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Natural selection shapes and regulates the population dynamics of birds and mammals

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Abstract I analyse for a natural selection component in the population dynamic regulation of birds and mammals. By fitting species-specific age-structured population dynamic models to 3,369 and 483 abundance timeseries for 900 and 208 species of birds and mammals, AIC model-selection includes natural selection in 79% to 92% of the accepted models, explaining 80% of the population dynamics variance, with median selection regulation being 1.2 (se:0.11) times stronger than density regulation. The selected life history changes produce damped to stable population cycles with median periods of seven and eight generations given stable cycles. These predictions solve several population cycle enigmas, highlighting the population dynamic implications of a natural selection that predicts also the life histories and abundancies of animals. All selection regulated models are available for online simulations at https://mrLife.org.

Keywords: Eco-evolutionary dynamics, natural selection, density dependence, population dynamics, population regulation, timeseries

1 Introduction

Although often neglected, a widespread absence of fit between population dynamic models and timeseries of abundance data remains one of the most enduring problems in population ecology (Myers 2018; Oli 2019; Andreassen et al. 2021). This reflects a contradiction where population regulation is supposed to shape the dynamics of natural populations, and traditional density regulated models fail to explain even simple population dynamic trajectories. Take for example a population that declines until the decline stops gradually and the population begins to increase. While this is observed often, density regulated populations will only increase, or decline, towards carrying capacity showing no change in the direction of growth (over-compensation from strong density regulation does not explain a gradual change in the direction of growth).

For the last century or so, variation in the environment and other species provided the conceptual solution to the lack of fit between density regulation theory and data. This explains population dynamic deviations that correlate with environmental fluctuations, inter-specific competition, predation, prey, and climatic change. But these extrinsic drivers are nearly always case specific, with the vast majority of the available timeseries of abundance data having no explicitly identified extrinsic cause. The extend of the extrinsic influence is therefore unresolved in the most cases, with the hypothesis serving more often as a convenient *ad hoc* explanation for the lack of fit between population regulation theory and data.

Delayed density regulated models provide another practical solution, generating "single-species" models that "explain" much of the observed dynamics (e.g. Turchin and Taylor 1992; Hörnfeldt 1994; Hansen et al. 1999). But there is usually not a proposed mechanism of cause and effect for the estimated delays, with the majority of the delayed density regulated studies turning the blind eye to the real problem: the lack of explicitly identified but necessary population dynamic interactions.

If we concentrate on studies with a mechanistic focus, population dynamic models have advanced by spatial synchrony (Ranta et al. 1995; Koenig 2002; Liebhold et al. 2004), stochasticity (Kaitala et al. 1996; McKane and Newman 2005), environmental oscillations (Post and Forchhammer 2002; Taylor et al. 2013), maternal effects (Ginzburg 1998), demographic details (Murdoch et al. 2002; McCauley et al. 2008), and higher-dimensional interactions (Tyson et al. 2010; Liu et al. 2013). But even this broader theoretical framework leaves several of the most essential population dynamic enigmas unresolved (Myers 2018; Oli 2019; Andreassen et al. 2021).

These unsolved issues include the presence of population cycles in the absence of cyclic species interactions. The isolated *Daphnia*-algae system analysed by Murdoch and McCauley (1985) is one example, where the population of *Daphnia* cycled with a stable period independently of the presence versus absence of a cycle in the algae. Similar observations include snowshoe

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hares that cycle in the absence of lynx (Keith 1963), and the absence of a firm predator-prey interaction for one of best documented cycles in forest insects (Berryman 1996).

Another paradox is the widespread presence of life history changes in phase with population dynamic cycles, changes that do not follow from density regulation, nor from interactions between predators and prey. Where predation affects survival, "most, if not all, cyclic rodent populations are characterised by phase related changes in body mass, social behaviour, ... and reproductive rates" (Oli 2019). These life history cycles are a serious problem for traditional population regulation theory because the reproductive rate stays low across the low phase of population cycles, where relaxed density regulation predicts a high reproductive rate (Myers 2018).

Other problems include that no experimental manipulation of predators and resources "has succeeded in stopping rodent population cycles anywhere" (Oli 2019), and "how can low amplitude cycles persist if high densities are required for the build-up of predators, parasitoids, pathogens or detrimental conditions" (Myers 2018).

While none of these issues question that external factors have a role to play in population dynamics, they hint at a theory that lacks an essential population dynamic mechanism. In my search for such a mechanism I take the parsimonious view that to explain the growth, abundance, and dynamics of natural populations we need first of all to understand how they regulate their own growth when other things are equal. Theoretically, it is not only density regulation, but also the intra-specific population dynamic feedback selection of interactive competition that regulates the growth and abundance shaping the dynamics of natural populations (Witting 1997, 2000b). Natural selection may thus be the main reason for the often-observed lack of fit between density regulation theory and data.

1.1 On natural selection regulation

With the Malthusian parameter r being the exponential growth rate of the population and the natural selection fitness of the individual (Fisher 1930), natural selection acts stronger on the demographic components of population dynamic growth than on other phenotypic traits.

Early work on the population dynamic implications of these naturally selected changes in the growth rate were verbal formulations relating primarily to the generation of population dynamic cycles (e.g. Elton 1924; Ford 1931; Voipio 1950; Chitty 1960; reviewed by Voipio 1988). These views failed to reach general consensus partially because the proposed process in the minds of many biologists (Chitty 1996), but not in the original formulations, became associated with the Wynne-Edwards (1959, 1993) hypothesis of self-regulation by group-selection, which was forcefully refuted (Maynard-Smith 1964; Wiens 1966; Williams 1966). The proponents also failed to develop population dynamic models that demonstrated the plausibility of the hypotheses (Voipio 1988), with a genetic model-interpretation with two phenotypes and no time-lag in the selection response failing to produce population dynamic cycles (Stenseth 1981, 1985).

But there is a delay of one generation from parents to offspring in the population response to natural selection making selection-driven population dynamic cycles plausible (Witting 1997, 2000b). These cycles depend on the magnitude of the selection response, and Sinervo et al. (2000) confirmed their existence by a two-strategy two-generation selection-driven oscillation in side-blotched lizard (*Uta stansburiana*). Other empirical studies soon documented a wide range of population dynamic implications of natural selection (e.g. Yoshida et al. 2003; Bell and Gonzalez 2009; Coulson et al. 2011; Turcotte et al. 2011; Bell 2017; Halley et al. 2021; Pavithran and Sujith 2022). I add to this list by analysing 3,852 population dynamic timeseries, in an attempt to estimate if natural selection is the missing population regulation that will make our single species model work as a first approximation to the population dynamics of birds and mammals.

To estimate the population dynamics of natural selection changes in the growth rate, I extend on a long tradition where the expected density dependent decline in r with increased abundance is incorporated structurally in population dynamic models and used to statistically estimate a parameter for the strength of the density regulation response of the population (e.g. Turchin and Taylor 1992; Sibly et al. 2005; Knape and de Valpine 2012). I follow Witting (1997, 2000b, 2013) and add the structure of the theoretically expected selection to these models estimating an extra parameter for the strength of the response to natural selection. The structure of the population dynamic model is in this way capturing the functional form of the natural selection force that selects, with the value of the estimated parameter capturing the response level of the population, with genetic evolution being one of many potential response components (Mameli 2004; Danchin et al. 2011).

To theoretically deduce the structural form of the expected natural selection I use individual selection

and the widely accepted game theoretical approach of Continuously Stable Strategies (CSS, Eshel 1983; Taylor 1989; Christiansen 1991) to balance the frequency-independent physiological selection of a continued increase in r (Fisher 1930) against the ecological selection of the intra-specific interactive competition for resources that follows from the population growth of the selected increase in r (Witting 1997, 2000a,b, 2002a). This interactive selection is density-frequency-dependent in the sense that the number of competitive encounters per individual is positively related to the density of the population, with the frequency distribution of the competitive abilities/qualities across the individuals in the population making the absolute competitive ability/quality of an individual relative by determining the percentage of the interactive competitive encounters that the individual wins. Combined with the energetic trade-off between competitive quality and the quantity/rate of reproduction (Smith and Fretwell 1974; Stearns 1992), this provides a population dynamic feedback where the frequency-independent selection of the physiology selects a decline in quality through the selection increase in intrinsic reproduction, and the resulting population growth and density-frequency-dependent interactive competition selects a decline in intrinsic reproduction through a selection increase in competitive quality.

