Decomposing social environment effects on eco-evolutionary dynamics: from density regulation to frequency-dependent selection

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Abstract

The density and frequencies of interacting phenotypes create a type of environment which affects both phenotypic selection and population growth. Fluctuations in population density create temporal variation in population mean fitness, driving population dynamics, while fluctuations in phenotypic frequencies create variation in the relative fitness of phenotypes through frequency-dependent selection. Different modelling frameworks have been used to study these (social) environment effects and the eco-evolutionary dynamics produced by their interaction. However, the diversity and mathematical complexity of these models can represent an obstacle for empiricists aiming to study the social factors shaping the eco-evolutionary dynamics of natural populations. Here, we reformulate components of these models using generalized linear regression equations to provide a statistical decomposition of how different frequency- and density-dependent processes influence phenotypic selection, population growth, and the expected equilibrium density and mean phenotype of a population. We complement these results with individual-based simulations to illustrate how quantifying the different ways the social environment affects an individual's fitness can improve our understanding of the feedback that links the evolutionary dynamics of phenotypes with the carrying capacity of natural populations.

Keywords: density-dependent selection, multiple regression, individual-based simulations, social evolution

Introduction

The interaction between ecological and evolutionary processes plays a fundamental role in shaping phenotypic diversity and the functioning of ecosystems (reviewed by Govaert *et al.*, 2019; Hendry *et al.*, 2018; Pelletier *et al.*, 2009). At the heart of such eco-evolutionary dynamics lies the feedback between population dynamics and phenotypic evolution. The social environment is a key mediator of this feedback because it evolves in response to selective pressures changing the demographic and phenotypic characteristics of populations. This will result in eco-evolutionary feedback because evolution alters the ecological context for selection, further affecting phenotypic evolution. Different modelling traditions have been used to study the consequences of these environmental feedbacks on the demographic characteristics of populations and the evolutionary dynamics of phenotypes (Abrams *et al.*, 1993; Boyce, 1984; Charlesworth, 1994; Engen *et al.*, 2020; Heino *et al.*, 1998; Lion, 2018; MacArthur, 1962; Mylius & Diekmann, 1995). These studies highlight that understanding the eco-evolutionary dynamics of populations requires quantifying the interactive effects of population density and phenotypic frequencies on population mean fitness and the relative fitness of phenotypes. These effects are rarely studied together in wild populations, leading to a potential mismatch between our theoretical understanding and the processes operating in nature. To address this gap, we use generalized linear regression models and individual-based simulations to illustrate the different ways the social environment can affect eco-evolutionary dynamics and encourage empiricists to quantify these social environment effects in natural populations.

We refer to the social environment as the density and frequency of conspecific phenotypes affecting an individual's survival and reproduction. Competitive and cooperative interactions shape the strength of density regulation and phenotypic selection (Frank, 1998; Haldane, 1956; Lack, 1954; West-Eberhard, 1979), making the social environment a key mediator of the eco-evolutionary dynamics of populations. Phenotypes mediating social interactions can influence population dynamics and/or phenotypic evolution whenever an individual's fitness is affected by its social environment (Travis *et al.*, 2013; Wolf *et al.*, 1999). We can think of traditional studies of density regulation and frequency-dependent selection as focusing on two ways the social environment affects the ecological

and evolutionary dynamics of populations. On the one hand, classic population ecology focuses upon the effects of population density on population growth, generally assuming that density-dependent effects on mean fitness are independent of individual phenotypes (Bellows, 1981; Gilpin & Ayala, 1973). On the other hand, studies of frequency-dependent selection in behavioural ecology (Krebs & Davies, 1993) focus on how the frequency of a phenotype affects its relative fitness, generally assuming that these effects are density-independent. The effects of most social interactions in natural populations lie somewhere in the middle, whenever the effect of population density on an individual's fitness depends on the individual's own phenotype and/or an individual's phenotype affects the fitness of others.

A historical, perhaps arbitrary, distinction can be made between evolutionary approaches initially designed to study the role of density-dependent selection on the evolution of life-history strategies versus those focusing on how the phenotypic and genetic characteristics of the social environment influence evolution. In the former, density-dependent theories of life-history evolution provided one of the first attempts to unite the fields of population ecology and population genetics, implying that the fitness of a genotype is not constant but depends upon population size (Anderson, 1971; Charlesworth, 1971; MacArthur, 1962). Considerable theoretical and empirical work has shown that density-dependent selection is a key determinant of the relationship between phenotypic variation and the carrying capacity of populations (Boyce, 1984; Charlesworth, 1994; Engen *et al.*, 2013, 2020; Joshi *et al.*, 2001; MacArthur & Wilson, 1967; Mueller *et al.*, 1991; Travis *et al.*, 2013; Wright *et al.*, 2019). In the latter, the theory of social evolution has a long tradition of exploring how the genetic and phenotypic characteristics of the social environment can affect short-term evolutionary change (Frank, 1998; Hamilton, 1964; Queller, 1985, 2017; Wolf *et al.*, 1999) and long-term evolutionary equilibria (Maynard Smith, 1982; McGill & Brown, 2007). In particular, game theory has focused on the evolution of the social environment and how it feeds back into patterns of phenotypic selection when the fitness of a strategy is frequency dependent (Araya-Ajoy *et al.*, 2020; Lion, 2018; McGill & Brown, 2007; Queller, 1984; Westneat, 2012).

Frequency-dependent selection has been used to refer to many different processes (see Discussion) and has been identified as a critical factor that will influence evolutionary dynamics (Fisher,

1958; Lande, 1976, 2007; Svensson & Connallon, 2018; Wright, 1948). The evolutionary importance of frequency-dependent selection in density-regulated populations was first acknowledged in the early mathematical formulations of evolutionary population genetics (Fisher, 1958; Wright, 1948), and quantitative genetic models have further elaborated on its effects on phenotypic evolution and population dynamics (Lande, 1976, 2007; Svensson & Connallon, 2018). For instance, recent quantitative genetics models in stochastic environments have shown that if the mean phenotype in the population modulates the strength of density regulation, then frequency- and density-dependent selection are intrinsically linked and jointly determine the expected equilibrium size and mean phenotype of a population (Engen *et al.*, 2020). Furthermore, the interaction between frequency- and density-dependent processes has been widely acknowledged in the theoretical population genetics (Heino *et al.*, 1998; Smouse, 1976) and is a key component of the adaptive dynamics framework (Brown, 2016; Lion, 2018). Despite their demonstrated importance in eco-evolutionary dynamics, empirical investigations rarely study frequency- and density-dependent processes together in a way that properly quantifies their dual effects on phenotypic evolution and population dynamics.

This paper uses statistical models commonly used by empiricists to decompose the effects of different frequency- and density-dependent processes on the eco-evolutionary dynamics of populations. We use generalized linear regression models to describe a set of scenarios where 1) population density, 2) the mean phenotype in the social environment and 3) their interaction affect the mean fitness of the population and the relative fitness of phenotypes. To demonstrate the importance of estimating these different social environment effects, we derive the relationship between the parameters of these models and the strength of selection on a phenotype, as well as the theoretical expectations for the population's equilibrium density and mean phenotype. We complement these results with individual-based simulations to reveal the underlying assumptions of these models, explore the robustness of their statistical implementation, and highlight how they can further our understanding concerning the role of the social environment in mediating the feedbacks linking phenotypic evolution with the carrying capacity of populations.

Methods

A basic model of selection in a density-regulated population

We can model selection in a density-regulated population by studying how population size and the phenotypes of individuals affect fitness (Fig. 1). We assume that a Gaussian fitness function approximates the effects of an individual's phenotype on its fitness (Fig. 1B). This model can be empirically parameterized as a Poisson (or negative binomial) regression:

$$v = \beta_0 + \beta_z z + \beta_q z^2 + \beta_n n \quad , \tag{1a}$$

$$W \sim Poisson(e^v) \quad , \tag{1b}$$

where W is the absolute fitness of individuals, and v represents the expected log fitness of an individual at a given time. In a multiple regression context, β_0 is a constant estimated as the intercept in the model and thus represents the mean fitness of the population when the population size is very small. Coefficients β_z and β_q describe the linear and quadratic components of the relationship between the phenotypic value (z) and log fitness (v) (Lande & Arnold, 1983). The effect of proportional increases in population size on the expected log fitness of individuals is described by the density regulation coefficient β_n, where n represents the (log) population size at different time points. This assumes a theta-logistic model with a θ less than one (Gilpin & Ayala, 1973). It is also possible to model a linear effect of absolute (i.e. not log) population size on log fitness, which is equal to the classic logistic model of density regulation (Bellows, 1981). Accounting for other forms of density regulation may require including additional non-linear terms. Formal modelling of density regulation using individual fitness data also requires the inclusion of random effects for year and individual. These have been left out here to allow for a more concise presentation of the equations. Table 1 describes all the symbols used in the paper.

To connect phenotypic evolution and population dynamics, we need a measure of fitness that varies at the level of each time step. Here, we focus on a fitness measure that reflects an individual's

demographic contribution to the population. This measure directly connects the average fitness of a population at each time step to changes in population size in the next time step. If we focus only on females, this will be the number of new females produced by a mother in a given time-step, plus its own survival into the next time-step (Sæther & Engen, 2015). In a closed population, summing the demographic contribution across all females to the next time-step will be equal to the expected female population size in the next time-step (year or breeding episode):

$$N_{t+1} = \sum(s_t + r_t) = \sum W_t = \bar{W} N_t \quad , \tag{1c}$$

where s are the values describing whether a given female survived or not to time-step $t+1$, and r represents the number of new females produced by each female in time step t that recruit to time step $t+1$. Hence, the mean fitness of the population ($\bar{W}$) in a given time-step multiplied by the female population size in that time-step (N_t) equals the expected female population size at time $t+1$. When the mean of this fitness measure is more than one, populations are expected to grow; if it is less than one, they are expected to decline.

One advantage of using a measure of individual demographic contributions as a fitness measure is that we can describe the characteristics of the density regulation function based upon the parameters in equation 1a. See the Discussion for details regarding the limitations of using this measure of fitness. The density regulation function describes the growth rate of a population as a function of the density-independent reproductive rate (r_0) and the strength of density regulation (γ). We can formulate the log growth rate of the population based on the mean log fitness of the population and the density regulation function. In the absence of immigration and in a deterministic environment, the growth rate of a population can be described as:

$$ln(\bar{W}) = ln(\frac{N_{t+1}}{N_t}) = n_{t+1} - n_t = \bar{v} = r_0 - \gamma n_t \quad , \tag{1d}$$

where n_t is the log population size at time t, and the log mean individual contribution $\bar{v}$ gives the expected change in log population size from time t to t+1 ($n_{t+1} - n_t$). From a statistical perspective, the density-independent growth rate and the strength of density regulation define the

intercept and the slope of the density regulation function. In the results section, we will show how the density regulation function can be described by the parameters of a multiple regression equation and how evolution can shape the equilibrium population size through its effects on the intercept and slope of the density regulation function.

