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Local adaptation of a parasite to solar radiation impacts disease transmission potential, spore yield, and host fecundity*

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Environmentally transmitted parasites spend time in the abiotic environment, where they are subjected to a variety of stressors. Learning how they face this challenge is essential if we are to understand how host–parasite interactions may vary across environmental gradients. We used a zooplankton–bacteria host–parasite system where availability of sunlight (solar radiation) influences disease dynamics to look for evidence of parasite local adaptation to sunlight exposure. We also examined how variation in sunlight tolerance among parasite strains impacted host reproduction. Parasite strains collected from clearer lakes (with greater sunlight penetration) were most tolerant of the negative impacts of sunlight exposure, suggesting local adaptation to sunlight conditions. This adaptation came with both a cost and a benefit for parasites: parasite strains from clearer lakes produced relatively fewer transmission stages (spores) but these strains were more infective. After experimental sunlight exposure, the most sunlight-tolerant parasite strains reduced host fecundity just as much as spores that were never exposed to sunlight. Sunlight availability varies greatly among lakes around the world. Our results suggest that the selective pressure sunlight exposure exerts on parasites may impact both parasite and host fitness, potentially driving variation in disease epidemics and host population dynamics across sunlight availability gradients.

KEY WORDS: ***Daphnia dentifera*, disease, fitness trade-off, local adaptation, *Pasteuria ramosa*, solar radiation.**

The abiotic environment can strongly impact host–parasite interactions and resulting disease outcomes (Wolinska and King 2009; Mostowy and Engelstädter 2011; Rogalski et al. 2017). For environmentally transmitted parasites, environmental stress may play a particularly important role: to infect a new host, transmission stages must spend time in the environment, where they are subjected to abiotic stressors. Moreover, anthropogenic environmental change is altering the nature and intensity of stressors faced by environmentally transmitted parasites, in some cases quite rapidly (Tucker and Williamson 2011; Lal et al. 2013). Environmental stress can drive adaptation in populations, influencing both their physiology and their interactions with other organisms (Bijlsma and Loeschcke 2005), and the expectation is that parasites should be able to adapt rapidly to environmental change (Cable et al. 2017). However, free-living stages of parasites can be particularly vulnerable to environmental stressors and may be limited in their ability to adapt to changing conditions (Phillips et al. 2017). Furthermore, in the face of significant selection pressure exerted by hosts it may be difficult for parasites to adapt to the additional stress exerted by the environment. Investigating the degree to which environmentally transmitted parasites can adapt to environmental stressors and how that influences their impacts on hosts may allow us to better predict how host–parasite interactions will change in a changing world.

One environmental stressor that can strongly influence parasite fitness is exposure to sunlight, particularly ultraviolet radiation (UVR) (in this manuscript sunlight is synonymous with solar radiation, inclusive of infrared, visible and ultraviolet radiation). Exposure to the shortest wavelengths of UVR that reach the Earth's surface, UV-B, promotes molecular and cellular damage

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(Rastogi et al. 2010) and can even be lethal to organisms (Vega and Pizarro 2000; Onzo et al. 2010). Navigating this stress likely shaped the ecology and evolution of life throughout its history (Hessen 2008). Parasites can be particularly sensitive to UVR—often more so than their hosts—such that sunlight exposure can dampen disease. For example, UVR can prevent coral bleaching by killing the UVR-sensitive *Vibrio shiloi* pathogen of corals in shallow, light-exposed reefs (Fine et al. 2007). In addition, municipal water managers take advantage of sunlight sensitivity in microorganisms by treating drinking water with UVR to eliminate viral, bacterial, and protistan human pathogens (King et al. 2008).

Some organisms have responded to UVR stress by avoiding or tolerating exposure (Hansson and Hylander 2009; Rastogi et al. 2010; Rautio and Tartarotti 2010) or by repairing the damage caused by UVR after it occurs (Rastogi et al. 2010; Miner et al. 2015). However, these adaptations can come at a cost. For example, although pigmentation can protect organisms from UVR damage (Hessen 1996), it also makes them more susceptible to visual predators (Hansson 2000). In addition, production of photoprotective pigments is associated with metabolic and fitness costs (Hessen 1996). Such trade-offs may explain why UVR avoidance and tolerance traits are more often found in populations experiencing greater exposure to sunlight (Stutzman 1999; Tartarotti et al. 2001; Miner et al. 2015).

