

Lag-induced critical transitions to extinction in replicating systems: the τ -tipping mechanism

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Replicating systems sustained by error-prone enzymatic amplification can undergo critical transitions between persistence and extinction. In RNA viruses, such transitions are classically governed by mutation rates and fitness landscape topographies, giving rise to error thresholds and lethal mutagenesis. Motivated by experimental evidence that polymerase-targeting antivirals limit replication, we analyze replicating systems with explicit time lags in replication-enzyme availability by means of delay differential equations. Our approach is not equivalent to a simple renormalization of the rate of replication, since explicit delays introduce memory and temporal nonlocality into the dynamics. We identify a lag-induced critical transition driven by the loss of temporal coordination between genome expression and replication. At fixed mutation and replication rates, populations cross an extinction threshold solely due to replication delays. In a delay-extended quasispecies model, the timing of replicase availability emerges as an independent dynamical control parameter, defining a route to extinction that is fundamentally distinct from transitions driven by mutation rates or replicative fitness. These results further suggest that perturbing the temporal coordination of viral replication may provide an alternative antiviral strategy for driving viral populations toward collapse. More generally, we propose that the extinction mechanism described here constitutes a form of lag-time-induced tipping, which we denote as τ -tipping.

I. INTRODUCTION

Replicating populations are nonequilibrium systems that can undergo sharp transitions between persistence and extinction. Classical theoretical approaches have emphasized replication, mutation, selection, and the topography of fitness landscapes as the primary determinants of these transitions. Within this setting, quasispecies theory provides a canonical framework for describing the collective dynamics of replicating genomes under high mutation rates [2, 5–8], with RNA viruses as a prototypical realization. A quasispecies is a genetically heterogeneous population maintained around a master sequence, whose dynamics can exhibit critical transitions in information fidelity and population survival. Several information and extinction thresholds have been identified, including the error threshold [5, 6], lethal mutagenesis [3, 4], and lethal defection [9]. Beyond their theoretical significance, these transitions underpin antiviral strategies aimed at driving viral populations to extinction [1, 10, 11].

Experimental and computational studies have provided evidence for distinct mutational transitions across RNA viruses. Error catastrophe, associated with the loss of genetic information beyond a mutation threshold, was first demonstrated in poliovirus treated with ribavirin [12] and later inferred in hepatitis C virus (HCV) data using quasispecies models [2], with computational studies suggesting that human immunodeficiency virus type-1 (HIV-1) operates near this threshold [13]. Lethal defection, driven by the accumulation of defective interfering genomes, has been observed in lymphocytic choriomeningitis virus and tobacco mosaic virus under fluorouracil treatment [14, 15]. Lethal mutagenesis, characterized by extinction through mutation accumulation under mutagenic pressure, has been reported in foot-and-mouth disease virus, HCV, HIV-1, influenza A virus, and, more recently, in SARS-CoV-2 [3, 16–19]. Together, these results demonstrate the broad relevance of informational and population-level thresholds in viral evolution and pathogenesis.

All previously characterized routes to viral quasispecies extinction rely on changes in parameters such as mutation rates, replication efficiencies, or competitive asymmetries between genomes. Here, we show that

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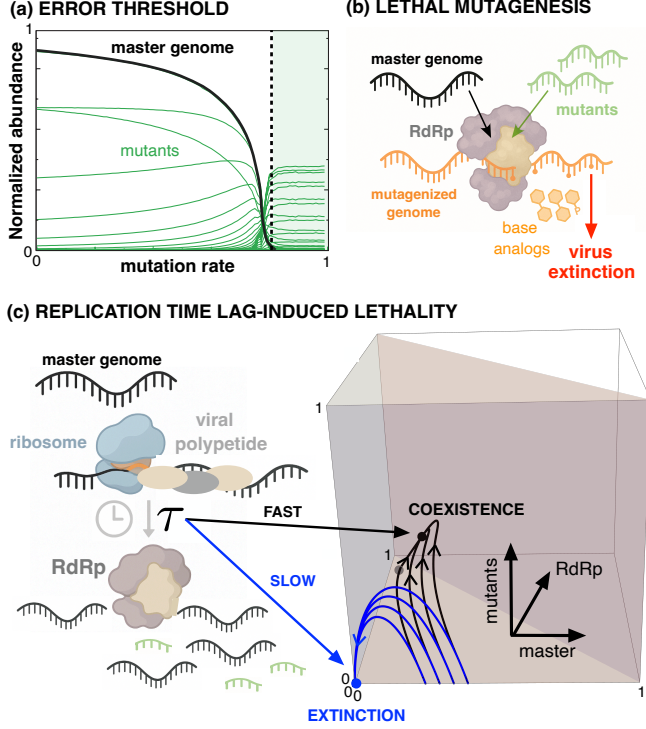