Individual-based simulation

We created an individual-based simulation (IBS) that uses the model described above as a foundation (see Appendix 1 for more details). The IBS focuses on females whose fitness can be affected by their phenotypes as a function of two social environment characteristics: population density and the average phenotype in the population. Interactions between females and their social environment are structured in discrete time steps describing sequential reproductive episodes within a population (e.g. years). The basic features of the IBS are that density regulation causes the population's average fitness to decrease with proportional increases in population density and that the fitness of each female can be affected by her phenotype as well as her social environment. Individuals can be present in more than one time step (i.e. overlapping generations), and population size and the mean phenotype are updated simultaneously for all individuals in each time step.

For simplicity, we assume female demographic dominance under a balanced sex ratio (Rankin & Kokko, 2007), as is common when studying population dynamics. The simulation starts with a population size N_1, which we set to 40 females for all simulations. The population at time step $t+1$ is a function of the number of adult individuals that survive time step t plus the individuals born in time step t recruiting to time step $t+1$. The mean phenotype in the next time step is then determined by the phenotypes of the surviving individuals and the new recruits. This reflects the dynamics of a closed population. We assume adult survival is not affected by an individual's phenotype or social environment and is modelled simply as a Bernoulli process. The average adult survival propensity thus defines the survival probability for all adults. The effects of a female's phenotype and social environment on the number of zygotes she produces is simulated as a Poisson process following equation 1a. For simplicity, we assume that the probability of a zygote produced

at time-step t to recruit to time step $t+1$ is not affected by its own phenotype, and the probability of zygote survival to recruitment is set to one. Therefore, the simulation's stochasticity is solely determined by the average zygote production in a given time step through the variance in the Poisson process and the average adult survival probability via the Bernoulli process.

The phenotypes of the females of the founder population are assumed to conform to a normal distribution with a mean of two and a variance of one. The phenotypes of new individuals are simulated as the average of the phenotype of the parents plus a random deviation reflecting Mendelian variance (half the genetic variance). A random male sires each zygote produced, assuming a balanced sex ratio, and the sire phenotypes conform to a normal distribution with the same mean and variance as the phenotypes of the reproducing females. The standing genetic variance at the beginning of the simulation was set to 1, and for simplicity, there is no phenotypic plasticity in the model. We simulated mutational variance, increasing the variance by half the standing genetic variance at each time step. This means that phenotypic variance at equilibrium is maintained only by mutation-selection balance.

In the following sections, we extend this basic IBS model to explore the different ways in which the social environment affects individual fitness (see Appendix 1 for the full equation). The specific forms in which an individual's phenotype and the characteristics of its social environment affect individual fitness are described by the multiple regression equations 1a, 3a, 4a, 5a, 6a, and 7a. For each simulated scenario using the IBS, we vary the strength of the effect of the social environment on fitness, and we analyze the output data of the IBS as we would an empirical data set. The fixed effect structure for the different scenarios followed the linear regression equation presented for each scenario. All models used to analyze the data included year and individual as random effects. A critical distinction between the simulation and the analyses is that in the individual-based simulation, the social environment only affects reproduction. However, we used statistical models to analyze the effects of the social environment on the total demographic contribution of individuals. This simplifies the presentation of the equations, but in empirical studies, these fitness components could be analyzed separately (see Discussion).

We then proceeded to compare the statistical estimates for the expected population size and mean phenotype derived from the multiple regression estimates against the corresponding observed mean phenotype and equilibrium size of the population for each IBS. We ran the IBS for 200 time steps, ensuring that populations arrived at the equilibrium values for the population density (Fig. 1C) and mean phenotype (Fig. 1D). We then explored how the length of the time series affected the accuracy of equilibrium estimates based on the parameters of the generalized linear mixed models assuming Poisson error distributions. We analyzed the last 10, 20, 30, 40 or 50 time steps of the different individual-based simulations (100 data sets per scenario). Code for simulation and statistical analysis can be found at https://github.com/YimenAraya-Ajoy/Social-environment-eco-evo-dynamics.

Results

Effects of the number of individuals

Density regulation(β_n)

Our first scenario describes a density-regulated population where the impact of density on an individual's fitness is independent of its phenotype, but there is selection on the phenotype that is independent of its social environment, $v = \beta_0 + \beta_z z + \beta_q z^2 + \beta_n n$ (eq. 1a). We model the effect of phenotypes on fitness, assuming a Gaussian fitness function. Therefore, the phenotypic value conferring the highest fitness (θ) is solely defined by the linear and non-linear (quadratic) effects of the phenotype on fitness:

$$\theta = \frac{-\beta_z}{2\beta_q} \quad . \tag{2a}$$

In this scenario, the optimal phenotype is independent of the social environment. The selection differential per time step is also unaffected by the social environment and is given by the covariance between the trait and relative fitness, which is a function of the strength of stabilizing selection and the difference between the population mean phenotype and the optimal phenotype (Lande, 1976).

We can estimate the selection differential for a given time step based upon the parameters of the regression Equation 1a (see Appendix 2 for more details):

$$\frac{1}{\bar{W}}Cov(z, W) = 2\beta_q(\bar{z} - \theta)\sigma_z^2 = (\beta_z + 2\beta_q\bar{z})\sigma_z^2 \quad . \tag{2b}$$

Phenotypic evolution is expected to "push" the population mean phenotypic value towards the optimum. For example, we might imagine the phenotypic trait to be body size, and that selection favours larger individuals because they can more easily capture some recently available larger prey items, causing an increase in the average size of the individuals in the population (Fig. 1D). When populations are perfectly adapted to their non-social environment, the optimum phenotype equals the mean equilibrium phenotype in the population ($\theta = \hat{z}$), and the population arrives at an equilibrium. In the early formulations of Wright's adaptive topography (Wright, 1931), this kind of evolution by natural selection was assumed to increase the population mean fitness, and implicit in this argument is that population size will increase. In a density-regulated population, positive evolutionary change in body size will result in an increase in population size (Fig. 1C), because evolution shapes the elevation of the relationship between log population size and log mean individual fitness (i.e. the population density-independent growth rate; Fig. 1A).

In this scenario, the population mean fitness is given by:

$$\bar{v} = \beta_0 + \beta_z\bar{z} + \beta_q(\bar{z}^2 + \sigma_z^2) + \beta_n n \quad . \tag{2c}$$

The effect of the social environment on population mean fitness through density regulation is captured by β_n. Since this scenario assumes a Gaussian fitness function which is independent of the social environment, both the mean phenotype $\bar{z}$ and its variance σ_z^2 affect population mean fitness through the non-linear effect of an individual's phenotype on its own fitness.

The equilibrium population size is given by the density-independent growth rate and the strength of density regulation $\frac{r_0}{\gamma}$. Rearranging equation 2c, we can infer the expected equilibrium

population size ($\hat{n}$) based upon the estimates of a linear regression:

$$\hat{n} = \frac{r_0}{\gamma} = \frac{\beta_0 + \beta_z \hat{z} + \beta_q(\hat{z}^2 + \sigma_z^2)}{-\beta_n} \quad . \tag{2d}$$

Equation 2d thus describes the way in which the equilibrium population size depends upon the phenotypic distribution through effects on the density-independent growth, but also through the effects of the social environment captured by the strength of density regulation. As implied by equation 2d, the IBS shows that as the strength of density regulation increases the number of individuals a population can sustain decreases (Fig. 2). For example, the strength of density regulation could reflect the degree of scramble competition affecting the number of recruits produced by a population breeding in a limited area (e.g. an island). As population size increases, females have fewer recruits because there are fewer resources for everyone. Variation among populations in the strength of the coefficient β_n in equation 1a could represent different ecological conditions affecting how proportional increases in the number of individuals (i.e. population density) affect the strength of competition.

Density-dependent selection (β_{zn})

The scenario described above assumes that the relative fitness of phenotypes is independent of population density. Therefore, variation in the number of individuals in the social environment only causes changes in mean fitness in the population. However, when there is density-dependent selection, the optimal phenotype also depends upon density, and variation in population density will generate differences in the relative fitness of phenotypes. We can model density-dependent selection by extending eq. 1a to include an interaction (β_{zn}) between population size and an individual's phenotype:

$$v = \beta_0 + \beta_z z + \beta_q z^2 + \beta_n n + \beta_{zn} z n \quad . \tag{3a}$$

The selection differential per time step is thus a function of density- and frequency-independent selection and population size through density-dependent selection:

$$\frac{1}{\bar{W}}Cov(z, W) = (\beta_z + 2\beta_q \bar{z} + \beta_{zn} n)\sigma_z^2 \quad . \tag{3b}$$

See Appendix 2 for more details on the derivation. The equilibrium phenotype in the population thus depends on adaptation to the abiotic environment and to the equilibrium population density:

$$\hat{z} = -\frac{(\beta_z + \beta_{zn}\hat{n})}{2\beta_q} \quad . \tag{3c}$$

With the equilibrium population size also depending upon the equilibrium phenotype through its effects on the density-independent growth rate and the strength of density regulation through β_{zn}:

$$\hat{n} = \frac{r_0}{\gamma} = -\frac{\beta_0 + \beta_z \hat{z} + \beta_q(\hat{z}^2 + \sigma_z^2)}{\beta_n + \beta_{zn}\hat{z}} \quad . \tag{3d}$$

For expanded versions of equations 3c and 3d, see Appendix 3.

Density-dependent selection will affect the equilibrium mean phenotype and shape population density by changing the slope of the relationship between population size and log mean fitness (Fig. 3, right-hand panels). Elaborating on the body mass example above, we can imagine that intermediate-sized individuals are favoured by density-independent selection. However, the fitness consequences of a phenotype can also depend upon density. This effect is captured by the strength of the coefficient β_{zn}, which may reflect different ecological conditions affecting the rate at which the optimal phenotype changes with population density. For instance, larger individuals may be favoured at higher densities due to their ability to capture greater numbers of prey more efficiently (i.e. scramble competition). This will result in a population of larger individuals being affected less by density than a population of smaller individuals. As population density increases, selection favours larger individuals, resulting in a larger equilibrium mean phenotype (Fig. 3, orange lines), leading to a larger population size due to the weaker effect of density regulation. The mean phenotype arrives at equilibrium when the costs of density-independent selection equal the benefits gained

through density-dependent selection. Our IBS shows that when selection is density-dependent then selection maximizes the function describing the equilibrium population size (Fig. 3, left-hand panels) in agreement with classic theoretical results (Engen *et al.*, 2013; MacArthur, 1962). However, as we will demonstrate in the following scenario, this is not necessarily the case when the absolute fitness of a phenotype is frequency-dependent.

