Review

Why intraspecific trait variation matters in community ecology

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Natural populations consist of phenotypically diverse individuals that exhibit variation in their demographic parameters and intra- and inter-specific interactions. Recent experimental work indicates that such variation can have significant ecological effects. However, ecological models typically disregard this variation and focus instead on trait means and total population density. Under what situations is this simplification appropriate? Why might intraspecific variation alter ecological dynamics? In this review we synthesize recent theory and identify six general mechanisms by which trait variation changes the outcome of ecological interactions. These mechanisms include several direct effects of trait variation *per se* and indirect effects arising from the role of genetic variation in trait evolution.

Trait variation within natural populations

One of Charles Darwin's greatest insights was recognizing that conspecific individuals differ in many traits including obvious features such as gender, age, or size, and also more subtle traits such as shape, behavior, or physiology. This variation provides the raw material for natural selection and thus is a key focus of evolutionary theory. By contrast, ecological theory typically focuses on predicting the dynamics of species' abundances over time without regard to particular phenotypes. Consequently, many models of species' interactions implicitly assume that all conspecific individuals are effectively interchangeable. In this paper we argue that this assumption is misleading and that intraspecific trait variation can substantially alter ecological dynamics.

Ecologists have long recognized that sexes [1] and age classes [2] differ in ecologically significant ways. However, phenotypic variation among individuals can generate variation in ecological attributes even within a sex and age class [3]. Within populations individuals can differ in

anti-predator defenses [4], parasite resistance [5,6] or exposure [7], abiotic tolerances [8], resource use [3], or competitive ability [4,9]. Such differences can, in turn, generate variance in demographic parameters [10,11]. For example, Southern sea otter (*Enhydra lutris nereis*) individuals differ substantially in diet, controlling for age, sex, and morphology [12]. Some individuals specialize in eating snails, others in eating crabs and abalone, whereas others are generalists. Owing to this diet variation, individual otters differ in energetic income and pathogen exposure [7].

The proximate mechanisms underlying such diet variation can be complex. In sea otters, foraging is a learned

Glossary

Eco-evolutionary dynamics: effects of ecological changes on evolutionary dynamics or the effects of evolutionary changes on ecological dynamics, feedbacks arise when a loop links both directions of effect.
Genetic drift: change in allele frequencies owing to random sampling during reproduction, especially strong in small isolated populations.
Jensen's Inequality: mathematical rule stating that the mean of a concave (convex) function of a variable trait is smaller (larger) than the value obtained by applying the function to the mean of the variable.
Narrow-sense heritability: the proportion of total phenotypic variation attributed to additive genetic variation, proportional to the rate of short-term responses to selection.
Niche complementarity: the tendency for phenotypically divergent individuals (or species) to compete less strongly.
Node degree: number of predator and prey links for a (trophic) species represented within a food web. Degree distribution describes the mean and variance of node degree across all nodes in a network.
Phenotype: the outward expression of an individual's genotype as affected by the environment, can include size, morphology, behavior, and physiology.
Phenotypic subsidy: a process by which the density of one phenotype is augmented by reproduction, behavioral switching, phenotypic plasticity, or migration by another phenotype.
Portfolio effect: negative covariances between the dynamics of subunits leads to a smaller total variance through averaging, applied here to phenotypic frequencies within a population.
Quantitative genetics: evolutionary approach to predicting the evolution of continuous traits assuming the action of many genes of small phenotypic effect.
Trait: any measurable feature of an individual organism, including phenotype as well as demographic parameters such as clutch size, growth, or longevity.

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behavior [12] that can respond flexibly to resource density [13]. In other organisms, diet variation reflects differences in morphology [14,15], digestive physiology [16], individual social status [17], or search images from past experience [18]. Each of these causes of variation can have genetic and environmental sources because most traits exhibit intermediate heritability [19,20].

Ecological consequences of trait variation

Trait variation among conspecific individuals has long been recognized, forming the basis of classic work in ecological genetics [21] and niche evolution [22]. However, ecologists' interest in such variation waned after the mid-1970s [23], and intraspecific trait variation has since been largely ignored by both empirical and theoretical ecologists. Recently, ecologists have gained renewed appreciation for the extent and community consequences of intraspecific variation [3,24]. Several experiments have manipulated intraspecific genetic diversity and found corresponding changes in population productivity and stability [25], coexistence [9,26,27], and ecosystem processes [28]. Intraspecific variation can thus have large ecological effects [24–30]. These results motivate the central question of this review: when and why do such effects emerge?

Despite a fast-growing literature on the ecology of trait variation, we lack a general framework for understanding the mechanisms by which trait variation influences ecological dynamics. Developing such a framework is key to determining when, and to what extent, intraspecific trait variation will alter population densities, transient dynamics, and persistence. These general questions can best be answered through the use of theory because there are currently too few studies for a meta-analysis. In this review we synthesize the relevant theoretical literature to build a framework for understanding the ecological effects of individual variation. Specifically, we consider the following

thought experiment: take an ecological model, and contrast the community dynamics with and without intraspecific trait variation. When and why does variation change the dynamics?

