allowing parameters to scale from linear forms ($\theta_x = 0$) to a quickly saturating shape (high values of θ_x). Since the contact rate between hosts (α) is expected to increase with provisioning (Becker et al., 2015; Boutin, 1990), we used the following functional form:

$$\alpha = \alpha_{max} - (\alpha_{max} - \alpha_{min})e^{-\theta_c \rho} \quad \text{Eq 1}$$

Because susceptibility to infection (δ) is hypothesised to decrease with provisioning (Becker et al., 2015; Sweeny et al., 2019) we used the following function:

$$\delta = \delta_{min} + (\delta_{max} - \delta_{min})e^{-\theta_s \rho} \quad \text{Eq 2}$$

where α_{min} and δ_{min} are the minimum values, and α_{max} and δ_{max} are the maximum values that contact rate and susceptibility could take. Thus, if $\rho = 0$, $\delta = \delta_{max}$ and $\alpha = \alpha_{min}$, the parameter baseline values. In an intensive resource provisioned scenario (high ρ) the maximum contact rate was assumed to be twice the baseline value ($\alpha_{max} = 2\alpha_{min/bl}$) and the minimum susceptibility was assumed to be half the baseline value ($\delta_{min} = \frac{\delta_{max/bl}}{2}$).

We investigated the net effect of host resource provisioning for each of the above parasite types as the difference in either mean parasite burdens for macroparasites or parasite prevalence for microparasites at equilibrium, between arbitrarily intensive provisioning ($\rho = 1$) and no provisioning ($\rho = 0$) scenarios. Here a value greater than 0 for these differences implies a net increase in overall prevalence or mean parasite burden due to provisioning, whereas a value less than 0 implies a net reduction due to provisioning. We explored how these changes for each parasite depended on the balance of the host defence and host contact sensitivities to resource provisioning (i.e., within $\theta_s - \theta_c$ parameter space). To simplify the analysis and presentation, we incorporated the effects of provisioning on host demography by varying host carrying capacity from K to $3K$, to reflect long-term increases in overall food availability due to increased provisioning (Becker et al., 2015; Ozoga & Verme, 1982). Hence, this analysis allowed us to explore how the overall changes due to provisioning vary with the sensitivity of the individual host demographic, contact rate or susceptibility parameters to provisioning.

Model parameterization

While our intention is to explore generic responses of each parasite type to provisioning, we fit each model to field data, to ensure we were using biologically plausible values. Specifically we parameterised each model using a specific parasite species from our four-year longitudinal dataset of 1453 individually-tagged wood mice (*Apodemus sylvaticus*) captured across four 70x70m grids, at Haddon Wood on the Wirral, UK between June 2009 and December 2012 (Knowles, Fenton, & Pedersen, 2012; Sweeny, Albery, Venkatesan, Fenton, & Pedersen, 2021). Wood mice in these populations are commonly infected by five different species of parasites, each of which corresponded to the different modelling frameworks described above (see Table 1 for details): *Heligmosomoides polygyrus* (macroparasite), *Eimeria hungaryensis* (SIS), Wood Mouse Herpes Virus (WMHV - SAL), *Bartonella* spp. (vector-borne), *Trypanosoma grosi* (vector-borne), and Cowpox virus (SIR). Full details of model parametrisation are provided in the Supplementary Material but, briefly, parameters were estimated using adaptive Monte Carlo Markov Chain Metropolis-Hastings (MCMC-MH) using the R package fitR (Camacho & Funk, 2019), assuming uniform priors. Model fitting was carried out in two stages. First, host demographic parameters were estimated by fitting the predicted total number of mice per week to the observed number of mice captured per week. Next, we estimated parasite infection-related parameters for the various parasite transmission models (see Table 1), through fitting each model individually. For the microparasite models, we used data on seroprevalence for Wood Mouse Herpes Virus (Knowles et al., 2012) and PCR diagnostics for *Trypanosoma grosi* and *Bartonella* spp. (Knowles et al., 2013; Withenshaw, Devevey, Pedersen, & Fenton, 2016), while we used faecal egg or oocyst counts (measured as eggs or oocysts per gram of faeces) for the gastrointestinal nematode *H. polygyrus* and the coccidian *E. hungaryensis*, respectively (Knowles et al., 2013). Because the seroprevalence of Cowpox was very low in the wood mouse population (<5%), the infection-related parameters were estimated using data from previous literature (Telfer et al., 2002); see Supplementary Material.

