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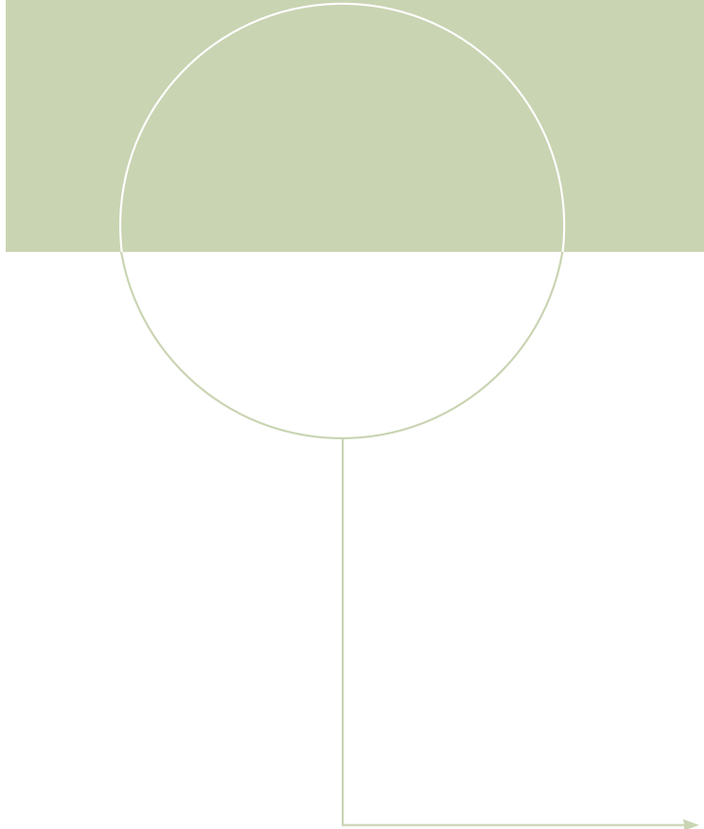

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The Role of Indirect Genetic Effects in Evolutionary Rescue

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The Role of Indirect Genetic Effects in Evolutionary Rescue

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Natural Resource Management (Biology)
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Abstract

Global environmental change is causing a rapid decline in biodiversity and associated ecosystem services. Understanding the mechanisms that affect a population's ability to adapt to environmental change is critical for informed conservation strategies, future research programs, and biodiversity forecasts, yet gaps remain between current theory and empirical observations. Theoretical and empirical studies indicate that indirect genetic effects, the causal effect of one individual's genotype on the phenotype of a conspecific, can alter the direction and pace of evolution, though they are often overlooked in the context of climate change adaptation. This thesis extends mathematical evolutionary rescue theory to explore how a directional interaction between two traits expressed by non-relatives can alter model predictions. Recognizing that multiple mechanisms can lead to adaptation, the model includes the potential for genetic correlation between traits and phenotypic plasticity in the focal trait. Relying on a combination of published estimates from wild populations and simulated data, we explore the conditions under which an interacting partner can counteract or exacerbate the demographic consequences imposed by directional environmental change. In the generalized model presented in this thesis, the indirect genetic effect altered base model predictions according to the interaction coefficient, ψ, and the interacting trait's environmental sensitivity of selection. Exploring the combined effect of IGEs and genetic correlation between interacting traits predictably altered model predictions, in some cases reversing the predicted outcome. This study is a first step to explore how evolutionary change in the social environment created by conspecifics can contribute to the evolutionary rescue or extinction of a population. This study also highlights the need for empirical estimates of key parameters in this model to assess the real importance of social interactions in the susceptibility of populations to environmental change.

Sammendrag

Globale miljøendringer fører til en kraftig nedgang i biodiversitet og de tilhørende økosystemtjenestene. Det å forstå mekanismene som påvirker en populasjons evne til å tilpasse seg disse miljøendringene er kritisk kunnskap for å kunne fatte kunnskapsbaserte bevaringsstrategier, fremtidig forskning og biodiversitet trender/prognoser, men fortsatt eksisterer det et gap mellom nåværende teori, og observasjoner. Teoretiske og empiriske studier tyder på at indirekte genetiske effekter, den kausale effekten av et individs genotype på fenotypen til et annet individ, kan endre retningen og evolusjonshastigheten, selv om de ofte blir oversett i sammenhengen mellom klimaendringer og tilpasninger til klimaendringer. Denne masteroppgaven viderefører matematikken bak teorien om evolusjonær unnsetning til å utforske hvordan et retningsbestemt samspill mellom to egenskaper uttrykt mellom ikke-beslektede individer kan endre forutsigelsene for modellen. Modellen anerkjenner at flere mekanismer kan føre til tilpasninger, og inkluderer derfor en mulig kovarians mellom direkte- og indirekte genetiske effekter, og fenotypisk plastisitet hos egenskaper i fokus. Ut fra en kombinasjon av publiserte estimater fra ville populasjoner og simulert data, utforsker vi måtene et samhandlende individ kan motvirke eller forverre de demografiske konsekvensene påført av deterministiske miljøendringer. I den generaliserte modellen presentert i denne oppgaven, endret den indirekte effekten prediksjonene til grunnmodellen i henhold til interaksjonskoeffisienten, og samspillet mellomegenskapenes følsomhet overfor seleksjonsmiljøet. Utforsking av den netto effekten fra flere mekanismer endret prediksjonene til modellen, som forutsatt. I visse tilfeller reverserte det også det forutsette utfallet. Dette studiet er et første steg på veien til å utforske hvordan evolusjonære endringer i et sosialt miljø skapt av ikke-beslektede individ kan bidra til den evolusjonære unnsetningen eller uttryddelsen av en populasjon. Skjønt, empiriske estimat av viktige parameter er nødvendig.

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Contents

1 Introduction

Land-use change, pollution, and rising CO_2 emissions are driving rapid declines in biodiversity and associated ecosystem services (Butchart et al., 2010; Dirzo et al., 2014; Parmesan & Yohe, 2003; Pimm et al., 2014). Understanding the mechanisms that underlie patterns of adaptation and extinction in response to rapid environmental change has important implications for conservation strategies, research programs, and global environmental targets. Instances of rapid evolution in nature (Carroll et al., 2007; Gingerich, 2009; Hendry & Kinnison, 1999) suggest that populations can locally adapt to changing environmental conditions, leading to their evolutionary rescue (Gonzalez et al., 2013; Hoffmann & Sgrò, 2011). Current knowledge in this area is based largely upon phenological and life-history traits in birds owing to long-term observational studies. Yet, even in these better-studied examples, gaps remain between current theory and its ability to describe observed patterns in the wild (Charmantier & Gienapp, 2014; Gienapp et al., 2008; Merilä et al., 2001). One possibility that often remains overlooked is the effect of social interactions on the evolutionary potential of populations and thus in their ability to adapt to environmental change.

Quantitative genetic theory can be used to study how, among conspecifics, the genotype of one individual can affect the trait expression of another (Griffing, 1967; Moore et al., 1997; Wolf et al., 1998). This indirect genetic effect (IGE) acts as a heritable component in the social environment of the focal individual and can alter the direction and pace of evolution (McGlothlin et al., 2010; Wolf et al., 1998). To understand how social interactions may contribute to population persistence, we must integrate quantitative genetic theory with population dynamics. Extending prior mechanistic models (Bürger & Lynch, 1995; Chevin et al., 2010; Lynch & Lande, 1993), we explore the role of IGEs in the evolutionary rescue of wild populations.

Social environments are an important determinant of phenotypic variation. Quantifying the genetic basis of social environments is key to understanding the total genetic variation in a trait and its potential to respond to selection. IGEs are perhaps most commonly studied in breeding programs to increase production and improve animal welfare (reviewed in Ellen et al., 2014). IGEs are expected in a range of behavioural traits from cooperation to aggression (reviewed in Bailey et al., 2018) and are a key

component of maternal effects theory, where transgenerational maternal genetic effects impact the offspring phenotypes (reviewed in McAdam et al., 2014; Rasanen & Kruuk, 2007). It is surprising that IGEs between conspecifics remain largely overlooked in the context of environmental change. Empirical estimates in this area are generally sparse, although IGEs have been detected in traits affecting disease response (Anacleto et al., 2019; Baud et al., 2017; Lipschutz-Powell et al., 2012), thermal tolerance (Mũnoz et al., 2014), breeding phenology (Brommer et al., 2015; Brommer & Rattiste, 2008; Gienapp et al., 2013; Teplitsky et al., 2010), range expansions (J. K. Bailey et al., 2014; Duckworth, 2009), and community structure (interspecific IGEs: Shuster et al., 2006; Whitlock et al., 2011). While generality regarding the magnitude of IGEs and their effect on evolution depends on further empirical estimates, accumulating evidence suggests that we are overlooking an important mechanism driving adaptive evolution.