A modest number of theoretical studies have investigated how intraspecific trait variation affects population dynamics [31–34], interspecific competition [35–37], and predator–prey or host–parasitoid systems [38–40]. These studies consider variation in diverse traits, including: (i) traditional phenotypes such as size or morphology; (ii) emergent traits such as competitive ability [41], prey attack rate [39], or vulnerability to enemies [42]; and (iii) fitness-related traits such as fecundity or survival [33]. Variation in these traits might be stochastic [32], environmentally induced [43], or genetic (Mendelian or quantitative; sexual or asexual [31,44]). These studies have found very different (and sometimes conflicting) results. For instance, alternate models find that trait variation increases or decreases extinction risk, depending on what trait and heritability is assumed [32,34]. Similarly, different models find that variation in competitive ability can reduce or increase equilibrium densities [45,46].

A few generalities emerge despite the diverse assumptions and predictions of these models. All relevant models invoke at least one of six general mechanisms by which trait variation alters ecological dynamics (Table 1). By clearly delineating these mechanisms we can better understand when it is necessary to consider trait variation. Below we describe each mechanism and highlight representative theoretical models. The mechanisms fall into two general categories: (i) direct ecological effects of trait variation *per se* regardless of its heritability (mechanisms 1–3) and (ii) indirect effects arising because genetic variation permits eco-evolutionary dynamics (mechanisms 4–6).

Table 1. Summary of the six mechanisms by which intraspecific trait variation could alter community structure or dynamics

| Mechanism	Name	Summary:		Genetic process	Examples:[e]
		Occurs when:	Variation alters:		
1	Jensen's Inequality	Ecological interactions depend nonlinearly on a variable trait.	Mean interaction strength.	NA	[31,38,40,41]
2	Increased degree	Trait variation changes the strength or number of species interactions.	Niche breadth.	NA	[36,42,46,94]
3	Portfolio effect	Phenotypes exhibit negative covariation in density over time.	Fluctuation in total population density.	NA[a]	[31,44,65]
4	Phenotypic subsidy	Genetic or developmental exchange between phenotypes decouples each phenotype's growth rates from its fitness.	Feasibility of ecological equilibria.	Recombination, assortment, or mutation[b]	[31,44,65,67]
5	Adaptive eco-evolutionary dynamics	Heritable trait variation permits rapid evolution.	Mean interaction strength.	Selection[c]	[39,69,75,77,95]
6	Trait sampling	Small population size permits stochastic sampling from trait distributions.	Mean fitness or interaction strength.	Drift[d]	[32,33,37,78,86,96]

Notes:

[a]Portfolio effect can be facilitated by a heritable basis distinguishing phenotypes.

[b]Phenotypic subsidy can also arise via phenotypic plasticity (developmental or behavioral).

[c]Adaptive phenotypic plasticity can drive similar outcomes.

[d]Sampling effects can arise via demographic stochasticity or genetic drift.

[e]This list is intended to be illustrative, not exhaustive, due to space constraints. We focus on relevant theory, because empirical studies of the ecological effects of intraspecific variation rarely distinguish among the mechanisms listed here.
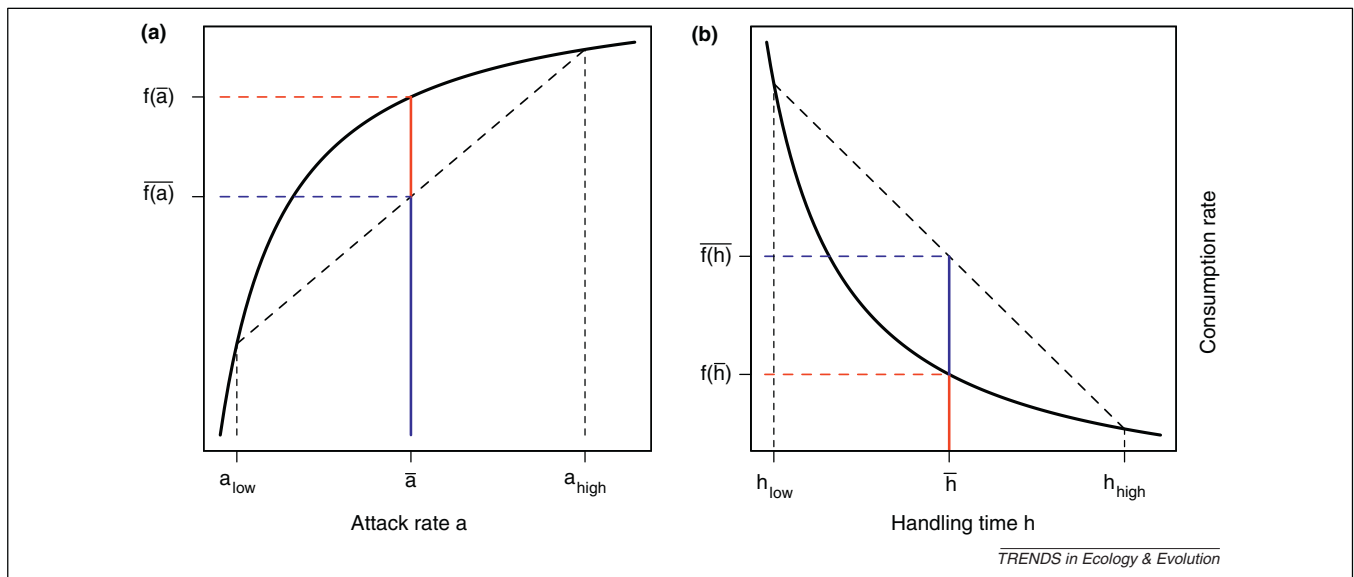
Figure 1. The effects of individual predator variation in attack rates and handling times on rates of prey consumption. In both figures, the consumption rate f – a function of an individuals' attack rate (a) and handling time (h) – is plotted as a solid black line (panels a and b respectively). For a dimorphic population consisting of individuals with low and high trait attack rates (or handling times), the average feeding rate $\overline{f(a)}$ is highlighted in blue and the feeding rate of the average individual $f(\bar{a})$ is highlighted in red. For any composition of individuals with low and high trait values, the average feeding rate lies on the diagonal dashed line. The feeding rate is a concave function of the attack rate, so the average feeding rate is lower than the feeding rate of the average individual. Conversely, feeding rate is a convex function of handling time and so variance in handling means the average feeding rate is higher than the feeding rate of the average individual.

