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Phenotypic plasticity promotes species coexistence

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Ecological explanations for species coexistence assume that species' traits, and therefore the differences between species, are fixed on short timescales. However, species' traits are not fixed, but can instead change rapidly as a consequence of phenotypic plasticity. Here we use a combined experimental–theoretical approach to demonstrate that plasticity in response to interspecific competition between two aquatic plants allows for species coexistence where competitive exclusion is otherwise predicted to occur. Our results show that rapid trait changes in response to a shift in the competitive environment can promote coexistence in a way that is not captured by common measures of niche differentiation.

Phenotypic plasticity allows genetically identical individuals to express different phenotypes in response to changes in abiotic or biotic conditions[1]. While plasticity in response to abiotic conditions is an important requirement of species coexistence in variable environments[2], the effects on coexistence of plasticity in response to competitors themselves has received less attention. Evidence from short-term studies suggests that plasticity is ubiquitous and can either increase[3,4] or decrease[5–7] the strength of competition between species. The expectation is that these changes will decrease or increase opportunities for species coexistence, respectively. However, theory demonstrates that changes in the strength of competition are not enough; plasticity can only promote coexistence if it reduces the strength of interspecific competition relative to the strength of intraspecific competition[8]. Only by doing so can plasticity buffer species from competitive exclusion by allowing species to recover after being perturbed to low density in a community. Given that plasticity can affect the strength of both inter- and intraspecific competition[9], and that no empirical study has quantified the effects of plasticity on recovery from low density, the net effect of plasticity on species coexistence remains, surprisingly, largely unknown[10].

Here we test the ability of plasticity in response to competitors to promote species coexistence by improving the ability of species to recover from low density in a community. The ability to recover from low density requires that a species benefits from a growth rate advantage over its high-density competitor. This requirement emphasizes that the most important phenotypic changes to understand when assessing the ability of plasticity to promote coexistence are those that occur when a species shifts from being common, and thus experiencing competition predominantly from conspecifics, to being rare, and thus experiencing competition predominantly from heterospecifics, in a community. Indeed, comparing the changes that occur when species shift from being common to rare in a community is a key tool for theoretical studies of species coexistence mechanisms[11–13].

We use a combined experimental–theoretical approach (Methods and Fig. 1) to quantify how plasticity occurring in response to conspecific versus heterospecific competitive environments influences coexistence of two globally distributed, co-occurring aquatic vascular plants, *Lemna minor* and *Spirodela polyrhiza*. These species have fast life cycles with asexual reproduction occurring every 3–7 days, providing an ideal system to assess the effects of plasticity across complete life cycles, including within and between generations[14]. In prior work, we found that genetic evolution alters competitive population dynamics of these species over ~10–15 generations[15]. Here, to isolate the influence of plasticity independent of evolution, we used populations of a single, asexually reproducing genotype of each species in our experiments.

In our study, we first manipulated each species' competitive environment, providing the opportunity for hundreds of individuals to plastically respond to communities dominated by either conspecific or heterospecific competitors. These are our 'plasticity-induction' treatments (Fig. 1). After this plasticity-induction phase, we used plants from each treatment in competition experiments designed to parameterize a mathematical model of competitive population dynamics[16]. Our empirically parameterized model provided estimates of the maximum finite rate of growth (λ_i) and sensitivity to interspecific competition (α_{ij}) for each species as the focal species i in each plasticity-induction treatment. Importantly, changes in the values of these population parameters in response to different competitive environments are the only way that plasticity in a focal species can increase its growth rate advantage when rare, when simultaneously accounting for the effects of plasticity in response to a conspecific competitive environment on λ_j and α_{jj} of the common 'resident' competitor (Methods). Therefore, using our estimates of these parameters and accounting for the correlated uncertainty in their estimation, we calculated each species' ability to recover from low density in a community (commonly known as a species' invasion growth rate[2,17]) for each species in each plasticity treatment to determine how plasticity in response to a change in competitive environment influences species coexistence.

Our results show that plasticity in response to heterospecific versus conspecific competitive environments results in higher invasion growth rates in both species (Fig. 2a and Supplementary Fig. 1; posterior probabilities of higher invasion growth rates in the heterospecific versus conspecific plasticity-induction treatments for *S. polyrhiza* and *L. minor*: ~0.99 and ~0.94, respectively). Taken together, the higher invasion growth rates caused large changes in the likely outcome of competition (Fig. 2b). In particular, plasticity in response to interspecific competition reduced the posterior probability of competitive exclusion from near certainty at ~0.99 down to ~0.62, or conversely, increased the posterior probability of coexistence from highly unlikely at ~0.01 up to ~0.38. While there was a larger positive effect of plasticity on *S. polyrhiza* leading to a

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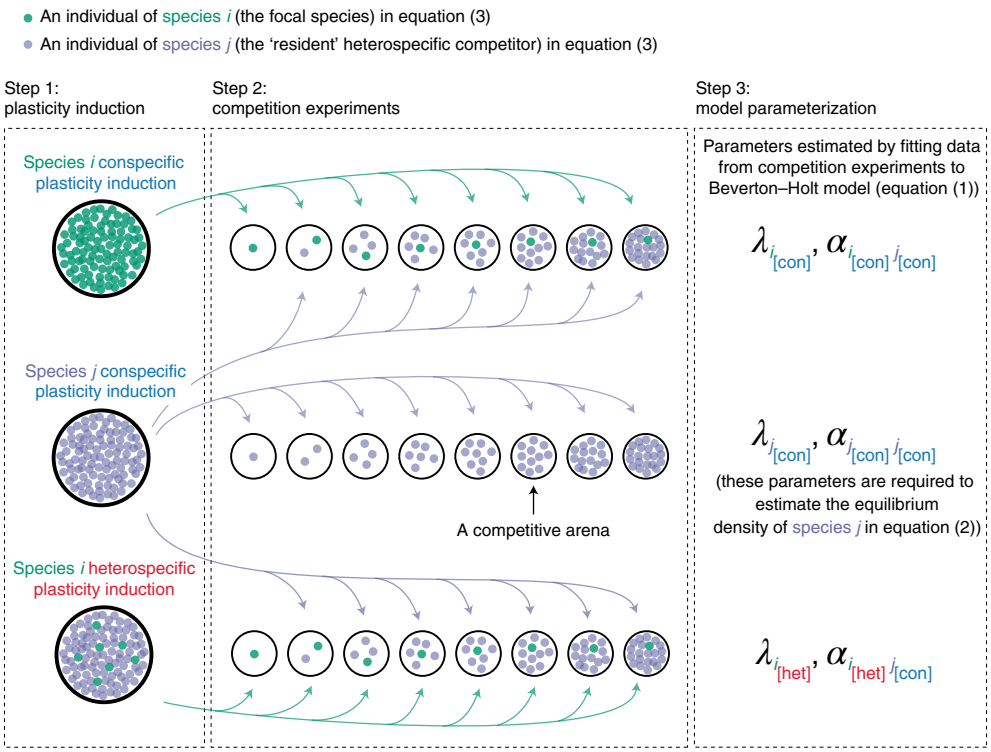

Fig. 1 | Schematic of study design. See Methods for an associated, detailed description of each step and the relationships between each step. Arrows from 'Step 1' to 'Step 2' indicate the source of the individuals of each species used in the competition experiments. Arrow colours correspond to the colours of species *i* and *j*. In 'Step 3', the terms in the subscripted square brackets indicate the plasticity-induction treatment—conspecific (con) or heterospecific (het)—associated with species *i* or *j* for that parameter. The methods were repeated for both species as the focal species *i*.