It is the population dynamic feedback between these two counteracting selection forces that regulates the growth of the population, selecting a CSS with limited population growth, a balanced life history, and a population dynamic equilibrium abundance with an intermediate level of interactive competition (Witting 1997, 2000b, 2002a). This selection is essential for the evolution of the balanced life histories of animals (Witting 2008, 2017b, 2024b) and it contrasts to other natural selection concepts that are population dynamically unbalanced with no feedback selected regulation. These include the frequency-independent selection of a continued increase in population dynamic growth and carrying capacity (Fisher 1930; Roughgarden 1971; Caswell 1989), and the arms race of density-invariant frequency-dependent interactive competition that selects a continued increase in competitive quality that drives the population to negative growth and extinction (Simpson 1953; Parker 1983; Haigh and Rose 1980; Maynard Smith 1982; Vermeij 1987). It is first when we add the density dependence of the frequency-dependent interactive competition to our model that we obtain a balanced feedback selected population dynamic regulation.

This feedback selected regulation is the predicted endpoint of an evolutionary unfolding where a continued selection increase in net energy for replication predicts the sequential natural selection of the major lifeforms (Witting 2002b, 2017b, 2024b). This selects energy requiring traits (like body mass, interactive behaviour, multicellularity, non-replicating males, and sexually replicating units) as competitive quality used in interactive competition, explaining evolutionary transitions from virus-like replicators, over prokaryote- and protozoa-like self-replicating cells, to multicellular sexually reproducing animals.

These theoretically predicted animals have theoretically predicted inter-specific allometries (Witting 1995, 2017a), with an inter-specific trait interpolation of the selected animal-model across more than hundred thousand life history data estimating equilibrium population dynamic life history models for approximately 90% of the birds and mammals of the world (Witting 2024a). I use these feedback selected species-specific models as the equilibrium age-structured population dynamic models of the present study. This allows me to focus my timeseries analysis on the estimation of the population regulation parameters and initial conditions that explain the observed dynamics.

These conditions select for enhanced competitive quality and decelerated population growth when the population density is above the naturally selected population dynamic equilibrium, and for decreased quality and accelerated growth when the population is below the equilibrium, generating population cycles that converge on hyperexponential growth at zero abundance (Witting 1997, 2000a,b). The predicted cycles are damped phase-forgetting in most cases, with amplitudes and cyclic regularities that depend on the magnitudes and regularities of external perturbations. The resulting dynamics can imitate the population cycles of forest insects (Witting 1997, 2000b), as well as the delayed recovery of large whales following commercial exploitation in past centuries (Holt 2004; Witting 2013).

The damping of the cyclic dynamics is inversely related to the population's response to the feedback selection. As the present study estimates the response statistically from timeseries of abundance data, we do not know its underlying form of heritability except that it is part of the complete general inclusive inheritance system of the population (Mameli 2004; Danchin et al. 2011). Population genetics is the traditional component of this inheritance, and other potentially important components include parental effects (Boonstra and Hochachka 1997; Inchausti and Ginzburg 2009), epigenetic inheritance (Richards 2006; Bossdorf et al. 2008), and long-term selected phenotypic plasticity that allow individuals to respond to e.g. cyclic changes in the selec-

tion pressure (Snell-Rood et al. 2018; Pfenning 2021). Cultural inheritance is another factor (Danchin et al. 2011; Whitehead et al. 2019), where most offspring may balance their quality/quantity investment in their offspring following the balance of their parents, with fewer offspring choosing another balance that keeps the cultural heritable variance of the population. To capture the cumulated response of all these processes I use continuous traits with a moderate proportional change that is similar in structure to the response of the secondary theorem of natural selection (Robertson 1968; Taylor 1996).

As it is the cumulated response of the complete inclusive inheritance system to the selection pressure of the selection gradient that generates the change in population dynamic growth, we cannot reject selection regulation as a valid hypothesis just because one inheritance component is insufficient to account for the observed change. It is like density regulation, where we know the overall structural shape of the response and do not reject the potential existence of a statistically estimated regulation just because we have not yet identified the underlying mechanistic details.

Several studies have estimated density regulation from statistical analyses of timeseries of abundance data (e.g. Turchin and Taylor 1992; Sibly et al. 2005; Knape and de Valpine 2012), but no study has systematically estimated the potential influence of selection regulation across a large number of species. I aim to estimate both regulations as it is possible to distinguish between them statistically when we fit population models to timeseries of abundance estimates. Density regulation sets the growth rate as a monotonically declining function of density, while feedback selection accelerates and decelerates growth as a function of the density frequency dependent selection. I use this difference to provide the first large-scale analysis that estimates the strength of density and selection regulation across almost four thousand populations of birds and mammals.

2 Method

2.1 Data

I use timeseries of abundance estimates from the Living Planet Index (LPI 2022), the North American Breeding Bird Survey (BBS; Sauer et al. 2017), the PanEuropean Common Bird Monitoring Scheme (EU; PECBMS 2022), the Netwerk Ecologische Monitoring (NET; Sovon 2022), the Swiss Breeding Bird Index (SWI; Knaus et al. 2022), the British Trust for Ornithology (BTO 2022), the Danish Ornithological Society (DOF 2022), and Svensk Fågeltaxering (SWE; SFT 2022).

Having different origins, these timeseries are of varying quality and length. To analyse a large number of timeseries I apply a moderate baseline filter to the data, and to identify results from long timeseries of high-quality data I select a restricted subset of controls also. The baseline timeseries include more than ten estimates over at least fifteen years, resulting in 3,369 accepted timeseries for 900 bird species and 483 timeseries for 208 mammal species, with all timeseries scaled for a geometric mean of unity.

The high-quality controls are standardised indices from point-counts of birds calculated from hundreds of separate indices for individual observers. The individual observers count in the same way at the same time each year on individual routes with a given number of geographically fixed point-counts. The calculation of these indices is very standardised, correcting for observer and severe weather. Given a sufficient number of routes, these timeseries are of high quality.

A potential issue with bird indices is that their geographical coverage may not necessarily reflect a population where the dynamics is spatially synchronised. I account for this by restricting my control timeseries to the population dynamics delineated indices (PDDIs) that Witting (2023b) compiled from the North American Breeding Bird Survey (Sauer et al. 2017). These are based on more than six million observations from USA and Canada, providing yearly abundance estimates for 51 years. Starting from a $15x7$ longitudinal/latitudinal grid of separate indices covering USA and southern Canada, the PDDIs combine neighbouring areas with synchronised indices delineating larger areas with desynchronised dynamics from one another, estimating 462 populations with different dynamics (Witting 2023b).

2.2 Population models

I use species-specific age-structured models to ensure that the estimated regulation and dynamics occur on the relevant biological timescale accounting for the demographic delays and population dynamic inertia of each species. If I use a non-structured model with yearly non-overlapping generations instead, I would convert much of the between year fluctuations in the abundance estimates into strong density regulation in over-compensatory models (Wolda and Dennis 1993). This may happen even when the true population trajectory is smooth, and the fluctuations in the abundance estimates follow from the estimation process only. Such

fluctuating sampling variation is widespread with natural populations, where it is common to count only a fraction of the total population with year-to-year variation in the available fraction.

To reduce this overfitting to random sampling variation further, I restrict the estimated strengths of regulation to avoid strong over-compensation, focussing on smooth population trajectories with additional sampling variation accounting for year-to-year zig-zag variation in the data (see appendix). This is a conservative method that aims to get the estimates of average regulation right in most cases, assuming that truly fluctuating dynamics is rare at the population level of birds and mammals.

As we cannot usually estimate the age-structured life history from abundance data, I obtain the equilibrium demographic parameters of the age-structured models of all species from Witting (2024a). These parameters are offspring survival [p_0 for age-class (a) zero], annual survival (p) for older age-classes ($a \geq 1$), age of reproductive maturity (a^*_m), and annual reproduction (m^*) of mature ($a \geq a^*_m$) females at population dynamic equilibrium (superscript $*$).

