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Effects of maternal disease history on provisioning, brooding, and offspring outcomes

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Abstract

Disease within a population has the ability to shape the development, evolution, and general performance of a species. Pathogen exposure to hosts can influence their physiology and behavioral patterns to further shape offspring immunity. Parental conditions experienced by offspring during early development can benefit survival and fitness (e.g. increasing provisioning rates), as well as help deter against similar diseases experienced by parents. By testing if parental behavior changes can better prepare offspring outcomes for disease exposure, such as disease severity or duration of infection, we can see the beneficial impacts it has on disease dynamics and host-pathogen processes. Incubation temperature, resource provisioning, and brooding time are parental behaviors that play an important role in improving offspring body condition. Here, I used a common avian host-pathogen system to test the hypothesis that prior pathogen exposure alters parental behaviors such as nest provisioning and brooding lengths in the domestic canary, affecting offspring outcomes to shape population-level disease dynamics. Female domestic canaries (*Serinus canaria domestica*) were infected experimentally with *Mycoplasmal conjunctivitis* (MG) then paired for breeding after recovery. Post laying, eggs were given to foster mothers in a 2x2 design: biological mother disease history (prior MG or sham exposure) by foster mother disease history (prior MG or sham exposure). Following offspring fledging, I examined video footage of exposed and unexposed foster mothers during chick rearing, recording number of trips to the nest, brooding events, feeding events and the amount of time spent on the nest. This study revealed that MG exposed foster mothers took fewer trips to the nest overall and, specifically, fewer feeding trips than control mothers. However, MG exposed foster mothers spent more time brooding nestlings than control mothers, suggesting that their offspring will be better equipped to handle disease relative to offspring of control mothers

because lower amounts of brooding during offspring development can cause increased susceptibility to infection. While we found no effect of treatment on offspring body condition, there may be other offspring outcomes that have more importance for disease risk. Examining prior disease exposure on maternal behaviors can help us to better understand the value of transgenerational effects in conservation studies.

Introduction

Disease is an important driver of population dynamics (Jesse et al., 2021; McCallum & Dobson, 1995). It can influence the evolution, behavior, reproduction, and abundance of wildlife species (Jesse et al., 2021). With increasing mortality rates, abundance within a population falls and could ultimately lead to extinction. In amphibians, the chytrid fungal epidemic is a prime example of how diseases can cause rapid declines in populations through high mortality rates and increased evolution of the disease (Scheele, 2019). Further, disease can worsen the effect of other stressors within an environment such as climate change or habitat fragmentation (McCallum & Dobson, 2002; Russell et al., 2020).

Effects of epidemics reach far beyond host mortality. In some cases, they lead to changes in reproductive investment, where hosts may put all their energy into investment because mortality is likely or reduce investment in reproduction to conserve energy for future opportunities (Brannelly et al., 2016). For example, immune responses trigger male blue-footed boobies (*Sula nebouxii*) to increase parental care efforts (Velando et al., 2006) whereas female alpine tree frogs (*Litoria verreauxii alpina*) show reductions in reproductive effort (Brannelly et al., 2021). Thus, when faced with infection or disease, animals can increase energy investments in provisioning and reproductive efforts that lead to better reproductive success and offspring survival (Klug & Bonsall, 2014a; Velando et al., 2006).

One such way parents might alter their reproductive investment is via parental care behaviors. Organisms often alter their behavior to mitigate environmental challenges, such as disease (Ruden & Adelman, 2021; Sudnick et al., 2021). For example, pathogens impose strong selective pressures on hosts, leading to behaviors that mitigate the impacts of disease (Ruden & Adelman, 2021). Further, many factors of maternal behavior and physiology can lead to

phenotypic variances in offspring, including their ability to resist disease or increase performance (Bernardo, 1996). For example, incubation temperature, oviposition sites, maternal size, and brooding time can influence offspring development (Heath et al., 1999; Mousseau & Fox, 1998a, 1998b). Similarly, resource provisioning (i.e. food and water) plays an important role in offspring body condition, growth rates, and nutrition (Amonett, 2020; Eggert et al., 1998; Hopkins et al., 2011; Mainwaring et al., 2012) and is important for offspring survival and reproductive success (Klug & Bonsall, 2014b). Thus, changes in provisioning due to parental disease history can shape offspring outcomes. These behavioral responses can affect the next generation's developmental environment, ultimately leading to transgenerational effects on offspring phenotypes (Monaghan, 2008). Meaning, the environment or stressors placed on the mother can influence offspring development and help prime offspring for similarly stressful environments (Mousseau & Fox, 1998a, 1998b; Rossiter, 1991).

