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Research Article

How does a bacterial infection affect an individual's foraging and risk management over time?

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Abstract The effects of host-parasite interactions can be measured in ecologically relevant ways through behavioural indicators based on foraging theory. These include, assessing how parasites alter foraging aptitudes, harvest rates, and risk management of foragers. Consequences of infection can be measured from individuals to ecosystems. At the individual level, sick animals need to trade-off finding food with remaining safe from predators, while potentially facing debilitating effects from infection. This potentially results in reduced foraging efficiencies, compromised risk awareness, and increased apprehension. To investigate this, we assessed how a *Mycoplasma* bacterial infection impacted individual Allenby's gerbils' foraging aptitudes, resource harvest rates, and anti-predator responses. We monitored individuals through three stages, from uninfected, to acutely (newly) and finally chronically (long term) infected. We identified three distinct responses. While acutely infected, some individuals increased their foraging effort in patches and spent less time vigilant. This may reflect increased future value of food for these individuals. Some individuals immediately reduced their foraging effort and displayed increased apprehension while acutely infected. This likely reflects a lethargy, where sick individuals are compromised in their ability to harvest seeds efficiently while also remaining vigilant. As all individuals became progressively sicker with a chronic infection, their foraging declined and apprehension levels increased. Two individuals employed a 'grab and go' foraging strategy to minimize time spent in patches. Foraging costs of long-term infection increase dramatically over time. These findings point to some behavioural plasticity in response to initial infection, yet the consequences of long-term infection are similar for all individuals.

Keywords apprehension; foraging; hosts; individuals; parasites; risk management

Introduction

Studies of how animals make foraging decisions have focused on a number of factors, including, habitat and patch suitability (Brown, 1988; Gorini *et al.,* 2012; Morris, 1992), the distribution of resources (Anderson *et al.,* 2004; Ben-Natan *et al.,* 2004), competition between hetero- and conspecifics (Berger-Tal *et al.,* 2015; Sinclair, 1985; Ziv *et al.,* 1993), and the presence and lethality of predators (Lima, 2002; Sih *et al.,* 1998). These factors significantly contribute to how animals utilize the landscape. Habitat selection and patch choice within habitats reflects a continuous trade-off between finding food and avoiding predators. As food resources and predation risk are often asymmetrically distributed across landscapes, and fluctuate with habitat structure, resource renewal, and the movement and density of predators, animals must continuously adjust their use of space in response to changing opportunities and costs of foraging (Brown and Kotler, 2004; Laundré *et al.,* 2001). Thus, animals typically forage across a landscape of fear, with resource poor areas of relative safety and areas of higher predation risk where food may be abundant (Laundré *et al.,* 2010). These factors all contribute to decision making regarding where and when to forage and how to manage risk within the environment (Brown, 1988; Padié *et al.,* 2015).

Another important factor that contributes to how animals forage and manage risk involves host-microbe interactions (Poulin, 1994). Complex microbial communities inhabit most multi-cellular organisms (Hungate, 2013; Moore, 2002). These commensal microbes can affect community dynamics and species interactions (McFall-Ngai *et al.,* 2013). Beyond the population effects, these microbial communities often have direct and indirect effects on an individual host's cytology, physiology, and behaviour (Moore, 2002). While some interactions are beneficial to the host, as in the case of symbiotic ruminant gut microfauna (Hungate, 2013), others are harmful, as in the case of pathogens and parasites (Gunderson, 2008; Norris *et al.,* 2013). These harmful host-microbe interactions often manifest as reduced body condition in the host through

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nutrient and amino acid deficiencies, and the alteration of the hosts' metabolism and digestive efficiencies (Moore, 2002; Thomas *et al.,* 2005). Additionally, parasites may alter the behaviour of the host, leading to greater risks of predation through suppression of predator avoidance strategies and manipulations of host habitat choice that can act to increase the transmission rates of the microbe to other host species (Moore, 2002).

For example, Lafferty and Morris (1996) observed that killifish (*Fundulus parvipinnis*) infected with trematodes employed behaviours, such as surfacing, flashing, and shimmying more frequently than uninfected individuals, which increased their conspicuousness and likelihood of predation by egrets and herons (the parasites' ultimate hosts). In another example, house sparrows (*Passer domesticus*) infected with malaria parasites had reduced intensities of escape behaviour (biting, struggling) when caught by a hawk compared to uninfected sparrows, leading to increased transmission rates of the parasite within the avian community (Garcia-Longoria *et al.,* 2015).