These models provide not only a species-specific equilibrium age-structure (c^*_a with $1 = \sum_{a\geq 0} c^*_a$) that I use as the initial age-distribution, but also a complete equilibrium age-structured population dynamic model with zero growth that I can iterate forwardly in age-unit-timesteps (often years but see appendix). A per-generation replication equation

$$\lambda^* = p_0 p^{a^*_m - 1} m^* t_r/2 = 1 \tag{1}$$

can also express each model, where $r^* = \ln \lambda^* = 0$ and $t_r = 1/(1-p)$ is the expected reproductive period given individuals that mature (Witting 2024a). By keeping these equilibrium demographic parameters fixed for each species, I estimate only the absolute abundance, the density and selection regulation parameters, and a few initial conditions from the abundance data.

I develop exponential, hyperexponential, density regulated, and selection regulated models for each population. I use likelihood fitting based on log normally distributed abundance data, apply a minimum fitting criterion, and find the best-fitting-hypothesis by the Akaike information criterion (AIC, Akaike 1973) to trade-off the number of parameters (from 2 to 5) against the likelihood of each model (see appendix for details).

As AIC selects selection-based models most often (see results), I run a second AIC-selection to estimate the best selection regulated models for all populations. In addition to a stable equilibrium, this second selection includes models with a linear trend in equilibrium density during the whole, or parts of, the data period. This allow me to quantify not only the relative strengths of regulation by density and selection, but to estimate also if local population trends are indicators of underlying changes in the external environment (assuming that a change in equilibrium reflects improved or deteriorating external factors).

I set regulation by density and selection to operate on the birth rate ($m = \tilde{m} m^*$) and age of reproductive maturity ($a_m = \tilde{a}_m a^*_m$) by changes in relative parameters ($\tilde{m}$ and $\tilde{a}_m$). These are constant in the exponential population model and set to unity ($\tilde{m}^* = 1$ and $\tilde{a}^*_m = 1$) at population dynamic equilibrium (m^* and a^*_m).

As I fit the 1+ abundance ($n = \sum_{a\geq 1} n_a$) of the population to the abundance data, the estimated multiplicative regulation on the birth rate includes regulation on offspring survival implicitly (as the number of age-class one individuals at time t is the number of births at time $t-1$ multiplied by offspring survival). Hence, I cover regulation on the three life history parameters that are most sensitive to density dependent changes, allowing for regulation on a_m for an extended analysis of the PDDI control timeseries only. The appendix describes the details of the population dynamic modelling based on selection regulation, with essential differences between the four population models described below.

Exponential growth. The exponential models are the life history models with p_0, p, a^*_m, and m^* obtained from Witting (2024a), $\tilde{a}_m = 1$, and relative birth ($\tilde{m}$) and initial abundance ($n_{t=0}$) at time-step zero ($t = 0$) estimated from data.

Hyperexponential growth. The age-structured abundance ($n_{a,t=0} = n_{t=0} c^*_a / \sum_{a\geq 1} c^*_a$) at time zero is the only initial condition of the exponential and density regulated models. A uniform $q_{a,t=0} = q_{t=0}$ vector of competitive quality (q) by age is an additional initial condition in the selection models where the projection of offspring quality ($q_{0,t}$) is given by selection acting on the quality of parent ($q_{a\geq a_m,t}$; Witting 1997, 2000b).

I use competitive quality as a joint measure of the energy requiring traits that intra-specific interactive competition selects to enhance the competitive ability of individuals (traits like body mass, group size, and interactive behaviour; Witting 1997, 2017b). The energetic quality-quantity trade-off

$$\tilde{m}_{a,t} = 1/q_{a,t} \tag{2}$$

is then defining the relative birth rate, with time used to build quality defining relative reproductive maturity

($\tilde{a}_m$) proportional to quality

$$\tilde{a}_{m,a,t} = q_{a,t} \tag{3}$$

when included as a selection response in the extended analysis of the PDDI data, with $q^* = 1$ for all a at equilibrium with no growth.

I evaluate selection across the overall intra-population phenotypic variation (subscript i) in individual competitive quality covering the intra- and inter-age-class variation implicitly. Following Witting (1997, 2000b), I calculate the selection gradient on log scale ($\partial r_{i,t}/\partial \ln q_{i,t}|_{q_{i,t}=q_t}$) at the limit of the average ($q_i = q$) quality in the population. Then, by extending the logic of the secondary theorem of natural selection (Robertson 1968; Taylor 1996) to inclusive inheritance (Mameli 2004; Danchin et al. 2011), the selected change in ln competitive quality—and thus indirectly in reproduction and reproductive maturity by eqns 2 and 3—is approximated as a product

$$-\gamma_\iota = \sigma\ \partial r_{i,t}/\partial \ln q_{i,t}|_{q_{i,t}=q_t} \tag{4}$$

between the selection gradient/pressure and the population response ($\sigma \geq 0$) of the entire inclusive inheritance system per unit selection, with γ_ι being a time invariant selection response for the hyper-exponential model, and $-\sigma$ reducing to the additive genetic variance for genetic evolution.

When there is no interactive competition and all individuals have equal access to resources, the intra-population variation in the growth rate is approximately $r_{i,t} \propto -\ln q_{i,t}$ from eqns 1 and 2, with a time invariant selection gradient of $\partial r_{i,t}/\partial \ln q_{i,t}|_{q_{i,t}=q_t} = -1$ with $\gamma_\iota = \sigma > 0$. This is the limit case of hyperexponential growth at zero population density. Yet, I allow for positive and negative γ_ι values to capture constantly accelerating ($\gamma_\iota > 0$) and decelerating ($\gamma_\iota < 0$) growth ($\gamma_\iota = 0$ is exponential growth). As the selection gradient on the per-generation growth rate is $\partial r_{i,t}/\partial r_{i,t}|_{r_{i,t}=r_t} = -\ \partial r_{i,t}/\partial \ln q_{i,t}|_{q_{i,t}=q_t}$, the acceleration/deceleration of the growth rate is

$$\dot{r} = dr/dt \approx \gamma_\iota \tag{5}$$

With the selection response acting on log scale, the average offspring quality (q_0) at time t is a product

$$q_{0,t} = q_t e^{-\gamma_\iota} \tag{6}$$

between the time invariant selection response $e^{-\gamma_\iota}$ and the average quality of the mature individuals

$$q_t = \frac{\sum_{a|a\geq a_{m,a,t}} q_{a,t} n_{a,t}}{\sum_{a|a\geq a_{m,a,t}} n_{a,t}} \tag{7}$$

With eqns 6 and 7 capturing the necessary response of selection for the age-structured population model, I do not have to include the intra- and inter-age-class variation in competitive quality and fecundity explicitly into the calculation of the selection response. Hence, I avoid dealing with complex individual-based models to track the differentiation in competitive qualities, resource availabilities, and reproductive rates across all the individuals in the population.

The hyperexponential model is structurally more complex than the exponential, yet it has only a single parameter (γ_ι) and two initial conditions ($n_{t=0}$ & $q_{t=0}$) to estimate from data.

Density regulated growth. The density regulated models resemble the exponential models, but they have a Pella and Tomlinson (1969) regulated relative reproduction

$$\tilde{m} = 1 + [\hat{m} - 1][1 - (n/n^*)^\gamma] \tag{8}$$

with three parameters (maximum relative birth rate $\hat{m}$; density regulation γ; equilibrium abundance n^*) and one initial condition ($n_{t=0}$) to estimate from data.

