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Intraspecific priority effects and disease interact to alter population growth

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Abstract. Intraspecific variation may shape colonization of new habitat patches through a variety of mechanisms. In particular, trait variation among colonizing individuals can produce intraspecific priority effects (IPEs), where early arrivers of a single species affect the establishment or growth of later conspecifics. While we have some evidence for the importance of IPEs, we lack a general understanding of factors affecting their presence or magnitude across a landscape. Specifically, IPEs should depend strongly on success of colonizers in the new habitat patch. This success hinges on interactions between colonizer traits and local selective pressures, but such context dependence remains unexplored experimentally. We addressed this gap by looking for the dynamical signature of IPEs in environments with and without a selective (parasite) pressure. We tested whether IPEs affected the population dynamics of a zooplankton host species (*Daphnia dentifera*) collected from two populations showing a tradeoff between growth rate and resistance to a fungal parasite (*Metschnikowia bicuspidata*). Differences in arrival order significantly altered population growth during a period of rapid resource depletion, driving large (up to 65%) differences in population abundance. Furthermore, the presence of IPEs was context dependent, as parasites reduced the impact of early arrivers on later arrivers. Such context-dependent IPEs, mediated by colonizer traits, colonization order, and selective pressures, may play an unanticipated role in the ecological and evolutionary dynamics of natural metapopulations. This mechanism highlights the overall importance of intraspecific variation for understanding ecological patterns.

Key words: colonization; Daphnia; *host–parasite dynamics; intraspecific variation; metapopulation dynamics;* Metschnikowia bicuspidata; *spatial structure*.

Introduction

Intraspecific variation is increasingly recognized as a major driver of contemporary ecological dynamics (Hughes et al. 2008, Bailey et al. 2009, Bolnick et al. 2011). This realization highlights the limitations of using a species' "average individual" to define its performance, function, or interactions. Intraspecific variation can alter or expand the role of a species through its influence on populations, communities, and ecosystems by a variety of mechanisms. For example, increasing population genetic diversity can alter the stability of population dynamics (Agashe 2009), enhance colonization success (Crawford and Whitney 2010), and increase biomass of plant populations (Cook-Patton et al. 2011). Similarly, differentiation among populations can influence community structure (Whitham et al. 2003, Hughes and Stachowicz 2004, Crawford and Rudgers 2012), change important abiotic conditions (Crutsinger et al. 2010), or alter the biomass and species richness of lower trophic levels (Palkovacs and Post 2009). Ignoring such variation inhibits our mechanistic understanding of natural patterns of species abundance and distribution.

Intraspecific variation may be particularly important during the colonization of new habitat patches, as new patches can receive colonists from a variety of neighboring source populations. Individuals from these distinct source populations often vary in their genes or traits, due to unique ecological and evolutionary pressures experienced in their natal patches (Hendry et al. 2002, Kawecki and Ebert 2004, Van Allen and Rudolf 2013). This among-patch variation can drive differences in the ability of colonists to survive and reproduce in a new patch. When colonists also vary in their order of arrival to this new patch, we may see that the order of colonization affects subsequent population dynamics. One mechanism driving this role of arrival order is the intraspecific priority effect (IPE), where early arrivers to a patch affect the establishment or growth of later arrivers. IPEs during colonization of habitat patches may play a pivotal, but underappreciated role in the ecological and evolutionary dynamics of newly established, local populations. Early arrivers to a new habitat, introduced at low densities, are expected to grow quickly and deplete existing resources. Late arrivers, then, would experience relatively intense

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intraspecific competition in addition to reduced resource levels, diminishing their establishment success. This general process drives within-host competition among parasite strains (de Roode et al. 2005, Ben-Ami et al. 2008), and is linked to the genetic structure of entire zooplankton metapopulations (Hebert and Moran 1980, Boileau et al. 1992, De Meester et al. 2002). The growth and success of early arrivers, however, should depend critically on the environmental conditions or selective pressures in the new patch. Poor performance of early arrivers, due to a variety of abiotic or biotic factors, may limit their effects on late arrivers. The presence, magnitude, and dynamical consequences of IPEs, then, fundamentally depend on an interaction between (1) intraspecific variation in colonists, (2) their order of arrival to the new habitat patch, and (3) the environmental pressures in that patch. This potential context dependence of IPEs remains untested empirically.

We addressed this gap with a mesocosm experiment. We tested whether IPEs could alter initial population growth of a single species in a new environment, and whether a biotic selective pressure (parasites) affected the presence or magnitude of IPEs. Using a zooplankton host–fungal parasite system, we manipulated the colonization order of host individuals from two populations into a new habitat patch. Animals in these populations differed in susceptibility to infection. Due to a previously documented trade-off between infection susceptibility and growth rate (Hall et al. 2010), we predicted that (1) these two populations would exhibit different growth in the new environment, (2) altering the order of arrival of populations would significantly alter population growth in the new habitat (indicating an IPE), and (3) a selective pressure (parasite) would affect the presence or strength of IPEs, via its effects on early arrivers. Our results indicate that intraspecific variation, differences in colonization order, and parasite-mediated selective pressures jointly determine the initial growth rate of a species when colonizing new habitat. They echo the dynamical signature of IPEs in the absence of parasites; however, parasites reduced the impact of early colonists, thereby inhibiting IPEs. These complex interactions may drive substantial variation in colonization dynamics across spatial landscapes.

