

Fluctuating Environments Favor Extreme Reproductive Delays and Penalize Intermediate Ones

Jorge Hidalgo ^{1,2,*} Lorenzo Fant ^{3,4} Rafael Rubio de Casas ^{5,6} and Miguel A. Muñoz ^{2,7}

¹Departamento de Física, *Universidad de Córdoba*, Córdoba 14071, Spain

²*Instituto Carlos I de Física Teórica y Computacional*, Granada 18071, Spain

³*Istituto Nazionale di Oceanografia e Geofisica Sperimentale (OGS)*, Trieste, Italia

⁴*National Biodiversity Future Centre (NBFC)*, Palermo Piazza Marina 61, Palermo 90133, Italy

⁵Departamento de Ecología, *Universidad de Granada*, Granada 18071, Spain

⁶Research Unit “Modeling Nature” MNat, *Universidad de Granada*, Granada 18071, Spain

⁷Departamento de Electromagnetismo y Física de la Materia, *Universidad de Granada*, Granada 18071, Spain



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The timing of reproduction is crucial for biological fitness. Reproductive delays carry an inherent demographic cost: an individual that takes longer to reproduce contributes later to population growth, *a priori*, without any compensating benefit. However, natural selection favors delayed reproduction under certain environmental conditions, a phenomenon that remains poorly understood. Here, we investigate how the environment can determine the evolution of particular life histories using a parsimonious delayed-logistic model. Individuals become reproductive only after a fixed lag whereas birth rates fluctuate under temporally correlated stochasticity. Numerical integration, analytical calculations and evolutionary simulations reveal that the joint effect of demographic memory and colored multiplicative noise generate a strongly nonmonotonic dependence of fitness on life history, with three distinct regimes of population performance: rapid reproduction maximizes linear growth but amplifies fluctuations and extinction risk; delayed reproduction buffers environmental variability, substantially increasing mean extinction times despite slower growth; strikingly, and central to our results, there is a broad band of maladaptive intermediate life histories that simultaneously reduce both growth and persistence, an effect that arises generically from the mismatch between reproductive timing and environmental autocorrelation. Our results show that reproductive timing is not merely a life-history parameter but an adaptive mechanism tuned to environmental time scales, and that “dangerous middle” maturation times can be inherently disfavored, with implications for understanding reproductive strategies across the tree of life.

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I. INTRODUCTION

Life history theory addresses how organisms allocate time and resources among growth, reproduction, and survival [1,2]. A key component is the duration of the reproductive maturation period—the interval from birth to first reproduction—which carries a direct fitness cost: delaying reproduction reduces an individual’s contribution to population growth. Despite this, maturation times vary over orders of magnitude across taxa, from hours in microbes to decades in long-lived plants, and even among closely related species (e.g., annuals vs trees in Euphorbia) [3,4]. Explaining the evolutionarily optimal duration of this period remains a central challenge, particularly in fluctuating environments where reproductive timing strongly affects both growth and persistence.

Reproductive maturation reflects diverse biological processes that shape life stages. In some cases, its costs are offset

by compensatory benefits: larger size at maturity can increase fecundity or competitive ability, and extended development may enhance adult survival [1]. However, such compensation is not universal. In many cases—such as dormancy, which acts as a quiescent interval during which individuals neither grow nor reproduce—its presence remains unclear.

Dormancy is widespread across bacteria, plants, fungi, insects, and even cancer cells [5–7]. It manifests itself in various forms, including microbial lag-time strategies tuned to environmental periodicity [8,9], synchronized reproductive cycles in insects [10], and broad germination-time distributions in plant seed banks under unpredictable rainfall [11–16]. Despite this diversity, common patterns emerge: dormancy duration is closely linked to the duration and predictability of unfavorable conditions, and its timing is under strong selection. Mechanisms enabling environmental sensing—such as cue-driven germination in plants [17,18], diapause termination in insects and zooplankton [19,20], or exit from bacterial latency [21,22]—allow organisms to resume activity under favorable conditions. Similar dynamics appear in cancer, where dormant tumor cells reactivate in response to microenvironmental cues [23–25]. At the same time, stochastic variation in dormancy duration is also common and can function as a bet-hedging strategy in unpredictable environments [26–28]. The coexistence of cue-driven responses and stochastic

*Contact author: jorge.hidalgo@uco.es

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strategies highlights a complex and not yet fully resolved phenomenology.

Most theoretical frameworks justify reproductive delay through compensatory trade-offs [29], but whether such compensation is necessary—or even adaptive—remains unclear. This raises a fundamental question: can intrinsically costly delays be favored solely due to environmental fluctuations? More specifically, how does the temporal structure of environmental variability shape optimal maturation time, and can selection favor both precise environmental tuning and stochasticity? While prior work has examined timing strategies under periodic or uncorrelated fluctuations [30–35], the role of temporal correlations has received less attention, despite strong empirical evidence that environmental variability is often autocorrelated (“colored” noise) [36–38].

Here, we address these questions using a minimal delayed-logistic model under stochastic environmental conditions with finite correlation time. Individuals become reproductive after a fixed delay α , introducing demographic memory, while birth rates fluctuate under temporally correlated noise. By isolating reproductive delay as a standalone trait, our framework complements models incorporating sensing or trade-offs and reveals how environmental correlations alone can shape the evolution of reproductive timing.

Our results uncover a rich and nonintuitive phenomenology. The interaction between delay and colored noise produces three dynamical regimes: short delays maximize mean growth but amplify fluctuations and extinction risk; long delays reduce variability and enhance persistence at the cost of slower growth; and, remarkably, intermediate delays perform worst, simultaneously lowering growth and resilience. This nonmonotonic behavior arises from a mismatch between the delay timescale and environmental autocorrelation. Using an evolutionary agent-based model, we further show that this landscape leads to bistability, with populations evolving toward either short or long delays while avoiding the maladaptive intermediate regime. This pattern echoes empirical observations such as bimodal germination strategies in plants, extended dormancy in periodical cicadas, and mixed phenotypes in microbial persisters and zooplankton propagules [39–43].

II. STOCHASTIC DELAYED-LOGISTIC MODEL FOR POPULATIONS WITH ENVIRONMENTAL NOISE AND DELAYED REPRODUCTION

Our model—illustrated in Fig. 1—considers a population undergoing logistic growth, governed by a differential equation that incorporates two key mechanisms: (i) environmental stochasticity and (ii) finite maturation time (interpreted as a reproductive delay) [30] (see upper panel of Fig. 1). In contrast to classical logistic models, reproduction is delayed and influenced by a correlated stochastic environment, while death takes place at a constant rate. In this formulation, the delay α represents the interval an individual must complete before it can reproduce. Consequently, the population density $x(t)$ evolves according to the following equation:

$$\dot{x}(t) = (b + \sigma \xi_\tau(t))x(t - \alpha) \left(1 - \frac{x(t)}{K}\right) - d x(t), \quad (1)$$

where d is the per capita death rate (unless otherwise stated, we set $d = 1$). Following reproduction at time $t - \alpha$, the offspring remains inactive/dormant for a fixed duration α and becomes active at time t , at which point it can reproduce or die. This leads to a delayed dependence in the birth term [44], which is proportional to the population at the earlier time $t - \alpha$. Furthermore, the reproduction rate is modulated by the carrying capacity K through a logistic saturation term $1 - x(t)/K$, evaluated at the time of becoming active. An extended formulation can incorporate α as a random variable drawn from a probability distribution, thus capturing individual-level variability in maturation times or dormancy: $x(t - \alpha) \rightarrow \int d\alpha \rho(\alpha)x(t - \alpha)$, where $\rho(\alpha)$ represents the distribution of reproductive delay durations across the population. Although this approach can describe more accurately many situations in nature—including diversification and bet-hedging strategies (see below)—for now we treat α as a fixed control parameter.

