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Ecological changes with minor effect initiate evolution to delayed regime shifts

P. Catalina Chaparro-Pedraza[1,2*] and André M. de Roos[1,3]

Regime shifts have been documented in a variety of natural and social systems. These abrupt transitions produce dramatic shifts in the composition and functioning of socioecological systems. Existing theory on ecosystem resilience has only considered regime shifts to be caused by changes in external conditions beyond a tipping point and therefore lacks an evolutionary perspective. In this study, we show how a change in external conditions has little ecological effect and does not push the system beyond a tipping point. The change therefore does not cause an immediate regime shift but instead triggers an evolutionary process that drives a phenotypic trait beyond a tipping point, thereby resulting (after a substantial delay) in a selection-induced regime shift. Our finding draws attention to the fact that regime shifts observed in the present may result from changes in the distant past, and highlights the need for integrating evolutionary dynamics into the theoretical foundation for ecosystem resilience.

Regime shifts in diverse ecosystems, including lakes, coral reefs, deserts, woodlands and oceans have been attributed to the occurrence of qualitatively different, alternative stable states (ASSs) under the same set of external conditions[1,2]. These abrupt transitions between contrasting alternative ecosystem states occur as a consequence of small changes in external conditions. Current environmental changes are likely to increase the frequency and severity of regime shifts[3,4], increasing incurred societal costs and making the need to uncover the factors that trigger regime shifts an urgent matter[5–7]. Existing theory considers these catastrophic transitions to result from changes in external conditions crossing a threshold or tipping point (Fig. 1a)[1,8]. To identify factors triggering regime shifts, analysis has focused on correlations between time series of external conditions and ecosystem variables[9,10]. However, changes in external conditions may also trigger evolutionary responses[11,12], as exemplified by the rapid changes in phenotypic traits of wild populations due to changes in selective pressure that have been documented for many species[13–16].

Whereas theory has considered that evolution may lead to species extinction[17], the possibility that current ecological changes may induce future regime shifts as a consequence of an evolutionary response has mostly been overlooked. Here we postulate an eco-evolutionary mechanism (Fig. 1b) in which changes in external conditions of a system with ASSs do not push the system beyond a tipping point and may only have minor, immediate, ecological effects, but drastically alter the fitness landscape and thus the selective pressures on phenotypic traits. The ensuing natural selection causes gradual phenotypic change, which after a considerable delay drives the phenotype beyond a tipping point and results in the ecosystem undergoing an abrupt regime shift in ecological state.

We illustrate this mechanism by incorporating evolutionary dynamics into an ecological system in which previous studies showed that regime shifts can occur[18]. The system consists of a population of individuals that use two different habitats in consecutive stages of their life history. The development of an individual during its life history is represented by its growth in body size. The population thus links two different (spatially) segregated ecological communities, in each of which only a part of the population (either the small juveniles or the immatures and adults) plays a functional role. Changes in, for example, mortality risk in either of the two habitats will therefore result in changes in the composition and functioning of both communities, such as illustrated by the study of Knight et al.[19], who showed that variation in mortality of juvenile dragonflies in ponds led to simultaneous changes in pollination success of plants growing around these ponds. In the extreme case, the changes in external conditions can even cause regime shifts to occur in both communities simultaneously[18]. Changes in mortality risk, however, also affect the optimal body size at which to switch habitats[20]. This life history trait is fundamental in determining individual fitness[21,22] and, therefore, is subject to natural selection. Nonetheless, the evolution of this trait has been overlooked when investigating regime shifts. We use a population model[23] in which individuals are born in the nursery habitat, where they grow until they reach a specific body size. At this body size, individuals switch to the adult habitat (we refer to the two habitats as nursery and adult habitat, even though individuals switch habitats before maturation and the adult habitat is therefore occupied by adults as well as large juveniles). We allow the body size at which individuals switch habitats to evolve (see Methods for details) and use an approach that combines population genetics and adaptive dynamics to assess the eco-evolutionary consequences of the selection process that results from a seemingly beneficial ecological change: a reduction in mortality in the adult habitat.

Results

A decrease in mortality in the adult habitat does not immediately cause a regime shift but triggers an evolutionary response that, after a substantial delay, results in one. Before mortality is decreased, the population is at an ecologically and evolutionarily stable equilibrium in which the trait value (in scaled units) is high (time 0 to 100 in Fig. 2). Lower mortality experienced by individuals in the adult habitat, which may for instance result from the loss of a predator or a decrease in exploitation rate, destabilizes the ecological and evolutionary dynamics. The ecological dynamics change from a stable equilibrium to oscillatory dynamics (limit cycle) (Fig. 2 at time 100). Population biomass in the adult habitat fluctuates around the

[1]Institute for Biodiversity and Ecosystem Dynamics, University of Amsterdam, Amsterdam, the Netherlands. [2]Eawag—Swiss Federal Institute of Aquatic Science and Technology, Dübendorf, Switzerland. [3]The Santa Fe Institute, Santa Fe, NM, USA. *e-mail: Catalina.Chaparro@eawag.ch

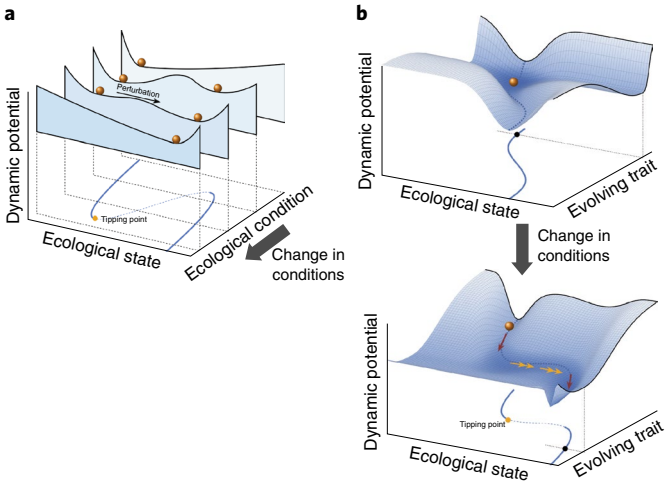

Fig. 1 | Potential ecological and evolutionary effects of changing conditions in ecosystems with ASSs. a, Existing theory in ecology postulates that regime shifts occur because external forces drive ecological conditions beyond a tipping point (yellow dot in bottom plane of plot; modified from ref. [1], Springer Nature Limited). **b**, We postulate an eco-evolutionary framework, in which changes in ecological conditions can also affect the fitness landscape of phenotypic traits. Initially the system is ecologically and evolutionarily stable. A change in ecological conditions (black arrow) with minor effects does not change ecological stability but alters selective pressures and thus destabilizes the evolutionary state (black dot in bottom plane of plots). Subsequently, natural selection causes gradual phenotypic change (brown single arrow) beyond a phenotype value at which an ecological tipping point occurs (yellow dot in bottom plane of plot). At this point, the system undergoes a delayed ecological regime shift (yellow double arrows).

same value as before the decrease in mortality, whereas population biomass in the nursery habitat on average increases. The population cycles are sustained if the trait variation is zero and mutations do not occur (Fig. 2 grey line), preventing natural selection to induce phenotypic changes. In contrast, with trait variation, individuals with a smaller trait value are selected for because the new conditions changed the selection pressures (black line in Fig. 2b). This evolutionary process initially stabilizes the ecological dynamics and dampens the oscillations, with food resource and population biomass densities slowly returning to similar values as occurred before the decrease in mortality (time 100 to 290, black line in Fig. 2). However, much later on, the evolutionary process causes a regime shift with substantially lower and higher population biomass levels in the nursery and adult habitat, respectively (after time 290, black line in Fig. 2). Although the change in total population biomass is small, the changes in population composition result in notable functional changes of the population. For instance, the increased population biomass in the adult habitat results in strong competition in this habitat, which causes reduced growth rates of larger individuals and a 32% decrease in maximum body size (detailed population compositions in the ASSs are shown in Extended Data Fig. 1). The regime shift therefore entails an abrupt change in the demographic composition of the population, which results in concomitant and similarly abrupt changes in the ecological communities linked by the population. As we limit our analysis, for simplicity, to the interaction of the consumer with its resources, these changes in the two ecological communities are reflected in the change in abundance of the food resources that it uses in the two different habitats. Lower amounts of trait variation cause slower evolutionary dynamics and, therefore, delay the upcoming regime shift. However, once the mean trait value reaches the threshold (tipping point) trait value, the regime shift trajectory followed by the population and its resource is not affected by the amount of trait variation in the population (see Extended Data Fig. 2).