Effects of the phenotypes in the social environment

Frequency dependence ($\beta_{\bar{z}}$)

The next scenario represents situations where individual fitness depends not only upon an individual's own phenotype, but also upon the phenotype of other individuals in the social environment. For example, as the mean body size of individuals in the population increases, the amount of resources available to each individual decreases due to contest competition, because individual fitness is more negatively affected by the presence of larger competitors. If we assume that individuals interact at random (i.e. "playing the field"; Maynard Smith, 1982), the effects of this particular aspect of the social environment on individual fitness can be captured by including the effect of the population mean phenotype $\bar{z}$ on the fitness. This effect can thus be included as another coefficient ($\beta_{\bar{z}}$) in the multiple regression equation as:

$$v = \beta_0 + \beta_z z + \beta_q z^2 + \beta_n n + \beta_{\bar{z}} \bar{z} \quad . \tag{4a}$$

In this formulation, the effects of the social environment and of the phenotype of an individual on its fitness are additive (Fig. 4), and therefore, changes in the mean phenotype in the population will not alter the relative fitness of phenotypes.

Equation 4a can be parameterized using the average phenotype with which a given individual interacts, and then the term $\beta_{\bar{z}}$ is related to what has been referred to as a 'social selection gradient'. Social selection gradients quantify the effect of an individual's social partner on its relative fitness within a given breeding episode (Wolf *et al.*, 1999). It has been shown that social selection gradi-

ents affect the expected response to selection when there is non-random assortment of individuals and/or social plasticity (McGlothlin *et al.*, 2010). When interactions between individuals are at random, as in the scenario we describe here, then the effect of the phenotype of individuals in the social environment on an individual's fitness does not create differences in the relative fitness of phenotypes. However, the effect of the phenotype of the average individual in the population on the reproductive success of others is expected to affect the mean fitness in the population and thus influence population growth (Fig. 2).

This frequency-dependent effect on fitness will partly define the relationship between the mean phenotype in the population and its mean fitness (Lande, 1976), further linking the evolutionary trajectory of the phenotype with the dynamics of population size through frequency dependence ($\beta_{\bar{z}}$). Rearranging equation 4a we get:

$$\hat{n} = -\frac{\beta_0 + (\beta_z + \beta_{\bar{z}})\hat{z} + \beta_q(\hat{z}^2 + \sigma_z^2)}{\beta_n} \quad , \tag{4b}$$

where we can see that the equilibrium population size $\hat{n}$ depends upon both the direct effect of the phenotype on fitness through the density-independent growth rate plus the indirect effects that the phenotype of an individual has on the fitness of others ($\beta_{\bar{z}}$). This follows previous work showing that two distinct processes define the effect of the mean phenotype of the population on average fitness (Engen *et al.*, 2020; Lande, 1976, 2007). On the one hand, it is determined by the direct effect of an individual's phenotype on its own fitness (β_z), and on the other by the impact that an individual's phenotype has on the fitness of others ($\beta_{\bar{z}}$). These effects both result in ways in which the intercept of the density regulation function depends upon the population mean phenotype (Fig. 2).

A key realization of early population genetics models was that under many types of frequency dependence, evolution will not always maximize the mean fitness in the population (Fisher, 1958; Wright, 1948). When the direct effect of phenotypes on fitness is positive, and there is also a positive social fitness effect (e.g. $\beta_z > 0$ and $\beta_{\bar{z}} > 0$), the equilibrium population size is larger, as compared with a case where the phenotypes of others have a negative effect on individual fitness (e.g. $\beta_z > 0$

and $\beta_{\bar{z}} < 0$). This first case may represent a (cooperative) social phenotype that allows each individual to utilize resources more efficiently (e.g. cooperative foraging in social spiders; Majer *et al.*, 2018). A population composed of more efficient individuals will free up more resources for use by other individuals in the population, thus increasing average fitness in the population and the population carrying capacity. The other case could represent a competitive phenotype, which allows each individual to monopolize more resources while reducing the resources available for other individuals in the population, thus decreasing its carrying capacity (Fig. 2, blue lines). Therefore, under frequency dependence, the phenotype that maximizes adaptation to the abiotic environment will not necessarily maximize the equilibrium population size. In the IBS scenario with negative frequency dependence lower phenotypic values increased the equilibrium population size, while in the IBS scenario with positive frequency dependence, it was larger phenotypes that maximized the equilibrium population size (Fig. 2).

Frequency-dependent selection ($\beta_{z\bar{z}}$)

We now focus on a scenario where the optimal phenotype depends upon the mean phenotype in the population. Therefore, changes in the mean phenotype change the relative fitness of different phenotypes. Following our body mass example, this might represent a situation where smaller body sizes are favoured when most individuals are large due to the ability of smaller individuals to keep breeding and better withstand the adverse effects of high competition due to reduced somatic maintenance. However, when the population is composed of mostly smaller individuals, selection favours larger bodies that can out-compete all the smaller individuals. This scenario characterizes (negative) frequency-dependent selection models, such as the hawk-dove game (Maynard Smith, 1982), where the fitness benefits of playing dove depend upon the frequency of hawks in the population, and vice versa. In a continuous trait, this leads to a type of balancing selection that results in an intermediate equilibrium mean phenotype. This is captured by the coefficient $\beta_{z\bar{z}}$, representing the interaction between an individual's phenotype and the mean phenotype in the population:

$$v = \beta_0 + \beta_z z + \beta_q z^2 + \beta_n n + \beta_{\bar{z}} \bar{z} + \beta_{z\bar{z}} z\bar{z} \quad . \tag{5a}$$

Fluctuations in the mean phenotype in the population cause changes in the selection differential at each time step due to frequency-dependent selection (Araya-Ajoy *et al.*, 2020; Queller, 1984):

$$\frac{1}{\bar{W}}Cov(z,W) = (\beta_z + 2\beta_q\bar{z} + \beta_{z\bar{z}}\bar{z})\sigma_z^2 \quad . \tag{5b}$$

See Appendix 3 for more details on the derivations, and Araya-Ajoy *et al.* (2020) for the effects of frequency-dependent selection on the selection differential when individual interactions are not random within a population. In the scenario we present here, the equilibrium phenotype is, therefore not only a function of adaptation to the abiotic environment but is also affected by the frequency-dependent selection coefficient:

$$\hat{z} = \frac{-\beta_z}{2\beta_q + \beta_{z\bar{z}}} \quad . \tag{5c}$$

In contrast to density-dependent selection (above), frequency-dependent selection can result in an equilibrium phenotypic value that does not necessarily maximize the expected population size (Fig. 3 middle panels). Under frequency-dependent selection, the effect of the frequency of a phenotype on its relative fitness will preclude adaptation to the non-social environment (eq. 5c).

Frequency-dependent selection is a core component of evolutionary game theory (Maynard Smith, 1982), and within this framework, it is often assumed that the population size is fixed. However, the frequency-dependent selection coefficient can also affect the size of the population:

$$\hat{n} = -\frac{\beta_0 + (\beta_z + \beta_{\bar{z}})\hat{z} + (\beta_q + \beta_{z\bar{z}})\hat{z}^2 + \beta_q\sigma_z^2}{\beta_n} \quad . \tag{5d}$$

Using the IBS, we show that this type of frequency-dependent selection is expected to affect both the equilibrium population size and equilibrium phenotype (Fig. 3 middle panels). Under this type of frequency-dependent selection (see Discussion for other types), the equilibrium mean phenotype will have a non-linear effect on the equilibrium population size (eq. 5d). It will affect the equilibrium population size because it will affect the elevation of the relationship between population size and mean fitness, but not the slope (Fig. 3 middle panels).

Interactions between density and phenotypic frequencies

Frequency-dependent density regulation ($\beta_{n\bar{z}}$)

When the density of the population affects the total amount of resources available, it is likely that this effect depends on the mean phenotype in the population. In other words, the strength of density regulation is frequency-dependent. In the case of the evolution of body mass, we can think about this as the biomass ($n\bar{z}$) of the population affecting individual fitness via competition for a given supply of food resources (Engen *et al.*, 2020; Owen-Smith, 2002). An increase in the number of heavier individuals of greater body mass will reduce the amount of resources disproportionately more *per capita*, as compared to an increase in the number of lighter individuals. This process can be captured by the coefficient $\beta_{n\bar{z}}$ in a linear regression equation, describing frequency-dependent density regulation as the effect on fitness of the interaction between population size (n) and the mean phenotype ($\bar{z}$) in the population:

$$v = \beta_0 + \beta_z z + \beta_q z^2 + \beta_n n + \beta_{\bar{z}}\bar{z} + \beta_{n\bar{z}} n\bar{z} \quad . \tag{6a}$$

The inclusion of the interaction term ($\beta_{n\bar{z}}$) thus redefines the coefficient β_n as the relationship between population size and log fitness when the mean phenotype of the population is zero, and the coefficient $\beta_{\bar{z}}$ as the effect of the average phenotype in the social environment on individual fitness when the population size is very small. Rearranging equation 6a, we can see that the expected equilibrium population size ($\hat{n}$) now depends upon the (equilibrium) population mean phenotype ($\hat{z}$) and how it modulates the strength of density regulation:

$$\hat{n} = -\frac{\beta_0 + (\beta_z + \beta_{\bar{z}})\hat{z} + \beta_q(\hat{z}^2 + \sigma_z^2)}{\beta_n + \beta_{n\bar{z}}\hat{z}} \quad . \tag{6b}$$

Here, the population's mean phenotype now moderates the strength of density regulation as a function of the coefficient $\beta_{n\bar{z}}$. In contrast to the scenario of density-dependent selection, frequency-dependent density regulation does not affect the relative fitness of different phenotypes across

breeding episodes. Nevertheless, it does affect the mean fitness of the population across breeding episodes. Frequency-dependent density regulation reflects how the impact of population density on an individual's fitness depends upon the phenotype of other individuals in the social environment, while density-dependent selection captures how the effects of population density on an individual's reproductive success depend upon its own phenotype.

Early theoretical models focusing on frequency-dependent interactions in density-regulated populations explored the consequences for evolutionary stability of different genotypes having a different impact on density regulation (Anderson & Arnold, 1983; Clarke, 1972). This idea has been recently extended to study the eco-evolutionary dynamics of phenotype-dependent contributions to density regulation in a quantitative genetic framework by Engen *et al.* (2020). We show how frequency-dependent density regulation can be estimated as a regression parameter, and how it affects the equilibrium size of the population through its effects on the slope of the effect of population size on mean fitness (Fig. 2).