FIG. 1. Critical transitions in error-prone self-replicating populations, such as RNA viruses. (a) The error threshold marks mutant dominance over the master genome [1, 2]. (b) Lethal mutagenesis causes extinction via mutation accumulation [3, 4]. (c) Replication time-lag-induced lethality identified in this study. Our model tracks master and mutant genomes replicated by the RNA-dependent RNA polymerase (RdRp), with a time lag τ in its functional availability. Phase-space trajectories are computed from Eqs. (1) for different situations: early RdRp production ($\tau = 0.65$, black) supports persistence, whereas long lags ($\tau = 50$, blue) cause collapse. Panels (a,b) show mutation- and fitness-driven transitions, while (c) shows extinction driven solely by replication timing at fixed mutation and replication rates.

extinction can instead arise solely from temporal constraints in replication, even when mutation and replication rates remain unchanged. This identifies the timing of functional replicase availability, relative to genome expression and replication, as an additional dynamical control parameter in quasispecies-inspired viral replication models, defining a distinct route to extinction beyond classical mutation- or fitness-driven mechanisms. In particular, the virus-encoded RNA-dependent RNA polymerase (RdRp), responsible for viral genome amplification, must first be translated from viral messenger RNAs by host ribosomes and acquire its proper folding and localization before becoming functional. Together with these steps, additional processes can introduce further time lags in replication dynamics (see Appendix A).

In this paper, we describe an alternative route to extinction arising from replicative time lags (τ) between genome replication and protein translation, with partic-

ular emphasis on the availability of the viral RdRp. We analyze a minimal dynamical model describing a replicating population given by a master sequence and an average defective mutant class [2, 20]. Enzymatic replication is explicitly incorporated by treating RdRp as a dynamical variable whose production and functional activation occur with finite time lags. These replicative lags, able to induce quasispecies extinction, may be considered as a novel control parameter in quasispecies dynamics, extending the theoretical framework of error-driven transitions and suggesting new paths in antiviral intervention.

II. QUASISPECIES MODEL WITH DELAYED RDRP AVAILABILITY

We consider a dynamical system derived from the Eigen-Schuster quasispecies model [5], in which the population fractions x_i of n replicating genomes evolve according to $\dot{x}_i = \sum_{j=1}^n x_j f_j Q_{ij} - \phi(\mathbf{x}) x_i$, where f_j denotes the replication rate of genome j , Q_{ij} the mutation matrix, and $\phi(\mathbf{x})$ the mean population fitness. Following a minimal reduction of this framework [21], we retain two effective classes: the master sequence x_0 and an aggregated mutant class x_1 , representing defective genomes generated from the master sequence and dependent on complementation by the master-derived RNA-dependent RNA polymerase (RdRp) [22]. Unlike the original Eigen-Schuster formulation, assuming a constant population, we allow the total population to vary in time, reflecting replication from a small initial pool of genomes. To explicitly account for enzymatic replication, we further incorporate the RdRp concentration, denoted by p , as a dynamical variable whose production and functional availability occur with a finite time lag τ . Thus, the model retains the mutation-selection structure of quasispecies theory, while embedding it in a mechanistic replication scheme in which polymerase production and functional availability are explicitly resolved. The model is given by:

$$\begin{aligned} \frac{dx_0(t)}{dt} &= (1 - \mu) \gamma x_0(t) r_0 p(t - \tau) \Phi(x) - \varepsilon_0 x_0(t), \\ \frac{dx_1(t)}{dt} &= p(t - \tau) [\mu \gamma r_0 x_0(t) + (1 - \gamma) r_1 x_1(t)] \Phi(x) - \varepsilon_1 x_1(t), \\ \frac{dp(t)}{dt} &= k x_0(t) [1 - p(t)] - \varepsilon_p p(t), \end{aligned} \quad (1)$$