Exposure to sunlight varies across space and over time. On larger spatial scales, high elevation as well as lower latitude locations experience relatively higher solar radiation intensity. Moreover, characteristics of the atmosphere, such as dust, smog, and cloud cover can affect the amount of sunlight that reaches the Earth's surface (Madronich et al. 1998). In aquatic systems, solar radiation intensity attenuates with depth, and the degree to which sunlight penetrates the water column depends in large part on differences in concentrations of dissolved organic carbon (DOC), which absorbs solar radiation (Morris et al. 1995; Rae et al. 2001). Land development and more frequent storms associated with climate change can drive lake browning, where increased levels of DOC can absorb a greater fraction of UVR (Williamson et al. 2016; Kritzberg 2017). UVR exposure also varies seasonally, with incident sunlight strength at its maximum in the summer. In addition, atmospheric ozone depletion drove greater UVR availability, though the success of the Montreal protocol prevented large-scale depletion of stratospheric ozone and associated dramatic increases in UVR (Williamson et al. 2014). Thus, both natural variation and human activities strongly influence light regimes, with some habitats receiving more sunlight, and some receiving less (Williamson et al. 2014).

Spatial and temporal variation in lake sunlight availability can affect the timing and size of disease epidemics of light-sensitive parasites (Overholt et al. 2012; Shaw 2019). Thus, we might expect the relative strength of the impacts of solar radiation on parasites and their interactions with hosts to vary among habitats. Here, we use a zooplankton–bacterial host–parasite system to examine how variation in lake sunlight environments affects the fitness of the light sensitive parasite as well as how the parasite affects its host. Our focal host is *Daphnia dentifera* Forbes (Hebert 1995), and our focal parasite is the environmentally transmitted, sterilizing, obligate killer, *Pasteuria ramosa* (Ebert et al. 1996). Although *Daphnia* are affected by UVR exposure (Rautio and Tartarotti 2010), they appear to be modestly impacted at levels of exposure that can be lethal to their parasites (Overholt et al. 2012). Previous work found that *Pasteuria* spores are sensitive to ambient solar radiation, including PAR and UVR (Shaw 2019; Overholt et al. 2020) but did not consider variation among populations in sunlight sensitivity.

Pasteuria epidemics occur in lakes that span a gradient of sunlight availability, though these epidemics tend to be smaller in clearer lakes (Shaw 2019). We took advantage of this natural variation by conducting a common garden experiment to examine whether *Pasteuria* populations are locally adapted to lake sunlight regimes. After finding evidence that *Pasteuria* populations from clearer lakes are less affected by sunlight exposure than strains from darker lakes, we examined whether this adaptation came at a cost to the parasites, in either their ability to infect *Daphnia* hosts or their reproduction within hosts postinfection. In addition, we examined how variation in parasite fitness in sunlight-exposed and unexposed conditions affected host fecundity.

Materials and Methods

HOST AND PARASITE

The host species in our study, *D. dentifera* Forbes, is a dominant freshwater microcrustacean of lakes in the Midwestern United States (Hebert 1995). *Daphnia* are cyclically parthenogenic, which allows the maintenance of clonal lineages in the laboratory. *Daphnia* play an important role in lake food webs as keystone herbivores of phytoplankton (Miner et al. 2012). *Daphnia dentifera* commonly suffer infection by our focal parasite, *P. ramosa*, a bacterial endoparasite in the *Bacillus* clade (Ebert et al. 1996). *Daphnia* encounter *Pasteuria* spores during filter feeding. Once consumed, *Pasteuria* spores bind to and then cross the host's gut epithelium and then replicate in the hemocoel (Ebert et al. 1996). *Pasteuria* infection results in host castration; although infected hosts may produce offspring early in the infection process and may also experience "castration relief" during late infection stages, many infected hosts never reproduce (Auld et al. 2012; Vale and Little 2012; Ben-Ami and Routtu 2013). In contrast, healthy mature *Daphnia* typically produce a clutch of offspring

every three to four days when cultured at 20°C. *Pasteuria* spores become mature inside the host, beginning around 15–20 days and are released upon host death (Ebert et al. 2016). Mature spores are also infectious if the host suffers early mortality, for example via sloppy predation by *Chaoborus* (Auld et al. 2014a). Significant genetic diversity has been observed within *Pasteuria* populations, likely driven by substantial rates of recombination (Andras and Ebert 2013), and we have found significant phenotypic diversity of *Pasteuria* within and among lakes (Shaw 2019). Rapid change in key traits related to infectivity, spore production, and host genotype specificity has been observed in *Pasteuria* populations both in the field and laboratory, over timespans as short as a few years to even a few months (Decaestecker et al. 2007; Auld et al. 2014a, 2014b). In addition, *Pasteuria* spores can remain viable for years in the lake environment before being consumed by a new host (Decaestecker et al. 2004, 2007). These conditions set the stage for the possibility that *Pasteuria* may adapt in response to environmental stress.