Materials and Methods

Study system.—Zooplankton species in freshwater habitats often adapt to local environmental conditions (e.g., De Meester 1996). This local adaptation, when combined with the small size of founder populations, drift, and bottlenecks, creates heterogeneity between populations inhabiting distinct patches, and also any colonists originating from them. Our focal species was the lake-dwelling cladoceran *Daphnia dentifera*, a seasonally dominant grazer in Midwestern (USA) lakes. *D. dentifera* hosts a variety of parasites and pathogens (Ebert 2005), and experiences seasonal epidemics of the fungal yeast parasite *Metschnikowia bicuspidata* (Metschnikoff). The size of seasonal epidemics depends on a number of factors (e.g., Hall et al. 2006, Duffy and Sivars-Becker 2007, Duffy and Hall 2008, Overholt et al. 2012) that drive significant variation in host and disease dynamics. Hosts consume infectious spores during feeding and die 10–14 days post-infection, releasing spores back into the water column (Green 1974). While non-castrating, this parasite reduces fecundity of infected hosts (Auld et al. 2012). Resistance to infection is tied to foraging ecology. Faster-feeding individuals and genotypes encounter more spores, increasing infection risk (Hall et al. 2010, 2012). However, faster feeders also produce more offspring (when consuming high-quality resources; Hall et al. 2010, 2012). These dual relationships with feeding can thus create a trade-off between fecundity and resistance (Hall et al. 2010), one that we show and use in this study.

Experimental design.—This study had two components. First, we assayed two natural lake populations of host *D. dentifera* (hereafter, host) for susceptibility to the fungal parasite *M. bicuspidata* (parasite) in a laboratory infection experiment. Second, we used a mixture of individuals from these two populations to examine the importance of IPEs in a mesocosm experiment. In this experiment, we manipulated the identity of the host population, their colonization order, and the presence or absence of the parasite in a $2 \times 2 \times 2$, fully factorial, randomized block design (plus two additional control treatments for 10 total treatments, $N = 5$ replicates). This design simulates colonization of new or recently reestablished habitat patches, such as temporary ponds that dry periodically.

Infection assay

We collected individuals from two lake populations (Canvasback and Downing) in the Greene Sullivan State Forest and Minnehaha Fish and Wildlife Area (Sullivan County, Indiana, USA) in early June 2011. We kept individuals in the laboratory (20°C, 16:8 h day : night cycle) in 1-L containers containing filter-sterilized (1-μm pore size) lake water and the algae *Scenedesmus obliquus* (~2 mg C/L) for approximately two generations to standardize maternal or environmental effects. These individuals represented polyculture subsets of natural lake populations, not isolated clonal lineages; polycultures better reflect the variability found in the field. To start infection assays, we added six ~6-d-old individuals to 150-mL beakers ($N = 20$ replicates per population) containing filtered lake water and algal food. We then added fungal spores, reared in vivo, at 200 spores/mL. Fungal spores have been raised in the laboratory on a single host clone since 2003. *M. bicuspidata* shows little genetic diversity across its range (Duffy and Sivars-Becker 2007), minimizing potential shared evolutionary history between particular host/parasite strains used in the experiment. After a 24-h exposure to spores and every three days thereafter, we transferred hosts into fresh media and food until visual inspection of infection status at 11 days post-exposure