Most studies investigating the negative effects of these host-parasite interactions have focused primarily on the physiological costs of infection (i.e. metabolism, deficiencies, immunity; Hempel, 2011). Other studies investigating the abilities of parasites to alter host behaviour, quantified the behavioural costs in terms of population fitness, with emphasis on fecundity, population growth rates, and long-term survival of populations (Finnerty *et al.,* 2018; Møller *et al.,* 1993). Often the effects of these host-parasite interactions are complex, varied, and very subtle, with many host-parasite interactions carrying low detectable physiological costs for the individual and minor fitness costs for the population (Hahn *et al.,* 2018; Jolles *et al.,* 2005). Yet, the impacts of these negative interactions may be better reflected in the larger ecological context, by quantifying the costs and benefits of infections to individuals in ecologically relevant ways using behavioural indicators based on foraging theory. These include quantifying the effects of host-parasite interactions through measuring foraging aptitudes (Brown, 1999), resource harvest rates (Kotler *et al.,* 2010), resource handling times and encounter rates (Kotler and Brown 1990), and risk management behaviour of individuals (Brown, 1988; Lima and Dill, 1990).

Several studies of host-parasite relationships have also highlighted that within populations, behavioural responses to parasite infections can reflect high degrees of individual variation (Dall *et al.,* 2004; Poulin, 1994, 2013; Seaman and Briffa, 2015). These individual responses to parasitic infections can reflect a combination of differences in personality (Dall *et al.,* 2004; Poulin, 2013), individual physiology (Isaksson *et al.,* 2013; Johnson and Hoverman, 2014), and genetic variation among individuals within a population (O'Brien and Evermann, 1988; Schmid-Hempel, 2003). For example, a study by Karvonen *et al.* (2004) observed that individual rainbow trout (*Oncorhynchus mykiss*) had considerably different avoidance behaviour response times to the presence of parasitic trematodes, which resulted in fish having different parasite loads and strongly affected the distribution of parasites within the population. House sparrows infected with the parasite *Haemoproteus* had weaker immune responses if their individual diurnal weight fluctuated more than individuals with smaller daily fluctuations in body mass (Navarro *et al.,* 2003). Hammond-Tooke *et al.* (2012) recorded high levels of individual variability in the use of three behaviours (activity, aggression, and boldness) by a fresh water fish species while infected with trematodes, and highlighted that these individual-level responses could be due to a fine level adaptive host manipulation strategy by the parasites to improve their transmission rates within the population. These studies emphasize that beyond the population level impacts of parasitic infections, individual behavioural responses can also vary. How this variability in behavioural response to infection might affect an individual's ability to forage efficiently and manage predation risk is largely unknown, and gives rise to a number of questions. Namely, how does infection influence the foraging aptitudes, harvest rates, and predator avoidance strategies of individual foragers? Do all individual foragers respond in a similar manner to infection? If not, how do their foraging and risk management responses vary, and how does this change with time since infection?

To address the above questions, we set up an experiment to investigate how infection with an endoparasitc bacteria influenced the foraging behaviour and risk management of individual foragers. More specifically, we ask to what degree does infection with the bacteria (*Mycoplasma haemomuris*-like bacterium; Kedem *et al.,* 2014) within the host Allenby's gerbils (*Gerbillus andersoni allenbyi*) influence the foraging aptitudes, resource harvest rates, and anti-predator responses of individual foragers. Previous studies investigating this microbe-host interaction have found that these bacteria cause very little physiological harm to the gerbils (Cohen *et al.,* 2018; Eidelman *et al.,* 2019). However, infection with the bacteria carries larger ecological costs at the population level, with decreased foraging aptitudes, lower harvest rates, reduced predator awareness, and high mortality rates (Makin *et al.,* 2020), all of which become more severe with time since infection. Here we present two alternative hypotheses: (1) all individual foragers will have a similar behavioural response to infection with the bacteria, resulting in a progressive decline in foraging efficiency (i.e. lower foraging effort), reduced harvest rates, and predator awareness with time since infection, or (2) there may be high levels of individual variation in response to infection that becomes masked at the population level, and this will be reflected in how each individual alters their foraging behaviour and risk management in response to the infection over time. Here, we focus on individuals, and follow particular gerbils through three different stages of infection, beginning as uninfected, becoming acutely (newly) infected, and finally progressing to being chronically (long-term) infected.

Materials and methods

This experiment was carried out over three months from December 2018 to February 2019 in a large, outdoor vivarium on the Sede Boker Campus of Ben-Gurion University of the Negev in Midreshet Ben Gurion, Israel (30.857274°N, 34.780942°E). This vivarium is fully enclosed with chicken-wire (15 × 37 × 4.5 m) and divided into four equally sized quadrants. Gerbils were released into the

vivarium two days prior to the start of the experiment. This allowed them time to acclimatize to their new surroundings. We managed to collect sufficient data from eight non-reproductive adult female Allenby's gerbils over the entire time course of infection to determine individual responses to the bacterial infection. This encompassed time periods when the gerbils were uninfected, when they carried an acute infection, and when they carried a chronic infection. These 8 individuals were a subset of a larger population study (Makin *et al.,* 2020), where we monitored the responses of 24 gerbils to different states of infection. Sample sizes for the number of individuals used was limited due to high recorded mortality rates of infected individuals due to owl predation (Makin *et al.,* 2020). Therefore, we could only obtain sufficient foraging and risk management data for 8 individuals who transitioned through all three states of infection (uninfected, acute and chronic).