COMMON GARDEN EXPERIMENT

Parasite spore collection and experimental sunlight exposure

We collected *D. dentifera* individuals that were heavily infected with *P. ramosa* from six lakes in southeastern Michigan and one lake in Indiana between mid-August and mid-September 2017. The study lakes span a gradient of sunlight availability (Table S1) and represent a subset of a larger set of lakes that are regularly monitored for disease dynamics in the summer and fall (Hall et al. 2011; Duffy et al. 2015). *Daphnia* samples were collected by pooling three vertical tows of the entire water column in the deep basin of the lake, using a 153 μm pore size Wisconsin net. We isolated *D. dentifera* individuals infected with *P. ramosa* by examining collected zooplankton under a dissecting microscope at 50× magnification.

We pooled groups of 20 *Pasteuria*-infected hosts from a given lake (hereafter a "strain") and stored them in 250 μL filtered lake water in small plastic tubes in the dark at 4°C. We collected 1–5 *Pasteuria* strains (i.e., 20–100 *Daphnia* infected with *Pasteuria*, separated into one to five separate tubes) from each lake (Table S2). On September 21, 2017, we homogenized each strain by pulverizing the infected hosts with a motorized pestle for 2 minutes. We then split the spore slurry for each individual strain evenly into two quartz vials (0.8 mL capacity). One vial was clear, allowing sunlight to penetrate ("sunlight-exposure treatment"). The second vial was covered in dark plastic to prevent any sunlight exposure ("control treatment"). The next morning, we suspended the clear and darkened vials of *Pasteuria* spores at a depth of 0.5 m below the surface of North Lake (the clearest study lake), leaving the vials suspended in the lake for four days.

Upon retrieving the vials, we transferred the *Pasteuria* spores from each vial into a small plastic tube, estimated the spore density for each strain × treatment using a Neubauer chamber cell (four counts per replicate), and stored the spores in the dark at −20°C for six months prior to the experiment. *Pasteuria* spores retain infectivity for at least 1 year when stored at −20°C (Duffy and Hunsberger 2019).

Laboratory infection assays

We conducted a common garden infection assay to (1) test our hypothesis that *Pasteuria* from clearer lakes would be relatively less impacted by exposure to sunlight, (2) determine whether *Pasteuria* populations from lakes spanning a sunlight availability gradient varied in key traits, and (3) examine how variation in *Pasteuria* transmission potential impacted *Daphnia* host reproduction.

Before beginning our common garden experiment, we maintained a single clonal genotype of *D. dentifera* under standardized conditions for three generations to minimize variation in host condition (Text S1). This clone originated from one of our study lakes (Midland Lake) and was chosen based on pilot studies showing it to be a good genetic match with *Pasteuria* from most of the study lakes. We placed 48- to 72-hour-old *Daphnia* individually into a well with 2 mL filtered lake water and 10,000 spores of a *Pasteuria* strain from either the sunlight-exposure or control treatment. We exposed 10–12 host replicates per parasite strain × treatment (based on availability of spores), for a total of 332 *Daphnia* across 14 strains from seven lakes.

We incubated the *Daphnia* for 48 hours and fed them 2×10^4 cells of *Ankistrodesmus* per day. This is half the typical food density, which promotes uptake of parasite spores by the host. The two-day incubations took place inside a darkened container to prevent light exposure from impacting the *Pasteuria* spores before infection (Text S2). We then transferred each *Daphnia* to individual beakers filled with 35 mL of spore-free filtered lake water. Hereafter, the *Daphnia* had no further opportunities for infection, as individuals were isolated in separate beakers, in spore-free water. We fed each *Daphnia* 1×10^6 cells of *Ankistrodesmus* four times a week and changed the water twice weekly. At each water change, we counted and removed any *Daphnia* offspring produced.

