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Experimental removal of nematode parasites increases growth, sprint speed, and mating success in brown anole lizards

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Abstract

Parasites interact with nearly all free-living organisms and can impose substantial fitness costs by reducing host survival, mating success, and fecundity. Parasites may also indirectly affect host fitness by reducing growth and performance. However, experimentally characterizing these costs of parasitism is challenging in the wild because common antiparasite drug formulations require repeated dosing that is difficult to implement in free-living populations, and because the extended-release formulations that are commercially available for livestock and pets are not suitable for smaller animals. To address these challenges, we developed a method for the long-term removal of nematode parasites from brown anole lizards (*Anolis sagrei*) using an extended-release formulation of the antiparasite drug ivermectin. This treatment eliminated two common nematode parasites in captive adult males and dramatically reduced the prevalence and intensity of infection by these parasites in wild adult males and females. Experimental parasite removal significantly increased the sprint speed of captive adult males, the mating success of wild adult males, and the growth of wild juveniles of both sexes. Although parasite removal did not have any effect on survival in wild anoles, parasites may influence fitness directly through reduced mating success and indirectly through reduced growth and performance. Our method of long-term parasite manipulation via an extended-release formulation of ivermectin should be readily adaptable to many other small vertebrates, facilitating experimental tests of the extent to which parasites affect host phenotypes, fitness, and eco-evolutionary dynamics in the wild.

KEYWORDS

Anolis sagrei, cost of parasitism, growth cost, mark-recapture study, performance cost, sexually transmitted parasite, survival

1 | INTRODUCTION

Parasites interact with nearly all free-living organisms and are known to impose a variety of costs on their hosts, including decreases in host growth, fat storage, and performance, increases in host metabolism and changes in host behavior (Binning et al., 2017; Careau et al., 2010; Cox et al., 2015; Eraud et al., 2005; Finnerty et al., 2017; Forbes et al., 2002; Hawley et al., 2012; Kelehear et al., 2019; Lafferty & Morris, 1996; Moore, 2013; Tierney et al., 1996; Wedekind & Milinski, 1996). The costs are in part mediated through activation of energetically expensive immune responses and collateral damage from inflammation and the production of reactive oxygen species (Ashley et al., 2012; Demas et al., 2011; Dowling & Simmons, 2009; Hasselquist & Nilsson, 2012; Sadd & Siva-Jothy, 2006). Ultimately, these costs are expected to accrue in terms of fitness, and parasites have frequently been found to reduce the survival and reproductive success of their hosts (Albery et al., 2021; Newey & Thirgood, 2004; Pai & Yan, 2003; Patterson et al., 2013; Robar et al., 2010; Schall, 1983; Wittman & Cox, 2021; however, Raveh et al., 2011, 2015). Through their detrimental effects on host fitness, parasites may regulate host populations and influence their evolutionary dynamics (Anderson & May, 1978; Pedersen & Fenton, 2015).

Early studies on the effects of parasitism in natural populations were predominantly observational, and their inferences often relied on age structure of parasite burden, associations between individual fitness and parasite burden, or associations between parasitism and proxies for host fitness (Coulson et al., 2018; Minchella & Scott, 1991; Pedersen & Fenton, 2015; Rousset et al., 1996). However, many processes can create positive or negative correlations between parasite burden and host characteristics in the absence of a direct causal relationship (e.g., individual hosts that feed more may grow more and also ingest more parasites; hosts in poor condition may have both weakened immune responses and lower clearance of parasites). Therefore, observational studies cannot establish the causal influence of parasitism on individual hosts or, by extension, on host populations. Experimental infection studies have increased our understanding of the costs of parasitism in terms of host survival and reproductive success, but they are generally performed in a laboratory or controlled environment in which organisms are removed from natural conditions that they experience in the wild (Atkinson et al., 1995; Blaser & Schmid-Hempel, 2005; Ebert et al., 2000; Ilmonen et al., 2008; Sharp & Vincent, 2015). The experimental removal of parasites in a framework that tracks individual hosts through time and under natural conditions provides a powerful approach to study the causal effects of parasitism on host populations. As this experimental approach has become more common, it has led to new insights on the effects of parasitism on host phenotypes (growth, body condition, performance, behavior) and fitness (survival and reproduction) while also increasing our understanding of coinfection dynamics and epidemiological processes (Binning et al., 2017; Budischak et al., 2016; Pedersen & Antonovics, 2013; Pedersen & Fenton, 2015; Sánchez et al., 2018, Sweeny et al., 2020). While the popularity of parasite removal experiments has grown, the taxonomic scope of such experiments in vertebrates has been focused on mammals and birds. Non-avian reptiles are poorly represented in experimental parasite removal and infection studies (Main & Bull, 2000; Oppliger et al., 1999), particularly for tests of the fitness costs of parasitism (Pedersen & Fenton, 2015). Expanding the taxonomic breadth of studies that experimentally test for costs of parasitism will allow for better comparative tests of theories relating pace-of-life and metabolic rate with immune strategy and parasite defense, and theories implicating parasitism in the evolution of endothermy (Casadevall & Damman, 2020; K. A. Lee, 2006; Logan, 2019; Sánchez et al., 2018; Sandmeier & Tracey 2014).

A common challenge associated with experimental parasite removal in the wild is the recommended timing for repeated dosing with antiparasite drugs (Barragry, 1987; Soll, 1989). Conducting frequent recaptures for retreatment is logistically difficult and potentially stressful for the host, and experimental subjects may be missed during a recapture census and not receive a supplemental dose. Yet, frequent dosing may be required if wild hosts are continually exposed to parasites, such that treated individuals face reinfection as the antiparasite drug is excreted and falls below therapeutic levels. Over time, these factors can reduce any differences in parasite prevalence and intensity between treatment groups while increasing variance within treatment groups, thereby complicating inferences about treatment effects, especially when the study is conducted over a time span that exceeds the half-life of the drug (Easterly et al., 1992; Ezenwa et al., 2010; Friant et al., 2016; Irvine et al., 2000; Knowles et al., 2013; Ranjan et al., 1997; Thomas & Morgan, 2013; Wahid et al., 1989). Therefore, extended-release drug formulations have many advantages over traditional drug formulations that require frequent dosing to maintain their effectiveness against parasites (Carlsson et al., 2012; Ezenwa et al., 2010).

Although extended-release formulations of antiparasite drugs have been developed for the livestock production industry and are commercially available, they are generally unsuitable for use in smaller animals that are often studied in ecological and evolutionary research (Boehringer Ingelheim, 2019; Geng et al., 2016; González Canga et al., 2009; Soll et al., 1990). This is because the mechanism of release for some commercially available products is only appropriate for ruminants, or because formulations for large animals are too concentrated to be accurately administered by simply reducing the volume given to small animals. Of the available extended-releases formulations, in situ gelling injections are particularly promising for long-term parasite removal in small vertebrates. Gelling compounds can be easily and affordably produced in the laboratory, allowing researchers to tailor the formulation to their specific host and parasite system and can sustain an effective level of drug release for many months (Geng et al., 2016).