The regime shift could not be determined when considering only the direct ecological consequences of the change in mortality. If individuals maintain the trait value that is evolutionarily stable for high mortality in the adult habitat, ASSs do not occur for any value of mortality in the adult habitat (Fig. 3a). The decrease in mortality merely causes the ecological dynamics to change from stable equilibrium to cyclic dynamics (yellow lines in Fig. 3a). In contrast, with lower mortality levels in the adult habitat, ASSs do occur for smaller trait values (Fig. 3b). Furthermore, the decrease in mortality in the adult habitat causes smaller trait values to be selected for (orange arrows). Natural selection thus drives the population to the region where ASSs exist and ultimately beyond the tipping point at which one of the ASSs disappears and a regime shift occurs (yellow vertical arrows at a scaled body size value of 0.24 in Fig. 3b).

The long-term ecological dynamics of the population depend on its evolutionary dynamics, either approaching a new ecological and evolutionary stable equilibrium after the first regime shift (Fig. 4a,c) or alternating perpetually with repeating regime shifts between the two ASSs (Fig. 4b,d). The latter scenario occurs when the evolutionary stable value for the trait corresponds to an equilibrium that is ecologically unstable, making the evolutionary endpoint ecologically unreachable (see Extended Data Fig. 3). Together with the ecological dynamics, the trait value oscillates over time (Fig. 4b,d) because in our model selection has opposing directions in the ASSs (see Extended Data Fig. 3). In the first scenario, the system will never return to its initial state without external intervention (in our case, an increase in mortality in the adult habitat), while in the latter scenario a return to the initial system state follows automatically from the intrinsic evolutionary dynamics. However, once in its original state an intervention to slow down the evolutionary process (for instance, by manipulating trait variation) will be necessary to avoid another regime shift to the alternative, potentially undesired state. Besides a change in the direction of selection, the evolutionary rates also differ substantially in the ASSs. Abrupt changes in the direction and strength of selection are not surprising given that the two ASSs correspond to qualitatively very different ecological conditions[1] causing selective forces to be different as well.

Discussion

Faced with novel conditions, populations can experience rapid contemporary evolution[16,24,25], which may mitigate ecological impacts on wild populations[12]. In accordance, we show that the evolutionary process in response to a change in external conditions seemingly restores the state of the two ecosystems linked by the population with a habitat switch. However, this apparent adaptive response later on drives the trait value beyond the tipping point causing a regime shift. Although phenotypic change in populations has been considered essential for adaptation to novel conditions[26], our results highlight that it can also precipitate the regime shift of ecosystems with ASSs because, paradoxically, it fuels the adaptive process that results in a delayed regime shift.

We illustrated this delayed regime shift using a population of a species with a habitat switch. In this system, the regime shift entails a dramatic change in the composition of the population that suddenly decreases and increases its biomass levels in the nursery and adult habitat, respectively. Although these changes in the biomass levels of different developmental stages occur without a major change in the total population biomass, their ecological consequences can result in cascading impacts on the local communities in either habitat. It is increasingly acknowledged that ecological differences among developmental stages within species can rival or even exceed differences between species[20,27]. For instance, increasing body size is correlated with increasing trophic levels[28]. Indeed, most predator species in aquatic ecosystems begin their life as larvae

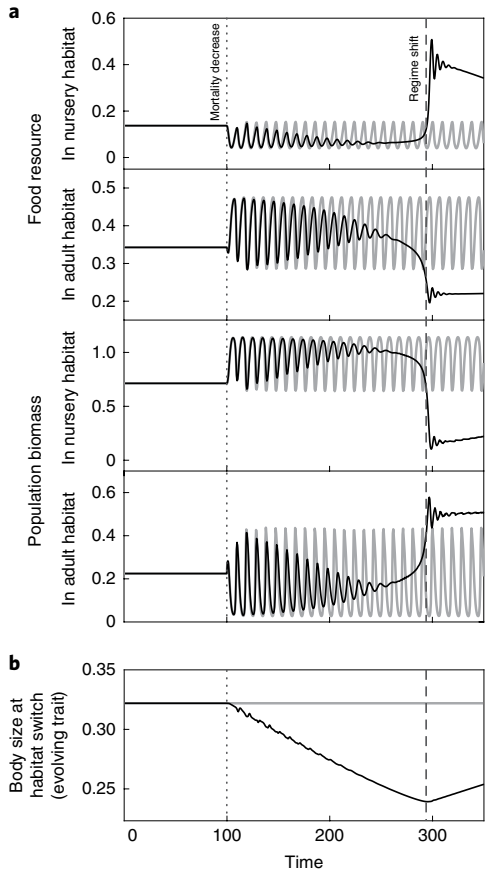

Fig. 2 | Eco-evolutionary dynamics of a population with a habitat switch experiencing a decrease in mortality in the adult habitat. a,**b**, Ecological (**a**) and evolutionary (**b**) dynamics of a population, whose individuals switch from a nursery to an adult habitat at a particular body size, before and after a reduction of mortality in the adult habitat (at time 100, vertical dotted line, from 2 to 1.5). Without trait variation in the population (grey lines), the change in mortality causes the stable equilibrium state to develop into stable population cycles, whereas when there is trait variation (black lines), a delayed regime shift occurs (vertical dashed line) after evolution to smaller size at habitat switch. Trait variation is represented with a truncated normal distribution with a minimum and maximum value equal to 80% and 120% of the mean trait value; mortality in the nursery habitat equals 0.8; other parameters are given in Table 1. All units are scaled units (see Methods).

