A quantitative genetics approach to the evolution of phenotypic (co)variance under limited dispersal, with an application to socially synergistic traits

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Abstract

Darwinian evolution consists of the gradual transformation of heritable quantitative traits due to natural selection and the input of random variation by mutation. Here, we use a quantitative genetics approach to investigate the coevolution of multiple traits under selection, mutation, and limited dispersal. We track the dynamics of trait means and variance-covariances between traits that experience frequency-dependent selection. Assuming a multivariate-normal trait distribution, we recover classical dynamics of quantitative genetics, as well as stability and evolutionary branching conditions of invasion analyses, except that due to limited dispersal, selection depends on indirect fitness effects and relatedness. In particular, correlational selection that associates different traits *within*-individuals depends on the fitness effects of such associations *between*-individuals. These kin selection effects can be as relevant as pleiotropy for correlation between traits. We illustrate this with an example of the coevolution of two social traits whose association within-individual is costly but synergistically beneficial between-individuals. As dispersal becomes limited and relatedness increases, associations between-traits between-individuals become increasingly targeted by correlational selection. Consequently, the trait distribution goes from being bimodal with a negative correlation under panmixia to unimodal with a positive correlation under limited dispersal. More broadly, our approach can help understand the evolution of intra-specific variation.

Keywords. Island model, G-matrix evolution, evolutionary branching, division of labour, social evolution

1 Introduction

Understanding how heritable quantitative traits are moulded by the processes of natural selection and mutation has been a longstanding goal of evolutionary biology. This research program has led to an abundant theoretical literature that seeks to understand the roles of ecology and genetics in the gradual transformation of phenotypes. Notwithstanding this abundance, models of gradual evolution usually follow one of two approaches, depending on whether the focus is put on ecological or genetic processes.

One approach consists in investigating the invasion success of a rare phenotypic mutant (i.e., an evolutionary invasion analysis, e.g. Michod, 1979, Eshel and Feldman, 1984, Parker and Smith, 1990, Eshel et al., 1997; also referred to as "Adaptive Dynamics", e.g., Dercole and Rinaldi, 2008, for a textbook treatment) and places emphasis on ecology (or on how organisms interact with one another via effects on resources and the environment). In most practical applications, this emphasis comes at the expense of genetics realism. In particular, trait dynamics inferred from invasion analyses most often assume that mutations have weak quantitative effects and

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are so rare (relative to the strength of selection) that at most two alleles can segregate in the population. In this case, a sensitivity analysis of the invasion fitness of a rare mutant in a resident monomorphic population that is at its ecological equilibrium (e.g., Michod, 1979, Eshel and Motro, 1981, Eshel and Feldman, 1984, Taylor, 1989, Parker and Smith, 1990, Charlesworth, 1994) can be used to understand gradual trait evolution and the ecological transformations due to this evolution (Metz et al., 1996, Geritz et al., 1998, Rousset, 2004, Dercole and Rinaldi, 2008, Metz, 2011). The approach of invasion analysis is therefore particularly well-suited to investigate the evolution of traits under ecological feedbacks and the frequency-dependent selection that emerges due to such feedbacks (e.g., Kisdi and Geritz, 2009, Lion, 2018, and references therein). This approach has revealed that in the presence of trade-offs, gradual evolution under ecological feedbacks often leads to the emergence of polymorphism. Here, the population evolves under directional selection to express a trait value such that any rare mutant has an advantage over the common resident (Eshel and Motro, 1981, Eshel, 1983, Taylor, 1989, Christiansen, 1991, Abrams et al., 1993b). As a result, the population subsequently splits into two lineages of distinct phenotypes, or morphs, in a process referred to as evolutionary branching (Geritz et al., 1998; see Rueffler et al., 2006, Kisdi and Geritz, 2009, for reviews).

By contrast to invasion analysis, evolutionary quantitative genetics models of gradual evolution tend to be more preoccupied with the genetic basis of traits (Roff, 1997, Lynch and Walsh, 1998). Importantly, quantitative genetics models envisage that there is substantial heritable phenotypic variation in the population. The continuum-of-alleles model, in particular, posits that quantitative traits are determined by a continuum of possible alleles produced by mutation (e.g., Kimura, 1965b, Latter, 1970, Fleming, 1979, Bürger, 1986). A quantitative genetics approach aims to investigate the roles of selection and mutation in the gradual evolution of a phenotypic distribution of arbitrary complexity. Due to the complication of dealing with multiple variants, however, analytical explorations of quantitative genetics models usually come at the expense of generality. Notably, the vast majority of quantitative genetics models of traits under frequency-dependent selection, which is either implicit or emerges from ecological interactions, focuses on the evolution of mean phenotypic values in the population, assuming that heritable phenotypic variation is constant (i.e., additive genetic variances and covariances are fixed, e.g., Lande, 1976, 1981, Iwasa et al., 1991, Gomulkiewicz and Kirkpatrick, 1992, Abrams et al., 1993a, Iwasa and Pomiankowski, 1995, Day and Taylor, 1996, Tazzyman and Iwasa, 2009, Nuismer et al., 2010, Connallon, 2015).

But phenotypic variance should be especially sensitive to frequency-dependent selection (because such selection regime either favours or disfavours rare variants that differ from the most common, it increases or decreases trait variance, respectively; Slatkin, 1980, Taper and Chase, 1985, Taylor and Day, 1997, Day and Proulx, 2004, Sasaki and Dieckmann, 2011, Wakano and Iwasa, 2013, Wakano and Lehmann, 2014, Débarre et al., 2014, Débarre and Otto, 2016). In fact, recent quantitative genetics models investigating populations of individuals experiencing frequency-dependent interactions have revealed links between the dynamics of phenotypic variance and evolutionary branching (Sasaki and Dieckmann, 2011, Wakano and Iwasa, 2013, Wakano and Lehmann, 2014, Débarre et al., 2014, Débarre and Otto, 2016; thereby extending the links between the dynamics of the phenotypic mean in quantitative genetics models and directional selection in invasion analysis models, Charlesworth, 1990, Iwasa et al., 1991, Taper and Case, 1992, Abrams et al., 1993a; for reviews: Abrams, 2001, Lion, 2018). In particular, evolutionary branching occurs in a quantitative genetics model when the phenotypic variance is predicted to grow without bound while the phenotypic mean is held constant, under the assumption that the phenotypic distribution is normal (this assumption allows to only have to consider the dynamics of the mean and variance of the phenotypic distribution, Wakano and Iwasa, 2013, Wakano and Lehmann, 2014, Débarre et al., 2014, Débarre and Otto, 2016). As evolutionary branching occurs, the variance may in fact converge to a bounded value (see Fig. 2e-f of Débarre and Otto, 2016), but these dynamics cannot be captured by models that assume that the phenotypic distribution is normal and thus unimodal (instead of a bi- or multi-modal distribution; see Sasaki and Dieckmann, 2011 and Appendix D of Débarre and Otto, 2016 for a relaxation of the unimodal assumption). In spite of this limitation, quantitative genetics approaches have been useful to investigate relevant factors for frequency-dependent selection and evolutionary branching, such as genetic drift (with fixed, Wakano and Iwasa, 2013, or fluctuating, Débarre and Otto, 2016, population size)

or the interaction between multiple traits (Débarre et al., 2014).

One factor that is particularly relevant for frequency-dependent interactions is limited dispersal. This is because limited dispersal creates genetic structure, whereby individuals that interact and compete with one another are more likely to share identical alleles than individuals randomly sampled from the population (Hamilton, 1964, Frank, 1998, Rousset, 2004). Using an invasion analysis, a number of models have investigated the conditions that lead to disruptive selection (usually followed by evolutionary branching) due to frequency-dependent interactions among individuals under limited dispersal (Day, 2001, Ajar, 2003, Rousset, 2004, Mullon et al., 2016, Parvinen et al., 2018; see also Svardal et al., 2015 for evolutionary branching due to spatial and temporal heterogeneities in selection). Using a quantitative genetics approach, Wakano and Lehmann (2014) (WL14 hereafter) found branching conditions equivalent to those obtained from invasion analysis by studying the dynamics of the variance of a trait under limited dispersal. The analysis of frequency-dependent and disruptive selection under limited dispersal has helped reveal further connections between invasion analysis and fundamental branches of evolutionary theory. In particular, Ajar (2003), WL14 and Mullon et al. (2016) expressed disruptive selection coefficients in terms of relatedness coefficients, which are quantities central to population genetics, kin selection and social evolution theory (i.e., the evolution of traits that influence the fitness of their actor and recipient, such as helping or harming, e.g., Hamilton, 1964, Frank, 1998, Rousset, 2004, Wenseleers, 2010; see also Kisdi, 2016 for a kin selection perspective on evolutionary branching of dispersal).

In this paper, we incorporate two further relevant factors that have previously been omitted in the gradual evolution of quantitative traits when selection is frequency-dependent and dispersal is limited. First, we consider the joint evolution of multiple traits (whereas WL14 focuses on a single trait). This enables us to investigate how phenotypic covariances among traits within individuals are moulded by frequency-dependent selection and pleiotropic mutations (i.e., when traits share a common genetic basis so that mutations have correlated effects across traits). Second, we model the coupled dynamics of the phenotypic means and (co)variances (whereas WL14 looks at the dynamics of the variance only once selection on means is negligible). This allows for a more complete picture of the dynamics of the phenotypic distribution. By expressing these dynamics in terms of relatedness coefficients, we further connect kin selection theory with the evolutionary quantitative genetics of multiple traits (Lande, 1979, Lande and Arnold, 1983, Phillips and Arnold, 1989, Brodie et al., 1995; in particular with the evolution of the **G**-matrix of additive genetic variance-covariance, Steppan et al., 2002, Arnold et al., 2008)

The rest of this paper is organised as follows. We describe the life-cycle and population structure under consideration in section 2. Our first result, presented in section 3.1, is an equation for the one-generational change of a multi-variate phenotypic distribution under limited dispersal, mutation, and selection. Next, in section 3.2, we present a closed dynamical system for the mean vector and variance-covariance matrix of the phenotypic distribution, under the assumption that the distribution in the whole population is normal. Further, we express this dynamical system in terms of effects on individual fitness and relatedness in section 3.3, and highlight some equilibrium properties of our dynamical system in section 3.4. In section 3.5, we apply our framework to study the coevolution of two traits that have socially synergistic effects between individuals. Finally, we discuss the implications of our results for understanding patterns of intra-specific variation, with special reference to social traits.

2 Model

We consider a population of haploid individuals, divided among an infinite number of groups, each of fixed size N (the total population size is therefore constant). Each individual bears a multidimensional phenotype of n quantitative traits that are genetically determined. The discrete-time life cycle of this population is as follows. (1) Adults reproduce clonally (producing offspring in sufficient number for the size of each group in the population to remain constant) then either survive or die, which frees up breeding spots. (2) The phenotype

of each individual independently mutates with probability ν, causing a random quantitative deviation in trait values. (3) Each offspring either remains in its natal group, or disperses to another randomly chosen group (i.e., we consider the island model of dispersal, Wright, 1931, Rousset, 2004). (4) Offspring compete locally in each group to fill open breeding spots, if any.

3 Results

3.1 Dynamics of the phenotypic distribution

In order to track phenotypic evolution in our population, we denote by $p_t(\mathbf{z})$ the phenotypic density distribution in the population at a demographic time point t, where $\mathbf{z} = (z_1, z_2, \ldots, z_n) \in \mathbb{R}^n$ is a vector collecting the variable z_a for each trait $a = 1, \ldots, n$. To capture the group structure of our population, we introduce the continuous distribution ϕ_t, which gives the density distribution of groups in the population with a certain composition of phenotypes at time t (i.e., $\phi_t(\mathbf{z}_1, \mathbf{z}_2, \ldots, \mathbf{z}_N)$ is the density distribution of groups in which individuals arbitrarily labelled 1 to N have phenotypes $\mathbf{z}_1, \mathbf{z}_2, \ldots, \mathbf{z}_N$, respectively).

In Appendix A, we show that the recurrence of the phenotypic distribution in the population from time t to $t+1$ (after one iteration of the life cycle) can be expressed as

$$p_{t+1}(\mathbf{z}) = (1-\nu) W(\mathbf{z}, \phi_t) p_t(\mathbf{z}) + \nu \int_{\mathbb{R}^n} v(\mathbf{z}', \mathbf{z}) W(\mathbf{z}', \phi_t) p_t(\mathbf{z}') \mathrm{d}\mathbf{z}'. \tag{1}$$

The first summand represents changes in the distribution due to individuals that have not mutated (with probability $1-\nu$), and the second summand, changes due to those that have (with probability ν; and where $v(\mathbf{z}', \mathbf{z})$ denotes the probability density function for the event that an individual mutates from $\mathbf{z}'$ to $\mathbf{z}$ given that a mutation has occurred). The quantity $W(\mathbf{z}, \phi_t)$ in eq. (1), which depends on the way phenotypes are distributed across groups (i.e., on ϕ_t), is a measure of fitness at the level of the phenotype: when $W(\mathbf{z}, \phi_t) > 1$, the frequency of $\mathbf{z}$ in the population increases due to selection, and conversely decreases when $W(\mathbf{z}, \phi_t) < 1$.

To gain insights into the fitness measure $W(\mathbf{z}, \phi_t)$, note first that recurrence eq. (1) has the same form as the classical recurrence of the phenotypic distribution in well-mixed populations under the continuum-of-alleles model (e.g., Kimura, 1965b, eqs. 1-2; Fleming, 1979, eq. 2.4; Bürger, 1986, eq. 1; Taylor and Day, 1997, eq. 1; Champagnat et al., 2006, eq. 4.1). In a well-mixed population of constant size, the relevant fitness measure is the *individual* fitness of a focal individual with phenotype $\mathbf{z}$, i.e., its expected number of successful offspring over one iteration of the life-cycle. Because individual fitness in a well-mixed population only depends on the focal phenotype $\mathbf{z}$ and the phenotypic distribution $p_t(\mathbf{z})$, it can simply be written as $w(\mathbf{z}, p_t(\mathbf{z}))$ (to distinguish between fitness at the phenotypic and individual level, we will denote the former by an upper case W and the latter by a lower case w). So in a well-mixed population of constant size, fitness at the phenotypic and individual levels align (i.e., $W(\mathbf{z}, \phi_t) = w(\mathbf{z}, p_t(\mathbf{z}))$ in eq. 1).

Defining fitness in terms of expected number of successful offspring is standard in social evolution theory (e.g., Hamilton, 1964, Rousset, 2004), and takes its roots in population dynamics: when $w(\mathbf{z}, p_t(\mathbf{z})) > 1$, the number of individuals with phenotype $\mathbf{z}$ increases and conversely decreases when $w(\mathbf{z}, p_t(\mathbf{z})) < 1$ (e.g., eq. 2.2 of Nagylaki 1992). As such, it is sometimes referred to as "absolute" fitness. Many quantitative genetics models, by contrast, employ the notion of "relative" fitness to track changes in phenotypic frequencies. This can stem from two non-mutually exclusive modelling choices: (1) one in fact considers the effect of the phenotype on a vital rate, $f(\mathbf{z}, p_t(\mathbf{z}))$ (such as fecundity or offspring survival), that influences the number of offspring that enter competition before regulation, which requires normalisation by mean vital rate, $W(\mathbf{z}, \phi_t) = w(\mathbf{z}, p_t(\mathbf{z})) = f(\mathbf{z}, p_t(\mathbf{z}))/[\int f(\mathbf{z}, p_t(\mathbf{z})) p_t(\mathbf{z}) \mathrm{d}\mathbf{z}]$; (2) the population size fluctuates, in which case it is necessary to normalise by mean fitness, $W(\mathbf{z}, \phi_t) = w(\mathbf{z}, p_t(\mathbf{z}))/[\int w(\mathbf{z}, p_t(\mathbf{z})) p_t(\mathbf{z}) \mathrm{d}\mathbf{z}]$. In our model, be-

cause group size and therefore population size is constant, $W(\mathbf{z},\phi_t)$ in eq. (1) can be viewed as an absolute measure of fitness.

In contrast to a well-mixed population, fitness at the level of the phenotype (or genotype) W and the fitness of a focal individual w do not necessarily align in a group-structured population, (Hamilton, 1964). To see this, consider that due to group structure, the fitness of a focal individual will further depend on the way phenotypes are distributed across groups (so on ϕ_t), and specifically on the collection of phenotypes carried by the individuals that belong to the focal group; we denote this collection by μ (formally, μ is a counting measure in our analysis – see Appendix A.1.2 – but for the purpose of the main text, it can simply be thought of as the phenotypic composition of the focal group). The fitness of a focal individual with phenotype $\mathbf{z}$ in a group-structured population can thus be written as a function: $w_\mu(\mathbf{z},\phi_t)$. In terms of this individual fitness function, we find that the fitness at the level of the phenotype that is relevant for phenotypic dynamics, $W(\mathbf{z},\phi_t)$ in eq. (1), is

$$W(\mathbf{z},\phi_t) = \int w_\mu(\mathbf{z},\phi_t)\, q_t(\mu|\mathbf{z})\mathrm{d}\mu, \tag{2}$$

where the integral runs through every possible group state, μ, and $q_t(\mu|\mathbf{z})$ is the probability density function for the event that an individual randomly picked from the collection of all carriers of the $\mathbf{z}$ phenotype in the population at time t resides in a group in state μ (see eq. A-17 in Appendix A for derivation). According to eq. (2), $W(\mathbf{z},\phi_t)$ corresponds to the expected number of successful offspring of an individual with phenotype $\mathbf{z}$, with expectation taken over all group states μ in which this individual can reside at time t.

An alternative interpretation for $W(\mathbf{z},\phi_t)$ can be reached by noting that because there is an infinite number of possible alleles, all individuals with the same phenotype $\mathbf{z}$ belong to the same genetic lineage. The function $q_t(\mu|\mathbf{z})$ in eq. (2) then corresponds to the probability that an individual sampled from this lineage at time t resides in a group in state μ. As such, $W(\mathbf{z},\phi_t)$ can be interpreted as the expected fitness of an individual randomly sampled from the lineage of individuals carrying phenotype $\mathbf{z}$ at time t. Eq. (1) then has an intuitive interpretation: if on average individuals from the $\mathbf{z}$-lineage produce more than one successful offspring at time t, this lineage will be larger at time $t+1$ and in a population of constant size, the frequency of individuals with phenotype $\mathbf{z}$ will increase. The fitness measure $W(\mathbf{z},\phi_t)$ can thus be seen as the multi-allelic version of the concept of a mutant's *lineage fitness* used previously in invasion analyses (which turns out to be equal to the mutant's growth rate when the mutant is rare in an otherwise monomorphic population, Lehmann et al., 2015, Mullon et al., 2016, Lehmann et al., 2016; see also Wild, 2011 for similar branching processes approach to social evolution in group-structured populations). We will therefore refer to $W(\mathbf{z},\phi_t)$ as the lineage fitness of phenotype $\mathbf{z}$, keeping in mind that unlike in invasion analyses, $W(\mathbf{z},\phi_t)$ here applies for any frequency of $\mathbf{z}$ (rare or common) and for any population composition (monomorphic or polymorphic).