Frequency-density-dependent selection ($\beta_{z\bar{z}n}$)

The natural extension of the frequency-dependent density regulation scenario is that the optimal phenotype depends on both the density and frequency of phenotypes in the social environment. In this scenario, density-dependent and frequency-dependent selection are inextricably intertwined because the relative fitness of a phenotype depends upon the number and phenotypes of other individuals in the social environment (Heino *et al.*, 1998; Smouse, 1976). An example could be a situation where the fitness benefits of larger, more competitive body sizes depend upon the biomass of the rest of the population. In the multiple regression equation, we therefore need to include a three-way interaction ($\beta_{z\bar{z}n}$) capturing the interplay between frequency- and density-dependent selection:

$$v = \beta_0 + \beta_z z + \beta_q z^2 + \beta_n n + \beta_{\bar{z}} \bar{z} + \beta_{n\bar{z}} n\bar{z} + \beta_{zn} zn + \beta_{z\bar{z}} z\bar{z} + \beta_{z\bar{z}n} z\bar{z}n \quad . \tag{7a}$$

Here, the coefficient $\beta_{z\bar{z}n}$ captures how the effect of population density on an individual's fitness depends upon the mean phenotype of individuals in the social environment and how this effect, in turn, depends upon the individual's own phenotype. The selection differential per time step is then also a function of population size and the mean phenotype of the population and their interaction (see Appendix 2):

$$\frac{1}{\bar{W}}Cov(z,W) = (\beta_z + [2\beta_q + \beta_{z\bar{z}} + \beta_{z\bar{z}n}n]\bar{z} + \beta_{zn}n)\sigma_z^2 \quad . \tag{7b}$$

In this equation, we can see the different ways in which the frequencies and density of the phenotypes in the social environment are expected to affect the selection differential. The equilibrium phenotype of the population thus depends upon the equilibrium population size,

$$\hat{z} = -\frac{(\beta_z + \beta_{zn}\hat{n})}{2\beta_q + \beta_{z\bar{z}} + \beta_{z\bar{z}n}\hat{n}} \quad , \tag{7c}$$

and the equilibrium size of the population, in turn, also depends upon the equilibrium phenotype,

$$\hat{n} = \frac{\beta_0 + (\beta_z + \beta_{\bar{z}})\hat{z} + (\beta_q + \beta_{z\bar{z}})\hat{z}^2 + \beta_q\sigma_z^2}{\beta_n + (\beta_{zn} + \beta_{n\bar{z}})\hat{z} + \beta_{z\bar{z}n}\hat{z}^2} \quad . \tag{7d}$$

The mutual dependency of the equilibrium mean phenotype and the equilibrium population size depends linearly on density-dependent selection (β_{zn}) and non-linearly on its interaction with frequency-dependent selection ($\beta_{z\bar{z}n}$). In this scenario, frequency-dependent selection and its interaction with density-dependent selection may also hinder adaptation to the abiotic environment (eq. 7c). Therefore, the equilibrium phenotype is not necessarily the one that maximizes the expected population size (Fig. 3, lower panels)

Interestingly, this type of complicated three-way dynamics was actually described in early population genetics models based upon the Lotka-Volterra formulations for multi-species interactions characterized by a matrix of competition coefficients describing how the abundances of each species or genotype affect the fitness of the other genotypes or species (Anderson & Arnold, 1983; Clarke, 1972). The three-way interaction $\beta_{n\bar{z}z}$ thus captures the differential contributions to density regula-

tion of different phenotypes, and the differential sensitivity to population density of those different phenotypes.

Discussion

Phenotypic evolution can cause changes in the social environment in terms of the number and the phenotypes of interacting individuals. These changes will alter the ecological context of selection, altering the strength of selection on the mean phenotype, with cascading effects on population dynamics and phenotypic change. We show how the characteristics of this feedback can be studied using generalized linear models quantifying the selective pressures creating variation in fitness among and within time steps. By combining individual-based simulations with mathematical descriptions, we highlight how parameters of a simple multiple regression relate to fluctuations of the selection differential and the feedback that determines the equilibrium phenotypic distribution in natural populations and how many individuals they can sustain. The exact links between the parameters in our models versus those describing the observed mean phenotype and average density of natural populations are obviously contingent on the various assumptions we highlight when describing the individual-based simulation. However, our goal here was to illustrate the importance of decomposing the different social environment effects on population mean fitness and the relative fitness of phenotypes in order to understand the relative contribution of frequency- and density-dependent processes on the eco-evolutionary dynamics of populations. We did so using generalized linear regression models in the hope that this will resonate with empiricists interested in a topic that is very well studied theoretically but less so empirically.

0.1 Evolution of population size

Evolution by natural selection can influence the equilibrium size of natural populations, shaping phenotypic traits affecting density-independent fitness and traits directly involved in how density affects the fitness of individuals. From a statistical perspective, evolution can be viewed as shaping population size by altering the intercept and/or the slope of the function describing the relationship

between population size and mean fitness. A greater carrying capacity will thus evolve when selection leads to a larger intercept value and/or a shallower slope in the effect of population density on population growth rate. This dichotomy can be seen in equation 7d, where density-independent effects on fitness are grouped in the numerator, whereas these density-dependent effects on fitness are grouped in the denominator.

The relationship between the phenotypic characteristics of a population and its density was the focus of early life-history studies framed in terms of *r*- versus *K*-selection (MacArthur & Wilson, 1967). *r*-selection occurs when populations are at low densities, and selection favours higher reproduction rates, as competition does not constrain individual fitness (Southwood, 1977). In contrast, as populations approach carrying capacity (K), selection favours traits that enhance an individual's ability to monopolize resources in crowded environments or increase their efficiency of resource utilization (Boyce, 1984). If selection favours traits enhancing cooperation and resource efficiency, it will incidentally increase the carrying capacity of populations, fitting the definition of *K*-selection as initially stated by MacArthur & Wilson (1967). One of the earlier criticisms of the *r*- versus *K*-selection dichotomy was that selection under crowded conditions does not necessarily result in higher carrying capacities (Boyce, 1984). This criticism partly stemmed from the observation that selection under crowded environments sometimes favours traits that result in costly investment in 'contest' competition, decreasing the expected equilibrium size of populations (Engen *et al.*, 2020; Joshi *et al.*, 2001). We show that the effect of phenotypic evolution on equilibrium population sizes can be inferred by the sign and magnitude of the regression coefficients $\beta_{\bar{z}}$ and $\beta_{z\bar{z}}$, which describe how the phenotypes in the social environment affect an individual's fitness, for instance, through the access to resources.

Selection under crowded conditions shaping the competitive ability of individuals has been studied under the concept of α-selection (Joshi *et al.*, 2001). An explicit distinction can, therefore, be made between the evolution of density-dependent selection strategies that increase tolerance to crowding through higher efficiency in resource use (*K*-selection) versus the evolution of strategies that competitively inhibit the fitness of others when population density is high (α-selection). Early population genetics models of frequency-density-dependent selection focused upon the evolutionary

consequences of interacting genotypes with different competition coefficients (α) and carrying capacities (Anderson & Arnold, 1983; Clarke, 1972). Models studying similar dynamics have recently been developed using a quantitative genetics framework to study eco-evolutionary dynamics in stochastic environments (Engen *et al.*, 2020). In line with these recent quantitative genetic models, we show how social traits can modulate the strength of density regulation when the effect of population density on individual fitness depends upon the phenotypes of the other individuals in the population ($\beta_{n\bar{z}}$) and/or when the effects of population density on an individual's fitness depend upon its own phenotype (β_{zn}). The relative magnitude of these two processes will reflect the contribution of α- versus K- selection to the equilibrium population size. Importantly, whenever these two processes occur together, density- and frequency-dependent selection are intrinsically linked because the effect of the density of conspecifics and their phenotypes on an individual's fitness depends upon its own phenotype ($\beta_{z\bar{z}n}$). In this case, it is difficult to derive the equilibrium values solely with the parameters from the multiple regression (but see Fig. A1 for a graphical approach).

The many types of frequency-dependent selection

The term 'frequency-dependent selection' is used to describe many different processes, and its definition has been extensively discussed, especially in the context of population genetics and the maintenance of polymorphisms (Ayala & Campbell, 1974; Gromko, 1977; Heino *et al.*, 1998). All uses of the term have in common that the fitness of a phenotype varies with its frequency in the population. However, it is important to distinguish between the different types of frequency dependence, as they have different consequences for phenotypic evolution and population dynamics. We can easily make these distinctions here using our statistical perspective because they are based upon whether the effects on individual fitness of the individual's phenotype versus its social environment are additive, multiplicative or relative.

There are instances where the effect of the average phenotype in the social environment and the effect of an individual's phenotype on its own fitness have additive effects ($\beta_z z + \beta_{\bar{z}}\bar{z}$). This situation has been shown to result in maladaptation and can thus affect population dynamics

(Lande, 1976) as we show with our IBS (Fig. 2). This type of fitness surface can be depicted as a plane (Fig. 4A & B), and so we do not refer to it as frequency-dependent selection but only as 'frequency dependence' because the relative fitness of individual phenotypes does not change as a function of their frequency in the population.

A much clearer type of actual frequency-dependent selection is when an individual's phenotype and the average phenotype in the social environment have multiplicative effects on fitness (Fig. 4C & D). This is the type of frequency-dependent selection we describe in the results section, where an individual's phenotype interacts with the average phenotype of its social environment to affect its fitness ($\beta_{z\bar{z}}\bar{z}z$). This can be represented as a warped fitness surface where the direct effect of a phenotype on fitness changes with the average phenotype in the social environment (Araya-Ajoy *et al.*, 2020).

There are, however, additional scenarios that we did not explore here in which the effect of a phenotype on fitness is relative to the average phenotype in the social environment ($\beta_z[z - \bar{z}]$). This type of frequency-dependent selection includes 'soft' selection (Bell *et al.*, 2021; Wallace, 1975). This can be thought of as a zero-sum game, where a fitness gain by one individual or phenotype results directly in a fitness loss by another. This type of frequency-dependent selection may have no net effect on the mean fitness in the population (Fig. 4E). This type of dynamic relates to a narrow definition that focuses upon types of negative frequency-dependent selection that result in the stable coexistence of polymorphisms (i.e. where the fitness of a phenotype decreases with its relative frequency in the population; (Fig. 4F). This process was at the centre of early developments of the concept of frequency-dependent selection in population genetics (Ayala & Campbell, 1974; Gromko, 1977; Heino *et al.*, 1998; McGill & Brown, 2007). In a quantitative genetic framework, it can be formulated as a type of disruptive selection (Araya-Ajoy *et al.*, 2023; Bürger & Gimelfarb, 2004; Rueffler *et al.*, 2006), where the effects of a phenotype depend on the absolute deviation from the average phenotype in the population ($[z - \bar{z}]^2$). The models presented here can be expanded to study the eco-evolutionary dynamics of this type of "relative" frequency-dependent selection and its interaction with population density.

Selection gradients and short-term responses to selection

Multiple regression is widely used to estimate the direct and indirect effects of phenotypes on reproductive success (Kingsolver & Diamond, 2011). These analyses directly relate to quantitative genetic theory for predicting evolutionary responses to selection (Lande & Arnold, 1983). In a social evolution context, this framework has been extended to study the impact of the social environment on relative fitness. The magnitude of these effects is generally measured as a social selection gradient (Wolf *et al.*, 1999), parameterized in either a neighbour-modulated approach (Okasha, 2006) or contextual analyses of fitness (Goodnight *et al.*, 1992; Heisler & Damuth, 1987). The multiple regression approach used here thus constitutes a critical conceptual and empirical tool to link processes relating phenotypic evolution and the social environment. However, studies on wild populations have been primarily focused on parameterizing their analyses in order to estimate gradients that can then be used to predict expected responses to selection (Lande & Arnold, 1983), in general across generations. It is much less common to parametrize selection models in order to study the responses to selection across time steps, or their consequences on population dynamics and the long-term equilibrium phenotype (but see Sæther *et al.*, 2021, 2016).