We ended the experiment 24 days after infection. This timing allowed the *Pasteuria* infections to progress to maturity, without killing many of the *Daphnia* hosts, ensuring the hosts' carapaces were intact and allowing for an accurate count of *Pasteuria* spore production. This approach mimics an environment in which a predator kills an infected host, which is likely given mortality rates in lakes and increased predation on infected hosts (Duffy and Hall 2008; Auld et al. 2014a). We assessed infection status by examining each *Daphnia* under a dissecting microscope at 50×

magnification. In cases where infection status was unclear, we examined *Daphnia* under a compound microscope at 400× magnification. To quantify spore yields (number of spores produced per host), we placed each infected *Daphnia* in 100 μL filtered lake water in a plastic microcentrifuge tube, homogenized the host using a motorized pestle, and counted four replicate subsamples with a Neubauer counting chamber at 400× magnification.

Measures of parasite transmission potential and host reproduction

Transmission potential is a component of parasite fitness that incorporates the likelihood of infection and the spore yield given infection. We calculated infectivity for each parasite strain from the sunlight-exposure and control treatments as the proportion of exposed *Daphnia* individuals that were infected by the end of the trial. Out of 332 replicates, 18 *Daphnia* that died when it was too early to determine infection status and five that were males were excluded from analysis. We calculated transmission potential by multiplying infectivity (number of infected hosts/number of exposed hosts) by mean spore yield (number of spores/infected host) for each parasite strain × treatment combination (Auld et al. 2014a). Thus, transmission potential gives the number of spores produced per exposed host individual. We estimated host fecundity by summing the total number of *Daphnia* offspring produced per individual by the end of the trial (through 24 days postinfection).

There was a poor genetic match between *Pasteuria* strains from Walsh Lake and North Lake and the *Daphnia* clone used in the infection assays. No *Daphnia* were infected by *Pasteuria* from Walsh Lake and only a single *Daphnia* individual was infected from North Lake *Pasteuria.* These two lakes were excluded from further analysis. For comparison, infectivity in the control treatment for the other five lakes ranged from 50% to 92% (with 0–58% infectivity in the sunlight-exposure treatment).

STATISTICAL ANALYSES

Evidence of local adaptation to light in Pasteuria

All statistical analyses were conducted using the statistical program R version 3.5.2 (R Core Team 2016). We used general linear mixed modeling (GLMM) to evaluate the extent to which sunlight exposure in the common garden experiment impacted *Pasteuria* transmission potential and to see if this impact varied according to the UVR environment (absorption coefficient a_{d320}, an index of UVR availability) in the study lake from which the *Pasteuria* strains were collected (i.e., whether there was a genotype by environment interaction). We included transmission potential as the response variable and a_{d320}, treatment (sunlight-exposed or control), and their interaction as fixed effects. We included the random effect of parasite strain to account for lack of independence in the data for host replicates exposed to parasite strains from the same lake. We used Akaike information criterion (AIC) to compare the saturated model with nested simpler models containing fewer fixed effects to evaluate the significance of the treatment × a_{d320} interaction (i.e., G × E) as well as the sunlight-exposure treatment (Table S3). We used the lme4 package to conduct GLMM analyses (Bates et al. 2015).

Parasite infectivity–spore yield trade-off

Parasite transmission potential depends on both infectivity (likelihood of infecting a host) and spore yield (replication within the host after infection). We used a mixed effects hurdle model to test whether either of these aspects of parasite fitness differed among *Pasteuria* strains collected from our study lakes that span a UVR availability gradient. The first part of the hurdle model included infection status of *Daphnia* individuals exposed to a given strain as the binomial response variable, and the second part evaluated spore yield of individual infected hosts as the response variable (with a zero-truncated negative binomial distribution). For both stages of the hurdle model we included a_{d320}, treatment, and their interaction as fixed effects. All models included parasite strain as a random effect. We evaluated whether infectivity or spore yield was explained by UVR penetration (a_{d320}) in the home lake environment, the sunlight-exposure treatment, or their interaction by comparing AIC of the saturated model with nested simpler models containing fewer fixed effects (Table S4). We used the glmmTMB package (Brooks et al. 2017) to conduct the hurdle model analysis.

Impact of parasite transmission potential on host reproduction

Using a GLMM with the lme4 package, we evaluated how variation in parasite transmission potential influenced mean *Daphnia* host fecundity, which considers reproduction at the population level (mean total number of offspring/replicate host for a given parasite strain × treatment). Mean host fecundity was the response variable, and transmission potential (mean spore yield weighted by infectivity of spores), treatment (sunlight-exposed or control), and their interaction were fixed effects. The models included parasite strain nested within lake as a random effect. We compared AIC values of the saturated model with simpler models including fewer predictors to evaluate the significance of each fixed effect (Table S5).