Evolution and demography are dynamically linked. Rapid evolution (or lack thereof) in response to altered selection pressures interacts with population demography to affect overall abundance and ultimately, population survival. Evolutionary rescue provides a framework to study the ability of rapid evolution to 'rescue' a population from negative demographic effects of environmental change (Gonzalez et al., 2013). Theoretical models illustrate how various mechanisms can contribute to the evolutionary rescue of populations (as reviewed in Gonzalez et al., 2013; Hoffmann & Sgrò, 2011; Kopp & Matuszewski, 2014). For example, phenotypic plasticity can accelerate the rate of adaptation (Chevin et al., 2010; Gienapp et al., 2013; Lande, 2009; Vedder et al., 2013), genetic correlations can bias the direction of adaptation (Hellmann & Pineda-Krch, 2007; Walsh & Blows, 2009) and social interactions between evolving species may disrupt predictions on the stability of demographic rates on a community level (Van Den Elzen et al., 2017; Yamamichi & Miner, 2015). However, each of these studies makes the standard quantitative genetic assumption that the social environment is not heritable.

Given the ubiquity of social interaction in nature, a generalized model of evolutionary rescue should account for the additional genetic variation arising through IGEs. We focus on the scenario where the IGE is a directional interaction between non-relatives. The model is based on prior theory by Chevin et al. (2010), which is an extension of an earlier model by Lynch and Lande (1993). The focus of this work is threefold:

(1) to derive a mechanistic model that integrates quantitative genetic theory of IGEs and population demography; (2) to illustrate potential bias in predicted thresholds of extinction when IGEs are not considered; and (3) to explore the interaction between IGEs and other mechanisms likely to occur simultaneously in the wild.

1.1 Modelling Indirect Genetic Effects

Under standard quantitative genetic theory, a trait (z) is partitioned into a heritable additive genetic effect (the 'breeding value', a) and a random, non-heritable environmental effect (e) where $z = a + e$. When the phenotype of an individual is influenced by its social environment, the environment can be further partitioned such that:

$$z = a + e + e_{social} \quad , \tag{1}$$

where e_{social} defines the social environment created through intraspecific interactions (Moore et al., 1997). There are two statistical frameworks for modelling the effect of IGEs on the phenotype of a focal individual: a trait-based approach and a variance-partitioning approach. The two methodologies are complementary, but they differ in the parameters they estimate and their empirical requirements. A more comprehensive review is provided by McGlothlin and Brodie III (2009) and Bijma (2014).

Trait-Based Framework

Under a trait-based approach, the social environment is defined by 'interacting phenotypes' (Moore et al., 1997). Assuming a pairwise interaction, (1) can be re-written:

$$z_f = a_f + e_f + \psi z_i \quad , \tag{2}$$

where subscripts f and i indicate parameters associated the focal and interacting social partners, respectively. ψ is an interaction coefficient that quantifies the causal effect of the interacting trait on the focal trait. ψ may also be referred to as social plasticity or social responsiveness (Dingemanse & Araya-Ajoy, 2015). Higher values of ψ are expected when the focal trait is highly responsive to the social environment. When the social trait affects the focal trait, but not the reverse, ψ can be estimated as a

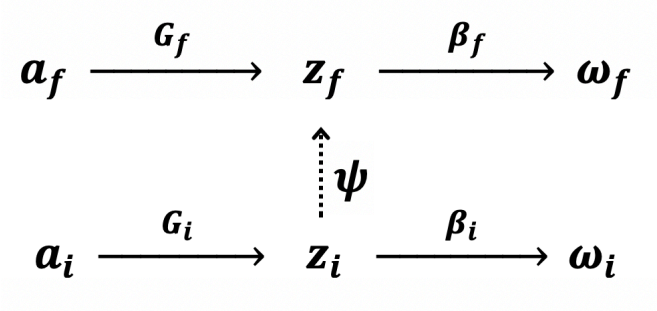

Figure 1: Path diagram illustrating how an indirect genetic effect (IGE) alters the genotype-phenotype-fitness map of a focal individual. Subscripts f and i denote focal and interacting partners, respectively. Solid arrows represent the direct genetic effect (DGE) of an individual's genes (a) on its own phenotype (z) and fitness (ω). The additive genetic variance (G) measures the covariance between the additive genetic effect (a) and phenotypic trait value (z). The directional selection gradient (β) describes the covariance between z and relative fitness, standardized by the total phenotypic variance. The interaction coefficient ψ (vertical dotted arrow) provides a pathway by which genes in the interacting trait can affect the focal trait.

regression coefficient or the standardized slope of the focal phenotype on the interacting phenotype. Traits can also have a reciprocal effect on each other within the span of an interaction, in which case the causal relationship between focal and interacting trait is subject to feedback (Moore et al., 1997; Wolf et al., 1998). Interaction coefficients can also be extended to include group effects (McGlothlin & Brodie III, 2009).

A non-zero interaction coefficient can alter the traditional mapping between genotype and phenotype. The interacting phenotype can itself be partitioned into genetic and environmental effects, where (2) can be re-written as:

$$z_f = a_f + e_f + \psi(a_i + e_i) \quad . \tag{3}$$

When the social trait has a heritable component, ψ provides a pathway by which the genotype of an interacting trait affects the phenotype of a focal partner (Figure 1). The expected evolutionary change in the mean phenotype is now a function of selection on its own genes, plus the response to selection on the social trait, mediated by ψ. Thus, in a trait-based method, the IGE depends on both non-zero ψ and genetic variance in the interacting trait, z_i.

A strength of the trait-based method is its explicitness about the trait(s) that mediate(s) the social interaction. However, a drawback for empiricists is that it requires

complete knowledge of the causal traits.

Variance-Partitioning Framework

IGEs can be also be modelled using a variance-partitioning approach, aptly named because it partitions the phenotypic variance of a focal trait into a direct and indirect genetic component. The social environment can be considered in terms of 'associative effects' rather than explicitly modelling the affecting traits (Griffing, 1967). Using the variance-partitioning framework, (1) can be extended such that:

$$z = a_D + e_D + a_A + e_A \tag{4}$$

where the trait value is a function of a direct genetic effect a_D from the genes of the focal individual, an indirect genetic effect a_A from the genes of the focal individual's social partners (or associates), and direct e_D and indirect e_A non-heritable environmental effects (Bijma, 2011; Griffing, 1967). Parameters in the trait-based framework are estimated using an extended version of the animal model that requires a multidimensional pedigree and associations between interacting individuals (Wilson et al., 2010). The variance-partitioning approach can easily accommodate group-wise interaction (Bijma, 2011), but it does not provide information on the direction or form of the IGE.

The variance-partitioning framework can be used to estimate the relative importance of IGEs, but it does not provide information on the direction or form of ψ. It is a popular method among empiricists when causal trait(s) may not be known.

1.2 Predicting Rates of Evolution

The multivariate breeder's equation is a foundational model often used to calculate the expected response of a fitness-related trait to selection under climate change. It describes the expected rate of phenotypic change from one generation to the next as a function of the additive genetic variance in the trait (G) scaled by the selection gradient (β), where $\Delta\bar{z} = G\beta$ (horizontal path: Figure 1). The expected change in the mean phenotype can be compared against the rate of climate change to infer whether the population has sufficient adaptive potential to track its environment (Hoffmann & Sgrò, 2011).

Adopting a trait-based approach, a non-zero interaction coefficient provides a pathway by which the response to selection in a focal trait is affected by its social environment (Figure 1). In this case, the expected phenotypic rate of change in the focal trait is a function of both DGEs and IGEs where:

$$\Delta \bar{z}_f = G_f \beta_f + \psi G_i \beta_i \tag{5}$$

(Moore et al., 1997; Wolf et al., 1998). Provided that $\psi \neq 0$, the IGE generates an additional source of genetic variation in z_f such that $G_f > 0$ is no longer a prerequisite for evolution. To illustrate how IGEs may affect the direction and pace of evolution, and thus the ability of populations to cope with climate change, we parameterize (5) using estimates from onset of breeding in wild bird populations.