Why does intraspecific variation affect ecological dynamics?

Mechanism 1: Jensen's Inequality

When an ecological interaction depends nonlinearly on a species' trait, variation around the trait mean can alter the average interaction strength. Jensen's Inequality [47] states that when x is a variable trait with mean $\bar{x}$, and $f(x)$ is a concave (convex) function of x, the average value of the function $\overline{f(x)}$ will be smaller (larger) than the function evaluated at the trait mean $f(\bar{x})$ (Figure 1). Therefore, if $f(x)$ is nonlinear and x is variable, then $\overline{f(x)}$ will typically differ from $f(\bar{x})$. Hence, populations with identical trait means but different variances will have different mean interaction strengths, thereby altering demographic rates and community dynamics.

Consider the Type II functional response often used in predator–prey models to relate an individual predator's feeding rate to its prey density. The Type II functional response saturates with increasing prey density R,

$$f(R, a, h) = \frac{aR}{1 + ahR} \qquad [1]$$

where a is a predator's attack rate and h is its handling time. If predators vary in attack or handling rates, then the population's functional response is not simply $f(R, \bar{a}, \bar{h})$, as is typically assumed in ecology [48], but instead is the average of individuals' functional responses $\overline{f(R, a, h)}$. Because Equation 1 is concave with respect to a, variation in attack rates will lower overall predation pressure (Figure 1a) potentially preventing predator–prey oscillations [38]. The same equation is convex with respect to h, so handling time variation increases predation pressure and destabilizes predator–prey interactions (Figure 1b). This example illustrates how variation in

two traits can yield contrasting ecological effects within a single model framework.

Nonlinear (convex or concave) functions such as the Type II functional response are widespread in ecological models [47,49]. Hence, Jensen's Inequality might be of nearly ubiquitous importance in ecology (Box 1). Importantly, ecological effects of Jensen's Inequality emerge irrespective of the genetic or environmental basis of trait variance, representing a direct consequence of trait variance rather than an effect of evolution (except where evolution alters the ecological function's curvature).

Mechanism 2: Increased degree

Intraspecific trait variation can alter the number and strength of a species' interspecific interactions thereby changing the topology and dynamics of species interaction networks. Roughgarden [22] pointed out that a species' diet diversity, D, could be decomposed into: (i) the average within-individual diet diversity and (ii) between-individual variation ($D_{total} = D_{within} + D_{between}$). Consequently, increased between-individual variation ($D_{between}$) inflates the population's total diet diversity (D_{total}), making the population more generalized even if individuals remain specialized (i.e. D_{within} is small and constant) [50].

Consider a food web in which each predator attacks all potential prey within a specified relative size range [51,52]. Intraspecific variation in consumer body size increases the range of acceptable prey sizes and thus the number of species the consumer population attacks. Equivalently, heterogeneous resources are vulnerable to more diverse predators. We thus expect more variable species to interact with more species; this greater connectivity is known as 'increased degree' in network theory (degree is the number of links from one node to other nodes in a network). Conversely, each pairwise link might be weaker because

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Box 1. Examples of nonlinear functions of variable traits

- *When fitness depends nonlinearly on any trait.* When a population is subject to stabilizing selection on a quantitative trait, any variance around the optimal trait value imposes a fitness load because most individuals deviate from the average (e.g. optimum) and hence have suboptimal fitness. Thus, the average fitness of the population $\overline{w(x)}$ is less than the fitness of the average phenotype $w(\bar{x})$. The opposite is true for populations under disruptive selection, $w(\bar{x}) < \overline{w(x)}$. Phenotypic variance can thus increase or decrease population growth rates depending on the mode and strength of selection acting on any given trait.

- *When population growth depends nonlinearly on fluctuating demographic rates.* Many models of population growth are nonlinear functions of individual fitness [86]. Consequently, among-individual variation in fecundity or survival alters mean population growth and abundance. Genetic variation in demographic rates is typically assumed to be small because selection eliminates low-fitness genotypes. However, additional fitness variation arises from stochastic events such as random dispersal and settlement in a heterogeneous environment, random variation in gene expression, as well as stochastic sampling of realized trait values (e.g. clutch size, longevity) from a shared probability distribution.