Results

PARASITE LOCAL ADAPTATION

Sunlight exposure strongly influenced *Pasteuria* transmission potential ($\chi^2 = 17.56$, $P < 0.001$), causing on average a 70% decrease in spore yield per exposed host (an average decrease of 88,043 spores/exposed host ± 16,986 SE). Most notably, the

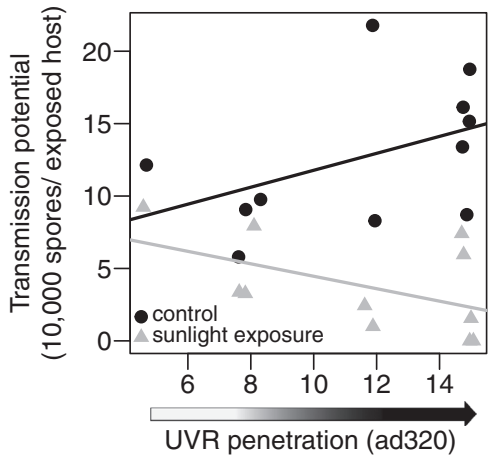

Figure 1. Sunlight exposure reduced parasite transmission potential, particularly for strains that originated from darker lakes. Lower a_{d320} values are relatively clearer lakes, with greater ultraviolet radiation (UVR) penetration. Each point represents the transmission potential for a parasite strain × treatment combination from a given lake (1–5 strain replicates per lake × treatment combination). Black circles represent the control treatment, and gray triangles represent the sunlight-exposed treatment. Data are slightly jittered along the *x* axis to show overlapping data points.

impact of sunlight exposure on transmission potential differed among *Pasteuria* strains according to the UVR environment of the lake of origin (significant a_{d320} × treatment interaction: $\chi^2 = 5.30$, $P = 0.021$). Sunlight exposure most strongly decreased transmission potential of *Pasteuria* strains from darker lakes (Fig. 1).

PARASITE TRADE-OFFS

Pasteuria strains from darker lakes (higher a_{d320}) were less likely to infect *Daphnia* than strains from clearer lakes (Fig. 2A; $\chi^2 = 11.41$, $P < 0.001$). Infection was much less likely with *Pasteuria* from the sunlight-exposed treatment (Fig. 2B, $\chi^2 = 74.02$, $P < 0.001$), but there was not a significant interaction between a_{d320} and treatment ($\chi^2 = 1.24$, $P = 0.27$). Thus, the pattern of lower infectivity of *Pasteuria* strains from darker lakes was consistent across the sunlight-exposure and control treatments.

For those *Daphnia* that were successfully infected, spore yield was higher for *Pasteuria* strains from darker lakes (significant effect of a_{d320}; Fig. 2C,D; $\chi^2 = 8.10$, $P = 0.004$). There was no difference in spore yield according to sunlight-exposure treatment ($\chi^2 = 0.69$, $P = 0.41$) and no significant interaction between a_{d320} and treatment ($\chi^2 = 0.32$, $P = 0.58$).

EFFECTS OF PARASITE TRANSMISSION POTENTIAL ON MEAN HOST FECUNDITY

The average fecundity of *Daphnia* replicates for a given strain × treatment combination declined with increasing *Pasteuria* transmission potential (significant effect of transmission potential on mean host offspring production: $\chi^2 = 16.28$, $P < 0.001$). *Daphnia* replicates that were exposed to *Pasteuria* strains that had been exposed to sunlight produced more offspring, on average (significant effect of treatment: $\chi^2 = 13.70$, $P < 0.001$; Fig. 3), a result of a combination of lower infectivity (Fig. 2) and resulting castration (Fig. S1) of the sunlight-exposure treatment. In addition, there was a significant interaction between experimental treatment and parasite transmission potential (Fig. 3; $\chi^2 = 10.41$, $P = 0.001$). We observed a negative relationship between *Pasteuria* transmission potential and mean *Daphnia* fecundity for the sunlight-exposure treatment (Fig. 3, separate GLMM for sunlight-exposure treatment data, with lake as a random effect: $\chi^2 = 13.10$, $P < 0.001$), but there was no relationship between *Pasteuria* transmission potential and *Daphnia* fecundity for the control treatment data (separate GLMM for control treatment data, with lake as a random effect: $\chi^2 = 0.49$, $P = 0.482$).