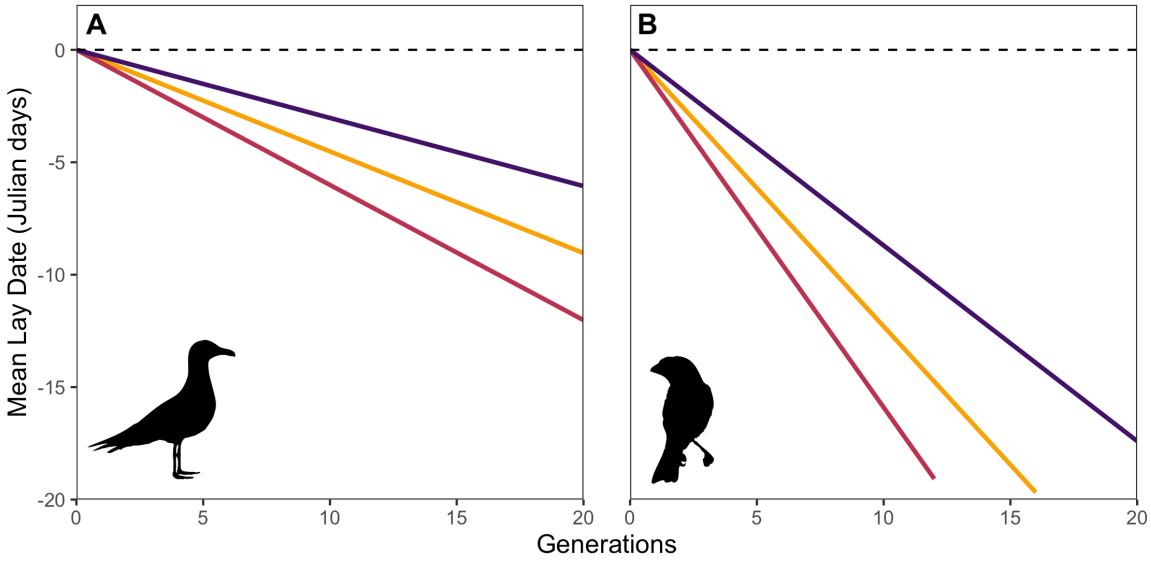

Figure 2: Naive quantitative genetic predictions of expected per-generation change in mean lay date in two wild bird populations: (A) the common gulls (*Larus canus*, Estonia) $G_f = 4.52$, $G_i = 1.49$ (Brommer & Rattiste, 2008); (B) song sparrows (*Melospiza melodia*, Canada) $G_f = 12.30$, $G_i = 3.60$ (Germain et al., 2016). Baseline predictions when $\psi = 0$ are indicated by the orange line. The pink line shows the predicted rate of change selection is convergent: $\psi = 2, \beta_f = -0.1, B_m = -0.1$. The purple line shows the predicted rate of change when selection is antagonistic: $\psi = 2, \beta_f = -0.1, B_m = 0.1$. The dashed horizontal line shows observed rate of change in mean lay date in each population.

Example: Avian Onset of Breeding

Onset of breeding in birds is an important life-history trait closely linked to fitness that often involves a social element if only due to the effects of male partners (Visser, 2008). Among avian species, egg-laying date is heritable with selection often favouring earlier lay dates in response to recent climate change. Advanced lay dates have been observed in natural populations (Both et al., 2006; Charmantier et al., 2008), but this observed shift often differs from predicted rates of microevolutionary change (Charmantier & Gienapp, 2014). In the case of avian lay dates, an IGE has been suggested to result from male provisioning of resources or securing of a territory used by the female (Brommer et al., 2015; Brommer & Rattiste, 2008). For example, males that provide more food or initiate earlier courtship could advance their female's lay date.

Despite lay date being a sex-limited trait, (2) shows how an IGE from a male breeding partner can bias the predicted rate of evolutionary change of the lay date expressed by females. Assuming a positive interaction coefficient, selection on the male IGE can increase the expected change of the female phenotype when it is convergent with selection on lay date, and can reduce the expected rate of phenotypic change when selection is antagonistic. Across study populations, the conventional model $\psi = 0$ is overly optimistic compared to the observed rate of change, suggesting that an evolutionary constraint could be preventing these populations from tracking their fitness optima. At least theoretically, IGEs that restrain the evolution of lay date could be one such source of constraint. However, these naive estimates do not account for different life histories and demographic characteristics of the populations, nor do they consider the effect of phenotypic plasticity in reproductive timing.

2 A General Model of Evolutionary Rescue

We now shift towards a mechanistic model that integrates quantitative genetic theory of IGEs with population demography to predict the potential for environmental rescue in a directionally changing environment. Consistent with earlier models we assume a large, isolated sexual population with non-overlapping generations that experiences neither genetic drift nor mutation (Bürger & Lynch, 1995; Chevin et al., 2010; Lynch & Lande, 1993). The environment is modelled via a single environmental parameter that changes linearly at a constant rate, η. The environmental parameter is often used to model global climactic phenomena (e.g. warming spring temperature), though it is equally applicable to deterministic local conditions (e.g. increasing salinity from winter runoff). Population persistence depends on a continuous, fitness-related trait (e.g. reproductive timing) tracking its phenotypic optimum, which changes proportionally with the environment. The potential for evolutionary rescue depends on the critical rate of environmental change predicted by the model. This represents an 'extinction threshold' (Lynch et al., 1991) beyond which the population cannot keep pace with the changing environment and the resulting increase in maladaptation is demographically unsustainable. By comparing this critical rate of environmental change under different scenarios and parameter values, we can evaluate the conditions that mitigate or intensify the threat of population extinction. This general framework can also be used as a heuristic tool to expand our understanding of the mechanisms underlying local adaptation to environmental change.

2.1 Base Model

Linking evolution with population dynamics requires a shift from thinking about evolution in terms of relative fitness (survival of the fitt*est*) to its consequences in terms of absolute fitness (Gonzalez et al., 2013; Kopp & Matuszewski, 2014). The mean absolute fitness of the population ($\bar{W}$) is the number of offspring in the next generation, that will be affected by survival, fecundity and mating success in the parent generation. $\bar{W} = 1$ indicates a stable population size where the parent generation is just replacing itself, $\bar{W} < 1$ indicates negative growth and $\bar{W} > 1$ indicates positive growth.

Mean population fitness can be derived by integrating over phenotypic and fitness

functions, both of which are assumed to be Gaussian (Lynch & Lande, 1993). For a deterministic environment, the maximum number of offspring produced in the next generation is reduced primarily by two fitness loads (Chevin, 2013). First is the 'lag load', defined by the deviation between the mean phenotype and optimum phenotype that maximizes fitness (Lande & Shannon, 1996; Maynard Smith, 1976). The lag load measures the maladaptation in the population, with mean population fitness decreasing non-linearly with an increasing lag load. Second is the 'standing load' that results from selection-induced mortality around the optimum (Lynch & Lande, 1993). It is inversely proportional to the genetic variance; the greater the variance in the distribution of phenotypes, the higher the number of individuals that will be selected against. A graphical explanation of the fitness loads is provided in Kopp and Matuszewski (2014). Both fitness loads are affected by the underlying additive genetic variance in the phenotype. Higher genetic variance allows the population to track its optimum more closely, reducing the lag load, but it also increases the proportion of individuals that will deviate from the optimum, increasing the standing load.

Selection must also be defined in terms of absolute fitness. When the environment changes, the strength of selection depends on the deviation between the population mean phenotype and the optimum at a particular point in time. The linear selection gradient can be defined relative to a shifting phenotypic optimum, θ:

$$\beta = \frac{\delta ln\bar{W}}{\delta \bar{z}} = \gamma(\bar{z} - \theta) \quad , \tag{6}$$

where $\gamma = 1/\omega^2 + \sigma_z^2$ is stabilizing selection, which is inversely proportional to the width of the fitness function ω^2 and the phenotypic variance σ_z^2 (Lande, 1979). The strength of selection is thus directly proportional to the deviation between the mean phenotype and the optimum, and inversely proportional to the width of fitness and phenotypic functions.

The population is initially well adapted such that $\bar{z} = \theta$. As the environment changes, selection increases as the the deviation between the mean phenotype and the optimum widens. Evolutionary theory predicts that the population eventually reaches a steady-state equilibrium lag (Lynch & Lande, 1993) where the rate of change in the population mean phenotype matches the rate of change of the phenotypic optimum ($\Delta\bar{z}/T = B\eta$),

otherwise it will go extinct. Here, B is referred to as a trait's environmental sensitivity of selection, which reflects how a change in the environment affects the optimum (Chevin et al., 2010), and η is the linear rate of change in the environment.

Assuming density independence on the basis that maladapted populations are unlikely to reach their carrying capacity, the population size N at time $t+1$ can be defined as the product of mean population fitness $\bar{W}$ and population size at time t, where $N(t+1) = \bar{W}N(t)$ (Chevin et al., 2010; Lynch & Lande, 1993). The population growth rate can be defined relative to the equilibrium lag:

$$ln\bar{W} = r_{max} - \frac{\gamma(\bar{z} - \theta)^2_{eq}}{2T} \quad , \tag{7}$$

where r_{max} is the maximum rate of intrinsic growth when every member of the population expresses the optimum phenotype, $(\bar{z} - \theta)_{eq}$ is the equilibrium lag and remaining parameters are as defined above. If the population loses pace with its shifting optimum, the lag load increases such that $\bar{W} < 1$ (or $ln\bar{W} < 0$). The maximum rate of environmental change that will allow for population persistence can be solved by substituting the equilibrium lag into (7) and setting $ln\bar{W} = 0$.