Table 1 | Scaled parameter values

Description	Symbol	Relation with unscaled parameters	Value
Ratio of maximum resource density in habitats 1 and 2	ρ	$\rho = \frac{D_2}{D_1}$	0.5
Resource growth rate	δ	$\delta = \theta\sqrt{\frac{s_m - s_b}{\epsilon_g a_1 D_1}}$	1
Mortality in habitat 1	η_1	$\eta_1 = \mu_1\sqrt{\frac{s_m - s_b}{\epsilon_g a_1 D_1}}$	Varied
Mortality in habitat 2	η_2	$\eta_2 = \mu_2\sqrt{\frac{s_m - s_b}{\epsilon_g a_1 D_1}}$	Varied
Attack rate ratio on food resource of habitats 1 and 2	q	$q = \frac{a_2}{a_1}$	1
Adult fecundity scaled constant	β	$\beta = \frac{(s_m - s_b)\epsilon_b}{\epsilon_g}$	2,000
Body size at the habitat switch	w_s	$w_s = \frac{s_s - s_b}{s_m - s_b}$	Evolving trait

at the base of food chains and as they growth larger their trophic level increases[29,30]. Therefore, juveniles have a very different ecological niche and function than adults of the same species. As a consequence, a sudden change in biomass of a particular developmental stage in the population may result in a change in the net effect of the population on the ecological system. This effect may be even stronger in populations with a habitat switch because their biomass is distributed over two different habitats and they link two separate ecological communities. Hence, if the juveniles in one habitat are mainly food source for larger predators due to their small size, and large juveniles and adults in the other habitat are mainly predators of other species, an abrupt change in the biomass of these stages would propagate through the two communities of which these different stages are part. Empirical evidence has demonstrated that ontogenetic habitat switches have important consequences across different communities and ecosystems: changes in abundance and composition of populations with a habitat switch have been shown to alter ecosystem functions such as pollination[19], as well as predator–prey interactions and energy pathways in ecosystems[31]. Furthermore, changes in the composition of populations can alter community functioning and ecosystem processes even before the collapse of any species in the community[32].

The regime shift presented here represents an abrupt transition between different demographic structures of a population that can result in simultaneous changes in the composition and functioning of the communities that this population inhabits. Such changes in the community composition and functioning can, in turn, alter ecosystem services. For instance, we show that, following the regime shift, the increase in biomass in the adult habitat causes reduced growth rates in this habitat and thus a substantial drop in maximum body size (32%). Several fish species of economic importance have life cycles that include a habitat switch, such as salmon, sturgeon and trout. Given that often large individuals have higher economic value per unit of biomass than small individuals[33], the regime shift may alter the harvestable biomass and economic profitability of a fish stock with a habitat switch.

In this system, the trait under selection is directly involved in the mechanism underlying the ASSs. Schreiber and Rudolf[18] showed that ASSs can occur in a population using two distinct habitats in different developmental stages and demonstrated that these ASSs arise from intrastage competition via food abundance. Since the timing of a habitat switch (that is, body size at habitat switch) regulates the flow of individuals between the two habitats, this trait has a direct influence on the strength of competition in both habitats, and thereby on the mechanism underlying the ASSs.

Likewise, we suggest that, in other systems with ASSs, selection can drive phenotypic traits beyond a tipping point when the traits under selection have a direct effect on the mechanism underlying ASSs. For instance, in shallow lakes where a clear plant-dominated state and an alternative turbid algae-dominated state co-occur, the competitive interaction between macrophytes and algae is key in the existence of these ASSs[34]. Therefore, changes in external conditions that trigger phenotypic changes in traits associated with competitive abilities of these species may cause selection-induced delayed regime shifts. Likewise, in aquatic food webs, ASSs corresponding to low- and high-density of predator populations co-occur, and predator–prey interactions are key in the existence of these ASSs[35,36]. Specifically, predatory species shape the size–structure of their prey populations but disturbances in predator mortality, caused for instance by fishing, can render the predator population incapable of shaping the prey size distribution. As a consequence, when harvesting levels exceed a tipping point, the system shifts to an alternative stable state of low-density of the predator population[35]. Size-selective harvesting of predatory fishes has triggered phenotypic changes in ecological and life history traits[37,38] that may affect predator–prey interactions, and these changes may result in selection-induced delayed regime shifts.

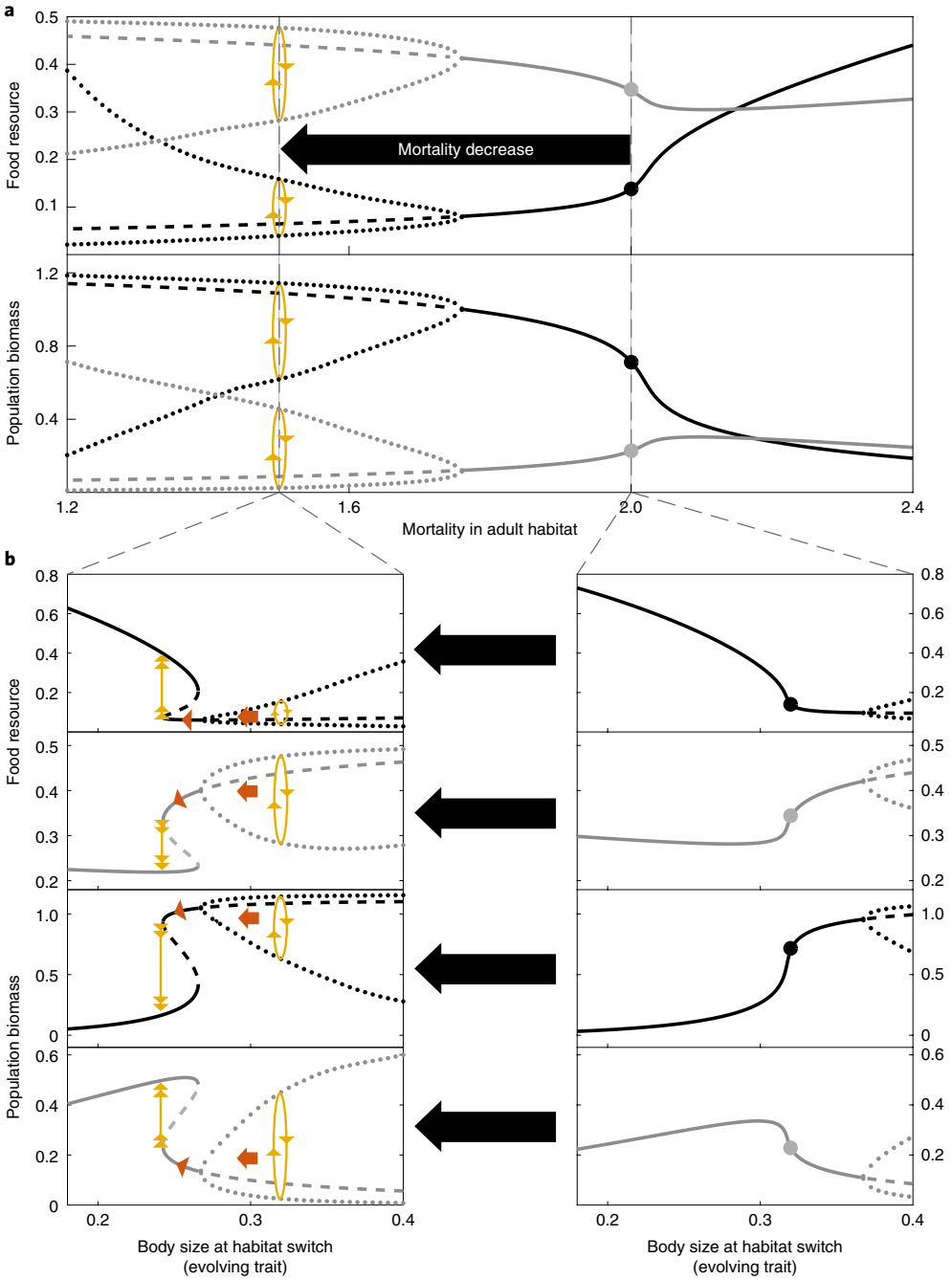

Fig. 3 | Ecological and eco-evolutionary consequences of a decrease in mortality risk. a,**b**, Direct ecological effects (**a**) and eco-evolutionary responses (**b**) following a decrease in mortality in the adult habitat (from 2 to 1.5). Stable (solid lines) and unstable (dashed lines) ecological equilibria as well as minimum and maximum values of oscillating dynamics (dotted lines) in the nursery (black) and adult habitat (grey) show that initially the system is ecologically and evolutionarily stable (**b** right panel, high mortality in adult habitat) without ASSs. The mortality reduction causes a destabilization of the ecological dynamics (black and grey dots to yellow cycling dynamics). In the novel conditions, ASSs emerge at lower trait values (**b** left panel, low mortality in adult habitat) while selection (orange arrows) gradually reduces the trait value (body size at habitat switch) until the critical trait value (tipping point) at which a regime shift occurs (yellow vertical arrows). Parameter values as in Fig. 2.