- *When food web links depend on body size.* Recent food web theory makes wide use of size-based niche models that describe the probability that a species with mean body size $\bar{x}_i$ consumes prey with mean size $\bar{x}_j$ [54,87]. However, if attack rate is a nonlinear function of two species' body sizes $f(x_i, x_j)$, the appropriate attack rate is not $f(\bar{x}_i, \bar{x}_j)$ but rather the weighted average attack rate across all predator/prey phenotype combinations. Consequently size variation alters food web structure (including the addition of size-dependent cannibalism [88]).

- *When any parameter is subject to metabolic scaling laws.* Size-structured food web models often use metabolic scaling laws to obtain species-specific demographic parameters such as birth or death rates and total consumption rates [54]. These rates are assumed to scale with body mass (M) according to a power law $y \sim aM^b$. Such power laws are considered a fundamental feature of biological systems [89]. However, their nonlinearity makes the use of mean body size misleading because a population's mean metabolic rate is not predicted by its mean body size ($\bar{y} \neq a\bar{M}^b$).

Other examples include nonlinear relationships between: light availability and photosynthesis, nutrient concentration and chemostat population growth, phenotypic similarity and intraspecific competition (Box 3).

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only a subset of conspecific individuals participates in a given interaction.

Changes in population diet diversity due to trait variation can have profound effects on density-dependence, interspecific competition, and food web structure. Greater population niche width alleviates intraspecific competi-

tion, particularly among divergent phenotypes (a phenomenon known as niche complementarity; Box 2). Increased niche width also affects the outcome of interspecific competition [36] by simultaneously increasing diet overlap with other species, and reducing the impact of this overlap because only a subset of individuals in each species are

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Box 2. Niche complementarity

Niche complementarity is the tendency for phenotypically divergent individuals (or species [90]) to compete less strongly. Models of intraspecific competition typically assume that population growth declines as a function of the total population density, N:

$$N_{t+1} = f(N_t)N_t. \qquad [\text{I}]$$

The function $f(N)$ represents density-dependent population growth, such as:

$$f(N) = \frac{\lambda}{1 + aN^b} \qquad [\text{II}]$$

where λ is the population's intrinsic growth rate, a measures the impact of density on individual fitness, and b reflects the type of competition (e.g. scramble versus contest [41]). The important point is that as total population size N increases, the denominator overwhelms the numerator and population growth approaches zero.

This model presumes that individuals compete equally with all members of the population. However, in variable populations competition can be strongest between phenotypically similar individuals [15,31], and weak or absent between phenotypically divergent individuals. For instance, imagine two predator phenotypes that each specializes on a different prey species. The predator phenotypes that eat species A will compete with each other and will not compete with the predator phenotype that exclusively eats species B. Thus, the fitness of an individual depends not on the total population size, but only on the abundance of competitively relevant phenotypes. This frequency-dependence can be modeled by replacing total population density N in Equation II with an 'effective population density' experienced by each phenotype z:

$$N_z = \int \rho(z')\alpha(z, z') \, dz' \qquad [\text{III}]$$

where $\rho(z')$ denotes the density of an alternative phenotype z'. This equation considers the density of every other phenotype ($\rho(z')$) weighted by its competitive effect on the focal phenotype z. This competitive effect, $\alpha(z, z')$, is the per capita decrease in population growth of phenotype z per individual of phenotype z', which is a declining function of the phenotypic difference between individuals:

$$\alpha\left(z, z'\right) = \exp\left(-\frac{\left(z - z'\right)^2}{2\sigma_\alpha^2}\right) \qquad [\text{IV}]$$

where σ_α^2 determines how quickly competition declines with phenotypic divergence. All else being equal, a population with greater phenotypic variance will experience weaker competition (diet specialization [low σ_α^2] has a similar effect). Consequently, a variable population reaches a higher carrying capacity and might be more stable than a homogenous one [31].

Niche complementarity is tightly tied to the changes in population niche width (degree) described in Mechanism 2. The model outlined above implicitly assumes that a more phenotypically variable population uses a wider range of resources thereby reducing niche overlap among individuals. We therefore do not consider niche complementarity to be a distinct mechanism in its own right.

Some models of niche complementarity also have elements that invoke other mechanisms. Niche complementarity has aspects that can be described as a portfolio effect. Competition generates positive covariances among phenotype densities over time because all phenotypes grow slower as density increases. By mitigating competition among phenotypes, niche complementarity reduces this density-dependence covariance in abundance, thus reducing fluctuations in overall population density (see Equation 2 in main text). Finally, niche complementarity sets the stage for frequency-dependent competition that can drive eco-evolutionary feedbacks, particularly in the form of disruptive selection that can maintain or inflate trait variance through time.

affected. Existing food web models provide some insight into how network structure might affect multitrophic dynamics. Increased average node degree of a network (more generalist species) enhances its structural robustness to secondary extinctions [53] and can stabilize network dynamics [54,55]. Even a few species with increased degree can alter network dynamics because generalists often serve as keystone species or link subnetworks [56]. Decreased interaction strength also promotes network persistence and stability by dampening consumer-resource oscillations [57,58]. Therefore among-individual trait variation should promote persistence and reduce oscillations by changing network connectance and interaction strengths. As with Jensen's inequality, this stabilizing effect is independent of trait heritability.