3.2 Tracking the dynamics of the phenotypic distribution

The dynamical equation for the phenotypic distribution eq. (1) has no straightforward solution, even when the population is well-mixed (Kimura, 1965b, Fleming, 1979, Lande, 1979, Bürger, 1986). Under limited dispersal, this problem is further complicated by the necessity of simultaneously tracking the dynamics of group composition ϕ_t. To proceed further in our analysis and track the dynamics of the phenotypic distribution, we therefore make some additional assumptions.

3.2.1 Weak selection, weak mutation, normal closure and quasi-equilibrium of local genetic associations

We first assume that selection is weak, in the sense that the phenotypic variance in the population is small (allowing for second-order approximation of fitness, see Appendix B.1.1 for details, and Iwasa et al., 1991 for a similar approach for quantitative genetics of traits under frequency-dependent selection in well-mixed pop-

ulations). Next, we assume that mutations are rare, so that we can ignore the joint effects of selection and mutation on the phenotypic distribution over one time period (see Appendix B.1.2).

Following previous authors (e.g., see Taylor and Day, 1997, Wakano and Iwasa, 2013, Débarre et al., 2014, Wakano and Lehmann, 2014, Débarre and Otto, 2016, for social traits), we further assume that the processes of selection and mutation are such that $p_t(\mathbf{z})$ is approximately multivariate normal (allowing for moment closure, see Appendix B.2.1). The assumption of normality is a strong one but it is noteworthy that it does not require that the realised distribution of phenotypes within a focal group at any given demographic time period is normal. In addition, the assumption of normality has been shown to give remarkably accurate predictions for the change of mean and variance, which is our main goal, even when selection generates significant deviations from normality (in well-mixed populations, Turelli and Barton, 1994). Under the assumption of normality, the distribution $p_t(\mathbf{z})$ is characterised by its mean vector $\bar{\mathbf{z}}_t = (\bar{z}_{1,t}, \bar{z}_{2,t}, \ldots, \bar{z}_{n,t})$, whose a-entry is the average value of trait a in the population at time period t, $\bar{z}_{a,t} = \int_{\mathbb{R}^n} z_a p_t(\mathbf{z}) \mathrm{d}\mathbf{z}$; and its variance-covariance matrix $\boldsymbol{G}_t$ whose (a,b)-entry is the (co)variance among traits a and b in the population at time period t, $\sigma_{ab,t} = \int_{\mathbb{R}^n} (z_a - \bar{z}_{a,t})(z_b - \bar{z}_{b,t}) p_t(\mathbf{z}) \mathrm{d}\mathbf{z}$. The dynamics of $p_t(\mathbf{z})$ can therefore be tracked through the dynamics of its mean vector $\bar{\mathbf{z}}_t$ and variance-covariance matrix $\boldsymbol{G}_t$.

But due to limited dispersal, the dynamics of $\bar{\mathbf{z}}_t$ and $\boldsymbol{G}_t$ still depend on time-dependent local genetic associations among individuals of the same group, which capture moments of the distribution of group composition ϕ_t. To close evolutionary dynamics on $\bar{\mathbf{z}}_t$ and $\boldsymbol{G}_t$ and avoid tracking the dynamics of ϕ_t, we assume that selection is weak relative to dispersal so that local genetic associations reach their steady state before significant changes have occurred in the phenotypic distribution, $p_t(\mathbf{z})$ (see section B.2.2 for details). This "quasi-equilibrium" assumption, which is frequently used in population genetic theory (e.g., Kimura, 1965a, Nagylaki, 1993, Kirkpatrick et al., 2002, Roze and Rousset, 2005, Lehmann et al., 2007, Roze and Rousset, 2008), finally allows us to characterise the dynamics of $p_t(\mathbf{z})$ entirely by the coupled dynamics of its mean vector $\bar{\mathbf{z}}_t$ and variance-covariance matrix $\boldsymbol{G}_t$.

3.2.2 Dynamics of phenotypic mean vector and variance-covariance matrix

Under the above assumptions, we show in Appendix B that the coupled changes of the phenotypic mean trait vector and variance-covariance matrix over one demographic time period are respectively given by

$$\Delta\bar{\mathbf{z}}_t = \boldsymbol{G}_t \boldsymbol{s}(\bar{\mathbf{z}}_t) \tag{3a}$$

$$\Delta\boldsymbol{G}_t = \boldsymbol{M} + \boldsymbol{G}_t \left(\boldsymbol{H}(\bar{\mathbf{z}}_t) - \boldsymbol{s}(\bar{\mathbf{z}}_t) \boldsymbol{s}(\bar{\mathbf{z}}_t)^{\mathrm{T}} \right) \boldsymbol{G}_t, \tag{3b}$$

where $\boldsymbol{s}(\bar{\mathbf{z}}_t) = (s_1(\bar{\mathbf{z}}_t), \ldots, s_n(\bar{\mathbf{z}}_t))^{\mathrm{T}}$ is a $n \times 1$ is vector of directional selection coefficients (or selection gradients) i.e., $s_a(\bar{\mathbf{z}}_t)$ is the first-order, marginal, effect of an (infinitesimal) change in trait a away from the population mean $\bar{\mathbf{z}}_t$ on lineage fitness ($s_a(\bar{\mathbf{z}}_t) = \partial W(\mathbf{z}, \phi_t)/\partial z_a$). The $n \times n$ matrix $\boldsymbol{M}$ collects the effects of mutation; its (a,b)-entry,

$$(\boldsymbol{M})_{ab} = \nu \underbrace{\int_{\mathbb{R}^n} (z_a - z'_a)(z_b - z'_b) \upsilon(\mathbf{z}', \mathbf{z}) \mathrm{d}\mathbf{z}}_{\sigma^{\mathrm{m}}_{ab}}, \tag{4}$$

is the product of the mutation probability, ν, with the (co)variance, σ^{m}_{ab}, in mutational effects on traits a and b conditional on the appearance of a mutation (which captures the pleiotropic effects of mutations on a and b: when $\sigma^{\mathrm{m}}_{ab} > 0$, mutations tend to change a and b in a similar way; and when $\sigma^{\mathrm{m}}_{ab} < 0$, in opposite ways). The $n \times n$ Hessian matrix $\boldsymbol{H}(\bar{\mathbf{z}}_t)$ collects the second-order effects of traits on lineage fitness; its (a,b)-entry $\boldsymbol{H}(\bar{\mathbf{z}}_t)_{ab} = h_{ab}(\bar{\mathbf{z}}_t)$ is the marginal effect of joint changes in traits a and b away from the population mean $\bar{\mathbf{z}}_t$ on lineage fitness ($h_{ab}(\bar{\mathbf{z}}_t) = \partial^2 W(\mathbf{z}, \phi_t)/(\partial z_a \partial z_b)$). Finally, the notation $\boldsymbol{s}(\bar{\mathbf{z}}_t)\boldsymbol{s}(\bar{\mathbf{z}}_t)^{\mathrm{T}}$ denotes the outer product between two column vectors, so that $\boldsymbol{s}(\bar{\mathbf{z}}_t)\boldsymbol{s}(\bar{\mathbf{z}}_t)^{\mathrm{T}}$ is $n \times n$ matrix with (a,b)-entry $s_a(\bar{\mathbf{z}}_t) s_b(\bar{\mathbf{z}}_t)$.

3.2.3 Directional, disruptive, and correlational selection coefficients

Dynamical equations (3) have the same form as in well-mixed populations (e.g., eqs. 1-2 of Phillips and Arnold, 1989, see also eq. 7 of Lande, 1979 and eqs. 6 and 15 of Lande and Arnold, 1983). But in such models, the effects of selection depend on the marginal effects of traits on *individual* rather than *lineage* fitness. Nevertheless, the parallels between eq. (3) and previous works allow us to use the same vocabulary and interpretations on the evolution of phenotypic means and (co)variances (Brodie et al., 1995). First, the evolution of the mean of each trait (eq. 3a) depends on the vector of directional selection (or the selection gradient), $\boldsymbol{s}(\bar{\mathbf{z}}_t)$, which points in the direction favoured by selection in multivariate phenotypic space (Lande, 1979). The effect of directional selection on the mean of each trait, however, is constrained by the genetic variation available and these constraints are captured by $\boldsymbol{G}_t$ in eq. (3a) (Lande, 1979).

Second, the evolution of the variance-covariance matrix $\boldsymbol{G}_t$ (eq. 3b) depends on the effects of mutations ($\boldsymbol{M}$), of directional selection ($\boldsymbol{s}(\bar{\mathbf{z}}_t)\boldsymbol{s}(\bar{\mathbf{z}}_t)^{\mathrm{T}}$), and of quadratic selection given by the matrix $\boldsymbol{H}(\bar{\mathbf{z}}_t)$ (Lande, 1979, Lande and Arnold, 1983, Phillips and Arnold, 1989). This matrix $\boldsymbol{H}(\bar{\mathbf{z}}_t)$ captures two relevant features of selection. First, the sign of its diagonal entry (a,a) indicates whether selection favours a decrease (when $h_{aa}(\bar{\mathbf{z}}_t) < 0$) or an increase (when $h_{aa}(\bar{\mathbf{z}}_t) > 0$) in the variance of trait a when this trait evolves in isolation of other traits (Phillips and Arnold, 1989), hence $h_{aa}(\bar{\mathbf{z}}_t)$ is reffered to as the coefficient of disruptive selection on trait a. Second, the off-diagonal entry (a,b) tells us whether selection favours a positive (when $h_{ab}(\bar{\mathbf{z}}_t) > 0$) or negative (when $h_{ab}(\bar{\mathbf{z}}_t) < 0$) covariance or correlation among traits a and b. The off-diagonal entry $h_{ab}(\bar{\mathbf{z}}_t)$ is therefore referred to as the coefficient of correlational selection among a and b (Lande and Arnold, 1983, Phillips and Arnold, 1989).

3.3 Selection in terms of individual fitness effects and relatedness coefficients

So far, the effects of limited dispersal on evolutionary dynamics (eqs. 1 and 3) have been hidden behind the notion of lineage fitness, $W(\mathbf{z},\phi_t)$. To highlight more tangibly how selection depends on limited dispersal, we express the selection coefficients ($\boldsymbol{s}(\bar{\mathbf{z}}_t)$ and $\boldsymbol{H}(\bar{\mathbf{z}}_t)$) in terms of the effects of traits on individual fitness and relatedness. For this, let us first rewrite the individual fitness of a focal individual, that we label as individual "i", as a function $w(\mathbf{z}_i,\mathbf{z}_{-i},\bar{\mathbf{z}}_t)$ of three arguments: (1) the phenotype of the focal individual, $\mathbf{z}_i = (z_{i,1},z_{i,2},\ldots,z_{i,n})$; (2) the collection of phenotypes of its $N-1$ neighbours $\mathbf{z}_{-i} = (\mathbf{z}_1,\ldots\mathbf{z}_{i-1},\mathbf{z}_{i+1},\ldots,\mathbf{z}_N)$ (where $\mathbf{z}_j = (z_{j,1},z_{j,2},\ldots,z_{j,n})$ is the phenotype of a neighbour indexed j); and (3) the average phenotype in the population $\bar{\mathbf{z}}_t$ (see eq. 15 for an example of such a fitness function). This individual fitness function is equal to the fitness function $w_\mu(\mathbf{z},\phi_t)$ that appears in eq. (2),

$$w(\mathbf{z}_i,\mathbf{z}_{-i},\bar{\mathbf{z}}_t) = w_\mu(\mathbf{z},\phi_t), \tag{5}$$

when focal phenotype is $\mathbf{z}_i = \mathbf{z}$, the state of the focal group is $\{\mathbf{z}_i\}\cup\mathbf{z}_{-i} = (\mathbf{z}_1,\ldots,\mathbf{z}_N) = \mu$, and groups other than the focal one are considered to be monomorphic for the population average $\bar{\mathbf{z}}_t$ (i.e., we consider that all individuals in other groups express $\bar{\mathbf{z}}_t$ so that the distribution ϕ_t is delta peaked on $\bar{\mathbf{z}}_t$; we can do this because the phenotypic distribution is assumed to be centred around $\bar{\mathbf{z}}_t$ with small variance and individuals from different groups interact at random in the island model; see Iwasa et al., 1991 for a similar approach in panmictic populations). We further introduce relatedness coefficients that will be relevant for selection: let $r_2^\circ(\bar{\mathbf{z}}_t)$ and $r_3^\circ(\bar{\mathbf{z}}_t)$ respectively be the probabilities that, in the absence of selection and when the population phenotypic average is $\bar{\mathbf{z}}_t$, two and three neighbours are identical-by-descent (i.e., have a common ancestor that resided in the focal group, which is in line with the definition of relatedness in the infinite island model, Rousset, 2004; see e.g., Taylor et al., 2007 for further considerations on relatedness in the finite island model).

3.3.1 Directional selection

We find that the selection gradient on a trait a can be expressed as

$$s_a(\bar{\mathbf{z}}_t) = \left.\frac{\partial w(\mathbf{z}_i,\mathbf{z}_{-i},\bar{\mathbf{z}}_t)}{\partial z_{i,a}}\right|_{\substack{\mathbf{z}_i=\bar{\mathbf{z}}_t\\ \mathbf{z}_{-i}=\bar{\mathbf{z}}_t}} + (N-1)r_2^\circ(\bar{\mathbf{z}}_t)\left.\frac{\partial w(\mathbf{z}_i,\mathbf{z}_{-i},\bar{\mathbf{z}}_t)}{\partial z_{j,a}}\right|_{\substack{\mathbf{z}_i=\bar{\mathbf{z}}_t\\ \mathbf{z}_{-i}=\bar{\mathbf{z}}_t}}, \tag{6}$$

where $\mathbf{z}_{-i} = \bar{\mathbf{z}}_t$ means that the derivative is evaluated when all neighbours express the mean phenotype $\bar{\mathbf{z}}_t$ ($\mathbf{z}_j = \bar{\mathbf{z}}_t$ for all $j \neq i$). The first derivative in eq. (6) captures the direct effect of trait a: the effect of a change in trait a in a focal individual on its own fitness. In a well-mixed population, this is all that matters for directional selection (i.e., $s_a(\bar{\mathbf{z}}_t) = \partial w(\mathbf{z}_i,\mathbf{z}_{-i},\bar{\mathbf{z}}_t)/\partial z_{i,a}$ when the population size is constant, Phillips and Arnold, 1989 [1]. The second derivative, which is weighted by pairwise relatedness $r_2^\circ(\bar{\mathbf{z}}_t)$, is the indirect effect of trait a: the effect focal fitness of a change in trait a in a neighbour of the focal (we arbitrarily chose this neighbour to be individual $j \neq i$). The selection gradient on trait a, eq. (6), is therefore the inclusive fitness effect of trait a (Hamilton, 1964, Rousset, 2004). Hence, in the absence of covariance among traits, the change in mean trait value is proportional to this trait's inclusive fitness effect (substituting eq. 6 into 3a with the off-diagonal elements of $\boldsymbol{G}_t$ all zeros). This finding is in line with much previous modelling work on the quantitative genetics of spatially- or family-structured populations (for e.g., Cheverud, 1985, Queller, 1992a,b, Frank, 1998, McGlothlin et al., 2014, Wakano and Lehmann, 2014).

3.3.2 Correlational and disruptive selection

We find that the correlational selection coefficient on two traits a and b (or the disruptive selection coefficient when $a = b$) can be expressed as the sum of two terms,

$$h_{ab}(\bar{\mathbf{z}}_t) = h_{\mathrm{w},ab}(\bar{\mathbf{z}}_t) + h_{\mathrm{r},ab}(\bar{\mathbf{z}}_t), \tag{7a}$$

where the first term,

$$\begin{aligned} h_{\mathrm{w},ab}(\bar{\mathbf{z}}_t) = &\left.\frac{\partial^2 w(\mathbf{z}_i,\mathbf{z}_{-i},\bar{\mathbf{z}}_t)}{\partial z_{i,a}\partial z_{i,b}}\right|_{\substack{\mathbf{z}_i=\bar{\mathbf{z}}_t\\ \mathbf{z}_{-i}=\bar{\mathbf{z}}_t}} + (N-1)r_2^\circ(\bar{\mathbf{z}}_t)\left.\frac{\partial^2 w(\mathbf{z}_i,\mathbf{z}_{-i},\bar{\mathbf{z}}_t)}{\partial z_{j,a}\partial z_{j,b}}\right|_{\substack{\mathbf{z}_i=\bar{\mathbf{z}}_t\\ \mathbf{z}_{-i}=\bar{\mathbf{z}}_t}} \\ &+ (N-1)r_2^\circ(\bar{\mathbf{z}}_t)\left(\left.\frac{\partial^2 w(\mathbf{z}_i,\mathbf{z}_{-i},\bar{\mathbf{z}}_t)}{\partial z_{i,a}\partial z_{j,b}}\right|_{\substack{\mathbf{z}_i=\bar{\mathbf{z}}_t\\ \mathbf{z}_{-i}=\bar{\mathbf{z}}_t}} + \left.\frac{\partial^2 w(\mathbf{z}_i,\mathbf{z}_{-i},\bar{\mathbf{z}}_t)}{\partial z_{i,b}\partial z_{j,a}}\right|_{\substack{\mathbf{z}_i=\bar{\mathbf{z}}_t\\ \mathbf{z}_{-i}=\bar{\mathbf{z}}_t}}\right) \\ &+ (N-1)(N-2)r_3^\circ(\bar{\mathbf{z}}_t)\left.\frac{\partial^2 w(\mathbf{z}_i,\mathbf{z}_{-i},\bar{\mathbf{z}}_t)}{\partial z_{j,a}\partial z_{k,b}}\right|_{\substack{\mathbf{z}_i=\bar{\mathbf{z}}_t\\ \mathbf{z}_{-i}=\bar{\mathbf{z}}_t}}, \end{aligned} \tag{7b}$$

depends on the effects of joint changes in traits a and b within- (first line of eq. 7b) and between-individuals (second and third line of eq. 7b) on focal fitness. The first derivative on the first line of eq. (7b) is the effect of a joint change in traits a and b in a focal individual on its own fitness, which can be viewed as the *direct* synergistic effects of traits a and b (Figure 1a). In a well-mixed population, there are no other effects participating to correlational selection (i.e., $h_{ab}(\bar{\mathbf{z}}_t) = \partial^2 w(\mathbf{z}_i,\mathbf{z}_{-i},\bar{\mathbf{z}}_t)/(\partial z_{i,a}\partial z_{i,b})$, Phillips and Arnold, 1989).