More than 30 different regime shifts have been documented in nature[39]. Whether selection can induce delayed regime shifts in all of them requires further research. We have demonstrated how selection-induced delayed regime shifts can be investigated and how evolutionary dynamics can be incorporated to study responses of ecological systems with ASSs. Following this approach, for each system in which ASSs have been documented, it is necessary to investigate how environmental changes trigger trait evolution of key species, namely those species involved in the mechanisms underlying ASSs, and whether the ensuing evolutionary responses can drive the system toward the vicinity of tipping points.

Besides ecological systems, delayed regime shifts may also occur in any system consisting of components with adaptable properties, such as human behaviour. The 2008 financial crisis, initiated by financial deregulation in the late 1970s but culminating in a collapse

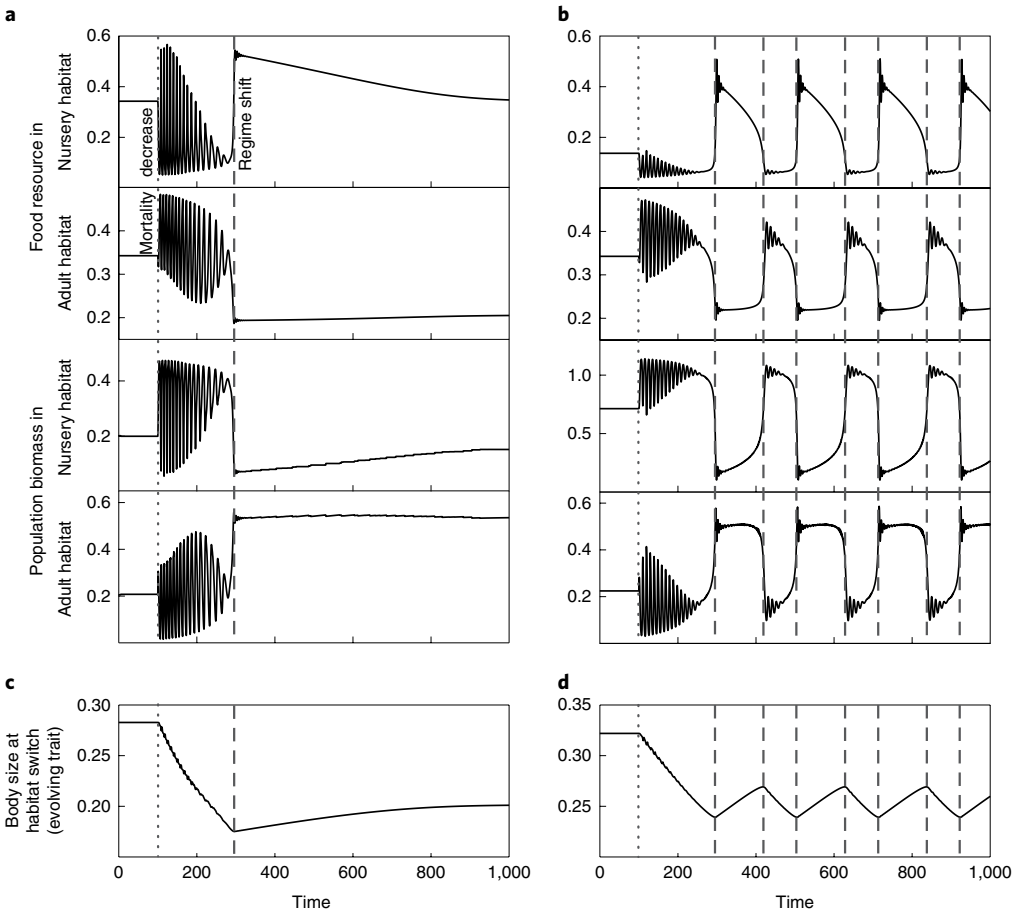

Fig. 4 | Eco-evolutionary dynamics of a population with a habitat switch experiencing a decrease in mortality in the adult habitat for two different regimes of mortality in the nursery habitat. a–**d**, Ecological (**a**,**b**) and evolutionary (**c**,**d**) dynamics before and after a reduction in mortality in the adult habitat (at time 100, dotted line) from 2 to 1.2 in **a** and **c** and from 2 to 1.5 in **b** and **d**. Single (**a**) or multiple (**b**) delayed regime shifts (at dashed vertical lines) occur after the change in mortality. Mortality in the nursery habitat equals 2 in **a** and **c** and 0.8 in **b** and **d**. Other model settings and parameters as in Fig. 2.

only years later, following a steady increase in risky financial lending practices[40], may serve as an example.

The mechanism underlying the occurrence of delayed regime shifts in ecological systems is the existence of ASSs for a range of trait values with natural selection driving the trait value beyond the tipping point. Such trait-based ASSs suggest that human-induced evolution, which is increasingly documented in wild populations[14,41,42], may be an important cause of regime shifts in ecosystems but with the environmental change that is ultimately unleashing the regime shift occurring long before the regime shift itself. The ultimate cause of a documented regime shift in present times may, in this case, be impossible to determine with the common praxis of analysing correlations between the system state and external factors affecting it. With environmental change come novel ecological conditions and phenotypic changes[11,12] that jointly can produce nonlinear dynamics with important consequences for communities and ecosystems. Understanding these dynamics, however, will prove challenging while ecological and evolutionary processes are studied in isolation. If we are to understand the responses of communities and ecosystems to environmental change, further knowledge of the interaction between ecological and evolutionary processes will be required.

Methods

The model. We consider a population structured by individual body size[23] s, which uses two different habitats in consecutive stages of their life history. In each habitat individuals exploit a different resource. Individual resource consumption, somatic growth, survival and reproduction follow continuous-time dynamics. We use a minimal model that includes only the three key ingredients to describe the dynamics of a population of individuals exploiting two different habitats in consecutive stages of their life history: (1) competition for food and food-dependent development in each of the two habitats resulting in density-dependent growth in body size and density-dependent reproduction; (2) a habitat switch during life history dependent on individual body size; and (3) differences in mortality rate experienced in the two habitats.

Life cycle. Individuals are born in habitat 1, the nursery habitat, with a size s_b where they remain until they reach body size s_s when they switch to habitat 2, the adult habitat. Juvenile individuals mature in habitat 2 and start to reproduce at a body size s_m ($s_b \leq s_s \leq s_m$).

Habitats. In each habitat, there is density-dependence mediated by food abundance, thus the food resource density declines through foraging by consumer individuals. In the absence of consumers, the resources are assumed to follow a semi-chemostat growth dynamics with productivity D and turnover rate θ (for an explanation and justification of this type of growth dynamics, see ref. [43]). In the absence of consumers, dynamics of the resource density F_1 and F_2 in the habitats 1 and 2, respectively, is given by:

$$\frac{\mathrm{d}F_i}{\mathrm{d}t} = D_i - \theta F_i \quad (1)$$

where i can take values 1 and 2 depending on the habitat the equation refers to.