In this study, we developed an extended-release formulation of the antihelminth drug ivermectin and then tested its efficacy for the removal of nematode parasites in the brown anole lizard, *Anolis sagrei*. Ivermectin is tolerated well by most mammals, amphibians,

fish, birds, and squamate reptiles, has broad-spectrum action against nematodes and arthropods, has a wide range between therapeutic and toxic doses, and can be used for extended periods with minimal side effects (Boyce et al., 1992; Camargo et al., 2013; Davies & Rodger, 2000; Jacobson, 2007; Langford et al., 2013; Letcher & Glade, 1992; Wilson & Carpenter, 1996). Ivermectin causes paralysis in nematodes and arthropods by selectively binding to and opening glutamate-gated chloride ion channels in nerve and muscle tissue (Turner & Schaeffer, 1989). We specifically tested the efficacy of an extended-release formulation of ivermectin for the removal of *Physaloptera* sp. (Physalopteridae) and *Cyrtosomum penneri* (Atractidae), two nematodes that are common parasites of *A. sagrei* adults (see Methods, below). In addition to testing the effects of extended-release ivermectin formulations on these two parasites in captivity (where posttreatment exposure to parasites is unlikely) and in the wild (where repeated exposure to parasites is likely), we also tested for any detrimental effects of ivermectin on performance by measuring sprint speed shortly after treatment. We then used this technique to test for predicted costs of nematode parasites with respect to performance, growth, survival, and mating success in adult *A. sagrei* as well as growth and survival in juvenile *A. sagrei*. Relative to individuals in control groups that received only the gelling vehicle for drug delivery, we predicted that individuals receiving an extended-release formulation of ivermectin would exhibit (1) reduced prevalence and intensity of infection by *Physalopetera* and *Cyrtosomum*, (2) increased sprint speed when measured 2 months posttreatment, (3) increased growth and mass gain as both juveniles and adults, (4) increased survival as both juveniles and adults, and (5) increased mating success, as measured by inferred copulation rates of males in each treatment.

2 | MATERIALS AND METHODS

2.1 | Lizard hosts and nematode parasites

The brown anole (*A. sagrei*) is a small lizard that is native to Cuba and The Bahamas and invasive across much of the southeastern United States, including our study populations in northeast Florida. Lizards used in our laboratory study were collected from Palm Coast, Florida (29°35′59″ N, 81°11′49″ W). Our field experiments were conducted on two nearby spoil islands located in the Guana Tolomato Matanzas National Estuarine Research Reserve (GTM NERR, 29°37′43″ N, 81°12′42″ W and 29°37′58″ N, 81°12′46″ W). Field work was conducted under permits from the GTM NERR and all procedures involving lizards were approved by the University of Virginia Animal Care and Use Committee (protocol 3896).

A. sagrei harbors a variety of internal macroparasites (Acanthocephala, Nematoda, Pentastomida, Trematoda), of which nematodes are usually the most prevalent (Goldberg & Bursey, 2000; Langford et al., 2013; Reedy et al., 2016; Thawley et al., 2019). In our study populations about half (56%) of all wild adult anoles are infected with *Physaloptera* sp. (Physalopteridae) and infection intensities range from 1 to 15 worms per infected anole (T. N. Wittman; unpublished data). *Physaloptera* is a large (5–15 mm total length) nematode that attaches to the mucosa of the stomach and uses the feces of anoles and other vertebrates to transmit its eggs, which are then ingested by intermediate arthropod hosts (e.g., Blattodea, Coleoptera, Hymenoptera, Orthoptera) that are common prey of anoles (Fincher et al., 1969; King et al., 2013; Lincoln & Anderson, 1975; Petri, 1950; S. H. Lee, 1957). A second nematode parasite, *C. penneri* (Atractidae), is much smaller (~1 mm length), resides in the rectum near the cloacal opening, reproduces viviparously within the host, and is only transmitted sexually (Langford et al., 2013). Infections of *C. penneri* can range upwards of 500 worms per anole, and nearly all adults harbor this parasite, whereas juveniles do not (Goldberg et al., 2002; Langford et al., 2013; Reedy et al., 2016). Experiments show that *C. penneri* does not infect anoles via oral transmission, nor does it infect snails or crickets that consume infected anole feces (Langford et al., 2013). However, 70% of male and 100% of female anoles acquire *C. penneri* after mating with an infected partner (Langford et al., 2013). Manipulations of reproduction via gonadectomy reduce the prevalence (percentage of individuals infected) of *C. penneri* infection in adult anoles of both sexes but have no effect on the prevalence or intensity (average number of parasites infecting individuals) of *Physaloptera* infection (Reedy et al., 2016). While some populations of *A. sagrei* harbor ectoparasites, we did not detect any ticks, mites, or other ectoparasites on the lizards used in this study (Reedy et al., 2016).

2.2 | Formulation of ivermectin gelling solution

We made an in situ gelling solution from the solvent N-methyl-2-pyrrolidone (NMP; Sigma-Aldrich; 328634; 15% v/v), the polymers polylactic acid (PLA; Polysciences; 22505; 5% m/v) and sucrose acetate isobutyrate (SAIB; Sigma-Aldrich; W518107; 85% v/v), and the active antiparasite drug ivermectin (IVM; Sigma-Aldrich; I18898; 1.5 μg/μl) (Camargo et al., 2013; Geng et al., 2016). Briefly, we melted PLA on a hot plate at 150°C, then added NMP and SAIB while stirring at 100°C until the PLA was fully dissolved and the solution was of uniform consistency, then reduced the heat to 50°C added ivermectin and continued stirring the solution until the ivermectin was fully dissolved. We stored this solution at 4°C and heated the solution at 50°C while stirring to reduce its viscosity prior to injection. Upon subcutaneous injection of this solution, the solvent diffuses into the aqueous environment of the organism while the hydrophobic polymers (SAIB and PLA) form a porous, semisolid gel containing the active drug ivermectin (Lin et al., 2012). This gel matrix then slowly degrades over several months, releasing ivermectin and the by-products lactic acid and sucrose (Göpferich, 1996; Phillips et al., 1976).