Chevin et al. (2010) use this above framework to model partially adaptive plasticity in a scenario of evolutionary rescue to predict the critical rate of environmental change:

$$\eta_c = \sqrt{\frac{2r_{max}\gamma}{T}}\frac{G}{|B - b|} \quad . \tag{8}$$

r_{max} is the maximum rate of intrinsic growth when every member of the population expresses the optimum phenotype and can be extended to include the costs associated with the cognitive and physiological machinery required for plasticity (eq. 2 in Chevin et al., 2010). T is population generation time in years. Generation time and maximum intrinsic growth rate have opposing effects on the extinction threshold where a higher generation time (e.g. age to sexual maturity) decreases the critical rate of environmental change, all else being equal, because higher rates of microevolution are required to keep pace with an environment that can change markedly between birth and age of sexual maturity (Chevin et al., 2010). γ is the strength of stabilizing selection, which combined with G, the additive genetic variance, determines the rate of microevolution. B is the environmental sensitivity of selection that measures how the

optimum phenotype changes with the environment and b is a linear reaction norm slope of the phenotype against the environment. The difference between these two slopes $|B - b|$ determines how much microevolution is required to track the environment.

2.2 Model Extension

We extend current evolutionary rescue theory by re-defining the expected change in the mean focal phenotype as a function of both direct- and indirect genetic effects (McGlothlin et al., 2010), including the possibility for genetic covariance as traits expressed by the population will be encoded on a shared genome. We consider a simple scenario where the interacting phenotype can exert an IGE on the focal trait, but not the reverse (a nonreciprocal, pairwise IGE; Moore et al., 1997). For instance, the focus could be the lay date of a female bird that is affected by her mate's ability to acquire a territory early in the breeding season. We use the term 'focal' to reflect the trait that is subject to the IGE (e.g. lay date) and 'interactant' to reflect the social partner's trait (e.g. territory acquisition).

Phenotypic Change

The trait of the social partner is a function of its direct genetic effect (DGE) and it may have a shared genetic basis with the focal trait, but it is not affected by IGEs. The expected generational rate of change in the mean phenotype of the interacting partner is consistent with other DGE models of correlated traits, where:

$$\frac{\Delta \bar{z}_i}{T} = \frac{G_i \beta_i + G_{fi} \beta_f}{T} \tag{9}$$

(Lande, 1979). The first composite term captures the direct genetic response to selection on the interacting trait as a function of the genetic variance in the interacting phenotype (G_i) and the selection gradient (β_i). The second composite term reflects how selection on the focal phenotype affects the interacting phenotype when traits genetically covary, where $G_{fi} = \rho\sqrt{G_f G_i}$ is the genetic covariance, G_f is the genetic variance in the focal trait, β_f is the directional selection gradient and ρ is the genetic correlation. We assume that there is no plasticity in the interacting trait.

Using a trait-based approach, we define the generational rate of change in the focal phenotype relative to the interacting phenotype, where:

$$\frac{\Delta \bar{z}_f}{T} = \frac{G_f \beta_f + G_{fi} \beta_i + \psi \Delta \bar{z}_i + b\eta T}{T} \quad . \tag{10}$$

Similar to (9), the first two terms capture the genetic response to direct selection and indirect selection through the genetic covariance, respectively. The third term is the IGE on the focal phenotype, which depends on the per-generation change in the interacting trait expressed by the social partner defined in (9), scaled by the coefficient of interaction, ψ. The fourth term accounts for the degree of phenotypic plasticity in the focal trait, where b is the slope of a linear reaction norm. Substituting (9) into (10), the equation can be re-written to explicitly show how evolution of the interacting trait affects evolution of the focal trait:

$$\frac{\Delta \bar{z}_f}{T} = \frac{G_f \beta_f + G_{fi} \beta_i + \psi (G_i \beta_i + G_{fi} \beta_f) + b\eta T}{T} \quad . \tag{11}$$

Equations (9-11) apply when all members in the population express both focal and interacting traits. The equations can accommodate sex-limited trait expression by scaling β_i and β_f by $\frac{1}{2}$ to account for the genetic contribution of each sex. Coming back to the example of female lay date, the evolutionary change in the average female lay date in the population will be a function of the direct selection on lay date, and selection on male's timing of territory establishment, modulated by the genetic correlation between the traits and the interaction coefficient, ψ.

Steady-State Equilibrium

Traits may differ in their respective sensitivity to the rate of environmental change due to differential trade-offs between fecundity, survival and mating success. This allows focal and interacting traits to track the same environmental parameter at different rates, potentially in different directions. For instance the change in optima due to environmental change for a male's timing to acquire a territory will be different compared to the female's optimal lay date, because of the survival costs associated to territorial defense. In this scenario, the steady-state equilibrium of the focal trait depends on the equilibrium of the interacting trait. Assuming that both focal and social partners have

sufficient evolutionary potential to reach equilibrium, and that neither perfectly tracks their respective optimum, the focal equilibrium lag is given by:

$$(\bar{z}_f - \theta_f)_{eq} = \frac{\eta T}{G_f \gamma_f (1-\rho^2)} (B_f^* - \psi B_i - \frac{G_{fi}}{G_i} B_i) \quad , \tag{12}$$

where $B_f^* = B_f - b$ is the environmental sensitivity of the focal trait after accounting for plasticity. From (12), the focal equilibrium lag does not depend directly on the genetic parameters of the social partner because it is assumed that the rate of phenotypic change is such that $\Delta \bar{z}_i = B_i \eta$. Thus, the interacting trait's environmental sensitivity of selection mediated by ψ determines the effect of the IGE at equilibrium.

Threshold for Extinction

The magnitude of the focal equilibrium lag affects the growth rate of the population according to (7). Assuming that population mean fitness is dependent on adaptation in the focal trait, the maximum rate of environmental change that can sustain positive growth is given by:

$$\eta_c = \sqrt{\frac{2 r_{max} \gamma_f}{T}} \frac{G_f (1-\rho^2)}{\left| B_f^* - B_i \left(\psi + \frac{\rho \sqrt{G_f G_i}}{G_i} \right) \right|} \quad , \tag{13}$$

where η_c is the critical rate of environmental change at the junction between population persistence ($\eta < \eta_c$) and extinction ($\eta > \eta_c$) in units of phenotypic measurement per year. The equation can also apply to sex-limited traits by scaling η_c by $\frac{1}{2}$. Model derivations are provided in Appendix I.

Equation (13) provides a general framework to model scenarios of evolutionary rescue in which IGEs, genetic correlations and/or phenotypic plasticity may be relevant. Setting parameters such that $\psi = 0$ and $\rho = 0$, (13) simplifies to (8). Equation (13) can serve as a generalized model that includes a potentially important missing link, that of the social environment.

3 Numerical Evaluations

Indirect Genetic Effects

ψ provides a pathway by which adaptive evolution in the social environment of a focal individual can counteract or intensify the demographic consequences of climate change. When the focal trait is not responsive to an interacting phenotype ($\psi = 0$), the social trait's response to environmental change does not affect evolutionary rescue. When $\psi \neq 0$, the rate and direction of evolution in the social trait affects the lag load according to the magnitude and direction of the interaction coefficient, ψ, and the difference between the focal and social trait's environmental sensitivities of selection, B. $B_f B_i > 0$ is analogous to a scenario of convergent selection where trait optima shift in the same direction (Figure 3-A) and $B_f B_i < 0$ reflects a scenario of antagonistic selection where focal and interacting trait optima shift in different directions (Figure 3-B).

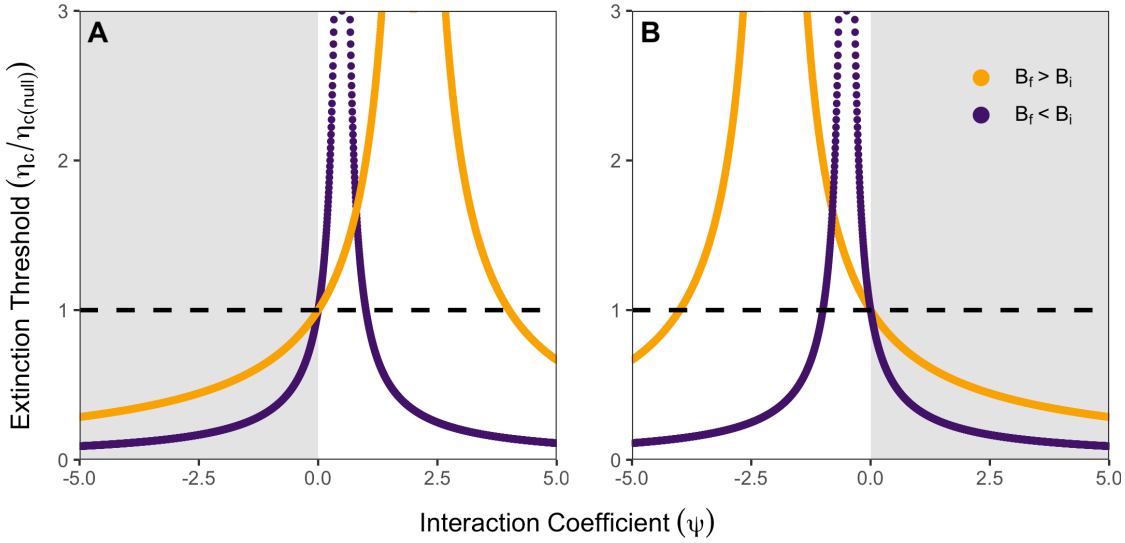

Figure 3: The effect of the interaction coefficient (ψ) and environmental sensitivity (B) on the standardized extinction threshold ($\eta_c/\eta_{c(null)}$) when focal and interacting trait optima are (A) convergent, $B_f B_i > 0$; and (B) antagonistic, $B_f B_i < 0$. The predicted extinction threshold is scaled by a null model ($\psi = 0, \rho = 0, b = 0$) such that the results are independent of input values for r_{max}, T, γ_f and G_f. The horizontal dashed line indicates model equality ($\eta_c/\eta_{c(null)} = 1$). The IGE model predicts a higher threshold for environmental change than the null model when points fall above the dashed line and a lower threshold for environmental change when points fall below the dashed line.