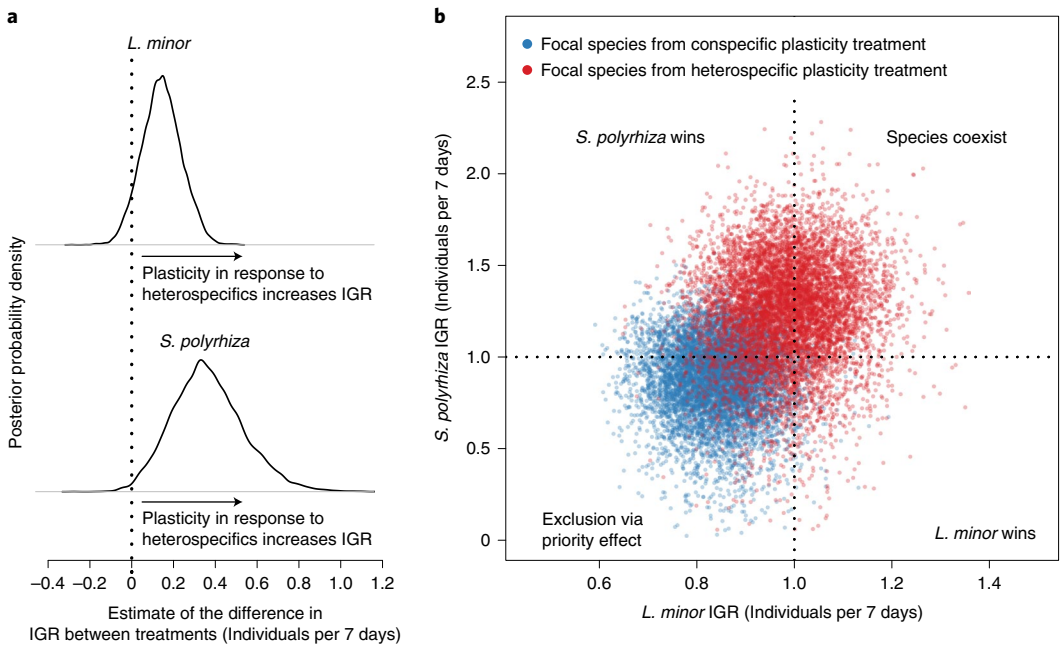

Fig. 2 | Effects of plasticity on species coexistence. a, Posterior probability densities of the difference in the invasion growth rates (IGRs) between plasticity treatments. Positive differences indicate that plasticity in response to interspecific competition causes invasion growth rates of the focal species to increase. **b**, The outcome of competition in each treatment based on mutual invasibility. Points show individual samples from the posterior distributions of the invasion growth rates, with colours indicating the plasticity-induction treatment of the focal invading species. In all cases, the resident heterospecific species was conditioned to its own conspecific plasticity-induction treatment, as would be the case in nature. Black dashed lines perpendicular to each axis indicate the boundary between negative and positive invasion growth rates. Species are expected to coexist when invasion growth rates of both species are positive.

relatively small increase in the posterior probability that *S. polyrhiza* would exclude *L. minor* (from ~0.30 to ~0.44), the very high posterior probabilities of higher invasion growth rates in both species suggests that plasticity in response to heterospecific competitors increases the likelihood that species will coexist (that is, there is an overall shift in the likely outcome of competition from the bottom left towards the top right in Fig. 2b). These results are consistent with observations that competitive exclusion did not occur in replicates of the heterospecific plasticity-induction treatment. The key strength of our experimental–theoretical approach, however, is that we are able to isolate the influence of plasticity on the predicted outcome of competition, independent of other coexistence mechanisms.

Our results provide empirical evidence for a rarely considered pathway to species coexistence. Most recent developments in theoretical and empirical work on coexistence assume that species' traits are fixed on ecological timescales[2,18,19]. On the basis of this assumption, stable coexistence can only arise if fixed species' traits allow intraspecific competition to exceed interspecific competition, or if specific life-history traits interact with environmental variation in ways that buffer species from competitive exclusion[2]. Neither of these conditions account for the possibility that species can rapidly alter their traits in response to changes in competitor identity. Given that trait plasticity in response to competition is common[9] and that there is evidence from other systems that the magnitude of plastic change can vary according to the intensity of interspecific competition[20], our results suggest that plasticity may provide an underexplored mechanism of coexistence.

A dominant framework over the past two decades interprets the tendency of species to coexist in terms of a balance between their niche differences and competitive differences (also known as species' average fitness differences)[19,21–23]. In general, niche differences provide growth rate advantages to rare over common species and thus promote coexistence, while competitive differences always favour one species over another (regardless of a species' relative abundance) and so promote competitive exclusion[2]. Our results suggest that plasticity complicates common theoretical and empirical measures of these quantities. In particular, in simple models of competition, species' maximum finite rates of growth (λ_i) are constant and contribute only to competitive differences driving exclusion[16]. By contrast, we found that plastic changes in λ_i provide an advantage to rare species and ultimately drive much of the observed change in invasion growth rates (Extended Data Fig. 1 and Supplementary Table 2). Thus, what is a driver of competitive exclusion in simple, fixed-trait competition models promotes coexistence when allowing for plasticity. Changes in species' sensitivity to interspecific competition (α_{ij}), which also takes a fixed value in simple competition models, also contributed to the changes in species' invasion growth rates (Extended Data Fig. 1). Similar dynamics have been proposed to occur as a consequence of rapid evolution, where rare species persist by evolving to improve their ability to respond to competition from common species[13,24–27]. Our study shows that these dynamics can emerge via plasticity even in the absence of genetic variance in competitive traits. The general implication of these findings is that while the concepts of niche and competitive differences may be useful, common measures of these quantities may not be useful if organisms rapidly change their traits in response to their competitive environment.

While the effects of plasticity on invasion growth rates were consistent across species, the effects of plasticity on morphological traits were not (Extended Data Fig. 2). For example, specific leaf area (SLA) is a trait commonly associated with competitive interactions between plants[19,28]. In our study, plasticity in response to heterospecific competitors caused the SLA of *L. minor* to increase and the SLA of *S. polyrhiza* to decrease (Extended Data Fig. 2), even though the direction of the effects of plasticity on the demographic and competitive rates of both species were the same. Plastic changes in morphological traits are the typical focus of studies of plasticity, but the contrast in our study between the morphological and the demographic/competitive effects of plasticity highlights the difficulty of making inferences about competitive outcomes from plasticity in morphological traits alone[19]. While other traits may have shown a clearer signal, evidence has suggested that it can be difficult to identify the specific traits driving competitive outcomes[19]. What was consistent between species in our study, however, was that plasticity in response interspecific competition caused the SLA of both species to converge with their heterospecific competitor (Extended Data Fig. 3). Together with our coexistence results, this suggests that trait convergence was associated with a higher likelihood of coexistence. While opposite to the common expectation that coexistence is promoted by trait divergence[23,29], trait convergence may be more likely when organisms compete for essential, non-substitutable resources[30].

Our study has some limitations to consider. First, our experiments were designed to isolate the influence of plasticity on the coexistence of two species in a constant environment. How these effects are mediated by additional species or varying environments deserves further study. Second, the ability of plasticity to promote coexistence will depend on the rate of plastic change relative to the rate of population change[31]; rapid declines in population density may exceed the capacity of plasticity to promote persistence[32]. Third, although the species were subject to high densities of competitors in both plasticity-induction treatments, the conspecific plasticity-induction treatment may have better suppressed growth. This may have affected the ontogeny of individuals in our experiments, which may have also influenced our results. Fourth, we used a single clone of each species in our experiments. More work is required to understand the influence of genotypic variation in plasticity and the interaction between plasticity and evolutionary changes on ecological dynamics[14].

There remains much to be learned about the effects of plasticity on the dynamics of ecological communities[10,33,34]. One of the major difficulties in this area is manipulating the presence versus absence of plasticity to isolate its effects on ecological dynamics[10,33]. We circumvented this technical problem by taking advantage of the importance of changes in the competitive environment for coexistence, allowing us to determine if plastic phenotypic changes buffer species from competitive exclusion when species are perturbed to low density in a community. While there are strong indications in many taxonomic groups that plasticity influences species interactions by changing functional traits, our work extends this effort by experimentally demonstrating that plasticity can stabilize species interactions, promoting species coexistence. Our results suggest that much of the field's focus on coexistence mechanisms assuming fixed traits may miss important processes that regulate the maintenance of species diversity in nature.