In my research, I examine how prior exposure to *Mycoplasma gallisepticum* (MG) in female domestic canaries (*Serinus canaria domestica*) affects provisioning and brooding behavior towards nestlings. MG is an avian bacterial pathogen that causes chronic respiratory disease by inflammation of air passageways in poultry and eye inflammation and mortality in wild house finches (*Haemorhous mexicanus*) (Kleven, 2008; Lu et al., 2017; Staley et al., 2017; Zhang et al., 2020). I predict that parents with a history of disease will increase their resource provisioning, resulting in offspring that are in better body condition prior to disease exposure. This would lead to increases in mass and fat reserves in the offspring raised by MG-exposed foster mothers due to higher frequency of feeding and visitations (more on bout periods) and overall greater attentiveness to offspring (longer brooding intervals). I will be able to draw conclusions on different parental behavioral patterns between the two by examining offspring

body condition. Having a better body condition would mean the increase in provisioning is worth the increased energy spent by the parent on provisioning (Hopkins et al., 2011; Kersten & Brenninkmeijer, 2008). Studying disease transmission and generational effects is important for avian health conservation and disease dynamics and can help us determine how to reduce severity effects within populations and limit large outbreaks.

Methods

Study species and husbandry. Domestic canaries were used to model the MG-house finch system. Canaries and house finches have similar pathogen loads, production-specific serum antibodies, pathology, and recovery time after exposure to MG (Hawley et al., 2011). Birds were housed in 30 x 18 x 18 cm cages with nesting material, seed, and water throughout the breeding season. Prior to breeding, half the mothers were inoculated with MG (n=21) suspended in Frey's media (5.00x107 CCU/mL; VA1994; E. Tulman, University of Connecticut) in both palpebral eye conjunctiva and the other half were inoculated with a sham solely of Frey's media (n=21). After recovering, females were paired with males for breeding (41-43 days after initial exposure). Males were then removed one week after the first egg was laid and housed separately throughout the entirety of the nestling rearing. Eggs were removed immediately after laying and given to foster mothers in a 2x2 design: biological mother disease history (prior MG or sham exposure) by foster mother disease history (prior MG or sham exposure) (Figure 2). There was a total of 22 foster mother nests (n=12 control nests; n=10 MG nests). Offspring mass and fat scores (0-3) were measured at 40 days old.

Video monitoring of female parental care behaviors. Video cameras mounted on stands were set outside the cages to record provisioning activity. Blind to treatment, I watched video data of all nests and noted provisioning behaviors such as brooding (time spent in the nest incubating the offspring), feeding (counted by the number of times nestlings are fed during on bouts), and number of overall trips to the nest.

Videos were recorded from the start of hatch date to date of fledging. Duration of time spent in the nest was recorded when the mother returned to the nest. Nest arrival and departure times were recorded and whether the mother was brooding, feeding, or both during that interval.

Videos were recorded each day between 8:00 and 20:00h about once an hour for 30 mins from hatching to fledging (about 17 days), resulting in a total of 2669 videos watched. Videos were separated based on mother ID, clutch number (1 or 2; only 5 mothers fostered a second clutch), and hatch date. After the offspring were fully fledged, I recorded mass and fat at fledging.

Statistical analysis. All statistics were conducted with R version 4.1.0 in R Studio (RStudio Team, 2021). To test for the effects of prior exposure to MG, days since hatch date, number of clutches, number of eggs hatched in a clutch, and the interaction between treatment and time on all response variables, we conducted linear mixed-effects models followed by ANOVAs. The response variables were: number of trips to the nest per minute (count of trips/length of video), proportion of time spent at the nest (time spent provisioning at nest/length of video), number of brooding trips per minute at the nest (count of brooding trips/length of video), and number of feeding trips per minute at the nest (count of feeding trips/length of video) (*lme4 & car* packages) (Bates et al., 2014).

For body condition models, we looked at the effect of prior exposure to Mycoplasma gallisepticum (MG), trips to the nest per minute, and the interaction between treatment and number of trips to the nest per minute on fat score and mass of 40 day old offspring. The same was done for proportion of time spent at the nest by examining the effect of prior exposure to Mycoplasma gallisepticum (MG), proportion of time spent at the nest, and the interaction between treatment and proportion of time spent at the nest on fat score and mass of 40 day old offspring. Both were conducted with linear mixed-effects models followed by ANOVAs.