IGEs cause a non-linear decrease in the predicted threshold for extinction with increasing ψ in the opposite direction of $B_f B_i$ (grey parameter space, Figure 3), such as when selection is convergent and ψ is negative (panel A) or when selection is antagonistic and ψ is positive (panel B). In either case, evolution in the interacting trait shifts the focal trait away from its phenotypic optimum and increases the lag load. The higher the interaction coefficient, the higher the predictions deviate from a model that does not consider IGEs, herein a 'null' model (points below the dashed line, Figure 3).

However, ψ in the direction of $B_f B_i$ does not necessarily mean the extinction threshold will increase (white parameter space). These scenarios include convergent selection and positive ψ (panel A) or antagonistic selection and negative ψ (panel B). In both cases, the IGE shifts the focal trait in the direction of its phenotypic optimum which can reduce the lag load compared to a null model (points above the dashed line), but can also shift the focal mean phenotype to the other side of its fitness peak, which increases the deviation with the focal optimum from the other direction and, can potentially increase the lag load beyond a null model (points below the dashed line).

Demographic and Genetic Constraint

Predictably, the IGE has a non-linear effect on the threshold for extinction where lower deviations between $B_f - \psi B_i$ allow the population to track faster rates of environmental change (higher extinction threshold). In the absence of genetic correlation and phenotypic plasticity, changes in the interactant phenotype drive the focal trait towards its optimum (Figure 4). The effect of the IGE on population persistence ultimately depends on the genetic and demographic characteristics of the population.

In Figure 4, the predicted extinction threshold is shown for two mammals at different ends the slow-fast continuum of life-history traits based on demographic parameters from the literature and simulated levels of genetic variance. The elephant represents a 'slow' species characterized by lower fecundity (r_{max}) and higher generation time (T) while the mouse represents a 'fast' species characterized by higher fecundity and lower generation time. In general, 'fast' species can adapt to higher rates of change (elevation of inflections points between plots: Figure 4) and are less sensitive to changes in the deviation between $B_f - \psi B_i$ (line curvature between plots: Figure 4). Similarly, populations with higher genetic variance can tolerate higher rates of environmental

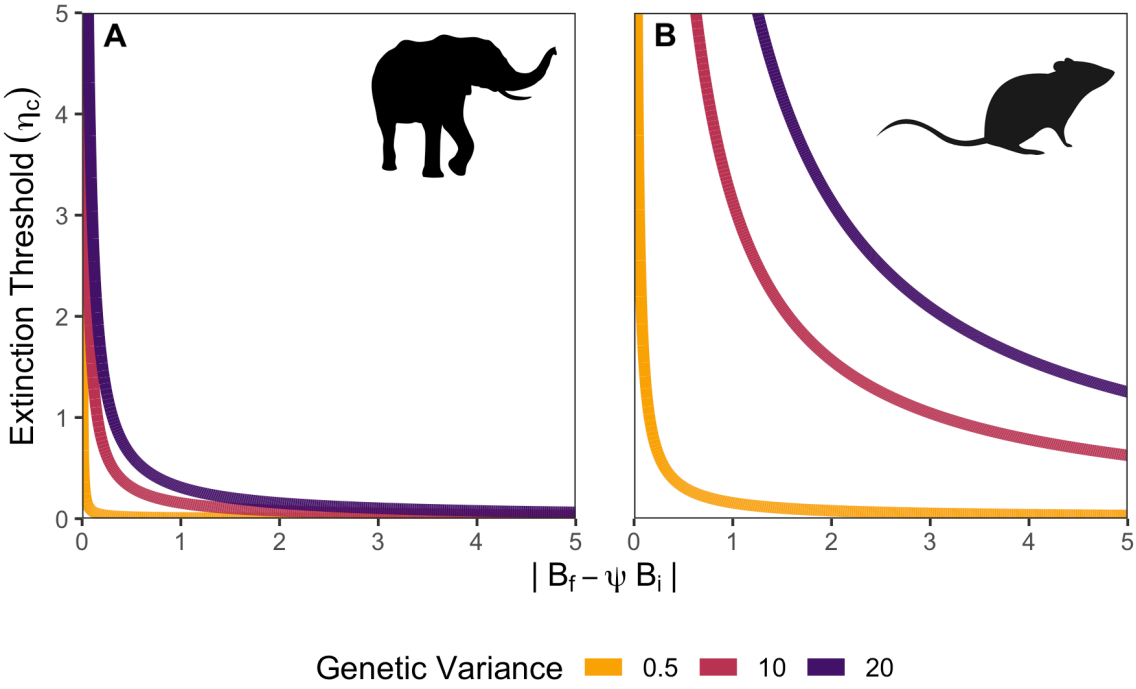

Figure 4: The effect of IGEs, direct genetic variance and population demography on the extinction threshold (η_c). The x-axis shows the deviation between the focal environmental sensitivity of selection and the effect of the IGE ψB_i. Colour indicates direct additive genetic variance in the focal trait. Panels differentate between demographic characteristics of two species: (A) an elephant (*Loxodonta* sp.): $r_{max} = 0.06$, $T = 10$ (Beeby & Brennan., 2008); and (B) a rodent (*Leporillus conditor*): $r_{max} = 1.47$, $T = 0.6$ (Hone & Forsyth, 2010). $\gamma = 0.02$ was used throughout all simulations.

change (elevation of the inflection point within plots: Figure 4). Populations with higher genetic variation are also less sensitive to changes in the deviation between $B_f - \psi B_i$ (change in line curvature within each plot: Figure 4).

Phenotypic Plasticity

Plasticity is expected to affect the adaptive potential of many traits impacted by climate change and its role in evolutionary rescue has been addressed in other theoretical studies (Chevin et al., 2010; Lande, 2009). We parameterize our general model using published data from a population of common gulls (*Larus canus*, Matsalu National Park, Estonia) and great tits (*Parus major*, Wytham Woods, UK). An estimate for female environmental sensitivity of selection was not available for the common gulls,

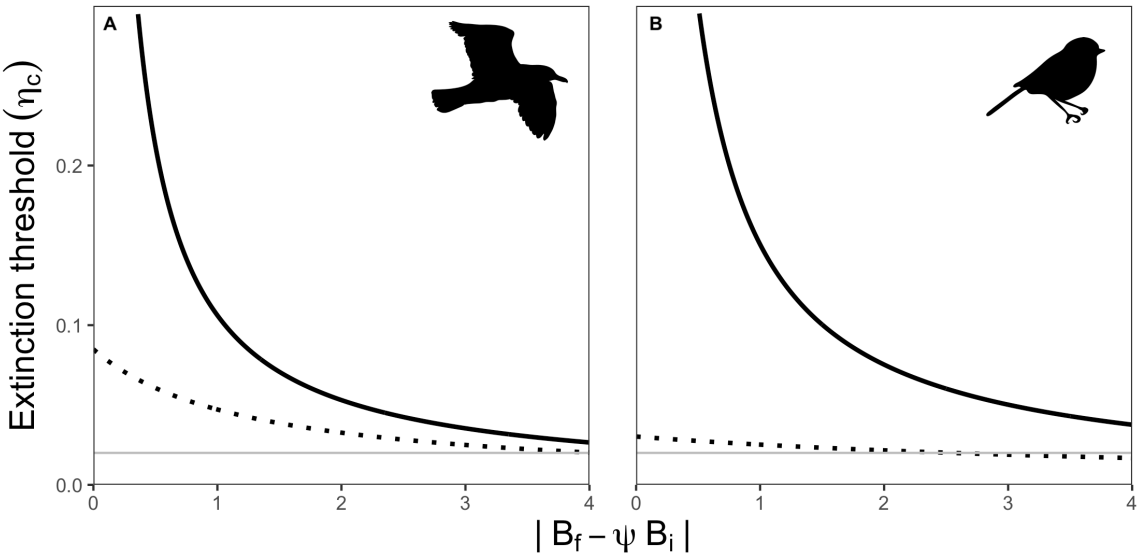

Figure 5: Comparing the effects of IGEs and phenotypic plasticity on the predicted extinction threshold of two wild bird populations using a combination of empirical and simulated data. (A) common gulls (*Larus canus*, Estonia); and (B) great tits (*Parus major*, United Kingdom). The x-axis shows the deviation between focal and ψ-mediated social environmental sensitivities resulting from convergent selection when $\psi B_i \geq B_f$. A solid lines shows model predictions when ψ is non-zero (plasticity is zero). A dashed line shows model predictions when both IGEs and plasticity are non-zero. Great tits: $G_f = 2.62,\ \gamma_f = 0.0061,\ B_f = -5.3,\ b = -4.98,\ r_{max} = 0.49,\ T = 1.81$ (Vedder et al., 2013). Common gull: $G_f = 4.52,\ b = -1.25,\ T = 3.7,\ r_{max} = 0.17$ (Brommer & Rattiste, 2008; Brommer et al., 2008; Niel & Lebreton, 2005). B_f and γ_f were not known in the common gull and were set to match the values of the great tits. r_{max} in the common gull was estimated by taking the average of other gull species.