where $\Phi(x) = 1 - x_0(t) - x_1(t)$ accounts for competition between genomes under a normalized carrying capacity. In the present minimal formulation, the explicit delay $\tau \geq 0$ is associated only with the functional availability of the RdRp. Additional delays linked to $(+) \rightarrow (-)$ and $(-) \rightarrow (+)$ strand synthesis are implicitly incorporated into the effective replication rates r_0 and r_1 . These replication rates are effective kinetic parameters, not microscopic elementary rates. They summarize the average net production of genomes per unit time after integrating over unresolved subprocesses. While a more detailed strand-resolved description could include separate delays for each replication step, the purpose of the

current model is to isolate the dynamical effects arising from delayed polymerase availability. Here, μ represents an effective transition rate that produces, during replication, an aggregated mutant class defective in functional RdRp, which replicates by complementation with the master RdRp. Following [22, 23], RdRp production is proportional to the amount of master genomes and bounded by logistic growth. Master and mutant genomes replicate at rates r_0 and r_1 , and decay at rates ε_0 and ε_1 , respectively. Viral replication is proportional to RdRp abundance, which becomes functional after a time lag τ , representing a causal constraint associated with RdRp synthesis and maturation that cannot be captured by instantaneous models. This delay introduces a de-synchronization between the translation of the viral genomes that first infect the cell into RdRp and their subsequent maturation and the moment in which these recently synthesized RdRp molecules start producing the progeny of viral genomes. A fraction $\gamma \in (0, 1)$ of RdRp replicates the master genomes, and thus a fraction $(1 - \gamma)$ replicates the mutants. For simplicity, we set $r_0 = r_1 = r$, and $\varepsilon_0 = \varepsilon_1 = \varepsilon_p = \varepsilon$, a choice that preserves the qualitative dynamics while enabling analytical tractability.

We note that the explicit replication lag τ introduced here is conceptually distinct from a simple reduction of the effective replication rates $r_{0,1}$. In standard quasispecies models, replication rates are instantaneous (Markovian) parameters encoding average amplification efficiency. By contrast, the delay term $p(t - \tau)$ introduces explicit dependence on past system states, rendering the dynamics nonlocal in time and intrinsically non-Markovian. As a consequence, increasing τ does not merely rescale replication efficiency but modifies the dy-

namical structure of the system through delayed feedback between genome abundance and polymerase availability. Indeed, time lags make the system become infinite-dimensional.

III. LAG-INDUCED DYNAMICAL TRANSITION

To assess the impact of time lags in RdRp synthesis and maturation, we first consider the instantaneous case ($\tau = 0$). The system (1) has three equilibrium points: $P_0 = (0, 0, 0)$, involving full extinction; $P_+ = (x_{0+}, x_{1+}, p_+)$ governing genomes-RdRp persistence; and $P_- = (x_{0-}, x_{1-}, p_-)$, with

$$x_{0\pm} = -2(\alpha + \beta)\varepsilon^2 / (k(\alpha + \beta)(\varepsilon - \beta) \pm \chi),$$

$$x_{1\pm} = -2\gamma\mu\varepsilon^2 / (k(\alpha + \beta)(\varepsilon - \beta) \pm \chi),$$

and $p_{\mp} = 2k(\alpha + \beta)\varepsilon / (k(\alpha + \beta)(\varepsilon + \beta) \mp \chi)$, where the upper sign corresponds to P_+ and the lower to P_- . Here,

$$\chi = \sqrt{-k(\alpha + \beta)(4\beta(\alpha + \gamma)\varepsilon^2 - k(\alpha + \beta)(\varepsilon - \beta)^2)},$$

$\alpha = r(1 - \gamma)$, and $\beta = \gamma(\mu - 1)$. Whenever the equilibria $P_{\pm}$ are real, numerical explorations verify that, for any value of the lag parameters, P_0 and P_+ are attractors, separated by the stable manifold of the saddle equilibrium P_- . Figure 2(a) shows a phase diagram with the population equilibria of master genomes at increasing γ and μ setting $\tau = 0$. The boundary between persistence and extinction of the master genome corresponds to a saddle-node bifurcation; extinction of both the master genome and mutants occurs for increasing mutation rates or decreasing fractions of RdRp replicating the master genome.