At the start of the experiment animals were confirmed by polymerase chain reaction (PCR) to be free of *Mycoplasma* sp. and *Bartonella* (Cohen *et al.,* 2018). The gerbils were kept isolated and cleaned for ectoparasites at least one month prior to the start of the experiment. All individuals were bled, and their samples were PCR tested to confirm their infection status (Cohen *et al.,* 2018). For the first round of the experiment these 8 individuals served as part of the control group, and were inoculated with negative-*Mycoplasma* sp. blood. In the second round, these individuals were injected with gerbil blood containing the *Mycoplasma* sp. 15 days prior to the start of the experiment, and they served as part of the acute infection treatment. Acute infection (peak infection intensity 10^6 copies/µl) occurs starting 15 days after initial infection and lasts 20 days (Cohen *et al.,* 2018). In the final round of the experiment, these same individuals were confirmed as chronically infected (low infection intensity 10^3 copies/µl) occurring > 65 days post infection. Subsequently, the *Mycoplasma* sp. infection continues at low infection intensities for the gerbil's entire life following the peak infection period (Cohen *et al.,* 2018; Eidelman *et al.,* 2019). For detailed laboratory protocols, see Cohen *et al.* (2018).

In the vivarium, we used artificial resource patches to provide seed resources for gerbils and quantify their foraging by measuring giving-up-densities (GUDs). GUDs provide a measure of the amount of food remaining in a patch once a forager has abandoned it, and allows us to measure the animal's foraging costs and benefits (Brown, 1988, 1992). Higher GUDs imply higher foraging costs (i.e. predation risk, metabolic, and missed opportunities).

We used plastic trays (30 × 30 × 10 cm) to provide 32 artificial resource patches spaced evenly across the vivarium. Each set of food patches were separated by 3.5 m in every quadrant. To replicate habitat features within the gerbils' landscape, we covered four artificial patches in each quadrant with a black shade-cloth covered low-lying wooden trellis (76 × 60 × 16 cm) topped with cut brush to simulate a bush. The other four trays per quadrant were placed in the open. The patches were set up in a grid with alternating open and bush microhabitats.

Each gerbil used in the experiment was injected with a uniquely numbered Passive Induction Transponder (PIT) tag for electronic identification, and each tray contained an integrated PIT tag antenna coupled with a reader and logger on which the time of each entry and exit and the identity of the forager within each patch was recorded. The PIT tag readers allowed us to calculate the cumulative duration of time (s) gerbils spent in foraging patches (Embar *et al.,* 2014).

For each replicate, GUD collection involved filling each tray with 3 g of millet seeds mixed into 3 l of sifted sand each night (18:00). The following morning after a night in which the gerbils could forage from the trays (07:00), we collected the remaining seeds from each tray and replenished seeds and sand to their original levels. Collected seeds were dried, cleaned of sand and debris, and weighed using an electronic balance (precision 0.005 g) to obtain the GUD for each tray per night. This process was repeated for 6 days. Following that, we captured all gerbils using Sherman traps for two nights. Gerbils were then rotated among the quadrants of the vivarium following a Latin square design to control for any idiosyncrasies associated with quadrant position. GUD data collection then continued for another 6 days.

To provide predation risk, a barn owl (*Tyto alba*) was allowed access to the vivarium for 3 nights of each 6-night sampling period on alternating nights. To minimize the chances of direct gerbil mortality, but still present a risk of predation, the owl was fed prior to being released each night (Embar *et al.,* 2014). All gerbils were captured, bled, and PCR tested after each experimental replication to ensure their infection status remained consistent throughout each trial period.

Statistical analyses

To compare the GUDs (g) and cumulative time (s) spent in resource patches by the 8 individual gerbils as they transitioned through infection states (uninfected, acute, and chronic), in response to the presence and absence of a predator (owl), and between safe and risky microhabitats, we ran generalized linear mixed effects models (GLMMs). Within each model, GUDs (g), and cumulative time (s) spent in patches served as the dependent variables, while infection status (uninfected, acute and chronic), predators (absence vs presence), and microhabitats (open and bush patches) served as explanatory variables, including interactions between variables. Quadrant and patch location nested within quadrant were included as random factors in the models to control for any idiosyncrasies associated with any one particular quadrant. For each GLMM, we ran Tukey HSD *post-hoc* tests to compare differences between treatment levels. All analyses were performed using R 3.25 (R Core Team 2014) using the lme4 package (Bates *et al.,* 2012), MASS package (Venables and Ripley 2002), and the multcomp package (Hothorn *et al.,* 2008).