Mechanism 3: Portfolio effect
Intraspecific trait variation can protect populations from extreme temporal fluctuations in population density. A population's density (N) is the sum of the densities of its constituent phenotypes (N_i). Fluctuations in population density (across time or space) depend on fluctuations in phenotype densities [59]:

$$\sigma_N^2 = \sum_i p_i^2 \sigma_{N_i}^2 + \sum_i \sum_j p_i p_j \sigma_{N_i,N_j} \qquad [2]$$

where p_i is the frequency of phenotype i, $\sigma_{N_i}^2$ is the temporal or spatial variance in its density, and σ_{N_i,N_j} is the covariance between phenotype densities. When phenotype densities exhibit negative covariances the second term of Equation 2 is negative and the whole population exhibits less density variation than individual phenotypes. This portfolio effect has been implicated in the long-term sustainability of the Alaskan Sockeye salmon fishery which depends on hundreds of discrete salmon runs [60]. Such negative covariances can have many sources, including intraspecific competition among individuals with diverse diets. When individuals compete more strongly with phenotypically similar conspecifics (Box 2), rare phenotypes are more fit than common ones. Doebeli [31] showed that this frequency dependence generates negative covariance between densities of asexual phenotypes, stabilizing total population size even when each phenotype exhibited oscillatory dynamics with its respective prey. Note that negative covariance among genotype abundances implies fluctuating genotype frequencies (evolution). However, the ecological effect can arise primarily via stabilized total population size rather than via trait evolution (see Mechanism 5).

Mechanism 4: Phenotypic subsidy
Various population genetic processes allow reproduction by each phenotype to increase the abundance of ('subsidize') other phenotypes within the same population thereby altering interactions with other species. Offspring are often genetically different from their parents owing to mutation, Mendelian segregation of alleles, independent assortment, or recombination among loci. Even clonal offspring can differ from their parents due to phenotypic plasticity [61–63]. This parent–offspring difference leads to a 'phenotypic subsidy', which we define as the increased

abundance of one phenotype owing to reproduction (or plasticity) by a different phenotype, that therefore produces fewer offspring of its own type. This subsidy is best illustrated by contrasting the dynamics of phenotypes produced via asexual versus sexual reproduction. In a strictly asexual system, the change in density of phenotype i depends only on its own abundance N_i, fecundity b_i and death rate d_i:

$$\frac{dN_i}{dt} = b_i N_i - d_i N_i. \qquad [3]$$

However, in a sexual organism the change in each phenotype's density is partially decoupled from its own fitness because of subsidies (see Box 3 for a detailed example). Consequently, the growth rate of each phenotype i depends on the fecundity, density, and the subsidy rates of all j phenotypes in the population to phenotype i:

$$\frac{dN_i}{dt} = \sum_j S_{ji} b_j N_j - d_i N_i. \qquad [4]$$

The subsidy S_{ji} is the fraction of phenotype j's offspring contributed to phenotype i. This fraction depends on the mating system and genetic mechanism of the subsidy.

By coupling population dynamics of different phenotypes a phenotypic subsidy can alter the equilibria and population dynamics of a species, as well as that of its resources and predators (Box 3). Phenotypic subsidies can sustain a given phenotype above the ecological equilibrium density it would experience in isolation, potentially preventing extinction of ecologically unfit forms. Conversely, a phenotype that would otherwise persist might go extinct by subsidizing unfit ('sink') phenotypes. Aside from influencing phenotypic variance, these subsidies can prevent populations from reaching ecologically determined equilibrium densities or trait distributions [22]. The resulting mismatch between ecologically determined equilibria and genetically permissible states can have cascading effects across other species (Box 3). For instance, if subsidies prevent extinction of an unfit phenotype, that phenotype's resources (and predators) would reach a lower (higher) abundance than would be the case without the subsidy. The coupling of genetically differentiated forms via sexual reproduction or mutation introduces a negative feedback akin to self-limitation, stabilizing total population size by reducing the incidence and amplitude of fluctuations [64]. All else being equal, these population and community effects will probably be stronger with greater phenotypic diversity and stronger reproductive coupling (e.g. in sexual populations).

Few models specifically consider how reproductive subsidies affect community dynamics [31,65]. However, we can extract some useful principles from spatial ecology models. The tendency for segregation and recombination to recreate otherwise moribund phenotypes resembles the 'rescue effect' in which immigration reduces the extinction risk of inherently unstable populations [66]. Models of patch dynamics can thus be reinterpreted to determine effects of subsidies: consumers in different patches can be redefined as different phenotypes, and dispersal between patches redefined as mutation. This reinterpretation suggests that

phenotypic subsidies are not always stabilizing: dispersal (mutation) between predators in distinct patches (genotypes) often generates oscillatory or chaotic dynamics [67].

Mechanism 5: Adaptive eco-evolutionary dynamics
Heritable intraspecific variation enables adaptive evolution in response to ecological interactions. There is growing realization that evolution can occur on timescales commensurate with population dynamics [68]. Selection mediated by ecological interactions can change trait means that, in turn alters ecological interactions [69,70]. Feedbacks between ecological processes and trait evolution ('eco-evolutionary dynamics') are of intense current interest [71,72], although they have been recognized for decades (e.g. ecological character displacement [73]). Because most traits exhibit at least some heritable variation [19,20], the most biologically relevant question is not whether eco-evolutionary feedbacks occur but: (i) how strongly does trait evolution affect ecology and (ii) how does the magnitude of additive genetic variation alter eco-evolutionary effects?