Crucially, environmental variability is incorporated into the model through a time-dependent birth rate, which fluctuates as a result of external conditions [45]. Specifically, the intrinsic birth rate is replaced by a stochastic term $b + \sigma \xi_\tau(t)$, where $\xi_\tau(t)$ represents environmental noise, with amplitude σ .

For simplicity, we represent environmental variability with a dichotomous continuous-time Markov noise (DMN) [46], with states ± 1 and symmetric transition rate τ^{-1} . This modeling choice is mathematically tractable, maintains non-negativity of rates, and encodes alternating “good” and “bad” conditions. As a consequence, the process exhibits a temporal autocorrelation function $\langle \xi_\tau(t)\xi_\tau(s) \rangle = \exp(-2|t - s|/\tau)$, with characteristic correlation time $\tau/2$. We note that an alternative formulation where environmental variability affects the death rate instead of the birth rate is also possible (see Appendix E).

The use of DMN ensures that the birth rate remains non-negative as long as $\sigma \leq b$, which is essential to prevent nonphysical scenarios in the presence of delay. In fact, if the coefficient multiplying the delayed term becomes negative, the dynamics can reach negative population densities [47,48]. For this reason, other forms of colored noise—such as Ornstein-Uhlenbeck processes—could induce negative fluctuations in the birth rate, potentially destabilizing the population. Nevertheless, we have verified that the qualitative behavior of the model remains robust under alternative noise models (see Appendix D).

It should be noted that the environmental noise term $\xi_\tau(t)$ could, in principle, be evaluated either at the moment of reproduction $t - \alpha$, i.e., when the offspring is generated, or at the activation time t , i.e., when it becomes capable of reproduction and subject to death. In our case, this choice is irrelevant because of the temporal translational invariance of the DMN. However, this distinction may become relevant in extended models with stochastic α or with multiple interacting populations coupled to the same environmental fluctuations.

This modeling framework enables us to investigate the interplay between the reproductive delay (α) and key features of environmental variability—such as its amplitude (σ) and temporal correlation (τ)—and how their interaction shapes the long-term dynamics of populations exposed to fluctuating environments.

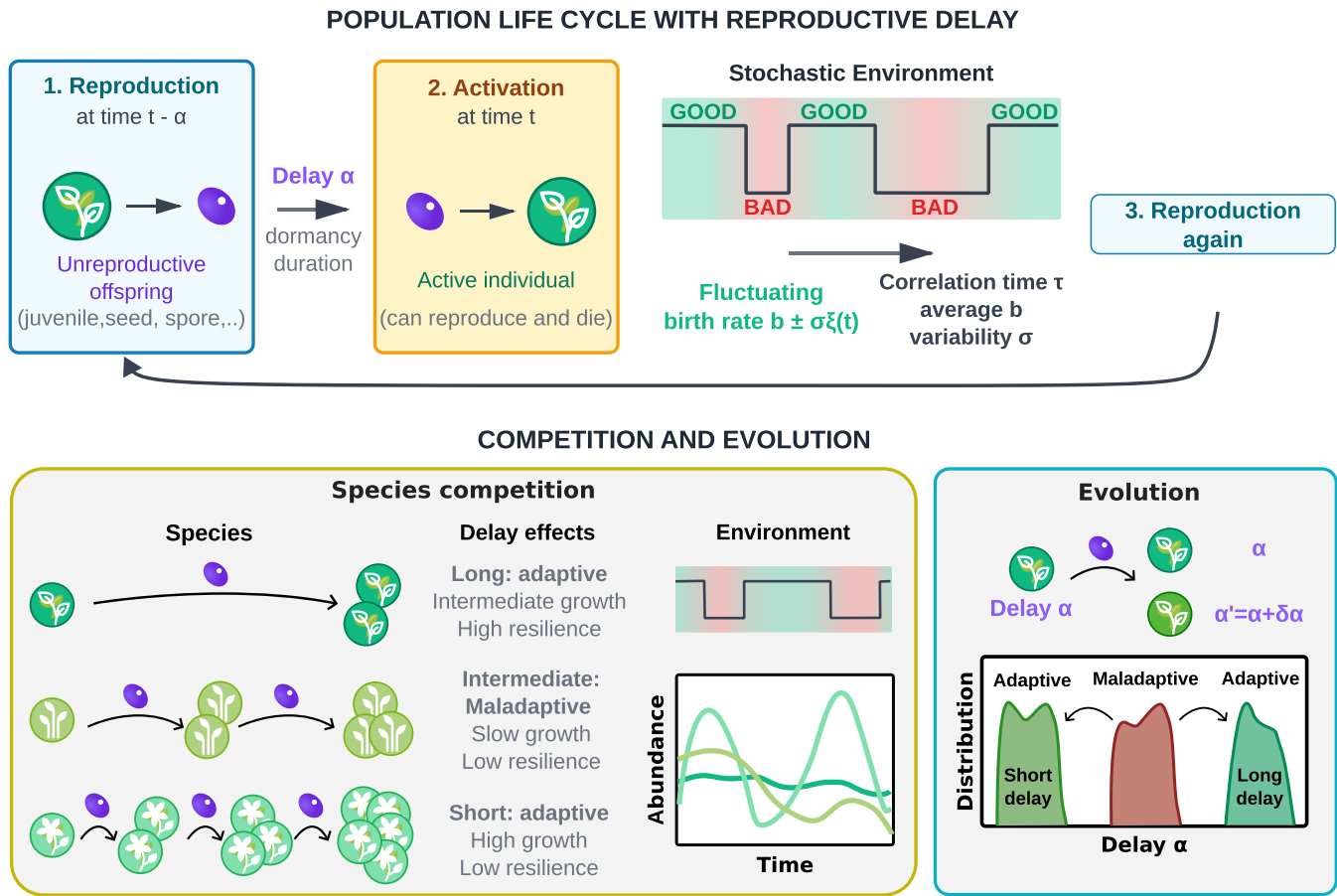


FIG. 1. Population life cycle with reproductive delay in a stochastic environment. Illustrated with plants, this framework can also be applied to other to organisms with maturation delay or dormancy. Individuals produce inactive propagules (e.g., seeds, spores, persister cells) at time $t - \alpha$, which remain in a quiescent state for a delay α before activating into reproductive, mortal individuals at time t . Active individuals experience alternating good and bad environmental conditions, represented as temporal fluctuations in the birth rate with mean b , amplitude σ and correlation time τ . We then use this framework to investigate (left) ecological competition among species that differ in reproductive delay and activation, and (right) evolutionary change in delay duration via single mutations that shift the delay from α to $\alpha' = \alpha + \delta\alpha$. In both cases, extreme strategies with either very long or very short delays gain a competitive advantage, whereas intermediate reproductive delay strategies are maladaptive and are progressively replaced by fitter ones.

We integrate Eq. (1) numerically using an explicit fourth-order Runge-Kutta method adapted to handle delay differential equations, following the approach described in Ref. [48] (see Appendix A). As is customary in delayed systems, initial conditions must be specified in the time interval $[-\alpha, 0]$. In our simulations, we set $x(t') = x(0)$ for $t' < 0$. This choice has no qualitative impact on the long-term dynamics, since the presence of noise rapidly erases the memory of the initial condition. It should be noted that the most interesting phenomenology of the model emerges when the average reproduction rate is just above the critical threshold, $b \gtrsim d$, where the interplay between environmental stochasticity and delayed reproduction leads to rich dynamical behavior. In this marginal regime, fluctuations and memory effects can significantly impact population persistence and variability, making the role of reproductive delay particularly relevant (see Appendix B). Such a regime is surely suited to describe populations in saturated environments where the overall growth rate is close to zero.