Results

Results of parental behavior

There was a marginal main effect of treatment for proportion of time spent on the nest, which is the amount of time the foster mother was provisioning (i.e. brooding or feeding) the chicks within a video. MG foster mothers spent more time provisioning at the nest than control foster mothers ($\beta(MG)$ = 0.116 ± 0.055 SE, t=2.115, p=0.135; Figure 1A & Table 1), but there were no other main effects of treatment on any other response variable (Table 2, 3, and 4). However, there were significant interactions between treatment and time on every predictor.

The interaction between treatment and time had a significant effect on the number of trips to the nest per minute (i.e. the number of times per minute the foster mother visited the nest within a video). Foster mothers with prior exposure to MG increased nest visits less over time relative to control mothers, which greatly increased nest visit frequency as chicks aged ($\beta(MG)$ = -0.002 ± 0.001 SE, t=-3.650, p=<0.001; Figure 1B & Table 2). The opposite was found for proportion of time spent on the nest, where MG foster mothers had a significantly shallower negative slope and spent more time with their offspring, mainly within the interval of 6-15 days post hatch ($\beta(MG)$ = -0.004 ± 0.002 SE, t=-2.58, p=0.01; Figure 1A & Table 1). The interaction between treatment and time had a significant effect on number of brooding trips to the nest per minute, where MG foster mothers had more brooding events relative to control foster mothers ($\beta(MG)$ = 0.001 ± <0.001 SE, t=2.117, p=0.034; Figure 1C & Table 3), and the number of feeding trips to the nest per minute, where control foster mothers had more feeding events relative to MG foster mothers ($\beta(MG)$ = -1.305e-03 ± <0.001 SE, t=-2.670, p=0.008; Figure 1D & Table 4).

For all response variables, there was a main effect of days since hatch date of offspring, with trips per minute increasing with time ($\beta(MG)$ = 0.003 ± <0.001 SE, t=9.359, p=<0.001; Figure 1B & Table 2) and feeding trips per minute increasing with time ($\beta(MG)$ = 0.002 ± <0.001 SE, t=6.837, p=<0.001; Figure 1D & Table 4). Meanwhile, proportion of time spent at the nest decreased with time ($\beta(MG)$ = -0.046 ± 0.001 SE, t=-48.532, p=<0.001; Figure 1A & Table 1) as did brooding trips per minute decreased with time ($\beta(MG)$ = -0.006 ± <0.001 SE, t=-29.794, p=<0.001; Figure 1C & Table 3).

We found a significant negative main effect of clutch number on trips per minute ($\beta(MG)$ = -0.013 ± 0.005 SE, t=-2.678, p=0.007; Figure 1B & Table 2), proportion of time spent at the nest ($\beta(MG)$ = -0.043 ± 0.016 SE, t=-2.747, p=0.006; Figure 1A & Table 1), and brooding trips per minute ($\beta(MG)$ = -0.007 ± 0.003 SE, t=-1.978, p=0.048; Figure 1C & Table 3). There was no significant effect of clutch number on the number of feeding trips (p=0.157; Table 4). For all response variables, mothers did less provisioning with the second clutch than the first. However, sample size was low due to only 5 out of 22 foster mothers having more than one clutch.

There was a positive significant main effect of number of hatched offspring in a clutch on total trips per minute ($\beta(MG)$ = 0.017 ± 0.002 SE, t=7.101, p=<0.001; Figure 1B & Table 2) and feeding trips per minute ($\beta(MG)$ = 0.020 ± 0.002 SE, t=8.832, p=<0.001; Figure 1D & Table 4), and a significant negative effect on proportion of time spent at the nest ($\beta(MG)$ = -0.021 ± 0.008 SE, t=-2.688, p=0.007; Figure 1A & Table 1). Meaning, mothers with more offspring take more trips and feeding trips to the nest and spend less time overall at the nest. There was no significant effect of number of hatched offspring on the number of broodings trips (p=0.888; Table 3).

Results for offspring outcomes

There was no significant effect of trips to the nest per minute or proportion of time spent at the nest on 40 day old fledgling fat score (all p-values > 0.41; Tables 5 & 6) or mass (all p-values > 0.39; Tables 7 & 8).