Box 3. A simple example of phenotypic subsidy

In a panmictic population with three Mendelian genotypes (*AA, Aa, aa*), reproduction by heterozygotes can produce homozygote offspring, and vice versa. This subsidy means that changes in each phenotype's abundance depends on the fitness (and relative abundance) of every phenotype. To illustrate the potential ecological impact of these phenotypic subsidies, we can contrast asexual versus sexual reproduction in a polymorphic consumer population. Let P_1, P_2, and P_3 denote the density of three consumer phenotypes that, respectively, consume resource species R_1, R_2, and R_3. Assuming Lotka-Volterra predator–prey dynamics, the abundance of each clonal consumer phenotype (P_i) and its resource (R_i) can be modeled as:

$$\frac{dP_i}{dt} = a_i b_i P_i R_i - d_i P_i, \quad\quad\quad\quad\quad [Ia]$$

$$\frac{dR_i}{dt} = r_i R_i \left(1 - \frac{R_i}{K_i}\right) - a_i b_i P_i R_i \quad\quad [Ib]$$

where a_i is the attack rate of predator i on resource i, b_i is the fecundity of the predator for each prey eaten, d_i is the predator per-capita death rate, r_i is the intrinsic growth rate of resource i and K_i is the carrying capacity of the resource. Each consumer-resource pair converges on its own independent equilibrium. The dark bars in Figure I represent the equilibrium densities of resource and consumer species with asexual reproduction in a numerical example in which P_2 is a much less efficient forager than P_1 or P_3 (P_2 goes extinct).

Sexual reproduction can be incorporated using some simple Mendelian rules, by assuming an equal sex ratio and random mating. For example, consider the birth rate of *aa* individuals. There are $\frac{1}{2}P_1$ female parents with genotype *aa* but only a fraction $S_{aa \to aa}$ of their offspring will be *aa*:

$$S_{aa \to aa} = p_{aa} + \frac{p_{Aa}}{2} \quad\quad\quad\quad\quad\quad [II]$$

where p_{aa} and p_{Aa} are genotype frequencies. Equation II takes into account the probability an *aa* female mates with each possible male genotype. It also accounts for the fraction of *aa* offspring that result from each possible pair of parents (1 from *aa mating with aa*, 0.5 from *aa mating with Aa* and 0 from *aa mating with AA*). Similarly, $S_{Aa \to aa} = \frac{p_{aa}}{2} + \frac{p_{Aa}}{4}$ and $S_{AA \to aa} = 0$. The total birth rate of genotype *aa* individuals (B_{aa}) can then be calculated, summing across all possible female genotypes, accounting for their densities, fecundities ($f_i = 2a_i b_i R_i$), and the subsidy fractions $S_{j \to i}$:

$$B_{aa} = \sum_j \frac{1}{2} P_j \, f_j S_{j \to i} \qu\quad\quad\quad\quad [III]$$

This birth rate is substituted in for the birth term $a_i b_i P_i R_i$ in Equation Ia.

Incorporating sexual reproduction dramatically alters the ratio of phenotypes and total population sizes as illustrated in Figure I (light bars). Because the homozygotes are equally fit and abundant in this numerical example, Mendelian segregation dictates a 1:2:1 ratio of predator phenotypes: random mating between homozygotes subsidizes the abundance of unfit heterozygotes. This genetic constraint on phenotype ratios is a substantial deviation from the P_1:0:P_3 ratio seen in the asexual model (dark bars, Figure Ib). The higher abundance of unfit heterozygotes places a substantial fitness load on the consumer

population whose total density is less than half that observed for asexual consumers. Correspondingly, resources abundances are on average both higher and more even.