Results

Giving-up-densities

The changes in GUDs reflected the foraging costs of infection and the shift in individual state as ecological costs changed with time since infection (uninfected, acute, and

Table 1. Generalized linear mixed effects models (GLMMs) comparing the mean GUDs (g), and cumulative duration of time spent in patches (s) by individuals (gerbil ID) with different infection levels (acute, chronic and uninfected) comparing the presence and absence of a predator (owls and no owls), and across bush and open microhabitats, including interactions between variables.[1]

Response	Variable	df	X^2	*P*-value	Significance
Mean giving-up-densities (g)	infection (acute vs chronic vs uninfected)	2	25.59	<0.001	***
	predators (owls)	1	0.09	0.767	
	microhabitat (bush vs open)	1	0.04	0.851	
	individuals (gerbil id)	7	15.77	0.027	*
	infection × predators	2	5.17	0.076	
	infection × microhabitat	2	3.18	0.203	
	infection × individuals	14	38.74	<0.001	***
	infection × predators × individuals	8	20.72	0.008	**
	infection × microhabitat × individuals	4	8.49	0.075	
Cumulative duration of time spent in patches (s)	infection (acute vs chronic vs uninfected)	2	13.86	<0.001	***
	predators (owls)	1	0.02	0.883	
	microhabitat (bush vs open)	1	7.86	0.005	**
	individuals (gerbil id)	7	17.39	0.015	*
	infection × predators	2	0.95	0.622	
	infection × microhabitat	2	7.76	0.021	*
	infection × individuals	14	28.36	0.012	*
	infection × predators × individuals	8	10.15	0.255	
	infection × microhabitat × individuals	4	16.96	0.002	**

1 Quadrant and patch position nested within quadrant were included as random factors in the models.

chronic). Gerbil giving-up-densities were significantly affected by their infection status (Table 1). Overall, when individuals were uninfected, they achieved the lowest average GUDs (highest foraging effort) in patches (1.95 ± 0.1 g). During acute infection with the *Mycoplasma* sp. bacteria, their average GUDs in patches increased by 5.4% to 2.11 ± 0.1g ($Z_{100,2}$ = -0.90, P = 0.638). As individuals transitioned from acutely to chronically infected over time, their average GUDs increased even further from 2.11 ± 0.1 to 2.76 ± 0.1g (21.4% increase; $Z_{100,2}$ = 3.54, P = 0.001). Thus, infection with the bacteria resulted in an average decline in foraging effort of 26.8% in patches over time ($Z_{81,2}$ = –4.09, P < 0.001). There was no significant effect of predator presence or differences in microhabitat features on the individual gerbils' GUDs (Table 1).

Although on average, GUDs increased from uninfected to acute to chronic, during the course of the infection, individual responses were more complex. For example, Individual (Ga-3888) GUDs were significantly lower (increased foraging effort by 34%) during the period of acute infection than when uninfected. Gerbils, Ga-9485 (8% lower GUDs) and Ga-9768 (10% lower GUDs; Figure 1), also increased their foraging effort while acutely infected, however, this was not statistically significant (Figure 1; Table 2).

In contrast, Ga-3855 (35% increase in GUDs), Ga-3863 (52% increase in GUDs), and Ga-9482 (3% increase in GUDs) significantly decreased their foraging efforts while acutely infected compared to when uninfected (Figure 1; Table 2). Additionally, both Ga-9435 (6%

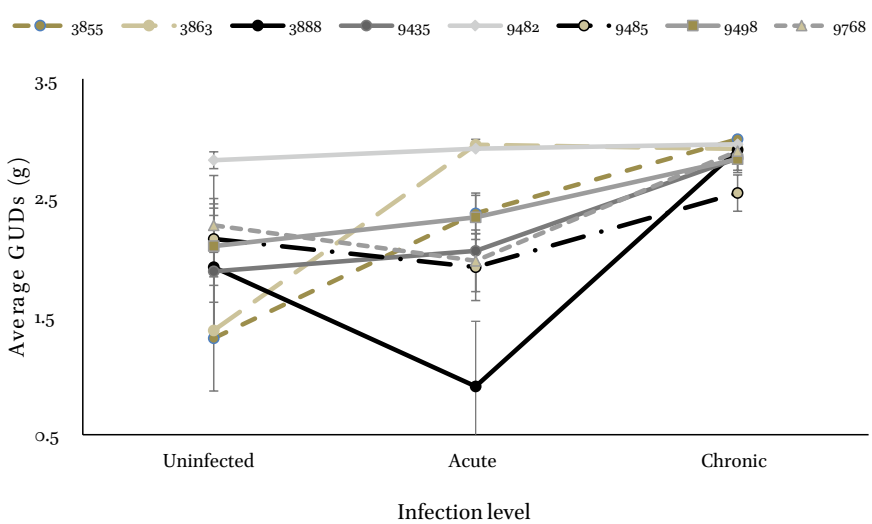

Figure 1. Mean gerbil giving-up-densities GUDs (g) in foraging patches comparing when the same individuals were uninfected, acutely infected (0-25 days after infection, peak intensity) and lastly chronically infected (> 65 days after initial infection) with the *Mycoplasma* sp. bacteria. Bars represent standard error.

