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Natural selection shapes the population dynamics of birds and mammals

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Abstract With eco-evolutionary processes explaining a wide range of ecological dynamics, I analyse for natural selection effects in the population dynamics of birds and mammals. By fitting single-species age-structured population dynamic models to 3,369 and 483 timeseries of 900 and 208 species of birds and mammals, I estimate that selection-based models are 780 (se:1.3) to 150,000 (se:2) times more probable than models with no selection. 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 estimated dynamics is damped to stable cyclic with median population periods around seven and eight generations for stable cycles. The estimated cycles follow from selected life history changes that solve several population cycle enigmas, highlighting the population dynamic implications of a natural selection that predicts also the life histories and abundancies of animals.

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

1 Introduction

Population regulation shapes the dynamics of natural populations, but density regulation does not 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 often observed, density regulated populations will only increase, or decline, towards carrying capacity showing no change in the direction of growth (overcompensation from strong density regulation does not explain a gradual change in the direction of growth).

For the last century or so, variation in environmental drivers provided a conceptual solution to the lack of fit between density regulation theory and data. This explains population dynamic deviations that correlate with environmental fluctuations, climatic change, interspecific competition, predators, and prey. But the vast majority of the available timeseries of abundance estimates have no associated data on inter-specific interactions. The extend of the extrinsic influence is therefore unresolved in most cases, with the hypothesis serving—more often—as a convenient *ad hoc* concept 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 population mechanistic focus, explanations 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 population dynamic enigmas unresolved (Myers 2018; Oli 2019; Andreassen et al. 2021).

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 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 regu-

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lation, 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 real 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 lingering 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).

These issues do not question that external factors affect population dynamics, but 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 eco-evolutionary feedback of intra-specific natural selection that regulates the growth and abundance shaping the dynamics of natural populations (Witting 1997, 2000b). Natural selection may thus be the reason for the often-observed lack of fit between density regulation theory and data.

1.1 On natural selection regulation

Early work on the population dynamic implications of 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 (e.g. 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), and a genetic model-interpretation with two phenotypes and no time-lag in the selection response failed to produce population cycles, although cycles seemed plausible given interactions with extrinsic factors (Stenseth 1981, 1985).

The population response to natural selection, however, is usually delayed one generation from parents to offspring. This allows for theoretical selection driven population cycles (Witting 1997, 2000b), as confirmed also empirically by Sinervo et al. (2000) who documented a two-strategy two-generation selection driven oscillation in side-blotched lizard (*Uta stansburiana*). These findings were soon followed by other empirical studies that 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 deal with the population dynamics of the selected changes in the growth rate, I use a general age-structured life history model with continuous traits and an energetic quality-quantity trade-off (Smith and Fretwell 1974; Stearns 1992) between competitive quality and reproduction (many of the early considerations included a somewhat similar trade-off). My analysis is furthermore based on a widely accepted game theoretical approach with individual selection and Continuously Stable Strategies (Eshel 1983; Taylor 1989; Christiansen 1991); a mathematical selection framework that I integrated into population dynamic theory about 25 years ago to describe the population dynamic implications of the eco-evolutionary feedback selection that follows from the density-frequency-dependent interactive competition among the individuals in the population (Witting 1997, 2000a,b, 2002a). This density-frequency-dependence of interactive competition is essential to ensure the selection of a balanced life history with intermediate growth, as frequency-independent selection selects a continued increase in population dynamic growth (Fisher 1930).

The feedback selection of my model is the endpoint of a directional evolutionary unfolding where a continued selection increase in net energy for replication predicts a sequential natural selection of major lifeforms from virus-like replicators, over prokaryote- and protozoa-like self-replicating cells, to multicellular sexually reproducing animals (Witting 2002b, 2017b, 2024b). The theoretically deduced inter-specific allometries (Witting 1995, 2017a) of the predicted animals were recently used to estimate population ecological life history models for approximately 90% of the birds and mammals of

the world (Witting 2024a). In the present study, I use these equilibrium life history models to deal with the population dynamics that follow when external factors determine the initial abundance and initial competitive quality of the eco-evolutionary feedback selection of population dynamic growth.