But when dispersal is limited (so that $r_2^\circ(\bar{\mathbf{z}}_t) > 0$ and $r_3^\circ(\bar{\mathbf{z}}_t) > 0$), three types of *indirect* synergistic effects become relevant for correlational selection. These are the effect of a change in: (i) both traits in one neighbour of the focal (second derivative on the first line weighted by the neutral probability that the focal and this neigh-

[1] When the size of the population fluctuates, $s_a(\bar{\mathbf{z}}_t) = \partial \log w(\mathbf{z}_i,\mathbf{z}_{-i},\bar{\mathbf{z}}_t)/\partial z_{i,a}$, due to normalisation of focal fitness with respect to mean fitness (see section 3.1; and eq. A6 of Iwasa et al., 1991 for how this holds when selection is frequency-dependent). If the size of the population fluctuates but selection is frequency-independent, then the selection gradient can be expressed as the derivative of the log of mean fitness in the population with respect to the trait under scrutiny (e.g., eq. 7b of Lande, 1979)

bour are identical-by-descent, $r_2^\circ(\bar{\mathbf{z}}_t)$, Figure 1b); (ii) in one trait in the focal and in the other in a neighbour (the two derivatives of the second line weighted by $r_2^\circ(\bar{\mathbf{z}}_t)$, Figure 1c); and (iii) in one trait in a neighbour and in the other in another neighbour indexed as k (last derivative weighted by the neutral probability that the focal and these two neighbours are identical-by-descent, $r_3^\circ(\bar{\mathbf{z}}_t)$, Figure 1d).

The second term of eq. (7a), $h_{\mathrm{r},ab}(\bar{\mathbf{z}}_t)$, is yet another synergistic effect relevant for correlational selection in group-structured populations. This term can be expressed as

$$h_{\mathrm{r},ab}(\bar{\mathbf{z}}_t) = (N-1)\frac{\partial w(\mathbf{z}_i,\mathbf{z}_{-i},\bar{\mathbf{z}}_t)}{\partial z_{j,a}}\bigg|_{\substack{\mathbf{z}_i=\bar{\mathbf{z}}_t\\ \mathbf{z}_{-i}=\bar{\mathbf{z}}_t}} \times \frac{\partial r_2(\mathbf{z})}{\partial z_b}\bigg|_{\mathbf{z}=\bar{\mathbf{z}}_t} + (N-1)\frac{\partial w(\mathbf{z}_i,\mathbf{z}_{-i},\bar{\mathbf{z}}_t)}{\partial z_{j,b}}\bigg|_{\substack{\mathbf{z}_i=\bar{\mathbf{z}}_t\\ \mathbf{z}_{-i}=\bar{\mathbf{z}}_t}} \times \frac{\partial r_2(\mathbf{z})}{\partial z_a}\bigg|_{\mathbf{z}=\bar{\mathbf{z}}_t}, \tag{7c}$$

where $\partial r_2(\mathbf{z})/\partial z_a$ is the effect of trait a on the probability of that two neighbours are identical-by-descent, or pairwise relatedness (and $\partial r_2(\mathbf{z})/\partial z_b$ the effect of trait b). So eq. (7c) reveals that correlational selection depends on the product between the indirect effect of one trait ($\partial w(\mathbf{z}_i,\mathbf{z}_{-i},\bar{\mathbf{z}}_t)/\partial z_{j,a}$ and $\partial w(\mathbf{z}_i,\mathbf{z}_{-i},\bar{\mathbf{z}}_t)/\partial z_{j,b}$), with the effect of the other trait on relatedness. Such synergy via relatedness (Figure 1e) reflects that in group structured populations, selection will favour a correlation among two traits when such a correlation results in indirect fitness benefits (e.g., trait a is cooperative, $\partial w(\mathbf{z}_i,\mathbf{z}_{-i},\bar{\mathbf{z}}_t)/\partial z_{j,a} > 0$) being preferentially directed towards relatives (e.g., trait b is the tendency to stay in natal group, $\partial r_2(\mathbf{z})/\partial z_b > 0$).

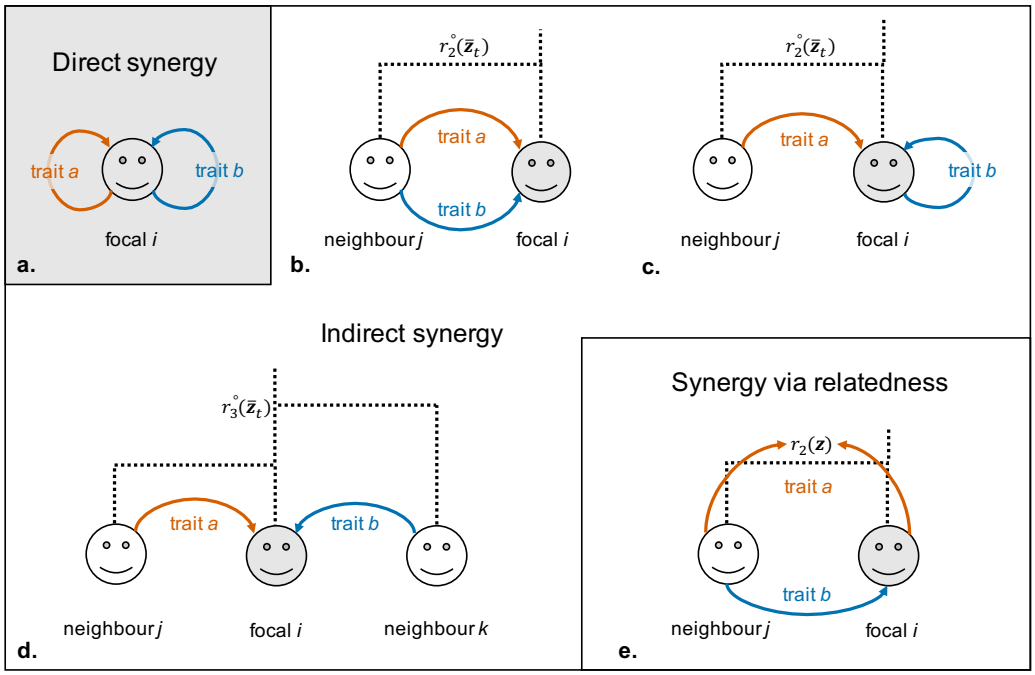

Figure 1: Within- and between-individual fitness effects relevant for correlational selection. As revealed by eq. (7), there are five types of fitness effects due to perturbations in two traits a and b that are relevant for correlational selection when dispersal is limited: **a.** effect of a joint changes in a and b within the focal individual (first term of eq. 7b); **b.** effect of joint changes in a and b within neighbours of the focal (second term of eq. 7b, weighted by neutral pairwise relatedness, $r_2^\circ(\bar{\mathbf{z}}_t)$); **c.** effect of joint changes in a and b between the focal (here b) and its neighbours (here, a; second line of eq. 7b, weighted by $r_2^\circ(\bar{\mathbf{z}}_t)$); **d.** effect of joint changes in a and b between neighbours of the focal (third line of eq. 7b, weighted by neutral three-way relatedness, $r_3^\circ(\bar{\mathbf{z}}_t)$); **e.** the effect of the indirect effect of one trait (here b) combined with the effect of the other (here a) on pairwise relatedness, which reflects the tendency of relatives to receive the effects of b (eq. 7c).

Group-structure and limited dispersal may thus lead to significant changes to the way selection moulds phenotypic correlations, especially when traits have synergistic effects that are either indirect (Figure 1b-d) or via relatedness (Figure 1e). This will be illustrated later when we study the coevolution of two social traits in sec-

tion 3.5. Before doing so, let us remark that when a single traits evolves ($n = 1$) and the selection gradient on this trait is zero ($s_a(\bar{\mathbf{z}}_t) = 0$), the change in phenotypic variance that we obtain (eq. 7 substituted into eq. 3b) reduces to previously derived expressions from quantitative genetics in the island model (eqs. 26 and 31 of Wakano and Lehmann, 2014). Further, eqs. (6)-(7) are consistent with evolutionary invasion analyses, i.e., with the first- and second-order effects of selection on the growth rate of a rare mutant that arises in a monomorphic group-structured population and that differs from the resident in a single (eqs. 8 & 9 of Ajar, 2003) or multiple (eqs. 12 & 13 of Mullon et al., 2016) traits. We discuss further the correspondence between quantitative genetics, invasion analyses, and adaptive dynamics models in the next section, in which we study the equilibrium properties of the phenotypic distribution.

3.4 Equilibrium properties of the phenotypic distribution

Eq. (3) with eqs. (6)-(7) is a closed dynamical system that allows to track the evolution of the mean trait value and of the (co)variance between traits. In this section, we first investigate key features of the equilibrium of these phenotypic dynamics, and then discuss their connections with notions of evolutionary stability that come from invasion analyses and adaptive dynamics.

3.4.1 Equilibrium mean trait values

We denote the mean trait vector and variance-covariance matrix of the equilibrium phenotypic distribution by $\bar{\mathbf{z}}^*$ and $\boldsymbol{G}^*$, respectively. Such equilibrium simultaneously satisfies $\Delta\bar{\mathbf{z}}_t = \mathbf{0}$ and $\Delta\boldsymbol{G}_t = \mathbf{0}$ (where $\mathbf{0}$ is used to denote a n vector and $n \times n$ matrix whose entries are all zero, respectively). Rather than solving both systems of equations simultaneously, we can use the fact that in eq. (3a), the matrix $\boldsymbol{G}$ is a positive-definite matrix with real-entries (since it is a variance-covariance matrix). From standard linear algebra theory (Hines, 1980, Leimar, 2005, 2009), it then follows that the equilibrium for the phenotypic means must satisfy

$$\boldsymbol{s}(\bar{\mathbf{z}}^*) = \mathbf{0}, \tag{8}$$

i.e., all selection gradients (eq. 6) vanish at $\bar{\mathbf{z}}^*$, independently of the $\boldsymbol{G}$ matrix.

We can further ask whether a population with a mean vector that is close to an equilibrium $\bar{\mathbf{z}}^*$ will eventually converge to it as a result of selection and mutation. From the fact that $\boldsymbol{G}$ is positive-definite, it can be shown (see Leimar, 2009, for e.g.) that a necessary condition for a population to converge to $\bar{\mathbf{z}}^*$ for all possible $\boldsymbol{G}$ matrices is that the Jacobian matrix $\boldsymbol{J}(\bar{\mathbf{z}}^*)$ of the selection gradients with (a, b) entry

$$\boldsymbol{J}(\bar{\mathbf{z}}^*)_{ab} = \left.\frac{\partial s_a(\bar{\mathbf{z}})}{\partial z_b}\right|_{\bar{\mathbf{z}}=\bar{\mathbf{z}}^*} \tag{9}$$

is negative-definite at $\bar{\mathbf{z}}^*$, which means that the symmetric real part of $\boldsymbol{J}(\bar{\mathbf{z}}^*)$, $(\boldsymbol{J}(\bar{\mathbf{z}}^*) + \boldsymbol{J}(\bar{\mathbf{z}}^*)^{\mathrm{T}})/2$ has only negative eigenvalues. This type of equilibrium is referred to as (strongly) convergence stable (Leimar, 2005, 2009).

3.4.2 Equilibrium variance-covariance matrix

The dynamics of the variance-covariance matrix can then be studied at a convergence stable equilibrium $\bar{\mathbf{z}}^*$ for mean trait values (eq. 8). In this case, the equilibrium $\boldsymbol{G}^*$ for the variance-covariance matrix solves

$$\boldsymbol{M} + \boldsymbol{G}^* \boldsymbol{H}(\bar{\mathbf{z}}^*)\boldsymbol{G}^* = \mathbf{0}. \tag{10}$$

Eq. (10) has an admissible solution (i.e., such that $\boldsymbol{G}^*$ is positive-definite) if, and only if, the Hessian matrix, $\boldsymbol{H}(\bar{\mathbf{z}}^*)$, is negative-definite (Bhatia, 2015). This corresponds to the case under which selection is stabilising at $\bar{\mathbf{z}}^*$. In fact, if $\boldsymbol{H}(\bar{\mathbf{z}}^*)$ is negative-definite, then the population will remain unimodally distributed around the mean vector $\bar{\mathbf{z}}^*$ and exhibit a variance-covariance matrix,

$$\boldsymbol{G}^* = \boldsymbol{M}\left[\boldsymbol{M}^{-1}\left(-\boldsymbol{H}(\bar{\mathbf{z}}^*)\right)^{-1}\right]^{1/2}, \tag{11}$$

where the operation $\mathbf{X}^{1/2}$ denotes the square root of $\mathbf{X}$ such that all the eigenvalues of $\mathbf{X}^{1/2}$ are positive (Bhatia, 2015; see also eq. 21c of Lande, 1980).

3.4.3 Connections with notions of stability from invasion analyses

Using a quantitative genetics approach, we have derived the conditions under which the multivariate phenotypic distribution of a dispersal limited population converges and remains at an equilibrium. Here, we highlight the connections between these conditions and notions of evolutionary stability that have emerged from invasion analyses and adaptive dynamics under limited dispersal.

Singular strategy. First, the selection gradient eq. (6) substituted into condition (8) is equivalent to the definition of evolutionarily singular strategies/phenotypes under limited dispersal (i.e., phenotypes which when expressed by the whole population, the gradient of invasion fitness is zero, e.g., Rousset, 2004; see also Geritz et al., 1998, for general definition).

Convergence stability. Second, the condition for a mean trait vector to be an attractor of directional selection (condition 9 with eq. 6) is equivalent to the condition for a multi-trait phenotype to be convergence stable in invasion analysis (Mullon et al., 2016; see also Brown and Taylor, 2010, for a graphical approach to the coevolution of two traits in a genetically structured population; and Leimar, 2009, Geritz et al., 2016, for general considerations on multi-trait in invasion analysis). It is noteworthy that in spite of this equivalence, the phenotypic dynamics envisaged by a quantitative genetics model (given by eq. 3a, see also eq. 7 of Lande, 1979, or eq. 1 of Phillips and Arnold, 1989) differ from the dynamics inferred from invasion analysis (which are captured by the so-called “canonical equation”, eq. 1 of Dieckmann and Law, 1996, or eq. 3 of Leimar, 2009). In a quantitative genetics model, the mean trait vector changes as a result of selection acting on a standing genetic variation, which is large enough to be captured by a statistical distribution ($\boldsymbol{G}_t$ in eq. 3a). Under the “canonical equation”, traits evolve under a trait substitution sequence, whereby selected mutants fix before other mutants arise, so that the population "jumps" from one monomorphic state to another and in principle cannot sustain polymorphism (see Fig. 1c, upper right panel of Champagnat et al., 2006, for a useful depiction of a trait substitution sequence; see Van Cleve, 2015, for a review of trait substitution sequence under limited dispersal).

Uninvadability. Third, the condition that $\boldsymbol{H}(\bar{\mathbf{z}}^*)$ with eq. (7) is negative-definite for the population to remain unimodally distributed around $\bar{\mathbf{z}}^*$ is consistent with the condition derived from invasion analyses for $\bar{\mathbf{z}}^*$ to be locally uninvadable (i.e., that any rare mutant that arises in a population for monomorphic for $\bar{\mathbf{z}}^*$ and that causes a slight deviation from $\bar{\mathbf{z}}^*$ eventually vanishes, Mullon et al., 2016; see also Ajar, 2003 for a single evolving trait in dispersal limited population; and Leimar, 2009, Geritz et al., 2016, for general considerations on multi-trait analyses).

Evolutionary branching. Invasion analyses have revealed that a phenotype that is convergence stable is not necessarily uninvadable (Eshel and Motro, 1981, Eshel, 1983, Taylor, 1989, Christiansen, 1991, Abrams et al., 1993b). In fact, when a singular phenotype is convergence stable but invadable, disruptive selection can lead

to evolutionary branching, whereby two lineages stably coexist in polymorphism (Metz et al., 1996, Geritz et al., 1998). When multiple traits are evolving, a sufficient condition for the initiation of evolutionary branching is that the Jacobian is negative-definite and the Hessian matrix is positive-definite at the singular phenotype $\bar{\mathbf{z}}^*$ (note that this does not ensure that the resulting polymorphism is stable, Geritz et al., 2016, for further considerations). In the context of quantitative genetics, this means that the mean trait vector is held at $\bar{\mathbf{z}}^*$ (as $\boldsymbol{J}(\bar{\mathbf{z}}^*)$ is negative-definite) while the dynamics of the variance-covariance matrix (eq. 3b) diverges to infinity (as $\boldsymbol{H}(\bar{\mathbf{z}}^*)$ is positive-definite). In other words, at the onset of evolutionary branching, directional selection maintains the population mean vector at $\bar{\mathbf{z}}^*$ all the while disruptive selection favours extreme phenotypes, leading to the explosion of the variance-covariance matrix (in line with previous quantitative genetics approaches to study evolutionary branching, Wakano and Iwasa, 2013, Débarre et al., 2014, Wakano and Lehmann, 2014, Débarre and Otto, 2016).

3.4.4 The moulding of phenotypic correlations by selection and mutation

Invasion analyses can be used to infer on the phenotypic correlations or associations generated by disruptive selection (by studying at the eigenvector associated with the greatest eigenvalue of $\boldsymbol{H}(\bar{\mathbf{z}}^*)$, which gives the axis in phenotypic space on which selection is disruptive and along which the population becomes dimorphic, Mullon et al., 2016, Geritz et al., 2016). This approach, however, only incorporates the effect of selection and is limited to studying phenotypic correlations at the onset of evolutionary branching (inferring on the long term outcome of evolutionary branching requires studying invasion in dimorphic populations, which is typically much more involved mathematically, e.g., Geritz et al., 1998, Sasaki and Dieckmann, 2011). A quantitative genetics approach such as ours here allows two further considerations on phenotypic correlations (e.g., Lande, 1980, Jones et al., 2007). First, it allows to incorporate the influence of pleiotropy (through the distribution of mutational input, captured by the variance-covariance matrix $\boldsymbol{M}$ in eq. 3b). Second, eq. (11) allows to study equilibrium phenotypic correlations as a balance between mutation and stabilising selection (and not only disruptive selection). We investigate this balance in more detail in the next section, in which we apply our quantitative approach to model the evolution of traits with socially synergistic effects.