Discussion

In this study, we examine how prior pathogen exposure can alter maternal effects, leading to differences in offspring outcomes. Using video monitoring, I tracked maternal behaviors in mothers either with MG history or without. We found that attentiveness to offspring differed from control foster mothers to MG foster mothers, with MG mothers spending more time with their offspring while control mothers make trips to the nest more frequently. Specifically, MG foster mothers made feeding trips to the nest less frequently than control mothers, but brooded more, meaning they spent more time with their chicks in the nest. While we did not find any significant differences in body condition between fledged offspring of foster mothers with or without prior MG exposure, there may be other consequences to the differences in brooding and feeding that are unrelated to body condition. This supports my hypothesis that prior pathogen exposure can lead to differences in maternal behaviors (i.e. increased time spent at the nest for MG foster mothers) that could help offspring combat similar diseases, but does not support that those behaviors shape offspring body condition.

Over time, control mothers took trips to the nest more frequently and had greater feeding visitations, the two likely related since the more the mother has to feed, the more visits she is making to do so. However, control mothers also showed fewer brooding visitations. This makes sense as the more time spent feeding their offspring and moving to and from the nest, the less time they have to brood their chicks. The opposite was true for MG mothers, who made fewer feeding trips but spent more time brooding offspring. Similar trade-off results were found in (Rosa' & Murphy', 2023) the Eastern Kingbird (*Tyrannus tyrannus*), where time spent at the nest attending to young negatively correlated with the amount of feeding trips to the nest. There is a difficulty for the mother to both feed and brood equally. Brooding is critical during early

offspring development by maintaining body temperatures for chicks. Chicks that experience lower temperatures during development have increased susceptibility to diseases from impaired immune systems, lower growth rates, and negative effects on performance (Fairchild, 2012). If brooding is more important than feeding frequency for offspring outcomes, offspring of control mothers may suffer harsher effects of disease than those of MG foster mothers.

Effects from maternal behaviors were not observed on any measures of offspring body condition, however, other factors could influence the differences seen in maternal provisioning behaviors between control and MG foster mothers. Further, we might not see effects on offspring body condition because there could be advantages to frequent feeding and brooding. The two are both beneficial for that particular outcome (Fairchild, 2012; Klug & Bonsall, 2014b). There are other outcomes not measured here that might be more important for disease risk.

We also found that regardless of prior infection status, mothers with greater numbers of offspring in a clutch take more trips to the nest as well as more feeding trips to the nest. This is likely an artifact of the feeding; the more offspring they have, the less time they can spend brooding them. With an increase in number of offspring, they also face the issue of begging intensity and must compensate for the decrease in brooding frequency with more feeding trips (Leonard, 2000). Clutch number was not highly considered due to the fact that only 5 out of the 22 mothers had second clutches.

MG epidemics in house finches have detrimental effects on their populations, with more than half of eastern populations decreasing in abundance since the emergence of MG in 1994 (Heylen et al., 2020; Hochachka & Dhondt, 2000). Because the disease is widespread and can be carried long distances by birds, it is important in determining which factors contribute to reduction of epidemic severity. Studying generational effects in diseased populations is

important for understanding how previous epidemics affect susceptibly of future generations and host-pathogen co-evolution. However, it is unclear whether maternal disease exposure plays a bigger role in offspring development and resistance to infection. Using avian-MG host pathogen systems we found that while effects of offspring body condition do not seem to be clearly shaped by the mother's prior exposure history to MG, differences in maternal behaviors can be attributed to prior exposure to MG. Epidemic models such as these can provide opportunities to better treat the spread of disease starting from small-scale populations.

Figure 1 | Effects of prior maternal exposure to *Mycoplasma gallisepticum* (MG) over time on **A**) proportion of time spent at the nest (β=-0.004, t=-2.580, p=0.01), **B**) number of trips per minute (β=-0.002, t=-3.650, p=<0.001), **C**) number of brooding trips per minute (β=0.001, t=2.117, p=0.034), and **D**) number of feeding trips per minute (β=-1.305e-03, t=-2.670, p=0.008).