Figure 2 illustrates the results of computer simulations. In particular, it shows a representative time series of the pop-

ulation density $x(t)$ for varying reproductive delay α , under conditions of pronounced environmental stochasticity. In a nutshell, we identify three regimes: for short delays (e.g., $\alpha = 0$), the population grows rapidly but remains prone to extinction due to strong fluctuations (note that true extinction cannot occur in the absence of demographic noise [49,50]); at intermediate delays (e.g., $\alpha = 1$), the system consistently collapses to extinction. For large delays (e.g., $\alpha \gtrsim 5$), the population stabilizes and oscillates around a steady value, with reduced amplitude in both positive and negative fluctuations.

A. Mean population diagram

We have explored the phase diagram of the stationary mean population density x^* as a function of reproductive delay α and the noise amplitude σ (see Fig. 3).

In the deterministic case ($\sigma = 0$), the population reaches the stationary value $x^* = (1 - d/b)K$, which is independent of reproductive delay. Once environmental fluctuations are introduced ($\sigma > 0$), the mean density becomes strongly delay-dependent: the largest values of x^* occur for vanishing delays

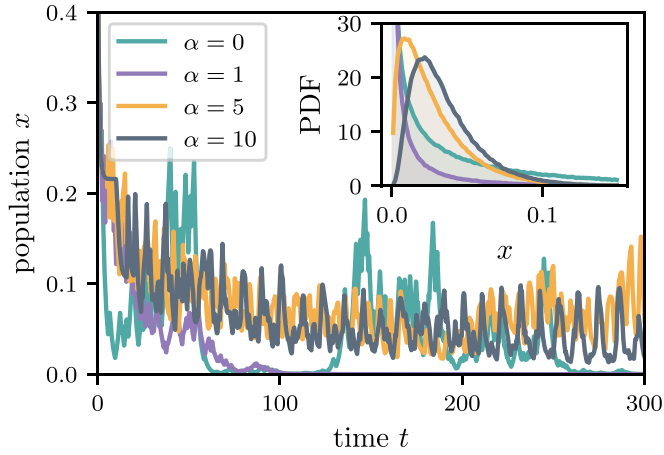


FIG. 2. Time series of the population density $x(t)$ for different values of the reproductive delay α , illustrating the effect of delayed reproduction under environmental noise. The inset shows the corresponding stationary probability density functions of $x(t)$, highlighting qualitative changes in population variability across delay regimes. Parameters: $b = 1.05$, $\tau = 1$, $\sigma = 0.5$, $d = 1$, $K = 1$.

($\alpha \rightarrow 0$), and to a lesser extent for very prolonged delays. For intermediate α , reproduction becomes significantly less effective, producing a pronounced depression in the population level. As σ increases, this depression deepens and broadens, forming a valley of low mean population that may eventually drive the system towards extinction (see Fig. 3, lower panel).

A qualitatively similar diagram is obtained in the (α, τ) plane for finite σ , where increasing τ has an effect analogous to increasing σ (see Appendix C). This will be further clarified in the following section.

B. Mean growth rate

To gain further insight into the aforementioned phenomenology, we analyze the linearized dynamics by taking the limit $K \rightarrow \infty$ in Eq. (1). This effectively removes density-dependent saturation effects and isolates the influence of reproductive delay and environmental variability on the intrinsic growth dynamics:

$$\dot{x}(t) = (b + \sigma \xi_\tau(t))x(t - \alpha) - d x(t). \quad (2)$$

In this regime, we compute the mean growth rate, defined as

$$G = \lim_{T \rightarrow \infty} \left\langle \frac{\log \left(\frac{x(T)}{x(0)} \right)}{T} \right\rangle, \quad (3)$$

where the average is taken over independent realizations of the environmental noise. In practice, the limit is approximated numerically by choosing a sufficiently large integration time, $T = T_{\max}$.

The main panel of Fig. 4 shows the mean growth rate G as a function of reproductive delay α , for fixed correlation time τ and varying noise amplitude σ . The growth rate is always maximal at $\alpha = 0$, which corresponds to the nondelayed reference model, i.e., the absence of an obligatory pre-reproductive interval. Its decay with increasing delays, however, depends strongly on the level of environmental variability. For small

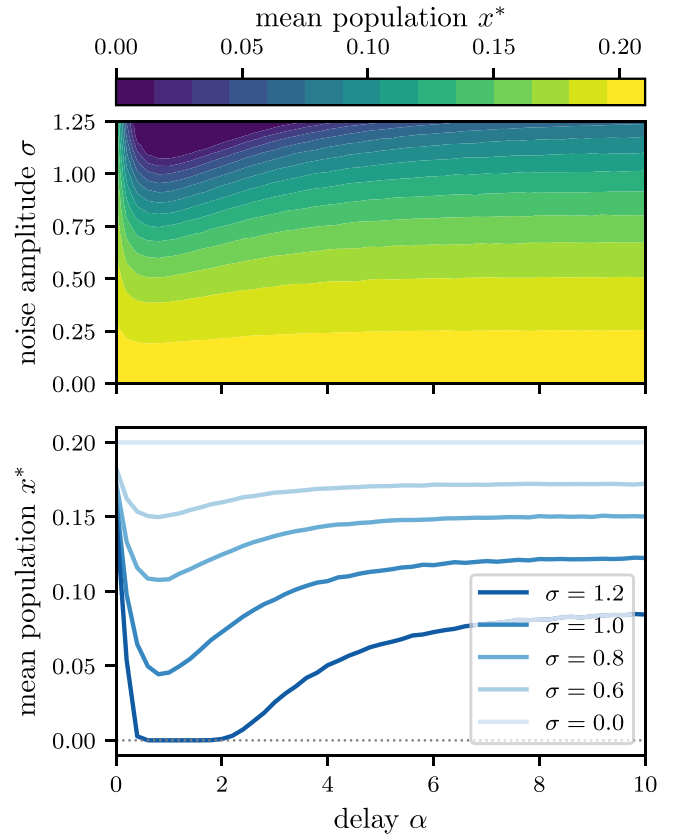


FIG. 3. Mean population density x^* as a function of reproductive delay α and noise amplitude σ . Upper panel: heat map of x^* in the (α, σ) plane. Lower panel: the same data shown as x^* versus α for different fixed values of σ , highlighting its nonmonotonic behavior. To calculate x^* , we numerically integrate Eq. (1) up to a burn-in time $T_{\min}$ to remove transients, compute the time average over the window $[T_{\min}, T_{\max}]$, and then average over independent noise realizations. Intermediate delays combined with strong noise leads to a marked decrease in population size, reflecting poor timing strategies. Parameters: $d = 1$, $b = 1.25$, $\tau = 1$, $K = 1$, $T_{\min} = T_{\max}/2$, $T_{\max} = 10^4$, averages over 10^3 realizations.

noise amplitudes, $G(\alpha)$ decreases monotonically. Beyond a certain threshold in σ , the curve develops a local minimum followed by a local maximum (i.e., via a fold, or saddle-node bifurcation in the language of dynamical systems [51]). Notably, for sufficiently large noise amplitudes, this local minimum becomes global, making intermediate delays the least favorable strategy. This result is consistent with the previous section for finite K , and depressions in $G(\alpha)$ at intermediate delay durations mirror the valley of low stationary densities reported for x^* (Fig. 3). Finally, in the asymptotic regime of prolonged delays, $\alpha \rightarrow \infty$, the influence of environmental variability vanishes and $G(\alpha)$ tends to the deterministic profile ($\sigma = 0$).