Estimated parameter values in Table 1 were assumed to be the baseline values ($\delta = \delta_{max}$ and $\alpha = \alpha_{min}$) corresponding to the scenario of no supplemental resource provisioning ($\rho = 0$). Under the scenario of arbitrarily intensive resource provisioning ($\rho = 1$), parameter values for δ and α were calculated using Equations 1 and 2 (Becker & Hall, 2014).

Table 1. Parasites from the *A. sylvaticus* system: model, type, transmission mode, diagnostic, and parameter values. $K = 42.12^*$ in all parasite models. *Parameter baseline values. See Supplementary Material for details of parameter estimation.

Parasite (Model)	Type	Transmission mode	Diagnostic	α_{min} $\rho = 0^*$	α_{max} $\rho = 1$	δ_{min} $\rho = 1$	δ_{max} $\rho = 0^*$
Cowpox virus (SIR)	Virus	Contact	Seroprevalence	0.1	0.2	0.053	0.105
<i>Eimeria</i> <i>hungaryensis</i> (SIS)	Coccidian	Environmental	Faecal eggs count	0.00001	0.0002	0.048	0.096
Herpesvirus (SAL)	Virus	Contact	Seroprevalence	0.1	0.2	Male to male: 0.128 Female to male: 0.212 Male to female: 0.074 Female to female: 0.078	Male to male: 0.255 Female to male: 0.424 Male to female: 0.148 Female to female: 0.156
<i>Bartonella</i> spp. (vector-borne)	Bacteria	Vector	PCR	0.01	0.02	0.408	0.815
<i>Trypanosoma</i> <i>grosi</i> (vector-borne)	Protozoan	Vector	PCR	0.01	0.02	0.053	0.106
<i>Heligmosomoides</i> <i>polygyrus</i> (macroparasite)	Nematode	Environmental	Faecal eggs count	0.001	0.002	0.103	0.205

211

212 **Results**

213 For all 5 parasite species, we found the same qualitative responses of parasite infection (either
 214 prevalence or mean burden) to host resource provisioning; all parasite infection measures increased
 215 when contact rate was highly sensitive to provisioning ($\theta_c \rightarrow 5$) and susceptibility sensitivity to
 216 provisioning was minimal ($\theta_s = 0$) (green regions, Figure 2). Hence, as may be expected, rapid
 217 increases in host contact rate (and hence transmission) due to provisioning (high θ_c), coupled with a
 218 negligible response of host immunity to provisioning (low θ_s) result in high levels of infection for all
 219 parasite types. Conversely, prevalence and infection burden for all parasites was the most reduced in
 220 the opposite scenario, when host susceptibility was highly sensitive to provisioning and the impact of
 221 provisioning on contact rate was minimal ($\theta_c = 0$, $\theta_s \rightarrow 5$; red regions, Figure 2). Hence, rapid
 222 increases in immunity due to provisioning (high θ_s), coupled with negligible changes to contact rate
 223 (low θ_c), result in low levels of infection, and this was true across all parasite types.

Figure 2. Resource-provision impacts parasite infection and burdens. The A row shows macroparasite mean burden change and B row to F row represent microparasites' prevalence changes (the difference in equilibrium prevalence between provisioned ($\rho = 1$) and un-provisioned scenarios ($\rho = 0$)). Columns illustrate different carrying capacities, varying from K to $3K$, representing increasing levels of host demographic response to provisioning. For all figures, X-axis represents the sensitivity of contact rate to provisioning (θ_c) and y-axis is the sensitivity of host susceptibility to provisioning (θ_s).