The perturbations of the initial 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 dependent 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 selected population cycle is inversely related to the full selection response of the growth rate that involves the complete general (Mameli 2004) inclusive (Danchin et al. 2011) inheritance system of the population. Genetics is one component of this inheritance, others include epigenetic inheritance (Richards 2006; Bossdorf et al. 2008), parental effects (Boonstra and Hochachka 1997; Inchausti and Ginzburg 2009), and long-term selected phenotypic plasticity (Snell-Rood et al. 2018; Pfenning 2021) that allow individuals to respond directly to the cyclic changes in the selection pressure. Cultural inheritance (Danchin et al. 2011; Whitehead et al. 2019) is another factor, 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 keep the cultural heritable variance of the population.

As it is the complete response of the inclusive inheritance system to the selection pressure of the selection gradient that generates the change in population dynamic growth, we cannot exclude selection regulation just because one inheritance component—like genetics—is insufficient to account for the observed change. It is like density regulation, where we do not exclude potential regulation just because we have not yet found the underlying mechanistic details.

Several studies estimate 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 presence and strength of density and selection regulation across almost four thousand populations of birds and mammals.

2 Method

2.1 Data

I obtained 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 applied a moderate baseline filter to the data, and to identify results from long timeseries of high-quality data I selected a restricted subset of controls also. The baseline timeseries included 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 were 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 age-structure models to obtain realistic estimates of regulation. If I used discrete models 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 sampling issues are widespread with natural populations, where it is common to count only part of the population with year-to-year variation in the available fraction.

To reduce this overfitting to random sampling variation, I use species-specific age-structured models that scale the estimated dynamics to the right biological timescale accounting for the demographic delays and inertia in the dynamics. I also restricted the estimated strengths of regulation to avoid strong overcompensation, focussing on smoot population trajectories with additional sampling variation accounting for the year-to-year zig-zag variation in the abundance data (see section 2.3). 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-structure from timeseries, I obtained the equilibrium demographic parameters of the age-structured models of all species from Witting (2024a). Keeping these parameters fixed for each species, I estimated only the absolute abundance, the density and selection regulation parameters, and few initial conditions from the abundance data. I used the equilibrium age-structure as the initial age-distribution and, when necessary, I rescaled the yearly parameters for 3, 6, or 12 time-steps per year keeping the projection timesteps shorter than the age of maturity.

I developed exponential, hyperexponential, density regulated, and selection regulated models for each population. I found 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. This allowed me to estimate the relative probability of models with (hyperexponential & selection regulated) and without (exponential & density regulated) selection.

As the AIC chose selection-based models most often (see result section), I ran a second AIC model-selection to estimate the best selection regulated models for all populations. In addition to a stable equilibrium, this second selection included models with a linear trend in equilibrium density. This allowed me to quantify not only the relative strengths of regulation by density and selection, but to estimate also if population trends are indicators of underlying changes in the external environment (assuming that a change in equilibrium reflects improved or deteriorating external factors).

I made regulation by density and selection 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 $\tilde{a}_m$) that were set to unity at population dynamic equilibrium (m^* and a_m^*). As I fitted the 1+ component of the population to the abundance data, the estimated regulation includes regulation on offspring survival implicitly. Hence, I covered 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 Supplementary Appendix describes the selection regulated model, with essential differences between the four population models described below.

Exponential growth. This model has constant life history parameters, with the relative birth rate ($\tilde{m}$) and the initial abundance (n_t) estimated from data.

Hyperexponential growth. The age-structured abundance ($n_{a,t}$) is the only initial condition of the exponential and density regulated models. A vector of competitive quality by age ($q_{a,t}$) that evolve by naturally selection is an extra initial condition in the selection models (Witting 1997, 2000b). This quality defines the age-structured relative birth rate

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

and relative reproductive maturity (when included for PDDI timeseries)

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

with $q^* = 1$ for all a at the equilibrium with no growth.