Selection regulated dynamics. The selection regulated models are structurally similar to the hyper-exponential models, but they have density and selection regulation acting on relative reproduction (and also on relative age of maturity for the extended PDDI analysis). This density regulation is a log-linear perturbation

$$\begin{aligned} m_{a,t} &= m^* \tilde{m}_{a,t}(n^*/n_t)^\gamma \\ a_{m,a,t} &= a^*_m \tilde{a}_{m,a,t}(n_t/n^*)^\gamma \end{aligned} \tag{9}$$

of the life history that evolves (changes in $\tilde{m}_{a,t}$ & $\tilde{a}_{m,a,t}$ by eqns 2, 3, and 10) around its evolutionary equilibrium ($\tilde{m}^* = \tilde{a}^*_m = 1$). Having a life history that is selected/adapted to the resource level at population dynamic equilibrium, the density regulation function of the selection regulated model operates as a local approximation around the equilibrium density, lacking the maximum growth (r_{max}) / birth ($\hat{m}$) rate parameter that is defined explicitly for zero density in traditional density regulation models (eqn 8).

Following Witting (1997, 2000b) in my appendix I derive the changes in competitive quality by the population dynamic feedback selection of density dependent interactive competition as

$$q_{0,t} = q_t(n_t/n^*)^{\gamma_\iota} \tag{10}$$

with q_t from eqn 7 and the selection induced acceleration/deceleration of the growth rate

$$\dot{r}_t \approx \gamma_\iota \ln(n^*/n_t) \tag{11}$$

being a log-linear function of the density dependent ecology. This model has three parameters (γ, γ_ι, & n^*) and two initial conditions ($n_{t=0}$ & $q_{t=0}$) to estimate from data.

The selection behind eqn 10 is based on the counteracting selection of the quality-quantity trade-off and the biased resource access from density-frequency-dependent interactive competition (Witting 1997, 2000b). The explicit modelling of the selection requires equations that account for the intra-population variation in competitive quality, the density dependence in the level of interactive competition, and the biased resource access from this competition. Yet, the population dynamic response of the selection converges on the population level response of eqn 10 (see appendix).

The dynamics of population dynamic feedback selection is cyclic, and I calculate the cycle period (T, in generations) and damping ratio (ζ) to describe it. The damping ratio is one for the monotonic return of density regulated growth ($\gamma_\iota = 0$ & $\gamma > 0$), it is between zero and one for damped cyclic dynamics ($0 < \gamma_\iota/\gamma <\approx 1$), zero for stable cycles ($\gamma_\iota/\gamma \approx 1$), and it declines to minus one for increasingly unstable cycles with amplitudes that increase over time instead of dampening out ($\gamma_\iota/\gamma >\approx 1$, see appendix).

3 Results

Population models for 2,058 bird and 290 mammal populations passed the minimum fitting criterion during the first round of AIC model selection. This selected selection-based models in 79% of the cases for both birds and mammals, with selection regulated models selected in 43% of the cases, followed by 35% hyperexponential, 14% exponential, and 7.6% density regulated models.

The selection effects were more pronounced for the PDDI control timeseries. Here, AIC included selection in 92% of 267 chosen models, with selection regulated models selected in 69% of the cases, followed by 23% hyperexponential models, 4.9% exponential, and 3.4% density regulated models.

With selection regulation covering all models (exponential when $\gamma = \gamma_\iota = 0$; hyperexponential when $\gamma = 0$ & $\gamma_\iota \neq 0$; density regulated when $\gamma > 0$ & $\gamma_\iota = 0$), I use the second AIC selection of selection regulated models with and without a change in equilibrium to describe the dynamics. This resulted in 2,801 accepted models (2,480 for birds; 321 for mammals) that explained 80% of the variance in the data, on average.

Fig. 1 shows the estimated dynamics of 24 populations, with the Supplementary Information showing all models. Where density regulated growth returns monotonically to carrying capacity (like top left plot in Fig. 1), the majority of the estimated selection regulated dynamics have damped to stable population cycles. Few populations show exploding cycles with negative damping ratios, but these may reflect estimation uncertainty.

The estimated median selection regulation (γ_ι) is 0.36 (se:0.01) for birds and 0.72 (se:0.032) for mammals, with median density regulation (γ) being 0.31 (se:0.0088) for birds and 0.31 (se:0.028) for mammals. The left plots in Fig. 2 show the distributions of the relative strength of selection regulation [i.e., $\gamma_\iota/(\gamma + \gamma_\iota)$] across all timeseries with accepted selection regulated models. With median estimates of 0.55 (se:0.0058) for birds and 0.58 (se:0.017) for mammals, selection regulation is equally or more important than density regulation in most populations, with median regulation ratios (γ_ι/γ) of 1.2 (se:0.11) and 1.4 (se:0.37). These results resemble those of the PDDI controls, where relative selection regulation [$\gamma_\iota/(\gamma + \gamma_\iota)$] is 0.53 (se:0.012) at the median across 399 selection regulated models. Allowing for regulation on reproductive maturity among the PDDI controls, 47% of 408 AIC-selected models were regulated through the reproductive rate and age of maturity, with a median relative [$\gamma_\iota/(\gamma+\gamma_\iota)$] selection regulation of 0.61 (se:0.019) across all models.

The distributions of regulation estimates cover the range from almost pure selection regulation to almost pure density regulation. Only 7.3% of the bird and 6.5% of the mammal populations have selection regulation below 10% of total regulation by density and selection. These results cannot support the hypothesis that natural populations of birds and mammals are density regulated predominantly. With the results of the first and second round of AIC selections agreeing on limited support for pure density regulation, the structural difference (eqn 8 vs. eqn 9) in the density regulation functions of the density regulated and selection regulated models is not influencing the overall conclusion.

The middle plots in Fig. 2 show the distributions of the estimated damping ratios. With median damping ratios around 0.12 (se:0.0071) and 0.062 (se:0.021) the population dynamics of birds and mammals is best characterised as cyclic. 83% of the bird populations, and 85% of the mammals, have damping ratios that are smaller than 0.5. Only 7.3% of the bird populations, and 7.8% of mammals, have strongly damped density regulation like growth with damping ratios above 0.9.

The right plots in Fig. 2 show the distributions of the periods of the population cycles. Only 3% of the estimated periods are shorter than five generations. The