2.3 | Experiment 1: Captive adult males

To test the safety and efficacy of our gelling compound, we captured 38 adult male *A. sagrei* lizards in Palm Coast, Florida (29°35′59″

N, 81°11′49″ W) in July 2018 and transported them to our animal facility at the University of Virginia. We housed each lizard individually in a plastic cage (30 x 20 x 20 cm; Lee's Kritter Keeper) with a strip of outdoor carpet as a substrate, a section of polyvinyl chloride pipe for perching and hiding, and a strip of fiberglass mesh for basking under two ReptiSun 10.0 UVB (ultraviolet B) bulbs (ZooMed) suspended above the cage. We maintained animals on a 13L:11D photoperiod with a temperature of 29°C during the day, 25°C at night, and constant 65% relative humidity. We misted each cage daily with deionized water. Three times per week, we fed 3–5 crickets of 1/2″ length (*Gryllodes sigillatus;* Ghann's Cricket Farm) to each lizard. We dusted the crickets twice weekly with a calcium supplement (Fluker's Repta Calcium with D_3; Fluker Farms), and once weekly with a vitamin supplement (Fluker's Reptile Vitamin). We allowed animals to acclimate to laboratory conditions for 30 days prior to treatment.

Seven days prior to and 60 days after treatment, we measured snout-vent length (SVL) to the nearest 1 mm with a ruler and body mass to the nearest 0.01 g with a digital balance (Ohaus Scout Pro: SP202). We assigned each lizard to one of two treatments: (1) ivermectin ($n = 19$), in which animals received 1 µl per g body mass of the 1.5 µg/µl ivermectin gelling solution described above (1.5 µg ivermectin per g body mass), and (2) control ($n = 19$), in which animals received 1 µl per g body mass of the gelling vehicle without ivermectin. We haphazardly assigned treatment groups to individuals and confirmed that SVL and mass did not differ between treatment and control groups prior to treatment (SVL: $t = 1.73$, $p = 0.09$; Mass: $t = 0.88$, $p = 0.38$). We injected the drug or vehicle compounds subcutaneously, approximately 5 mm posterior and medial to the right shoulder, using a 25 µl Hamilton syringe (702 LT SYR) and a 26-gauge needle. We used 70% isopropyl alcohol swabs to sanitize the site of injection and the needle before and after each injection, and we used a new needle every five injections.

Upon injection of the gelling solution, dissipation of the solvent causes an initial burst release of the drug before the polymer matrix forms (Geng et al., 2016). Because of the initial burst of ivermectin released into the blood, any potential toxic effects of ivermectin are likely to occur in the days immediately following injection. Impaired locomotor performance is a common side effect of ivermectin toxicity (Clayton et al., 2013; Kim & Crichlow, 1995; Lovell, 1990; Verdú et al., 2018). To assess the effect of the gelling vehicle and the burst release of ivermectin on lizard performance during this immediate posttreatment period, we compared the maximum sprint speed of lizards 2 days pretreatment with the same measure at 2 days posttreatment. To test whether experimental removal of parasites improves performance, we also measured maximum sprint speed at 2 months posttreatment. We measured sprint speed at the same temperature and humidity in which lizards were housed (29°C, 65% relative humidity) by racing each lizard up a flat wooden track at an incline of 45.° The track was 1.2 m in length and 6 cm in width with sides 8 cm in height. We shrouded the upper end of the track to offer lizards a refugium to sprint toward. Four infrared sensors were spaced 10 cm apart starting 40 cm up the track (TrackMate Racing IRSENSORS). We recorded the time a lizard passed each sensor using TrackMate Racing Sc Timer software (Version 9.42), then calculated velocity (cm/s) over each 10 cm interval between sensors, which resulted in three measures of velocity per trial. We raced each lizard in three successive trials and selected the highest velocity across the nine measures (3 intervals x 3 trials) as the maximum sprint speed for each individual. Two lizards had maximum sprint speeds that were over 100 cm/s faster than the next fastest lizard in the data set. These values were significant outliers as assessed by Rosner's test, so these individuals were removed prior to analysis.

To test for a potential detrimental effect of either the gelling vehicle or the burst release of ivermectin on sprint speed, we used a linear mixed-effects model with time (2 day pretreatment, 2 day posttreatment) and treatment (ivermectin, control) as fixed effects with interaction, plus individual ID as a random effect. A significant decrease in sprint speed at 2 day posttreatment (main effect of time) would indicate a detrimental effect of the gelling vehicle or injection procedure, whereas a significant decrease in only the ivermectin group (time x treatment interaction) would indicate a detrimental effect of ivermectin. To test for the effect of parasites on sprint speed, we used a similar mixed-effects model with a different posttreatment time point (2-m posttreatment), with the expectation (subsequently confirmed) that ivermectin would eliminate parasites by that point. We predicted that any beneficial effects of parasite removal on performance would be evident as a significant time x treatment interaction. We also tested for effects of body length and body mass on sprint speed, but neither were significant, so they were not included as covariates in the model. We additionally tested if ivermectin males experienced a significant increase in sprint speed 2 m posttreatment compared to their pretreatment values. To do this, we used a mixed-effect model that included only ivermectin males, with time as a fixed effect and individual ID as a random effect. For visualization, we expressed change in performance by subtracting each male's pretreatment sprint speed from its posttreatment speed.

We tested for effects of parasite removal on growth in SVL (mm/d) and mass (g/d) using linear models with a fixed effect of treatment and initial body size (SVL or mass) as a covariate. Initial size was included as a covariate because growth rates decrease with size. At 70 days posttreatment, we euthanized each lizard, dissected out its gastrointestinal tract, and stored it in 100% ethanol. We sectioned the gastrointestinal tract into the stomach, small intestine, large intestine, and rectum, then counted all nematode parasites in each section under a x10 stereoscope. The two nematode genera are easily distinguished by their relative size and location; *Physaloptera* sp. is 5–15 mm in length and found embedded in the gastric mucosa, whereas *C. penneri* is only 1 mm in length and mainly found in the rectum and the posterior end of the large intestine. To test for treatment effects on prevalence of parasite infection (proportion of hosts infected), we used generalized linear models with a binomial error distribution and each individual scored as infected (1) or not (0) by each parasite type. To test for treatment effects on intensity of parasite infection (number of parasites per host), we used count data for each parasite type and generalized linear models with a negative

binomial error distribution and a log link function (Alexander, 2012). We performed all analysis in R v4.0.2 (R Core Team, 2020).

2.4 | Experiment 2: Wild adults

Using a population of *A. sagrei* on a small spoil island located in the GTM NERR (29°37′43″ N, 81°12′42″ W), we captured adult males and females at the beginning of the breeding season (March 2019) and treated individuals with an injection of either (1) our gelling formulation of ivermectin ($n = 91$ males, 90 females), or (2) the gelling vehicle as a control ($n = 87$ males, 87 females). Prior to treatment, we gave each individual a unique toe clip for identification, measured its SVL and body mass (see above), and sorted individuals by these measures of size to create size-matched treatment groups within each sex. Treatment groups did not differ in initial size for males (SVL: $t = 0.06$, $p = 0.95$; mass: $t = 0.36$, $p = 0.71$) or females (SVL: $t = 0.85$, $p = 0.39$; mass: $t = 0.57$, $p = 0.56$). Within 24 h of capture and treatment, we released each animal at its site of initial capture.