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 competitive quality—and thus also indirectly in reproduction and reproductive age—is

$$q_{0,t} = q_t e^{-\gamma_\iota} \tag{3}$$

with average offspring quality (q_0) being a product between the average quality of the mature individuals

$$q_t = \frac{\sum_{a|a_{m,a,t} \leq a} q_{a,t} n_{a,t}}{\sum_{a|a_{m,a,t} \leq a} n_{a,t}} \tag{4}$$

and a selection response $e^{-\gamma_\iota}$, where

$$\gamma_\iota = -\sigma \; \partial r_i / \partial \ln q_i|_{q_i=q} \tag{5}$$

is the product between the selection gradient ($\partial r_i / \partial \ln q_i|_{q_i=q}$) across variants ($i$) and the population response ($\sigma \geq 0$) of the inclusive inheritance system per unit selection, with σ 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 of a discrete model is $r_i \propto -\ln q_i$ from eqn 1, with a selection gradient of $\partial r_i / \partial \ln q_i|_{q_i=q} = -1$ with $\gamma_\iota = \sigma > 0$. This is the limit case of hyperexponential growth at zero population density. Yet, I allowed 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 $-\partial r_i / \partial \ln q_i|_{q_i=q}$, the acceleration/deceleration of the growth rate is

$$\dot{r} = dr/dt = \gamma_\iota \tag{6}$$

The intra-population variation and the resulting population response of eqns 5 and 6 represent the underlying natural selection. Yet, it is not necessary to include the modelling of the intra-population variation into the population dynamic equations, which use only the average response of eqns 3 and 4.

The hyperexponential model is structurally more complex than the exponential, but it has only a single parameter (γ_ι) and two initial conditions (n_t & q_t) to estimate from data.

Density regulated growth. I use the Pella and Tomlinson (1969) formulation

$$\tilde{m} = 1 + [\hat{m} - 1][1 - (n/n^*)^\gamma] \tag{7}$$

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

Selection regulated dynamics. The selection regulated model includes density regulation

$$\begin{aligned} m_t &= m^* \tilde{m}_t (n^*/n_t)^\gamma \\ a_{m,t} &= a^*_m \tilde{a}_{m,t} (n_t/n^*)^\gamma \end{aligned} \tag{8}$$

as a log-linear perturbation of a life history that evolves (changes in $\tilde{m}_t$ & $\tilde{a}_{m,t}$ by eqns 1, 2, and 9) around its evolutionary equilibrium ($\tilde{m} = \tilde{a}_m = 1$; with density regulation on a_m occurring only with a_m selection in the extended PDDI analysis).

Witting (1997, 2000b) derived the changes in competitive quality by the population dynamic feedback selection of density dependent interactive competition as

$$q_{0,t} = q_t (n_t/n^*)^{\gamma_\iota} \tag{9}$$

with the selection induced acceleration/deceleration of the growth rate

$$\dot{r} = \gamma_\iota \ln(n^*/n_t) \tag{10}$$

being a log-linear function of the density dependent ecology.

The selection behind eqns 9 and 10 is based on the biased resource access from the density frequency dependent interactive competition. The explicit modelling of the selection requires equations that account for the intra-population variation in competitive quality and resource access (Witting 1997, 2000b). But the population dynamic equations use only the population level response of eqn 9.

The dynamics of population dynamic feedback selection is cyclic. Thus, I calculated the cycle period (T, in generations) and damping ratio (ζ) to characterise the dynamics. 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

$$\zeta = \frac{1}{\sqrt{1 + 4\pi^2/\delta^2}} \tag{11}$$

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 increase 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.

The selection regulated model has three parameters (γ, γ_ι, & n^*) and two initial conditions (n_t & q_t) to estimate from data.