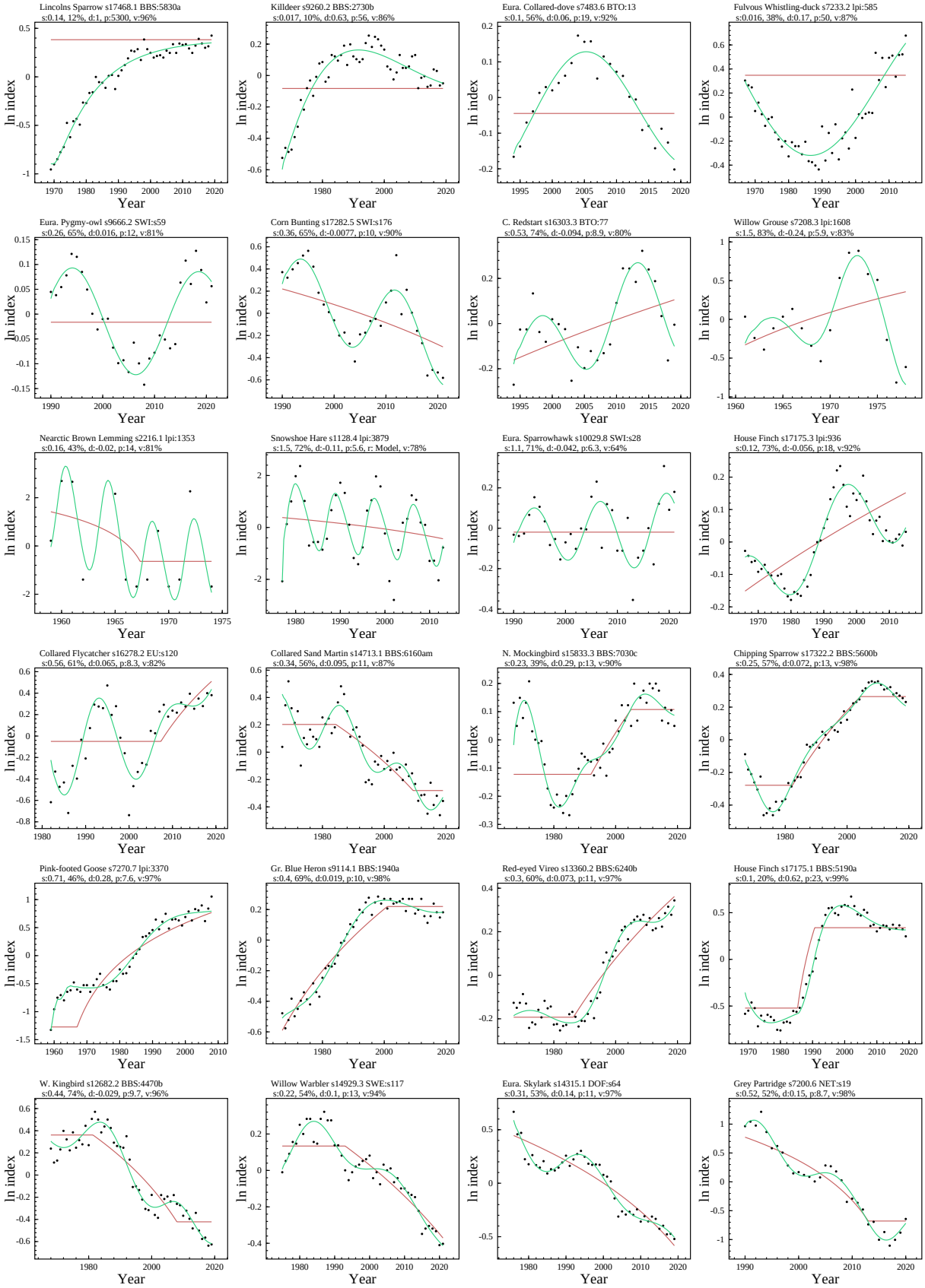

Figure 1: Examples of selection-regulated models fitted to population dynamic timeseries. Dots are index series of abundance, red lines the estimated equilibria, and green curves the model projections. Headings: name; id. nr; data reference; s:γ_ι & $\gamma_\iota/(\gamma_\iota+\gamma)$ in %; d:damping ratio; p:period in generations; v:explained variance in %.

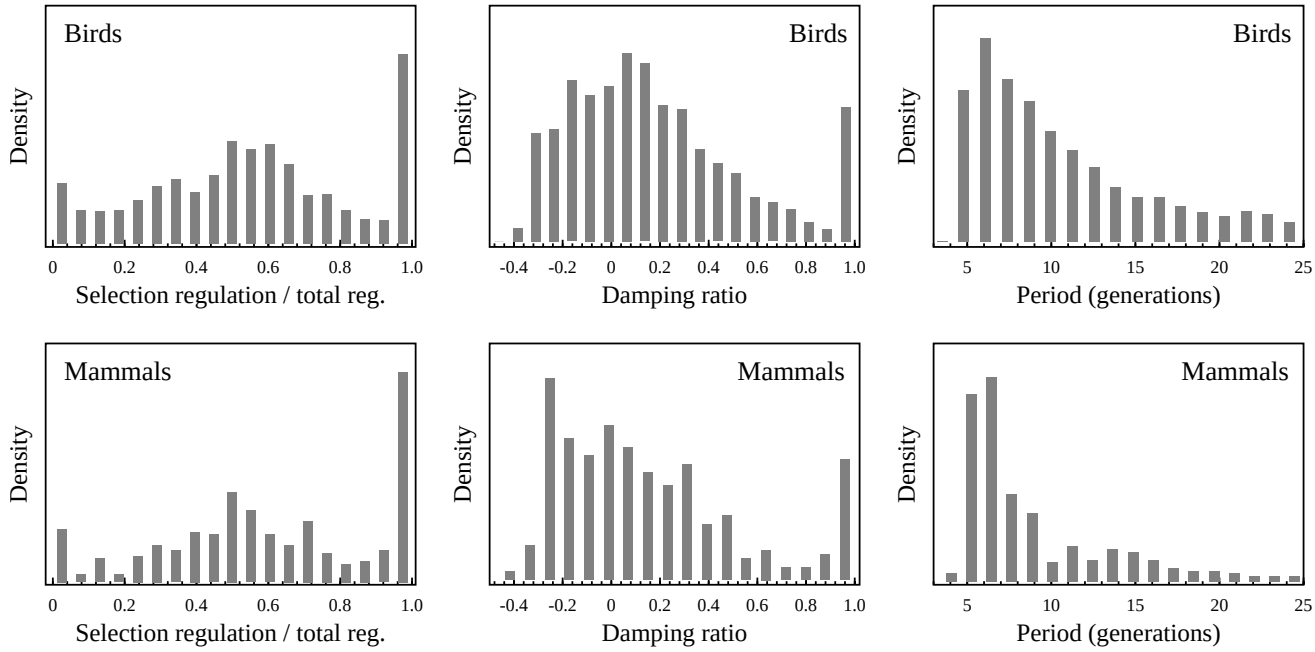

Figure 2: Distributions of the strength of selection regulation [divided by total regulation; $\gamma_\iota/(\gamma_\iota+\gamma)$], the damping ratio, and population period of the final AIC selected models for 2,480 bird and 321 mammal populations.

distributions have long tails toward long population periods, and they peak in the lower range with 46% of all birds, and 62% of all mammals, having periods below 10 generation. Median estimates are 11 (se:86) generations for birds and 7.9 (se:180) for mammals, and the period increases with stronger damping. The median period increases from 8.3 (se:0.99) and 7.2 (se:0.85) generations for birds and mammals with stable cycles (damping ratios around zero), to 28 (se:9.5) and 15 (se:1.8) for damping ratios around 0.8. This suggests that the selection response is usually somewhat slowly in the sense that it needs several generations to complete a selection cycle.

Across the AIC-chosen selection regulated models, the estimated equilibrium abundancies increase for 30% and 21% of the bird and mammal timeseries and decline for 29% and 14%. Yet, these apparent changes in the extrinsic environment do not determine the estimated local direction of growth. Where density regulated populations tend to decline only if the environment deteriorates with a declining equilibrium abundance, the selection regulated populations often decline about 50% of the time when the equilibrium is stable, declining, or increasing (Fig. 1, second line). For cases where the estimated trajectories are either declining or increasing locally, 76% of the population dynamic declines were not associated with an estimated decline in the equilibrium abundance, and that 77% of the population dynamic increases were not associated with an equilibrium increase. In fact, 23% of the local population declines had increasing equilibria, and 27% of the local increases had declining equilibria.

4 Discussion

By focussing on internal population regulation, I accounted for 80% of the variance in timeseries trajectories for 2,480 populations of birds and 321 populations of mammals. I used the life history models of Witting (2024a) to construct complete equilibrium age-structured population dynamic models, with the joint effects of density and selection regulation requiring the statistical estimation of only three parameters (density regulation, selection regulation, and equilibrium abundance) and two initial conditions (abundance and quality). While my estimates are model dependent, I used additional variance to account for random environmental perturbations and fluctuating abundance estimation, and I used trends in the equilibrium abundance to capture directional changes in extrinsic factors like habitats, resources, and predators. My models do not account for secondary phase related effects from e.g. cyclic changes in predation mortality, but I avoided most of these cases by excluding models with non-random residuals from my analysis.

I used AIC-selection to choose the best population

model for each timeseries, with selection-based models selected for 79% to 92% of the analysed timeseries, with median selection regulation estimated 1.2 (se:0.11) times stronger than density regulation. This identifies selection regulation as important and pure density regulation (with no selection) as the exception rather than the rule, i.e. unsuited as base-case regulation in birds and mammals. My statistical analysis does not resolve the underlying mechanisms of the population responses to the theoretically predicted density and selection pressures, but I can nevertheless conclude that the population dynamics of birds and mammals support the population regulation structure of natural selection. This involves the selection of life history cycles that last several generations, cycles that accelerate and decelerate population growth below and above the population dynamic equilibrium.

With selection regulation included we obtain a flexible single-species model that describes a broad range of the observed population dynamic trajectories (Fig. 1). These are cyclic with median damping ratios around 0.12 (se:0.0071) and 0.062 (se:0.021) for birds and mammals respectively, and population dynamic periods that increase with increased damping, with medians around 8.3 (se:0.99) and 7.2 (se:0.85) generations for stable cycles with damping ratios around zero.