nor was an estimate for the strength of stabilizing selection according to model assumptions (see Vedder et al., 2013, for discussion). Incomplete data were substituted with estimates from the great tits for illustrative purposes. We focus on a scenario of concordant selection, assuming that the evolutionary interests of males and females are aligned (maximizing reproductive success) though they may differ in magnitude owing to differential survival costs between the sexes (Brommer & Rattiste, 2008). We assume the interaction coefficient is positive where, for example, earlier acquisition of a breeding territory by the male has an advancing effect on the lay date of his mate.

Figure 5 depicts a scenario of convergent selection where the effect of the IGE ψB_i exceeds the environmental sensitivity of the female trait B_f. Assuming zero plasticity, lower deviations in $B_f - \psi B_i$ increase the predicted extinction threshold (black line:

Figure 5). When IGEs and plasticity are both included in the model, the predicted extinction threshold is much lower (dashed line: Figure 5). Across the subset of parameter space shown in Figure 5, including phenotypic plasticity and IGEs concurrently reduces the potential for population persistence, which seems counter-intuitive. This highlights an artefact of the model based on the assumption that parameters are additive and fixed. For example, higher plasticity in great tits generates a more pessimistic prediction in the combined scenario compared to the common gulls because a small deviation in $B_f - \psi B_i$ is offset by a higher degree of plasticity. This assumes birds do not adjust their plastic response for the the effect of the IGE. Redefining the combined effect such that $b - \psi B_i < B_f$ allows plasticity to adjust according to the IGE up to the value of b without overshooting the phenotypic optimum.

Genetic Correlation Between Interacting Partners

Pleiotropy and linkage disequilibrium can generate genetic covariance between traits such that their evolution is codependent proportional to the magnitude of the genetic correlation, ρ. The genetic correlation between two traits measures how genes simultaneously affect the phenotypes expressed by focal and social partners. For example, a positive genetic correlation implies alleles that increase the focal trait value also increase the social trait value, while a negative genetic correlation implies alleles that increase the focal trait value decrease the social trait value, and *vise versa*. In the common gulls, for example, a strong negative cross-sex genetic correlation was estimated between males and females, suggesting that females with earlier onset of breeding produce sons with a delaying effect on their breeding partners (Brommer & Rattiste, 2008).

When traits expressed by interacting partners are genetically correlated, the social partner's impact on evolutionary rescue depends on the combined effect of the genetic correlation and ψ. For example, a negative genetic correlation that increases the threat of extinction when ψ is zero (Figure 6-J), can be masked by a positive IGE (Figure 6-K). In the common gulls, where the cross-sex genetic correlation is negative, a model that considers neither IGE nor genetic correlation predicts an extinction threshold of 0.021 °C/year (assuming the same paramters as Figure 5, $B_i = 2$, $G_i = G_f$ and $b = 0$). Including the negative genetic correlation in the model reduces this estimate by almost

a half (0.12 °C/year), adaptation is constrained. A negative interaction coefficient ($\psi = -2$), further constrains the population and the extinction threshold is almost three times worse than the null model (0.0075 °C/year). However, a positive interaction coefficient ($\psi = 2$) masks the negative effect of the genetic correlation and the predicted extinction threshold is higher than the null model (0.37 °/year).

Correlations between interacting partners can lead to non-intuitive interactions and they are dependent on the scale in which ψ is measured. Nevertheless, some general conclusions can be drawn from the model. For example, the effect of ψ and ρ is cumulative when they are in the same direction. When ψ and ρ differ in directions, for example if there is a negative cross-sex genetic correlation between breeding partners and the interaction coefficient is positive (e.g. females advance their lay dates in response to earlier courtship feeding), then predicted extinction threshold depends on the net effect of ψ and $\rho\sqrt{G_f/G_i}$. When these two terms are equal, the focal trait is unaffected by the environmental sensitivity of the interactant because the effect of the IGE and the genetic correlation effectively cancel out. Otherwise, the effect of the social partner is biased towards the direction with the higher magnitude. When $|\psi| > |\rho\sqrt{G_f/G_i}|$, ψ masks the effect of the genetic correlation whereas if $|\psi| < |\rho\sqrt{G_f/G_i}|$, ψ may reduce the effect of the genetic correlation but the effect of the social partner is biased in the direction of the genetic correlation. The interaction also depends on the relative genetic variance, where $\sqrt{G_f/G_i}$ scales effect correlation coefficient ρ and relative sensitivity of selection between the traits (Figure 5).

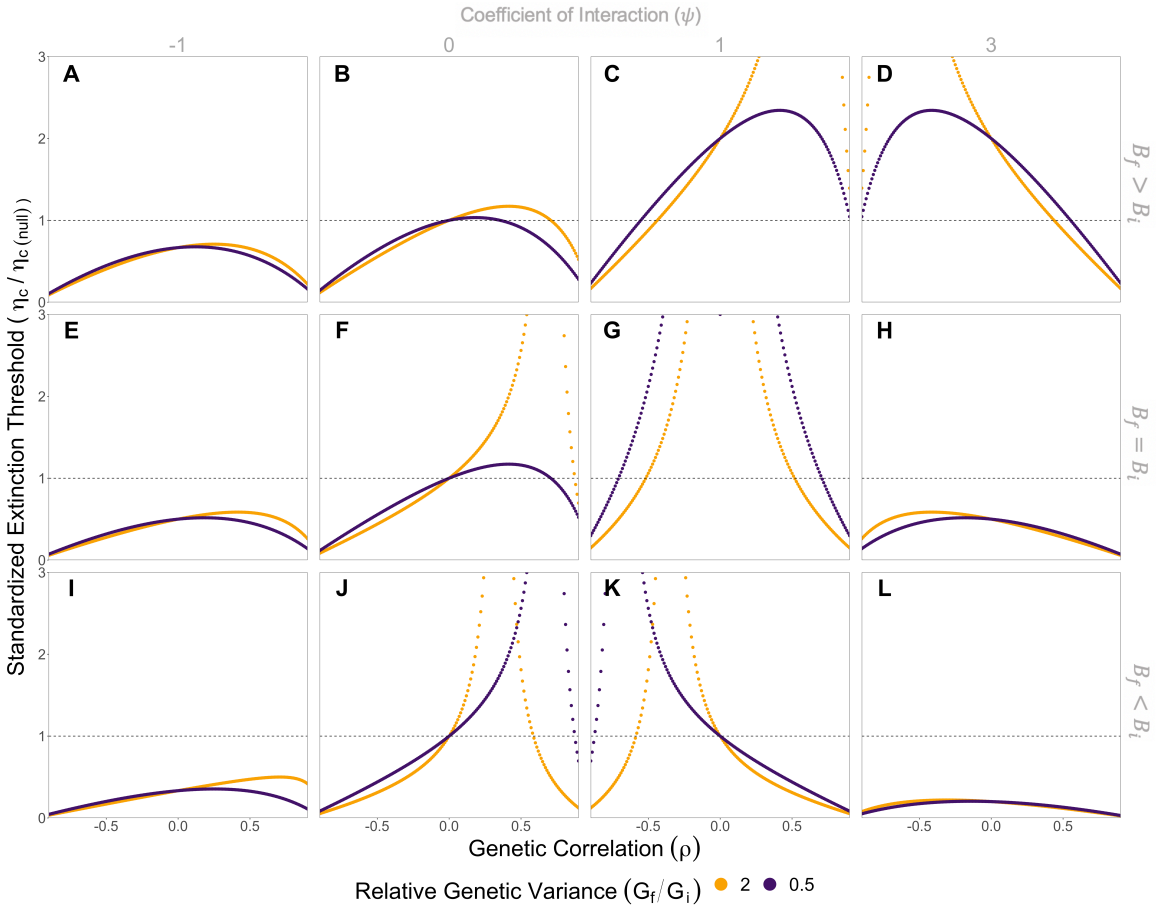

Figure 6: The effect of genetic correlation on the standardized extinction threshold when environmental sensitivities are aligned ($B_f B_i > 0$). Columns present different values of ψ and rows present relative environmental sensitivities between traits. The relative genetic variance between traits is indicated according to the legend, where $G_f/G_i = 2$ indicates that there is twice as much genetic variance in the focal trait relative to the social trait. The extinction threshold predicted by the generalized model is scaled by a null model, model equivalence is indicated by the horizontal dashed line.