Individual dynamics. Individuals are assumed to feed on the food resource at a rate $a_i F_i$ and to grow at a rate

$$g_1(F_1) = \epsilon_g a_1 F_1 \quad (2)$$

in habitat 1, and

$$g_2(F_2) = \epsilon_g a_2 F_2 \tag{3}$$

in habitat 2, where ϵ_g is a proportionality constant relating the growth rate in biomass to the ingestion rate of food.

Adults reproduce at a rate

$$b(F_2) = \epsilon_b a_2 F_2 \tag{4}$$

where ϵ_b is a proportionality constant relating the reproduction rate (in terms of offspring per unit of time) in biomass to the ingestion rate of food. Individuals die at a mortality rate μ_1 in the habitat 1 and μ_2 in the habitat 2.

Ecological dynamics. Using standard methods to formulate size-structured populations from individual life history processes[44], the population model based on the individual life history above is described by the following set of equations.

Dynamics of the food resource density in the habitat 1 is given by

$$\frac{\mathrm{d}F_1}{\mathrm{d}t} = D_1 - \theta F_1 - a_1 F_1 \int_{s_b}^{s_s} n_1(t,s)\mathrm{d}s \tag{5}$$

and in the habitat 2 by

$$\frac{\mathrm{d}F_2}{\mathrm{d}t} = D_2 - \theta F_2 - a_2 F_2 \int_{s_s}^{\infty} n_2(t,s)\mathrm{d}s \tag{6}$$

In equations (5) and (6), $n_1(t,s)$ and $n_2(t,s)$ are the density functions of the size distribution of the population in habitat 1 and habitat 2, respectively. The dynamics of these size-dependent density functions are given by:

$$\frac{\partial n_1(t,s)}{\partial t} + g_1(F_1)\frac{\partial n_1(t,s)}{\partial s} = -\mu_1 n_1(t,s) \tag{7}$$

$$\frac{\partial n_2(t,s)}{\partial t} + g_2(F_2)\frac{\partial n_2(t,s)}{\partial s} = -\mu_2 n_2(t,s) \tag{8}$$

In these equations, the abundance of individuals in either habitat $n_i(t,s)$ as a function of time t and their body mass s is dependent on the individual growth $g_i(F_i)$ and mortality μ_i rates. The entry of individuals in either habitat—newborns in habitat 1 and individuals switching to habitat 2—are described by the boundary conditions

$$g_2(F_2)n_2(t,s_s) = g_1(F_1)n_1(t,s_s) \tag{9}$$

$$g_1(F_1)n_1(t,s_b) = b(F_2)\int_{s_m}^{\infty} n_2(t,s)\mathrm{d}s \tag{10}$$

Equations (9) and (10) provide the boundary condition at the switching size s_s from habitat 1 to habitat 2 and at the birth size s_b corresponding to the total population birth rate, respectively. The total biomass of individuals in habitat 1 equals

$$\int_{s_b}^{s_s} s\, n_1(t,s)\mathrm{d}s \tag{11}$$

and in habitat 2

$$\int_{s_s}^{\infty} s\, n_2(t,s)\mathrm{d}s \tag{11}$$

We have non-dimensionalized the model (see Supplementary Information) and, as a result, the scaled model is described by the following set of equations:

$$\frac{\mathrm{d}F_1}{\mathrm{d}t} = 1 - \delta F_1 - \int_0^{w_s} \gamma_1(F_1)m_1(t,w)\mathrm{d}w \tag{12}$$

$$\frac{\mathrm{d}F_2}{\mathrm{d}t} = \rho - \delta F_2 - \int_{w_s}^{\infty} \gamma_2(F_2)m_2(t,w)\mathrm{d}w \tag{13}$$

$$\frac{\partial m_1(t,w)}{\partial t} + \gamma_1(F_1)\frac{\partial m_1(t,w)}{\partial w} = -\eta_1 m_1(t,w) \tag{14}$$

$$\frac{\partial m_2(t,w)}{\partial t} + \gamma_2(F_2)\frac{\partial m_2(t,w)}{\partial w} = -\eta_2 m_2(t,w) \tag{15}$$

with the boundary condition for switching from habitat 1 to habitat 2

$$\gamma_2(F_2)m_2(t,w_s) = \gamma_1(F_1)m_1(t,w_s) \tag{16}$$

and for the population birth rate

$$\gamma_1(F_1)m_1(t,0) = \beta\gamma_2(F_2)\int_1^{\infty} m_2(t,w)\mathrm{d}w \tag{17}$$

In this system $\gamma_1(F_1) = F_1$ and $\gamma_2(F_2) = qF_2$. The scaled body size variable w is related to the original body size measure following $w = (s - s_b)/(s_m - s_b)$ and maturation occurs at $w = 1$.

In the scaled system of partial differential equations, the parameters relate to the parameters in the unscaled model according to Table 1. The results presented in this study are carried out using the non-dimensional form of the model, which is parameterized following de Roos and Persson[23].

Eco-evolutionary dynamics. In this study, we are interested in understanding the eco-evolutionary consequences of the selection process in the body size at habitat switch (the evolving individual trait) when individual mortality varies in either habitat. To investigate this, we study the evolutionary dynamics on ecological timescales using a quantitative genetics approach for life history traits in a structured population[45]. The body size at habitat switch is considered to be a quantitative trait controlled by a number of loci of small effect. The trait is distributed according to a truncated, approximately normal, distribution (more specifically, a Bates distribution of degree three), with mean $\overline{w_s}$ and a minimum and a maximum value $(1-\sigma)\overline{w_s}$ and $(1+\sigma)\overline{w_s}$, respectively. Given that individual fecundity $\beta\gamma_2(F_2)$ is identical for every adult, selection depends only on survival. Hence, the mean trait value of offspring born at every time t equals the mean trait value of the reproducing (adult) part of the population at this time. The rate of change of the mean trait value of offspring born at time t equals the rate of change of the mean trait value of the adult population. The trait variation 2σ is assumed constant. A detailed model description of the eco-evolutionary dynamics can be found in the Supplementary Information (see Supplementary note 1).

In addition, we assess the possibility of evolutionary endpoints on the unstable ecological equilibrium using an adaptive dynamics approach for size-structured populations[46].