An important methodological step when performing selection analyses, that may preclude studying the dual role of the social environment on phenotypic selection and population growth, concerns the standardization of individual measures of fitness by the mean fitness in the population (i.e. calculating relative fitness), and also the scaling of the phenotypic trait by (subtracting) its mean and (dividing by) its standard deviation (De Lisle & Svensson, 2017). These standardizations should be avoided in the context we describe here because they do not allow the simultaneous quantification of the drivers of selection and population dynamics. Re-scaling population mean fitness to one within each selection event will obscure any changes in mean fitness across selection episodes. Standardizing the phenotype by its mean (mean-centring per selective episode) will likewise conceal the changes in selective pressures due to changes in the mean phenotype of the social environment (Araya-Ajoy *et al.*, 2020). Estimating the selection gradients expected to result in short-term responses to selection without standardizing phenotype or fitness is possible. After fitting the sta-

tistical models, the regression coefficients can be standardised to facilitate comparative analyses between traits within or across studies (Dingemanse *et al.*, 2021). In Appendix 1 & 3, we show how the log-linear model presented here relates to selection analyses on relative fitness.

In our presentation of the different frequency and density processes, we use a fitness measure that links individual demographic contributions to the next time-step with the expected changes in population size. We combine annual survival and annual reproduction in a single metric to make the links between phenotypic selection and population dynamics easier to discuss. However, empirical analyses may focus on the separate vital rates. Furthermore, this fitness measure (eq. 1c) needs to be modified when studying the dynamics of males and females in a population. For the log mean individual fitness to add up to the population growth rate, the number of male and female recruits produced by each individual must be divided by 2 (i.e. $w = r/2 + s$) because each recruit has a mother and a father (Sæther & Engen, 2015). However, empirical investigations involving both sexes would instead need to transform the fitness data by multiplying the survival of each individual by two instead of dividing the number of recruits by 2 (i.e. $w = r + 2s$). This ensures that the measure of fitness used in the regression analysis is an integer and can thus be parameterised as a Poisson or negative binomial model. It is then necessary to subtract $\ln(2)$ from the estimated β_0 in the multiple regression to obtain the correct value for the growth rate of the population when its size is small.

It is also very important to note that using 'mixed' measures of fitness in selection analyses can lead to biased estimates of selection and inaccurate inferences about the strength of selection on adult phenotypes. This is because such fitness measures combine different fitness components (e.g. juvenile survival and adult fecundity), making it challenging to distinguish selection on offspring traits from selection on adult traits and obscuring the distinction between selection and inheritance. Again, researchers should analyze each fitness component separately to obtain a more accurate understanding of how selection operates on each trait and how this affects the short-term responses to selection (Hadfield & Thomson, 2017).

Detailed individual long-term studies are necessary to parameterize the models we present here.

We used our IBS models to explore how the length of the time series affects the accuracy and precision of different parameters. These analyses show that estimates are unbiased within the kinds of time spans we explored. However, as expected, the precision increases with the length of the time series (Fig. A2). There are an increasing number of study populations where these types of data are now available (Sheldon *et al.*, 2022). Furthermore, under certain assumptions, similar inferences can be made using fitness proxies to study the types of models we present here. Most importantly, this manuscript highlights the importance of long-term studies collecting detailed individual-based data.

Conclusions

Empirical quantification of the impact of the social environment on phenotypic selection and population growth is critical for our understanding of the interplay between ecological and evolutionary processes in wild populations. By examining the statistical interactions between processes that generate temporal variation in mean fitness versus processes that cause variation in the relative fitness of phenotypes, we can gain valuable insights into the mechanisms underlying the complex eco-evolutionary dynamics driving phenotypic evolution and population dynamics. Regression analysis, commonly used in selection studies, can be used to quantify the ways that variation in the social environment can cause changes in the mean fitness of a population and the relative fitness of phenotypes. Empirical estimates of the parameters quantifying these processes will provide key insights into how the social dynamics of species affect the relationship between the equilibrium mean phenotype of a population and the number of individuals it can sustain. This will improve predictions concerning population-level responses to environmental change in order to make better-informed conservation and management decisions based on the social dynamics of species.

Author contributions

Discussions between YAA, JW, SE and BES lead to the idea of the paper. YA and JW wrote the first draft. YAA and SE derived the equations. YAA and MM performed the simulations. All authors contributed to the later versions of the paper.

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Data Accessibility Statement

Code and simulated data can be found in https://github.com/YimenAraya-Ajoy/Social-environment-eco-evo-dynamics..

Conflict of interest

Authors declare no conflicts of interest.

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1 Main text tables

Parameter	Description
z	Individual phenotypes
$\bar{z}$	Average yearly phenotype of the population
$\hat{z}$	Equilibrium mean phenotype in the population
σ_z^2	Phenotypic variance
v	Individual latent (log) fitness
W	Individual fitness
$\bar{W}$	Average yearly fitness in the population
n	Population sizes
$\hat{n}$	Equilibrium population size
β_n, b_n	Density regulation coefficient
β_z, b_z	Linear selection coefficient, relating phenotypes to absolute fitness
β_q, b_q	Quadratic selection coefficient, relating phenotypes to absolute fitness
β_{zn}, b_{zn}	Coefficient describing density-dependent selection
$\beta_{\bar{z}}$, $b_{\bar{z}}$	Coefficient describing the effects of the average phenotype in the population on individual fitness
$\beta_{z\bar{z}}$, $b_{z\bar{z}}$	Coefficient describing frequency-dependent selection
$\beta_{n\bar{z}}$, $b_{n\bar{z}}$	Coefficient describing frequency-dependent density regulation
$\beta_{z\bar{z}n}$, $b_{z\bar{z}n}$	Coefficient describing the link between density- and frequency-dependent selection
θ	Optimal phenotype independent of the social environment; $\frac{\beta_z}{-2\beta_q}$.
r_0	Density-independent growth rate.
γ	Strength of density regulation.

Table 1: Description of the parameters. Those denoted with a 'β' refer to the estimates from the data in the regression model of fitness, and coefficients denoted with an 'b' refer to the equivalent parameter values determining recruit production in the individual-based simulation model that generated those data.

Scenario	b_n	b_z	b_q	b_{zn}	$b_{\bar{z}}$	$b_{z\bar{z}}$	$b_{n\bar{z}}$	$b_{z\bar{z}n}$
Base scenario	-0.35	0.20	-0.01	0.000	0.00	0.000	0.000	0.0000
Selection	-0.50	0.00	-0.01	0.000	0.00	0.000	0.000	0.0000
Selection	-0.35	0.20	-0.01	0.000	0.00	0.000	0.000	0.0000
Density regulation	-0.32	0.20	-0.01	0.000	0.00	0.000	0.000	0.0000
Density regulation	-0.40	0.20	-0.01	0.000	0.00	0.000	0.000	0.0000
Density-dep. selection	-0.35	0.20	-0.01	-0.005	0.00	0.000	0.000	0.0000
Density-dep. selection	-0.35	0.20	-0.01	0.003	0.00	0.000	0.000	0.0000
Frequency dependence	-0.35	0.20	-0.01	0.000	-0.02	0.000	0.000	0.0000
Frequency dependence	-0.35	0.20	-0.01	0.000	0.02	0.000	0.000	0.0000
Frequency-dep. selection	-0.35	0.20	-0.01	0.000	0.00	-0.005	0.000	0.0000
Frequency-dep. selection	-0.35	0.20	-0.01	0.000	0.00	0.002	0.000	0.0000
Frequency-density-dep.	-0.35	0.20	-0.01	0.000	0.00	0.000	-0.005	0.0000
Frequency-density-dep.	-0.35	0.20	-0.01	0.000	0.00	0.000	0.003	0.0000
Freq.-dens.-dep. selec.	-0.35	0.20	-0.01	0.000	0.00	0.000	0.000	-0.0005
Freq.-dens.-dep. selec.	-0.35	0.20	-0.01	0.000	0.00	0.000	0.000	0.0003

Table 2: Values in the individual based simulations. The b values are the coefficients used in the IBS, determining the effects on recruit production of the social environment and an individual's phenotype. These are analogous to the β parameters that describe the effects on individual fitness, which are presented in the main text. Individual recruit production when population sizes were very small was set to 1.1, and the average survival probability was set to 0.475 for all simulations.

2 Main text figures

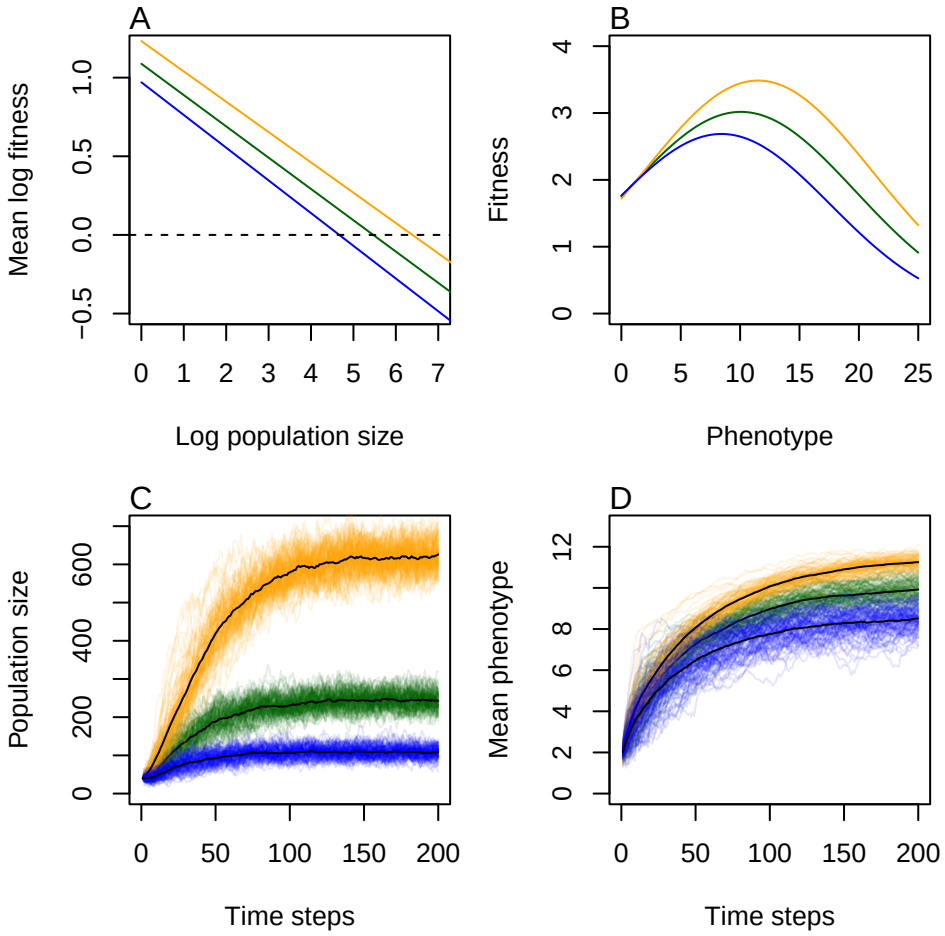

Figure 1: Selection in a density regulated population. (A) The relationship between log population size and the log mean fitness of the population. Population size is expected to arrive at an equilibrium value when log mean fitness equals zero (dashed line). (B) The non-linear function relating individual phenotypes to their mean fitness. (C) The trajectory of population size, and (D) the trajectory of the mean phenotype ($\bar{z}$), from individual-based simulations across 200 time steps for three scenarios given by the colour-coded functions in A and B. In each scenario, we varied the linear effect of individual phenotype on the log fitness parameter (β_z) while keeping all others constant: yellow=0.12; black=0.11; green=0.10. Each simulation is shown in C and D, with the solid lines representing the means for 200 simulations.