2.3 Model fitting & model selection

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

$$\ln L = -\sum_t \frac{[\ln(\tilde{n}_t/n_t)]^2}{2cv_t^2} + \ln cv_t \qquad (12)$$

where $\tilde{n}_t$ is the 1+ index estimate in year t, n_t the corresponding model estimate, and $cv_t = \sqrt{\tilde{cv}_t^2 + cv^2}$ with $\tilde{cv}_t$ being the coefficient of variation of the index estimate, and cv the additional variance of the data (the latter estimated by the likelihood function).

I projected each model for 100,000 random parameter sets and applied 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 converted 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. I calculated the fraction of the AIC selected models that included selection, and the distribution of the probability ratio $p(s/d) = e^{(\alpha_s - \alpha_n)/2}$ of selection (s) versus non-selection (d) models, with the s and d models being hyperexponential versus exponential, or density regulated versus selection regulated, depending upon the best AIC-fitting model.

To avoid fitting fluctuating dynamics to between-year variation of uncertain abundance estimates, I placed an upper limit of 1.5 on the estimates of γ and γ_ι. To reduce other confounding effects, fitted models were analysed further only if the mean of the residuals were not significantly different from zero ($p < 0.05$ student't), there were no significant autocorrelation in the residuals (lag 1 and 2), no significant correlation between the residuals and the model, and the model explained at least 50% of the variance. I allowed one year of catastrophic survival for ten populations with a crash in abundance, and for seven large whale populations I subtracted annual catches from the dynamics following Witting (2013; data from https://iwc.int).

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 AIC selection chose selection-based models in 79% of the cases (79% for birds & 79% for mammals), with the selection-based models being 780 (se:1.3) times more probable on average (geometric mean) than population dynamic models with no selection (based on relative AIC). The AIC selection chose selection regulated models 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, the AIC included selection in 92% of 267 chosen models, with selection-based models being 150,000 (se:2) times more probable on average than non-selection models. The AIC selection chose selection regulated model 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