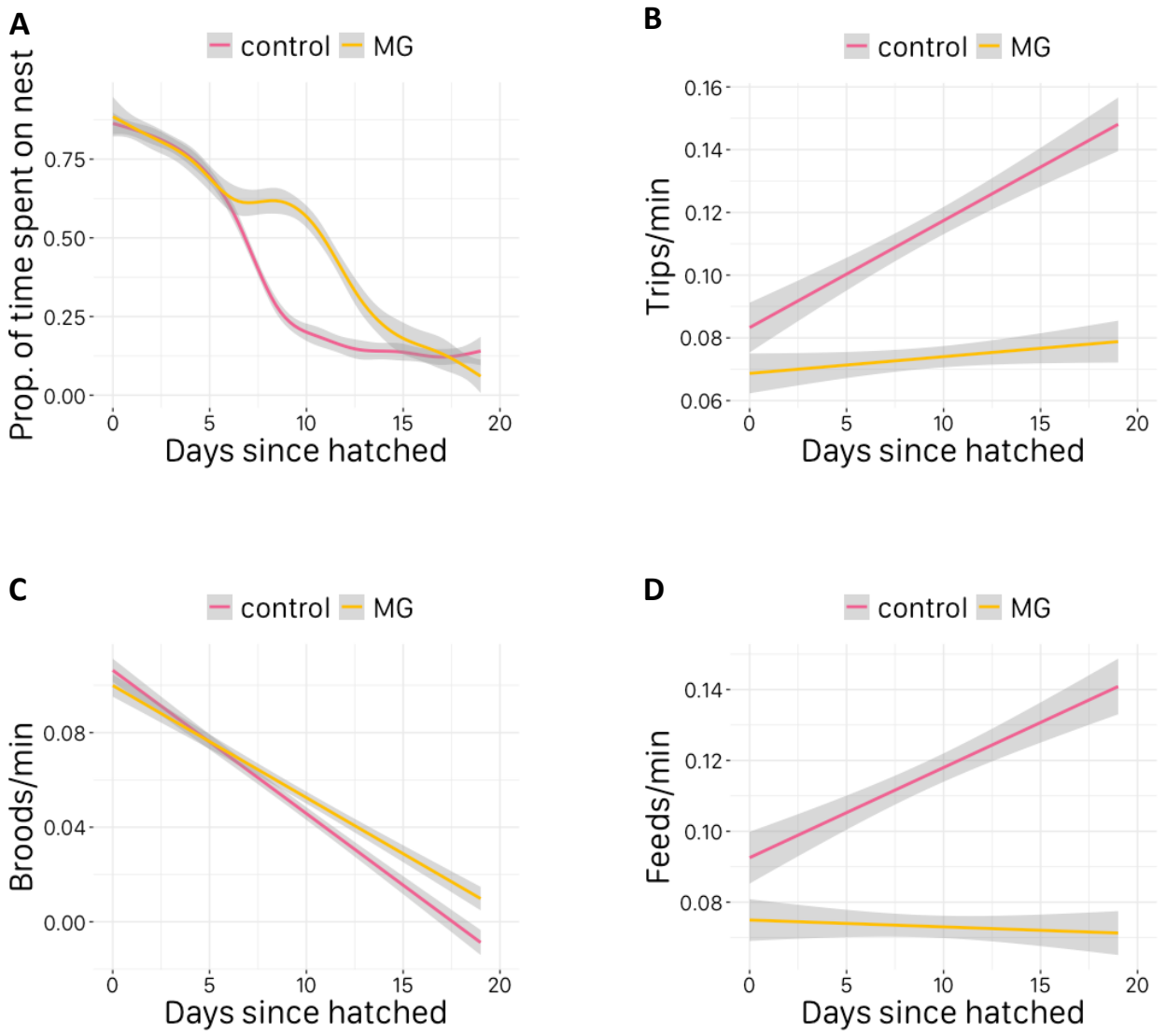

Figure 2 | 2x2 cross-fostering design: biological mother disease history (prior MG or sham exposure) by foster mother disease history (prior MG or sham exposure).