Table 2. Gerbil giving-up-densities (GUDs) in response to different levels of *Mycoplasma* infection comparing across treatment (uninfected vs acute vs chronic) levels obtained from the Tukey HSD *post-hoc* tests.

Level of *Mycoplasma* infection	df	Z	*P*-value	Significance
Individuals – uninfected to acute				
Ga-3888	2	3.63	<0.001	***
Ga-9485	2	0.42	0.905	
Ga-9768	2	0.67	0.779	
Ga-3855	2	–4.12	<0.001	***
Ga-3863	2	–49.09	<0.001	***
Ga-9482	2	–2.61	0.023	*
Ga-9435	2	–0.85	0.656	
Ga-9498	2	–0.98	0.588	
Individuals – acute to chronic				
Ga-3888	2	–3.84	<0.001	***
Ga-9485	2	–1.93	0.125	
Ga-9768	2	–2.56	0.027	*
Ga-3855	2	–3.88	<0.001	***
Ga-3863	2	–0.57	0.830	
Ga-9482	2	–0.95	0.592	
Ga-9435	2	–3.32	0.002	***
Ga-9498	2	–3.42	0.003	***
Individuals – uninfected to chronic				
Ga-3888	2	–8.81	<0.001	***
Ga-9485	2	–1.02	0.557	
Ga-9768	2	–1.51	0.282	
Ga-3855	2	–4.93	<0.001	***
Ga-3863	2	–35.28	<0.001	***
Ga-9482	2	–0.68	0.766	
Ga-9435	2	–3.32	0.002	***
Ga-9498	2	–3.18	0.007	***

increase in GUDs) and, Ga-9498 (8% increase in GUDs) decreased their foraging efforts when acutely infected, however, this was not statistically significant (Figure 1; Table 2).

As time since infection increased and individuals became chronically infected, their foraging aptitudes declined within patches. Specifically, both Ga-3888 (66% decrease in foraging effort; Figure 1), and Ga-9768 (31% decrease in foraging effort) after foraging more intensively while acutely infected, significantly increased their GUDs while chronically infected (Table 2). This was also observed as a trend for Ga-9485 (21% increase in GUDs), but was not statistically significantly different. The foraging efforts of Ga-3855 (21% increase in GUDs), Ga-9435 (26% increase in GUDs; Figure 1), and Ga-9498 (16% increase in GUDs), continued to decline as they reached a state of chronic infection. There was no statistically significant change in GUDs for Ga-3863 (1% increase in GUDs), and Ga-9482 (1% increase in GUDs; Figure 1) as they shifted from acutely to chronically infected (Table 2).

Overall, in the end, gerbils chronically infected all had decreased foraging effort in patches as costs increased. There was a significant decline in foraging effort (i.e. higher GUDs) of 56% for Ga-3855, 51% for Ga-3863, 33% for Ga-3888, 32% for Ga- 9435, and 24% for Ga-9498 (Table 2). While, GUDs increased for Ga-9482 (4% increase; Figure 1), Ga-9485 (13% increase) and Ga-9768 (21% increase) as they transitioned from uninfected to chronically infected, it was not statistically significantly different.

Time spent in patches

The cumulative amount of time gerbils spent in patches differed significantly with their infection status (Table 1). On average, uninfected (41 ± 7 min) gerbils spent 17% more time in patches than chronically infected individuals (34 ± 4 min; $Z_{88,2} = 3.65$; $P = 0.009$). At the same time, there was no significant difference in time spent in patches when individuals were uninfected or acutely infected (5% more time spent by uninfected; 39 ± 6 min; $Z_{88,2} = -0.96$; $P = 0.586$). While acutely infected, individuals spent 13% more time in patches than when they were chronically sick ($Z_{102,2} = 2.36$; $P = 0.018$). Unexpectedly, predator presence had no effect on the cumulative time individuals spent in patches (Table 1). However, there were microhabitat differences, with individuals spending more time on average per patch in bush microhabitats (40 ± 7 min) than open microhabitats (29 ± 6 min; Table 1). Within infection treatments, uninfected gerbils spent significantly more time in bush microhabitats (48 ± 8 min) than open microhabitats (17 ± 6 min; $Z_{37,2} = 2.12$; $P = 0.034$). Surprisingly, when these gerbils became chronically infected, they shifted their patch use and spent more time in riskier open patches (40 ± 7 min) than in safer bush patches (30 ± 5 min; $Z_{51,2} = 2.33$; $P = 0.018$). No significant difference in time spent in bush and open patches was observed when gerbils were acutely infected ($Z_{51,2} = -0.46$; $P = 0.644$).