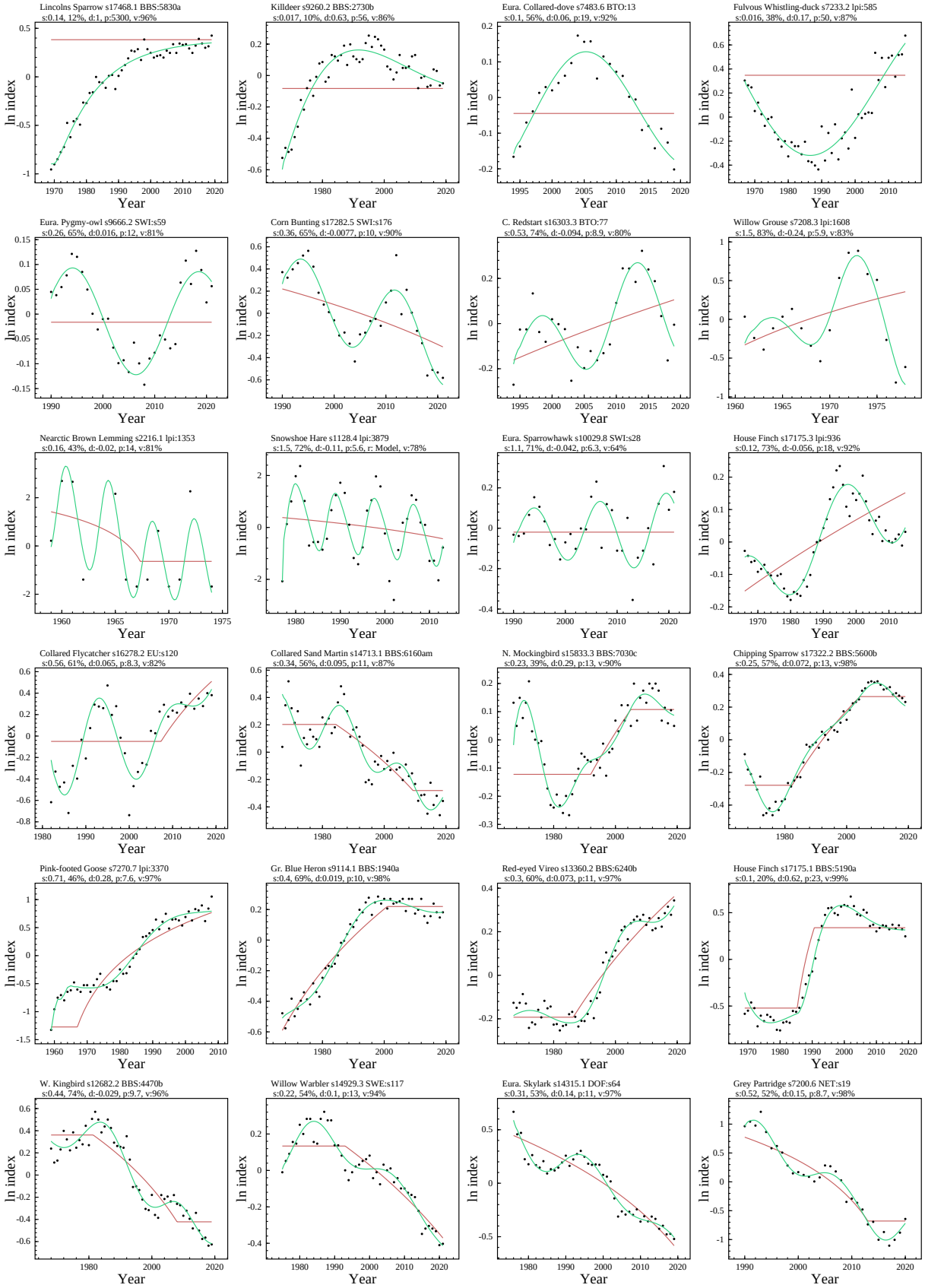

Figure 1: Examples of selection-regulated models fitted to population dynamic timeseries. Dots are index series of abundance, red lines the estimated equilibria, and green curves the model projections. Headings: name; id. nr; data reference; s:γ_ι & $\gamma_\iota/(\gamma_\iota+\gamma)$ in %; d:damping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