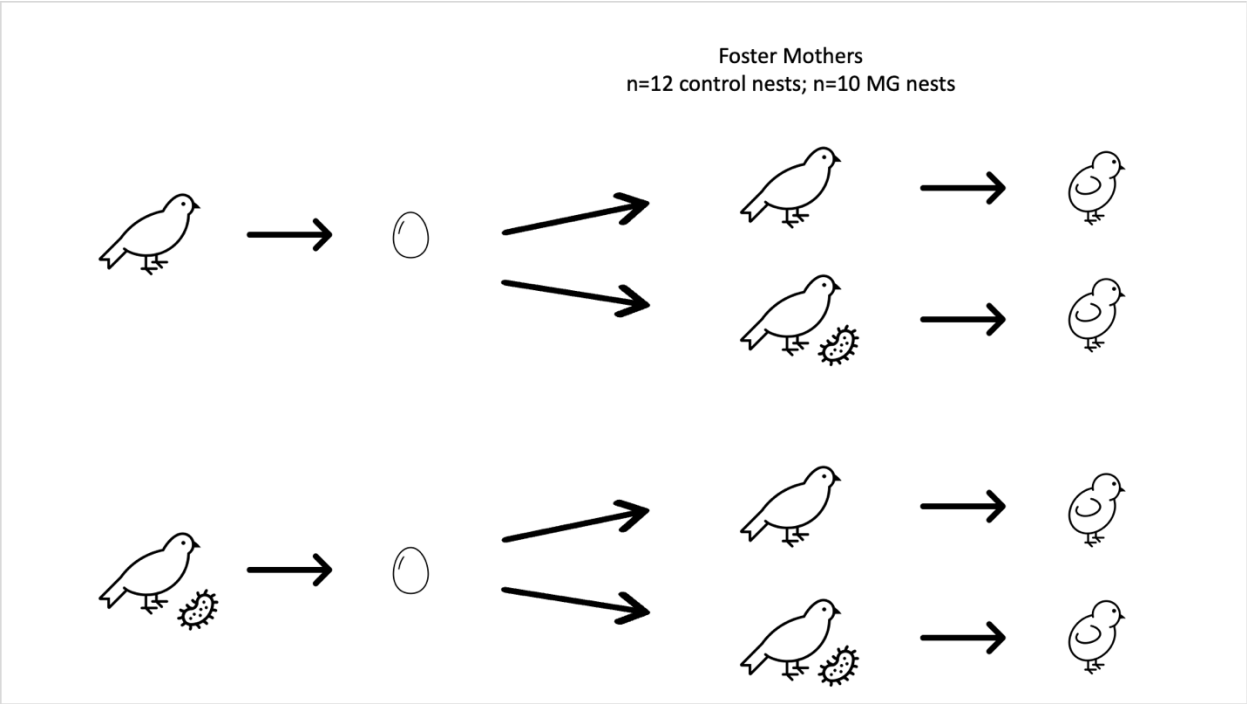

Table 1 | Results from the linear mixed-effects model examining the effect of prior exposure to Mycoplasma gallisepticum (MG), days since hatch date, number of clutches, number of eggs hatched in a clutch, and the interaction between treatment and time on proportion of time spent at the nest and ANOVA.

Linear mixed effects model results			
	Coefficient	SE	t value
Intercept	0.938	0.048	19.668
Treatment MG	0.116	0.055	2.115
Day	-0.046	0.001	-48.532
Clutch	-0.043	0.016	-2.747
Number of hatched offspring	-0.021	0.008	-2.688
F.Treatment MG:day	-0.004	0.002	-2.580

ANOVA results			
	Chi squared	df	p value
Treatment	2.24	1	0.135
Day	3713.089	1	<0.001
Clutch	7.545	1	0.006
Number of hatched offspring	7.226	1	0.007
Treatment MG:day	6.655	1	0.01

Table 2 | Results from the linear mixed-effects model examining the effect of prior exposure to Mycoplasma gallisepticum (MG), days since hatch date, number of clutches, number of eggs hatched in a clutch, and the interaction between treatment and time on number of trips to the nest per minute and ANOVA.

Linear mixed effects model results			
	Coefficient	SE	t value
Intercept	0.052	0.013	3.950
Treatment MG	0.008	0.014	0.533
Day	0.003	<0.001	9.359
Clutch	-0.013	0.005	-2.678
Number of hatched offspring	0.017	0.002	7.101
Treatment MG:day	-0.002	0.001	-3.650

ANOVA results			
	Chi squared	df	p value
Treatment	0.46	1	0.498
Day	78.640	1	<0.001
Clutch	7.17	1	0.007
Number of hatched offspring	50.419	1	<0.001
Treatment MG:day	13.324	1	<0.001

Table 3 | Results from the linear mixed-effects model examining the effect of prior exposure to Mycoplasma gallisepticum (MG), days since hatch date, number of clutches, number of eggs hatched in a clutch, and the interaction between treatment and time on number of brooding trips per minute at the nest and ANOVA.