The reported behavior becomes even more pronounced as the average birth rate b approaches the death rate d (see Appendix B), indicating that proximity to the extinction threshold magnifies the impact of reproductive delays and environmental correlations on G .

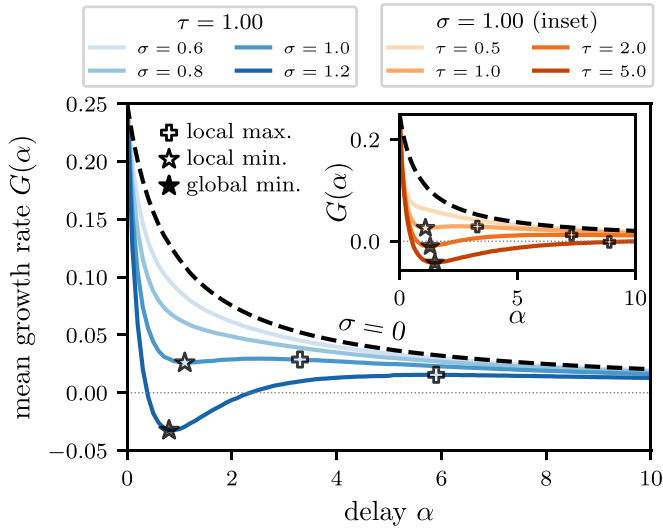


FIG. 4. Mean growth rate G as a function of reproductive delay α , for different values of the noise amplitude σ (main panel) and correlation time τ (inset), in the model with unbounded growth, $K = \infty$ (numerical results from simulations). While G is maximal at $\alpha = 0$ for all cases, increasing σ or τ leads to the emergence of a local minimum at intermediate delays that eventually becomes global (indicated with different symbols). The dashed lines correspond to the analytical solution of the deterministic case, $\sigma = 0$ [see Eq. (4)]. Parameters: $d = 1$, $b = 1.25$, $T_{\max} = 10^3$, averages over 10^4 independent realizations.

A similar qualitative behavior is observed when keeping σ fixed and increasing the correlation time τ (see inset of Fig. 4).

1. Analytical calculation

We develop a semianalytical framework to further investigate the influence of each parameter and timescale on $G(\alpha)$. The literature provides general approaches to treat either stochastic differential equations with delay and white noise [52], or nondelayed systems subject to colored noise [53], but a unified framework that simultaneously accounts for both delay and time-correlated noise is extremely challenging and, to the best of our knowledge, still lacking. Here, we do not attempt to address this general problem, but rather aim to derive an approximate expression tailored to our model. To this end, let us first analyze the deterministic case ($\sigma = 0$), where the population grows exponentially with a growth rate G_0 , $x(t) = x(0)e^{G_0 t}$. Introducing this expression into Eq. (2), one finds that

$$G_0 = be^{-\alpha G_0} - d = b_{\text{eff}} - d, \quad (4)$$

where we have introduced the effective birth rate $b_{\text{eff}} = be^{-\alpha G_0}$ for convenience. This implicit equation for $G_0(\alpha)$ is well known in the literature on delayed population models [47] and must be solved numerically (dashed line in Fig. 4).

It is instructive to analyze the behavior of $G_0(\alpha)$ at small and large delays. For short delays, expanding the exponential in Eq. (4) to first order gives $G_0 \simeq (b - d)/(1 + \alpha b) \simeq (b - d)(1 - \alpha b)$ (with $\alpha \ll b^{-1}$). This also shows that the initial decay scale of both G_0 and b_{eff} is set by b^{-1} . For large

delays, setting $y = \alpha G_0$ shows that y remains bounded and the balance $be^{-y} \rightarrow d$ is required, hence $G_0(\alpha) \simeq \ln(b/d)/\alpha$.

In the presence of fluctuations, we can introduce the ansatz $x(t) = x(0)e^{-\int_0^t g(s)ds}$ in Eq. (2), where $g(t)$ is the *instantaneous* growth rate (note that $\lim_{t \rightarrow \infty} \langle g(t) \rangle = G$). We then obtain an equation for $g(t)$ that must be solved self-consistently:

$$g(t) = (b + \sigma \xi_\tau(t)) e^{-\int_{t-\alpha}^t ds g(s)} - d. \quad (5)$$

The integral can be estimated in two asymptotic limits. For small delays ($\alpha \ll b^{-1}$), we approximate the integral by evaluating it at time t , $\int_{t-\alpha}^t g(s)ds \simeq \alpha g(t)$. Then we write $g(t) = G_0 + \sigma g_1(t)$ and Taylor expand $e^{-\alpha g_1(t)}$ to linear order in α . We further assume that the noise amplitude σ is small and discard all mixed terms of order $\sigma^2 \alpha$. Many terms cancel, yielding an explicit equation for $g_1(t)$:

$$g_1(t) = \frac{b_{\text{eff}}/b}{1 + b_{\text{eff}}\alpha} \xi_\tau(t). \quad (6)$$

This result can be introduced in Eq. (5) to compute the mean growth rate G for small delays:

$$\begin{aligned} G_{\text{small}} &= \left\langle (b + \sigma \xi_\tau(t)) e^{-G_0 \alpha} \left(1 - \sigma \frac{b_{\text{eff}}/b}{1 + b_{\text{eff}}\alpha} \int_{t-\alpha}^t \xi_\tau(s) ds \right) \right\rangle \\ &= G_0 - \sigma^2 \frac{(b_{\text{eff}}/b)^2}{1 + b_{\text{eff}}\alpha} \int_{t-\alpha}^t \langle \xi_\tau(t) \xi_\tau(s) \rangle ds \\ &= G_0 - \frac{1}{2} \sigma^2 \tau \frac{(b_{\text{eff}}/b)^2}{1 + b_{\text{eff}}\alpha} (1 - e^{-2\alpha/\tau}), \end{aligned} \quad (7)$$

where we have used $\langle \xi_\tau(t) \xi_\tau(s) \rangle = e^{-2|t-s|/\tau}$.

On the other hand, for large delays ($\alpha \gg b^{-1}$), the integral can be replaced by its mean value, $\int_{t-\alpha}^t g(s)ds \simeq \alpha G_{\text{large}}$, and taking the average in Eq. (5) yields an implicit equation for G_{large} that coincides with Eq. (4). Therefore,

$$G_{\text{large}} = G_0, \quad (8)$$

as indeed is observed in Fig. 4.

We finally introduce a heuristic expression that interpolates between the two regimes, $G(\alpha) = f(\alpha) G_{\text{small}} + (1 - f(\alpha)) G_{\text{large}}$, where $f(\alpha)$ is an interpolation function, e.g., $f(\alpha) = (1 + \alpha b)^{-1}$; other choices for this function yield similar performance and do not significantly affect the forthcoming results. Substituting the expressions, we obtain

$$G = G_0 - \frac{1}{2} \sigma^2 \tau \frac{(b_{\text{eff}}/b)^2}{(1 + b_{\text{eff}}\alpha)(1 + b\alpha)} (1 - e^{-2\alpha/\tau}). \quad (9)$$

We have assessed the validity of this approximation in Fig. 5 (solid line), comparing it with numerical simulations for increasing values of σ at fixed τ (upper panel), and increasing τ at fixed σ (lower panel). Overall, the agreement is good: it captures the qualitative dynamics and the locations of the local extrema. This provides a semianalytical expression that captures the dependence of the growth rate on the model parameters.