Methods

Conceptual and theoretical background. To quantify the ability of two species to coexist, theory commonly relies on the ability of each species to increase from low density in the presence of its heterospecific competitor, which is at its single-species equilibrium density (the mutual invasibility criterion)[17,35]. Invasion growth rates can be estimated empirically by parameterizing a model that describes the dynamics of the competing species. In our study, we use the Beverton–Holt competition model[36], which provided the best fit to our data (Supplementary Fig. 2 and Supplementary Table 1). The model takes the form:

$$N_{i,t+1} = N_{i,t}\frac{\lambda_i}{1 + \alpha_{ii}N_{i,t} + \alpha_{ij}N_{j,t}} \quad (1)$$

where $N_{i,t}$ is the number of individuals of species i at time t, λ_i describes per capita offspring production in the absence of competitors (that is, the maximum finite growth rate) and α_{ii} and α_{ij} are the intra- and interspecific competition coefficients, respectively. A second equation with subscripts reversed describes the dynamics of species j.

Solving equation (1) (with j rather than i as the focal species) for the single-species equilibrium density, $\widehat{N}$, of species j gives:

$$\widehat{N}_j = \frac{\lambda_j - 1}{\alpha_{jj}}. \quad (2)$$

By substituting the right-hand side of equation (2) for $N_{j,t}$ in equation (1) and by replacing $N_{i,t}$ in the denominator of the right-hand side of equation (1) with zero (indicating that the initial density of species i is arbitrarily close to zero), we can derive an expression for the invasion growth rate of species i:

$$\frac{N_{i,t+1}}{N_{i,t}} = \frac{\lambda_i}{1 + \alpha_{ij}\frac{\lambda_j - 1}{\alpha_{jj}}}. \quad (3)$$

This equation demonstrates that the ability of species i to have positive invasion growth rate is determined by the parameters λ_i, α_{ij}, λ_j and α_{jj}.

Most attempts to understand coexistence assume that these parameters are fixed, at least in the absence of environmental variation[2]. However, if plasticity causes these rates to vary depending on the competitive environment, the outcome of competition may change. For example, if species i changes its phenotype in response to high densities of heterospecific competitors, then this may cause an increase in its invasion growth rate by increasing its maximum finite growth rate, λ_i, and/or by decreasing its sensitivity to interspecific competition, α_{ij}. Indeed, as indicated by equation (3), these changes are the only way that plasticity in the focal species i can reduce the likelihood that it will be competitively excluded. Therefore, quantifying how λ_i and α_{ij} change when species change from high to low density in a community (that is, are competing predominantly with conspecifics versus heterospecifics) is the most relevant comparison for understanding if plasticity can buffer species from competitive exclusion by increasing invasion growth rates. Consistent with this logic, in our study we first induced plasticity in each species in response to either conspecifics or heterospecific competitive environments (Fig. 1). We then estimated invasion growth rates for each species as the focal species i as a consequence of changes in λ_i and α_{ij} between these plasticity-induction treatments.

Calculating invasion growth rates also requires an estimate of the equilibrium density of the common 'resident' species, which requires estimates of the resident's λ_j and α_{jj} according to equation (2). Consistent with the logic of an invasion condition where the invader is introduced into a system where the resident is close to its equilibrium density, we estimated these parameters for the resident species j after it had plastically responded to its own conspecific competitive environment as would be the case in a resident-invader scenario in nature. That is, while we do not explicitly quantify the effects of plasticity on α_{jj}, our methods generate estimates of α_{jj} that have been influenced by plasticity in a way consistent with the requirement that coexistence is promoted when intraspecific competition is greater than interspecific competition (that is, $\alpha_{jj} > \alpha_{ij}$; Supplementary Information).

Study species. *L. minor* and *S. polyrhiza* are small, morphologically simple aquatic plants, composed of a floating frond with small rootlets attached to the underside[37,38]. Reproduction occurs via asexual budding and is rapid, with population doubling times of 3–7 days under ideal conditions[37,38]. Experiments were done using single genotypes of each species, which were isolated by culture from single individuals collected from ponds in northern Switzerland. Using single, asexually reproducing genotypes enabled us to isolate the influence of plasticity independent of changes in genotype frequency (that is, evolution). We assume that *de novo* mutations are unlikely to alter population-level demographic rates over the short duration of our study. For our experiments, individuals of each species were first propagated in single-species monocultures in nutrient solution.

Plasticity-induction treatments. The first step in our experimental procedure (Fig. 1) required inducing plasticity in each of our study species in response to conspecific and heterospecific competitive environments. In these 'plasticity-induction' treatments, we exposed individuals of *L. minor* to high densities of itself (*L. minor* conspecific treatment) or we exposed individuals of *L. minor* (at very low densities) to high densities of *S. polyrhiza* (*L. minor* heterospecific treatment; Fig. 1). Similarly, for *S. polyrhiza* we exposed individuals to high densities of itself (*S. polyrhiza* conspecific treatment) or we exposed individuals of *S. polyrhiza* (at very low densities) to high densities of *L. minor* (*S. polyrhiza* heterospecific treatment).

To establish these treatments, we first placed high densities of each species into circular plastic containers (222 mm diameter, 5.8 l), with 37 and 36 containers for *L. minor* and *S. polyrhiza*, respectively. Each container contained 1,250 ml of Hoagland's nutrient solution[39]. The plants were initially at sufficiently high densities to cover the water surface but without excessive overlap of fronds (~1,900 fronds of *L. minor* at ~5.5 fronds cm^{-2}, ~1,300 fronds of *S. polyrhiza* at ~3.8 fronds cm^{-2}). 19 and 18 of the containers for *L. minor* and *S. polyrhiza*, respectively, became the conspecific plasticity-induction treatments. To establish the heterospecific plasticity-induction treatments, we added ~20 fronds of *L. minor* to the remaining 18 high-density *S. polyrhiza* containers (*L. minor* heterospecific treatment) and ~20 fronds of *S. polyrhiza* to the remaining 18 high-density *L. minor* containers (*S. polyrhiza* heterospecific treatment).

We randomly arranged the containers in a climate chamber where they were kept for five weeks, allowing sufficient time for multiple generations to plastically respond to their competitive environment. Our manipulation allows for plasticity to occur both within generations and across generations (that is, transgenerational plasticity). The chambers were on a 16/8 h day/night cycle, with daytime temperatures set at 23 °C and night-time temperatures at 21 °C. During the day, plants were illuminated with fluorescent grow lights. To compensate for evaporation, transpiration and nutrient uptake, 250 ml of nutrient solution was added to each container weekly. During the plasticity-induction period, we regularly redistributed clusters of fronds in the heterospecific plasticity-induction treatment to ensure that local conspecific densities remained low so that these individuals experienced competition predominantly from heterospecifics. We similarly disturbed fronds in the other treatment. In our laboratory setting, this manipulation should mimic small disturbances that these non-sessile floating plants would normally be exposed to (for example, wind on a lake, movement of animals, gentle currents and so on). Nevertheless, if conspecific clumping of rare species persists in nature, then plasticity may occur differently.

We used $n = 9$ replicates of each of plasticity-induction treatment per species to estimate invasion growth rates via a series of competition experiments. The remaining replicates were used to assess the effects of plasticity on morphological traits.

Competition experiments. Step 2 (Fig. 1) in our experimental procedure required parameterizing a model of competitive population dynamics for the species in each plasticity-induction treatment. Doing so allowed us to estimate invasion growth rates. We could have measured growth rates of small numbers of individuals from each treatment introduced into equilibrium densities of the heterospecific competitor to estimate invasion growth rates[17]. However, our approach also allows us to identify which components (demographic and competitive rates) of a species' invasion growth rate are influenced by plasticity. Therefore, after the plasticity-induction phase, we included the individuals from each plasticity treatment in competition experiments designed to parameterize a dynamic model of competitive population dynamics, according to established methods[15,16]. These methods rely on measurements of short-term species' growth rates across a gradient of competitor densities[16,19,40,41].