We resampled the island in May, July, and October of 2019. During each recapture census, we measured SVL and body mass to calculate growth in length (mm/d) and mass (g/d). We re-treated the animals with ivermectin or control injections calibrated to their new body mass during the July census. Because our final October sampling effort was not exhaustive, we did not include this census in capture-mark-recapture models of survival and recapture rate. Individuals captured in October were euthanized and dissected to assess treatment effects on parasite load, as described above, with the addition of sex as a fixed effect. We did not detect any significant treatment × sex interactions for the prevalence or intensity of either parasite, so we present results from models without an interaction term. We tested for effects of parasite removal on growth in SVL (mm/d) and mass (g/d) using linear models with a fixed effect of treatment and initial body size (SVL or mass) as a covariate. Initial size was included as a covariate because growth rates decrease with size. Because the distributions of the covariates (initial mass and initial SVL) have little overlap between the sexes, we tested for treatment effects on growth separately for adult males and females. To assess homogeneity of slopes, we tested for an interaction between initial size and treatment. For growth in body mass of adult males, we found a significant interaction between treatment and initial body mass, which we included when testing for main effects of treatment. To make the main effect in this interaction model interpretable as an effect at mean body mass, we transformed initial body mass to have a mean of zero. There were no significant interactions between initial body size and treatment for any other models of adult growth (SVL growth in males: initial SVL × treatment: $F_{1,96} = 1.50$, $p = 0.22$; SVL growth in females: initial SVL × treatment: $F_{1,94} = 0.29$, $p = 0.59$; mass growth in females: initial mass × treatment: $F_{1,94} = 0.08$, $p = 0.78$), so we present the results from analysis of covariance without the interaction term. Numbers of recaptured adults were low in July and October, so we restricted our analysis of growth to the interval between March and May.

We used generalized linear models with a binomial error distribution and a logit link function to test for effects of sex, treatment, and their interaction on apparent survival (observed survival, uncorrected for our estimated probability of recapture) between March and May and between March and July. One male was injured upon capture in May and had to be euthanized; this animal was censored when measuring survival between March and July. To estimate survival while also estimating and accounting for recapture probability between censuses in March, May, and July, we built Cormack-Jolly-Seber capture-mark-recapture models using the Rmark interface for the program MARK (Laake, 2013). We used a model comparison approach to test for the effects of treatment, sex, their interaction, and time on monthly survival rate and recapture rate. We tested the significance of factors using log likelihood ratio tests between full and reduced models (Supporting Information: Table S1).

In May, during the peak of the breeding season, we performed an additional experiment to test whether parasite removal increased the mating success of males. On our first recapture day, we exclusively captured males, then grouped them according to their treatment (ivermectin, control, or previously unmanipulated). Immediately prior to release, we dusted the venter of each male with nontoxic fluorescent powder (A/AX Series; Day-Glo Color Corp.) of a color unique to its treatment group (ivermectin, $n = 40$; control, $n = 37$; unmanipulated, $n = 73$). We allowed males to mate undisturbed for 2 days, then returned to the island to capture both experimental and unmanipulated females and checked their venters under UV (ultraviolet) light to detect any fluorescent powder transferred during copulation. Females with two or more different colors of powder ($n = 14$) were counted as two or more copulation events. To estimate the population mean copulation rate, we divided the total number of inferred copulation events by the total number of recaptured females. To test for an effect of parasite removal on male copulation success, we assessed whether the observed number of copulations in each group differed from null expectation using a χ^2 test with 2° of freedom. We calculated the expected number of copulations for each of the three groups of males by multiplying the relative frequencies of powdered males in each group by the total number of inferred copulation events.

2.5 | Experiment 3: Wild juveniles

Using a separate population of *A. sagrei* on a different small spoil island in the GTM NERR (29°37′58″ N, 81°12′46″ W), we captured juvenile males and females during July 2019, at which point juveniles ranged from about 1–70 days of age, depending on their hatch date (hatching typically begins in the last week of May). We gave each animal a unique toe clip, measured its SVL, and mass, then treated all individuals that weighed at least 0.5 g with an injection of either (1) our in situ gelling formulation of ivermectin ($n = 40$ males, 37 females), or (2) the gelling vehicle as a control ($n = 45$ males, 38 females). Prior to assigning treatment, we sorted individuals by SVL

and mass to create size-matched treatment groups within males (SVL: $t = 0.02$, $p = 0.99$; mass: $t = 0.35$, $p = 0.72$) and females (SVL: $t = 0.05$, $p = 0.96$; mass: $t = 0.18$, $p = 0.85$). Within 24 h, we released each treated individual at its site of capture. We resampled the island extensively in October 2019 to measure growth and survival. We tested for treatment effects on growth in SVL (mm/d) and in body mass (g/d) using linear models with fixed effects of sex and treatment plus initial size as a covariate. We did not find a significant interaction between initial body size and sex (SVL growth: $F_{1,88} = 1.39$, $p = 0.24$; Mass growth: $F_{1,88} = 0.46$, $p = 0.49$), so we analyzed males and females together in a single model with a fixed effect of sex. Further we did not find a significant interaction between initial body size and treatment (SVL growth: $F_{1,88} = 1.37$, $p = 0.25$; Mass growth: $F_{1,88} = 0.84$, $p = 0.36$), or between sex and treatment for either SVL growth ($F_{1,88} = 0.39$, $p = 0.53$), or mass gain ($F_{1,88} = 0.13$, $p = 0.71$), so we present results from models without this interaction term. We used a generalized linear model with a binomial error distribution and a logit link function to test for effects of sex, treatment, and their interaction on apparent survival of juveniles between July and October. Because we only have one resampling period, we cannot estimate recapture rate and survival independently.

3 | RESULTS

3.1 | Efficacy of parasite removal treatment

In the laboratory (Experiment 1), one injection of the gelling ivermectin formulation achieved complete removal of both gastric *Physaloptera* (Prevalence: $\chi^2_1 = 17.51$, $p < 0.0001$) and cloacal *Cyrtosomum* (Prevalence: $\chi^2_1 = 25.12$, $p < 0.0001$) in adult males (Figure 1a–b). In control males, the prevalence of *Physaloptera* infection was 53% (10 of 19), and the prevalence of *Cyrtosomum* infection was 68% (13 of 19). In the field (Experiment 2), adult males and females did not differ in the prevalence or intensity of infection by either parasite, and two injections of the gelling ivermectin formulation reduced the prevalence of *Physaloptera* infection (sex: $\chi^2_1 = 0.14$, $p = 0.70$; treatment: $\chi^2_1 = 6.70$, $p = 0.009$), the intensity of *Physaloptera* infection (sex: $\chi^2_1 = 0.12$, $p = 0.73$; treatment: $\chi^2_1 = 8.12$, $p = 0.004$), the prevalence of *Cyrtosomum* infection (sex: $\chi^2_1 = 1.79$, $p = 0.18$; treatment: $\chi^2_1 = 13.40$, $p = 0.0002$), and the intensity of *Cyrtosomum* infection (sex: $\chi^2_1 = 0.87$, $p = 0.34$; treatment: $\chi^2_1 = 3.95$, $p = 0.046$) (Figure 1c–d).