Linear mixed effects model results			
	Coefficient	SE	t value
Intercept	0.111	0.009	12.032
Treatment MG	0.003	0.010	0.250
Day	-0.006	<0.001	-29.794
Clutch	-0.007	0.003	-1.978
Number of hatched offspring	<0.001	0.002	0.141
Treatment MG:day	0.001	<0.001	2.117
ANOVA results			
	Chi squared	df	p value
Treatment	0.853	1	0.356
Day	1200.107	1	<0.001
Clutch	3.913	1	0.048
Number of hatched offspring	0.02	1	0.888
Treatment MG:day	4.483	1	0.034

Table 4 | Results from the linear mixed-effects model examining the effect of prior exposure to Mycoplasma gallisepticum (MG), days since hatch date, number of clutches, number of eggs hatched in a clutch, and the interaction between treatment and time on number of feeding trips per minute at the nest and ANOVA.

Linear mixed effects model results			
	Coefficient	SE	t value
Intercept	0.048	0.013	3.679
Treatment MG	<0.001	0.015	-0.005
Day	0.002	<0.001	6.837
Clutch	<0.001	0.005	-1.415
Number of hatched offspring	0.020	0.002	8.832
Treatment MG:day	0.001	<0.001	-2.670

ANOVA results			
	Chi squared	df	p value
Treatment	0.638	1	0.425
Day	41.911	1	<0.001
Clutch	2.002	1	0.157
Number of hatched offspring	78.006	1	<0.001
Treatment MG:day	7.128	1	0.008

Table 5 | Results from the linear mixed-effects model examining the effect of prior exposure to Mycoplasma gallisepticum (MG), trips to the nest per minute, and the interaction between treatment and number of trips to the nest per minute on fat score of 40 day old offspring and ANOVA.

Linear mixed effects model results			
	Coefficient	SE	t value
Intercept	1.514	0.578	2.618
Treatment MG	-1.737	1.192	-1.457
Trips to nest per minute	0.454	4.771	0.095
Treatment MG:Trips to nest per minute	16.008	14.236	1.125

ANOVA results			
	Chi squared	df	p value
Treatment	1.661	1	0.197
Trips to nest per minute	0.251	1	0.616
Treatment MG:Trips to nest per minute	1.265	1	0.261

Table 6 | Results from the linear mixed-effects model examining the effect of prior exposure to Mycoplasma gallisepticum (MG), proportion of time spent at the nest, and the interaction between treatment and proportion of time spent at the nest on fat score of 40 day old offspring and ANOVA.

Linear mixed effects model results			
	Coefficient	SE	t value
Intercept	1.293	0.638	2.027
Treatment MG	-0.204	0.776	-0.263
Proportion of time spent at nest	0.676	1.492	0.453
Treatment MG:Proportion of time spent at nest	-0.898	1.738	-0.517

ANOVA results			
	Chi squared	df	p value
Treatment	2.533	1	0.112
Proportion of time spent at nest	<0.001	1	0.985
Treatment MG:Proportion of time spent at nest	0.267	1	0.605

Table 7 | Results from the linear mixed-effects model examining the effect of prior exposure to Mycoplasma gallisepticum (MG), trips to the nest per minute, and the interaction between treatment and number of trips to the nest per minute on 40 day old offspring mass and ANOVA.

Linear mixed effects model results			
	Coefficient	SE	t value
Intercept	18.067	1.150	15.710
Treatment MG	1.664	2.815	0.591
Trips to the nest per minute	5.548	9.102	0.610
Treatment MG:Trips to nest per minute	- 12.441	33.804	- 0.368

ANOVA results			
	Chi squared	df	p value
Treatment	0.673	1	0.412
Trips to nest per minute	0.281	1	0.596
Treatment MG:Trips to nest per minute	0.136	1	0.713

Table 8 | Results from the linear mixed-effects model examining the effect of prior exposure to Mycoplasma gallisepticum (MG), proportion of time spent at the nest, and the interaction between treatment and proportion of time spent at the nest on 40 day old offspring mass and ANOVA.

Linear mixed effects model results			
	Coefficient	SE	t value
Intercept	19.606	1.257	15.592
Treatment MG	- 0.633	1.551	- 0.408
Proportion of time spent at nest	- 2.352	2.912	- 0.808
Treatment MG:Proportion of time spent at nest	2.926	3.443	0.850

ANOVA results			
	Chi squared	df	p value
Treatment	0.476	1	0.490
Proportion of time spent at nest	0.028	1	0.868
Treatment MG:Proportion of time spent at nest	0.722	1	0.396

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