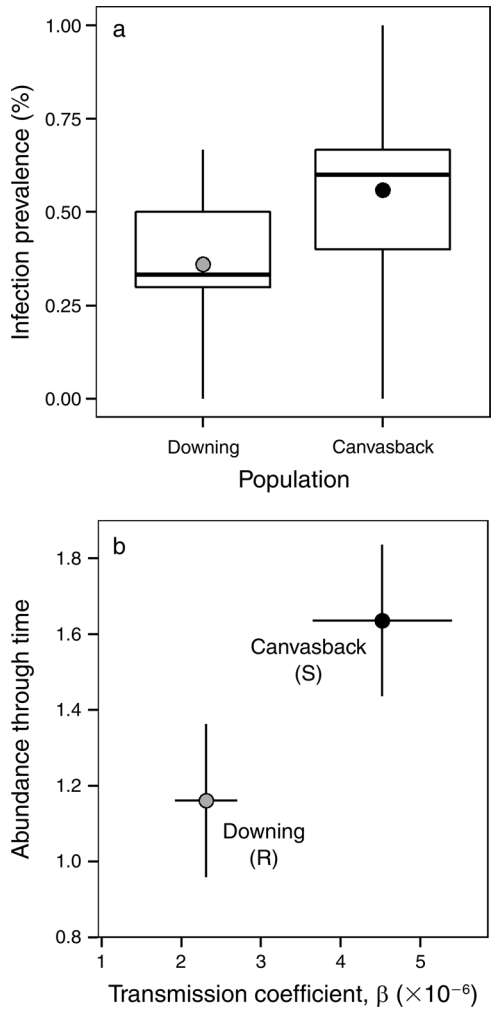

FIG. 1. (a) Infection prevalence in a laboratory-based infection assay for two lake populations of a zooplankton host-grazer (*Daphnia dentifera*) used in the mesocosm growth experiment. Solid horizontal lines represent lake medians, circles represent lake means (different colors represent different lake populations), boxes capture middle 50% of values (interquartile range [IQR]), and whiskers extend to highest and lowest non-outlier (1.5 × IQR). (b) Relationship between disease transmissibility (β) in the infection assay and population abundance over time for parasite-free monocultures in the mesocosm experiment (measured as coefficients from generalized additive mixed effect model, GAMM; points are parameter estimate means, error bars show ±SE). The faster-growing population (Canvasback) is more vulnerable to infection (i.e., more susceptible [S]) than the slower-growing, more resistant (R) one (Downing).

(25× magnification). We recorded infection status and the number of individuals surviving to diagnosis.

Mesocosm experiment

Order of arrival.—The two source populations differed in resistance to infection (see *Results*, Fig. 1). We added individuals from these populations to indoor mesocosms (plastic tanks filled with 40 L of treated tap water, arranged in a randomized block design) at two different times, 10 days apart (day 0 [early addition] and day 10 [late addition]). Treatments either received individuals from the same population during each colonization event ("single" population treatments), or individuals from a different population during each colonization event (multiple populations, "sequential" arrival, Table 1). One treatment received individuals from both populations during each addition ("simultaneous" arrival). Densities were identical for all additions (~0.5 individuals/L). Mesocosms were inoculated with algae (*Scenedesmus obliquus*) and nitrogen and phosphorus fertilizer were added at a 14:1 mass ratio (280 μg N/L, 20 μg P/L), with subsequent weekly additions (assuming 5% loss per day and exponential decay). Tanks were lit on a 16:8 h day : night schedule. Sampling occurred twice per week, when ~5% of the total tank volume was removed and all individuals counted. Chlorophyll *a*, a surrogate for resource density, was quantified using chilled ethanol extraction followed by narrow band fluorometry (Webb et al. 1992, Welschmeyer 1994) using a Trilogy laboratory fluorometer (Turner Designs, Sunnyvale, California, USA). This experiment ran for 29 days, approximately five to six generations of *D. dentifera*.

Fungal infections.—Fungal spores were introduced to half of the experimental treatments at a low dose (~2 spores/mL), 7 days after the initial addition of hosts. Spores were cultured and infection was diagnosed as described for the infection assay.

Statistical analyses

Infection assay.—To compare resistance to infection between populations, we used binomial generalized linear models (GLM; logit link function; Pinheiro et al. 2013) in the statistical program R (R Core Development Team 2012). We ignored replicates in which fewer than two individuals survived or could not be scored unambiguously. We also calculated the rate of parasite transmission, β, for lake populations. This value is useful as a per-capita measure of infection risk, and complements the population-level differences in infec-

TABLE 1. Experimental treatments describing order of addition of individuals of a zooplankton grazer-host (*Daphnia dentifera*) from a more susceptible but faster-growing population (susceptible, S) or a slower-growing, more resistant population (resistant, R) to new habitat patches.

Treatment	Early-arriver identity (day 0)	Late-arriver identity (day 10)	Population type
S,S	susceptible	susceptible	single
R,R	resistant	resistant	single
S,R	susceptible	resistant	multiple (sequential)
R,S	resistant	susceptible	multiple (sequential)
SR,SR	both	both	multiple (simultaneous)

Notes: Planned contrasts between treatments are as follows: contrast 1 demonstrates S,S vs. R,R, and contrast 2 demonstrates S,R vs. R,S. All treatments are crossed with presence (+) or absence (–) of fungal parasite *Metschnikowia bicuspidata*, added on day 7.

tion prevalence. We followed previous methods for calculating β (Civitello et al. 2012). Briefly, we can assume that uninfected (susceptible, S) hosts decrease after contact with spores (Z) following a simple transmission model: $dS/dt = -\beta SZ$. We can then predict the proportion infected in the experiment, p_I, using the integrated form of this model: $p_I = 1 - \exp(-\beta Z t_E)$, where t_E is the time of experimental exposure of hosts to spores. Higher values of β indicate lower host resistance (i.e., higher susceptibility). We used the binomial distribution to find an estimate of β that best fit the infection data (using maximum likelihood). We tested for significant differences between populations using bootstrapped confidence estimates and a likelihood ratio test.