Model analysis. We use the Escalator Boxcar Train method[44,47] to carry out numerical simulations of the non-dimensional form of the model. The central idea of this method is to group individuals into cohorts. In the absence of genetic variation, these cohorts are collections of individuals that are born with the same size at birth within a short period of time; therefore, each cohort can be characterized by the number of individuals and their average body size. To implement genetic variability in the population, we consider each of these cohorts to be subdivided into ten subcohorts, which are identical in their body size at birth and maturation but differ in the value of the body size at habitat switch. Individuals within a subcohort are considered identical. Each of the ten subcohorts is assigned its own phenotype: five with lower and five with higher w_s values than $\overline{w}_s^*$, equidistantly separated by a factor of $2\sigma\overline{w}_s^*/10$. Newborn individuals are distributed over the subcohorts with different w_s in such a way that the trait distribution reflects a discrete approximation to the Bates distribution truncated at $(1-\sigma)\overline{w}_s^*$ and $(1+\sigma)\overline{w}_s^*$ that is described above. The dynamics of the population are followed by numerically integrating the ordinary differential equations for each subcohort separately. Specifically, we carry out simulations to investigate the eco-evolutionary dynamics before and after a reduction of the mortality rate η_2 in habitat 2 from 2 to 1.5 when the mortality rate η_1 in habitat 1 is constant and equal to 0.8 (Figs. 2, 3 and 4b,d), and after a reduction of the mortality η_2 in habitat 2 from 2 to 1.2 when the mortality rate η_1 in habitat 1 is constant and equal to 2 (Fig. 4a,c). In both cases, genetic variation was represented with a truncated normal distribution with σ equal to 0.2. The effect of lower genetic variation $\sigma = 0.1$ was also investigated and its results are shown in Extended Data Fig. 2. We use the PSPManalysis software package[48] to numerically compute and continue the ecological equilibrium of the non-dimensional form of the model as a function of the model parameters. This approach is complementary to the population genetics approach because it enables us to detect and continue the unstable equilibrium occurring between the two ASSs, which is not possible with dynamics simulations. We use a combination of the EBT and PSPManalysis package to visualize stable and unstable equilibrium values as well as maximum and minimum densities during limit cycles as a function of mortality in habitat 2 and body size at habitat switch (Fig. 3). The PSPManalysis package also enables us to detect evolutionary endpoints irrespective of whether the equilibrium is ecologically stable or unstable by calculating the selection gradient using the adaptive dynamics approach. Hence, by using a combination of EBT and PSPManalysis package we detect evolutionary endpoints in the ecological setting (Extended Data Fig. 3c,d).

Reporting Summary. Further information on research design is available in the Nature Research Reporting Summary linked to this article.

Data availability

No data were collected or used in this study.

Code availability

The implementation of the Escalator Boxcar Train numerical method and the PSPM package used to analyse the model can be found in https://staff.fnwi.uva.nl/a.m.deroos/EBT/Software/index.html and https://staff.fnwi.uva.nl/a.m.deroos/PSPManalysis/index.html, respectively.

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References

1. Scheffer, M., Carpenter, S., Foley, J. A., Folke, C. & Walker, B. Catastrophic shifts in ecosystems. *Nature* **413**, 591–596 (2001).
2. May, R. M. Thresholds and breakpoints in ecosystems with a multiplicity of stable states. *Nature* **260**, 471–477 (1976).
3. Drijfhout, S. et al. Catalogue of abrupt shifts in Intergovernmental Panel on Climate Change climate models. *Proc. Natl Acad. Sci. USA* **112**, E5777–E5786 (2015).
4. Winkelmann, R. et al. Trajectories of the Earth system in the Anthropocene. *Proc. Natl Acad. Sci. USA* **115**, 8252–8259 (2018).
5. Scheffer, M. et al. Early-warning signals for critical transitions. *Nature* **461**, 53–59 (2009).
6. Guttal, V. & Jayaprakash, C. Changing skewness: an early warning signal of regime shifts in ecosystems. *Ecol. Lett.* **11**, 450–460 (2008).
7. Carpenter, S. R. et al. Early warnings of regime shifts: a whole-ecosystem experiment. *Science* **332**, 1079–1082 (2014).
8. Scheffer, M. *Critical Transitions in Nature and Society* (Princeton Univ. Press, 2009).
9. DeMenocal, P. et al. Abrupt onset and termination of the African Humid Period: rapid climate responses to gradual insolation forcing. *Quat. Sci. Rev.* **19**, 347–361 (2000).
10. Hare, S. R. & Mantua, N. J. Empirical evidence for North Pacific regime shifts in 1977 and 1989. *Prog. Oceanogr.* **47**, 103–145 (2000).
11. Walther, G. R. Community and ecosystem responses to recent climate change. *Phil. Trans. R. Soc. B* **365**, 2019–2024 (2010).
12. Parmesan, C. Ecological and evolutionary responses to recent climate change. *Annu. Rev. Ecol. Evol. Syst.* **37**, 637–669 (2006).
13. Singer, M. C., Thomas, C. D. & Parmesan, C. Rapid human-induced evolution of insect–host associations. *Nature* **366**, 681–683 (1993).
14. Allendorf, F. W. & Hard, J. J. Human-induced evolution caused by unnatural selection through harvest of wild animals. *Proc. Natl Acad. Sci. USA* **106**, 9987–9994 (2009).
15. Olsen, E. M. et al. Maturation trends indicative of rapid evolution preceded the collapse of northern cod. *Nature* **428**, 932–935 (2004).
16. Palumbi, S. R. & Mu, P. Humans as the World's greatest evolutionary force: the pace of human-induced evolution. *Science* **293**, 1786–1791 (2001).
17. Parvinen, K. & Dieckmann, U. Self-extinction through optimizing selection. *J. Theor. Biol.* **333**, 1–9 (2013).
18. Schreiber, S. & Rudolf, V. H. W. Crossing habitat boundaries: coupling dynamics of ecosystems through complex life cycles. *Ecol. Lett.* **11**, 576–587 (2008).
19. Knight, T. M., McCoy, M. W., Chase, J. M., McCoy, K. A. & Holt, R. D. Trophic cascades across ecosystems. *Nature* **437**, 880–883 (2005).
20. Werner, E. E. & Gilliam, J. F. The ontogenetic niche and species interactions in size-structured populations. *Ecology* **15**, 393–425 (1984).
21. Diehl, S. & Eklov, P. Effects of piscivore-mediated habitat use on resources, diet, and growth of perch. *Ecology* **76**, 1712–1726 (1995).
22. Hobson, K. A. Tracing origins and migration of wildlife using stable isotopes: a review. *Oecologia* **120**, 134–326 (1999).
23. de Roos, A. M. & Persson, L. Competition in size-structured populations: mechanisms inducing cohort formation and population cycles. *Theor. Pop. Biol.* **63**, 1–16 (2003).
24. Hendry, A. P., Farrugia, T. J. & Kinnison, M. T. Human influences on rates of phenotypic change in wild animal populations. *Mol. Ecol.* **17**, 20–29 (2008).
25. Bone, E. & Farres, A. Trends and rates of microevolution in plants. *Genetica* **112–113**, 165–182 (2001).
26. Lande, R. & Shannon, S. The role of genetic variation in adaptation and population persistence in a changing environment. *Evolution* **50**, 434–437 (1996).
27. Woodward, G. & Hildrew, A. G. Body-size determinants of niche overlap and intraguild predation within a complex food web. *J. Anim. Ecol.* **71**, 1063–1074 (2002).
28. Brose, U. et al. Predicting the consequences of species loss using size-structured biodiversity approaches. *Biol. Rev.* **92**, 684–697 (2017).
29. Boyle, P. R. & Boletzky, S. Cephalopod populations: definition and dynamics. *Phil. Trans. R. Soc. B.* **351**, 985–1002 (1996).
30. de Roos, A. M. & Persson, L. *Population and Community Ecology of Ontogenetic Development* (Princeton Univ. Press, 2013).
31. Hansen, J. H. et al. Ecological consequences of animal migration: prey partial migration affects predator ecology and prey communities. *Ecosystems* **22**, 1–15 (2019).
32. Rudolf, V. H. W. & Rasmussen, N. L. Population structure determines functional differences among species and ecosystem processes. *Nat. Commun.* **4**, 2318 (2013).
33. Zhou, S. et al. Ecosystem-based fisheries management requires a change to the selective fishing philosophy. *Proc. Natl Acad. Sci. USA* **107**, 9485–9489 (2010).
34. Scheffer, M., Hosper, S., Meijer, M., Moss, B. & Jeppesen, E. Alternative equilibria in shallow lakes. *Trends Ecol. Evol.* **8**, 275–279 (1993).
35. de Roos, A. M. & Persson, L. Size-dependent life-history traits promote catastrophic collapses of top predators. *Proc. Natl Acad. Sci. USA* **99**, 12907–12912 (2002).
36. Gårdmark, A. et al. Regime shifts in exploited marine food webs: detecting mechanisms underlying alternative stable states using size structured community dynamics theory. *Phil. Trans. R. Soc. B.* **370**, 20130262 (2015).
37. Biro, P. A. & Post, J. R. Rapid depletion of genotypes with fast growth and bold personality traits from harvested fish populations. *Proc. Natl Acad. Sci. USA* **105**, 2919–2922 (2008).
38. Swain, D. P., Sinclair, A. F. & Hanson, J. M. Evolutionary response to size-selective mortality in an exploited fish population. *Proc. Biol. Sci.* **274**, 1015–1022 (2007).
39. Rocha, J. C., Peterson, G. D. & Biggs, R. Regime shifts in the Anthropocene: drivers, risks, and resilience. *PLoS ONE* **10**, e0134639 (2015).
40. Brunnermeier, M. K. Deciphering the liquidity and credit crunch 2007-08. *J. Econ. Perspect.* **23**, 77–100 (2009).
41. Sih, A., Ferrari, M. C. O. & Harris, D. J. Evolution and behavioural responses to human-induced rapid environmental change. *Evol. Appl.* **4**, 367–387 (2011).
42. Skelly, D. K. et al. Evolutionary responses to climate change. *Conserv. Biol.* **21**, 1353–1355 (2007).
43. Persson, L., Leonardsson, K., de Roos, aM., Gyllenberg, M. & Christensen, B. Ontogenetic scaling of foraging rates and the dynamics of a size-structured consumer-resource model. *Theor. Pop. Biol.* **54**, 270–293 (1998).
44. de Roos, A. M. in *Structured-Population Models in Marine, Terrestrial, and Freshwater Systems* (eds Tuljapurkar, S. & Caswell, H.) 119–204 (Springer Science & Business Media, 1997).
45. Lande, R. A quantitative genetic theory of life history evolution. *Ecology* **63**, 607–615 (1982).
46. Durinx, M., Metz, J. A. & Meszéna, G. Adaptive dynamics for physiologically structured population models. *J. Math. Biol.* **56**, 673–742 (2008).
47. de Roos, A. M. Numerical methods for structured population models: the Escalator Boxcar Train. *Numer. Methods Partial Differ. Equ.* **4**, 173–195 (1988).
48. de Roos, A. M. *PSPManalysis: A Package for Numerical Analysis of Physiologically Structured Population Models* (Institute for Biodiversity and Ecosystem Dynamics, 2018); https://bitbucket.org/amderoos/PSPManalysis/