3.5 Application to the coevolution of two synergistic social traits

We now apply the quantitive genetics approach elaborated above to study the coevolution of two social traits under limited dispersal. Our main goal is to illustrate the potential significance of indirect synergistic effects for the moulding of phenotypic correlations when dispersal is limited (Figure 1b-d).

3.5.1 Two public goods model

We model the coevolution of two nonnegative quantitative traits, labelled 1 and 2, that each capture participation to a different public good. For examples, in group living mammals, one trait could be the time/energy invested into foraging for the group's offspring, and the other, investment into defending the group by standing sentry against predators (e.g., Carter et al., 2014); in microorganisms, each trait could be the production of a specific amino-acid that is released into the external environment from which it can then be absorbed and used by group members (e.g., D'Souza and Kost, 2016).

Benefits and costs. We assume that both public goods are shared equally among group members, and that individuals receive extra benefits from obtaining both goods together. The benefits, $B(\mathbf{z}_i, \mathbf{z}_{-i})$, received by a focal individual (with traits $\mathbf{z}_i$ in a group composed of $\mathbf{z}_{-i}$) can then be written in terms of the group trait

averages ($\hat{z}_1 = \sum_{j=1}^{N} z_{j,1}/N$ and $\hat{z}_2 = \sum_{j=1}^{N} z_{j,2}/N$) as

$$B(\mathbf{z}_i, \mathbf{z}_{-i}) = b(\hat{z}_1 + \hat{z}_2) + b_{\mathrm{M}} \hat{z}_1 \hat{z}_2, \tag{12}$$

where the parameter $b > 0$ tunes the independent benefit of each public good produced (assumed to be the same for both goods for simplicity); and parameter $b_{\mathrm{M}} > 0$, the multiplicative benefits of receiving both goods together. Conversely, participation to both public goods simultaneously is assumed to be extra costly, for instance because the different goods call upon different biological functions that are costly to co-maintain, so that the cost $C(\mathbf{z}_i)$ paid by a focal individual (with traits $\mathbf{z}_i$) can be written as

$$C(\mathbf{z}_i) = \frac{c}{2}(z_{i,1}^2 + z_{i,2}^2) + c_{\mathrm{M}} z_{i,1} z_{i,2}, \tag{13}$$

where the parameter $c > 0$ tunes the independent cost of each trait, and parameter $c_{\mathrm{M}} > 0$, the multiplicative costs of the traits. The fecundity of a focal individual, $f(\mathbf{z}_i, \mathbf{z}_{-i})$, is then the difference between the benefits received and the costs paid,

$$f(\mathbf{z}_i, \mathbf{z}_{-i}) = 1 + B(\mathbf{z}_i, \mathbf{z}_{-i}) - C(\mathbf{z}_i), \tag{14}$$

where 1 is the baseline fecundity when no one in the group participates to either public good ($z_{i,1} = z_{i,2} = 0$ for all i).

These benefits (eq. 12) and costs (eq. 13) entail that it is best for a focal individual to express a negative *within*-individual association between traits (if expressed at all), and simultaneously be in a group in which traits are positively associated *between*-individuals. Such a configuration is possible when the population is well-mixed (so that there are no genetic correlations – or no relatedness – among individuals of the same group), but difficult when individuals of the same group are related due to limited dispersal. As relatedness increases, associations within- and between-individuals become aligned due to the co-inheritance of linked traits (in fact, the covariance between-traits between-individuals is equal to the product of pairwise relatedness with total covariance in the absence of selection; i.e., the between-individuals covariance of traits a and b is equal to $r_2^{\circ}(\bar{\mathbf{z}}_t)\sigma_{ab,t}$, see eq. SI-23 in Supplementary Information). We therefore expect limited dispersal to be relevant to the coevolution of the two traits of our model and to the way selection associates these traits within individuals.

Fitness. Before proceeding to the analysis, let us give the individual fitness function of a focal individual $w(\mathbf{z}_i, \mathbf{z}_{-i}, \bar{\mathbf{z}}_t)$. For this model, we assume that all adults die after reproduction (so that the population follows a Wright-Fisher life cycle). In this case, individual fitness is,

$$w(\mathbf{z}_i, \mathbf{z}_{-i}, \bar{\mathbf{z}}_t) = \underbrace{\frac{(1-m) f(\mathbf{z}_i, \mathbf{z}_{-i})}{(1-m)\sum_{i=1}^{N} f(\mathbf{z}_i, \mathbf{z}_{-i})/N + m f(\bar{\mathbf{z}}_t, \bar{\mathbf{z}}_t)}}_{w^{\mathrm{P}}(\mathbf{z}_i, \mathbf{z}_{-i}, \bar{\mathbf{z}}_t)} + \underbrace{\frac{m f(\mathbf{z}_i, \mathbf{z}_{-i})}{f(\bar{\mathbf{z}}_t, \bar{\mathbf{z}}_t)}}_{w^{\mathrm{D}}(\mathbf{z}_i, \mathbf{z}_{-i}, \bar{\mathbf{z}}_t)}, \tag{15}$$

where $0 < m \leq 1$ is the dispersal probability. Individual fitness is the addition of two terms: (1) the expected number of offspring of the focal that successfully establish in their natal group, $w^{\mathrm{P}}(\mathbf{z}_i, \mathbf{z}_{-i}, \bar{\mathbf{z}}_t)$, which is the ratio of the number of philopatric offspring of the focal to the total number of offspring that enter the competition in the focal group; and (2) the expected number of offspring of the focal that successfully settle in other groups, $w^{\mathrm{D}}(\mathbf{z}_i, \mathbf{z}_{-i}, \bar{\mathbf{z}}_t)$, which is the ratio of offspring the focal sends in a non-focal group to the expected number of offspring in such a group (fitness function of the form eq. 15 is standard under limited dispersal, e.g., Rousset, 2004, Ohtsuki, 2010).

Relatedness. The final pieces of information that are necessary to apply our framework are the neutral relatedness coefficients, $r_2^{\circ}(\bar{\mathbf{z}}_t)$ and $r_3^{\circ}(\bar{\mathbf{z}}_t)$, and the effect of each trait on pairwise relatedness ($\partial r_2(\mathbf{z})/\partial z_a$). These expressions, which have been derived elsewhere for the Wright-Fisher life-cycle considered here (e.g., Rousset,

2004, Ajar, 2003, Ohtsuki, 2010, Wakano and Lehmann, 2014), are given in Appendix B.2.2 (eqs. B-21-B-22).

3.5.2 Analysis

We now proceed to analyse the evolution of both social traits using the approach established in section 3.2. We first focus on the equilibrium properties of the phenotypic distribution.

Convergence of mean trait values. Substituting eq. (15) and pairwise relatedness coefficient (eq. B-21) into eq. (6), we obtain that the selection gradient vector is

$$\boldsymbol{s}(\bar{\mathbf{z}}_t) = [1 - r_2^\circ(\bar{\mathbf{z}}_t)] \begin{pmatrix} b/N - c\bar{z}_{1,t} + \bar{z}_{2,t}(-c_\mathrm{M} + b_\mathrm{M}/N) \\ b/N - c\bar{z}_{2,t} + \bar{z}_{1,t}(-c_\mathrm{M} + b_\mathrm{M}/N) \end{pmatrix} + \mathcal{O}(\epsilon^2), \tag{16}$$

where ϵ is a parameter capturing the magnitude of the effect of the public good on fecundity (i.e., ϵ is of the order of b, b_M, c, c_M). Solving eq. (16) for zero then yields the unique singular strategy

$$\bar{\mathbf{z}}^* = (\bar{z}_1^*, \bar{z}_2^*) = \left(\frac{b/N}{c + c_\mathrm{M} - b_\mathrm{M}/N}, \frac{b/N}{c + c_\mathrm{M} - b_\mathrm{M}/N}\right), \tag{17}$$

which unsurprisingly decreases with costs c and c_M, and increases with "direct" benefits b/N and b_M/N (as an individual recoups a share $1/N$ of its participation to each public good). Note that this singular strategy does not depend on dispersal (or relatedness). This is due our assumptions that group size is fixed and that generations are non-overlapping (in which case indirect fitness benefits of interacting with relatives are "cancelled" by the fitness costs of kin competition, e.g., Taylor, 1992).

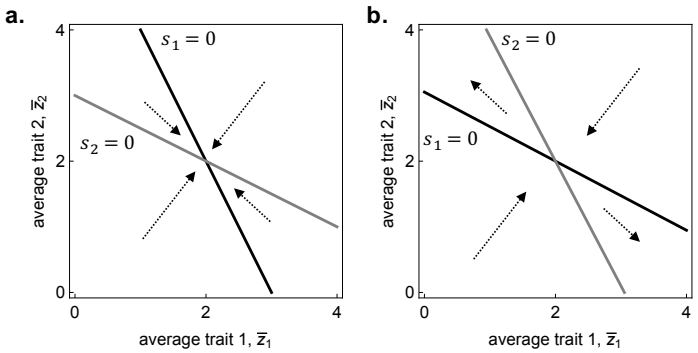

Figure 2: Directional selection on synergistic social traits. Qualitative dynamics of population means due to selection when equilibrium eq. (17) is: **a.** an attractor (with $b/N = 3$, $b_\mathrm{M}/N = 1.5$); **b.** a repeller (with $b/N = 5.8$, $b_\mathrm{M}/N = 0.1$). Solid lines show when the selection gradient eq. (16) on each trait vanishes (black for trait 1, $s_1(\bar{z}_1, \bar{z}_2) = 0$; grey for trait 2, $s_2(\bar{z}_1, \bar{z}_2) = 0$; other parameters: $c = 1$, $c_\mathrm{M} = 2$).

To establish whether the phenotypic distribution will converge to have mean $\bar{\mathbf{z}}^*$, we substitute eq. (16) into the symmetric part of the Jacobian matrix eq. (9), which we evaluate at the equilibrium eq. (17). It is straightforward to show that the two eigenvalues of the resulting matrix are given by

$$[1 - r_2^\circ(\bar{\mathbf{z}}_t)]\left\{-c - c_\mathrm{M} + b_\mathrm{M}/N, -c + c_\mathrm{M} - b_\mathrm{M}/N\right\} + \mathcal{O}(\epsilon^2). \tag{18}$$

Both are negative provided

$$-1 < \frac{-c_\mathrm{M} + b_\mathrm{M}/N}{c} < 1, \tag{19}$$

i.e., when the difference between the multiplicative costs, c_M, and direct multiplicative benefits, b_M/N, is small compared to the independent cost, c. In that case, the population will evolve to have mean given by eq. (17)

and produce an equal amount of each public good (Figure 2a). Otherwise, the population will evolve to express a single trait and thus produce a single public good (depending on initial conditions, Figure 2b). Eqs. (17) and (19) reveal that limited dispersal does not influence the evolution of the mean of the phenotypic distribution. But what about the shape of the distribution around this mean?

Stabilisation of the distribution around the mean. Assuming eq. (19) holds true, whether or not the population distribution stabilises around the equilibrium trait values (eq. 17) depends on the Hessian matrix, $\boldsymbol{H}(\bar{\mathbf{z}}^*)$. Let us start with analysing the diagonal elements of $\boldsymbol{H}(\bar{\mathbf{z}}^*)$, which reveal whether selection on each trait is independently stabilising or disruptive. Substituting eq. (15) and relatedness coefficients (Appendix B.2.2) into eq. (7) for traits 1 and 2 (i.e., $a=b=1$ and $a=b=2$), and evaluating it at equilibrium eq. (17), we obtain that the diagonal entries of $\boldsymbol{H}(\bar{\mathbf{z}}^*)$ are

$$h_{11}(\bar{\mathbf{z}}^*) = h_{22}(\bar{\mathbf{z}}^*) = -\left[1 - r_2^{\circ}(\bar{\mathbf{z}}_t)\right]c + \mathcal{O}(\epsilon^2). \tag{20}$$

Since $0 \leq r_2^{\circ}(\bar{\mathbf{z}}_t) < 1$, the diagonal entries of $\boldsymbol{H}(\bar{\mathbf{z}}^*)$ are always negative, which means that selection on each trait is stabilising when they evolve independently from one another.

Whether selection is stabilising when both traits co-evolve also depends on the correlational coefficient of selection, $h_{12}(\bar{\mathbf{z}}^*)$. In particular, stabilising selection requires that: (1) $h_{11}(\bar{\mathbf{z}}^*) < 0$ and $h_{22}(\bar{\mathbf{z}}^*) < 0$; and (2) $h_{12}(\bar{\mathbf{z}}^*)^2 < h_{11}(\bar{\mathbf{z}}^*)h_{22}(\bar{\mathbf{z}}^*)$, i.e., that the correlational selection coefficient is weak relative to the strength of stabilising selection on both independent traits; this is because a 2×2 symmetric matrix Hessian matrix is negative-definite if and only if its diagonal entries are both negative and the off-diagonal satisfies condition (2) (e.g., Horn and Johnson, 2012). Condition (2) can equivalently be written as

$$-1 < \rho_s^* = \frac{h_{12}(\bar{\mathbf{z}}^*)}{\sqrt{h_{11}(\bar{\mathbf{z}}^*)h_{22}(\bar{\mathbf{z}}^*)}} < 1, \tag{21}$$

where ρ_s^* is the strength of correlational selection, relative to the strength of stabilising selection on each independent trait at $\bar{\mathbf{z}}^*$. If eq. (21) does not hold, then selection is disruptive due to correlational selection.

The correlational coefficient of selection is derived by first substituting eq. (15) into eq. (7) with $a=1$ and $b=2$, and second, evaluating the result at equilibrium eq. (17). This yields

$$h_{12}(\bar{\mathbf{z}}^*) = \left[1 - r_2^{\circ}(\bar{\mathbf{z}}_t)\right]\left[-c_M + \left(1/N + \alpha(N-1)/N\right)b_M/N\right] + \mathcal{O}(\epsilon^2), \tag{22}$$

where α is a function that decreases as dispersal and group size increases (i.e., α decreases as relatedness coefficients decrease, see Figure 3a). Eq. (22) reveals that as α (and relatedness) increases, the within-individual association favoured by selection goes from negative (Figure 3b-c, grey region) to positive (Figure 3b-c, black region). This is because as relatedness increases, indirect synergistic effects become increasingly targeted by correlational selection (Figure 1b-d).

Substituting eqs. (20) and (22) into eq. (21), we find that selection is stabilising around $\bar{\mathbf{z}}^*$ when

$$-1 < \rho_s^* = \frac{-c_M + \left(1/N + \alpha(N-1)/N\right)b_M/N}{c} < 1, \tag{23}$$

which reveals that high relatedness, or large α, favours stabilising selection (Figure 3b-c, dark grey and black regions), and conversely, low relatedness, or small α, favours disruptive selection and thus polymorphism (when eq. 19 holds but eq. 23 does not, Figure 3b-c, light grey region). This finding is in line with a recent computational eco-evolutionary model which found that when species can evolve cross-feeding interactions, mutualistic coexistence is compromised by spatial structure and limited dispersal (Oliveira et al., 2014). This is also in line with previous results on the evolution of single traits that have found that evolutionary branching is

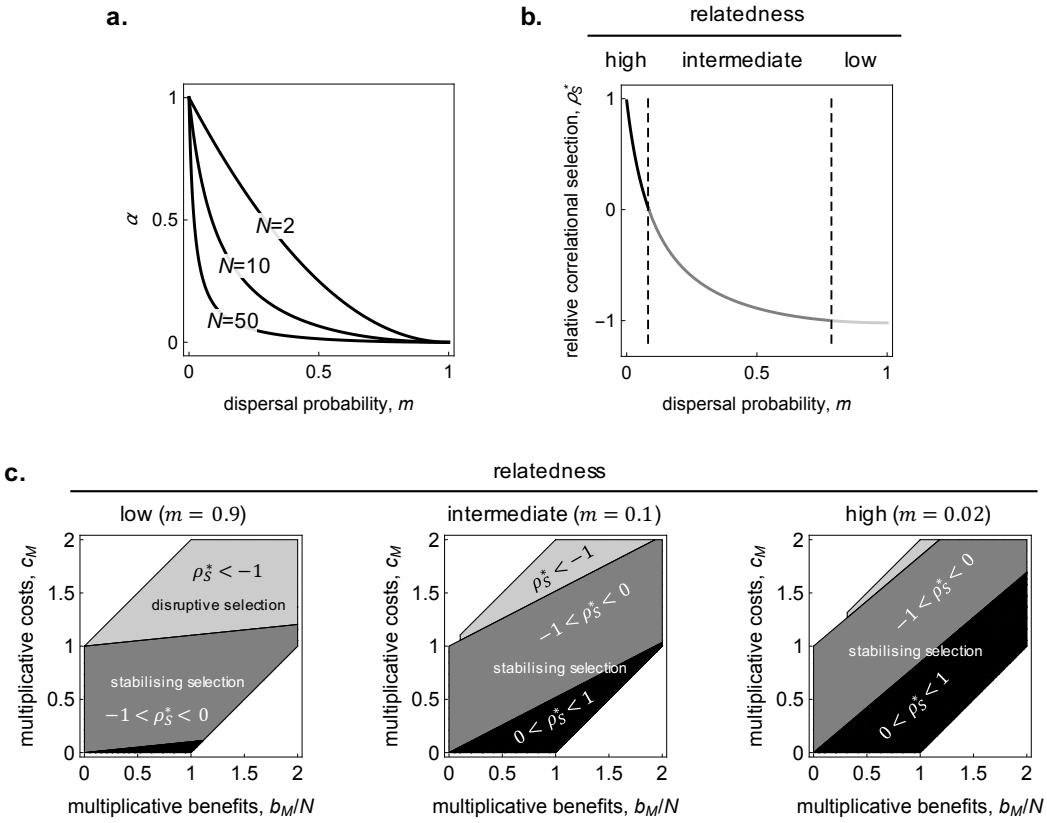

Figure 3: Correlational selection on synergistic social traits. **a.** Factor $\alpha = (1-m)^2(3N-2-m(N-2))/[3N-2+(N-1)(N-2)(1-(1-m)^3)]$ to multiplicative benefits in the coefficient of correlational selection (see eq. 22). **b.** Relative correlational selection, ρ_s^* (eq. 23), as a function of dispersal m, with critical levels of dispersal for which: $\rho_s^* < -1$ (light grey); $-1 < \rho_s^* < 0$ (dark grey); and $0 < \rho_s^* < 1$ (black, with $N = 10$, $b/N = 0.03$, $b_M/N = 1.8$, $c = 0.8$, $c_M = 1$). **c.** Parameter combinations (with $N = 10$, $c = 1$) for which correlational selection at the equilibrium eq. (17) is: (1) strongly negative (and causes selection to be disruptive, $\rho_s^* < -1$ & eq. 23 does not hold, light grey regions); (2) negative (and selection is stabilising, $-1 < \rho_s^* < 0$ & eq. 23 holds, dark grey regions); and (3) positive (and selection is stabilising, $0 < \rho_s^* < 1$ & eq. 23 holds, black regions). White regions correspond to parameter combinations under which the equilibrium is not evolutionary convergent (i.e., eq. 19 does not hold).

inhibited by limited dispersal (e.g., Day, 2001, Ajar, 2003, Wakano and Lehmann, 2014, Parvinen et al., 2017). In such models and ours, limited dispersal inhibits evolutionary branching because it creates genetic correlations among competing individuals, so that a mutant cannot be as different to common types as in well-mixed population. As a result, frequency-dependent disruptive selection is weaker under limited dispersal.