The noise-induced correction in Eq. (9) exhibits a single peak (see Fig. 5) from the competition between the decreasing term $\frac{(b_{\text{eff}}(\alpha)/b)^2}{(1 + \alpha b_{\text{eff}}(\alpha))(1 + b\alpha)}$ and the increasing factor $(1 - e^{-2\alpha/\tau})$. Each factor acts on its own scale: the former decays over

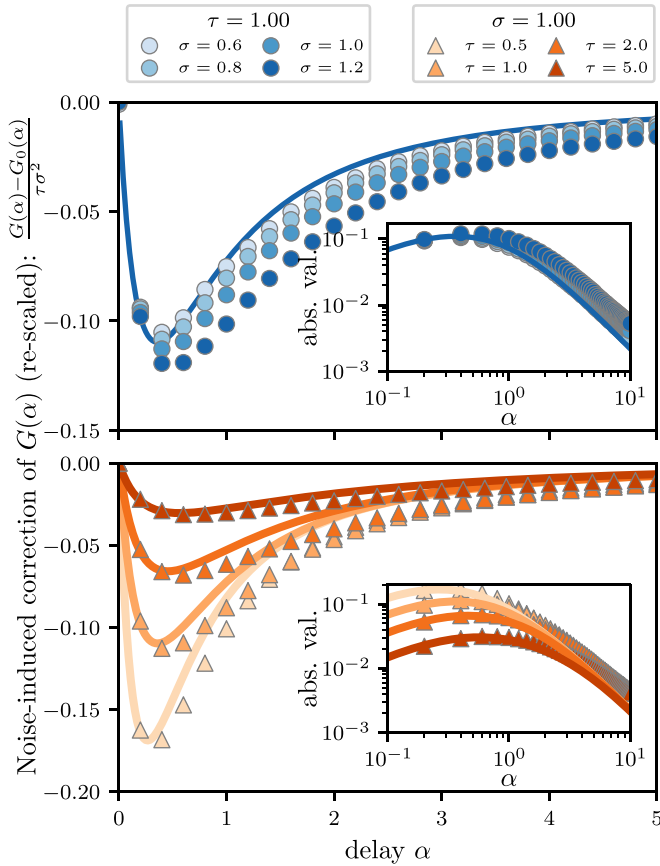


FIG. 5. Noise-induced correction to the mean growth rate as a function of reproductive delay α , rescaled by the factor $\tau\sigma^2$. Solid lines correspond to the approximation given in Eq. (9), while points denote numerical simulation results. Top panel: fixed correlation time $\tau = 1$ and varying noise amplitude σ . Bottom panel: fixed $\sigma = 1$ and varying τ . Insets show the same data (in absolute value) on log-log scale, highlighting the asymptotic decay at large values of α . Parameters: $d = 1$, $b = 1.25$.

b^{-1} (via b_{eff} and the denominators), while the latter grows over the environmental correlation time $\tau/2$. Although we cannot provide a closed-form expression for the peak location—marking the time of worst performance—its scale is set by the shorter limiting timescale, $\alpha_* \sim \min\{\tau/2, b^{-1}\}$.

C. Extinction times

We now explore the resilience of the population to external perturbations by analyzing how delay duration impacts the mean extinction time. It is important to note that in order to observe true extinction ($x = 0$), demographic fluctuations must be included in Eq. (1). In the absence of demographic noise, the absorbing state cannot be reached in finite time [49].

To overcome this limitation, we adopt the following standard approach: we introduce an artificial absorbing boundary at $x = 1/N$, effectively mimicking the population density corresponding to a single individual in a finite population of size N . Starting from a given initial history—here $x(t) = K/2$ for $t \in [-\alpha, 0]$, with results robust to other smooth histories—we compute the mean first-passage time $\bar{T}$ to this boundary,

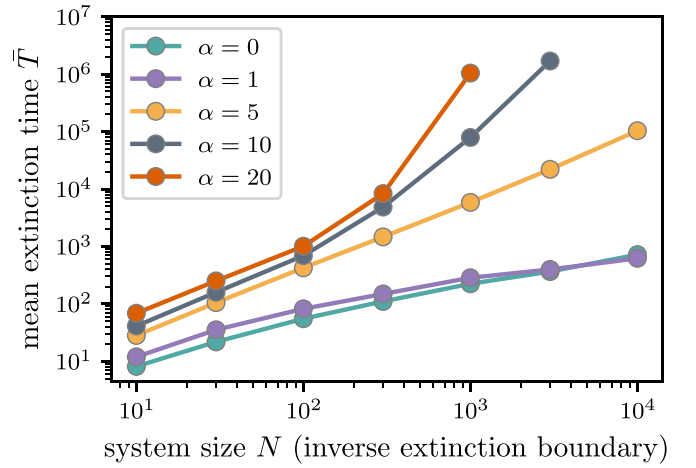


FIG. 6. Mean extinction time $\bar{T}$ (measured as the time to reach the absorbing boundary $x = 1/N$) as a function of system size N , for different values of the reproductive delay α . Parameters: $d = 1$, $b = 1.05$, $\tau = 1$, $\sigma = 0.5$, $K = 1$. Each point represents an average over 10^3 realizations.

which we use as a proxy for the mean extinction time. This procedure yields results consistent with more elaborate demographic simulations while remaining computationally efficient [54,55].

Figure 6 shows the dependence of $\bar{T}$ on the effective system size N for different values of reproductive delay α . In all cases, $\bar{T}$ increases with N . Interestingly, prolonged dormancy consistently leads to longer mean extinction times. For the set of parameters chosen here, distinct scaling regimes emerge depending on α : $\bar{T}$ grows relatively slowly with N when delays are short (i.e., sublinearly), indicating limited resilience. In contrast, for longer delays, the population becomes more stable, and $\bar{T}$ increases more rapidly with N . This behavior is consistent with the trends observed in Fig. 2 (where we use the same parameter values to facilitate comparison), where large delays suppress both positive and negative fluctuations, thereby reducing extinction risk. This result highlights the stabilizing effect of reproductive delay under environmental variability.

So far, we have analyzed two key indicators of population performance: the ability to grow under unbounded conditions, captured by the mean growth rate G (in the limit $K \rightarrow \infty$), and the ability to persist over time, measured by the mean extinction time $\bar{T}$ (with an absorbing boundary at $x = N^{-1}$). In Fig. 7, we make an explicit comparison between these two quantities both by fixing the correlation time τ and varying the noise amplitude σ (upper panel), and by fixing σ and varying τ (lower panel). Each point corresponds to a different value of reproductive delay α .

As expected, the system faces a trade-off: short delays maximize growth (G) but results in shorter extinction times ($\bar{T}$), whereas prolonged lags promote resilience at the cost of slower growth. These differences become more pronounced under strong (σ large) or persistent (τ large) environmental fluctuations. Interestingly, both panels reveal a nonmonotonic behavior: the curve bends backward for intermediate values of α , simultaneously lowering G and $\bar{T}$. These regimes