4 Model Implications

Theory predicts that indirect genetic effects (IGEs) can alter the pace and direction of evolution (Wolf et al., 1998). Such social evolutionary effects are considered an important driving force in phenotypic evolution that may help to explain unanswered questions in evolutionary ecology (Kruuk et al., 2008; Pujol et al., 2018; Visser, 2008; Wilson, 2014). We have extended mathematical theory to explore how adaptive evolution of the social environment can affect population persistence by constraining or reinforcing adaptive evolution. We show that IGEs and genetic correlation can bias model predictions and that considering these mechanisms concurrently can change the predicted outcome. Empirical estimates of IGEs, particularly in fitness-related traits that are expected to shift under environmental change, support the idea that IGEs are an important factor in evolutionary dynamics. Additional heritable variation provided by IGEs may, therefore, be a consequential determinant in population-level response to climate change and other local environmental stressors. However, IGEs are often overlooked. The model we have presented is a first step in understanding the complex evolutionary dynamics that drive population-level changes. We have presented a simple scenario in social evolutionary theory where the interaction is nonreciprocal and individuals are not related. Relaxing either of these assumptions would likely lead to a very different result, though the framework we have outlined can provide a basis for future extensions.

Evolutionary rescue models can be used to help clarify the mechanisms that allow a population to overcome the demographic consequences of environmental change. Our findings suggest that models that do not account for IGEs may be missing an important source of heritable variation in the population that can buffer or intensify the threat of extinction (Figure 3). For example, overlooking IGEs that drive a focal trait towards its optimum could lead to a pessimistic prediction that underestimates the extinction threshold of the population. Ignoring IGEs that drive the focal trait away from its optimum would have the opposite effect, where estimates are overly optimistic about the degree to which a population can track its environment. The numerical evaluations of the model agree qualitatively with a wealth of quantitative genetic theory exploring how IGEs constrain or accelerate the predicted response to selection (Bijma et al.,

2007; McGlothlin et al., 2010; Moore et al., 1997; Wilson, 2014; Wolf et al., 1998).

We find that not all populations will be equally affected by IGEs, according to their demographic and genetic characteristics (Figure 4). From a conservation perspective, this suggests that populations with low genetic variance and/or slow life histories may stand to gain from 'adaptive' IGEs and more to lose when IGEs are 'maladaptive'. In the defined context of evolutionary rescue, populations that lack the evolutionary potential to keep pace with environmental change are dependent on other mechanisms (Carlson et al., 2014), we show that this can also include IGEs. This highlights the value of considering the ecological consequences of social evolution.

Our findings are with consistent theoretical arguments at the intersection between social evolution and evolvability. An abiding question in evolutionary ecology centers around a lack of response to selection, so-called evolutionary stasis (Hansen & Houle, 2008). There is a wealth of theory exploring how IGEs can constrain or accelerate the predicted response to selection (McGlothlin et al., 2010; Moore et al., 1997; Wilson, 2014; Wolf et al., 1998) and a key concept in IGE theory illustrates how a focal trait can evolve in the absence of genetic variation, provided the social environment is heritable. While accumulating estimates of IGEs establishes a genetic basis underlying behavioural, morphological, and physiological traits, evidence of their effect driving selection is rather sparse (Falconer, 1955; Muir, 2005).

Empirical evidence shows that IGEs and genetic correlations can have a large effect on the estimated heritable variation and, inferentially, the observed response to selection. IGEs can increase heritable variation (Costa e Silva et al., 2013; Germain et al., 2016) or remove heritable variation despite estimate of ordinary heritability (Costa e Silva et al., 2013; Ellen et al., 2016). IGEs estimated in sex-limited life-history traits (Brommer & Rattiste, 2008; Ellen et al., 2016; Gienapp et al., 2013; Teplitsky et al., 2010) further underscore the need to broaden our understanding of the total available heritable variation available for selection. In the case of the red-billed gulls *Larus novaehollandiae scopulinus*, for example, egg-laying date was not heritable in females but significantly heritable in males (Teplitsky et al., 2010), providing a clear example of how the evolution of the focal trait may be highly dependent on genetic variation in a social trait.

There are many mechanisms that can lead to adaptation and striking a balance between predictive ability and model simplicity is an enduring challenge in evolutionary ecology (Urban et al., 2016). For example, considering the effect of DGEs, IGEs and the genetic correlation between traits can completely reverse model predictions (Figure 6) and there is an empirical basis for IGEs masking pleiotropic effects between traits (Costa e Silva et al., 2017). The argument for concurrent consideration of IGEs and genetic correlations been argued by others (Moore & Pizzari, 2005; Wilson, 2014). Our model shows how genetic correlations between interacting traits can result in non-intuitive predictions, such as when the IGE masks the effect of the genetic correlation.

While the model undoubtedly requires empirical validation and further insight on key assumptions, its predictions and limitations can be used to guide further study. For example, the model suggests that IGEs in the direction of selection do not necessarily lead to population adaptation. Where quantitative genetic models would generally equate higher rates of phenotypic change to a higher likelihood of adaptive tracking, our model suggests that higher rates of evolution driven by IGEs may increase population maladaptation if there is a wide difference between focal and social environmental sensitivities (Figure 3). This could reflect a scenario where the focal trait would be relatively insensitive to environmental change, but a strong IGE from a more environmentally sensitive interacting trait forces the focal trait to outpace its optimum. Given that the model is highly sensitive to deviations in the denominator (Gienapp et al., 2013), the extent of maladaptation is likely an artifact of the model, but furthering our understanding of the conditions under which IGEs can lead to the evolutionary rescue or extinction of a population can help to generate testable predictions and inform research programs.

For example, in a scenario of sexually antagonistic selection where the evolutionary interests of potential mates diverge, it is expected that one sex should evolve a counteracting trait (Moore & Pizzari, 2005), such that the response trait could lead to the evolutionary rescue of the population by reducing the demographic consequences of sexual conflict (Svensson, 2019). Other leading questions center upon the conditions under which ψ would evolve (Chenoweth et al., 2010). For example, theoretical stud-

ies explore how environmental change affects the evolution of phenotypic plasticity and the maternal effects coefficient in offspring (Ezard et al., 2014; Kuijper & Hoyle, 2015).

5 Limitations and Assumptions

The functional form of the model likely contributed to an unexpected result. For example, modelling IGEs and plasticity concurrently predicted a significant reduction in the extinction threshold compared to separate models of each parameter. This occurs because the extinction threshold depends on the combined effect of the parameters, which are assumed to be fixed. The sensitivity of selection represents an 'optimal reaction norm' (Gienapp et al., 2013) where reaction norms of social and phenotypic plasticity can bring the population closer to its optimum by reducing the difference with B_f. The combined sum of parameters allows two seemingly adaptive mechanisms to increase the distance from B_f and it suggests that an individual cannot adjust its level of plasticity towards changes from the social environment. Redefining the domain of the denominator in (13) reflects a more intuitive example where plasticity adjusts according to the abiotic and the social environment.

Another contributing factor to the difficulty in modelling both social and phenotypic plasticity relates to the assumption that the parameters are fixed. Genetic variation in phenotypic plasticity can cause directional selection under changing environmental conditions (Gavrilets & Scheiner, 1993; Lande, 2009). Experimental evolution shows that ψ is also a heritable property that can respond to selection (Chenoweth et al., 2010) and may vary among individuals (Brooks & Endler, 2001) and populations (N. W. Bailey & Zuk, 2012). Improving our understanding of the interaction between these two mechanisms would likely benefit from a more dynamic modelling approach (see Chevin & Lande, 2011; Kazancıoğlu et al., 2012; Lande, 2009).

Most parameters in the model are held constant for simplicity. In reality, the fitness function is likely to change over time, and sustained directional selection may reduce genetic variation (and the width of the phenotypic function), although mechanisms like IGEs (Danielson-François et al., 2009) and genetic correlations may help to sustain variation. The genetic covariance structure may also be environmentally dependent (Sgrò & Hoffmann, 2004) and, if generated by linkage disequilibrium, would break down through recombination. Demographic parameters may also vary across time, if for example environmental change alters resource availability (Sæther et al., 2016), though the intrinsic growth rate may be constrained in situations of environmental

extremes (Chevin & Hoffmann, 2017).

Furthermore, we have defined population mean fitness according to the focal phenotype, such as the effect of males on female laying date (Teplitsky et al., 2010). This assumption is largely contextual. The trait expressed by social partners may have a more direct effect on population dynamics depending on the causal phenotype. For example if the effect of partner disease infectivity on host susceptibility affects both the social and focal partners' survival (Lipschutz-Powell et al., 2012), then relaxing this assumption and using a multivariate fitness function may be more representative. Another avenue of social evolutionary theory which we have omitted is the effect of social selection, where the social selection gradient is the covariance between the phenotype of the social partner and the fitness of the focal individual. In this case, the IGE would affect both the focal phenotype and fitness (McGlothlin et al., 2010). But this too is dependent on context and should be incorporated into a model that involves relatives or reciprocal interactions are introduced (Bijma et al., 2007).