Discussion

Exposure to ambient solar radiation strongly impacted parasite transmission potential, with *Pasteuria* from darker lakes particularly sensitive to sunlight, suggesting local adaptation (Fig. 1). This adaptation appears to be associated with both a cost and a benefit for the parasite. *Pasteuria* strains from clearer lakes produced fewer spores per infected host (Fig. 2C,D) but were more capable of causing infection, compared to strains from darker lakes (Fig. 2A,B). These influences of solar radiation on parasite infectivity, spore yield, and resulting transmission potential were reflected in host reproduction. *Daphnia* populations that were exposed to parasites from the sunlight-exposure treatment had higher fecundity, on average, than the group of *Daphnia* exposed to parasites from the corresponding control group (Fig. 3). However, parasite strains that had relatively high transmission potential following sunlight exposure more strongly reduced fecundity of their *Daphnia* hosts (Fig. 3, sunlight-exposure treatment). Notably, parasite strains whose transmission potential was least impacted by sunlight exposure were as virulent to their hosts as all strains from the control treatment (Fig. 3). Thus, adaptation to sunlight exposure in this virulent parasite effectively removed the fecundity benefit that host populations experienced when their sunlight-sensitive parasites were exposed to solar radiation.

In our experiment, exposure to ambient sunlight harmed *Pasteuria* spores, reducing their ability to infect their *Daphnia* host (Fig. 2A,B). Our study was not designed to separate the effects of UVR from other wavelengths of light, such as photosynthetically active radiation (PAR). However, we know that *Pasteuria* is sensitive to both visible light and UVR (Overholt et al. 2020). Our incubation depth of 0.5 m in North Lake allowed exposure to about 14% of available UVR and 80% of PAR based on measures