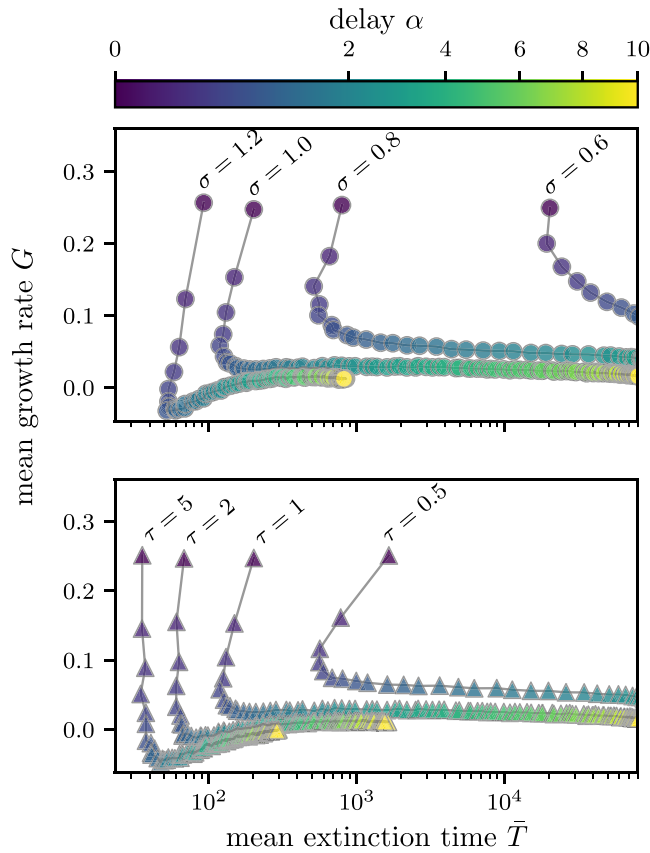


FIG. 7. Trade-off between mean growth rate G and mean extinction time $\bar{T}$ for different values of the reproductive delay α . Each curve corresponds to a different level of environmental variability. Parameters: $d = 1$, $b = 1.25$, $\tau = 1$ for variable σ (top panel) and $\sigma = 1$ for variable τ (bottom panel). To calculate G , we set $K = \infty$ and $T_{\max} = 10^3$, and to calculate $\bar{T}$ we set $K = 1$, $N = 10^3$. In both cases, we average over 10^3 realizations.

correspond to the least favorable strategies identified in previous sections.

III. AGENT-BASED, EVOLUTIONARY DYNAMICS

The results developed so far show that demographic delay interacting with temporally correlated environmental variability generates a fitness landscape with two adaptive peaks and a maladaptive intermediate region. However, these analyses quantify growth and persistence, not evolutionary success. In real biological systems, natural selection operates on populations composed of individuals with heritable variation, demographic stochasticity, and finite lifetimes. Whether the nonmonotonic structure identified above truly translates into evolutionary dynamics is therefore not guaranteed.

To test whether the predicted bistability represents a genuine evolutionary mechanism, we developed an explicit agent-based model in which the reproductive delay is an evolving trait subject to mutation and natural selection (see lower panel of Fig. 1). The ecological setting matches the stochastic environment studied in Sec. II: resources fluctuate between “good” and “bad” states according to a dichotomous Markov process with correlation time τ . Agents consume

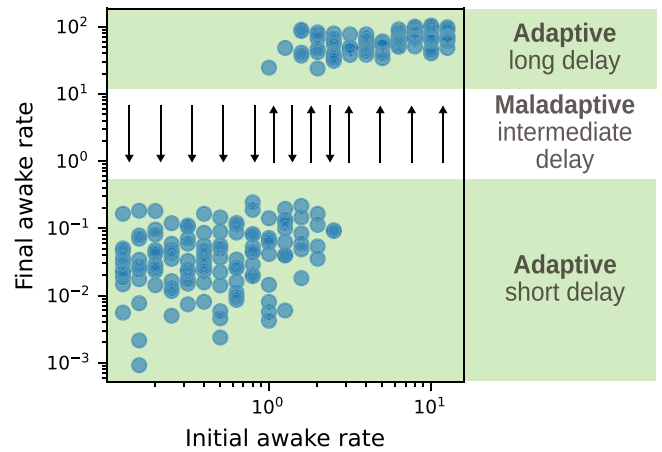


FIG. 8. Evolutionary validation of the predicted bistable reproductive-delay strategies. Final population-mean delay rates α are plotted against their initial values across independent evolutionary simulations. The population consistently evolves toward one of two adaptive attractors: a short-delay strategy or a long-delay strategy, while avoiding intermediate values. Populations initiated in the maladaptive region never remain there; instead, natural selection drives them toward one of the two fitness peaks predicted by the delayed-logistic analysis. Parameter values: $\mu = 100$, $\sigma = 90$, $d = 0.1$, $v = 0.01$, and $\tau = 4$; simulation time is 2×10^5 .

resources, reproduce proportionally to intake, and generate propagules that remain inactive until they establish. Importantly, each individual carries an inheritable activation rate α , which governs the probability per unit time that a propagule becomes active. While this formulation introduces natural variability in delay duration, its role here is not to modify the phenomenology of delay, but to allow delay strategies to evolve in a biologically realistic manner. Mortality affects only active agents, enabling dormant propagules to form a persistent reservoir analogous to seed banks or microbial persisters. All ecological and evolutionary events are simulated in continuous time using a Gillespie algorithm [56], ensuring full demographic and environmental stochasticity.

Despite these additional sources of variability and the lack of any imposed fixed delay, the evolutionary outcomes closely mirror the predictions derived from the delayed-logistic framework. As shown in Fig. 8, populations starting with low values of α evolve toward a stable short-delay strategy, while those starting with large α converge to a stable long-delay strategy. Crucially, populations initiated with intermediate α do not remain there. Instead, stochastic evolutionary trajectories drift away from the maladaptive region, ultimately reaching one of the two adaptive strategies. This confirms that the performance valleys and peaks identified in Sec. II represent evolutionary features of the fitness landscape generated by our model, rather than artifacts of fixed-delay assumptions or idealized population-level dynamics.

The emergence of evolutionary bistability in this fully stochastic model parallels empirical observations of timing strategies in nature, such as those found in microbial persisters, plant seed banks, and other systems where populations diversify into fast-activating and long-dormant phenotypes.

These results demonstrate that the dual adaptive strategies revealed by our analysis are robust to demographic noise, architecture of the reproductive delay, and details of trait inheritance, reinforcing the view that the interaction between demographic memory and environmental temporal structure fundamentally shapes the evolution of reproductive maturation times.

IV. CONCLUSIONS AND DISCUSSION

We have investigated how the duration of the reproductive delay shapes population performance in environments characterized by temporal correlations. Using a minimal delayed-logistic model with stochastic birth rates, we showed that delayed reproduction and colored environmental noise interact in a highly nontrivial way, producing distinct dynamical regimes and revealing a general principle: the adaptive value of reproductive delay depends critically on the relationship between the delay time and the environmental autocorrelation timescale.

Our analysis demonstrates that maturation time plays a dual, tightly balanced role as a reproductive delay. Very short delays maximize the growth rate, exploiting favorable conditions but amplifying susceptibility to rare downturns. Conversely, prolonged delays buffer environmental variability, enhancing resilience and substantially increasing extinction times even if at the cost of limited growth. Most strikingly, our work reveals the emergence of maladaptive intermediate delays—lag times that simultaneously reduce both growth and persistence below the values of both adaptive phases. This nonmonotonous dependence on the reproductive delay arises robustly from the mismatch between demographic memory and the temporal structure of environmental variability, and it appears across numerical simulations, analytical approximations, and evolutionary models.

These findings show that the fitness landscape of reproductive maturation is not smooth but organized around a bistable structure, with two adaptive peaks separated by a broad maladaptive valley of intermediate delay. This bistability is confirmed in a fully stochastic, evolutionary agent-based model: populations consistently evolve toward either short or long reproductive delays, avoiding the intermediate region. Such evolutionary outcomes match empirical patterns observed in diverse biological systems, including bimodal germination strategies in plant seed banks—where mixed populations of fast-germinating individuals and a persistent dormant reservoir coexist [57–63]—and phenotypic diversification into active and dormant subpopulations in microbes. It has also been posited that positively autocorrelated environments should select for long diapause in insects [64]. In cancer, transitions between quiescent and proliferative states suggest that both extremes can contribute to the survival and spread of tumors. Our results can provide a theoretical framework to understand why intermediate, partially active states where cells are neither fully dormant nor fully proliferative, are particularly susceptible to therapeutic intervention [23–25]. Further research should expand on this to clarify why intermediate or transitional dormancy stages are maladaptive and to what extent this can be manipulated by exogenous factors.