Mesocosm experiment.—Abundance data were analyzed using generalized additive mixed effects models (GAMM; Wood 2006). GAMMs account for nonlinear population growth using flexible, nonlinear functions while also estimating fixed effect parameters (Hastie and Tibshirani 1987). More specifically, we fit a penalized regression spline to represent nonlinear change in abundance over time, with treatment as a fixed factor modifying the underlying growth model (see also Appendix: Table A1). Individual treatment coefficients, then, represent population abundance over time (i.e., growth rate): higher coefficients indicate higher abundance. This model also contained a smoothing term for the abundance of an unwanted species, *Daphnia pulicaria*. This species persisted in tanks throughout; ignoring this species in the model did not change results, but nonetheless this term is included in *Results*. Spatial block was added as a random effect. To account for repeated sampling through time, we included an autoregressive correlation structure (individual tank observations repeated over time). We fit the model with a quasi-approximation to the Poisson error distribution (log-link function) due to significant over-dispersion in host abundance (Crawley 2007).

The three-way interaction from this model was pertinent for showing potential IPEs. This interaction, identity of early arrivers × identity of late arrivers × presence or absence of fungal parasite (i.e., early × late × parasite), represents the biological interaction of intraspecific variation (population identity), arrival order (i.e., Table 1), and selection pressure (presence/absence of parasite). We tested for overall significance of interaction terms in the factorial model (without the simultaneous arrival treatments, which were not implemented factorially). Additionally, we implemented planned, two-tailed contrasts outlined in Table 1. These contrasts conveyed two important pieces of information. Contrast 1 tested for differences in population growth or disease dynamics among the source populations. These differences enhanced detection and interpretation of an IPE. Contrast 2 tested whether variation in arrival order altered population growth or disease dynamics in multiple-sequential treatments. By our definition, an IPE would manifest as a significant difference in population abundance, by way of contrast 2; each treatment should bear the population growth signature of the early arriver, identified in contrast 1.

The three-way interaction in our model approached statistical significance (see *Results*). To better understand these dynamics, we fit follow-up models with and without the parasite. This allowed us to determine the effect of early arrivers, late arrivers, and their interaction with and without the parasite (Appendix: Table B2.). We used post-hoc contrasts to test for differences among specific treatments, adjusted for multiple comparisons (Holm 1979). These post-hoc contrasts included the simultaneous arrival (SR,SR) treatments, which were not included in any of the factorial models (Fig. 3).

We further investigated the effect of early arrivers on late arrivers by analyzing rates of resource (chlorophyll *a*) decline from days 9–16. Early arrivers take time to establish, acquire resources (Appendix: Fig. A1), and grow to reproductive age or size. The 9–16 day window represents the analogous time for late arrivers to establish and grow, where they face resource declines driven primarily by the established early arrivers. Resource dynamics during this period, then, may affect the growth or establishment of late arrivers. We analyzed the rate of decrease in chlorophyll *a*, log(day 9 resource density) − log(day 16 resource density)/7 days, with mixed-effect models (spatial block as a random effect). We examined the three-way interaction between early-arriver identity, late-arriver identity, and the presence or absence of the parasite. We used post-hoc comparisons to test whether the simultaneous arrival treatments differed from the others (adjusting for multiple comparisons; Holm 1979). Infection prevalence data were not analyzed statistically, as prevalence remained generally below the detection level.

Results

Microcosm infection assay

Our two lake populations, Canvasback and Downing, differed significantly in infection prevalence (binomial GLM, $\chi^2_{1,27} = 4.80$, $P = 0.028$; Fig. 1a.) and the index of resistance, β (log-likelihood ratio test, $D = 4.804$, $P = 0.017$, df = 1; Fig. 1b.). Hereafter, we refer to these polyculture populations as susceptible (S, Canvasback) and resistant (R, Downing).

Mesocosm experiment

Overall population abundance was affected by the three-way interaction between the identity of early arrivers, the identity of late arrivers, and presence or absence of a parasite in the simulated habitat patch (GAMM; early × late × parasite, $r^2 = 0.621$, $F_{1, 334.5} = 3.68$, $P = 0.056$). We clarified these IPEs with planned contrasts. Contrast 1 showed that our single population treatments receiving colonists from the more susceptible population (S,S) grew to higher abundance with (S,S,+) and without (S,S,−) the parasite, compared to the