We model the fitness consequences of the IGE relative to its effect on the lag load (equation 10) as this captures how the effect of the IGE on the focal phenotype alters population maladaptation and its effect on mean population fitness. We ignore the fitness trade-off between the lag load and the standing load when additional genetic variance is introduced through the IGE. Using a similar model, Lande and Shannon (1996) show that the fitness costs from an increasing standing load are counterbalanced by the decrease in the lag load when the environmental change is deterministic. Under different forms of environmental change, this assumption may have a greater impact but in the current context seems trivial.

The model is based on many simplifying assumptions. We have overlooked other processes that change allelic frequency such as genetic drift, mutation, and gene flow that have been the focus of other theoretical studies (Bürger & Lynch, 1995; Kirkpatrick & Peischl, 2013; Lynch & Lande, 1993; Orr & Unckless, 2014), and the contribution of population size. We have also excluded demographic and environmental stochasticity, which may intensify the threat of extinction (Lynch & Lande, 1993). This model, like many before it, assumes discrete-non overlapping generations. Adapting the model to account for generational overlap can help tailor the model towards a broader range of populations to which it is intended.

6 Empirical Challenges

Both evolutionary rescue and social evolution are fields in which theory has outpaced empirical validation. Empirical estimates are a critical missing link in our understanding of the factors that contribute to the evolutionary rescue of populations in the wild. Much of the empirical literature on evolutionary rescue involves experimental evolution of antibiotic resistance and the adaptation of yeast to saline environments (Bell & Gonzalez, 2009; Martin et al., 2013), though some models have been parameterized using data from wild populations (Gienapp et al., 2013; Radchuk et al., 2019; Vedder et al., 2013). Trait-based models of IGEs requires estimates of ψ, though there are only seven studies that formally estimated the interaction coefficient in behavioural traits in arthropods and fish (Hunt et al., 2019). Most empirical estimates of IGEs use a variance partitioning approach such that causal traits are not known. This can shed light on the relative importance of IGEs in the system. IGE theory can stand to greatly benefit from empirical estimates of key parameters (e.g. ψ) needed to parameterize theoretical models across traits and taxa. While the data requirements make estimates in wild populations especially challenging, studies such as Brommer et al. (2015) and Brommer and Rattiste (2008) provide a pathway forward. Estimating the parameters required to validate theoretical models of IGEs and evolutionary rescue presents an exciting challenge to empiricists. An increasing number of estimates relying on variance partitioning method highlights the relative importance of IGEs as an important determinant in evolution, but a significant obstacle remains in estimating the interaction coefficient in natural systems.

The environmental sensitivity of selection is a parameter that is often overlooked by empiricists, but it continues to appear in the theoretical literature (Chevin et al., 2010; Connallon & Hall, 2016; Marshall et al., 2016). The environmental sensitivity of selection quantifies the covariance between the abiotic environment and phenotypic selection and identifies specific agents of selection needed to study genetic change alongside other mechanisms like plasticity. Differences in the environmental sensitivities between interacting and focal partners is a key parameter in this model, suggesting that it should be emphasized in future work seeking to understand the mechanisms underlying observed patterns of adaptation and extinction.

7 Conclusions

This thesis extends mathematical theory to explore the relative importance of indirect genetic effects (IGEs) in the evolutionary rescue of a population experiencing directional change. The model indicates that IGEs can mitigate or intensify the demographic consequences of environmental change. Mean population fitness was more sensitive to IGEs when populations had slower life-histories or lower standing genetic variation in the focal trait. We extended the model to include a genetic correlation between interacting traits. Considering the combined effects of IGEs and a genetic correlation altered model predictions where, for example, declines in population mean fitness from a negative genetic correlation could be masked by IGEs, or *vise versa*. This highlights the value of multi-mechanism models in bridging knowledge gaps in population-level response to a changing environment. We also generalized prior theory to incorporate adaptation through phenotypic plasticity, though simulations suggest that the interaction between IGEs and plasticity is not adequately captured by the model. The effect of the IGE in the model was mediated by the interaction coefficient, ψ and the environmental sensitivity of selection. A lack of empirical estimates of key parameters prevents empirical validation of the model at this time. Accumulating empirical estimates of IGEs, particularly in fitness-related traits expected to shift under environmental change, suggests that we have been overlooking a source of heritable variation provided by IGEs. Our model suggests that IGEs can be an important determinant in population-level response to climate change and should be the focus of further study to better our understanding of the role social interactions play in population dynamics. Further empirical study into the genetic basis of these interactions will broaden our understanding of the mechanisms that drive adaptation and extinction.

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Appendix I. Model Derivations

Here we derive the maximum rate of environmental change for a focal trait (z_f) that may be affected indirectly by a nonreciprocal indirect genetic effect (IGE) from an interacting trait (z_i) and/or a genetic correlation. Let $y = [f, i]$ be an index indicating properties measured on focal individuals (f) and interacting traits expressed by social partners (i). We start with the base assumption that all individuals in the population express both focal and social traits (i.e. the level of aggression in focal individuals depends, in part, on the body mass of their social partners). We then show how the model can be extended to model sex-limited traits. The expected generational rate of change in the mean focal phenotype is given in the main text as:

$$\frac{\Delta \bar{z}_f}{T} = \frac{G_f \beta_f + G_{fi} \beta_i + b_f \eta T + \psi \Delta \bar{z}_i}{T} \tag{1}$$

and the rate of change in the social trait as:

$$\frac{\Delta \bar{z}_i}{T} = \frac{G_i \beta_i + G_{fi} \beta_f}{T} \quad . \tag{2}$$

When the population reaches equilibrium, the phenotypic rate of change for each trait matches the rate of change in their respective optima (θ_i). Substituting $\Delta \bar{z}_i / T = B_i \eta$ into (1) and (2) and solving for the focal equilibrium lag yields:

$$(\bar{z}_f - \theta_f)_{eq} = \frac{\eta T}{G_f \gamma_f (1 - \rho^2)} (B_f^* - \psi B_i - \frac{G_{fi}}{G_i} B_i) \tag{3}$$

where $B_f^* = B_f - b_f$ is the focal sensitivity to environmental change after accounting for phenotypic plasticity. The equilibrium lag is a measure of maladaptation, it quantifies how far the population mean trait deviates from its optimum. To derive the critical rate of environmental change, we are interested in the maximum degree of maladaptation the population can tolerate while still being able to replace itself. The growth rate of the population can be expressed in relation to the equilibrium lag:

$$r = \frac{\gamma_f (\bar{z}_f - \theta_f)_{eq}^2}{2T} \quad . \tag{4}$$

When $r = 0$ (or $ln\bar{W}_f = 1$), the population is just replacing itself. Substituting (3) into (4) and setting $r = 0$ gives the critical rate of environmental change:

$$\eta_c = \sqrt{\frac{2r_{max}\gamma_f}{T}} \left(\frac{G_f(1-\rho^2)}{B_f^* - B_i(\psi + \frac{G_{fi}}{G_i})} \right) \tag{5}$$

Sex-limited traits

If modelling sex-limited traits (i.e. female egg-laying date is affected by an IGE from her breeding partner), equations (1) and (2) must account for the genetic contribution of males and females in the population:

$$\frac{\Delta\bar{z}_f}{T} = \frac{\frac{1}{2}G_f\beta_f + \frac{1}{2}G_{fi}\beta_i + b_f\eta T + \psi\frac{1}{2}\Delta\bar{z}_i}{T} \tag{6}$$

and

$$\frac{\Delta\bar{z}_i}{T} = \frac{\frac{1}{2}G_i\beta_i + \frac{1}{2}G_{fi}\beta_f}{T} \quad . \tag{7}$$

The equilibrium lag of the focal trait can the be expressed as:

$$(\bar{z}_f - \theta_f)_{eq} = \frac{2\eta T}{G_f\gamma_f(1-\rho^2)}(B_f^* - \psi B_i^* - \frac{G_{fi}}{G_i}B_i) \quad . \tag{8}$$

The growth rate described in (4) remains the same. Substituting (8) into (4) when $r = 0$ yields the sex-limited critical rate of environmental change:

$$\eta_c = \sqrt{\frac{r_{max}\gamma_f}{2T}} \left(\frac{G_f(1-\rho^2)}{B_f^* - B_s(\psi + \frac{G_{fi}}{G_i})} \right) \tag{9}$$

which can be re-written to resemble (5), where:

$$\eta_c = \frac{1}{2}\sqrt{\frac{2r_{max}\gamma_f}{T}} \left(\frac{G_f(1-\rho^2)}{B_f^* - B_i(\psi + \frac{G_{fi}}{G_i})} \right) \quad . \tag{10}$$

Therefore, to extend the critical rate of environmental change to a scenario where traits are sex-limited requires that η_c be scaled by $1/2$.