It is important to emphasize, however, that these biological connections should be understood as qualitative analogies rather than direct quantitative applications of the model. Our framework applies most directly to life histories that incorporate a clearly defined, unambiguous reproductive delay: individuals or propagules go through a lag phase before contributing to population growth, activation after this lag is not explicitly conditioned on environmental sensing, and environmental variability acts through the reproductive term. These assumptions leave out several mechanisms that are central to some specific biological systems. For instance, in microbial persistence, dormancy usually arises through phenotypic heterogeneity, with only a minority of cells entering low-activity persister states, rather than as an obligatory stage in the life cycle of all newborn cells [28,42]. In seed germination, exit from dormancy often depends on environmental cues whose reliability shapes germination timing and the evolution of bet-hedging strategies [17,18]. In tumor progression, quiescent and proliferative states are shaped by cell-state heterogeneity and micro-environmental feedbacks, rather than by a uniform post-division delay imposed on every cell [23,24]. Hence, our results should be interpreted as identifying a minimal mechanism by which reproductive delays and correlated environmental noise generate nonmonotonic fitness landscapes, rather than as a full mechanistic model that can be applied directly to any of these systems.

Although our results may lack the necessary realism to fit specific biological examples, the qualitative patterns they describe are likely representative of many real-world scenarios. Reproductive delay is not a passive effect in life-history but an adaptive mechanism tuned to the timescales of environmental-variability autocorrelation. Consequently, organisms should evolve reproductive lag times that match the predictability of their environment. Other authors have shown empirically that dormancy can be regarded as insurance that organisms overcome the (potentially long) stretch of unfavorable conditions and that reproduction coincides with a favorable window (e.g., a specific seasonal cycle) [17,64,65]. Alternatively, selection could favor “*r* strategists” that forego lags in reproductive maturation to ensure rapid exploitation of the environment, while it seems unlikely that intermediate strategies can persist in autocorrelated environments. We believe that this study provides a minimal but powerful theoretical foundation for understanding how environmental heterogeneity shapes adaptive reproductive delay strategies across biological domains. It opens the door to exploring evolutionarily stable distributions of lag times, multimodal bet-hedging strategies, and the ecological and physiological constraints that may shape the observed diversity of reproductive delay mechanisms in nature.

Beyond its biological implications, the present work underscores fundamental theoretical challenges. The model highlights the complexity of delayed, non-Markovian stochastic processes driven by correlated multiplicative noise near absorbing boundaries, an area where rigorous analytical tools remain limited. Our approximate analytical treatment provides initial insight, but future progress will require developing general methods to handle delay kernels, correlated noise, and extinction dynamics in a unified framework.

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DATA AVAILABILITY

Simulation and analysis codes used in this work are openly available on Zenodo [66].

APPENDIX A: NUMERICAL INTEGRATION

We use an explicit Runge-Kutta method of fourth order adapted to handle delay differential equations, following the procedure outlined in Ref. [48]. The key idea is to discretize time with a fixed step Δt and store the past history of the solution in a buffer or ring array, enabling efficient evaluation of the delayed term $x(t - \alpha)$ by interpolation. In our simulations, we typically use time steps $\Delta t \lesssim 0.01$, which ensures numerical stability and accuracy across the range of delays and noise intensities considered.

For the linearized dynamics, we perform the numerical integration after a change of variables. Since, in the absence of saturation, Eq. (2) exhibits unbounded exponential growth or decay, it is more natural to integrate it in logarithmic space. Such transformations have also been used in related problems to preserve positivity and avoid numerical artifacts in the direct integration [67]. In particular, defining $z = \log x$, Eq. (2) becomes

$$\dot{z} = (b + \sigma \xi_\tau(t)) e^{z(t-\alpha)-z(t)} - d. \quad (\text{A1})$$

No additional stochastic calculus (e.g., Itô corrections) is required, as the noise has finite correlation time.

APPENDIX B: DEPENDENCE ON THE AVERAGE BIRTH RATE b

Figure 9 shows the dependence of the mean growth rate $G(\alpha)$ on the reproductive delay α for different values of the mean birth rate b . As expected, the overall growth rate G decreases as b approaches the critical threshold d . More interestingly, the emergence of least favorable strategies—signaled by local minima in $G(\alpha)$ —becomes more pronounced in this marginal regime. This reinforces the idea that proximity to the

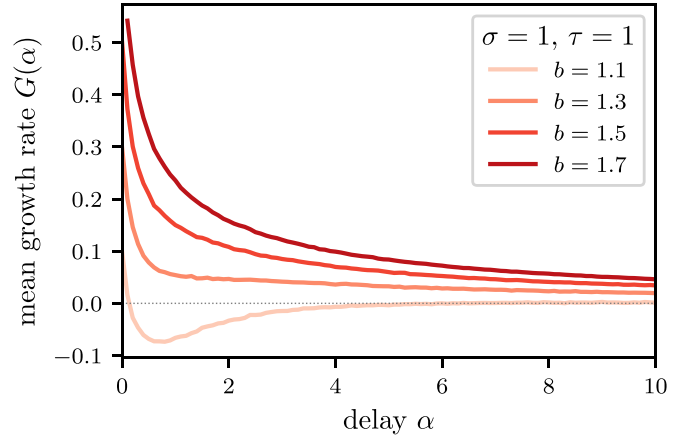


FIG. 9. Mean growth rate G as a function of the reproductive delay α for different mean birth rates b (simulations with the model presented in the main text). The nonmonotonic behavior of $G(\alpha)$ and the emergence of least favorable strategies become more evident when $b \rightarrow d$, approaching the extinction threshold. Parameters: $d = 1$, $\sigma = 1$, $\tau = 1$, $T_{\max} = 10^3$, averages over 10^2 independent realizations.

extinction threshold amplifies the influence of reproductive delay and environmental structure on population performance.

APPENDIX C: MEAN POPULATION DIAGRAM IN THE (α, τ) PLANE

We explore the stationary mean population density x^* as a function of reproductive delay α and the noise correlation time τ (Fig. 10). For finite noise amplitude ($\sigma = 1$), increasing τ produces an effect qualitatively similar to increasing σ in the main text: intermediate values of α lead to a marked reduction in x^* , generating a broad minimum in the population density. This confirms that both the intensity and the temporal correlations of environmental fluctuations shape population viability in a comparable way.

APPENDIX D: ENVIRONMENT MODELED AS AN ORNSTEIN-UHLENBECK PROCESS

The environment can be also modeled as an Ornstein-Uhlenbeck process:

$$\dot{\xi}_{\text{OU}}(t) = -\frac{1}{\tau_{\text{OU}}} \xi_{\text{OU}} + \sqrt{\frac{2}{\tau_{\text{OU}}}} \eta(t), \quad (\text{D1})$$

where $\eta(t)$ is a zero mean, delta-correlated white Gaussian noise. In this case, the environment has zero mean and characteristic time of temporal correlation τ_{OU} [68].

As discussed in the main text, care is needed when integrating regimes where the effective birth rate can become negative: even the simple delay equation $\dot{x} = -x(t - \alpha)$ has the (*a priori* stable) fixed point $x^* = 0$ losing stability beyond a critical delay, and the same mechanism appears in models with delayed birth and death terms [47,48]. For this reason, in simulations we enforce a non-negative birth rate to avoid unphysical destabilization induced by delay.