Effect of selection on phenotypic correlation. Putting our stability analyses together (especially eqs. 17, 19, 22, and 23) and validating them using individual-based simulations (see Appendix C for details), we find that there are three possible outcomes for the phenotypic distribution once it has converged to be unimodal around the equilibrium eq. (17) due to selection: (1) when relatedness is low, correlational selection is negative and strong enough to make selection disruptive, which leads to the stable coexistence of individuals specialised in producing a single public good (Figure 4a). In this case, evolutionary dynamics follow so-called "Black queen" dynamics (Morris et al., 2012, Morris, 2015, with special reference to microorganisms): individuals first evolve to produce the same amount of leaky product that is shared among individuals, but the costly maintenance of both traits leads to specialisation in a single product and the evolution of cross-feeding among types. (2) Over a critical level of relatedness, selection becomes stabilising but correlational selection remains negative,

which prevents evolutionary branching and thus specialisation, but still results in a negative association among traits within individuals (Figure 4b). (3) Over another threshold of relatedness, correlational selection becomes positive, so that the traits become positively associated within individuals (Figure 4c). Hence, even though limited dispersal and relatedness have no bearing on the mean of the phenotypic distribution in our model (eqs. 17 and 19), indirect synergistic effects entail that relatedness has a significant influence on the shape of this distribution (which goes from being bimodal with a negative correlation under panmixia to unimodal with a positive correlation under limited dispersal, Figure 4).

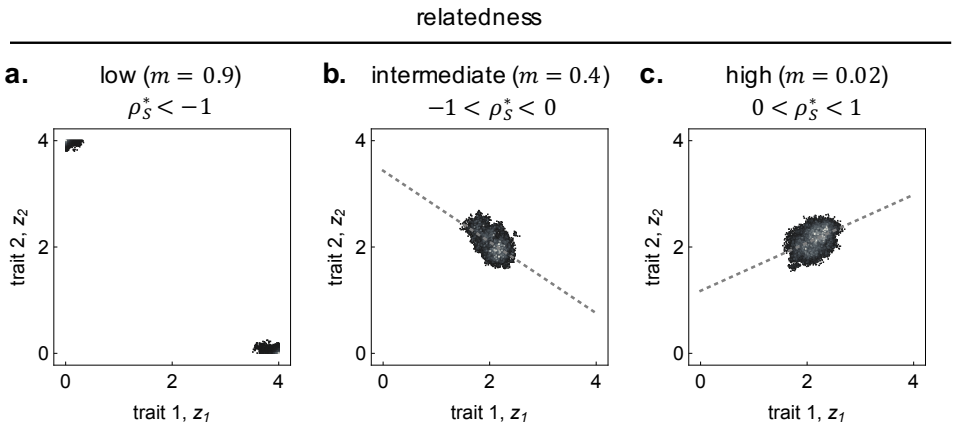

Figure 4: The effect of relatedness and indirect synergy on the phenotypic distribution. Equilibrium phenotypic density distribution, $p_t(\mathbf{z})$, of a simulated population, initially monomorphic for both traits at equilibrium $(2,2)$ (population composed of 1000 groups of size $N = 10$; sampled every 500 generations for 20'000 generations after 30'000 generations of evolution; other parameters: $b_M/N = 1.$, $\nu = 0.01$, $\sigma_{11}^m = \sigma_{22}^m = 0.02$, $\sigma_{12}^m = 0$; see Appendix C for details on simulations). **a**. Strong negative association with social polymorphism (with $b/N = 0.2$, $c = 0.1$, $c_M = 1$). **b**. Negative association (correlation = -0.67, $p < 10^{-10}$; with $b/N = 2.2$, $c = 1$, $c_M = 1.1$). **c**. Positive association (correlation = 0.45, $p < 10^{-10}$; with $b/N = 0.1$, $c = 1$, $c_M = .05$)

Effect of pleiotropy on phenotypic correlation. So far, our analysis has focused on the effects of selection on the stability of jointly evolving traits (an analysis that could have equally been performed using invasion analysis; see Mullon et al., 2016, for such an approach to the joint evolution of multiple traits under limited dispersal). But selection is not the only relevant process for the way phenotypic distributions are shaped. As highlighted by the present quantitative genetic approach, the equilibrium variance-covariance matrix of the phenotypic distributions also depends on the patterns of mutation (captured by matrix $\boldsymbol{M}$ in eq. 11). In particular, pleiotropy is expected to influence the correlations among traits within individuals at an evolutionary equilibrium.

In order to investigate the joint effects of pleiotropy and correlational selection, let us assume that the variance of mutational effect on both traits is the same ($\sigma_{11}^m = \sigma_{22}^m = \sigma_m$), in which case the variance-covariance matrix of mutation effects can be written as

$$\boldsymbol{M} = \nu\sigma_m \begin{pmatrix} 1 & \rho_m \\ \rho_m & 1 \end{pmatrix}, \tag{24}$$

where $\rho_m = \sigma_{12}^m/\sigma_m$ is the correlation of the effect of mutations on traits 1 and 2. The parameter $-1 < \rho_m < 1$ thus captures the degree of pleiotropy between both traits (when it is zero, both traits change independently due to mutation, when it is positive, they tend to change in similar ways, and when it is negative, in opposite ways).

Substituting eqs. (20), (22) and (24) into eq. (11), we find that the correlation ρ_{12}^* among traits 1 and 2 at equi-

librium is

$$-1 < \rho^*_{12} = \frac{\sigma^*_{12}}{\sqrt{\sigma^*_{11}\sigma^*_{22}}} = \frac{\rho_m + \rho^*_s}{1 + \rho_m\rho^*_s + \sqrt{(1-\rho^2_m)(1-\rho^{*2}_s)}} < 1, \tag{25}$$

where ρ^*_s is given in eq. (21). This shows that at equilibrium, the sign of the correlation among between two traits reflects the balance, $\rho_m + \rho^*_s$, between the degree of pleiotropy, ρ_m, and the relative strength of correlational selection ρ^*_s (see Figure 5a; note that eq. 25 can be directly deduced from eq. (11) whenever the variance of mutational effect on both traits is the same, $\sigma^m_{11} = \sigma^m_{22}$, and the coefficients of disruptive selection on independent traits are equal, $h_{11}(\bar{\mathbf{z}}^*) = h_{22}(\bar{\mathbf{z}}^*)$, see eq. 8 of Jones et al., 2007). Since limited dispersal and relatedness has a significant influence on relative correlational selection ρ^*_s (eq. 21), it can affect the correlation ρ^*_{12} among traits in the population as much as pleiotropy, ρ_m.

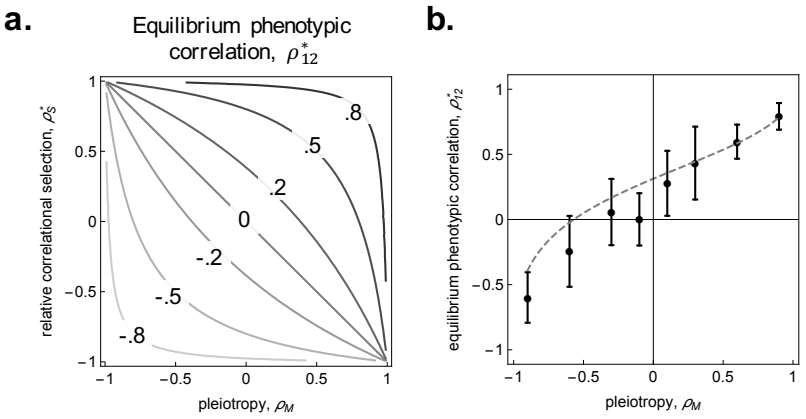

Figure 5: The effect of pleiotropy on phenotypic correlation. a. Contours of predicted phenotypic correlation among traits 1 and 2 at mutation-selection balance, ρ^*_{12}, according to pleiotropy, ρ_m, and the relative strength of correlational selection, ρ^*_s (from eq. 25). **b**. Predicted phenotypic correlation among traits 1 and 2 (dashed grey curve, from eq. 25), and corresponding observations from individual based simulations of a population initially monomorphic for $(2,2)$ divided among 1000 groups of size $N = 10$ (black dots, averaged correlation over 20'000 generations after 30'000 generations of evolution, error bars indicate standard deviation; other parameters: $m = 0.05$, $b/N = 0.2$, $b_M/N = 1$, $c = 1$, $c_M = 0.1$, $\nu = 0.01$, $\sigma^m_{11} = \sigma^m_{22} = 0.02$; see Appendix C for details).

We additionally checked that our model captured pleiotropy correctly by comparing the phenotypic correlation among the two traits at equilibrium predicted by our model (eq. 25) and that observed in simulations for different levels of pleiotropy. We found that model predictions and observations from simulations also matched well in the presence of pleiotropy (Figure 5b).

Dynamics of the distribution. We further tested the accuracy of our dynamical model by comparing individual-based simulations with numerical recursions of eqs. (3). We found that simulated populations tend to have lower phenotypic variance that eqs. (3) would predict (Figure 6). This is probably due to global genetic drift, which our model ignores and which depletes phenotypic variance (as in well-mixed populations, e.g., Wakano and Iwasa, 2013, Débarre and Otto, 2016), and/or the presence of phenotypic skew, which is ignored under our assumption that the phenotypic distribution in the population is normal, but which can influence the dynamics of phenotypic variance (Appendix B, eq. B-18). Nonetheless, we observed overall a good qualitative fit between the predicted and observed dynamics of the phenotypic distribution (Figure 6). This suggests that the assumption of normality yields accurate predictions for the change of mean and variance when dispersal is limited (like in well-mixed populations, Turelli and Barton, 1994).

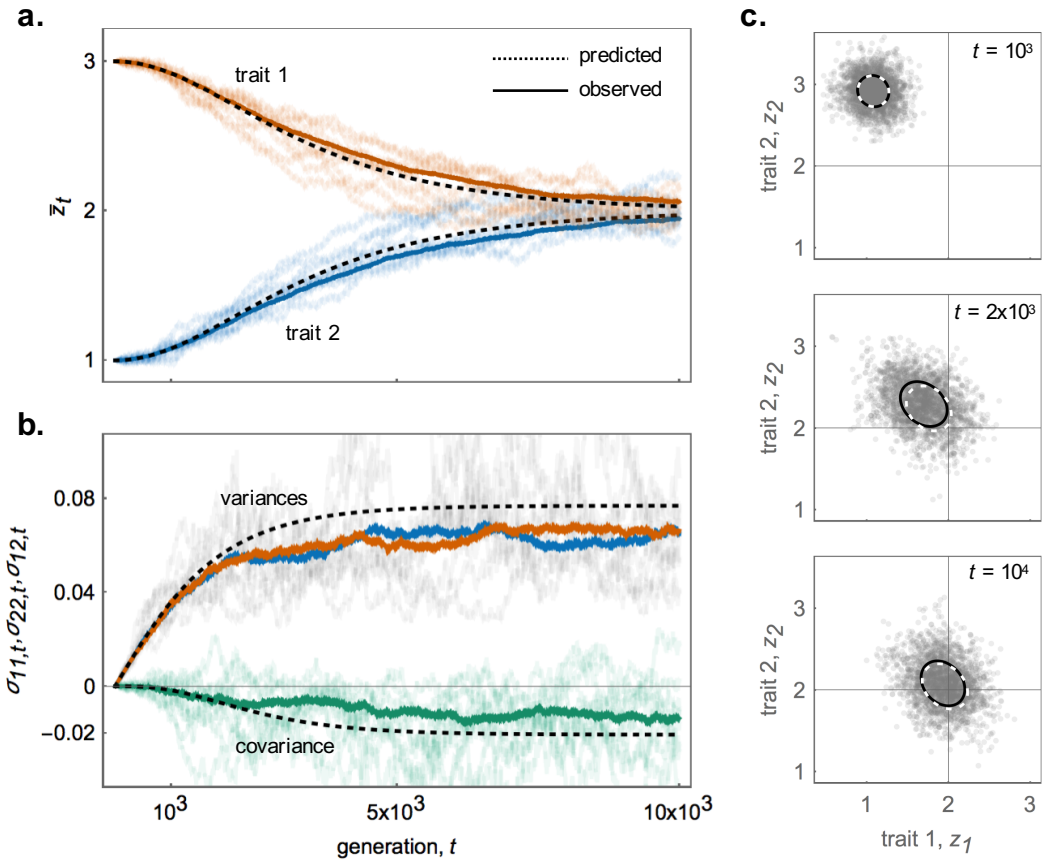

Figure 6: Observed and predicted evolution of the phenotypic distribution, $p_t(\mathbf{z})$. The observed (full lines, from individual based simulations) and predicted (dashed lines, from eq. 3) evolution of the traits' means (**a**. trait 1 in orange and 2 in blue), variances (**b**. trait 1 in orange and 2 in blue) and covariance (**b**. green) for 64 replicates (10 randomly chosen replicates in lighter shade, average over all 64 replicates in darker shade, initial population monomorphic with $z_1 = 3$ and $z_2 = 1$, distributed over 1000 groups of size $N = 10$, other parameters: $m = 0.4$, $b/N = 14.8$, $b_M/N = 0.1$, $c = 5$, $c_M = 2.5$, $\nu = 0.1$, $\sigma^m_{11} = \sigma^m_{22} = 0.02$, $\sigma^m_{12} = 0$; see Appendix C for details). **c**. Snapshot of the population (2'500 individuals randomly sampled across 64 replicates shown by grey points) and variance-covariance ellipses given by the (right) eigenvectors of the **G** matrix (observed across all 64 replicates in full lines and predicted in dashed), at generations: 1'000 (top panel); 2'000 (middle panel); and 10'000 (bottom panel).

4 Discussion

In this paper, we have modelled the evolution of the distribution of genetically determined, quantitative, phenotypic traits under limited dispersal, frequency-dependent selection and pleiotropic mutation. By doing so, we have generalised two classical quantitative genetics results to include limited dispersal, first for the general recurrence formula eq. (1) of the phenotypic distribution under the continuum of alleles model (Kimura, 1965b, Fleming, 1979, Lande, 1979, Bürger, 1986); and second for the closed dynamical system eq. (3) of the vector of means and matrix of variance-covariance when the distribution is normal (Lande, 1979, Lande and Arnold, 1983, Phillips and Arnold, 1989). In both cases, genetic structure due to limited dispersal leads to the replacement of individual fitness in classical quantitative genetics equations by lineage fitness, which is the fitness of a typical carrier of a given phenotype (randomly sampled from the lineage of all members carrying that phenotype). This fitness depends on the phenotypes expressed in the whole population and how they are distributed among groups (eq. 2).

The lineage fitness concept, which is is useful to understand the nature of selection in subdivided popula-

tions, was derived in the context of evolutionary invasion analyses (Lehmann et al., 2015, Mullon et al., 2016, Lehmann et al., 2016, see Akçay and Van Cleve, 2016 for discussion). Comparing such invasion analyses results with our own results reinforces existing links between concepts of evolutionary stability and evolutionary quantitative genetics: the vector of means evolves to convergence stable phenotypic values (eqs. 8-9) and the distribution remains unimodal around such values when they are locally uninvadable or may become bimodal when they are invadable (eq. 10; in agreement with previous results in well-mixed populations, see Charlesworth, 1990, Iwasa et al., 1991, Taper and Case, 1992, Abrams et al., 1993a, Abrams, 2001, Lion, 2018, for the dynamics of the mean, and Sasaki and Dieckmann, 2011, Wakano and Iwasa, 2013, Débarre et al., 2014, Débarre and Otto, 2016, for the dynamics of the variance; under limited dispersal, see Cheverud, 1985, Queller, 1992a,b, Frank, 1998, McGlothlin et al., 2014 for the dynamics of the means, and Wakano and Lehmann, 2014 for the dynamics of the variance of a single trait around a singular strategy). While it may be felt that these links are intuitive and must hold, their demonstration required surprisingly lengthy and tedious calculations due to limited dispersal (see Appendix and the online Supplementary Information).

Contrary to an invasion analysis, a quantitative genetics approach allows to specify the phenotypic distribution at a mutation-selection balance (by using eq. 11). In particular, it allows to study the effects of selection and mutation on the phenotypic associations that emerge among traits at equilibrium (eq. 25). Our analyses of such associations suggest that kin selection due to limited dispersal can mould phenotypic associations as much as pleiotropic mutations (eq. 25 and Fig. 5). By expressing correlational selection on traits in terms of their direct and indirect fitness effects, we gained insights into the influence of kin selection on phenotypic associations (eq. 7, which is in line with previous results from invasion analyses, Mullon et al., 2016). Motivated by our explicit formula for the variance-covariance matrix (eq. 11) and our example (section 3.5), we complement here the discussion found in Mullon et al. (2016) (based on an invasion analysis) on the implications of kin selection for the evolution of within-individual phenotypic associations. As indicated by the decomposition of correlational selection eq. (7a), there are two ways kin selection influences such associations.