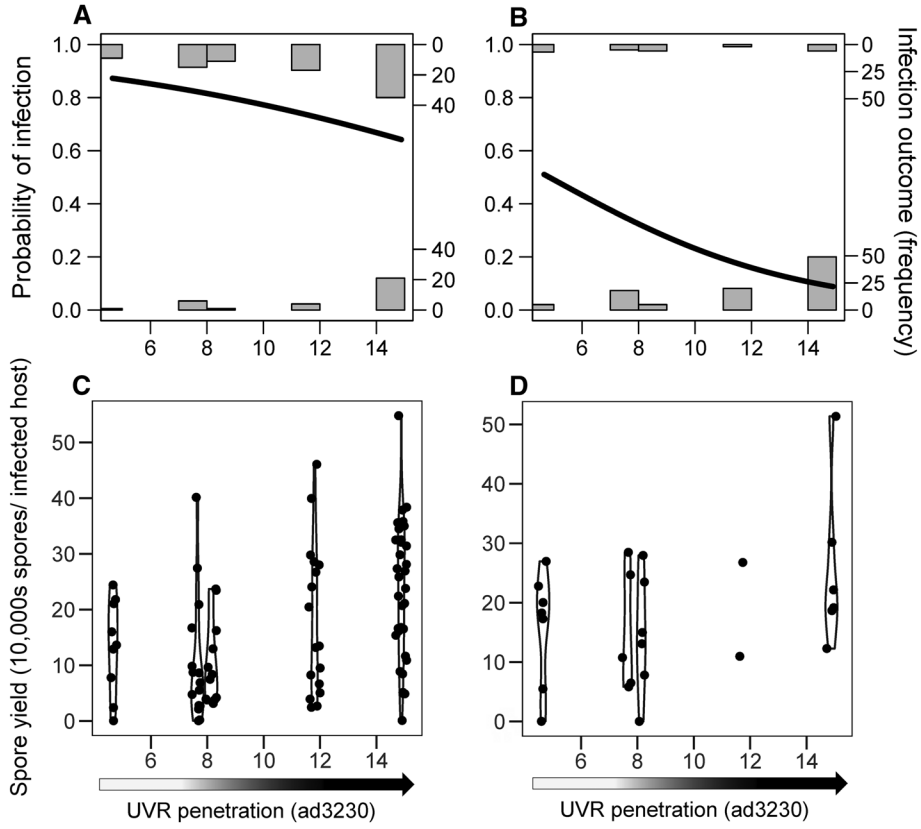

Figure 2. **Pasteuria strains from darker lakes were less infectious but yielded more spores. Panels A and C include data from the control treatment; panels B and D show the sunlight-exposure treatment. Lower a_{d320} values are relatively clearer lakes with greater ultraviolet radiation (UVR). (A and B) The black line (left axis) shows the probability of infection: in the control treatment, approximately 90.1% of individuals became infected when exposed to spores from the clearest lake, versus approximately 62.5% from the darkest lake. In the sunlight-exposure treatment, approximately 58.3% of individuals became infected when exposed to spores from the clearest lake, versus 10.9% from the darkest lake. The bars (right axis) show binned data of frequency of infected (top) and uninfected (bottom) individuals for different a_{d320} values. Each bar combines all *Daphnia* individuals from the parasite strain replicates for a given lake that were either uninfected (bottom) or infected (top); because there were different numbers of parasite strain replicates for different lakes, the total frequencies differ across lakes, with lakes with more replicates having larger total frequencies. (C and D) Spore yield from infected hosts (uninfected hosts not included in analysis/plot).**

of a_{d320} and a_{d440}, respectively. Thus, it is possible that exposure to UVR, or PAR, or both drove the patterns observed in our experiment.

We observed evidence of a trade-off between *Pasteuria* infectivity and spore yield. *Daphnia* were more often infected by *Pasteuria* strains from clearer lakes (Fig. 2A,B), but these parasite strains from clearer lakes produced fewer spores per infected host (Fig. 2C,2D). Sunlight exposure greatly diminished infectivity, regardless of the lake of origin of the spores. However, for those hosts that became infected, spore yields were unaffected by the sunlight-exposure treatment. Together these infectivity and spore yield gradients led to big differences in how transmission potential for parasite strains from clearer and darker lakes were impacted by sunlight exposure. High spore yields for parasite strains from darker lakes contributed to high transmission potential in the control treatment; however, sunlight exposure greatly diminished transmission potential for these strains from darker lakes, relative to strains from clearer lakes (Fig. 1). This evidence of a trade-off between infectivity and spore yield supports the idea that resistance to sunlight exposure is metabolically costly. Other researchers have seen reduced metabolic function in organisms with increased ability to tolerate or avoid UVR damage (Raven 1991; Hessen 1996). As spore production is known to be limited by resource availability (Hall et al. 2009; Cressler et al. 2014), our results suggest that the ability to withstand sunlight exposure may draw resources away from *Pasteuria* reproduction.

It is unclear why parasite strains from clearer lakes would be better able to infect *Daphnia* hosts when the spores were incubated in the dark (control treatment). One possibility is that, despite taking care to avoid sunlight exposure outside of the experimental treatment, *Pasteuria* spores were exposed to solar radiation during the process of collecting infected hosts and

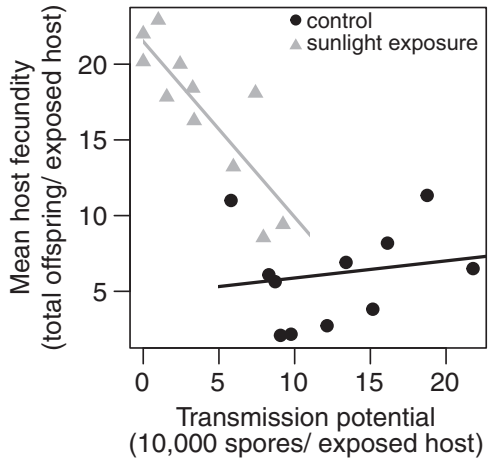

Figure 3. Mean host fecundity decreased with increasing parasite transmission potential (spore yield weighted by infectivity) in the sunlight-exposure treatment (gray) but not the control treatment (black). Each data point represents the mean host fecundity and transmission potential for a particular parasite strain × treatment combination.

processing the spores. This modest exposure to sunlight may have resulted in some spore mortality during experimental set up, with relatively lower rates of mortality among sunlight-adapted strains from the clearer lakes. However, it is also possible that *Pasteuria* UVR defenses increase the chance of infecting a host. *Pasteuria* infection of *Daphnia* is a complex yet well-studied process involving several key stages, including spore activation upon contact with the host, which involves shedding the outer spore shell, as well as attaching to and penetrating the host gut (Ebert et al. 2016). One possibility is that if sunlight-tolerant *Pasteuria* protect themselves with an altered spore shell, perhaps this also increases the odds of shedding this outer shell, leading to an increase in activation rate. Another possibility is that UVR chemical or physical protective barriers aid in gut attachment and penetration or increase resistance to immune system attacks. Further research investigating *Pasteuria* phenotypic responses to sunlight exposure could provide insight into the mechanisms driving both decreased spore yields and increased infectivity in sunlight-adapted parasite strains.