For a single replicate, we first describe how we assessed the effects of plasticity in *L. minor* when competing with *S. polyrhiza* as the resident species (that is, when *L. minor* is species i in equation (3)). We allowed low densities (0.65 individuals cm^{-2}) of *L. minor* that had been growing in a single replicate of either the conspecific or heterospecific plasticity-induction treatment to grow and compete for one week against a range of densities (0, 1.3, 3.3, 7.4, ~15.8, ~23.3, ~28.7, ~34 individuals cm^{-2}) of *S. polyrhiza* that had been growing in a single replicate of its own conspecific plasticity-induction treatment. Each density combination was placed in a single competitive arena—an open-ended vertical tube, 2.8 cm in diameter—that was inserted into a polystyrene frame floating in a large plastic tub (64 × 36 × 20 cm) that was filled to 15 cm depth with Hoagland's nutrient solution. The exact number of individuals placed in each competitive arena was assessed via manual counting (for small numbers of individuals) or from image analysis of photographs (for large numbers of individuals). Image analysis was done using the 'imfindcircles' function in MATLAB[42]. After seven days, we counted the final population size of the focal species in each competitive arena. We then calculated the population growth rate in each density combination (that is, $N_{i,t+1}/N_{i,t}$, with time step equal to seven days).

Using a model fitting process described below, we fit the competition model (equation (1)) to these data to estimate the maximum finite growth rate of *L. minor* (λ_L) and *L. minor*'s response to interspecific competition from *S. polyrhiza* (α_{LS}) for each *L. minor* plasticity-induction treatment. Here, the subscripts L and S refer to *L. minor* and *S. polyrhiza*, respectively. In additional competitive arenas placed in the same tub, we grew *S. polyrhiza* alone at low density (0.325 individuals cm^{-2}), as well as across the same range of *S. polyrhiza* densities listed above. Individuals of *S. polyrhiza* used to create this density gradient were taken from the same replicate of the conspecific plasticity-induction treatment used for the *L. minor* competition trials (Fig. 1). This single-species density gradient enabled us to estimate λ_S and α_{SS} from the conspecific plasticity-induction treatment, as is required to estimate *S. polyrhiza*'s equilibrium density (equation (2)). Thus, the complete set of competition trials for a single replicate of the plasticity-induction treatments allowed us to estimate *L. minor* invasion growth rates in each plasticity treatment following equation (3).

We repeated these experiments in the same plastic tub for a single replicate of *S. polyrhiza* as the focal species. Here we allowed low densities of *S. polyrhiza* (0.325 individuals cm^{-2}) from each treatment to compete against a range of densities of *L. minor* from its own conspecific plasticity treatment (0, 2.9, 8.4, ~19.5, ~35.6, ~53.6, ~67.7, ~83.4 individuals cm^{-2}), and we also grew *L. minor* from the conspecific plasticity treatment alone at low density (0.65 individuals cm^{-2}) and alone at each of the *L. minor* densities listed above. These competition trials enabled us to estimate λ_S and α_{SL} in each plasticity-induction treatment and λ_L and α_{LL} for *L. minor* as the resident species. The entire procedure for both species as focal species was repeated in separate tubs for each of the nine replicates.

We cannot rule out additional plastic changes occurring during the one-week duration of our competition experiments, and our parameter estimates implicitly

include any influence of plasticity that did occur during this time. To the extent that plasticity did occur during these experiments, this should cause the performance of individuals from the different plasticity treatments to converge (because they are exposed to the same range of densities, regardless of plasticity induction), thus weakening the effects of our plasticity-induction treatment.

Model fitting and analyses of effects of plasticity on coexistence. We fit the competition model (equation (1)) to the growth rate data from the competition experiments. This is a common approach for parameterizing dynamic models in a range of taxa[16,19,40,41], including in our system where this approach has been shown to do an excellent job of predicting longer-term, multigenerational competitive population dynamics[15]. We fit the model using a Bayesian analytical approach using Stan[43] together with the R[44] package brms v.2.13.5 (ref. [45]). We describe the details below, but first note that we initially fit seven candidate competition models to the data. Model selection via leave-one-out cross validation using the loo[46] package in R indicated that the Beverton–Holt model (equation (1)) provided the best fit to our data (Supplementary Fig. 2 and Supplementary Table 1). We proceed with the description of our analyses on that basis, although we note that our qualitative results did not change regardless of which of the three best-performing models were selected (Supplementary Fig. 3).

A single Beverton–Holt model was fit with species, initial conspecific and heterospecific competitor density in the competition trials and plasticity-induction treatment as predictors (Supplementary Information). We included plasticity-induction replicate ($n = 9$) as a random 'blocking' factor in our competition model fits. For the lambda (λ_i) terms, we used uniform priors bounded between 0 and 5. For the competition coefficients (α_{ii} and α_{ij}), we used uniform priors bounded between 0 and 0.05. These priors constrain competition coefficients to be positive (that is, competitive, not facilitative), which is biologically reasonable given previous work on interactions between these species[15]. More generally, these prior distributions broadly reflect the direction and magnitude of demographic and competitive rates previously observed in this system[15], and constraining priors to be positive was not only biologically reasonable but improved model convergence. We used half Student's-t priors for the random-effect terms (the default in brms). The distribution of our response variable (growth rates all positive real numbers) suggested a log-normal likelihood, which required that we log transform the right-hand side of the model to preserve its nonlinear functional form. To fit the models, we ran 4 Markov chain Monte Carlo (MCMC) chains for 4,000 iterations with a warm-up period of 2,000 iterations, for a total of 8,000 samples. We checked for model convergence by visual analysis of traceplots and assessment of convergence diagnostics (potential scale reduction factor ($\widehat{R}$) = ~1) and we used graphical posterior predictive checks to assess the adequacy of the final model.

To assess the effects of plasticity on coexistence, we estimated each species' invasion growth rate according to equation (3) using each of the 8,000 correlated samples of the competition model parameters for each species in each treatment from our model fit. This approach allowed us to estimate the propagated uncertainty in our estimate of each species' invasion growth rate in each treatment, accounting for the correlated uncertainty in our estimates of the underlying competition model parameters. For each species, we then derived the posterior distributions of the difference in invasion growth rates between plasticity treatments, again accounting for the correlated uncertainty in the estimates of the invasion growth rates themselves. The posterior distributions of the difference in invasion growth rates between plasticity treatments allowed us to assess the plausibility that plasticity in response to heterospecific competition promotes coexistence by increasing invasion growth rates.

Trait measurements and analyses. We haphazardly sampled ~60 and ~30 fronds of *L. minor* and *S. polyrhiza*, respectively, from each of the additional replicates of each treatment. We photographed these fronds and quantified total frond area in each sample using ImageJ[47]. We quantified area per frond by dividing total frond area by the number of fronds in the sample. We assessed root length as the longest root of a single, haphazardly chosen cluster of fronds from each replicate. We measured dry mass by first removing all roots and turions (*S. polyrhiza* dormant resting stages[37]) and then dried the fronds at 70 °C for 24 hours before weighing. With these data, we calculated SLA and the ratio of root length to frond dry mass, with one estimate per replicate. We assessed the effects of the plasticity treatments on each trait using linear models with species and plasticity-induction treatment as the predictors and improper flat priors (a uniform distribution over the real numbers, which is the default 'uninformative' prior in brms) on each coefficient. We modelled SLA using a Gaussian likelihood and the ratio of root length to frond dry mass using a Gamma likelihood with a log-link function.