The first is through the fitness effects that traits have when co-expressed among relatives, so when traits have indirect synergistic effects (eq. 7b, Figure 1b-d). Under limited dispersal, selection favours an association among two traits within individuals, when such an association between individuals has indirect fitness benefits. Due to such kin selection effects, different levels of dispersal can lead to significantly different evolutionary outcomes for phenotypic associations, as highlighted by our example on the coevolution of two traits whose association within-individual is costly but beneficial between-individuals due to synergy (Figure 4). In this example, we saw that while relatedness has a substantial influence on the shape of the phenotypic distribution, it has no effect on the mean of this distribution (Figure 4, eqs. 17 and 18). Hence, these effects of genetic structure on phenotypic evolution would have gone unnoticed from the study of the dynamics of the mean only (which is the focus of the vast majority of study of quantitative genetics in family-structured populations, e.g., Cheverud, 1985, Queller, 1992a,b, Frank, 1998, McGlothlin et al., 2014), or from the analysis of the selection gradient vector only (as done in the majority of evolutionary analyses to synergistic social traits, e.g., Gandon, 1999, Perrin and Mazalov, 2000, Reuter and Keller, 2001, Lehmann and Perrin, 2002, Rousset and Gandon, 2002, Gardner and West, 2004, Leturque and Rousset, 2004, Hochberg et al., 2008, Brown and Taylor, 2010, Kuijper and Johnstone, 2017). Overall, this example highlights that when traits have indirect synergistic effects between individuals (Figure 1b-d), relatedness is relevant for the way natural selection moulds phenotypic associations within individuals.

A relevant pair of traits likely to be influenced by such kin selection effects is costly helping and punishment, which have synergistic indirect benefits when expressed by different individuals (e.g., Raihani et al., 2012, and references therein). According to our results, kin selection should favour a positive association among helping and punishment, which interestingly, has been observed in humans (Fehr and Gächter, 2000). Another pair of traits whose evolution is likely to be influenced by their joint expression in different individuals is the production and exploitation of a public good, such as the secretion and use of siderophores by microorganisms (West et al., 2006). Under limited diffusion of siderophores and limited bacterial dispersal (Nadell et al., 2009, Kümmerli et al., 2014, Ross-Gillespie et al., 2015), we thus expect kin selection effects to be ecologically relevant for

how secretion and use of siderophores are associated, and more generally for patterns of multi-trait social variation within microbial populations (Cordero and Polz, 2014, van Gestel et al., 2015, Özkaya et al., 2017, Schiessl et al., 2019).

The second way kin selection influences phenotypic associations is via the combination of the indirect effect of one trait with the effect of the other on the tendency to interact with relatives ("synergy via relatedness", eq. 7c, Figure 1e). Specifically, selection favours an association among two traits when it results in fitness benefits being preferentially directed towards relatives or fitness costs towards non-relatives. For example, if trait a has positive indirect fitness effects (e.g., altruistic helping) and trait b decreases the tendency to interact with relatives (e.g., dispersal), then selection favours a negative correlation between traits a and b (e.g., Koella, 2000, Purcell et al., 2012, Mullon et al., 2018). We refer readers interested in this effect to Mullon et al. (2016), in which it is discussed at greater length, in particular in the context of dispersal syndromes (Edelaar and Bolnick, 2012, Ronce and Clobert, 2012).

More generally, our evolutionary perspective on phenotypic associations may be useful to empiricists who investigate correlational selection among traits in experimental or natural population (e.g., Blows and Brooks, 2003, Blows, 2007, for reviews, and ch. 30 of Walsh and Lynch, 2018). Based on Lande and Arnold (1983) paper, the typical starting point of such studies is to perform a quadratic regression of individual fitness on the multiple traits expressed by this individual (for e.g., eq. 30.11 of Walsh and Lynch, 2018). The linear regression coefficients are collected in a vector usually denoted $\boldsymbol{\beta}$ with entry β_a interpreted as directional selection on trait a, and the quadratic coefficients in a matrix $\boldsymbol{\gamma}$ with entry γ_{ab} interpreted as correlational selection on traits a and b (in our notation, $\beta_a = \partial w(\mathbf{z}_i, \mathbf{z}_{-i}, \bar{\mathbf{z}}_t)/\partial z_{i,a}$ and $\gamma_{ab} = \partial^2 w(\mathbf{z}_i, \mathbf{z}_{-i}, \bar{\mathbf{z}}_t)/(\partial z_{i,a} \partial z_{i,b})$). This correspondence between selection on traits and regression coefficients on individual fitness, however, is only valid in well-mixed populations. Indeed, as our analysis has shown, $\boldsymbol{\beta}$ and $\boldsymbol{\gamma}$ are respectively equal to the selection gradient $\boldsymbol{s}(\bar{\mathbf{z}}_t)$ and Hessian matrix $\boldsymbol{H}(\bar{\mathbf{z}}_t)$, only when all relatedness coefficients are zero (eq. 6 and 7). For populations that are genetically structured, empirical estimates of selection on multiple traits require to: first regress individual fitness on the traits of the focal individual and on those of its social partners; and second, weigh these indirect fitness effects by relatedness coefficients (according to eqs. 6 and 7). While such considerations have long been established for the directional selection gradient (so considering only linear regression coefficients, $\boldsymbol{\beta}$, e.g., Cheverud, 1985, Queller, 1992a,b, Frank, 1998, McGlothlin et al., 2014, see also ch. 5 of Walsh and Lynch, 2018), our analysis has further quantified the relationship between correlational selection and quadratic regression coefficients ($\boldsymbol{\gamma}$, eq. 7, Figure 1b-d).

In practice, it is likely to be challenging to obtain reliable estimates of all the quadratic regression coefficients necessary to quantify the strength and direction of correlational selection (eq. 7). But our results can nevertheless be of use when designing experimental assays or interpreting collected data. For instance, our results show that for traits that underlie social behaviours, such as mating, aggression or cooperation, there is little reason to believe that quadratic regression coefficients on individual fitness alone are relevant estimates of correlational selection. A corollary to this is that when there is mismatch between phenotypic correlations among two traits observed in a population on one hand, and the quadratic regression coefficient on individual fitness from experimental assays on the other (e.g., Bell and Sih, 2007, Adriaenssens and Johnsson, 2012, Han and Brooks, 2013, Akçay et al., 2015), this may indicate the presence of indirect synergistic fitness effects among traits and genetic structure in the population (rather than genetic constraints).

Our results further provide insight into the effects of limited dispersal on how selection influences the $\mathbf{G}$ matrix of additive genetic variances-covariances (Steppan et al., 2002, Arnold et al., 2008). Previous theoretical works have studied how linkage disequilibrium, pleiotropy and epistasis influence $\mathbf{G}$ under selection (Lande, 1980, 1984, Turelli, 1985, Turelli and Barton, 1990, Revell, 2007, Jones et al., 2014), but the effects of limited dispersal on $\mathbf{G}$ have either been assessed in the absence of selection (Lande, 1992), or when selection is frequency-independent (Jones et al., 2004, Guillaume and Whitlock, 2007, Guillaume, 2011, Björklund and Gustafsson, 2015). Here, we have shown that kin selection effects due to limited dispersal are relevant for the way selection favours phenotypic associations (i.e., for correlational selection, eq. 7), which in the long run can lead to

genetic correlations through genetic integration (Sinervo and Svensson, 2002, Roff and Fairbairn, 2012). Of course, our model ignores many relevant features for quantitative genetics, such as environmental effects, genetic dominance, genetic linkage or sexual reproduction. Incorporating such features into our framework is likely to make the analysis of selection more complicated, but it would allow to study important questions on the genetic basis of variation, such as how genetic architecture and its evolution influence trait associations (e.g., Saltz et al., 2017).

One other significant limitation to our present approach is that it assumes that the phenotypic distribution is normal. This assumption is likely to be violated under frequency-dependent selection, which can lead to skewed and complicated distributions. In particular, the normal assumption precludes investigating what happens to the phenotypic distribution once evolutionary branching has occurred (like adaptive dynamics models based on the invasion analyses of monomorphic populations). To relax this assumption would entail tracking the dynamics of higher moments of the phenotypic distribution. One possible way to retain some mathematical tractability would be to use the oligomorphic approximation proposed by Sasaki and Dieckmann (2011). This approximation decomposes a multimodal trait distribution into a sum of unimodal distributions, each corresponding to a morph. Applying Sasaki and Dieckmann (2011)'s approach, which was developed for a large and well-mixed population, to a dispersal limited one, would be an interesting avenue of future research, as well as including class-structure (e.g., age- or sex-structure).

To conclude, we have derived a quantitative genetics framework to study gradual evolution of multiple traits under frequency-dependent selection and pleiotropic mutations when dispersal is limited. This framework is especially relevant to study how associations within individuals between social traits emerge in response to mutation and selection under limited dispersal. Our results could therefore help understand patterns of intraspecific variation in social behaviour (such as behavioural syndromes, Dall et al., 2004, Dingemanse et al., 2012; social niche specialisation, Bergmüller and Taborsky, 2007, Montiglio et al., 2013; or social division of labour, Boehm, 2002, Wright et al., 2014), which are increasingly thought to be ecologically significant (Bolnick et al., 2011, Wolf and Weissing, 2012, Sih et al., 2012, Canestrelli et al., 2016, Chaturvedi et al., 2017, Estrela et al., 2019). More broadly, by connecting different branches of theoretical evolutionary biology, from invasion analysis to adaptive dynamics to quantitative genetics, the present framework further bolsters the notion that whatever modelling approach is taken, natural selection cannot be divorced from kin selection when dispersal is limited (Hamilton, 1964, Frank, 1998, Rousset, 2004, van Baalen M, 2013, Lehmann et al., 2016).

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Appendix

A Phenotypic distribution dynamics

In this appendix, we derive eq. (1) of the main text.

A.1 Process construction

We first lay the foundations of our analysis by describing how phenotypic evolution in our model population (see section 2) is represented mathematically.

A.1.1 Markov chain

The phenotypic state, or state for short, of a group at given time point is given by the set of phenotypic values of all individuals residing in that group, $\{\mathbf{z}_1,\dots,\mathbf{z}_N\}$ (where $\mathbf{z}_i = \{z_{i,1},...,z_{i,n}\} \in \mathbb{R}^n$ is the phenotype of individual indexed $i \in \{1,\dots,N\}$). The state of each group in the population changes stochastically from one time period to the next (i.e., after one iteration of the life cycle) due to selection, mutation and dispersal. We assume that these changes can be modelled as a discrete time Markov chain on a continuous state space (as traits are continuous; see Meyn and Tweedie, 2009, for general state spaces). Because groups affect one another through dispersal, the transition kernel of a group depends on the state of all the other groups. But since there is an infinite number of groups and there is no isolation-by-distance (i.e., all groups are equally connected to one another through dispersal), the infinite set of interacting Markov chains (one for each group) can be described as a single Markov chain (for a focal group), whose kernel is a function of the expected value of the process (see Chesson, 1981, 1984, for ecological models). In other words, we can focus on the stochastic dynamics of a focal group and ignore the stochasticity stemming from groups other than the focal one.

A.1.2 Markov chain in terms of counting measures

Note that to describe the state of a focal group, the order of elements in $\{\mathbf{z}_1,\dots,\mathbf{z}_N\}$ does not matter (because there is no class structure in our population, we do not care which specific individual carries a given phenotype within a group). What matters is how many individuals carry which phenotypes. We can thus represent the state of the focal group by a function, a *counting measure*, that counts the number of individuals with phenotypes that belong to an arbitrary set. Specifically, the counting measure, denoted μ, takes a subset $E \subseteq \mathbb{R}^n$ and sends it to a non-negative integer by counting the number of individuals within the focal group with phenotypes that belong to E according to the following definition

$$\mu(E) = \sum_{i=1}^{N} \delta_{\mathbf{z}_i}(E), \tag{A-1}$$

where δ is the dirac measure,

$$\delta_{\mathbf{z}_i}(E) = \begin{cases} 1, & \text{if } \mathbf{z}_i \in E; \\ 0, & \text{otherwise,} \end{cases} \tag{A-2}$$

(p. 51 of Harris, 1963, p. 3 of Daley and Vere-Jones, 2003; see also pp. 228-229 of Bürger and Bomze, 1996 for further formal considerations on using counting measures to describe a population under the continuum-of-alleles model). Applied to a single phenotypic value $\mathbf{z} = \{z_1,...,z_n\} \in \mathbb{R}^n$, where z_a is the value of trait a, $\mu(\mathbf{z})$

returns the number of individuals with phenotype $\mathbf{z}$, and applied to the whole set of possible phenotypic values, it returns group size, $\mu(\mathbb{R}^n) = N$.

Under definition eq. (A-1), each possible state that a group can assume is uniquely determined by a specific counting measure, i.e., for each unordered set of N vectors in $\mathbb{R}^n$, there exists a unique counting measure (p. 52 of Harris, 1963 and p. 7 of Daley and Vere-Jones, 2003). We can therefore study the dynamics of the state of the focal group by studying the dynamics of its equivalent counting measure (Daley and Vere-Jones, 2003, p. 13-15). So, if S_t denotes the (random) counting measure of a focal group at time period t, we can study the Markov chain $\{S_t\}$ on the space of all finite counting measures, which we write as $\mathcal{S}$. This type of construction has so far primarily been used to study phenotypic evolution in populations that are well-mixed and when time is continuous (e.g., Bürger and Bomze, 1996, Oechssler and Riedel, 2001, Champagnat et al., 2006, Champagnat and Lambert, 2007; but see Morale et al., 2005, Simon, 2008, for populations in explicit space).

A.1.3 State dynamics

To describe the stochastic dynamics of the state of a focal group, we let

$$\phi_t(T) = \Pr\left[S_t \in T\right], \tag{A-3}$$

denote the probability that a focal group is in a state that belongs to a subset $T \subseteq \mathcal{S}$ at time period t (equivalent to eq. 3.3 of Harris, 1963, p. 55). Since there is an infinite number of groups, $\phi_t(T)$ also gives the distribution of group states in the whole population. The dynamics $\phi_t(T)$ are governed by the Markov kernel transition function,

$$P(T|\mu;\phi_t) = \Pr\left[S_{t+1} \in T|S_t = \mu;\phi_t\right], \tag{A-4}$$

which is the probability that a group will be in a state that belongs to a subset $T \subseteq \mathcal{S}$ at time period $t+1$, given that it was in state μ at time period t and that the population distribution of states is ϕ_t (this is a non-homogeneous Markov chain, eq. 6.1 of Harris, 1963, p. 60).

State dynamics, or the probability that the focal group is in a state that belongs to $T \subseteq \mathcal{S}$ at time period $t+1$, can then be written as

$$\phi_{t+1}(T) = \int_{\mathcal{S}} \int_{T} P(\mu'|\mu;\phi_t)\phi_t(\mu)\mathrm{d}\mu'\mathrm{d}\mu, \tag{A-5}$$

i.e., the sum of weighted probabilities of going from all states $\mu \in \mathcal{S}$ to states $\mu' \in T$. Because one iteration of the life cycle (from t to $t+1$) encompasses many events, like selection, mutation, and dispersal, the transition kernel for our model is difficult to characterise (studies like Champagnat et al., 2006, are capable of constructing explicit transition kernels by considering time steps small enough so that only one event can occur per step). To model the evolutionary process in a more practical way, we will focus on the dynamics of the distribution of phenotypes across the entire population rather than on the dynamics of the distribution of group states $\phi_t(T)$.

A.2 Recurrence for the phenotypic distribution

The distribution of phenotypes across the entire population at time t is given by the density function

$$p_t(\mathbf{z}) = \int_{\mathcal{S}} \frac{\mu(\mathbf{z})}{N}\phi_t(\mu)\mathrm{d}\mu, \tag{A-6}$$

where $\mu(\mathbf{z})/N$ is the frequency of individuals with phenotype $\mathbf{z}$ within a group in state μ (recall that all groups have the same size N). Using eq. (A-5), the phenotypic distribution at time period $t+1$ can be written as

$$p_{t+1}(\mathbf{z}) = \int_{\mathcal{S}} \frac{\mu(\mathbf{z})}{N} \phi_{t+1}(\mu) \mathrm{d}\mu = \frac{1}{N} \int_{\mathcal{S}} \int_{\mathcal{S}} \mu'(\mathbf{z}) P(\mu'|\mu;\phi_t) \phi_t(\mu) \mathrm{d}\mu' \mathrm{d}\mu = \frac{1}{N} \int_{\mathcal{S}} \lambda_\mu(\mathbf{z},\phi_t) \phi_t(\mu) \mathrm{d}\mu, \tag{A-7}$$

where

$$\lambda_\mu(\mathbf{z},\phi_t) = \int_{\mathcal{S}} \mu'(\mathbf{z}) P(\mu'|\mu;\phi_t) \mathrm{d}\mu' \tag{A-8}$$

is the expected number of individuals with phenotype $\mathbf{z}$ residing in a focal group at time $t+1$, given that this focal group was in state μ at time t (and the population state distribution was ϕ_t). We can decompose this expected number as

$$\lambda_\mu(\mathbf{z},\phi_t) = \lambda^{\mathrm{P}}_\mu(\mathbf{z},\phi_t) + \lambda^{\mathrm{I}}_\mu(\mathbf{z},\phi_t), \tag{A-9}$$

where $\lambda^{\mathrm{P}}_\mu(\mathbf{z},\phi_t)$ is the expected number of philopatric individuals (i.e., surviving adults or offspring that have remained in their natal group) and $\lambda^{\mathrm{I}}_\mu(\mathbf{z},\phi_t)$ is the expected number of immigrant offspring (i.e., coming from other groups) with phenotype $\mathbf{z}$. We aim to express these expected numbers in terms of the fitness of individuals at time t.