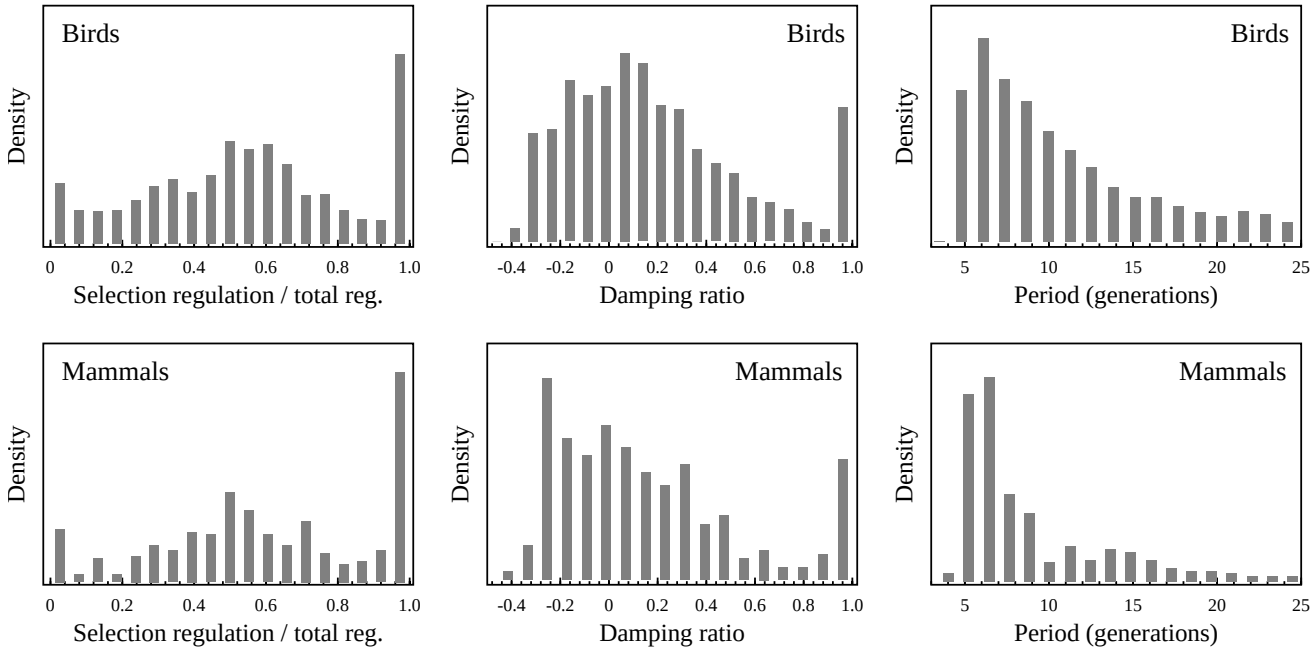

Figure 2: Distributions of the strength of selection regulation [divided by total regulation; $\gamma_\iota/(\gamma_\iota+\gamma)$], the damping ratio, and population period of the final AIC selected models for 2,480 bird and 321 mammal populations.

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.

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 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.

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 primarily 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 account for the demographic delays and inertia in the dynamics of each species. This allowed me to construct complete age-structured population dynamic models, where the joint effects of density and selection regulation required 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. The population dynamics of birds and mammals support the population regulation structure of natural selection, with accelerating and decelerating 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 mechanisms 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 natural populations 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 mechanism of 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 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 also 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 dependence and trophic interactions do not explain them (at least not straightforwardly). And, 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 just secondary side effects with no relation to the underlying cause of the cycles. They are instead the necessary proximate cause that drives the population cycles, and most likely the result of the proposed eco-evolutionary feedback selection. Following a population equilibrium 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 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 growth direction, the direction of growth 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 e.g. 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 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, with all body mass variants in the population being essentially equally fit at evolutionary equilibrium (Witting 1997). As this selection scales with the selected net energy of the organism, 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).

This natural selection attractor selects life histories that are adapted to the resources at population dynamic equilibrium, with density regulation selected as a local response of a life history that evolves around its adaptive optimum with zero growth ($r^* = 0$) at equilibrium. This breaks down the assumed fixed r_{max} to r^* tension of traditional density regulation, which requires a rarely to never observed famine that supresses optimal growth (r_{max}) at zero abundance to zero growth (r^*) at equilibrium. This natural selection breakdown of famine, and associated evolutionary adjustment of density regulation, makes additional sense in light of a non-selection population regulation theory that apparently cannot explain the abundancies of animals (May 2020). The end-result of population dynamic feedback selection is a balanced nature, where the world is green, and the majority of individual animals are healthy because the relative depletion of resources plays only 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.

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Supplementary Information

si-appendix Model appendix

si-plot Population plots

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5 Model appendix

The age-structure of each model is determined from three species specific parameters obtained from Witting (2024). These are the age of reproductive maturity (first reproductive event) in years ($\tilde{a}_m$), the annual rate of reproduction at population dynamic equilibrium ($\tilde{m}^*$), and the reproductive period ($\tilde{t}_r$). These parameters were converted to the appropriate timescale, with $a_m = \tilde{a}_m/\Delta t$, $m^* = \tilde{m}^*\Delta t$, and $t_r = \tilde{t}_r/\Delta t$ with $\Delta t \ll \min(\tilde{a_m}, \tilde{t}_r)$ being the timestep of the simulation model in years. The survival of all 1+ age-classes was then calculated as $p = (t_r - 1)/t_r$, with age-class zero survival being $p_0 = 2/t_r m^* p^{a_m - 1}$.