We also assessed how plasticity affected the difference in SLA between the competing species. These analyses were done in a manner consistent with the character-displacement literature, which tends to expect trait divergence in response to interspecific competition[23]. In our experiments, this expectation means that trait differences between species should be greater when species are able to plastically respond to their heterospecific competitor (that is, when the focal species comes from the heterospecific plasticity-induction treatment). To determine if plasticity caused traits to diverge, for each species as the focal species we derived the posterior probability distribution of the difference between plasticity treatments in the magnitude of the trait difference from the heterospecific competitor's trait values (Extended Data Fig. 3). These distributions were derived by calculating the difference between the SLA estimates from the posterior distributions of SLA from the linear model described above. Greater interspecific trait differences in the heterospecific plasticity treatment would indicate trait divergence because of plasticity in response to interspecific competition.

Reporting summary. Further information on research design is available in the Nature Research Reporting Summary linked to this article.

Data availability

The data that support the findings of this study are available via Zenodo at https://doi.org/10.5281/zenodo.5726004.

Code availability

The R code used to analyse the data is available via Zenodo at https://doi.org/10.5281/zenodo.6844013.

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References

1. Pigliucci, M. *Phenotypic plasticity: Beyond Nature and Nurture* (Johns Hopkins Univ. Press, 2001).
2. Chesson, P. Mechanisms of maintenance of species diversity. *Annu. Rev. Ecol. Syst.* **31**, 343–366 (2000).
3. Aerts, R., Boot, R. G. A. & Van Der Aart, P. J. M. The relation between above- and belowground biomass allocation patterns and competitive ability. *Oecologia* **87**, 551–559 (1991).
4. Ashton, I. W., Miller, A. E., Bowman, W. D. & Suding, K. N. Niche complementarity due to plasticity in resource use: plant partitioning of chemical N forms. *Ecology* **91**, 3252–3260 (2010).
5. Pfennig, D. W., Rice, A. M. & Martin, R. A. Ecological opportunity and phenotypic plasticity interact to promote character displacement and species coexistence. *Ecology* **87**, 769–779 (2006).
6. van Kleunen, M. & Fischer, M. Adaptive evolution of plastic foraging responses in a clonal plant. *Ecology* **82**, 3309–3319 (2001).
7. Relyea, R. A. Competitor-induced plasticity in tadpoles: consequences, cues, and connections to predator-induced plasticity. *Ecol. Monogr.* **72**, 523–540 (2002).
8. Broekman, M. J. E. et al. Signs of stabilisation and stable coexistence. *Ecol. Lett.* **22**, 1957–1975 (2019).
9. Callaway, R. M., Pennings, S. C. & Richards, C. L. Phenotypic plasticity and interactions among plants. *Ecology* **84**, 1115–1128 (2003).
10. Turcotte, M. M. & Levine, J. M. Phenotypic plasticity and species coexistence. *Trends Ecol. Evol.* **31**, 803–813 (2016).
11. Chesson, P. in *Unity in Diversity: Reflections on Ecology after the Legacy of Ramon Margalef* (eds F. Valladares et al.) 119–164 (Fundación Banco Bilbao Vizcaya Argentaria, 2008).
12. Ellner, S. P., Snyder, R. E. & Adler, P. B. How to quantify the temporal storage effect using simulations instead of math. *Ecol. Lett.* **19**, 1333–1342 (2016).
13. Vasseur, D. A., Amarasekare, P., Rudolf, V. H. W. & Levine, J. M. Eco-evolutionary dynamics enable coexistence via neighbor-dependent selection. *Am. Nat.* **178**, E96–E109 (2011).
14. Hendry, A. P. Key questions on the role of phenotypic plasticity in eco-evolutionary dynamics. *J. Hered.* **107**, 25–41 (2016).
15. Hart, S. P., Turcotte, M. M. & Levine, J. M. Effects of rapid evolution on species coexistence. *Proc. Natl Acad. Sci. USA* **116**, 2112–2117 (2019).
16. Hart, S. P., Freckleton, R. P. & Levine, J. M. How to quantify competitive ability. *J. Ecol.* **106**, 1902–1909 (2018).
17. Grainger, T. N., Levine, J. M. & Gilbert, B. The invasion criterion: a common currency for ecological research. *Trends Ecol. Evol.* **34**, 925–935 (2019).
18. Letten, A. D., Ke, P.-J. & Fukami, T. Linking modern coexistence theory and contemporary niche theory. *Ecol. Monogr.* **87**, 161–177 (2017).
19. Kraft, N. J. B., Godoy, O. & Levine, J. M. Plant functional traits and the multidimensional nature of species coexistence. *Proc. Natl Acad. Sci. USA* **112**, 797–802 (2015).
20. Pfennig, D. W. & Murphy, P. J. How fluctuating competition and phenotypic plasticity mediate species divergence. *Evolution* **56**, 1217–1228 (2002).
21. Adler, P., HilleRisLambers, J. & Levine, J. A niche for neutrality. *Ecol. Lett.* **10**, 95–104 (2007).
22. Barabás, G., D'Andrea, R. & Stump Simon, M. Chesson's coexistence theory. *Ecol. Monogr.* **88**, 277–303 (2018).
23. Pfennig, D. W. & Pfennig, K. S. *Evolution's Wedge: Competition and the Origins of Diversity* (Univ. California Press, 2012).
24. Ayala, F. J. Reversal of dominance in competing species of *Drosophila*. *Am. Nat.* **100**, 81–83 (1966).

25. Pease, C. M. On the evolutionary reversal of competitive dominance. *Evolution* **38**, 1099–1115 (1984).
26. Pimentel, D., Feinberg, E. H., Wood, P. W. & Hayes, J. T. Selection, spatial distribution, and the coexistence of competing fly species. *Am. Nat.* **99**, 97–109 (1965).
27. Lankau, R. A. & Strauss, S. Y. Mutual feedbacks maintain both genetic and species diversity in a plant community. *Science* **317**, 1561–1563 (2007).
28. Kunstler, G. et al. Plant functional traits have globally consistent effects on competition. *Nature* **529**, 204–207 (2016).
29. Stuart, Y. E. & Losos, J. B. Ecological character displacement: glass half full or half empty? *Trends Ecol. Evol.* **28**, 402–408 (2013).
30. Abrams, P. A. Alternative models of character displacement and niche shift. 2. Displacement when there is competition for a single resource. *Am. Nat.* **130**, 271–282 (1987).
31. Chevin, L. M., Lande, R. & Mace, G. M. Adaptation, plasticity, and extinction in a changing environment: towards a predictive theory. *PLoS Biol.* **8**, e1000357 (2010).
32. Harmon, E. A. & Pfennig, D. W. Evolutionary rescue via transgenerational plasticity: evidence and implications for conservation. *Evol. Dev.* **23**, 292–307 (2021).
33. Forsman, A. Rethinking phenotypic plasticity and its consequences for individuals, populations and species. *Heredity* **115**, 276–284 (2015).
34. Brass, D. P. et al. Phenotypic plasticity as a cause and consequence of population dynamics. *Ecol. Lett.* **24**, 2406–2417 (2021).
35. Macarthur, R. H. & Levins, R. The limiting similarity, convergence, and divergence of coexisting species. *Am. Nat.* **101**, 377–385 (1967).
36. Beverton, R. J. H. & Holt, S. J. *On the Dynamics of Exploited Fish Populations* (UK Ministry of Agriculture, Fisheries and Food, 1957).
37. Landolt, E. *Biosystematic Investigations in the Family of Duckweeds (Lemnaceae), Vol. 2*: *The Family of Lemnaceae—A Monographic Study,* Vol.1 (Geobotanischen Institute, ETH Zürich, 1986).
38. Wang, W. et al. The *Spirodela polyrhiza* genome reveals insights into its neotenous reduction fast growth and aquatic lifestyle. *Nat. Commun.* **5**, 3311 (2014).
39. Hoagland, D. R. & Arnon, D. I. *The Water-Culture Method for Growing Plants without Soil* (College of Agriculture, Agricultural Experiment Station, Univ. California, 1950).
40. Inouye, B. D. Response surface experimental designs for investigating interspecific competition. *Ecology* **82**, 2696–2706 (2001).
41. Law, R. & Watkinson, A. R. Response-surface analysis of two-species competition: an experiment on *Phleum arenarium* and *Vulpia fasciculata*. *J. Ecol.* **75**, 871–886 (1987).
42. MATLAB v.9.0 (MathWorks, 2016).
43. *Stan Modeling Language Users Guide and Reference Manual*, v.2.27 (Stan Development Team, 2021); https://mc-stan.org
44. Vehtari, A., Gelman, A. & Gabry, J. Practical Bayesian model evaluation using leave-one-out cross-validation and WAIC. *Stat. Comput.* **27**, 1413–1432 (2017).
45. Bürkner, P.C. brms: an R package for Bayesian multilevel models using Stan. *J. Stat. Softw.* https://doi.org/10.18637/jss.v080.i01 (2017).
46. Vehtari, A. et al. loo: efficient leave-one-out cross-validation and WAIC for Bayesian models, v.2.4.1 (2020).
47. ImageJ (US NIH, 1997–2016).