A.2.1 Individual fitness

The number, $\lambda^{\mathrm{P}}_\mu(\mathbf{z},\phi_t)$, of philopatric individuals with phenotype $\mathbf{z}$ can be expressed in terms of fitness components of individuals at time t as

$$\lambda^{\mathrm{P}}_\mu(\mathbf{z},\phi_t) = \int_{\mathbb{R}^n} \mu(\mathbf{z}') w^{\mathrm{P}}_\mu(\mathbf{z}',\phi_t) u(\mathbf{z}',\mathbf{z}) \mathrm{d}\mathbf{z}', \tag{A-10}$$

where $w^{\mathrm{P}}_\mu(\mathbf{z}',\phi_t)$ is philopatric fitness: it is the expected number of offspring produced by a *single* individual (including itself if it survives) bearing $\mathbf{z}'$ a time t (given that it resides in a state μ group); and $u(\mathbf{z}',\mathbf{z})$, is the p.d.f. for the event that the offspring produced by an individual with phenotype $\mathbf{z}'$ has phenotype $\mathbf{z}$. Note that we assume that surviving adults and offspring mutate alike. While this is relevant to unicellular organisms, an application specific to multicellular organisms would require distinguishing between two components of philopatric fitness: adult survival and offspring production. This would only complicate eq. (1) but would not affect our other results presented in the main text (eq. 3 onwards) as we later assume that mutations are rare, so that the chances of mutating during an individual's lifetime would be unlikely (Appendix B.1.2).

Likewise, we can write the expected number of immigrant offspring as

$$\lambda^{\mathrm{I}}_\mu(\mathbf{z},\phi_t) = \int_{\mathbb{R}^n} \int_{\mathcal{S}} \mu'(\mathbf{z}') w^{\mathrm{D}}_{\mu,\mu'}(\mathbf{z}',\phi_t) u(\mathbf{z}',\mathbf{z}) \phi_t(\mu') \mathrm{d}\mu' \mathrm{d}\mathbf{z}', \tag{A-11}$$

where $w^{\mathrm{D}}_{\mu,\mu'}(\mathbf{z}',\phi_t)$ is the expected number of successful emigrant offspring of a *single* individual with phenotype $\mathbf{z}'$, given that it resides in a group in state $\mu' \in \mathcal{S}$, and that the colonized group (i.e., the group the offspring lands in) was in state μ at time t.

Substituting eqs. (A-10) and (A-11) into eq. (A-9), which is in turn substituted into eq. (A-7), the phenotypic distribution at $t+1$ reads as

$$p_{t+1}(\mathbf{z}) = \frac{1}{N} \int_{\mathcal{S}} \int_{\mathbb{R}^n} \left(\mu(\mathbf{z}') w^{\mathrm{P}}_\mu(\mathbf{z}',\phi_t) + \int_{\mathcal{S}} \mu'(\mathbf{z}') w^{\mathrm{D}}_{\mu,\mu'}(\mathbf{z}',\phi_t) \phi_t(\mu') \mathrm{d}\mu' \right) u(\mathbf{z}',\mathbf{z}) \mathrm{d}\mathbf{z}' \phi_t(\mu) \mathrm{d}\mu. \tag{A-12}$$

By exchanging integral variables μ and μ' in the second summand within brackets, we obtain

$$p_{t+1}(\mathbf{z}) = \frac{1}{N}\int_{\mathcal{S}}\int_{\mathbb{R}^n} \mu(\mathbf{z}')\left(w_\mu^{\mathrm{P}}(\mathbf{z}',\phi_t) + w_\mu^{\mathrm{D}}(\mathbf{z}',\phi_t)\right)u(\mathbf{z}',\mathbf{z})\mathrm{d}\mathbf{z}'\phi_t(\mu)\mathrm{d}\mu, \tag{A-13}$$

where

$$w_\mu^{\mathrm{D}}(\mathbf{z}',\phi_t) = \int_{\mathcal{S}} w_{\mu',\mu}^{\mathrm{D}}(\mathbf{z}',\phi_t)\phi_t(\mu')\mathrm{d}\mu' \tag{A-14}$$

is the expected number of successful dispersing offspring produced by an individual with phenotype $\mathbf{z}'$, given that this individual resides in a group in state μ at time t.

Individual fitness is then defined as

$$w_\mu(\mathbf{z}',\phi_t) = w_\mu^{\mathrm{P}}(\mathbf{z}',\phi_t) + w_\mu^{\mathrm{D}}(\mathbf{z}',\phi_t), \tag{A-15}$$

which gives the expected number of successful offspring produced by an individual with phenotype $\mathbf{z}'$, given that this individual resides in a group in state μ at time t (and the population state distribution was ϕ_t). In terms of this individual fitness function, the phenotypic distribution at time $t+1$ (eq. A-13) reads as

$$p_{t+1}(\mathbf{z}) = \frac{1}{N}\int_{\mathcal{S}}\int_{\mathbb{R}^n} \mu(\mathbf{z}')w_\mu(\mathbf{z}',\phi_t)u(\mathbf{z}',\mathbf{z})\mathrm{d}\mathbf{z}'\phi_t(\mu)\mathrm{d}\mu. \tag{A-16}$$

A.2.2 Lineage fitness

To go from eq. (A-16) to eq. (1) of the main text, let us define

$$W(\mathbf{z}',\phi_t) = \int_{\mathcal{S}} w_\mu(\mathbf{z}',\phi_t)q_t(\mu|\mathbf{z}')\mathrm{d}\mu, \tag{A-17}$$

where

$$q_t(\mu|\mathbf{z}') = \frac{\mu(\mathbf{z}')}{N}\frac{\phi_t(\mu)}{p_t(\mathbf{z}')} \tag{A-18}$$

is the p.d.f. for the event that an individual resides in a group in state μ at time t given that this individual bears phenotype $\mathbf{z}'$. In other words, $q_t(\mu|\mathbf{z}')$ gives the probability that an individual, randomly sampled at time t from the collection of individuals with phenotype $\mathbf{z}'$ in the population (the "$\mathbf{z}'$-lineage"), resides in a group in a state μ. As such, $W(\mathbf{z}',\phi_t)$ (eq. A-17) is the expected fitness of a member of the $\mathbf{z}'$-lineage at time t (where expectation is taken over all possible groups this member can belong to) and a multi-allelic version of *lineage fitness* (Lehmann et al., 2015, Mullon et al., 2016, Lehmann et al., 2016).

Substituting eq. (A-17) into eq. (A-16), we obtain that the individual phenotypic density distribution is

$$p_{t+1}(\mathbf{z}) = \int_{\mathbb{R}^n} W(\mathbf{z}',\phi_t)u(\mathbf{z}',\mathbf{z})p_t(\mathbf{z}')\mathrm{d}\mathbf{z}', \tag{A-19}$$

which combines the forces of mutation and selection on phenotypic change. To start disentangling these, note that when the probability of a mutation is independent from parental phenotype, the p.d.f. for the event that the offspring of an individual with phenotype $\mathbf{z}'$ has phenotype $\mathbf{z}$ can be expressed as

$$u(\mathbf{z}',\mathbf{z}) = (1-\nu)\delta(\mathbf{z}'-\mathbf{z}) + \nu\upsilon(\mathbf{z}',\mathbf{z}), \tag{A-20}$$

where ν is the probability that an offspring has a mutant phenotype (i.e., $1-u(\mathbf{z},\mathbf{z}) = \nu$ for all $\mathbf{z}$), $\delta(\mathbf{z}'-\mathbf{z})$ is the Dirac delta function, and $\upsilon(\mathbf{z}',\mathbf{z})$ is the conditional probability of mutating from $\mathbf{z}'$ to $\mathbf{z}$ given that a mutation has occurred. So the first term of eq. (A-20) captures the event of no mutation, in which case the offspring

has the same phenotype than its parent, and the second term captures the event of a mutation. Substituting eq. (A-20) into eq. (A-19), we finally obtain eq. (1) in the main text, as required.

B The dynamics of trait means and variance-covariance

Here, we derive eqs. (3)-(7) of the main text, which govern the closed dynamics of trait means and variance-covariance. As mentioned in the main text, this derivation hinges upon several assumptions that we detail below.

B.1 Weak selection and mutation

B.1.1 Weak selection

We first assume that the phenotypic distribution, $p_t(\mathbf{z})$, is peaked around the population mean $\bar{\mathbf{z}}_t = \int_{\mathbb{R}^n} \mathbf{z} p_t(\mathbf{z}) \mathrm{d}\mathbf{z}$ (i.e., the phenotypic variance is small). We can thus approximate lineage fitness, $W(\mathbf{z}, \phi_t)$, as a second-order Taylor expansion around $\bar{\mathbf{z}}_t$. The computation of this approximation is straightforward and only requires careful bookkeeping. But because it is long and tedious, we have relegated step-by-step calculations to the online Supplementary Information and only report our result here.

We show (see eq. SI-49 in the Supplementary Information) that as a second-order Taylor expansion around $\bar{\mathbf{z}}_t$, lineage fitness can be written as

$$W(\mathbf{z}, \phi_t) = W(\bar{\mathbf{z}}_t, \phi_t) + \sum_{a=1}^{n} \xi_t(z_a) s_{a,t}(\bar{\mathbf{z}}_t) + \frac{1}{2} \sum_{a=1}^{n} \sum_{b=1}^{n} \xi_t(z_a) \xi_t(z_b) h_{ab,t}(\bar{\mathbf{z}}_t) + \mathcal{O}(\xi_t^3), \tag{B-1}$$

where

$$W(\bar{\mathbf{z}}_t, \phi_t) = 1 - \frac{1}{2} \sum_{a=1}^{n} \sum_{b=1}^{n} \sigma_{ab,t} h_{ab,t}(\bar{\mathbf{z}}_t) + \mathcal{O}(\xi_t^3) \tag{B-2}$$

is the lineage fitness of the average phenotype $\bar{\mathbf{z}}_t$; $\xi_t(z_a) = z_a - \bar{z}_{a,t}$ denotes the difference between a value z_a and the average trait value a; $\sigma_{ab,t} = \int_{\mathbb{R}^n} \xi_t(z_a) \xi_t(z_b) p_t(\mathbf{z}) \mathrm{d}\mathbf{z}$ is the (co)variance among traits a and b in the population; $s_{a,t}(\bar{\mathbf{z}}_t)$ is the first-order effect of change in trait a away from $\bar{\mathbf{z}}_t$ on lineage fitness (i.e., $s_{a,t}(\bar{\mathbf{z}}_t) = \partial W(\mathbf{z}, \phi_t)/\partial z_a|_{\mathbf{z}=\bar{\mathbf{z}}_t}$); $h_{ab,t}(\bar{\mathbf{z}}_t)$ is the second-order effect of a joint change in traits a and b away from $\bar{\mathbf{z}}_t$ on lineage fitness (i.e., $h_{ab,t}(\bar{\mathbf{z}}_t) = \partial^2 W(\mathbf{z}, \phi_t)/\partial z_a \partial z_b|_{\mathbf{z}=\bar{\mathbf{z}}_t}$); and ξ_t is the maximum deviation between individual trait value in the population and the population mean trait value at time t. We detail the first- and second-order effects below.

The first-order effect is given by

$$s_{a,t}(\bar{\mathbf{z}}_t) = \left. \frac{\partial w(\mathbf{z}_i, \mathbf{z}_{-i}, \bar{\mathbf{z}}_t)}{\partial z_{i,a}} \right|_{\substack{\mathbf{z}_i=\bar{\mathbf{z}}_t \\ \mathbf{z}_{-i}=\bar{\mathbf{z}}_t}} + (N-1) r^{\circ}_{2,t}(\bar{\mathbf{z}}_t) \left. \frac{\partial w(\mathbf{z}_i, \mathbf{z}_{-i}, \bar{\mathbf{z}}_t)}{\partial z_{j,a}} \right|_{\substack{\mathbf{z}_i=\bar{\mathbf{z}}_t \\ \mathbf{z}_{-i}=\bar{\mathbf{z}}_t}}, \tag{B-3}$$

where individual fitness, $w(\mathbf{z}_i, \mathbf{z}_{-i}, \bar{\mathbf{z}}_t)$, is written as in the main text eq. (5) and $r^{\circ}_{2,t}(\bar{\mathbf{z}}_t)$ is a neutral time-dependent coefficient of pairwise relatedness (i.e., the probability that two individuals sampled at random within a group at time t are identical-by-descent in the absence of selection).

The second-order effect is given by

$$h_{ab,t}(\bar{\mathbf{z}}_t) = h_{\mathrm{w},ab,t}(\bar{\mathbf{z}}_t) + h_{\mathrm{r},ab,t}(\bar{\mathbf{z}}_t), \tag{B-4a}$$

with

$$h_{\mathrm{w},ab,t}(\bar{\mathbf{z}}_t) = \frac{\partial^2 w(\mathbf{z}_i,\mathbf{z}_{-i},\bar{\mathbf{z}}_t)}{\partial z_{i,a}\partial z_{i,b}}\bigg|_{\substack{\mathbf{z}_i=\bar{\mathbf{z}}_t\\ \mathbf{z}_{-i}=\bar{\mathbf{z}}_t}} + r^{\circ}_{2,t}(\bar{\mathbf{z}}_t)(N-1)\frac{\partial^2 w(\mathbf{z}_i,\mathbf{z}_{-i},\bar{\mathbf{z}}_t)}{\partial z_{j,a}\partial z_{j,b}}\bigg|_{\substack{\mathbf{z}_i=\bar{\mathbf{z}}_t\\ \mathbf{z}_{-i}=\bar{\mathbf{z}}_t}}$$
$$+ r^{\circ}_{2,t}(\bar{\mathbf{z}}_t)(N-1)\left(\frac{\partial^2 w(\mathbf{z}_i,\mathbf{z}_{-i},\bar{\mathbf{z}}_t)}{\partial z_{i,a}\partial z_{j,b}}\bigg|_{\substack{\mathbf{z}_i=\bar{\mathbf{z}}_t\\ \mathbf{z}_{-i}=\bar{\mathbf{z}}_t}} + \frac{\partial^2 w(\mathbf{z}_i,\mathbf{z}_{-i},\bar{\mathbf{z}}_t)}{\partial z_{i,b}\partial z_{j,a}}\bigg|_{\substack{\mathbf{z}_i=\bar{\mathbf{z}}_t\\ \mathbf{z}_{-i}=\bar{\mathbf{z}}_t}}\right) \tag{B-4b}$$
$$+ r^{\circ}_{3,t}(\bar{\mathbf{z}}_t)(N-1)(N-2)\frac{\partial^2 w(\mathbf{z}_i,\mathbf{z}_{-i},\bar{\mathbf{z}}_t)}{\partial z_{j,a}\partial z_{k,b}}\bigg|_{\substack{\mathbf{z}_i=\bar{\mathbf{z}}_t\\ \mathbf{z}_{-i}=\bar{\mathbf{z}}_t}},$$

and

$$h_{\mathrm{r},ab,t}(\bar{\mathbf{z}}_t) = (N-1)\frac{\partial w(\mathbf{z}_i,\mathbf{z}_{-i},\bar{\mathbf{z}}_t)}{\partial z_{j,b}}\bigg|_{\substack{\mathbf{z}_i=\bar{\mathbf{z}}_t\\ \mathbf{z}_{-i}=\bar{\mathbf{z}}_t}} \times \frac{\partial r_{2,t}(\mathbf{z})}{\partial z_a}\bigg|_{\mathbf{z}=\bar{\mathbf{z}}_t} + (N-1)\frac{\partial w(\mathbf{z}_i,\mathbf{z}_{-i},\bar{\mathbf{z}}_t)}{\partial z_{j,a}}\bigg|_{\substack{\mathbf{z}_i=\bar{\mathbf{z}}_t\\ \mathbf{z}_{-i}=\bar{\mathbf{z}}_t}} \times \frac{\partial r_{2,t}(\mathbf{z})}{\partial z_b}\bigg|_{\mathbf{z}=\bar{\mathbf{z}}_t}, \tag{B-4c}$$

where $r^{\circ}_{3,t}(\bar{\mathbf{z}}_t)$ is the neutral time-dependent three-way relatedness (i.e., the probability that three individuals sampled at random within a group at time t are identical-by-descent in the absence of selection); and $\partial r_{2,t}(\mathbf{z})/\partial z_a$ is the marginal effect of a change in trait a on time-dependent pairwise relatedness.

The first (eq. B-3) and second (eq. B-4) order effects are the same as the selection gradient (eq. 6) and correlational selection (eq. 7) of the main text, respectively, with the exception that relatedness coefficients are time-dependent in eqs. (B-3) and (B-4) and independent in eqs. (6) and (7). We will specify in section B.2.2 below how we can get rid of this time dependence, but first, we need to make a further assumption.

B.1.2 Weak mutation

Second, we assume that mutations are rare, with the probability of mutating, ν, of the order $\mathcal{O}(\xi_t^2)$. Under this assumption, note that $\nu W(\mathbf{z},\phi_t) = \nu + \mathcal{O}(\xi_t^4)$ (as $\sigma_{ab,t} \sim \mathcal{O}(\xi_t^2)$ in eq. B-2). We can therefore rewrite eq. (1) of the main text as

$$p_{t+1}(\mathbf{z}) = W(\mathbf{z},\phi_t)p_t(\mathbf{z}) + \nu\left(\int_{\mathbb{R}^n} p_t(\mathbf{z}')\upsilon(\mathbf{z}',\mathbf{z})\mathrm{d}\mathbf{z}' - p_t(\mathbf{z})\right) + \mathcal{O}(\xi_t^3), \tag{B-5}$$

where the first term captures the effects of selection only, and the next term, the effects of mutation only. Eq. (B-5) takes the same functional form as classical recurrence for the phenotypic distribution in well-mixed populations when selection and mutation are weak (under the continuum-of-alleles model, e.g., eq. 1 of Bürger, 1986; for fluctuating population size, see eq. 4.9 of Champagnat et al., 2006), but with lineage, $W(\mathbf{z},\phi_t)$, instead of individual fitness. Next, we use eq. (B-5) to derive recurrence equations for the changes in mean trait values and the phenotypic variance-covariance matrix over one time period.

B.1.3 Dynamics of the mean trait values

By definition, the change in the mean of trait a over one time period is

$$\Delta\bar{z}_{a,t} = \bar{z}_{a,t+1} - \bar{z}_{a,t} = \int_{\mathbb{R}^n} \xi_t(z_a)p_{t+1}(\mathbf{z})\mathrm{d}\mathbf{z}. \tag{B-6}$$

Substituting eq. (B-5) into eq. (B-6), we obtain

$$\Delta\bar{z}_{a,t} = \int_{\mathbb{R}^n} \xi_t(z_a) W(\mathbf{z},\phi_t)\, p_t(\mathbf{z})\mathrm{d}\mathbf{z} + \nu\left(\int_{\mathbb{R}^n}\int_{\mathbb{R}^n} \xi_t(z_a)\, p_t(\mathbf{z}')\, \upsilon(\mathbf{z}',\mathbf{z})\mathrm{d}\mathbf{z}'\mathrm{d}\mathbf{z}\right) + \mathcal{O}(\xi_t^4), \tag{B-7}$$

But since the effects of mutation are assumed to be unbiased, we have

$$\int_{\mathbb{R}^n}\int_{\mathbb{R}^n} \xi_t(z_a)\, p_t(\mathbf{z}')\, \upsilon(\mathbf{z}',\mathbf{z})\mathrm{d}\mathbf{z}'\mathrm{d}\mathbf{z} = 0. \tag{B-8}$$

Eq. (B-7) then reduces to

$$\Delta\bar{z}_{a,t} = \bar{z}_{a,t+1} - \bar{z}_{a,t} = \int_{\mathbb{R}^n} \xi_t(z_a) W(\mathbf{z},\phi_t)\, p_t(\mathbf{z})\mathrm{d}\mathbf{z} + \mathcal{O}(\xi_t^4), \tag{B-9}$$

which corresponds to the first term of the Price equation: the change in average trait value in a population is equal to the covariance between trait and fitness (Price, 1970; see eq. 3 of Frank, 1997).