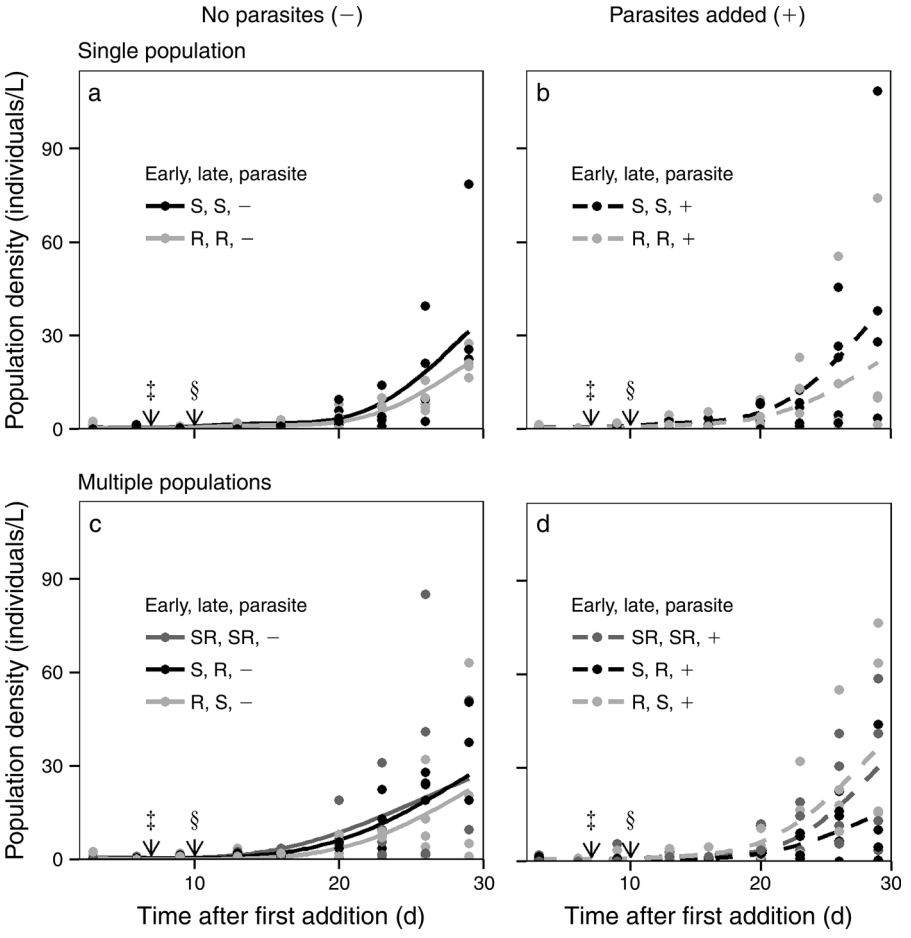

FIG. 2. Population growth of experimental populations of *Daphnia dentifera* upon colonization of a new habitat patch. Line color represents the order in which individuals from susceptible (S, Canvasback) and resistant (R, Downing) host populations were added to tanks (black indicates S early, light gray indicates R early; Table 1). Line type indicates presence (+, dashed lines) or absence (–, solid lines) of a fungal parasite. Early addition of individuals occurred on day 0, fungal inoculations (‡) on day 7, and late host addition (§) on day 10. (a, b) Single-population treatments received individuals from the same population during early and late additions (Table 1, contrast 1 [S,S vs. R,R]). (c, d) Multiple population treatments received either a mix of both populations simultaneously (i.e., SR, SR, dark gray lines), or received individuals from each population sequentially (black indicates S early, light gray indicates R early, Table 1, contrast 2 [S,R vs. R,S]). Lines reflect best-fit models (GAMM) of *Daphnia* abundance, points are *Daphnia* density data.

resistant-only treatments with (R,R,+) and without (R,R,–) the parasite; S,S,– > R,R,–, $P < 0.001$; S,S,+ > R,R,+, $P = 0.001$; Figs. 1b, 2a, b, and 3).

In the sequential treatments, however, order of arrival significantly altered host abundance (contrast 2). Without parasites (–; solid lines in Figs. 2 and 3), populations receiving colonists from faster-growing population S before receiving colonists from slower-growing population R (S,R,–) had higher abundance than the alternate arrival order (S,R,– > R,S,–; $P = 0.038$). Differences in arrival order produced a 16.4% difference in final mean population size. However, in parasite addition treatments (+; dashed lines, Figs. 2b and 3), populations receiving immigrants from R before S grew faster (S,R,+ < R,S,+; $P < 0.001$). This difference in arrival order led to an even larger (65.6%) difference in final mean population size and drove the marginal three-way interaction.