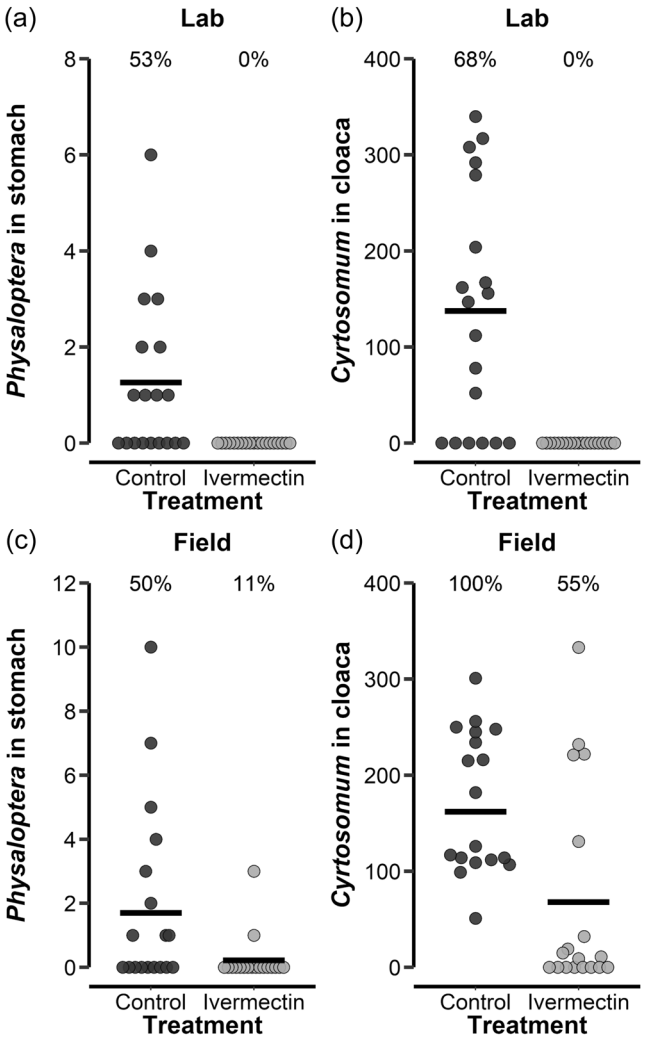

FIGURE 1 Nematode counts in adult brown anoles in the control (black) and ivermectin (gray) treatment groups based on dissections from the lab experiment on adult males (a and b) and the field experiment on adult males and females (c and d, sexes pooled). Points are jittered horizontally to avoid overplotting. Bars give the mean for each treatment group. Percentages above each column of data indicate the prevalence of infection (percentage of lizards infected). Ivermectin reduced the prevalence and intensity of both nematode parasites in both the lab and the field.

3.2 | Effects of parasite removal on performance

In the laboratory study of adult males (Experiment 1), treatment groups did not differ in sprint speed prior to treatment, and neither the injection itself nor the drug ivermectin had any short-term effect on sprint speed (time: $\chi^2_1 = 0.17$, $p = 0.67$; treatment: $\chi^2_1 = 2.13$, $p = 0.14$; time × treatment: $\chi^2_1 = 0.20$, $p = 0.65$) (Figure 2a). However, ivermectin treatment significantly increased sprint speed above that of control animals at 2 months posttreatment (time: $\chi^2_1 = 1.31$, $p = 0.25$; treatment: $\chi^2_1 = 0.56$, $p = 0.45$; time × treatment: $\chi^2_1 = 6.14$, $p = 0.01$) (Figure 2b). Further, ivermectin treatment significantly increased sprint speed at 2 months posttreatment above the pretreatment sprint speeds for the same individuals ($\chi^2_1 = 3.97$, $p = 0.046$).

3.3 | Effects of parasite removal on growth

In the laboratory study of adult males (Experiment 1), we did not detect any effects of ivermectin treatment on growth in SVL (treatment: $F_{1,35} = 0.51$, $p = 0.48$; initial SVL: $F_{1,35} = 11.12$, $p = 0.002$)

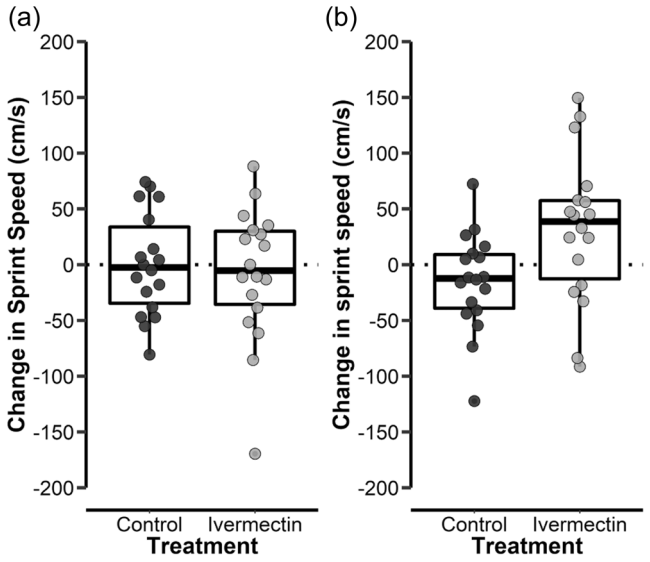

FIGURE 2 Change in sprint speed for adult males in the lab experiment from (a) 2 days pretreatment to 2 days posttreatment, or (b) 2 days pretreatment to 2 months posttreatment. Symbols represent individual males and box-and-whisker plots report medians (heavy line), upper and lower quartiles (boxes), and ranges (whiskers) in each treatment group. There was no short-term effect of either the vehicle (control) or the drug (ivermectin) on sprint speed (a). Parasite removal significantly increased sprint speed at 2 months posttreatment. This increase was significantly greater than the change in sprint speed for the control group (b). For simplicity, these figures depict change in sprint speed, whereas the analysis used a repeated-measures, mixed-effect linear model including both pre- and posttreatment measures.