Substituting eq. (B-1) into eq. (B-9), we obtain that the change in the mean of trait a is,

$$\Delta\bar{z}_{a,t} = \sum_{b=1}^{n} \sigma_{ab,t} s_{b,t}(\bar{\mathbf{z}}_t) + \frac{1}{2}\sum_{b=1}^{n}\sum_{c=1}^{n} \kappa_{abc,t} h_{bc,t}(\bar{\mathbf{z}}_t) + \mathcal{O}(\xi_t^4), \tag{B-10}$$

which depends on the skew,

$$\kappa_{abc,t} = \int_{\mathbb{R}^n} \xi_t(z_a)\xi_t(z_b)\xi_t(z_c)\, p_t(\mathbf{z})\mathrm{d}\mathbf{z}, \tag{B-11}$$

in the population at time period t (in line with e.g., eq. 8a of Wakano and Iwasa, 2013 and eq. A20 b of Débarre and Otto, 2016 in well-mixed populations; eq. 17 of Wakano and Lehmann, 2014 for the island model).

B.1.4 Dynamics of the phenotypic variance-covariance

By definition, the change in the (co)variance (within individuals) between two traits a and b over one time period is

$$\begin{aligned}\Delta\sigma_{ab,t} = \sigma_{ab,t+1} - \sigma_{ab,t} &= \int_{\mathbb{R}^n} (z_a - \bar{z}_{a,t+1})(z_b - \bar{z}_{b,t+1})\, p_{t+1}(\mathbf{z})\mathrm{d}\mathbf{z} - \sigma_{ab,t} \\ &= \int_{\mathbb{R}^n} \left(\xi_t(z_a)\xi_t(z_b) - \sigma_{ab,t}\right) p_{t+1}(\mathbf{z})\mathrm{d}\mathbf{z} - \Delta\bar{z}_{a,t}\Delta\bar{z}_{b,t}.\end{aligned} \tag{B-12}$$

Substituting eq. (B-5) into the above, we obtain

$$\begin{aligned}\Delta\sigma_{ab,t} = &\int_{\mathbb{R}^n} \left(\xi_t(z_a)\xi_t(z_b) - \sigma_{ab,t}\right) W(\mathbf{z},\phi_t)\, p_t(\mathbf{z})\mathrm{d}\mathbf{z} \\ &+ \nu\left(\int_{\mathbb{R}^n}\int_{\mathbb{R}^n} \xi_t(z_a)\xi_t(z_b)\, p_t(\mathbf{z}')\, \upsilon(\mathbf{z}',\mathbf{z})\mathrm{d}\mathbf{z}'\mathrm{d}\mathbf{z} - \sigma_{ab,t}\right) - \Delta\bar{z}_{a,t}\Delta\bar{z}_{b,t} + \mathcal{O}(\xi_t^5).\end{aligned} \tag{B-13}$$

The bracketed term in the second line of eq. (B-13), which captures the effects of mutations (from parents with phenotype $\mathbf{z}'$ to descendant with phenotype $\mathbf{z}$), can be simplified by first writing out the product of deviations in terms of parental phenotype as

$$\xi_t(z_a)\xi_t(z_b) = \xi_t(z'_a)\xi_t(z'_b) + (z_a - z'_a)(z_b - z'_b) + \xi_t(z_a)(z_b - z'_b) + \xi_t(z_b)(z_a - z'_a), \tag{B-14}$$

and second, by noting that since mutations are assumed to be unbiased, the covariance between parental phenotype and mutation effect is zero:

$$\int_{\mathbb{R}^n} \left(\xi_t(z_a)(z_b - z_b') + \xi_t(z_b)(z_a - z_a')\right) \nu(\mathbf{z}', \mathbf{z}) \mathrm{d}\mathbf{z} = 0. \tag{B-15}$$

Using eqs. (B-14)-(B-15), the effect of mutations in eq. (B-13) can then be written as

$$\begin{aligned} \int_{\mathbb{R}^n}\int_{\mathbb{R}^n} \xi_t(z_a)\xi_t(z_b) p_t(\mathbf{z}') \nu(\mathbf{z}', \mathbf{z}) \mathrm{d}\mathbf{z}' \mathrm{d}\mathbf{z} &= \int_{\mathbb{R}^n}\int_{\mathbb{R}^n} \left(\xi_t(z_a')\xi_t(z_b') + (z_a - z_a')(z_b - z_b')\right) p_t(\mathbf{z}') \nu(\mathbf{z}', \mathbf{z}) \mathrm{d}\mathbf{z}' \mathrm{d}\mathbf{z} \\ &= \sigma_{ab,t} + \sigma^{\mathrm{m}}_{ab}, \end{aligned} \tag{B-16}$$

where we have defined, $\sigma^{\mathrm{m}}_{ab} = \int_{\mathbb{R}^n} (z_a - z_a')(z_b - z_b') \nu(\mathbf{z}', \mathbf{z}) \mathrm{d}\mathbf{z}$, as the (co)variance in mutational effects on traits a and b. Substituting eq. (B-16) into eq. (B-13), we obtain that the change in the (co)variance between two traits a and b over one time period is

$$\begin{aligned} \Delta\sigma_{ab,t} &= \nu\sigma^{\mathrm{m}}_{ab} + \int_{\mathbb{R}^n} \left(\xi_t(z_a)\xi_t(z_b) - \sigma_{ab,t}\right) W(\mathbf{z}, \phi_t) p_t(\mathbf{z}) \mathrm{d}\mathbf{z} - \Delta\bar{z}_{a,t}\Delta\bar{z}_{b,t} + \mathcal{O}(\xi_t^5) \\ &= \nu\sigma^{\mathrm{m}}_{ab} + \int_{\mathbb{R}^n} \xi_t(z_a)\xi_t(z_b) \left(W(\mathbf{z}, \phi_t) - 1\right) p_t(\mathbf{z}) \mathrm{d}\mathbf{z} - \Delta\bar{z}_{a,t}\Delta\bar{z}_{b,t} + \mathcal{O}(\xi_t^5), \end{aligned} \tag{B-17}$$

where to go from the first to the second line, we have used the fact that mean lineage fitness is one: $\int_{\mathbb{R}^n} W(\mathbf{z}, \phi_t) p_t(\mathbf{z}) \mathrm{d}\mathbf{z} = 1$ (since the population size is constant).

Substituting eq. (B-1), and the change in mean, eq. (B-10), into eq. (B-17), we obtain after some re-arrangements that the one-generational change in phenotypic (co)variance between traits a and b is

$$\begin{aligned} \Delta\sigma_{ab,t} = \nu\sigma^{\mathrm{m}}_{ab} &+ \left(W(\bar{\mathbf{z}}, \phi_t) - 1\right)\sigma_{ab,t} + \sum_{c=1}^{n} \kappa_{abc,t} s_{c,t}(\bar{\mathbf{z}}_t) \\ &+ \frac{1}{2}\sum_{c=1}^{n}\sum_{d=1}^{n} \sigma_{abcd,t} h_{cd,t}(\bar{\mathbf{z}}_t) - \sum_{c=1}^{n}\sum_{d=1}^{n} \sigma_{ac,t}\sigma_{bd,t} s_{c,t}(\bar{\mathbf{z}}_t) s_{d,t}(\bar{\mathbf{z}}_t) + \mathcal{O}(\xi_t^5), \end{aligned} \tag{B-18}$$

which depends on the fourth central moment of the phenotypic distribution,

$$\sigma_{abcd,t} = \int_{\mathbb{R}^n} \xi_t(z_a)\xi_t(z_b)\xi_t(z_c)\xi_t(z_d) p_t(\mathbf{z}) \mathrm{d}\mathbf{z} \tag{B-19}$$

(in line with e.g., eq. 8b of Wakano and Iwasa, 2013 and eq. A24 b of Débarre and Otto, 2016 in well-mixed populations; eqs. B1-B8 of Wakano and Lehmann, 2014 for the island model with a single trait).

B.2 Closure assumptions

Finally, we close the dynamical system for the means and (co)variances (given by eqs. B-10 & B-18). We achieve this closure in two steps.

B.2.1 Normal closure

First, we assume that the phenotypic distribution, $p_t(\mathbf{z})$, is normal. Under this assumption, the skew in the phenotypic distribution is zero, $\kappa_{abc,t} = 0$, and the fourth central moments can be expressed in terms of the (co)variances, $\sigma_{abcd,t} = \sigma_{ab,t}\sigma_{cd,t} + \sigma_{ac,t}\sigma_{bd,t} + \sigma_{ad,t}\sigma_{bc,t}$. Substituting these relationships into eqs (B-10) and

(B-18), we obtain that the one-generational changes in means and covariances are respectively given by

$$\begin{aligned}\Delta\bar{z}_{a,t} &= \sum_{b=1}^{n}\sigma_{ab,t}s_{b,t}(\bar{\mathbf{z}}_t)+\mathcal{O}(\xi_t^4)\\ \Delta\sigma_{ab,t} &= \nu\sigma_{ab}^{\mathrm{m}}-\sum_{c=1}^{n}\sum_{d=1}^{n}\sigma_{ac,t}\sigma_{bd,t}s_{c,t}(\bar{\mathbf{z}}_t)s_{d,t}(\bar{\mathbf{z}}_t)+\frac{1}{2}\sum_{c=1}^{n}\sum_{d=1}^{n}\left(\sigma_{ac,t}\sigma_{bd,t}+\sigma_{ad,t}\sigma_{bc,t}\right)h_{cd,t}(\bar{\mathbf{z}}_t)+\mathcal{O}(\xi_t^5).\end{aligned} \tag{B-20}$$

In vector and matrix form, eq. (B-20) corresponds to eq. (3) of the main text, except that in eq. (B-20), the selection coefficients depend on time t (due to time-dependent relatedness coefficients, $r^{\circ}_{2,t}(\bar{\mathbf{z}}_t)$, $r^{\circ}_{3,t}(\bar{\mathbf{z}}_t)$, and $\partial r_{2,t}(\mathbf{z})/\partial z_a$). We get rid off of this dependency and finally achieve closure in the next section.

B.2.2 Quasi-equilibrium

Our second step to close the dynamical system eq. (B-20) is to assume that dispersal is strong enough (relative to selection) so that genetic associations between individuals within groups reach their steady-state values before any significant changes has occurred in the phenotypic distribution, $p_t(\mathbf{z})$, at the population level. This *quasi-equilibrium* assumption, which is frequently used in population genetic and social evolution theory (e.g., Kimura, 1965a, Nagylaki, 1993, Kirkpatrick et al., 2002, Roze and Rousset, 2005, Lehmann et al., 2007, Roze and Rousset, 2008) is in line with our assumption that selection is weak. It entails that we can evaluate $r^{\circ}_{2,t}(\bar{\mathbf{z}}_t)$, $r^{\circ}_{3,t}(\bar{\mathbf{z}}_t)$, and $\partial r_{2,t}(\mathbf{z})/\partial z_a$ in eqs. (B-3)-(B-4) at their quasi-equilibrium, i.e., we take the limits $\lim_{\tau\to\infty} r^{\circ}_{2,\tau}(\bar{\mathbf{z}}_t) = r^{\circ}_{2}(\bar{\mathbf{z}}_t)$, $\lim_{\tau\to\infty} r^{\circ}_{3,\tau}(\bar{\mathbf{z}}_t) = r^{\circ}_{3}(\bar{\mathbf{z}}_t)$, and $\lim_{\tau\to\infty}\partial r_{2,\tau}(\mathbf{z})/\partial z_a|_{\mathbf{z}=\bar{\mathbf{z}}_t} = \partial r_2(\mathbf{z})/\partial z_a|_{\mathbf{z}=\bar{\mathbf{z}}_t}$, while holding $p_t(\mathbf{z})$ constant (we thus denote by $r^{\circ}_2(\bar{\mathbf{z}}_t)$,$r^{\circ}_3(\bar{\mathbf{z}}_t)$, and $\partial r_2(\mathbf{z})/\partial z_a|_{\mathbf{z}=\bar{\mathbf{z}}_t}$, the steady-state values of neutral pairwise relatedness, neutral three-way relatedness, and the first-order perturbation of pairwise relatedness, respectively). Substituting these steady-states into the selection coefficients eqs. (B-3)-(B-4) (now independent of time so written as $s_a(\bar{\mathbf{z}}_t)$ and $h_{ab}(\bar{\mathbf{z}}_t)$), which are in turn substituted into eq. (B-20), we finally obtain the closed dynamical eqs. (3) of the main text.

Computing relatedness coefficients. Computing relatedness coefficients under neutrality (i.e., $r^{\circ}_2(\bar{\mathbf{z}}_t)$, $r^{\circ}_3(\bar{\mathbf{z}}_t)$), which is standard in population genetics, uses identity-by-descent arguments (e.g., Karlin, 1968, Rousset, 2004). When generations are non-overlapping (i.e., a Wright-Fisher life cycle), for example, the relevant relatedness coefficients for our approach are given by

$$\begin{aligned}r^{\circ}_2(\bar{\mathbf{z}}_t) &= \frac{(1-m)^2}{N-(N-1)(1-m)^2}\\ r^{\circ}_3(\bar{\mathbf{z}}_t) &= \frac{(1-m)^3(1+3(N-1)r^{\circ}_2(\bar{\mathbf{z}}_t))}{N^2-(N-1)(N-2)(1-m)^3},\end{aligned} \tag{B-21}$$

(e.g., eqs. 12a & 12b of Ohtsuki, 2010; see also Table 1 of Mullon et al., 2016 for the Moran model). Calculating the first-order effect of selection on pairwise relatedness, $\partial r_2(\mathbf{z})/\partial z_a$, however, is more complicated. Under the quasi-equilibrium assumption, a perturbation of genetic associations between individuals will depend on first-order perturbations of individual fitness and neutral relatedness coefficients (see Roze and Rousset, 2008 for a general treatment, in particular their eq. 67). So far, the first-order effect of selection on pairwise relatedness, $\partial r_2(\mathbf{z})/\partial z_a$, has been explicitly derived for two standard life-cycles, the semelparous Wright-Fisher life-cycle (in which all adults die after reproduction; see eq. 18 of Ajar, 2003 and eq. 28 of Wakano and Lehmann, 2014) and the iteroparous birth-death Moran life-cycle (in which a single adult dies after reproduction in each group;

see eq. 14 of Mullon et al., 2016). In both cases, the effect of selection on relatedness can be written as

$$\frac{\partial r_2(\mathbf{z})}{\partial z_a}\bigg|_{\mathbf{z}=\bar{\mathbf{z}}_t} = \kappa \frac{r_2^\circ(\bar{\mathbf{z}}_t)}{1-m}\Bigg[\left[1+(N-1)r_2^\circ(\bar{\mathbf{z}}_t)\right]\frac{\partial w^{\mathrm{P}}(\mathbf{z}_i,\mathbf{z}_{-i},\bar{\mathbf{z}}_t)}{\partial z_{i,a}}\bigg|_{\substack{\mathbf{z}_i=\bar{\mathbf{z}}_t\\ \mathbf{z}_{-i}=\bar{\mathbf{z}}_t}} + \left[2r_2^\circ(\bar{\mathbf{z}}_t)+(N-2)r_3^\circ(\bar{\mathbf{z}}_t)\right](N-1)\frac{\partial w^{\mathrm{P}}(\mathbf{z}_i,\mathbf{z}_{-i},\bar{\mathbf{z}}_t)}{\partial z_{j,a}}\bigg|_{\substack{\mathbf{z}_i=\bar{\mathbf{z}}_t\\ \mathbf{z}_{-i}=\bar{\mathbf{z}}_t}}\Bigg] \tag{B-22}$$

where $\kappa = 2$ for the Wright-fisher and $\kappa = 1$ for the Moran life cycle (watch out for an unfortunate typo in eq. 15 of Mullon et al., 2018, which has "$\kappa = N$" under the Moran life cycle).

C Individual-based simulations

We performed individual based simulations for a population composed of N_{d} groups, each populated by N individuals, using Mathematica 11.0.1.0 (Wolfram Research, 2016). Starting with a monomorphic population, we track the evolution of the multidimensional phenotypic distribution under the constant influx of mutations. Each individual $i \in \{1,\ldots,N_{\mathrm{d}}N\}$ is characterised by two traits $(z_{i,1}, z_{i,2})$. At the beginning of a generation, we calculate the fecundity f_i of each individual according to its traits and those of its neighbours (using eq. 14). Then, we form the next generation of adults by sampling N individuals in each group with replacement according to parental fecundity, but to capture limited dispersal, the fecundity of each individual from the parental generation is weighted according to whether or not they belong to the group on which the breeding spot is filled: if an individual belongs to the same group in which a breeding spot is filled, its weighted fecundity is $f_i(1-m)$, where m is the dispersal probability; if it belongs to another group, its weighted fecundity is $f_i m/(N_{\mathrm{d}}-1)$ (as a disperser is equally likely to reach any other group, it lands with probability $1/(N_{\mathrm{d}}-1)$ in a focal group). Once an individual is chosen to fill the breeding spot, it mutates with probability ν, in which case we add to parental values a perturbation that is sampled from a multivariate normal distribution with mean $(0,0)$ and variance-covariance matrix $\begin{pmatrix}\sigma^{\mathrm{m}}_{11} & \sigma^{\mathrm{m}}_{12}\\ \sigma^{\mathrm{m}}_{12} & \sigma^{\mathrm{m}}_{22}\end{pmatrix}$. The resulting phenotypic values are truncated to remain between 0 and 4. We repeat the procedure for a fixed number of generations (see Figures for parameter values).