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Author contributions

C.H., J.M.L., M.M.T. and S.P.H. conceived the problem and designed the experiments; C.H. did the experiments; S.P.H. did the analyses; C.H. and S.P.H. wrote the first draft of the manuscript; S.P.H. led the subsequent writing and all authors contributed to revisions.

Competing interests

The authors declare no competing interests.

Additional information

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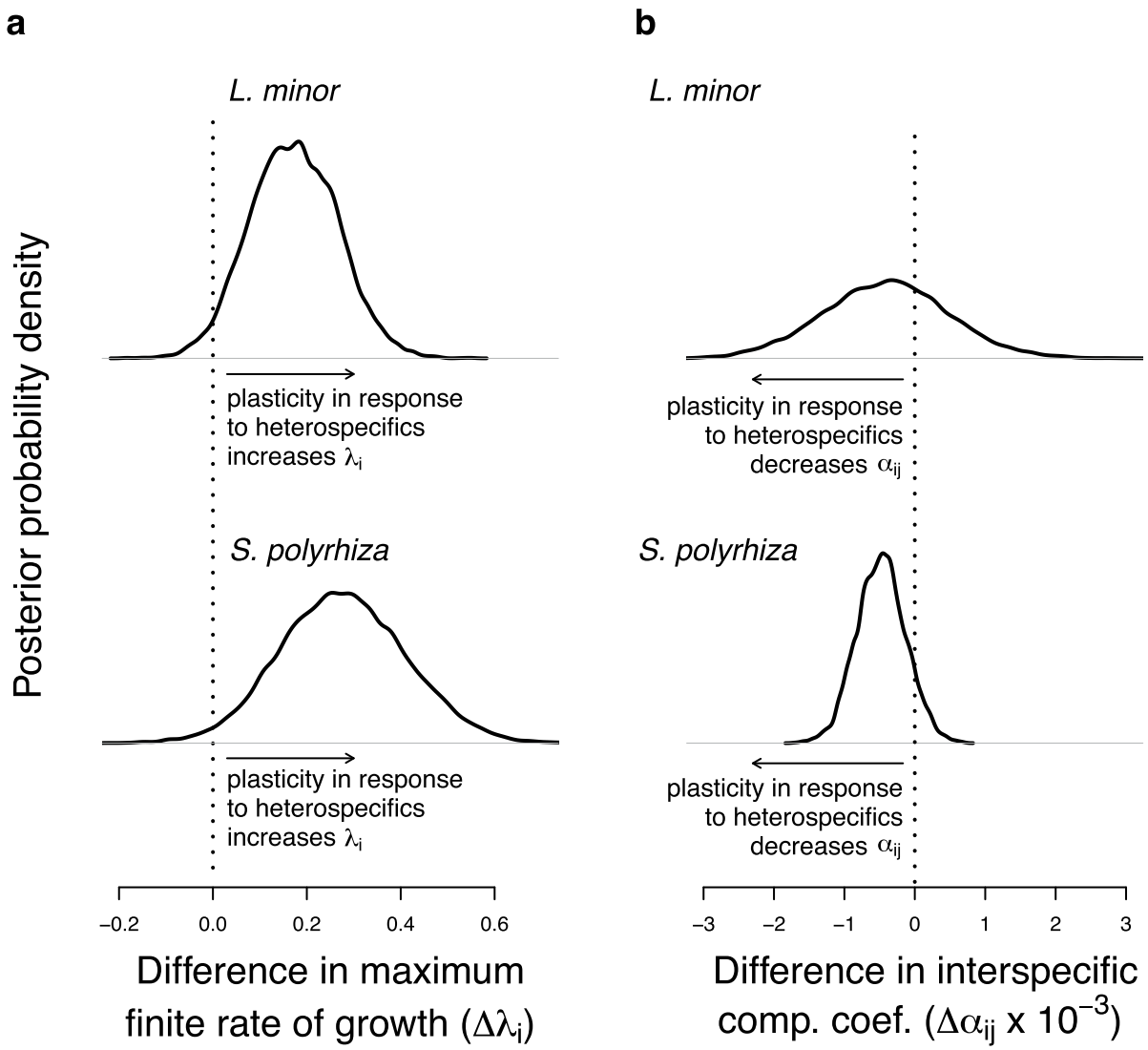
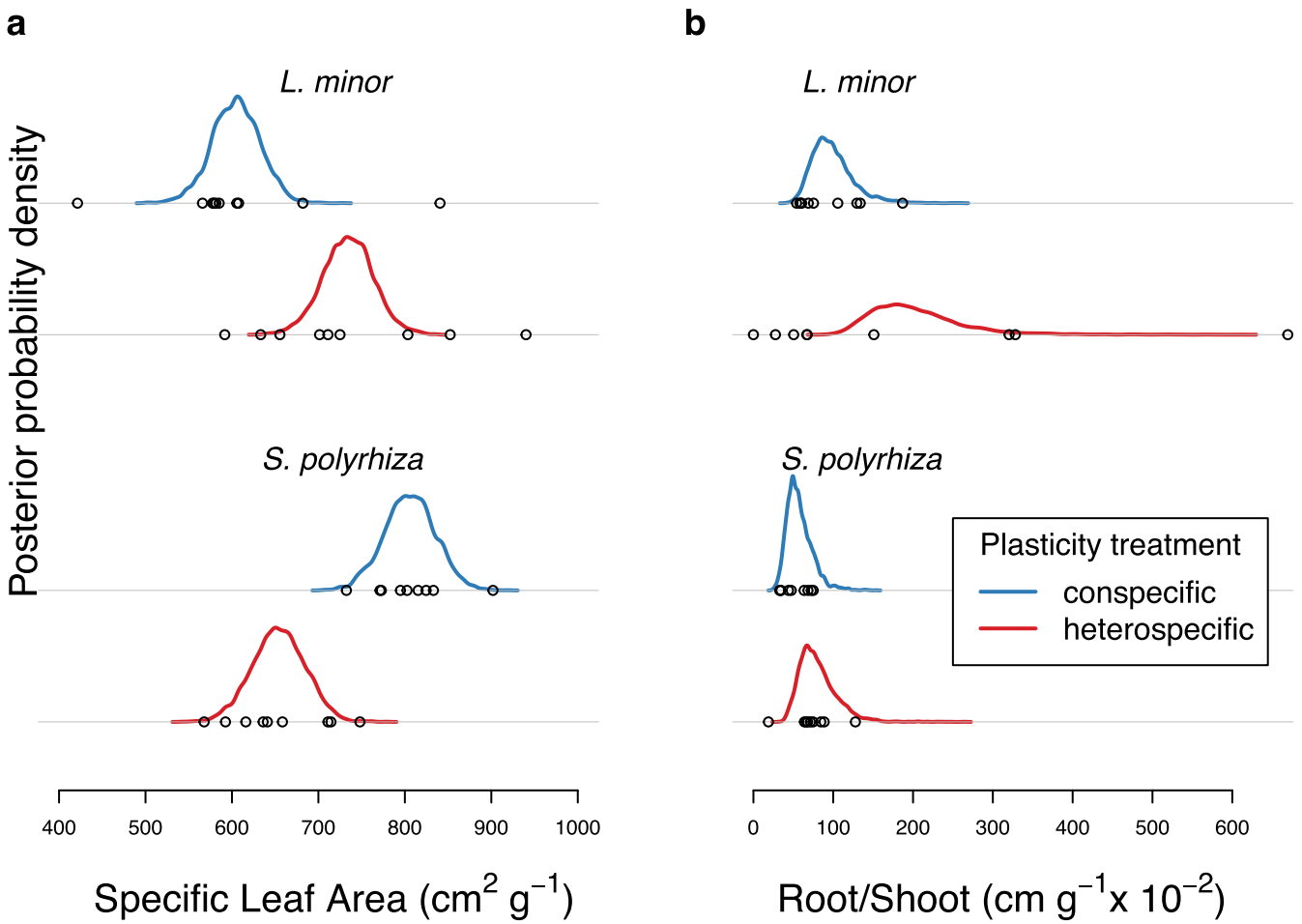
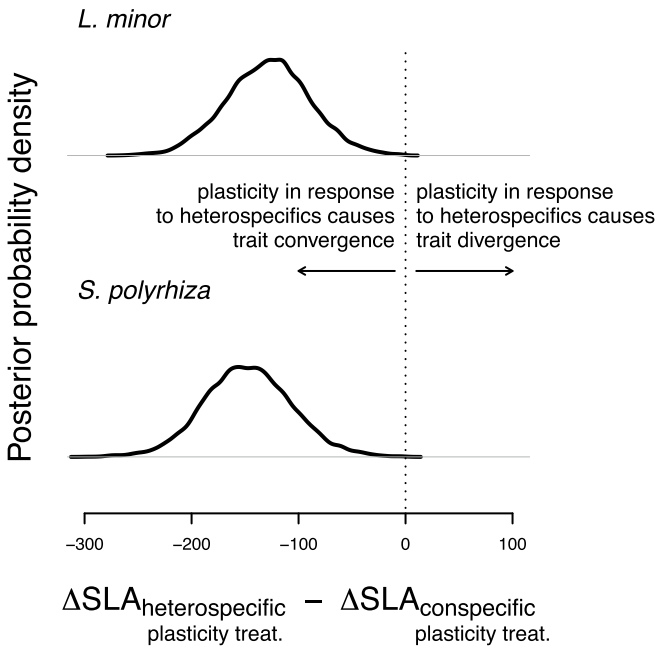