Figure 2 illustrates the effect of the *Mycoplasma* sp. bacteria in determining individual foraging behaviour and risk management by plotting the quitting harvest rate (QHR) against the GUDs for each individual's state of infection (uninfected, acute, chronic). Curves with shallower slopes reflect greater vigilance use, and average GUDs closer to the origin reflect greater time allocation (Brown, 1999; Kotler *et al.,* 2010). Not all individuals responded similarly. We can recognize three distinct manners in which gerbils altered risk management according to their infection status. Following acute infection with the bacteria, Ga-3888, Ga-9485, and Ga-9768 decreased their use of vigilance (i.e. lower apprehension, steeper harvest rate curves) in patches and allocated more time to foraging, harvesting at a faster rate, thus, achieving lower GUDs compared to when they were uninfected. Notably, Ga-3888, spent very little time vigilant (steep curve) in patches and maintained a high quitting harvest rate at a lower GUD than when uninfected. With the onset of a chronic infection, all three gerbils significantly reduced their time allocation to foraging (higher GUDs) and became more apprehensive, thus, allocating more time to vigilance. This was an extreme shift for Ga-3888 and Ga-9768, who both effectively ceased harvesting seeds from patches and spent most of their time in patches vigilant.

By comparison, Ga-9435, Ga-9482 and Ga-9498, decreased their time allocation to foraging (higher GUDs) and increased their use of vigilance in patches (shallower curves) with the progression of the infection with a rank