We predicted that sunlight exposure would drive variation in *Pasteuria* transmission potential, which would then influence mean host reproduction. Our results supported this hypothesis, but also suggest that the effect of *Pasteuria* transmission potential on host reproduction saturates. Reduced *Pasteuria* transmission potential due to reduced infectivity in the sunlight-exposure treatment provided relief to the *Daphnia* host, allowing fewer animals to be infected and castrated, yielding an increase in mean host fecundity (Fig. S1, and Figs. 2, 3). However, *Pasteuria* fitness levels beyond ∼75,000 spores/exposed host did not yield additional decreases in host fecundity. Although there was great variation in baseline *Pasteuria* transmission potential among strains that had been maintained in the dark, exposure to these spores had an equally negative impact on host fecundity (Fig. 3, control treatment). Notably, some of the most sunlight-tolerant *Pasteuria* strains that were exposed to ambient sunlight reduced host reproduction about as much as strains that were unexposed to solar radiation. Thus, it appears that local adaptation to sunlight exposure can reduce or even eliminate substantial gains in host reproduction provided by exposure to solar radiation.

How might parasite adaptation and the associated costs and benefits, influence disease dynamics in the field? Although sunlight exposure and intensity may limit the size, duration, and timing of *Pasteuria* epidemics in the field (as seen in Shaw 2019), our results suggest that adaptation to solar radiation could contribute to higher rates of disease transmission in clearer lakes than we might expect otherwise. Although sunlight exposure may benefit *Daphnia* by reducing infection by this castrating obligate killer, increased sunlight tolerance in the parasite may remove some of this benefit.

Sunlight penetration is changing in lakes around the world owing to impacts associated with climate change and land use (Williamson et al. 2014, 2016). We might expect to see earlier onset of disease, with larger, longer lasting epidemics in these darker lakes. Without the need to invest in costly UVR protection mechanisms, *Pasteuria* in these darker lakes may also produce more spores per infected host, further boosting transmission potential. Alternatively, as *Pasteuria* strains from darker lakes appear to be less infectious, it is possible that lake browning could lead to no net change in transmission. Longer term studies could help elucidate connections between changing sunlight availability and shifts in disease dynamics.

Identifying evolutionary responses to environmental stress and the impacts that local adaptation might have on species interactions are key challenges for evolutionary ecology. Solar radiation is a widespread environmental stressor known to strongly affect many organisms. Our finding that adaptation to solar radiation is linked with changes in key parasite traits as well as host–parasite interactions highlights the importance of considering evolutionary interactions beyond host–parasite coevolution in trying to untangle the drivers of disease. Further research into the physiological and genetic mechanisms associated with solar radiation adaptation will help us better understand why we see impacts on parasite spore yields and infectivity. Field research and experiments can help us uncover the consequences for host populations as well as disease dynamics and any cascading effects on ecosystem function. As human activities alter the availability of solar radiation in lake environments globally, this work will aid in predicting how these changes in sunlight exposure might influence disease.

AUTHOR CONTRIBUTIONS

MAR and MAD designed the experiment and MAR conducted the experiment. MAR conducted statistical analyses and drafted the manuscript with input from MAD. MAR and MAD edited the manuscript.

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DATA ARCHIVING

Data are available on Dryad: https://doi.org/10.5061/dryad.2jm63xskd (Rogalski and Duffy 2020).

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Supporting Information

Additional supporting information may be found online in the Supporting Information section at the end of the article.

Table S1. Chemical and physical characteristics of study lakes where *Pasteuria* spores were collected.

Table S2. Details on the dates when *Pasteuria*-infected *Daphnia* hosts were collected to yield the *Pasteuria* strains used in this study.

Table S3. Nested set of GLMM models used to test the effects of lake UVR availability (ad320), experimental treatment (sunlight-exposure vs. control), and their interaction in explaining *Pasteuria* transmission potential.

Table S4. Nested set of models used to test the effects of lake UVR availability (ad320), sunlight-exposure treatment, and their interaction in explaining *Pasteuria* infectivity (step 1 of hurdle model) and spore yield (step 2 of hurdle model).

Table S5. Nested set of GLMM models used to test the effects of transmission potential, experimental treatment (sunlight-exposure vs. control), and their interaction in explaining mean host offspring production (fecundity).

Figure S1. As expected, uninfected hosts produced many more offspring during the experiment than infected hosts did.