Our follow-up analyses revealed how parasites modulated the importance of invasion order. In systems without parasites, early arrival of individuals from the susceptible population (i.e., S,S,– and S,R,– treatments) increased population abundance compared to early arrival of individuals from the resistant population (i.e., R,R,– and R,S,– treatments). However, late arrival of S individuals did somewhat offset the low abundance of early R in the R,S,– treatment (Fig. 3). The increase in abundance between R,R,– and R,S,–, while not significant after adjusting for multiple comparisons, resulted in a significant early × late arriver interaction for population abundance ($F_{1, 163.5} = 4.57$, $P = 0.0341$; Fig. 3). With parasites, in contrast, we found significant but independent effects of early arrivers ($F_{1, 163.5} = 3.52$,

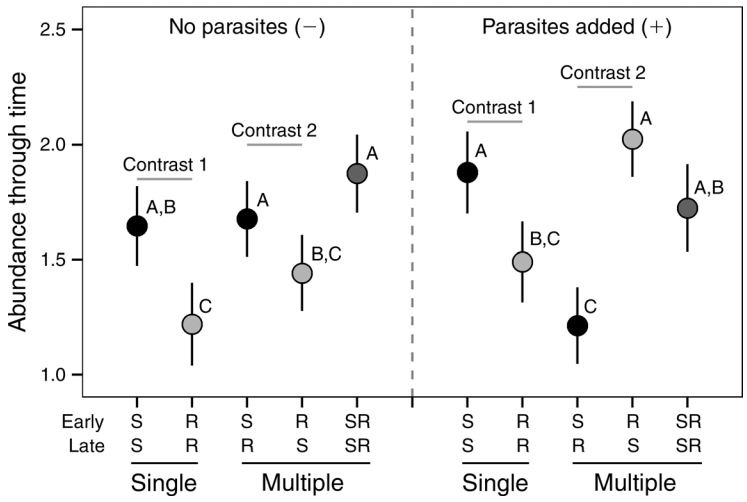

FIG. 3. GAMM-derived estimates of *Daphnia* abundance through time (coefficient means ± SE) for experimental populations, showing influence of early- and late-arriver identity when parasites are present or absent. Circle color indicates identity of early arriver (black indicates S, light gray indicates R, dark gray indicates SR). Gray bars connect treatments compared with the four main planned contrasts (Table 1). Treatments sharing the same letter are not significantly different after adjusting for multiple comparisons. Comparisons are only made within parasite present/absent treatments.

$P = 0.015$) and late arrivers ($F_{1, 163.5} = 17.43$, $P < 0.0001$) on population abundance. Thus, with parasites, early arrival of R individuals led to higher abundance than early arrival of S individuals. However, this effect of early arrival of R individuals is small compared to the strong effect of late arrival of S individuals, which significantly increases overall population abundance during the experiment (Fig. 3). Therefore, with parasites, identity of early arrivers becomes less important than identity of later arrivers.

Post-hoc comparisons with the simultaneous arrival treatments provide further insight. In parasite-free environments, simultaneous arrival treatments were not different from treatments that received early S (S,S or S,R; Fig. 3) but they were significantly higher than treatments which received R first (R,R and R,S; Fig. 3). In contrast, when parasites were present, the simultaneous arrival treatments only differed from treatments with early S and late R (Fig. 3).

Chlorophyll a density.—Resource abundance declined during the course of the experiment (Appendix: Fig. A1). However, rates of resource decline during the key period of late-arriver establishment (days 9–16) depended on an interaction between early-arriver identity and presence of the parasite ($F_{1,34.6} = 10.5$, $P = 0.003$). In the absence of the parasite, early-arriving R individuals (R,R,–, R,S,– treatments) reduced resources at a faster rate than early-arriving S individuals (S,S,–, S,R,–; Fig. 4). The presence of the parasite reversed this pattern: early-arriving S individuals resulted in faster rates of resource decline than early-arriving R individuals (Fig. 4). After correcting for multiple comparisons, the simultaneous arrival treatments (SR,SR) were not significantly different from any others (Fig. 4).

Infection prevalence.—Infected hosts were observed only on the last sampling date (day 29), and at low prevalence (i.e., <5%). Many of the infected individuals came from only two tanks, and therefore prevalence data were not analyzed statistically.

DISCUSSION

Intraspecific variation drives important differences in local populations and communities (Hughes et al. 2008, Bailey et al. 2009, Bolnick et al. 2011), but its importance in spatially structured landscapes remains relatively unknown. We found strong effects of arrival order on *Daphnia* population growth in new environments, but that these effects were mediated by the presence or absence of a parasite. In particular, we found the signature of IPEs in parasite-free environments, where early arrivers affected late arrivers. Parasite presence, however, reduced the importance of early arrivers, inhibiting IPEs. The magnitude of differences in population abundance we observed, in conjunction with the context-dependent importance of arrival order, has substantial implications for within- and among-population dynamics in spatial landscapes.