4.1 Evidence of mechanisms

Apart from estimating the similarity between model predictions and abundance data, my analysis provides by itself no direct evidence of the density and selection regulation responses that drive the proposed dynamics. Yet, empirical studies have already documented several of the underlying selection mechanisms. The study by Sinervo et al. (2000) e.g. documents a natural population with a density-frequency-dependent feedback selection of interactive competition, including a selection for increased competitive quality and decreased growth above the average abundance, and selection for the opposite below. Where their clear-cut two-strategy system produces a stable two-generation oscillation, the gradual selection of the continuous traits that I study typically produces damped cycles with periods that last more than seven generations.

Turcotte et al. (2011) studied the alternative case with growing populations at a low abundance and documented, as predicted, a selection acceleration of the population dynamic growth rate by up to 40% over few generations, contrasting to pure density regulation that predicts a constant or declining growth rate for similar situations. And the selection of faster-spreading Covid-19 variants (Halley et al. 2021; Pavithran and Sujith 2022) document the limit case with a constantly accelerating growth rate and a hyper-exponential (instead of exponential) increase in abundance (Witting 2000a), as expected for pure r-selected replicators like virus and prokaryotes (Witting 2017b).

Other interesting cases are the upswings from a declining to an increasing population during the low phases of population cycles, which are predicted by the selection acceleration of the growth rate. This is a special case of evolutionary rescue, where selection accelerates the growth rate and prevents a population from going extinct by turning a population decline into an increase (e.g. Gomulkiewicz and Holt 1995; Agashe 2009; Bell and Gonzalez 2009; Ramsayer et al. 2013; Bell 2017). There are also other eco-evolutionary studies that document a wider array of population dynamic responses to natural selection (e.g. Thompson 1998; Yoshida et al. 2003; Hairston et al. 2005; Saccheri and Hanski 2006; Coulson et al. 2011; Hendry 2017; Brunner et al. 2019).

The documentation of the underlying responses to selection regulation in birds and mammals is more indirect, but there is nevertheless a wide range of evidence from the otherwise unresolved issues with population dynamic cycles. Here selection regulation predicts that the proximate cause of the cyclic dynamics is a special type of phase related life history change that does not follow from density dependence or predator-prey interactions. This prediction includes selection for lower reproduction, larger body masses, increased competitive behaviour (like aggression), larger kin groups, and more interacting males at high population densities, and selection for the opposite at low densities (Witting 1997, 2000b). This aligns with plenty of empirical evidence that includes a body mass cycle in the *Daphnia* experiments of Murdoch and McCauley (1985), with larger individuals occurring mainly in the late peak phase of the cycle, and smaller individuals mainly in the early increasing phase (Witting 2000b). Similar changes in body mass are widespread in voles and lemmings with cyclic dynamics (e.g. Boonstra and Krebs 1979; Lidicker and Ostfeld 1991; Norrdahl and Korpimäki 2002), and they have been observed in snowshoe hare (Hodges et al. 1999) and cyclic forest insects (Myers 1990; Simchuk et al. 1999).

As predicted, the percentage of males increases in small rodents when densities are high, while females predominate during the low phase (Naumov et al. 1969). Other cases of an increased male fraction with increased density include white-tailed deer (*Odocoileus virginianus*) (McCullough 1979) and northern elephant

seal (*Mirounga angustirostris*) (Le Boeuf and Briggs 1977). Individuals of voles and red grouse (*Lagopus lagopus scotica*) are, again as predicted, more aggressive at high than low population densities, with red grouse having larger kin groups evolving during the increasing phase of a cycle (e.g. Boonstra and Krebs 1979; Piertney et al. 2008).

While most of these empirical studies failed to identify the underlying cause of the observed phase related life history changes, the view that they are unrelated to natural selection seems untenable as the density-frequency-dependent feedback selection of interactive competition explicitly predicts them, while density regulation and trophic interactions do not explain them (at least not straightforwardly).

As there can be no cycle in abundance unless there is a cycle in at least one life history parameter, the phase related life history cycles are not secondary side effects with no relation to the underlying cause of the cycles. They are instead the necessary proximate cause that drives the population dynamics, and most likely the result of the proposed population dynamic feedback selection. Following a population dynamic perturbation, the predicted feedback cycle starts when the associated change in the level of interactive competition selects a life history change that initiates the first population cycle, with the resulting cyclic change in abundance repeating the cyclic selection that keeps the cyclic dynamics for its extended, but often damped, existence.

Low amplitude cycles are no longer a problem because the cyclic dynamics of selection regulation do not depend on high-density amplitudes for the build-up of predators, pathogens, or other detrimental factors. Selection regulation accelerates and decelerates population dynamic growth in smaller or larger steps around the equilibrium abundance, with deceleration above the equilibrium in high-amplitude cycles selecting a reproductive rate that stays low during the low phase of the cycle until the following phase of increase.

4.2 Conceptual implications

The estimated dynamics, and widely observed phase related life history changes, support population dynamic feedback selection as a common regulator of population dynamics, in addition to density regulation. This has population dynamic implications that make sense in a broader eco-evolutionary perspective.

A partial decoupling of population growth from the state of the environment is one intriguing consequence of selection regulation. Pure density regulation with no selection implies that a gradual change from a growing to a declining population, or from a declining to a growing population, requires a corresponding change in the extrinsic environment. This have led to the concept of indicators, where the population dynamic behaviour of indicator species is supposed to reflect the underlying state of the environment (e.g. LPI 2022; PECBMS 2022). While environmental changes may change the direction of growth, the direction may also change by selection in the absence of environmental changes. In other words, the growth of populations are not necessarily indicators of the environment. For about 75% of the estimated local population declines in this study I estimated no deteriorating environment.

Other essential results follow from the predicted population dynamic equilibrium, where the population dynamic feedback of interactive competition selects the population dynamic equilibrium abundance as an integral part of the selection attractor that determines the overall life history of a species (Witting 1997, 2008, 2024b). This selection scales with the selected net energy of the organism, and it explains part of the observed inter-specific variation in abundance as an allometric response to the naturally selected variation in net energy and mass (Witting 1995, 2023a), resolving issues with a density regulation theory that does not explain the abundance of animals (May 2020).

This naturally selected life history attractor selects net assimilated energy into population growth until the population dynamic equilibrium abundance generates a level of interactive competition where the monopolisation of resources by the larger-than-average individuals balances the quality-quantity trade-off that selects for smaller masses. This distribution of resources makes all the body mass variants approximately equally fit, adapting the life history to the resource level at population dynamic equilibrium (Witting 1997). Density regulation is thus selected as a local response of a life history that evolves around an adaptive optimum with zero growth ($r^* = 0$), and this breaks down the assumed r_{max} to r^* tension of traditional density regulation that requires a rarely to never observed famine to supress optimal growth (r_{max}) at zero abundance to zero growth (r^*) at equilibrium. With zero population growth being part of the naturally selected life history optimum, selection is actively removing the famine of the traditional density regulation concept, selecting a balanced nature where the world is green, the majority of individual animals are healthy, and the depletion of resources plays a minor role for the evolution of animal abundancies (Witting 1997, 2008).

Population dynamics is then a temporal disturbance in the naturally selected population dynamic life history

equilibrium. Here it is mainly the response of the feedback selection on the age-structured demography that determines the period of the cyclic dynamics, the magnitude of the environmental perturbation that determines the amplitude, and the strength of density regulation relative to the selection response that determines the damping of the cycle. The eco-evolutionary population dynamic feedback may in this way select the life histories and abundancies of natural populations, with the interplay between the feedback selection, density regulation, and varying environment shaping the dynamics (visit https://mrLife.org to examine your own online simulations of the estimated selection regulated models).