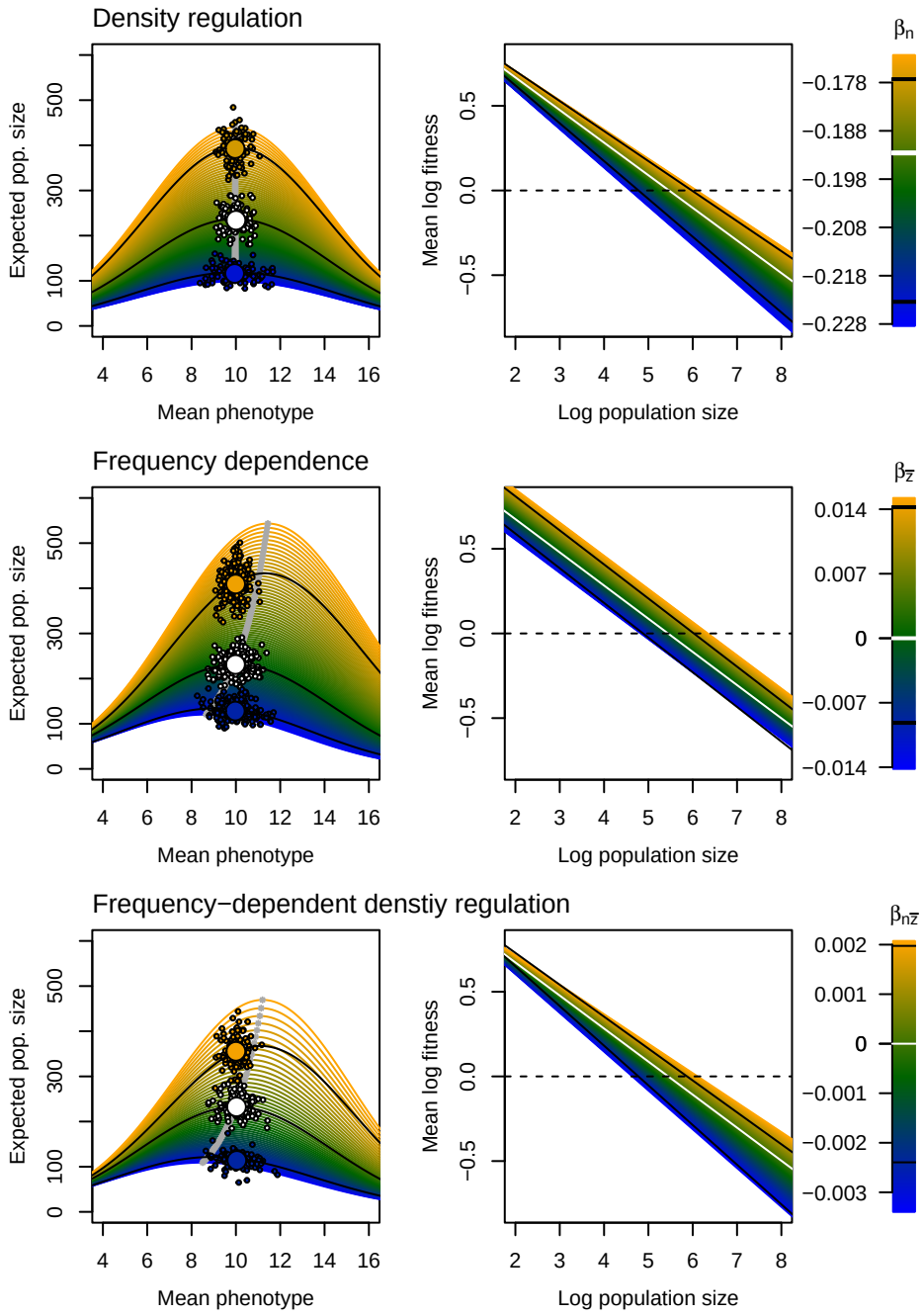

Figure 2: Results of the individual-based simulations for the scenarios of density-regulation (upper panels), frequency-dependence (middle panels) and their interaction (lower panels). See Table 2 for all parameter values. The colours in the different top, middle and lower panels represent the strength of the coefficients β_n, $\beta_{\bar{z}}$ and $\beta_{\bar{z}n}$, respectively. In the left-hand panels, the coloured lines show the predicted values for the expected equilibrium population size ($\hat{n}$) as a function of the mean phenotype in the population ($\bar{z}$), based upon the equations presented in the main text. The many smaller dots represent the equilibrium mean phenotype and population size from each individual-based simulation, whereas the larger dots represent the averages across all the simulations for a given scenario. The black lines represent the predicted values based on the (average) estimates from analyzing the individual-based simulation. The white dots represent the reference scenario, where the only effect of the social environment on fitness is mediated by density regulation. The right-hand panels represent the expected relationship between the log population size and mean log fitness. Black lines represent the predicted values derived from analyzing the individual-based simulations, and white lines are again from the reference scenario.

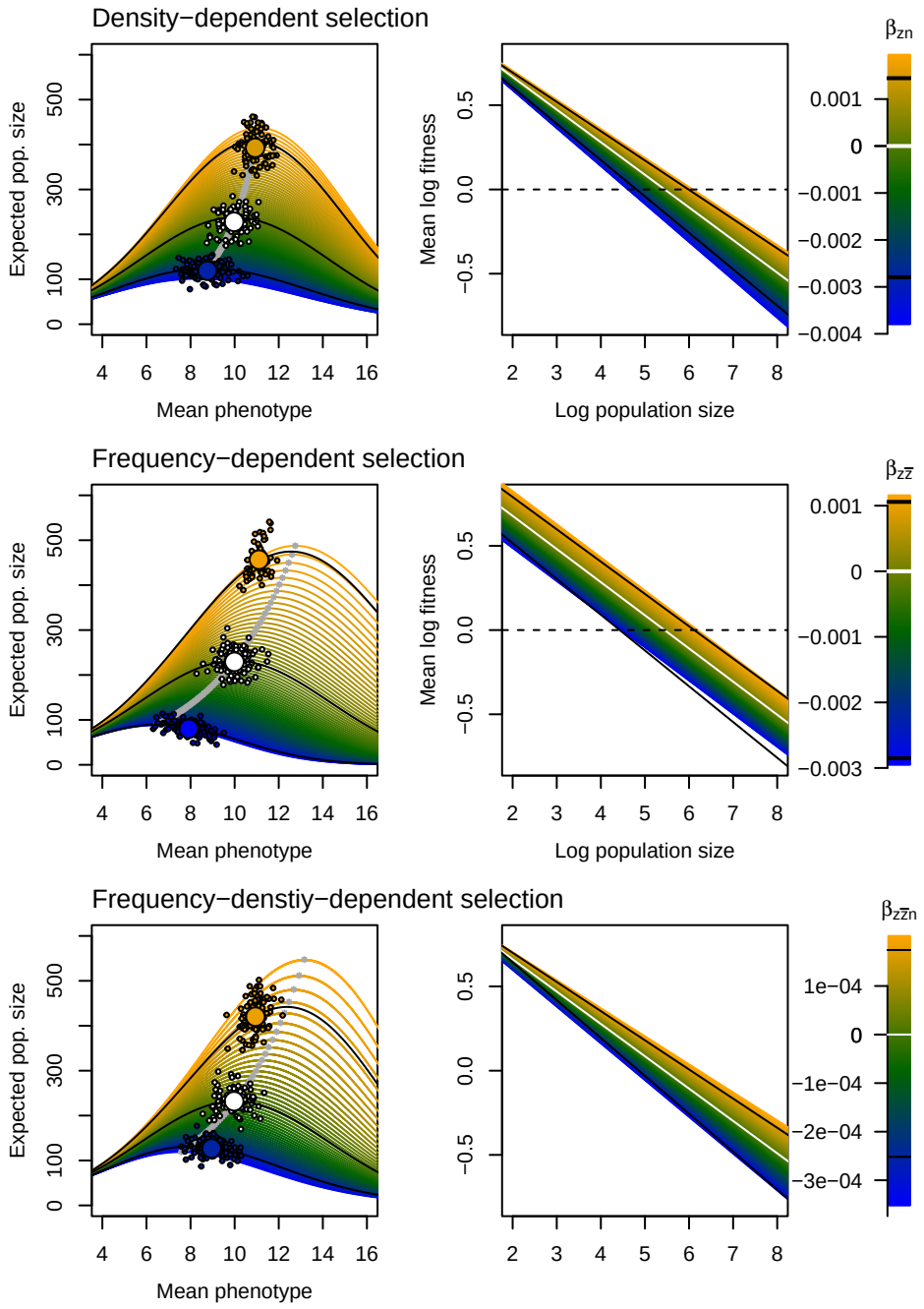

Figure 3: Results of the individual-based simulation for the scenarios of density-dependent selection (upper panels), frequency-dependent selection (middle panels) and their interaction (lower panels). See Table 2 for all parameter values. The colours in the different top, middle and lower panels represent the strength of the coefficients β_{zn}, $\beta_{z\bar{z}}$ and $\beta_{z\bar{z}n}$, respectively. In the left-hand panels, the coloured lines show the predicted values for the expected equilibrium population size ($\hat{n}$) as a function of the mean phenotype in the population ($\bar{z}$), based upon the equations presented in the main text. The many smaller dots represent the equilibrium mean phenotype and population size from each individual-based simulation, whereas the larger dots represent the averages across all the simulations for a given scenario. The black lines represent the predicted values based on the (average) estimates from analyzing the individual-based simulation. The white dots represent the reference scenario, where the only effect of the social environment on fitness is mediated by density regulation. The right-hand panels represent the expected relationship between the log population size and mean log fitness. Black lines represent the predicted values derived from analyzing the individual-based simulations, and white lines are again from the reference scenario.