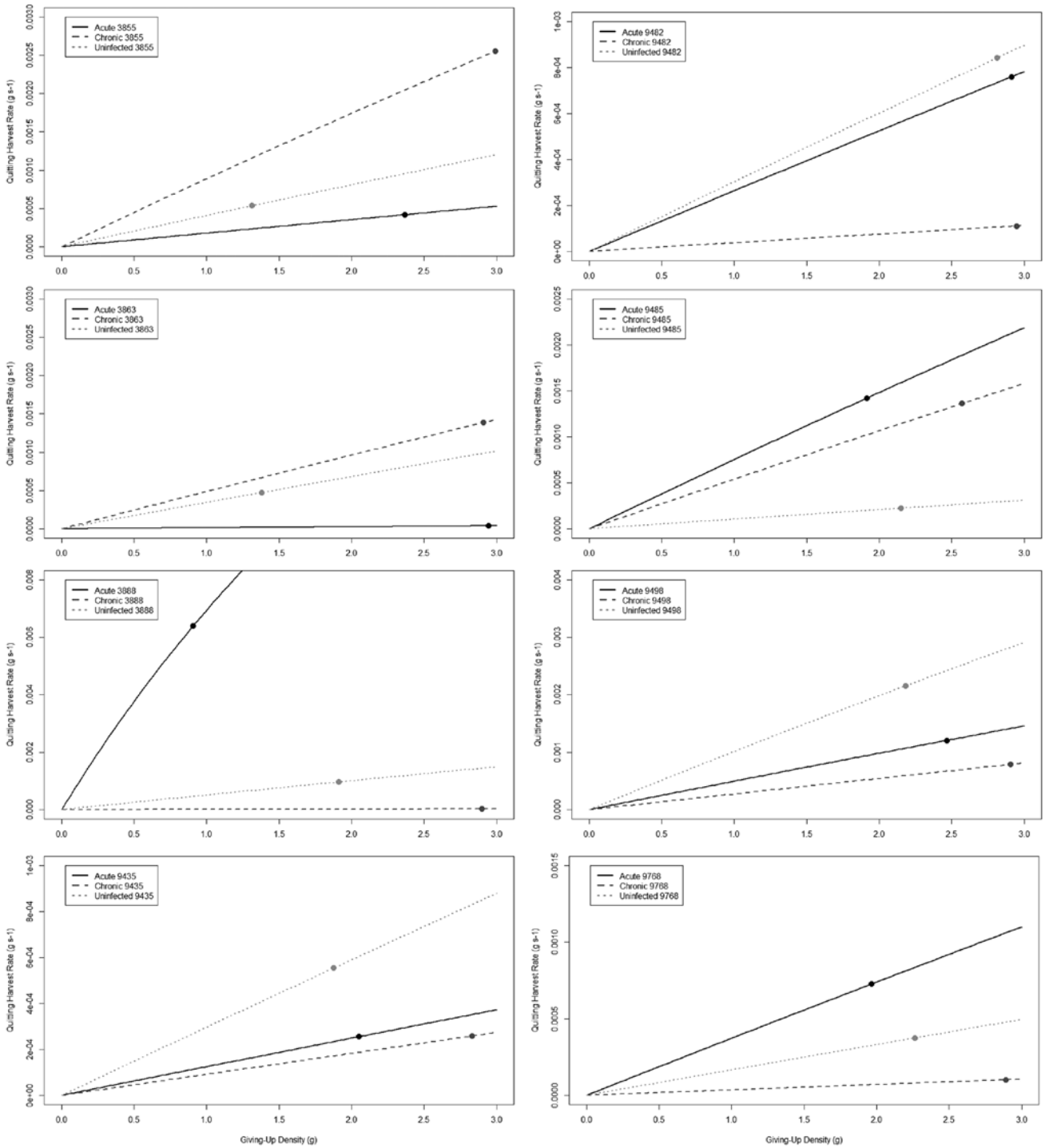

Figure 2. Sets of quitting harvest rate curves for each of the gerbils as they shifted from uninfected to acutely infected, and finally chronically infected. Curves of different slopes indicate differences in apprehension, with slower harvest rate curves (shallow slopes reflective of slower harvest rates for any given seed density) indicating a more vigilant forager. If foragers have similarly sloped harvest rate curves, but GUDs vary, this indicates differing use of time allocation for risk management, with lower GUDs reflecting greater time allocation to feeding (Kotler *et al.,* 2010).

ordering of Uninfected > Acute > Chronic. While uninfected, they were less apprehensive and allocated more time to feeding (lower GUDs) at a trade-off of lower vigilance. However, as they became progressively sicker their time allocation to feeding decreased (higher GUDs), and they maintained higher levels of vigilance in patches. This suggests that foraging costs increased dramatically with time since infection for these individuals.

Lastly, Ga-3855 and Ga-3863, were less apprehensive while chronically infected than when either were uninfected or acutely infected. However, this did not translate to increased time allocation to feeding. Rather, these individuals used time allocation to manage risk with higher GUDs and QHRs in riskier patches. Therefore, they seem to have adopted a 'grab and go' foraging strategy (St Juliana *et al.,* 2017), skimming seeds off the top of the patches

for a high quitting harvest rate at a high GUD. In contrast, when these individuals were uninfected, they traded off time allocation to foraging with remaining vigilant more effectively, achieving lower GUDs while also remaining alert for potential predators. While acutely infected, these individuals displayed their highest levels of vigilance and allocated less time to feeding, achieving much slower QHRs.

Discussion

Host-parasite interactions are often complex and subtle, with a range of potentially harmful effects for the host, including, changes in physiological state (through metabolic and energy deficiencies), reduced fitness (lower fecundity, survival rates), and manipulation of host behaviour (Eidelman *et al.,* 2019; Hudson *et al.,* 1992; Moore, 2002). In particular, changes in host behaviours that increase potential parasite transmission rates or in response to the onset of serious illness can carry significant ecological costs. Like injury, infection can limit an animal's ability to forage, reducing its aptitudes for harvesting resources, increasing search and handling times, limiting predator awareness, and suppressing predator avoidance strategies. At the population level (Makin *et al.,* 2020), this was observed for infected gerbils, with decreased foraging efficiencies, reduced predator awareness, and increased mortality rates with time since infection, following the rank order, uninfected > acute > chronic. The debilitating effects of infection followed a clear pattern with acutely infected (short term) gerbils displaying greater foraging aptitudes and predator awareness than chronically infected (long term) gerbils. However, within infection states and at the individual level and in line with our second hypothesis, we observed that the gerbils' foraging behaviour and risk management reflected high levels of individual variation in their shifts from uninfected to infected. The high degree of variation in individual responses was masked at the population level (Makin *et al.,* 2020) but became evident at the individual level.

We observed three distinct responses by individuals in response to initial infection. While acutely infected with the *Mycoplasma* sp., three individuals responded by increasing their time allocation to foraging, achieving lower GUDs, and displaying diminished levels of apprehension compared to when they were uninfected. This may be due to a couple of factors. Firstly, these individuals' perception of their environment may have become impaired, such that infection with the bacteria limited their ability to differentiate between periods of risk and relatively safety, extending to their use of microhabitats and the presence and absence of predators. This led them to miss-assess risk and so achieve lower GUDs despite foraging during riskier periods, when they faced an increased risk of direct mortality from the owl. Nonetheless, despite a miss-assessment of increased local risk, and subsequent increased foraging efforts, they managed to avoid being killed. In contrast, while uninfected these same individuals would have perceived differences in risk between microhabitats and responded to predator presence by abandoning patches earlier at higher GUDs.

Secondly, increased foraging effort from patches by these individuals potentially has to do with the future value of food. Allenby's gerbils are known to maintain seed caches in their burrows (Kotler, 1997). Therefore, if the future value of food increases for sick animals, infected gerbils may increase their time allocation to harvesting seeds early in their illness to counter potential subsequent lean periods when chronic infection later reduces their foraging aptitudes (Kotler *et al.,* 1999; Van der Merwe *et al.,* 2014). However, why this increased foraging activity while acutely infected was not observed for the other five individuals is unknown. Alternatively, although the *Mycoplasma* sp. bacteria carries relatively low physiological costs for Allenby's gerbils, it can still potentially cause low levels of energy deficiencies (Cohen *et al.,* 2018; Messick, 2004). Therefore, for these individuals, increased foraging efforts while acutely infected may have compensated for this loss of energy.