With $x \gg a_m$ being the maximum lumped age-class, the number $n_{a,t}$ of individuals of age $0 < a < x$ at timestep t is

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

and the number in age-class x

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

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

$$\hat{n}_t = 0.5 \sum_{a \geq 1} n_{a,t} + n_{a,t-1}. \tag{15}$$

Together with the quality-quantity trade-off, the competitive qualities of the individuals define their relative birth rate

$$\tilde{m}_{a,t} = 1/q_{a,t} \tag{16}$$

as well as their relative age of reproductive maturity

$$\tilde{a}_{m,a,t} = q_{a,t} \tag{17}$$

with the population dynamic equilibrium having $q^* = 1$ for all a. More generally $q_{a,t} = q_{a-1,t-1}$ and

$$q_{x,t} = \frac{q_{x,t-1} p_x n_{x,t-1} + q_{x-1,t-1} p_{x-1} n_{x-1,t-1}}{n_{x,t}} \tag{18}$$

assuming that there is no change in the quality of a cohort over time. The quality of offspring

$$q_{0,t} = \frac{\sum_{a|a_{m,a,t} \leq a} q_{a,t} n_{a,t}}{\sum_{a|a_{m,a,t} \leq a} n_{a,t}} \left(\frac{\hat{n}_t}{\hat{n}^*}\right)^{\gamma_\iota} \tag{19}$$

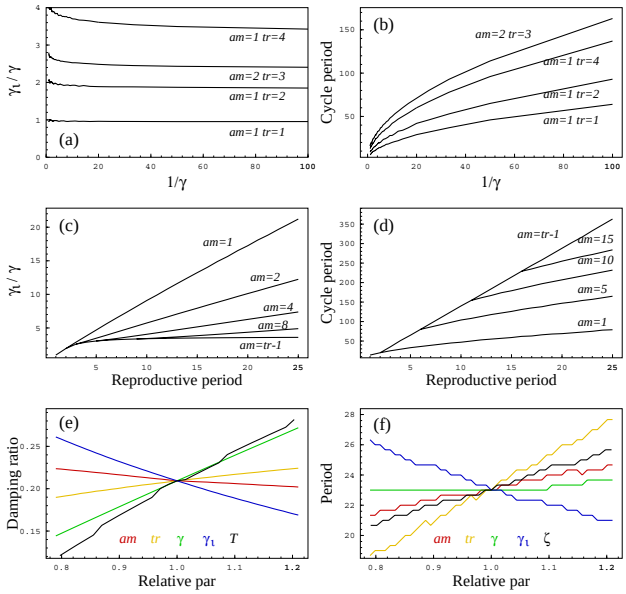

Figure 3: **Dynamic behaviour. Plot a to d:** The γ_ι/γ-ratio, and period (in years), of a stable population cycle ($\zeta = 0$) as a function of $1/\gamma$ (plot **a** and **b**) and the reproductive period (plot **c** and **d** for $\gamma = 0.2$), for different combinations of tm and tr. **Plot e 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.

is the average quality of the mature component multiplied by the density dependent selection, with γ_ι being the selection response.

Density regulation

$$\begin{aligned} m_{a,t} &= m^* \tilde{m}_{a,t} (\hat{n}^*/\hat{n}_t)^\gamma \\ a_{m,a,t} &= a_m^* \tilde{a}_{m,a,t} (\hat{n}_t/\hat{n}^*)^\gamma \end{aligned} \tag{20}$$

is formulated as a log-linear deviation from the equilibrium life history, with γ being the strength of regulation, and the number of offspring in age-class zero being

$$n_{0,t} = 0.5 \sum_{a|a_{m,a,t} \le a} m_{a,t} n_{a,t}. \tag{21}$$

The initial conditions of an iteration are the same quality across all individuals and the initial abundance with a stable age-structure

$$c_a = l_a / \sum_{a \ge 0} l_a \tag{22}$$

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

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