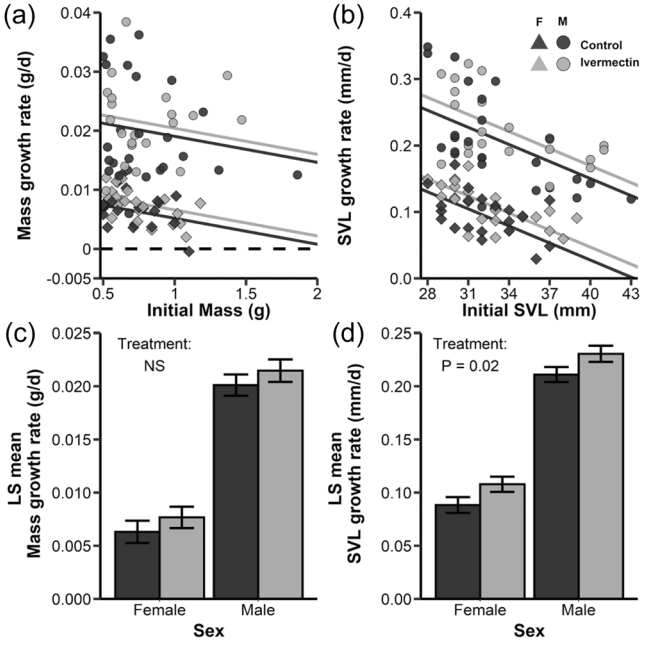

FIGURE 3 Effects of parasite removal on growth rate from July to October 2019 for body mass (g/d) (a–c) and SVL (mm/d) (b–d) for juvenile males and females. a and b show individual growth with best-fit least squares regression lines for each group. c and d give estimated marginal means (±1SE) for each sex and treatment group, as well as *p* values for the main effect of treatment from models with sex and initial size (mass or SVL) as a covariate. There is a significant effect of treatment on SVL growth rate in juveniles (b–d). SVL, snout-vent length.

or growth in mass (treatment: $F_{1,35} = 0.60$, $p = 0.44$; initial mass: $F_{1,35} = 13.16$, $p = 0.009$). In the field study of adult males (Experiment 2), we did not detect any effects of ivermectin treatment on growth in SVL (treatment: $F_{1,97} = 0.06$, $p = 0.79$; initial SVL: $F_{1,97} = 112.41$, $p < 0.0001$) or growth in mass (treatment: $F_{1,96} = 1.57$, $p = 0.21$; initial mass: $F_{1,96} = 0.029$, $p = 0.86$; treatment × initial mass: $F_{1,96} = 8.01$, $p = 0.006$) (Supporting Information: Figure S1). The significant interaction was included because males treated with ivermectin exhibited the expected negative relationship between initial body mass and growth whereas control males did not (Supporting Information: Figure S1). In the field study of adult females (Experiment 2), we did not detect any effects of ivermectin treatment on growth in SVL (treatment: $F_{1,95} = 0.05$, $p = 0.82$; initial SVL: $F_{1,95} = 118.27$, $p < 0.0001$) or growth in mass (treatment: $F_{1,95} = 0.02$, $p = 0.88$; initial SVL: $F_{1,95} = 75.59$, $p < 0.0001$) (Supporting Information: Figure S1). For juveniles in the field (Experiment 3), males grew more quickly than females and treatment with ivermectin significantly increased growth in SVL in both sexes (treatment: $F_{1,89} = 5.36$, $p = 0.02$; initial SVL: $F_{1,89} = 49.37$, $p < 0.0001$; sex: $F_{1,89} = 207.67$, $p < 0.0001$), but ivermectin did not significantly increase mass gain (treatment: $F_{1,89} = 1.31$, $p = 0.25$; initial mass: $F_{1,89} = 3.30$, $p = 0.07$; sex: $F_{1,89} = 132.38$, $p < 0.0001$) (Figure 3a–d).

3.4 | Effects of parasite removal on survival

For adults in the field (Experiment 2), apparent survival was similar between sexes and treatment groups from March to May (sex: $\chi^2_1 = 0.24$, $p = 0.61$; treatment: $\chi^2_1 = 0.40$, $p = 0.52$; sex × treatment: $\chi^2_1 = 0.64$, $p = 0.42$) and from March to July (sex: $\chi^2_1 = 2.01$, $p = 0.16$; treatment: $\chi^2_1 = 0.22$, $p = 0.63$; sex × treatment: $\chi^2_1 = 0.04$, $p = 0.83$) (Table 1). Our best-supported CJS capture-mark-recapture model had a single recapture rate (0.84) that did not vary across treatment groups or sexes, and a time-varying survival rate (survival probability per month) (March to May: 0.77, May to July: 0.69; $\chi^2_1 = 4.15$, $p = 0.04$) (Supporting Information: Table S1). We found no evidence for effects of treatment or sex on either survival (sex: $\chi^2_1 = 1.65$, $p = 0.20$, treatment: $\chi^2_1 = 0.39$, $p = 0.53$, sex × treatment: $\chi^2_1 = 0.20$, $p = 0.65$) or recapture probability (sex: $\chi^2_1 = 0.46$, $p = 0.49$, treatment: $\chi^2_1 = 0.28$, $p = 0.60$, sex × treatment: $\chi^2_1 = 0.51$, $p = 0.46$). For juveniles in the field (Experiment 3), apparent survival was similar between the sexes and between treatment groups (sex: $\chi^2_1 = 1.18$, $p = 0.27$; treatment: $\chi^2_1 = 0.14$, $p = 0.70$; sex × treatment: $\chi^2_1 = 0.69$, $p = 0.41$) (Table 1).

3.5 | Effects of parasite removal on mating success

We detected 77 copulations across 272 recaptured females (0.28 mating frequency, $n = 63$ females copulated at least once). The

TABLE 1 Summary of survival estimates across ages, time intervals, sexes, and treatment groups for the two field studies involving adults (Experiment 2) and juveniles (Experiment 3)

Age	Interval	Sex	Treatment	Alive	Dead	Survival	1 SE
Adult	March to May	Male	Control	47	40	0.54	0.053
			Ivermectin	56	35	0.62	0.051
		Female	Control	53	34	0.61	0.052
			Ivermectin	54	36	0.60	0.052
Adult	March to July	Male	Control	15	72	0.17	0.041
			Ivermectin	18	72	0.20	0.042
		Female	Control	21	66	0.24	0.045
			Ivermectin	23	67	0.26	0.046
Juvenile	July to October	Male	Control	25	20	0.55	0.074
			Ivermectin	21	19	0.54	0.077
		Female	Control	22	16	0.58	0.080
			Ivermectin	25	12	0.68	0.077

Note: The standard error was calculated as $\sqrt{\frac{probability\ of\ survival \times probablity\ of\ mortality}{n}}$.