Extended Data Fig. 1 | The effect of plasticity on demographic and competitive rates. a) Maximum finite rate of growth (λ_i); and b) sensitivity of focal species *i* to interspecific competition (α_{ij}). Plots show the posterior probability densities of the difference in these competition model parameters between treatments, accounting for correlations in the posterior distributions of the parameters. The vertical dotted lines indicate zero difference between treatments. Positive values indicate parameter values are higher in the heterospecific plasticity-induction treatment. Higher λ_i and lower α_{ij} for focal species *i* in the heterospecific plasticity-induction treatment will increase invasion growth rates (see equation 3), and therefore the arrows below the plots indicate the direction of parameter change that would favour coexistence by increasing invasion growth rates. Posterior probabilities for increases in each species maximum finite rate of growth (λ_i) were 0.98 and 0.97 for *S. polyrhiza* and *L. minor*, respectively. Posterior probabilities for decreases in each species' sensitivity to interspecific competition were 0.92 and 0.68 for *S. polyrhiza* and *L. minor*, respectively. Point estimates (means) and variability (standard deviations and 2.5 & 97.5 quantiles) calculated from the posterior distributions for each competition model parameter are provided in Supplementary Table 2.



Extended Data Fig. 2 | The effect of plasticity on functional traits. a) Specific leaf area (SLA); and b) the ratio of root length to frond biomass. Plots show the posterior probability densities for the estimate of each trait value, for each species and plasticity treatment. Raw data are shown as points on the x-axis.



Extended Data Fig. 3 | The effect of plasticity on trait divergence. The plots show the posterior probability densities of the difference in specific leaf area (SLA) between the heterospecific competitor and a focal species from the heterospecific vs. conspecific plasticity-induction treatments. The traits of the heterospecific competitor were measured after its own plasticity in response to itself (that is in its own conspecific plasticity-induction treatment). Positive values indicate that trait differences between species increase (that is, there is trait divergence) when the focal species is exposed to interspecific competition (that is, in the heterospecific plasticity treatment) and negative values indicate plasticity causes trait differences between species to decrease (that is, there is trait convergence) when the focal species is exposed to interspecific competition. These contrasting possibilities are indicated by the arrows either side of the dotted vertical line, which represents zero trait divergence/convergence.

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Software and code

Policy information about availability of computer code

Data collection

MATLAB v. 9.0 (The MathWorks Inc., Natick, Massachusetts, 2016) was used to assist in counts of individuals in the competition trials.

The open-source image analysis software ImageJ (version 1.51) was used to assess frond area.
Citation (also appears in manuscript): Rasband, W.S., ImageJ, U. S. National Institutes of Health, Bethesda, Maryland, USA, https://imagej.nih.gov/ij/, 1997-2018.

Data analysis

macOS Big Sur 10.16

R version 4.0.4 (2021-02-15)
citation: R Core Team (2021). R: A language and environment for statistical computing. R Foundation for Statistical Computing, Vienna, Austria. URL https://www.R-project.org/
Platform: x86_64-apple-darwin17.0 (64-bit)

Attached base R packages: parallel, stats, graphics, grDevices utils, datasets, methods, base
Other attached R packages (name_version, and not including dependencies): other attached packages (name_version): brms_2.16.1, cluster_2.1.0, cmdstanr_0.4.0, coda_0.19-4, devtools_2.4.2, ggplot2_3.3.5, gridExtra_2.3, lme4_1.1-26, MASS_7.3-53, Matrix_1.3-2, mvtnorm_1.1-2, nlme_3.1-152, Rcpp_1.0.7, rethinking_2.13, rpart_4.1-15, rstan_2.21.1, sparr_2.2-15, spatstat_2.2-0, spatstat.core_2.3-0, spatstat.data_2.1-0, spatstat.geom_2.2-2, spatstat.linnet_2.3-0, StanHeaders_2.21.0-7, usethis_2.0.1

Stan version 2.27
citation: Stan Development Team (2021) Stan modeling language users guide and reference manual. doi:https://mc-stan.org

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The data that support the findings of this study are available in a Zenodo repository at http://doi.org/10.5281/zenodo.5726004.

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Study description

The methods, including all treatment factors, interactions, design structures, and nature and number of experimental units and replicates are described in detail in the Methods and Supplementary Materials of the manuscript, including in Supplementary Fig. 1. Here we provide an abbreviated summary of the most salient details, but please refer to the manuscript for full description of all details.

The study assesses the effect of phenotypic plasticity in response to interspecific competition on the ability of two species of floating aquatic plants (Lemna minor and Spirodela polyrhiza) to coexist. There was one experimental treatment with two levels (high-frequency and low-frequency plasticity-induction). For our coexistence analyses there were n=9 replicates. For our assessment of the effects of plasticity on morphological traits, there were n = 9 replicates for S. polyrhiza in each treatment, and n=10 and n=9 replicates for L. minor in the high- and low-frequency plasticity-induction treatments, respectively. Please see Methods for full description of the experimental units during the plasticity-induction phase of the study.

Individuals of both species from the plasticity-induction treatments associated with our coexistence analyses (n=9 for both species, as described above) were included in a series of competition trials. These competition trials generated data which were then fit to a model of competitive population dynamics. Please see Methods for full description of the experimental units for these competition trials. We fit all the data from the competition trials from all plasticity-induction replicates in a single model fit and included replicate as a random 'blocking' factor in our analyses (i.e. our statistical model was hierarchical) to account for the structure in our experimental design.