Many other population genetic processes can generate ecologically relevant phenotypic subsidies. First, quantitative phenotypic traits are typically the result of additive effects of many loci of small effect. Segregation and recombination of these genes determine the trait distribution (often normally distributed) which might not match the ecologically-determined optimal trait distribution [31]. Second, sexual reproduction is a form of subsidy because the production of new males is determined by female fecundity. When males and females consume different prey [1], the males' prey might contribute little to the species' abundance because male abundance is determined by the availability of the females' prey.
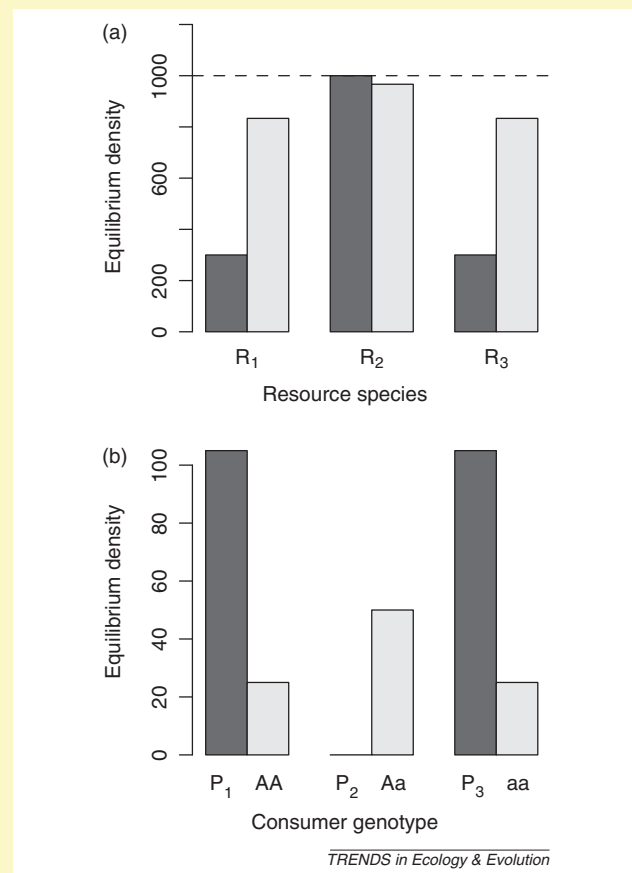
Figure I. Equilibrium abundance of three consumer genotypes (b: asexual, dark bars; sexual, light bars) and their respective three prey resources (a). In (a) a horizontal line indicates the carrying capacity of all three resources (K = 1000). All parameters are identical across consumer-resource pairs (r = 1.5, a = 0.01, b = 0.05, d = 0.15) except that P_2 (sexual genotype Aa) has attack rate a = 0.001.

Heritability determines evolutionary rates, and thus the relative speed of evolutionary and ecological dynamics. For instance, in a model of interspecific competition for multiple essential resources, high heritability allows rapid evolutionary character convergence thereby facilitating coexistence. However, low heritability slows evolution sufficiently that ecological dynamics dominate and exclusion ensues [74]. Trait variation also allows adaptation to changing environments. Greater additive genetic variance decreases extinction risk because more variable populations adapt faster and thus more closely track changing trait optima [75]. This faster adaptation to environmental change also alters the outcome of interspecfic competition [76]. A relatively underexplored question is whether similar feedbacks between ecology and trait distributions can arise from adaptive phenotypic plasticity instead of evolution.

Eco-evolutionary dynamics have been convincingly demonstrated in numerous controlled experiments [69,72] and in theoretical models [77]. Fewer studies have specifically evaluated how genetic variance affects these dynamics [30,39], and most fail to distinguish between eco-evolutionary versus non-evolutionary effects of variation (Mechanisms 1–3). Doing so requires factorial manipulation of both phenotypic variance and heritability, which is difficult. Theory can thus play a crucial role in partitioning effects arising from different components of phenotypic variation. For example, Schreiber et al. (unpublished) studied an apparent competition model (one predator, two prey) with genetic or environmental trait variation among predators that influenced attack rate on the two prey. Total phenotypic variance determined equilibrium population densities, whereas heritability affected the rate of transient dynamics and stability around equilibria. Hence, both genetic and environmental trait variance have appreciable and non-equivalent effects.

Mechanism 6: Trait sampling

In small populations, ecological and evolutionary sampling of individual variation can change trait means and variances thereby altering population dynamics. Demographic variance [32,33,78,79] is the variance in a population's vital rates arising from two sources: (i) stochastic sampling of individuals with different demographic rates and (ii) conventional demographic stochasticity arising from the discrete probabilistic nature of births and deaths. For instance, mean clutch size can fluctuate through time because the individuals who (randomly) succeed in reproducing in different years happen to have different expected fecundity, or because clutch size varies randomly among individuals with equal expected fecundity. Demographic variance (and trait sampling in general) will be most pronounced in small populations, and can affect long-term population dynamics [32,33,78], population persistence [79], and species coexistence [37].

An analogous process could drive maladaptive eco-evolutionary feedbacks. Genetic drift is the random change in allele frequencies resulting from stochastic sampling of alleles from the preceding generation (independent of demographic stochasticity). By altering trait means and variances, genetic drift might alter demographic rates or interspecific interactions. In small populations, drift can cause substantial departures from phenotypic optima thereby reducing mean fitness. Consequently, population density declines still further and drift becomes even more pronounced. This feedback might generate an 'extinction vortex' [80,81] with cascading effects on an entire community.

Combining mechanisms

The six mechanisms outlined above imply that trait variation alters ecological dynamics when: (i) ecological parameters are nonlinear functions of the trait(s), (ii) trait variance affects niche width or network topology, (iii) phenotypes exhibit asynchronous fluctuations in density over time, (iv) phenotypic subsidies decouple genetic and ecological equilibria, (v) ecology both drives and responds to trait evolution and (vi) trait variation is stochastically sampled in small populations. Additional mechanisms might still be uncovered.

For trait variation to be ecologically *unimportant*, all of these mechanisms must be jointly ruled out or have weak effects. Because each highlighted mechanism is general, intraspecific trait variation can have nearly ubiquitous ecological effects. These effects can cascade throughout a community of interacting species and substantially alter equilibrium densities [46] and change conditions for population stability or multispecies coexistence [31,37].