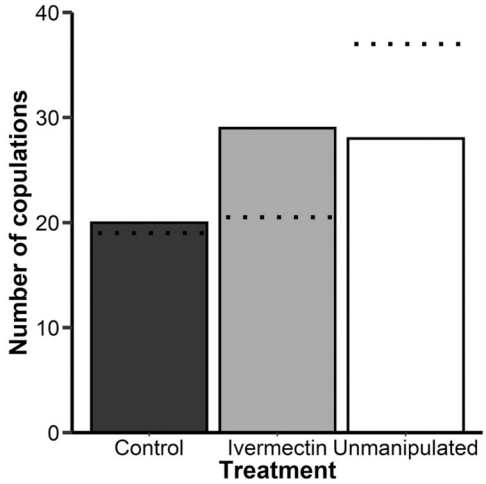

FIGURE 4 Bars give the total number of copulations of adult males in each treatment group, based on the number of females recaptured with fluorescent powder corresponding to that group. Dashed lines give the expected number of copulations (under the null hypothesis of no difference between groups) based on the total number of powdered males in that group and the total number of copulations detected across all groups. Ivermectin males had more copulations than expected while unmanipulated males had fewer copulations than expected.

number of copulations attributed to each of the three male groups differed slightly from the null expectation based on their frequencies in the population ($\chi^2_1 = 5.94$, $p = 0.051$), such that more copulations were attributed to males treated with ivermectin than expected, fewer copulations were attributed to unmanipulated males than expected, and the number of copulations attributed to control males was similar to the null expectation (Figure 4).

4 | DISCUSSION

We found that removal of nematode parasites from anole hosts with a novel, in situ gelling formulation of ivermectin increased the sprint speed and mating success of adult males and also increased growth in length for juveniles of both sexes. We interpret these results as evidence for costs of parasitism with respect to performance, juvenile growth, and one aspect of reproductive fitness, although we found no evidence for any effects of parasite removal on the survival of juveniles or adults of either sex. We did not measure reproductive success directly, but it is possible that the positive effects of parasite removal on juvenile growth that we observed could indirectly enhance both male mating success and female fecundity by increasing adult body size, which correlates with reproductive success (Duryea et al., 2016; Kamath & Losos, 2018). Non-avian reptiles are poorly represented in experimental parasite removal and infection studies (Main & Bull, 2000; Oppliger et al., 1999), particularly for tests of the fitness costs of parasitism (Pedersen & Fenton, 2015). Expanding the taxonomic breadth of studies that experimentally test for costs of parasitism will allow for better comparative tests of theories relating pace-of-life and metabolic rate with immune strategy and parasite defense (K. A. Lee, 2006; Sánchez et al., 2018; Sandmeier & Tracey, 2014).

We found that the small, sexually transmitted nematode *C. penneri* occurred at a higher prevalence and intensity of infection than the large, trophically transmitted nematode *Physaloptera* sp., and we found no evidence for a sex difference in the prevalence or intensity of infection by either parasite. These patterns are largely consistent with other studies of parasitism in the brown anole (Goldberg & Bursey, 2000; Goldberg et al., 1994; Norval et al., 2011; Reedy et al., 2016). Although ivermectin eliminated both parasites in the laboratory, it was somewhat more effective at lowering the

prevalence and intensity of infection by *Physaloptera* compared to *Cyrtosomum* in the field (Figure 1), perhaps because of the high prevalence of *Cyrtosomum* in the untreated portion of the adult population (100%) and the high likelihood of reinfection during mating (Langford et al., 2013), which appears to happen frequently based on our fluorescent powdering data (23% of females had mated at least once within several days of their capture). Although our experimental design does not allow us to disentangle the independent effects of *Physaloptera* and *Cyrtosomum* on anoles, the detrimental effects on growth, performance, and mating success that we observed in control animals may be largely due to *Physaloptera*. Nematodes in the genus *Physaloptera* embed in the gastric epithelium and feed on gastric mucosa and blood, which causes tissue damage in the mucosal lining of the stomach, inflammation, and invasion of the gastric tissue with immune cells (Hoseini et al., 2014; Naem et al., 2006). By contrast, *C. penneri* is a sexually transmitted nematode infecting the rectum of its hosts, where it may feed primarily on feces rather than host tissue. In *Sceloporus* lizards, infection by *C. penneri* was not associated with inflammation of the rectum or tissue damage, but its potential pathological effects have not been well studied (Pearce, 1973). Therefore, any costs stemming from *C. penneri* may reflect collateral damage from immune activation and associated tissue inflammation, rather than direct damage to host tissues by the parasite.

Neither the gelling injection vehicle nor the ivermectin treatment had any detrimental short-term (2 days) effect on sprint speed in adult males. However, 2 months posttreatment, males given ivermectin were free of both species of nematode parasite and had significantly increased sprint speed, whereas males given the control vehicle showed no change in sprint speed. This increase in performance of the ivermectin group is likely due to the concomitant reduction in parasitism and not due to ivermectin itself, because ivermectin tends to depress the central nervous system and reduce motor coordination (Moreira et al., 2017). A meta-analysis across host and parasite taxa showed that, on average, parasitism reduces host performance, and that endurance measures tend to be reduced more than speed (McElroy & de Buron, 2014). However, of the previous studies investigating the relationship between parasitism and running performance in lizards, none involved nematodes, and all were observational. Reductions in performance were associated with parasitism by apicomplexans (haemogregarines, *Plasmodium*), but not by mites or ticks (Ekner-Grzyb et al., 2013; Garrido & Pérez-Mellado, 2014; Main & Bull, 2000; Oppliger et al., 1996; Schall et al., 1982). One mechanism through which parasitism may affect performance is by compromising the ability of blood to effectively transport nutrients and oxygen to muscles under stress. In the lizard *Lacerta vivipara* hemogregarine parasitism is associated with reduced hemoglobin, increased numbers of immature red blood cells, and reduced mean sprint speed (Oppliger et al., 1996). Although this mechanism is intuitive for hemogregarines and other blood parasites, there is also some evidence that *Physaloptera* infections can cause anemia (Al-Obaidi, 2012; Lértora et al., 2016). Protein loss from healing damaged gastric epithelium due to parasitic nematodes also leads to a decrease in enzymes and myoglobin used for oxygen transport during muscle activity (Fuge et al., 1968). Additionally, the energetic cost of mounting an immune response may trade off with performance. Lizards treated with the bacterial cell wall component lipopolysaccharide (LPS) to induce an immune response have reduced sprint speed, possibly due to altered energy balance favoring immune response (Hudson et al., 2021; Zamora-Camacho et al., 2014). However, in mammals the antimicrobial immune response induced by LPS is distinct from the response against macroparasites and involves proinflammatory type 1 responses (Annunziato et al., 2014; Ashley et al., 2012; Spellberg & Edwards, 2001).