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Author contributions

P.C.C.-P. and A.M.deR. designed methodology and gave final approval for publication. P.C.C.-P. conceived the ideas, analysed the results and led the writing of the manuscript. A.M.deR. developed the model formulation and contributed to later versions of the manuscript.

Competing interests

The authors declare no competing interests.

Additional information

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Correspondence and requests for materials should be addressed to P.C.C.-P.

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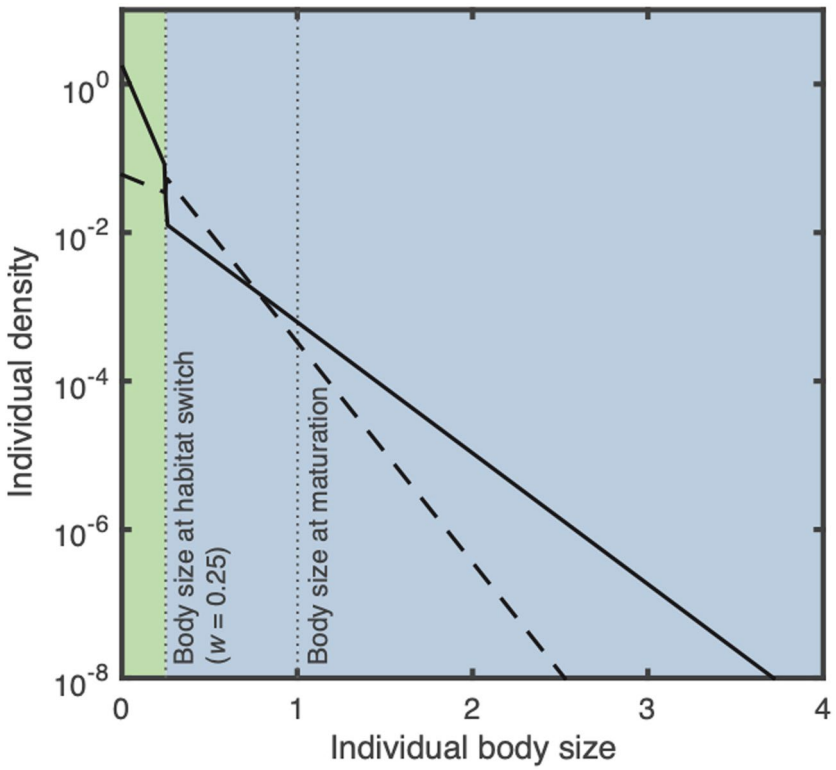
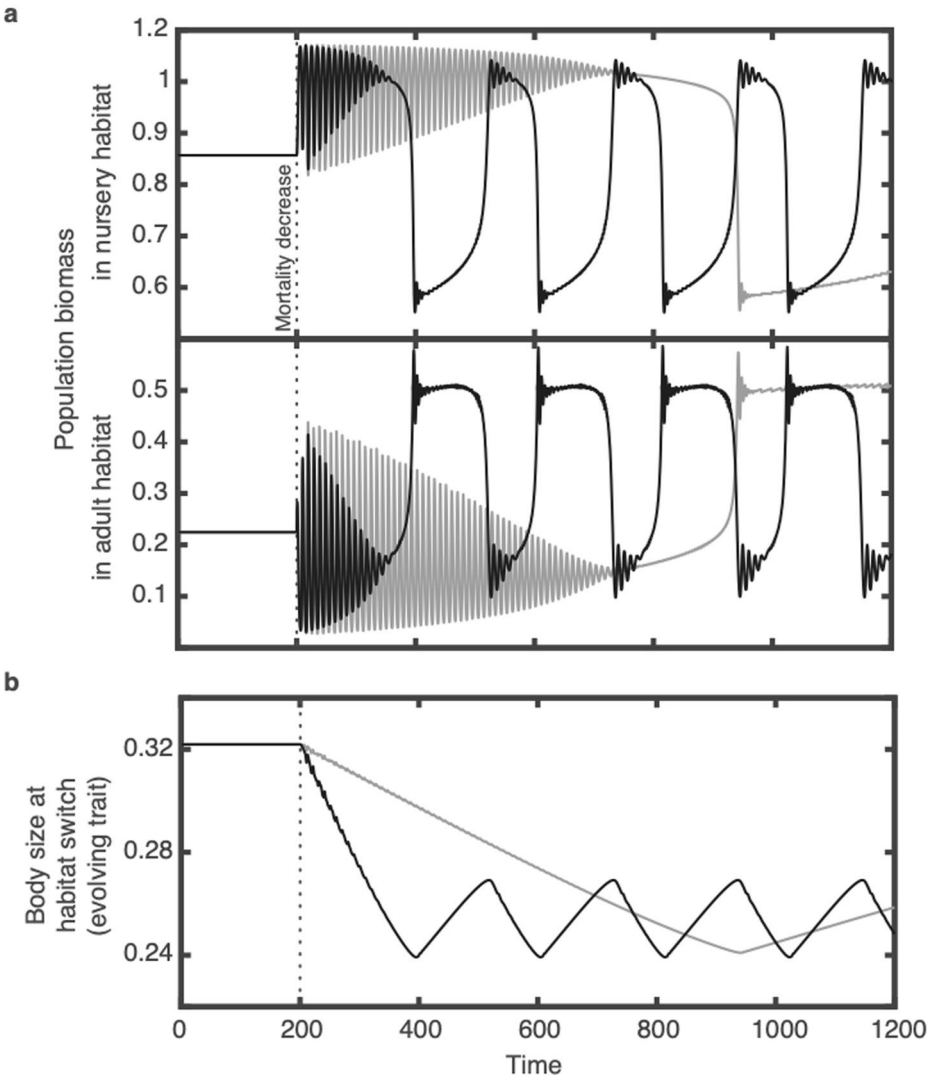
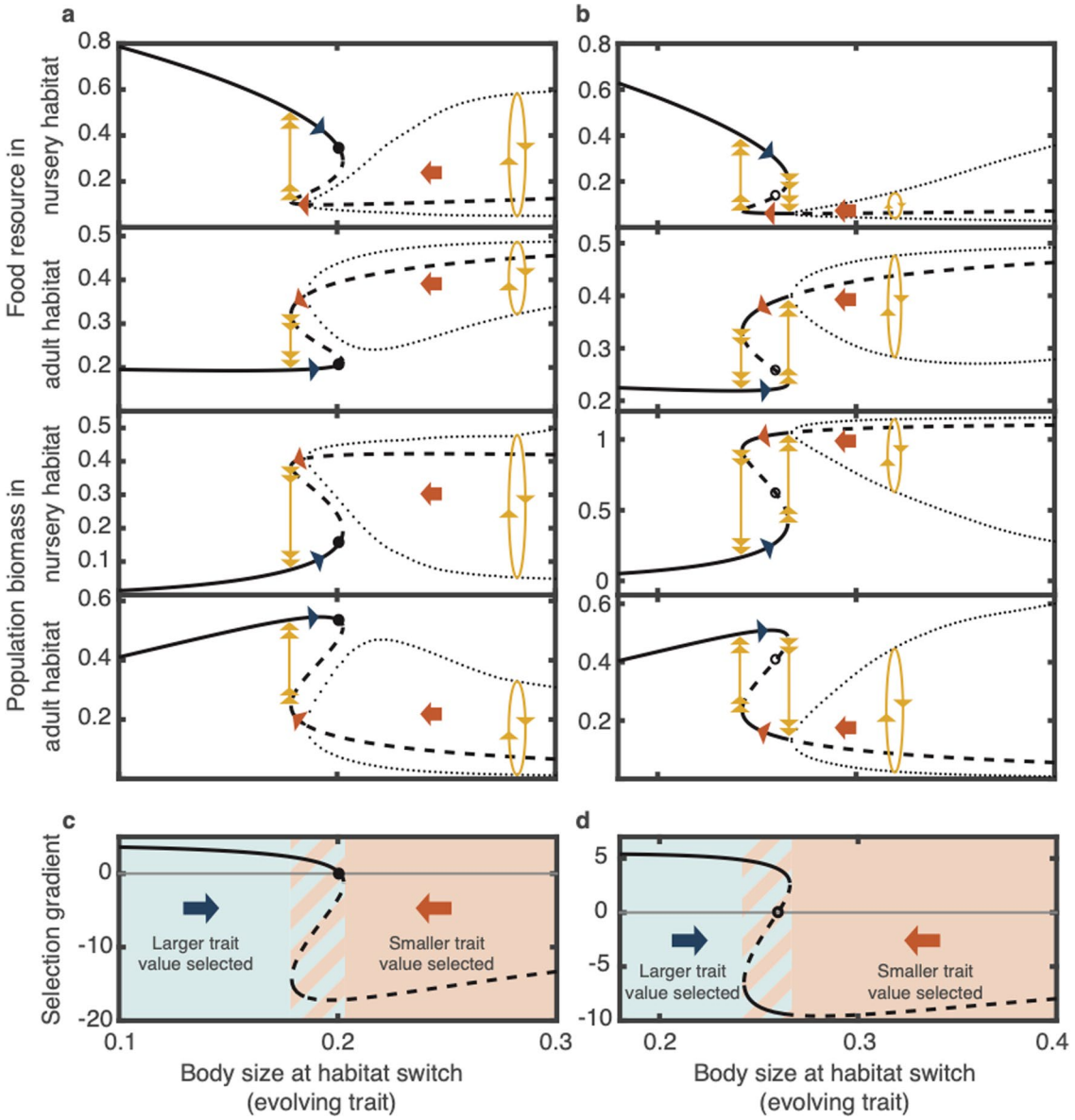


Extended Data Fig. 1 | Population compositions in the ASSs when the average body size at habitat switch equals 0.25. The two ASSs correspond to low (solid line) and high biomass levels (dashed line) in the nursery habitat, and to high (solid line) and low biomass levels (dashed line) in the adult habitat. These alternative stable population compositions represent the population structure approximately at time 260 in Fig. 2 (before the regime shift in Fig. 2) and approximately at time 340 in Fig. 2 (after the regime shift in Fig. 2). The regime shift observed in Fig. 2 leads to a decrease in population density in the nursery habitat (green region) and an increase in population density in the adult habitat (blue region), mainly as a consequence of an increase in the density of immature individuals (smaller than the size at maturation). This increase in density of immature individuals in the adult habitat results in increased competition in this habitat that produces a reduction of 32% in the maximum asymptotic body size after the regime shift (reduction from 3.71 to 2.52). Parameter values as in Fig. 2.