In contrast, the five other individuals responded to being acutely infected by immediately decreasing their time allocation to foraging (i.e. higher GUDs) and displayed increased levels of apprehension (i.e. vigilance) in a manner similar to chronically infected gerbils. This suggests that for these individuals the onset of acute infection carried immediate increased foraging costs, with sick individuals compromised in their ability to forage efficiently whilst managing predation risk. Potential explanations for this observation are that sick gerbils are more lethargic, and thus, have reduced harvest rates while foraging and are more susceptible to predation. They therefore increased their investment in vigilance behaviour at the expense of finding food (Makin *et al.,* 2020). Increased predation rates on individuals in poor health has been well documented (Isomursu *et al.,* 2008; Møller and Nielsen, 2007; Tizard, 2008). For example, a study in Quebec, Canada, found that moose (*Alces alces*) infected with tapeworms had higher rates of predation by wolves (*Canis lupus*) than individuals without the parasites (Joly and Messier, 2004). A multisite analysis investigating rates of predation in 150 bird species infested with brood parasites determined that species with higher prevalence rates of parasites had higher risks of predation by hawks, and that protozoan infections were a common cause of predator-mediated death (Møller and Nielsen, 2007). Furthermore, Makin *et al.* (2020) observed similar patterns in gerbils, with chronically ill animals having the highest rates of predator-induced mortality, followed by those who were acutely infected, while those individuals who were uninfected had by far the lowest overall mortality rates.

With the shift of infection state from acute to chronic, gerbils faced increased foraging costs likely due to progressive debilitation. This caused the three gerbils that had lower GUDs, and reduced apprehension while foraging when initially sick (acute infection) to subsequently decrease their time allocation to foraging and increase their levels of vigilance. This shift was particularly extreme for one individual who effectively ceased foraging from

patches with the onset of chronic infection. With the transition in individual state from acute to chronic infection, three of the individuals continued to exhibit a progressive decline in foraging efficiency and further increased their use of vigilance to manage risk. Thus, for these individuals, a continued decline in health dramatically increased their foraging costs. Lastly, two of the individuals switched their foraging and risk management strategies by significantly reducing their use of vigilance (no apprehension) while foraging. However, they did not increase their time allocation to feeding. Rather, these individuals manifest high quitting harvest rates at high GUDs. This suggests that they adopted a 'grab and go' strategy (St Juliana *et al.,* 2017), skimming seeds off the top of patches to reduce the amount of time spent foraging, and thus reduce their potential encounter rates with owls. This 'grab and go' strategy was observed by St Juliana *et al.* (2017), who determined that under periods of greater risk, Allenby's gerbils favour this harvesting strategy, collecting seeds quickly, filling their mouths, and abandoning patches to carry seeds to feed in safety in nearby burrows or to cache seeds for future use. This strategy limits their exposure to predators provided individuals limit the number of trips made between burrows and resource patches (St Juliana *et al.,* 2017).

Despite gerbils adopting varied foraging and risk management strategies while acutely infected, ultimately the ecological costs of chronic infection were the same for all individuals. As individuals became sicker, they exhibited increased lethargy through reduced foraging efforts in patches, higher GUDs, reduced time spent in patches, and lower levels of predator awareness. This observation supports our first hypothesis. Thus, while the immediate ecological costs of acute infection for individuals varied, long term infection carries increasing foraging costs for all. This highlights that the effective quality of time dedicated to harvesting resources and managing predation risk becomes compromised gradually with time since infection.

We acknowledge that the inferences drawn from these findings may be limited by a small sample size for recorded individuals. A further study is required to expand on these findings. However, despite the small samples size, our study shows a clear pattern of changing foraging efficiency and predator awareness for individuals in response to changing states of infection. Thus demonstrating how infected gerbils vary their individual foraging behaviour and predator avoidance over time in response to the debilitating effects of a bacterial infection.

In conclusion, although all individuals began in a similar state and ended up carrying similar ecological costs (i.e. high GUDs, high vigilance) with the onset of long-term infection, their journey from uninfected to chronically infected revealed high levels of individual variation. The observed differences in the individual responses during initial (acute) infection reflects a degree of behavioural plasticity potentially due to a number of individual specific traits. These may include, potential differences in the present and future value of food to individuals, with those that have larger caches of seeds placing lower value on immediate foraging gains (Kotler *et al.,* 1999; Van der Merwe *et al.,* 2014). Additionally, differences in physiological state (immunity to infection, fitness costs; Hempel, 2011; Schmid-Hempel, 2003) and host personality (fear responses; Dall *et al.,* 2004; Poulin, 2013) may have contributed to the observed variation in individual foraging behaviour and risk management.

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Data availability

The datasets generated during and analysed during the current study are available from the corresponding author on reasonable request.

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