We found no effects of parasites on growth in either length or mass of adult anoles in either captivity or the wild (Supporting Information: Figure S1.). Although growth is indeterminate in *A. sagrei*, it decreases asymptotically with body size. Therefore, adults that are not rapidly growing may not face growth costs from parasitism, or the similar costs may be more difficult to detect than in juveniles, where we found small but significant effects of parasite removal on growth (Figure 3a–d). When expressed relative to the average initial SVL for each sex, individuals treated with ivermectin grew 0.019 mm more per day than control individuals, corresponding to a 20% increase in growth rate of females and a 10% increase in growth rate of males. These growth costs may have important consequences for fitness as adults, given that body size at the beginning of the breeding season is positively associated with offspring production that year (Duryea et al., 2016).

Growth costs of parasitism have been found in a variety of taxa, including birds (Fassbinder-Orth et al., 2018; O'Brien & Dawson, 2007), amphibians (Finnerty et al., 2017), mammals (Sacks & Blejwas, 2000; Stien et al., 2002), fish (Hansen et al., 2006; Hoffnagle et al., 2006), arthropods (Botto-Mahan et al., 2017; Polak, 1998), annelids (Field & Michiels, 2005), and molluscs (O'Connell-Milne et al., 2016). These studies also encompass a broad range of parasite types, suggesting that growth reduction is a general cost of parasitism. However, many other studies have found no effect of parasitism on growth or body size (Hillegass et al., 2010; Reed et al., 2012; Roznik et al., 2020; Tompkins et al., 1999), and in some systems, parasites are known to increase the body size of their hosts, though usually at the expense of reproduction (Ebert et al., 2004; Sorensen & Minchella, 2001). Variability in the reported growth costs of parasitism is likely due in part to variation in study methodology (e.g., observational study, experimental infection, experimental removal, effectiveness of removal, reinfection rate, length of study, field vs. laboratory, measure of body size) as well as the specifics of the host–parasite system (variation in parasite virulence and host tolerance), and the time period of the study relative to host lifespan.

Parasites are likely more costly when resources are limited or during energetically demanding periods of the host life cycle, such as during rapid growth periods preceding maturity or during reproduction (Albery et al., 2021; Bruns et al., 2017; Francis, 1961). Because parasites directly utilize host resources, they potentially decrease the total amount of resources that hosts can allocate toward growth, somatic maintenance, and reproduction (de Jong, 1993; Sheldon &

Verhulst, 1996; van Noordwijk & de Jong, 1986; Zera & Harshman, 2001). This reduction may be especially detrimental for juveniles that are in a period of rapid, energetically demanding growth. In the chipmunk (*Tamias striatus*), botfly parasitism depresses growth and increases resting metabolic rate in juveniles, but not in adults (Careau et al., 2010). Juvenile chickens that are experimentally infected with the nematode *Ascaridia galli* have decreased mass gain and increased mortality, and this effect is greatest in younger chicks (Ackert & Herrick, 1928). In terms of growth and survival, juveniles may be less likely to compensate for the increased energetic demands imposed by parasitism because their foraging opportunities are potentially more limited than those of adults, and because they cannot divert energy from reproduction. In addition to shrinking the pool of available resources through direct consumption, parasites may reduce the size of the resource pool that can be allocated to growth, given that many hosts show a depressed feeding rate upon infection (Arneberg et al., 1996; Adamo 2005, Finnerty et al., 2017; Sargent et al., 2014). Further, parasites may render foraging more energetically costly for their hosts. In European shags (*Phalacrocorax aristotelis*), there is a positive association between parasite burden and energy expended on foraging flights (Hicks et al., 2018). The energetic cost of mounting an immune response to an active nematode infection may also trade off against growth, and growth costs of immune activation have been seen in many organisms (Bascuñán-García et al., 2010; Bonneaud et al., 2016; Demas et al., 2011; Devevey et al. 2008; Uller et al., 2006).

Male anoles with their parasites removed had greater mating success than expected, while the mating success of control and unmanipulated males was at or below expectation, as inferred through transfer of florescent powder to the female venter, which presumably occurred during copulation. Parasite removal may have increased the competitive ability of males in intrasexual interactions, thereby increasing their access to females (Gómez-Llano et al., 2020). Additionally, parasite removal could have increased the number of encounters a male has with females. Tick removal increases range size in the common pheasant (*Phasianus colchicus*), and thereby increases the likelihood of males acquiring mates (Hoodless et al., 2002). In *A. sagrei*, male home range size is positively associated with female encounter rate, and the proportion of a female's offspring sired (Kamath & Losos, 2018). Parasites have been shown to reduce reproductive success and offspring production in other species (Newey & Thirgood, 2004; Patterson et al., 2013; Worden et al., 2000; however, see Raveh et al., 2011), but further work involving genetic parentage assignment will be necessary to determine whether the effects of parasite removal on juvenile growth and male mating success that we observed translate into increased reproductive success.

Across a variety of host–parasite systems, parasites usually reduce host survival (Robar et al., 2010; Wittman & Cox, 2021). Therefore, it is somewhat surprising that we found no effects of nematode parasitism on survival in juvenile or adult brown anoles. Although experimental data for non-avian reptiles are scarce, observational studies of mite and hemogregarine parasitism have shown little evidence for survival costs in this group (Bonneaud et al., 2017; Brown et al., 2006; Paterson & Blouin-Demers, 2020; Sorci et al., 1996; but see Shaner et al., 2013), suggesting some level of mortality tolerance to parasites. The immune strategy of short-lived ectotherms may rely more heavily on constitutive rather than induced aspects of the immune system, or on tolerance (reducing the fitness costs of a given parasite burden) than on resistance (reducing the parasite burden) than long-lived ectotherms or endotherms with a comparable pace-of-life (Palacios et al., 2010; Previtali et al., 2012; Råberg et al., 2009; Sandmeier & Tracy, 2014). Alternatively, mortality may be largely stochastic in this system, such that any effects of parasites on survival are obscured by other sources of mortality, such as predation, which may occur randomly with respect to parasite load.

5 | CONCLUSION

Our extended-release formulation of ivermectin safely and effectively decreased nematode parasitism of brown anole hosts in the laboratory and field, suggesting that this new technique has promise for use in field studies of parasitism in a variety of vertebrate host species. Our experimental results indicate that nematode parasites can impose costs in terms of growth, performance, and mating success, but we see no evidence for a survival cost of parasitism in *A. sagrei*. The lack of a survival cost is somewhat surprising given substantial evidence for this cost in a variety of other vertebrate and invertebrate hosts, though lizards are poorly represented in experimental tests for survival costs of parasitism in the wild. Given that our results suggest a variety of modest costs of parasitism that may collectively influence lifetime reproductive success, future studies should seek to measure survival, mating success, and total lifetime reproductive success to understand how parasites directly and indirectly affect host fitness.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

DATA AVAILABILITY STATEMENT

The data used in all analysis is publicly available through the Open Science Foundation, and can be accessed through the following link, https://doi.org/10.17605/OSF.IO/JQ8UM

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