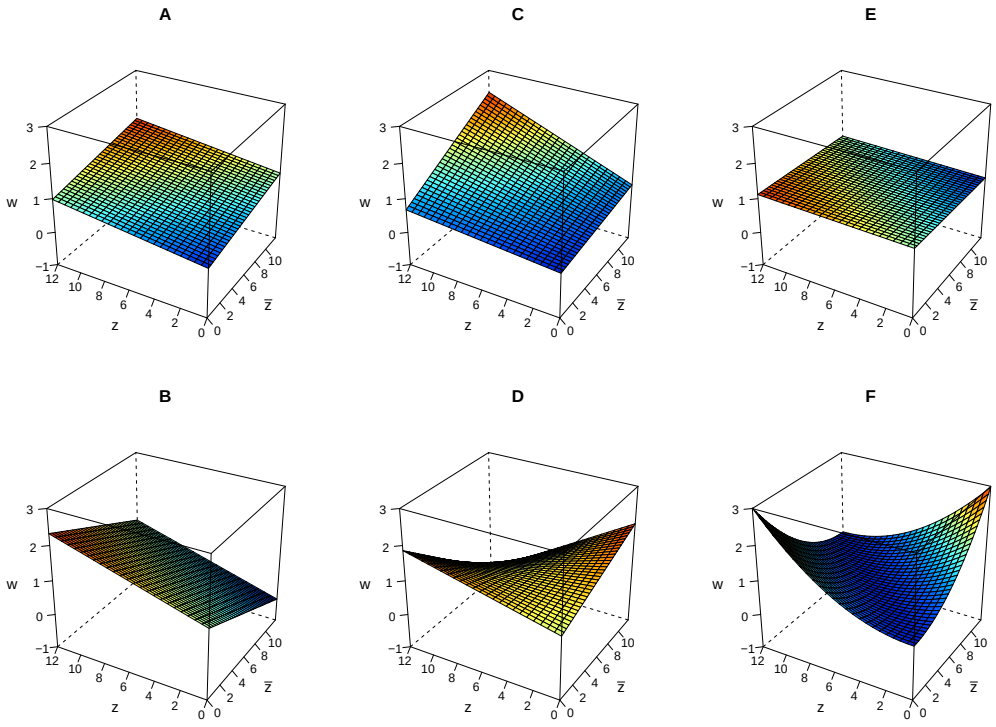

Figure 4: Fitness surfaces for different types of frequency-dependent selection. The left-hand panels represent scenarios of (A) positive and (B) negative frequency dependence in which the effects on fitness of an individual's phenotype and that of its social environment have additive effects ($\beta_z \mathbf{z} + \beta_{\bar{z}} \bar{\mathbf{z}}$). The middle panels represent scenarios of (C) positive and (D) negative frequency-dependent selection in which an individual's phenotype interacts with its social environment to affect its fitness ($\beta_{z\bar{z}} \bar{\mathbf{z}} \mathbf{z}$), such that the fitness function depends upon a product of the mean phenotype in the social environment. The right-hand panels (E) and (F) represent scenarios of frequency-dependent selection in which the effects of an individual's phenotype on its fitness are relative to the average phenotype in its social environment, with (E) showing a scenario of positive selection for having a higher phenotypic value than that of the average individual in the social environment ($z - \bar{z}$), and (F) a scenario of negative frequency-dependent selection (and the evolution of polymorphisms) in which the effects of the phenotype depend upon its absolute deviation from the mean phenotype in the social environment ($(z - \bar{z})^2$).

Supplementary material

Appendix 1: Equations and approximations for the individual-based simulation

In the individual-based simulation (IBS), deterministic variation in fitness within and among time steps is underpinned by variation in recruit production, assuming that individuals have the same underlying survival probability ϕ. The equation underpinning the IBS and the statistical model that can be used to estimate the effects on recruit production (r) of an individual's phenotype (z), population size (n) and the mean phenotype in the population ($\bar{z}$) can be written as:

$$\rho = b_0 + b_n n + b_z z + b_q z^2 + b_{\bar{z}} \bar{z} + b_{n\bar{z}} n\bar{z} + b_{z\bar{z}} z\bar{z} + b_{zn} zn + b_{z\bar{z}n} z\bar{z}n + e \quad , \tag{A1.1a}$$

$$r \sim Poisson(e^{\rho}) \quad . \tag{A1.1b}$$

Here b_0 is the average log recruit production in the population when it is very small. Population size effects on the log number of recruits is described by the density regulation coefficient b_n. An individual's recruit production can be affected by its own phenotype as a function of the linear (b_z) and quadratic (b_q) effects of the phenotype on fitness. The number of recruits produced by an individual can also depend upon the (average) phenotype ($\bar{z}$) of the individuals in the population modulated by the coefficient $b_{\bar{z}}$. Furthermore, the average phenotype in the population can also modulate the strength of density regulation as a function of the interaction coefficient $b_{n\bar{z}}$. The optimal phenotype depends upon the number of individuals in the population (b_{zn}) and also upon the mean phenotype in the population ($b_{z\bar{z}}$). Ultimately, the relationship between phenotype and fitness may also depend upon an interaction between the number of individuals and the phenotype of the average individual in the population ($b_{z\bar{z}n}$).

Based upon the equations presented here, we can approximate the strength of selection per generation due to reproduction using the log-linear effects of an individual's phenotype (z), population size (n) and the mean phenotype in the population ($\bar{z}$) on recruit production. This can be

expressed as:

$$W(z; n, \bar{z}) = \phi + r(z; n, \bar{z}) \quad , \tag{A1.2}$$

where ϕ is the average survival probability and r is the expected number of recruits produced by a phenotype. Assuming a linear model on log recruit production ($\rho = \ln r$), as we assume in the IBS, at a given value of $\bar{z}$ and n the mean log number of recruits fluctuates around the expected value $\ln \bar{r}$, which is the expected value of ρ with respect to variation in z in the population. The deviation of each phenotype's expected log recruit production (ρ) can thus be expressed as:

$$\Delta\rho = \rho - \bar{\rho} \quad . \tag{A1.3}$$

The first-order approximation of r is thus:

$$r = e^{\bar{\rho}+\Delta\rho} \approx \bar{r} + \bar{r}\Delta\rho \quad . \tag{A1.4}$$

The corresponding approximation to the fitness function at $\bar{z}$ and n is now:

$$W(z, n, \bar{z}) = \phi + \bar{r} + \bar{r}\Delta\rho(z, n, \bar{z}) \quad , \tag{A1.5}$$

and since the mean fitness at $\bar{z}$ and n is $\bar{w} = \phi + \bar{r}$, the relative fitness is:

$$\frac{W}{\bar{W}} = 1 + \frac{\bar{r}}{\phi + \bar{r}}\Delta\rho(z, n, \bar{z}) \quad . \tag{A1.6}$$

The strength of selection is now given by the gradient of mean relative fitness taken only with respect to $\bar{z}$, obtained by averaging across the distribution of z in the population at a given time step. The strength of directional selection will vary as a function of how far the mean phenotype of the populations is from the optimum phenotype. While under density- and frequency-dependent selection, it will depend upon n and z, or both. The gradient of $\Delta\rho$ can be considered as the covariance between the trait and relative fitness at a time step (episode) where the population size is n and the mean phenotype $\bar{z}$. Since the gradient of $\Delta\rho$ is the same as that of ρ, under certain

assumptions of inheritance (see Discussion in the main text), we can write the expected response to selection as:

$$\Delta\bar{z} = h^2\sigma_z^2 \frac{\bar{r}}{\phi + \bar{r}} \nabla\bar{\rho}(\bar{z}, n, \bar{z}^*) \quad , \tag{A1.7}$$

where h^2 is the heritability, and the selection gradient is taken with respect to $\bar{z}$. Note that we insert $\bar{z}^* = \bar{z}$ to clarify that the selection gradient is with respect to the direct effect of the mean phenotype on fitness. Notice that at equilibrium, defined by $\bar{W} = 1$, we have $\bar{r} = 1 - \phi$ giving:

$$\Delta\bar{z} = \frac{h^2 P \nabla\bar{\rho}(\bar{z}, n, \bar{z}^*)}{T} \quad , \tag{A1.8}$$

where $T = 1/(1-\phi)$ is the generation time measured at equilibrium. For a fluctuating population, however, the factor $\bar{r}/(\phi + \bar{r})$ is a function of $\bar{z}$ and N, and therefore fluctuates around T. In a finite population, with zero mean and variance in $\Delta\bar{z}$ during a generation, the genetic drift is $h^2P/(N)$, and during a time step $h^2P/(NT)$, giving:

$$\Delta\bar{z} = h^2 P \frac{\bar{r}}{\phi + \bar{r}} \nabla\bar{\rho}(\bar{z}, N, \bar{z}^*) + \sqrt{h^2P/(NT)}\, U_{drift} \quad , \tag{A1.9}$$

where U_{drift} is a standard normal variable.

It is important to note that $\Delta\bar{z}$ will represent the strength of selection on the mean phenotype when survival (ϕ) is independent of the phenotype and when the population is at equilibrium. We present an analogous equation expressed in terms of the estimates of a regression on individual fitness in Appendix 3.

Scenario	**Gradient** ($\nabla\bar{v}$)
Direct selection $W(z,n)$	$b_z + 2b_q\bar{z}$
Density dep.-selection $W(z,n)$	$b_z + 2b_q\bar{z} + b_{zn}n$
Frequency dep.-selection $W(z,\bar{z})$	$b_z + 2b_q\bar{z} + b_{z\bar{z}}\bar{z}$
Dens.- freq.-dep. selection $W(z,n,\bar{z})$	$b_z + 2b_q\bar{z} + b_{z\bar{z}}\bar{z} + (b_{zn} + b_{z\bar{z}n}\bar{z})n$

Table A1.1: Equations to predict the fluctuations in mean fitness and thus the effects on population size, as well as the expected changes in mean phenotype in the population based on the equations presented in the text. For the definitions of parameters see Table 1 in the main text.