However, although all parasites showed the same qualitative responses as θ_s and θ_c varied, the different parasite types differed considerably in their quantitative responses to provisioning (i.e., in the magnitude of changes in prevalence or mean infection burden, as indicated by the intensities of colours in Figure S1, which presents the same results as Figure 2, but re-expressed so the changes in prevalences are plotted on the same scale for all microparasites). For clarity, we initially describe these results assuming no host demographic change to provisioning (carrying capacity fixed at the baseline value of K ; Fig S1 left-hand column), but below we consider the effects of incorporating longer-term host demographic changes due to resource provisioning. When the carrying capacity is fixed at K , the directly-transmitted SIR microparasite showed the strongest responses overall to provisioning, with ~20% increase in prevalence due to provisioning for high θ_c (rapid increase in contact rate due to provisioning) and low θ_s (negligible change in immunity due to provisioning), and ~70% decrease in prevalence due to provisioning for low θ_c and high θ_s (Figure S1, row B). The SAL and SIS microparasites also showed a reasonably strong (~10%) increase in prevalence due to provisioning for high θ_c and low θ_s , but very little change in prevalence for all other combinations of parameters (Figure S1, rows C, D). Comparatively however, the two vector-borne parasites presented very minor quantitative variations in prevalence due to provisioning across all combinations of θ_c and θ_s (Figure S1, rows E, F).

When we included the effect of longer-term host demographic responses to provisioning, modelled as fixed changes in host carrying capacity, the varying parasite types responded differently. As carrying

capacity was increased from K to $3K$, changes in macroparasite mean burden became more extreme (Figure 2, row A); with both increases in burden under high contact rate sensitivity ($\theta_c \rightarrow 5$), and decreases in burden under high susceptibility sensitivity ($\theta_s \rightarrow 5$), becoming intensified with higher host carrying capacities. Conversely, the opposite pattern occurred for the microparasite SIR model, where the change in prevalence became less dramatic as carrying capacity increased (e.g., whereas high θ_c was associated with >20% increase in prevalence when the carrying capacity was kept at K (Figure 2 row B, left-hand plot), increasing the carrying capacity to $3K$ resulted in <10% increase in prevalence (Figure 2 row B, right-hand plot)). The SAL and SIS models however showed mixed responses to rises in carrying capacity; the magnitude of positive changes in prevalence due to provisioning (i.e., in the region of high θ_c and low θ_s) were neutralized as carrying capacity increased for both parasites (Figure 2 rows C and D), whereas the region of negative changes in prevalence due to provisioning (i.e., in the region of low θ_c and high θ_s) initially intensified as carrying capacity was increased from K to $2K$, but then diminished as it was increased further to $3K$.

Finally, the two vector-borne parasites showed different predicted responses to changes in host demography; *Bartonella* spp. (relatively high baseline prevalence, reflecting high estimated baseline host susceptibility; see Supplementary Material) was predicted to show broadly the same pattern regardless of carrying capacity variations, with both positive and negative changes in prevalence being approximately equal as carrying capacity changed (Figure 2, row E). On the other hand, changes in host prevalence for *T. grosi* (low baseline prevalence, with low estimated baseline host susceptibility) became intensified as carrying capacity increased, similarly to that seen for changes in macroparasite mean burden (Figure 2, row F).

Discussion

Overall, our analyses revealed common epidemiological responses of a wide range of different parasite types to resource provisioning of their host. In particular, for all parasites, we demonstrated that responses to provisioning could be driven in opposite directions dependent on which host

pathways are most altered by provisioning (Becker & Hall, 2014): increases in contact rate and population size due to provisioning could boost parasite prevalence, whereas decreases in host susceptibility might decrease it. However, we also found some notable differences, particularly in the magnitude of responses by the different parasites, suggesting specific epidemiological outcomes could strongly depend on parasite type (micro- or macroparasite), the duration of infection (whether SIS, SIR or SAL), and whether the parasite is directly, environmentally, or vector-borne transmitted (Altizer et al., 2018). These analyses therefore highlight the importance of understanding the key aspects of host-parasite biology, to properly infer effects of resource provisioning on host-parasite interactions.

We found that different parasite types, such as the macroparasite *H. polygyrus* and vector-borne parasites, which strongly depend on host demographic factors such as changes in host population size, were likely to show extreme changes as carrying capacity increases due to provisioning. Our analyses demonstrated that the macroparasite, and the vector-borne parasite with low baseline prevalence, showed the greatest changes in mean worm burden and prevalence, respectively, to host demographic responses to provisioning. In general, for directly transmitted parasites, however, increases in host carrying capacity due to provisioning translated into limited increases, and sometimes even net decreases, in infection prevalence. These predictions are supported by observations from larger vampire bat (*Desmodus rotundus*) colonies in livestock areas, where provisioning was found to impact host population size, but no effect of rabies virus exposure was observed (Streicker et al., 2012). However, our analysis also showed that directly transmitted parasites that do not confer lasting immunity (i.e., the SIS model) presented the lowest parasite prevalence at intermediate population sizes, particularly if host susceptibility rapidly declines with provisioning (high θ_s). This finding supports previous research suggesting that at an intermediate demographic response to provisioning, parasite persistence (as measured by its R_0) attains its minimum, although susceptibility would have to be more affected by provisioning compared contact rate (Becker et al., 2015).