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Supplementary Information

si-appendix Appendix

si-plot Population plots

si-cpp Population model in c++

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5 Appendix

5.1 Selection regulated model

I use the population dynamic equilibrium ($*$) age-structured demography of the species-specific life history models of Witting (2024a). These formulate the age-structured demography from a per-generation replication rate of unity

$$\lambda^* = l_0 m^* t_r/2 = 1 \tag{12}$$

where $r^* = \ln \lambda^* = 0$, and the survival of offspring to the age of reproductive maturity (a^*_m) is $l_0 = p_0 p^{a^*_m - 1}$, with p_0 being survival of age-class zero individuals, p survival of older individuals, m^* the equilibrium reproductive rate of mature females (assuming an even sex ratio), and $t_r = 1/(1-p)$ the expected reproductive period given individuals that mature.

The age-structure is parameterised from the annual estimates $\tilde{a}_m$, $\tilde{m}^*$, and $\tilde{t}_r$ for models iterated in annual timesteps $\Delta t = 1$. But for species with a small $\tilde{a}_m$ and/or short $\tilde{t}_r$ I use shorter timesteps to ensure $\Delta t \ll \min(\tilde{a_m}, \tilde{t}_r)$. The parameters of my age-structured iteration models are then $a_m = \tilde{a}_m/\Delta t$, $m^* = \tilde{m}^* \Delta t$, and $t_r = \tilde{t}_r/\Delta t$, with the survival of individuals in 1+ age-classes being $p = (t_r - 1)/t_r$, and age-class zero survival being $p_0 = 2/t_r m^* p^{a_m - 1}$ from eqn 12.

I use population dynamic models that simulate at the level of the average values of each age-class, with $x \gg a_m$ denoting the maximum lumped age-class. The number $n_{a,t}$ of individuals in age-class $0 < a < x$ at timestep t is then

$$n_{a,t} = p_{a-1} n_{a-1,t-1} \tag{13}$$

with the number in age-class x being

$$n_{x,t} = p_x n_{x,t-1} + p_{x-1} n_{x-1,t-1} \tag{14}$$

with $p_a = p_0$ for $a = 0$ and $p_a = p$ for $a \geq 1$.

I let selection operate on the competitive qualities (q) of individuals, with quality defining a relative intrinsic birth rate $\tilde{m}$ by the quality-quantity trade-off

$$\tilde{m}_{a,t} = 1/q_{a,t} \tag{15}$$

and time required to build quality defining the relative intrinsic age of reproductive maturity

$$\tilde{a}_{m,a,t} = q_{a,t} \tag{16}$$

in proportion to quality, with all individuals having $q^* = 1$, $\tilde{m}^*_{a,t} = 1$, and $\tilde{a}^*_{m,a,t} = 1$ at the naturally selected population dynamic equilibrium. Assuming no

change in the quality of a cohort over time, we have $q_{a,t} = q_{a-1,t-1}$ and

$$q_{x,t} = \frac{q_{x,t-1}p_x n_{x,t-1} + q_{x-1,t-1}p_{x-1}n_{x-1,t-1}}{n_{x,t}} \quad (17)$$

with offspring quality given by the population level selection response of eqn 26.

To formulate density dependence let the number of individuals in each age-class relate to time just after each timestep transition, with offspring at t being produced by the $t-1$ individuals that survive to the $t-1 \to t$ transition, with the density dependent ecology being approximated by the average 1+ abundance of the two timesteps:

$$\hat{n}_t = 0.5 \sum_{a \geq 1} n_{a,t} + n_{a,t-1}. \quad (18)$$

Density regulation is then defining the realised age-structured reproduction and age of maturity

$$\begin{aligned} m_{a,t} &= m^* \tilde{m}_{a,t} (\hat{n}^*/\hat{n}_t)^\gamma \\ a_{m,a,t} &= a^*_m \tilde{a}_{m,a,t} (\hat{n}_t/\hat{n}^*)^\gamma \end{aligned} \quad (19)$$

as a log-linear deviation from the intrinsic equilibrium scaled parameters $m^* \tilde{m}_{a,t}$ and $a^*_m \tilde{a}_{m,a,t}$, with γ being the strength of regulation, and the number of offspring in age-class zero being

$$n_{0,t} = 0.5 \sum_{a|a \geq a_{m,a,t}} m_{a,t} n_{a,t}. \quad (20)$$

As it is the fitness variation among the quality variants in the population that imposes natural selection, I use the population level model of eqn 12 to turn the intra-population phenotypic variation (denoted by subscript i) in quality and fitness into a population level response that determines the average competitive quality of the offspring in age-class zero. For this I follow Witting (1997, 2000b) and define the intra-population variation in relative fitness as

$$r_{i,t} \propto -\ln q_{i,t} + \psi \tilde{\gamma} \ln n_t (\ln q_{i,t} - \ln q_t) \quad (21)$$

with the variant specific quality term $(-\ln q_{i,t})$ representing the influence of the quality-quantity trade-off (eqn 15) on the growth rate (eqn 12), and the right-hand part of the equation representing the density-frequency-dependent selection of interactive competition, with $\tilde{\gamma} \ln n_t$ being the density dependence in the level of interactive competition, and ψ the intra-population differentiation in resource access across the differentiation in competitive quality ($\ln q_{i,t} - \ln q_t$; q_t is average quality at timestep t) per unit interactive competition. This generates the selection gradient/pressure

$$\partial r_{i,t} / \partial \ln q_{i,t}|_{q_{i,t}=q_t} = \psi \tilde{\gamma} \ln n_t - 1 \quad (22)$$

and the following population response

$$d \ln q_t / dt = (\psi \tilde{\gamma} \ln n_t - 1)\sigma \quad (23)$$

from a proportional response ($\sigma \geq 0$) of the inclusive inheritance system (Mameli 2004; Danchin et al. 2011) when applying the logic of the secondary theorem of natural selection (Robertson 1968; Taylor 1996). The response of eqn 23 transforms into a multiplicative formulation

$$\begin{aligned} q_t &= q_{t-1} e^{(\psi \tilde{\gamma} \ln n_t - 1)\sigma} \\ &= q_{t-1} n_t^{\psi \tilde{\gamma} \sigma} e^{-\sigma} \\ &= q_{t-1} (n_t/n^*)^{\psi \tilde{\gamma} \sigma} n^{*\psi \tilde{\gamma} \sigma} e^{-\sigma} \end{aligned} \quad (24)$$

that reduces to

$$q_t = q_{t-1} (n_t/n^*)^{\gamma_\iota} \quad (25)$$

as eqn 22 defines the selection determined equilibrium abundance as $n^* = e^{1/\psi\tilde{\gamma}}$, with $\gamma_\iota = \psi\tilde{\gamma}\sigma$ being the selection response parameter. We may thus approximate average offspring quality

$$q_{0,t} = \frac{\sum_{a|a \geq a_{m,a,t}} q_{a,t} n_{a,t}}{\sum_{a|a \geq a_{m,a,t}} n_{a,t}} \left(\frac{\hat{n}_t}{\hat{n}^*} \right)^{\gamma_\iota} \quad (26)$$

as the average quality of the mature component multiplied by the density-frequency-dependent selection.

As I keep the species-specific equilibrium demographic models of Witting (2024a) constant, I am only estimating three parameters (n^*, γ, γ_ι) and two initial conditions ($n_{t=0}$, $q_{t=0}$) from the abundance data. For the initial conditions of an iteration, I use the same quality across all individuals and distributes the initial abundance according to the stable age-structure

$$c_a = l_a / \sum_{a \geq 0} l_a \quad (27)$$

where $l_0 = 1$, $l_a = p_0\, p^{a-1}$ for $1 \leq a < x$, and $l_x = p_0\, p^{x-1}/(1-p)$.