Appendix 2: Expected selection diferentials

We can approximate the selection gradients that contribute to the expected evolutionary change in the mean phenotype of the population from one time step to the next based on the log-linear effects of the phenotype on fitness (w). It is important to keep in mind here that we are focusing on several time steps together, where both the mean fitness of the population and the selection gradients fluctuate due to changes in the number of individuals in the population and its mean phenotype. The different scenarios presented in the main text assume that fitness (w) is dependent upon an individual's phenotype (z), population size (n) and the mean phenotype of the population ($\bar{z}$). The fitness function can thus be written as:

$$W(z;n,\bar{z}) \quad . \tag{A2.1}$$

The models presented in the main text describe the effects of phenotypes and population size on log fitness, $\ln w$, which can thus be thought of as a population growth rate measure (v). Therefore, the underlying fitness model is: $\ln W = v(z;n,z)$. At a given value of $\bar{z}$ and n, the population growth rate fluctuates around the expected value $\bar{v} = ln\bar{W}$, which is the expected value of v with respect to variation in z in the population within a time-step. The deviation Δv of each phenotype's expected growth rate v from the population mean growth rate ($\bar{v}$) in a given time-step can thus be expressed as:

$$\Delta v = v - \bar{v} \quad . \tag{A2.2}$$

We can infer a phenotypes fitness (W) from its growth rate (v) using a first order approximation, where

$$\boldsymbol{W} = e^{\bar{v}+\Delta v} \approx \bar{W} + \bar{W}\Delta v \quad . \tag{A2.3}$$

The corresponding approximation to the fitness function at $\bar{z}$ and n can thus be expressed as:

$$W(z, \bar{z}, n) = \bar{W} + \bar{W}\Delta v(z, n, \bar{z}) \quad , \tag{A2.4}$$

where the expected population size in the next time step is equal to $ne^{\bar{v}}$, and the relationship between the phenotype and relative fitness can then be expressed as:

$$\frac{W}{\bar{W}} = \frac{\bar{W} + \bar{W}\Delta v(z, n, \bar{z})}{\bar{W}} = 1 + \Delta v(z, n, \bar{z}) \quad . \tag{A2.5}$$

The selection gradient is now given by the gradient of relative fitness taken only with respect to the $\bar{z}$ resulting from averaging over the distribution of z in the population. The gradient of Δv can be thought of as the covariance between the trait and relative fitness for a given time step (episode) where the population size is n and the mean phenotype $\bar{z}$. Note that log fitness is very similar to dividing fitness by its mean, and thus the model involving v is a very close approximation to a model of relative fitness ($\frac{w}{\bar{w}}$). The selection gradient for a given episode can thus be approximated based on the estimates (β) from the log-linear effects on fitness. The directional selection gradient will vary as a function of how far the mean phenotype of the population is from the optimum phenotype, while under density- and frequency-dependent selection it will also depend upon n and z, respectively, and in the most complex scenario we have sketched in the main text, it can depend upon both. Since the gradient of Δv is the same as that of $\bar{v}$, we can write the expected evolutionary change as:

$$\Delta\bar{z} = h^2 P \nabla \bar{v}(\bar{z}, N, \bar{z}^*) \quad , \tag{A2.6}$$

where h^2 is the heritability, P is the phenotypic variance, and the gradient is taken with respect to $\bar{z}$. Note that we insert $\bar{z}^* = \bar{z}$ to specify that the gradient here is with respect to the direct

effect of the phenotype on fitness. It is very important to clarify that this equation can be used to predict the expected evolutionary change in our simulation because fitness variation is solely caused by effects on recruit production and we are assuming a very simplistic mode of inheritance (e.g. no permanent environmental effects). Utilizing the number of recruits as a measure of fitness has been shown to provide estimates of selection gradients that will produce biased estimates of the expected evolutionary change. If the aim is to estimate the expected evolutionary change from one episode to the next, it is important to disentangle the effects of the phenotype on fecundity (zygotes produced), juvenile survival and adult survival. Appendix 1 provides an alternative way to estimate these selection gradients, which can provide more precise statistical estimates when the effects of the phenotype on individual fitness are only mediated through fecundity.

Appendix 3: Supplementary equations for the density-dependent selection scenario

The equilibrium population size when there is density-dependent selection can be expressed as:

$$\hat{n} = -\frac{2\beta_n\beta_q - \beta_{zn}\beta_z - 2\sqrt{\beta_{zn}^2\beta_q^2\sigma_z^2 + \beta_n^2\beta_q^2 - \beta_n\beta_{zn}\beta_q\beta_z + \beta_{zn}^2\beta_q\beta_0}}{\beta_{zn}^2}. \tag{A3.1}$$

The equilibrium phenotype when there is density-dependent selection can be expressed as:

$$\hat{z} = -\frac{2\beta_n\beta_q + \sqrt{\beta_{zn}^2\beta_q^2\sigma_z^2 + \beta_n^2\beta_q^2 - \beta_n\beta_{zn}\beta_q\beta_z + \beta_{zn}^2\beta_q\beta_0}}{\beta_{zn}\beta_q}. \tag{A3.2}$$

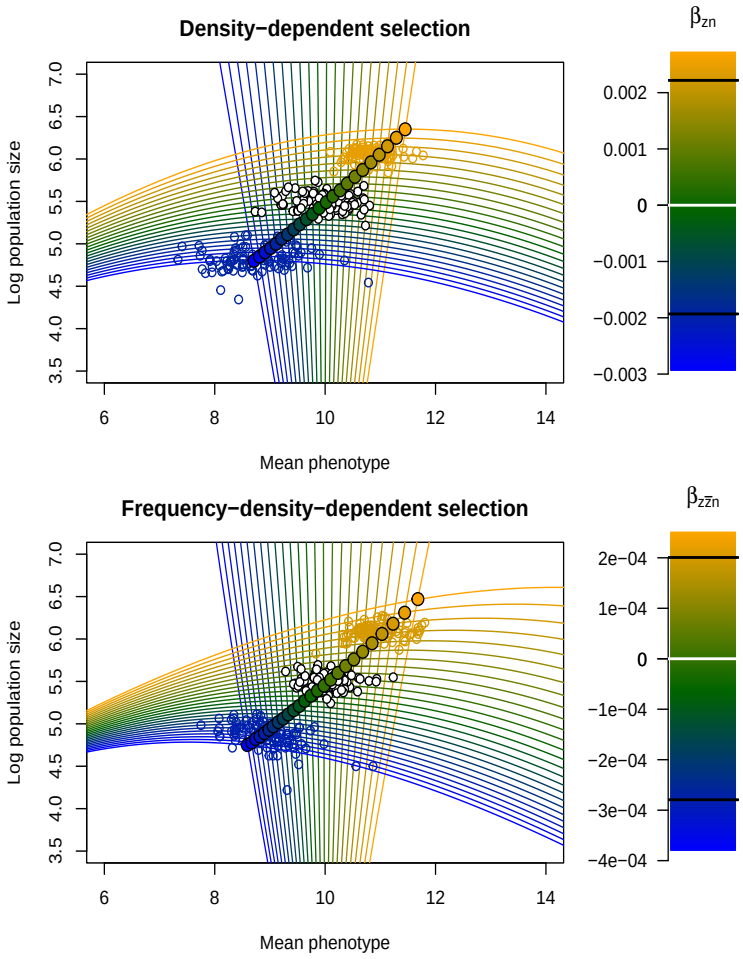

Figure A1: Equilibrium mean phenotypes and population densities for density- and frequency-density-dependent selection. In these scenarios, the optimal phenotype partly depends upon the population density, while population density also depends upon the optimal phenotype. For (A) the density-dependent selection scenario, we can derive the analytical expectation of the equilibrium population density and mean phenotype based upon the parameters of the multiple regression equation (see Appendix 3). However, this is often not possible, and an alternative approach is to use the parameters from the multiple regression equation to predict the expected mean phenotype for a given (log) population size, depicted here as the more vertical lines, and the predicted (log) population size for a given mean phenotype, represented here by the more horizontal lines. The point of intersection of these two lines, denoted by the filled dots, corresponds to the equilibrium mean phenotype and population size in each case. Colours represent the strength of the coefficients β_{zn} and $\beta_{z\bar{z}n}$ capturing how the optimal phenotype depends upon the characteristics of the social environment. The non-filled dots represent the equilibrium mean phenotype and population densities from different individual-based simulation scenarios (see Table 2). The white dots represent the reference scenario, where the only effect of the social environment on fitness is mediated by density regulation.

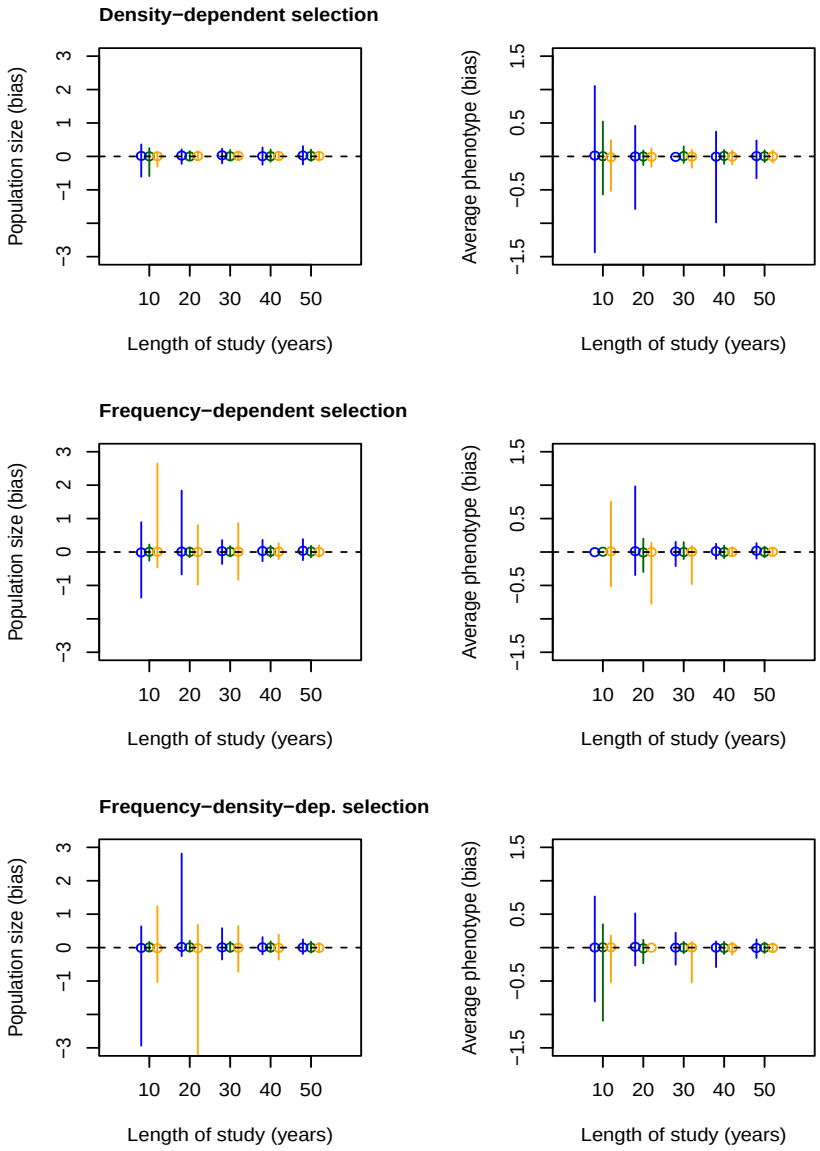

Figure A2: Distributions of the deviations (bias) of the estimated equilibrium population sizes and mean phenotypes based upon the multiple regression parameters from the observed equilibrium mean phenotype and population size in the individual-based simulation. The results are shown from analyzing a different number of time steps (e.g. years) from the data generated by the individual-based simulations. We analyzed the last 10, 20, 30, 40 or 50 time steps of the different individual-based simulations (100 data sets per scenario). We aimed to explore how the length of the time series affected the accuracy (and bias) of these equilibrium estimates based upon the parameters of the generalized linear mixed models. The median (points) and 95% confidence intervals of the differences are presented. All models used to analyze the data included year and individual as random effects. The fixed effect structure and error distributions were presented in the main text equations. Colours correspond to the different magnitudes of social environment effects on fitness. Blue shows the scenarios with negative effects of the social environment, and yellow shows the scenarios with positive social environment effects. The colour coding corresponds to the one in the main text