Extended Data Fig. 2 | Eco-evolutionary effects of trait variation in the population. a) Ecological and **b**) evolutionary dynamics before and after a reduction of mortality in the adult habitat (vertical dotted line, from 2 to 1.5). When trait variation is represented with a truncated normal distribution with a minimum and maximum value equal to 80% and 120% (black lines) the regime shift occurs at time 390, whereas when the minimum and maximum value equal to 90% and 110% (grey lines) the regime shift occurs at time 940. Mortality in habitat 1 is 0.8, other parameters as in Table 1 (see Methods).



Extended Data Fig. 3 | Long-term stability of the system. Population biomass and food resource densities in the nursery and adult habitat and selection gradient as a function of body size at habitat switch after a decrease in mortality when the evolutionary endpoint occurs **a**) in one of the alternative stable ecological equilibrium resulting in a single regime shift (dynamics shown in Fig. 4a) and **b**) in the unstable equilibrium resulting in repeated delayed regime shifts (dynamics shown in Fig. 4b). Ecologically stable (solid lines) and unstable (dashed lines) equilibrium values are indicated with black lines as well as minimum and maximum densities during oscillatory dynamics (dotted lines). The direction of selection is indicated with thick arrows (orange when negative and blue when positive) and ecological dynamics with double vertical arrows (yellow). The evolutionary endpoint is indicated with a circle (open circle in case it corresponds to an unstable ecological equilibrium, filled circle if it correspond to a stable ecological equilibrium). The direction of selection (bottom plots) is positive at low values of the trait (blue shaded area), negative at high values (pink shaded area) and either negative or positive at intermediate values of the trait (mixed shaded area), depending on which of the two ASSs the population is in. Parameter values as in Fig. 4.

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Corresponding author(s): Catalina Chaparro-Pedraza

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