The six mechanisms are not mutually exclusive. Heritable trait variation in both a predator and its prey can influence population dynamics simultaneously via Jensen's Inequality, a portfolio effect, phenotypic subsidy, and eco-evolutionary feedbacks [31]. However, little is known about the relative frequency or strengths of these different pathways. To complicate matters, the mechanisms listed above might interact. Fluctuations in one species' population size due to demographic stochasticity can alter another species' growth via Jensen's Inequality. Interactions can also arise from eco-evolutionary change in trait variances that alters the potential for other variation-based effects. Finally, intra- and inter-specific diversity could have parallel and perhaps interacting effects on population dynamics (Box 4).

Future directions

Given the general, multifaceted, and potentially large ecological effects of trait variation, we need to revisit classic ecological models and ask how the magnitude and heritability of trait variation alters previous conclusions. In doing so, we should evaluate: (i) the magnitude of ecological effects of trait variance; (ii) the relative contributions of the various mechanisms described above; and the consequences of (iii) heritable, environmental, or stochastic variation; (iv) the genetic architecture (one-locus, quantitative genetic, or explicit multi-locus models); (v) asexual versus sexual reproduction; and (vi) variation in multiple traits. Addressing these questions will require new approaches for fusing ecological models with population and quantitative genetics. For discrete phenotypic variation one could separately model the density of each phenotype within a species (coupled by reproduction), or track overall density and allele frequencies in each species. For continuous traits, one could describe each species with three equations that track

Box 4. Intra- versus inter-specific diversity

The influence of intraspecific variation on population dynamics has a well studied analogue: multiple species within a community are sometimes aggregated into guilds to define a particular ecosystem service or function [91]. The mean total function (e.g. total biomass) or stability (temporal variability in function) of the system can change as species are added to the community: typically both measures improve but saturate as richness increases [91]. This community effect of species richness is commonly attributed to increased functional diversity as species are added, increasing resource availability or resistance to consumers or environmental conditions [91]. Similar effects can arise from functional diversity among individuals within populations, increasing equilibrium density in response to increasing variation [45,46,92].

Furthermore, when environmental conditions fluctuate over time, communities (or populations) possessing a diverse set of functional traits have a higher probability of including species (genotypes) that persist through particular environmental conditions, thereby stabilizing function [91]. For instance, negative covariance among edible and less-edible phytoplankton morphotypes dampens temporal fluctuations in total biomass during summer when competition is most intense [93]. Similar results have been found for competing phenotypes, where variation in a quantitative trait generated strong frequency-dependent competition and negative covariance among phenotypes due to niche complementarity [31]. This is an example of the portfolio effect that can arise both for species diversity and intraspecific variation.

Our limited understanding of how genetic variation is maintained under stabilizing selection in real populations is fundamentally similar to the classic question in community ecology: Why do many species persist under what seems to be strong selection for a single or few species? Potential answers to these two questions have many similarities including temporal and/or spatial variation in selection, mutation-drift balance (colonization–extinction balance in community ecology), and negative frequency-dependence. Conversely, the two can differ in important ways. For example, phenotypic subsidies due to interbreeding are possible within species but not between them.

changes in population density, trait mean, and trait (co)-variance. However, modeling the evolution of trait (co)variances is a continuing challenge in quantitative genetics. Computationally intensive individual-based models with explicit multilocus genetics offer one solution.

Intraspecific variation has been particularly lacking in models of species-rich communities. Some food web models incorporate changing topologies via trait evolution or behavioral prey switching [61,82] but even these typically assume homogenous conspecifics at each time step. Incorporating trait variation into food webs might change their topology (see Mechanism 2) and dynamics [83]. Such effects presumably depend on where in a food web one adds trait variance. Unfortunately, empirical studies of diet variation typically focus on single species, giving little guidance as to how variance is distributed across multiple interacting species [84].

One limitation for theoreticians is the scarcity of empirical data regarding the causes, patterns, and consequences of within-population ecological variance [3]. The majority of empirical studies of ecological variation among individuals simply test the null hypothesis that individuals are identical but do not quantify the magnitude of variation or determine its mechanistic or genetic basis [3]. To predict the consequences of trait variation, ecologists need to: (i) identify the genetic and phenotypic causes of ecological variation and (ii) quantify ecological variation across multiple species, trophic

levels, or entire communities. Ideally, such information could guide theoreticians in developing biologically realistic models that can be parameterized with empirical data to yield testable quantitative predictions.

Most importantly, we need more experiments that test the ecological effects of trait variation within species. The typical approach is to manipulate trait (or genetic) variance within populations, and measure the resulting difference in population or community dynamics. A major challenge for such studies is to clearly distinguish between the direct and indirect (evolutionary) effects of trait variance outlined in this paper. This will require careful experimental designs that either manipulate phenotypic variance among genetically identical individuals (thereby preventing evolution), or that compare experimental replicates in which evolution is allowed versus inhibited (e.g. [85]). Clearly, many of the mechanisms outlined above will be best tested by studies that fuse quantitative models with empirical data. For instance, the ecological effects of Jensen's Inequality are best studied by empirically estimating the nonlinear relationships between variable traits and their functional consequences. Such studies will be a challenging and possibly unwelcome complication for ecologists. However, theory indicates that ecologists cannot safely ignore intraspecific variation, owing to its large effects on community dynamics.

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