Research sample

We used Lemna minor and Spirodela polyrhiza as the study species in our experiments. These species are small aquatic plants belonging to the Araceae family. The plants are morphologically simple, composed of a floating frond with small rootlets attached to the underside. Reproduction occurs via asexual budding of daughter fronds and is rapid, with population doubling times of 3-7 days under ideal conditions. All experiments were done using a single genotype of each species, which were isolated by culture from single individuals collected from ponds in northern Switzerland. The Lemna minor clone was collected by Simon Hart and Martin Turcotte from a pond in Leuggern, Aargau, Switzerland. The Spirodela polyrhiza clone was provided by ZHAW Zurich University of Applied Sciences, and was also originally collected from a pond in Zurich, Switzerland. The S. polyrhiza clone is maintained in single-

genotype culture in the Landolt Duckweed Collection (http://www.duckweed.ch/) in Zurich, Switzerland.

Using single asexually-reproducing genotypes enabled us to isolate the influence of plasticity independent of changes in genotype frequency (i.e., evolution) in our experiments. For our experiments, individuals of each species were first propagated in single-species monocultures in nutrient solution.

Sampling strategy

Our study is based on an experiment with one experimental treatment with two treatment levels (high-frequency and low-frequency plasticity-induction). As described above, for our coexistence analyses there were n=9 replicates for each species in each treatment. These nine-replicates were included as a random 'blocking' factor in competition experiments used to parameterize a model of competitive population dynamics on which our conclusions are based. For our assessment of the effects of plasticity on morphological traits, there were n=9 replicates for S. polyrhiza in each treatment, and n=10 and n=9 replicates for L. minor in the high- and low-frequency plasticity induction treatments, respectively.

We did not use specific statistical methods to predetermine sample sizes. Sample sizes were determined based on previous work on our study system and had sufficient power to identify biologically-meaningful changes in the outcome of competition and in species' traits. Moreover, it is our understanding that estimating invasion growth rates of vascular plants via regression analyses using data from nine independent replicates across multiple experimental treatment levels represents a very high level of experimental effort in our field.

Data collection

The data on which our coexistence conclusions are based consists of measurements of population sizes of each species of plant in a series of competitive arenas at two time-points (initial [day 0] and final [day 7]). Each competitive arena contained a different combination of species and densities taken from different replicates of our plasticity-induction treatments (see Methods for full details). At the beginning of the competition trials, the exact number of individuals placed in each competitive arena was assessed via manual counting when populations were small (for L. minor populations of less than 46 individuals and for S. polyrhiza populations of less than 117 individuals), or from image analysis of photographs when populations were large. After 7 days we counted the final population size of the focal species in each competitive arena. Again, when the numbers of individuals of the focal species were small counts were done manually and directly, and when numbers of individuals were large, we took photographs and subsequently counted the numbers of individuals in these photographs using image analyses. Counts of individuals from photographs were done with the assistance of the MATLAB function, 'imfindcircles'. Manual counts were done by Mr Cyrill Hess (first author) and research assistants (Andrea Reid, Annette Bieger, Marc-Jacques Mächler, Simon Schmid, Laurie Willi, Mateus de Oliveira Negreiros). Image analysis was done by Mr Cyrill Hess. Data were added directly to an Excel spreadsheet.

Trait data was also collected for each species in each replicate of each plasticity-induction treatment. These measurements were done by haphazardly sampling ~60 and ~30 fronds of L. minor and S. polyrhiza, respectively, from each of the additional replicates of each treatment. We photographed these fronds and quantified total frond area in each sample using the open-source image analysis software, ImageJ. Area per frond was quantified by dividing total frond area by the number of fronds in the sample. Root length was assessed as the longest root of a single haphazardly chosen cluster of fronds from each replicate. Dry mass was estimated by first removing all roots and turions (S. polyrhiza dormant resting stages) and then drying the fronds at 70□C for 24 hours before weighing. All trait measurement were done by Mr Cyrill Hess (first author), with technical assistance from Andrea Reid and Annette Bieger.

Timing and spatial scale

The plasticity induction period began on 8th and 9th March, 2018. Plasticity-induction occurred in circular plastic containers (222 mm diameter, 5.8 L). The competition experiments were initiated between the 10th and 12th April, 2018, and were concluded 7 days later. Each density combination in the competition trials was placed in a single competitive arena – an open-ended vertical tube, 2.8 cm diameter – that was inserted into a polystyrene frame floating in a large plastic tub (64□36□20 cm) that was filled to 15cm depth with Hoagland's nutrient solution. Because of the large number of competition trials it was not possible to initiate the plasticity-induction treatments, and initiate and conclude the competition trials, for all replicates on a single day. However, the plasticity manipulations and competition trials for each replicate were set up and concluded over the course of a few hours, justifying our inclusion of a random replicate-level 'blocking' factor when fitting our competition data to the model of competitive population dynamics.

Trait measurements (photographs of frond area, and root and dry-weight measurements) were done between 12th and 14th April, 2018 (i.e. these measurements were done in the days between the initiation and conclusion of the competition trials).

Data exclusions

As described in our methods: "We note that we excluded from our analyses one data point from one replicate that showed an unusually high growth rate of L. minor in response to competition from S. polyrhiza. Excluding this data point was conservative for showing effects of plasticity on coexistence, as omitting this point decreased the difference in invasion growth rates between plasticity treatments. For reproducibility, we have included this data point in our published dataset." This decision was not based on pre-established exclusion criteria. As noted, we have included this data point in our published dataset.

In addition, we note that we initially set up 10 (rather than 9) replicates of the low-frequency plasticity-induction treatment for measurements of L. minor traits. However, in one replicate (replicate 4) we failed to take a photograph of the fronds after the plasticity-induction phase. This meant that we were unable to assess frond area in this replicate, which subsequently meant that we were unable to estimate SLA or root-shoot ratios for this replicate. It is for this reason that replicate 4 is missing from our published dataset (although dry-weight and root-length measurements can be provided for this replicate if requested).

Reproducibility

We initiated the experiment twice. The first run of the experiment (in December 2017) did not produce interpretable results because of high, unexplained mortality of both species across replicates and treatments during the plasticity-induction phase. This run of the experiment was done in a shared greenhouse at the ETH Research Station for Plant Sciences in Lindau, Switzerland. For the second run of the experiment we moved to a dedicated growth chamber at the Zurich campus of ETH Zürich. It is the second run of the experiment that we present in the current manuscript. We have not repeated the experiment subsequently.

Randomization

Initially, all of the 37 containers of L. minor and 36 containers of S. polyrhiza were treated the same way – that is, high densities of each species were added to each container from our single-species monoculture stocks. Each container was then haphazardly assigned to either the low-frequency or high-frequency plasticity-induction treatment. All containers were then randomly arranged in a climate chamber for the plasticity-induction period. Random arrangement of containers in the climate chamber was done by assigning random numbers associated with different locations in the growth chamber to the plasticity-induction containers.

For the competition trials, individuals from each replicate of the appropriate plasticity-induction treatment were haphazardly assigned to each competitive arena in the competition trials. Treatments and density combinations were randomly arranged in tubs for the competition experiments. Random arrangement was done using random numbers to assign competitive arenas to different locations within each floating raft in each tub.

Blinding

Competitive arenas were randomly arranged within tubs. Final populations within competitive arenas were quantified with reference to the position of the competitive arenas within tubs and/or the order with which photographs of the competitive arenas were taken, but without reference to the plasticity-induction treatment associated with each competitive arena. Thus, our estimates of final population sizes on which all our main conclusions rest were blind with respect to the levels of the plasticity-induction treatment.

Measurements of frond weight and root-length were not blind to experimental treatment. However, measurements of frond area (and thus SLA and root-shoot ratios – our traits of interest) were partially blind in that these measurements were done without explicit reference to experimental treatments by post-processing of photos using image analysis software (ImageJ), which enables automated estimation of area.

Did the study involve field work? ☐ Yes ☒ No

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☒	☐ Dual use research of concern

Methods

n/a	Involved in the study
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