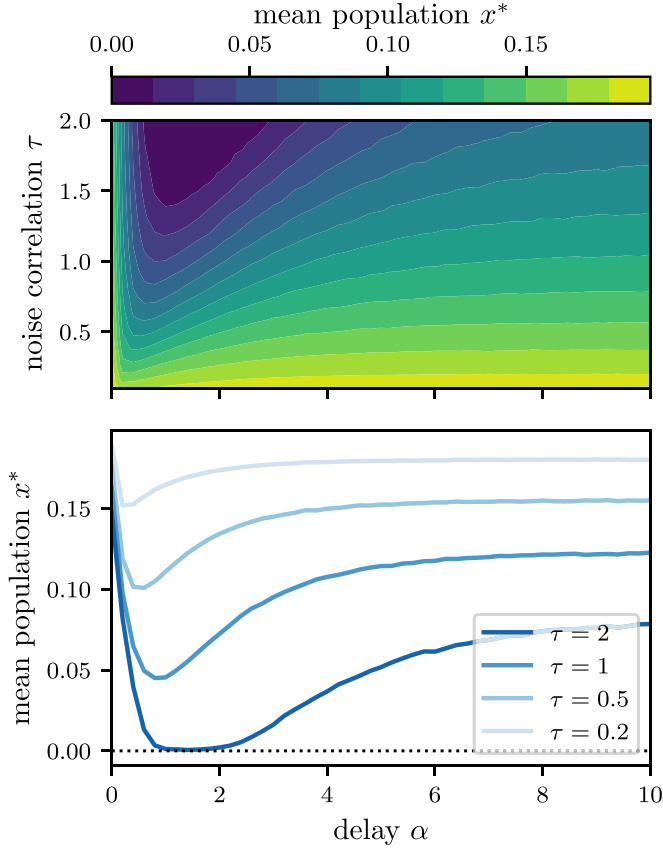


FIG. 10. Mean population density x^* as a function of reproductive delay α and noise correlation time τ . Upper panel: heat map of x^* in the (α, τ) plane. Lower panel: the same data shown as x^* versus α for different fixed values of τ , highlighting its nonmonotonic dependence. Increasing τ leads to a deepening and widening of the minimum at intermediate delays, analogous to the effect of increasing noise amplitude σ . Parameters: $d = 1$, $b = 1.25$, $\sigma = 1$, $K = 1$, $T_{\min} = T_{\max}/2$, $T_{\max} = 10^4$, averages over 10^3 realizations.

To address this issue, we model the birth rate as $\max(0, b' + \sigma \xi_{\text{OU}}(t))$ (i.e., no offspring are produced when the environmental variable falls below a threshold). However, when $\sigma \gtrsim b'$, the time-averaged birth rate b differs from b' . To enable fair comparisons across σ , we therefore choose $b' = b'(\sigma)$ so that the average birth rate is fixed to b . Results are shown in Fig. 11. The same phenomenology as in the case studied in the main text (with dichotomic noise) emerges, highlighting the robustness of the observed behavior.

APPENDIX E: ENVIRONMENTAL VARIABILITY IN THE DEATH RATE

In the main text, we considered environmental variability acting on the birth process. Here, we explore an alternative scenario in which fluctuations instead affect the death rate. Since reproductive delay modulates only the birth term, the delay remains confined to reproduction, and the stochastic differential equation becomes

$$\dot{x}(t) = bx(t - \alpha) \left(1 - \frac{x(t)}{K}\right) - (d + \sigma \xi_\tau(t))x(t). \quad (\text{E1})$$

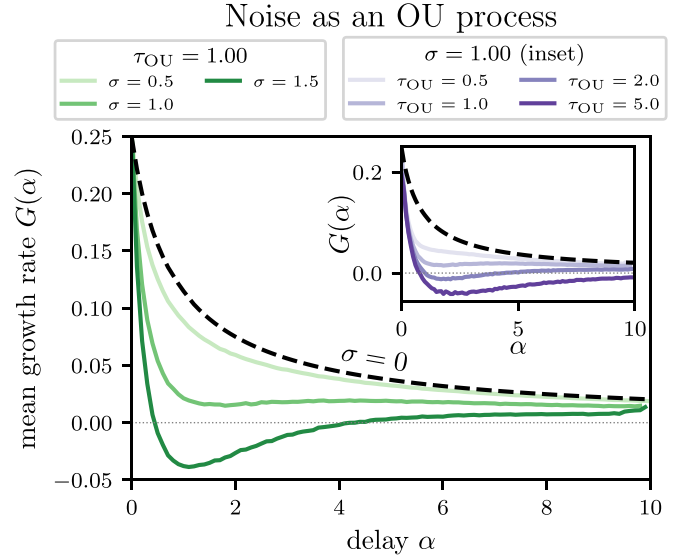


FIG. 11. Mean growth rate G as a function of the reproductive delay α when the birth rate is driven by an Ornstein-Uhlenbeck process and rectified as $\max(0, b' + \sigma \xi_{\text{OU}}(t))$. Main panel: dependence on the noise amplitude σ at fixed τ_{OU} ; inset: dependence on the correlation time τ_{OU} at fixed σ (simulation results). In all cases, the same phenomenology emerges as for dichotomic noise (see main text). To enable a fair comparison, b' is chosen so that the time-average of the birth rate equals b . Parameters: $d = 1$, $b = 1.25$; $b'(\sigma=0.5) = 1.249$, $b'(\sigma=1) = 1.194$, $b'(\sigma=1.5) = 1.035$; $T_{\max} = 10^3$; averages over 10^3 independent realizations.

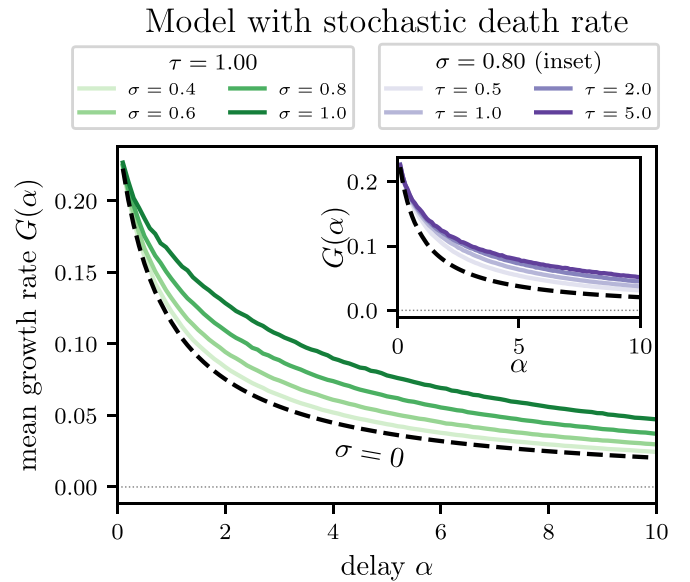


FIG. 12. Mean growth rate G as a function of the reproductive delay α , for different values of the noise amplitude σ (main panel) and correlation time τ (inset), in the linearized model with stochastic death rate (simulation results). In contrast to the case with noise in the birth rate, here $G(\alpha)$ decreases monotonically with increasing delay, regardless of σ or τ . This highlights the crucial role of the coupling between noise and delay in generating nonmonotonic behaviors. Parameters: $d = 1$, $b = 1.25$, $d = 1$, $T_{\max} = 10^3$, averages over 10^3 independent realizations.

Figure 12 shows the results of integrating this model in the linear regime ($K \rightarrow \infty$), using DMN. In contrast to the case where fluctuations act on the birth rate, here the mean growth rate $G(\alpha)$ decays monotonically with the reproductive delay α , without developing any local extrema. This indicates that the emergence of least favorable strategies—i.e.,

intermediate values of α that simultaneously reduce growth and persistence—is not a generic consequence of delay under noise. Instead, such regimes result specifically from the direct coupling between reproductive delay and environmental variability, which is absent when noise acts solely on the instantaneous death rate.

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