IPEs: the signature in population growth

Our natural lake populations differed in a key trait (infection susceptibility; Fig. 1a) that could produce the signature of IPEs on population growth. Variation in susceptibility among populations commonly occurs in host–pathogen systems, as either adaptive (e.g., Duffy et al. 2012) or potentially neutral (e.g., Carlsson-Graner and Pettersson 2005) responses to local conditions. Due to a previously documented trade-off (Hall et al. 2010), we predicted that this variation in susceptibility would

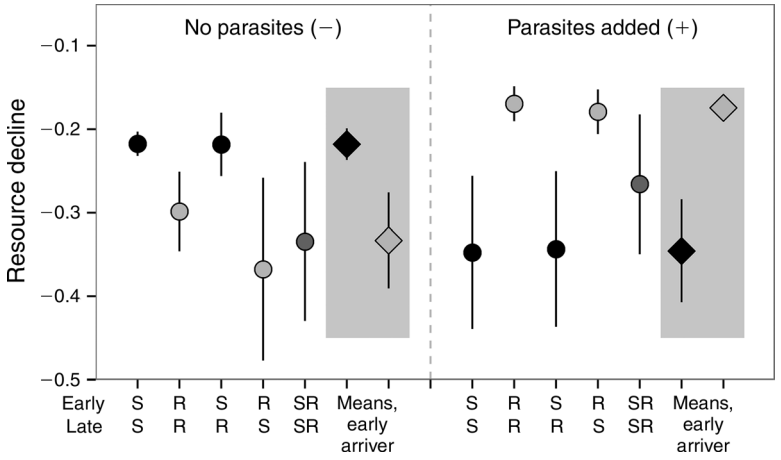

FIG. 4. Resource decline (chlorophyll *a*, μg/L, log-transformed) between day 9 and day 16. Higher values indicate fewer resources consumed per day during this window. Analyses indicate that early-arriving R individuals resulted in faster rates of resource decline in parasite-free environments, but slower rates of decline in the presence of parasites. Treatment labels are as in Fig. 3, with the addition of diamonds representing the mean rate of decline for each early-arriver treatment (black indicates early S, gray indicates early R). The simultaneous arrival treatments (SR,SR) were not significantly different from any other treatments after correcting for multiple comparisons, and were not used to calculate early-arriver means.

correlate positively with population growth over time. We recovered this relationship (trade-off) in our experiments: the single-population treatments colonized by the more susceptible (S) population grew to higher abundance during the experiment than those colonized by the more resistant population (R, contrast 1; Figs. 1 and 3). These overall trait differences between populations of origin allowed us to detect and interpret the signature of IPEs on population abundance over time. Population growth determined by the identity (and traits) of the early arriver would support an IPE in the strictest sense.

Arrival order influenced population growth, but the signature of IPEs (i.e., the importance of early arrivers) on growth depended on parasites. Without parasites, population growth was strongly affected by identity of early arrivers. In general, early arrival of individuals from the fast-growing, susceptible (S) population resulted in higher population abundance. Contrast 2 showed that early arrival of S individuals, followed by R individuals, led to higher abundance than when S arrived after R. This general, positive effect of early S emerged even when S individuals arrived at half density (in the simultaneous arrival treatment, at a 50:50 ratio S:R). These findings highlight the influence of the identity of the early arriver in parasite-free treatments. However, parasite presence, even at very low density, reversed this pattern (e.g., contrast 2, S,R,+ < R,S,+; Fig. 3). While the identity of early arrivers did affect populations (i.e., initial colonization by individuals from the more resistant population meant slightly faster growth), the identity of late arrivers had a much stronger impact. More specifically, with parasites, populations grew to highest abundance when fast-growing S individuals colonized late. This result suggests that the virulent parasite negatively affected early arrivers, reducing their influence on later arrivers.

Intraspecific priority effects: mechanisms

Mechanistically, these IPEs likely arose from resource competition between early and late arrivers. Early arrivers consume shared algal resources and convert this energy to offspring; depletion during this period limits resources available to colonists that arrive late. The signature of this resource-based mechanism appeared in sequential treatments without parasites. From a population perspective, the identity of the early arriver drove abundance, even with slow-growing early arrivers (specifically contrast 2, R,S,− < S,R,−; Fig. 3). This result suggests that early R limited late S (an IPE). Supporting this idea, resources declined rapidly when R colonized before S (Fig. 4). This reduction in resource abundance likely exerted a strong, negative effect on late S individuals. Parasites, however, alleviated the negative effect of early R on late S. In the presence of parasites, algal resources declined at a slower rate with early R (Fig. 4), presumably via a reduction in individual resource acquisition rate. Since resources declined at a lower rate with parasites, populations with late S arrival grew like those in which S arrived early (i.e., R,S,+ vs. S,S,+; Fig. 3). Parasite presence, then, inhibited the IPE inferred from parasite-free growth data.