5.2 Selection regulated dynamics

Witting (1997, 2000b) described the population dynamic behaviour of a discrete selection-regulated model with non-overlapping generations. This model has damped population cycles when $\gamma_\iota < \gamma$, neutrally stable

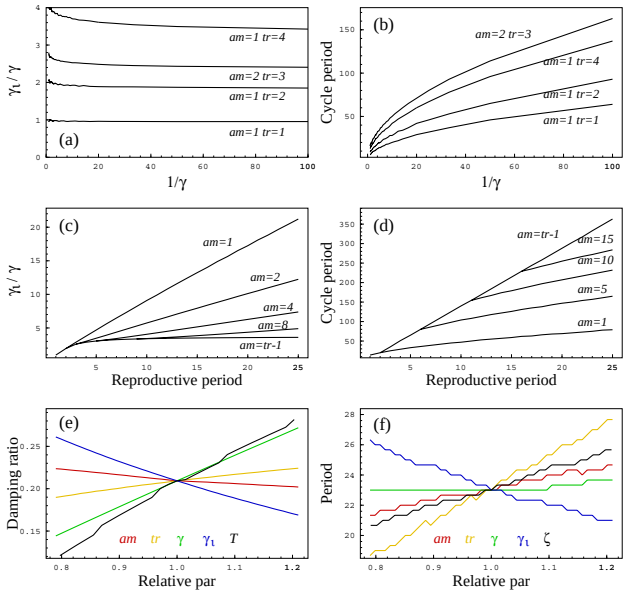

Figure 3: **Dynamic behaviour. Plot a to d:** The γ_ι/γ-ratio, and period (in years), of a stable population cycle ($\zeta = 0$) as a function of $1/\gamma$ (plot **a** and **b**) and the reproductive period (plot **c** and **d** for $\gamma = 0.2$), for different combinations of tm and tr. **Plot e and f:** The damping ratio (ζ) and population period (T) as a function of the parameters $x \in \{am, tr, \gamma, \gamma_\iota, \zeta, T\}$, relative ($x/\hat{x}$) to $\hat{x} \in \{am = 1, tr = 2.8, \gamma = 0.51, \gamma_\iota = 0.76, \zeta = 0.21, T = 23\}$. The dependence on T in plot **e**, and on ζ in plot **f**, is given by their responses to changes in γ_ι.

cycles when $\gamma_\iota = \gamma$, and repelling cycles when $\gamma_\iota > \gamma$. The population period of the stable cycles increases from four to an infinite number of generations as the $\gamma_\iota = \gamma$ parameters decline from two to zero. For a given γ the period increases with a decline in γ_ι, i.e., with an increasingly damped cycle. When, for a stable cycle, $\gamma_\iota = \gamma$ increases from two to four, there is an extra period in the amplitude of the population period, with the latter declining monotonically to two generations, with the dynamics becoming chaotic when $\gamma_\iota = \gamma$ increases beyond four.

The age-structured model with overlapping generations behave in a similar way, but the dynamics depend on the age of reproductive maturity (a_m) and the reproductive period [$t_r = 1/(1-p)$]. The age-structured model converges on the discrete model as $a_m \to 1$, $t_r \to 1$, and $p \to 0$. With no regulation on maturity, the period (T) of the stable population cycle remains a declining function of γ (Fig. 3b), with the slope/exponent (β) of the $\ln T \propto \beta \ln \gamma$ relation being -0.5 (estimated by linear regression). The cyclic dynamics become more and more stable with a decline in γ_ι, but the damping is also dependent on a_m and t_r. The stable cycle, e.g., has a γ_ι/γ ratio that increases beyond unity as a_m and t_r increase above unity (Fig. 3a and c). For any given combination of a_m and t_r, the stable cycle has a γ_ι/γ ratio that is almost constant (Fig. 3a).

For a given γ, the period of the stable population cycle increases almost linearly with an increase in a_m and t_r (Fig. 3d), with the period dependence on γ being somewhat elevated relative to the discrete model where $a_m = t_r = 1$ (Fig. 3b). Hence, for populations where γ is independent of a_m and t_r, we can expect an approximate linear relation between the population period T and life history periods like a_m and t_r.

When only one parameter is altered at the time, the period is almost invariant of γ (Fig. 3f). This reflects that the decline in period with an increase in γ for dynamics with a given damping ratio, is counterbalanced by the increase in period that is caused by the increased stability of the cycle, as the γ_ι/γ ratio—that defines the damping ratio—declines with the increase in γ. For single parameter perturbations, the damping ratio is usually most strongly dependent on γ and γ_ι, showing only a small increase with t_r and a small decline with an increase in a_m (Fig. 3e).

To describe the cyclic dynamics, I calculated the cycle period (T, in generations) and damping ratio (ζ). The damping ratio is zero for a stable cycle, increasing to unity for the monotonic return of density regulated growth. I calculated the damping ratio

$$\zeta = \frac{1}{\sqrt{1 + 4\pi^2/\delta^2}} \tag{28}$$

by the logarithmic decrement $\delta = \ln(n_{p,1}/n_{p,2})$ of the two successive abundance peaks ($n_{p,1}$ and $n_{p,2}$) that follow from an equilibrium population that is initiated with a positive growth rate where $q_{a,t} = 2q^*/3$. The estimated period (T) is the number of generations between these two abundance peaks.

When the γ_ι/γ-ratio increases above one the dynamics become unstable with amplitudes that increase over time instead of dampening out. In these cases, I reverted $n_{p,1}$ and $n_{p,2}$ in the estimate of $\delta = \ln(n_{p,2}/n_{p,1})$ and multiplied the ratio by minus one, so that negative ζ values refer to exploding cycles, with the rate of explosion increasing as ζ declines from zero to minus one.

5.3 Model fitting & model selection

I use the likelihood (L) maximum to estimate the parameters of all models given log normally distributed

abundance data

$$\ln L \propto -\sum_t \frac{[\ln(\tilde{n}_t/n_t)]^2}{2cv_t^2} + \ln cv_t \tag{29}$$

where $\tilde{n}_t$ is the 1+ index estimate in year t, n_t is the corresponding model estimate, and the coefficient of variation of the index estimate on normal scale ($\tilde{cv}_t$) used in an approximation of the σ parameter of the log normal distribution [as $\sigma^2 = \ln(1 + cv^2)$], with $cv_t = \sqrt{\tilde{cv}_t^2 + cv^2}$ including an additional variance term (cv^2) of the data relative to the model projection, with cv estimated by the likelihood function (an additional $\ln \tilde{n}_t$ term is not included explicitly in the likelihood as it is redundant, i.e., always summing to the same value for a given timeseries).

I project each model for 100,000 random parameter sets and apply a Quasi-Newtonian minimiser to find a local likelihood maximum for the 100 best fits, with the global maximum being the maximum across the local maxima. I convert the global likelihood maximum of each model to AIC [$\alpha = 2(k - \ln L)$, k nr. of model parameters] to select the best model for each timeseries, calculating the fraction of the AIC selected models that include selection.

I include the fit of the additional variance (cv) in the models that are AIC selected. Relative to a case with no additional variance, this increases the relative likelihood of models with a poor fit, increasing their likelihood of being AIC-selected. Given that the non-selection models most often have the poorest fit to the abundance data, the exponential and density regulated models are slightly overrepresented among the AIC-selected models relative to a case with no additional variance, and this makes my conclusion on the importance of including selection into population dynamic modelling somewhat conservative.

To reduce the fitting of fluctuating dynamics to between-year variation of uncertain abundance estimates, I place an upper limit of 1.5 on the estimates of γ and γ_ι. To reduce other confounding effects, I use a minimum fitting criterion and analyse fitted models further only if the mean of the residuals is not significantly different from zero ($p < 0.05$ student's t), there is no significant autocorrelation in the residuals (lag 1 and 2), no significant correlation between the residuals and the model, and the model explains at least 50% of the variance.

A second model-selection includes five selection-regulated models. In addition to the original model with a stable equilibrium abundance, it includes four versions with a linear trend in the population dynamic equilibrium (n^*): *i*) a trend that covers the whole data period [1 extra parameter (final n^*) fitted by the likelihood]; ii) a trend that starts after the first data year [2 extra parameters: trend starting year and final n^*]; iii) a trend that ends before the last data year [2 extra parameters: final n^* and trend ending year]; and iv) a trend that starts after the first year and ends before the last year [3 extra parameters: start and end year of trend plus final n^*], with a minimum allowed trend period of five years. Following eye inspection of all fits, I allow AIC to select one year of catastrophic survival for ten populations with an obvious crash in abundance, adding two extra likelihood fitted parameters (year and year-specific 1+ survival). For seven large whale populations I subtract annual catches from the dynamics following Witting (2013; data from https://iwc.int).