Prevalence was predicted to increase for both directly and environmentally-transmitted microparasites, when the response of host aggregation behaviour to provisioning is stronger compared to host immune defence and demographic responses. An example of this scenario has been reported in European greenfinches (*Chloris chloris*), in which aggregation around bird feeders increases transmission of the parasite *Trichomonas gallinae* (Lawson et al., 2012). Similarly in Elk (*Cervus elaphus*) populations, supplemental feed stations were shown to increase contact rates and lead to higher *Brucella abortis* exposure (Cross, Edwards, Scurlock, Maichak, & Rogerson, 2007). In contrast, host susceptibility can also be more affected by improved nutrition compared to changes in contact rate. One study on lace monitors (*Varanus varius*) foraging on human garbage, found that body condition improvement was associated with lower intensity of blood parasites compared to non-provisioned individuals (Jessop et al., 2012). Another example of this scenario occurs in wood mice infected with *H. polygyrus*, in which resource provisioned mice were less susceptible to infection, cleared worms more efficiently, and maintained better body condition (Sweeny et al., 2019). These examples suggest that increased host aggregation, and therefore increased transmission, is not always an outcome of provisioning, or at least not sufficient to overcome potential beneficial impacts of provisioning on the host's physiology. Indeed, changes in host contact rates due to resource provisioning are likely to depend explicitly on the spatial arrangement of resources, thus, when food is aggregated, contact rate between hosts could increase, as observed in urban feral cats showing smaller foraging ranges and higher territory overlap compared to rural cats (Schmidt, Lopez, & Collier, 2007). However, if food is more evenly scattered across the landscape, contact rate could actually decrease with provisioning. Spatially explicit models could be useful for studying how resource provisioning modifies contact rates and therefore parasite persistence (Plowright et al., 2011).

In this study, we assumed that any impact of resource provisioning on host body condition would be positive, and therefore decrease host susceptibility to parasite infection. However, this may not always be the case, and it is important to consider both resource quantity and quality; it is quite possible that although resource quantity might increase, for example through increased availability of human

garbage, quality could be poorer compared to a baseline scenario, thereby increasing host susceptibility to infection (Maggini, Wintergerst, Beveridge, & Hornig, 2007; Van Heugten, Coffey, & Spears, 1996). For instance, rock iguanas feeding from carbohydrate-rich food sources provided by tourists, were associated with changes in nutritional status and higher hookworm burdens compared to non-supplemented individuals (Knapp et al., 2013). Furthermore, although we considered a range of parasite types, we did not consider either multi-host systems or parasites with complex life cycles involving intermediate hosts. Such systems would require consideration of how those alternative host species themselves respond to resource provisioning, potentially resulting in more complex net responses of such parasites to provisioning. Future theoretical studies on the role of provisioning on parasite transmission more generally could therefore involve even more complex systems than those considered here.

In conclusion, we demonstrate that there are general responses to provisioning that are consistent across a wide range of parasite types and transmission modes; thus, our work contributes to the understanding and comparison of increased provisioning impacts on different parasites in the same host species to allow comparison. In particular, we demonstrated that considering general trade-offs of provisioning-altered parameters is crucial for determining parasite persistence. However understanding the key parasite characteristics and host pathways driving these patterns is important, as resource provision can impact infectious disease dynamics in opposite directions. For instance, population increases in vector-borne systems could lead to increments in parasite prevalence. Hence it is crucial to recognise system-specific variations, such as specific host behaviours at the individual level, parasite interactions with the host immune system, and how natural resource availability shapes host demographics, in order to fully understand and predict epidemiological responses to provisioning.

Data Availability Statement

Data available from <https://github.com/dieraz/prov-theo.git>

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