High population abundance in the R,S,+ treatment also diminished the likelihood of an alternative explanation for the IPE without parasites. In this other hypothesis, early R did not limit late S colonists via resource depletion. Instead, fast-growing S individuals simply arrived late (day 10 of the 30-day experiment), producing intermediate abundance (as hinted by the nonsignificant trend, Fig. 3). Yet, with parasites, late S

achieved high abundance, despite being absent for the first third of the experiment. This counterpoint shows that length of time to grow was not the only factor driving low overall abundance in the R,S,– vs. S,R,– treatments. Variation in resource decline rates offers a plausible mechanism driving the shift in importance from early arrivers (without parasites) to late arrivers (with parasites). Overall, we infer that early R inhibited late S without a parasite, evidenced by the population growth signatures and rates of resource decline. Parasite presence alleviated the negative effect of early R on late S, presumably by indirectly increasing resource availability for fast-growing late arrivers.

Our inference of IPEs, mediated through resources and parasites, warrants some caution, however. Ideally, we would have genotypic data that identified the source of individuals (clonal genotypes) at the end of the experiment. Without these data, we cannot definitively conclude whether early arrivers (in any of our treatments) altered the establishment success of late arrivers. We use population growth signatures as a proxy to infer the composition of individuals and traits in a habitat. This metric cannot precisely identify the composition of individuals within a population. Yet, it identifies significant IPE-mediated variation in growth rate, an important trait at the population level with potential consequences for natural communities. Additionally, we need an explanation for the mechanism by which parasites potentially altered feeding rates of their hosts. Ongoing work shows that exposure to parasite spores can reduce feeding rates, and that this effect differs among individual clonal genotypes (S. R. Hall, *unpublished data*). Parasites might also act through some unknown, additional pathway, further complicating comparisons between parasite + and − treatments. With these limitations in mind, we still believe that parasite-mediated resource acquisition rate of early arrivers most likely explained the presence or absence of IPEs.

Overall, the experiment offers four major conclusions. First, we saw the signature of IPEs on population growth early in the colonization process. Second, the presence of parasites shifted the relative importance of early and late arrivers; we interpret this as context dependence of IPEs (with the noted caveats). Mechanistically, this shift may be due to different rates of resource reduction for each population in environments with and without the parasite. Third, this context dependency arose despite very low parasite inoculation and subsequently low abundance. Although we did not observe large epidemics in this experiment, the parasite did maintain itself in vivo throughout (indicating some sustained transmission). The strong effects of parasites, even at the low levels in this experiment, make some sense in light of *Daphnia* growth patterns. During a nearly exponential increase, parasite-induced mortality of even a few colonists, or reductions in resource consumption (and thus later reproduction) may have large effects on initial population dynamics. The seemingly insignificant prevalence of parasites in our experiment could have surprising effects in an IPE context, especially in conjunction with their visible effects on resource decline rates. Lastly, the simultaneous arrival treatments suggest an advantage for having a mixture of individuals colonize a habitat patch. These treatments appear to buffer populations against the reduced abundance caused by certain orders of arrival, and may stabilize dynamics among distinct habitat patches. Overall, this work shows strong interactive effects of intraspecific variation, arrival order, and environmental context on population dynamics. Understanding the role of intraspecific variation on a landscape scale may help to explain the substantial heterogeneity often found among habitat patches (e.g., Sutcliffe et al. 1996, Cottenie et al. 2003).

Ecological and evolutionary implications

Though our study does not address long-term effects of IPEs, the differences in aggregate abundance we observed here may represent significant variation in interspecific competitive ability (e.g., Wilbur 1982); variation in establishment success of heterospecific individuals during this early, transient colonization stage would likely affect later community composition (Fukami 2004, Olito and Fukami 2009). This potential role of IPEs in interspecific competition remains unexplored, but would complement the large body of work examining priority effects between species (Chase 2003). IPEs may be more common in systems characterized by rapid population growth and clear spatial structure (though see Sunahara and Mogi [2002], Eitam et al. [2005] for examples of IPEs among cohorts), including pioneer species in disturbed landscapes (e.g., Chapin et al. 1994), metapopulations with high patch turnover (e.g., Hanski 2011), boundary zones of range expansion for invasive species (e.g., Sakai et al. 2001), and pathogen interactions within and among hosts (de Roode et al. 2005, Ben-Ami et al. 2008, Hoverman et al. 2013).

Our study supports the premise that early arrivers can limit the growth and/or establishment of late arrivers. If true, the long-term population genetics of local populations may reflect effects of prior colonization history and intraspecific competition, in addition to (or even rather than) contemporary ecological and evolutionary factors. This complicates the traditional idea that genotypes simply increase in frequency where they are favored, because IPEs could limit their ability to establish in a favored patch in the first place. Our results illustrate that early ecological dynamics and context could facilitate these long-term evolutionary patterns, in some cases giving early arrivers an advantage. This can buffer relatively maladapted populations against immigration, allowing for further local adaptation and evolutionary change (Boileau et al. 1992, De Meester et al. 2002, Fukami et al. 2007, Knope et al. 2012). We anticipate that future research will

further link IPEs to ecological and evolutionary dynamics.

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Supplemental Material

Ecological Archives

The Appendix is available online: http://dx.doi.org/10.1890/13-1958.1.sm