Numerical integration [24] of eqs. (1) confirms the analytical predictions. For $\gamma = 0.9$ and $\mu = 0.4$ [Fig. 2(a)], trajectories converge to the persistence equilibrium. Increasing the replication time lag τ redirects the same initial conditions toward extinction, without changing mutation rates or replication fitness. Figure 2(b) displays orbits in the phase space achieving persistence for $\tau = 0$ (blue trajectory) and extinction for larger lags (red and green orbits). As a result, increasing τ reshapes the basins of attraction, markedly expanding the extinction basin, as quantified in Fig. 3. This figure displays the basins of attraction of P_0 and P_+ , showing, in a colour gradient, the time it takes for each initial condition to achieve the corresponding attractor. Notice that the extinction basin enlarges as the time lag τ increases [see panels (a-c)]. Moreover, the time series in panel (d) show, for $\tau > 0$, an initial fast decrease in genome abundance, reflecting the regime in which viral genomes are lost faster than functional RdRp becomes available.

To clarify how changes in τ alter the basins of attraction, we next consider a simplified model with the same qualitative dynamics of eqs. (1). Let us introduce a minimal two-variable model consisting of a replicator x that

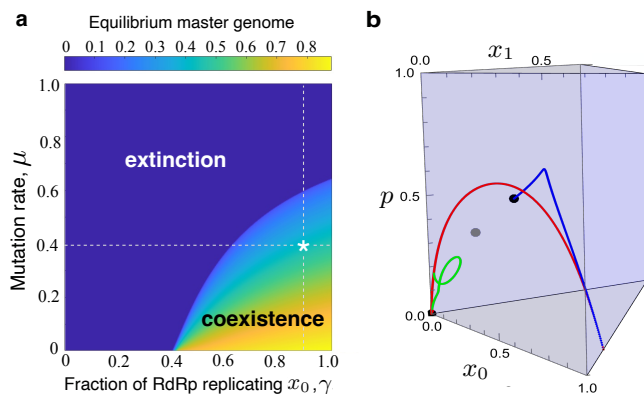


FIG. 2. (a) Phase diagram of the master genome equilibrium obtained from eqs. (1) for $\tau = 0$, showing persistence at low μ and large γ . The blue area corresponds to the full collapse of the system. (b) Phase portrait at $(\gamma, \mu) = (0.9, 0.4)$ [asterisk in (a)] showing three trajectories with the same initial condition ($x_0(0) = 0.8$, $x_1(0) = 0.2$, and $p(0) = 0$) for $\tau = 0$ (blue), $\tau = 25$ (red trajectory overlapped with the green one), and $\tau = 50$ (green). Increasing τ drives extinction. Black and gray dots denote stable equilibria and saddles. Here we used $r = 0.5$, $k = 0.5$ and $\varepsilon_{0,1,p} = 0.14$.

encodes its polymerase y with logistic growth and linear degradation. The model reads:

$$\begin{aligned}\frac{dx(t)}{dt} &= x(t) y(t - \tau) [1 - x(t)] - \varepsilon x(t), \\ \frac{dy(t)}{dt} &= x(t) [1 - y(t)] - \varepsilon y(t).\end{aligned}\quad (2)$$

This system has three equilibrium points given by the origin $P_0 = (0, 0)$, and the pair $P_{\pm} = (x_{\pm}, x_{\pm}/(x_{\pm} + \varepsilon))$, with

$$x_{\pm} = \frac{1}{2} \left[(1 - \varepsilon) \pm \sqrt{1 - 2\varepsilon - 3\varepsilon^2} \right].$$

The equilibrium points $P_{\pm}$ exist for $0 \leq \varepsilon \leq 1/3$. Linear stability analysis shows that the origin has eigenvalues $\lambda_{1,2} = -\varepsilon$ and is therefore locally asymptotically stable. The evaluation of the Jacobian shows that $\det J(P_-) < 0$, thus indicating that P_- is a saddle point, while $\det J(P_+) > 0$ and $\text{tr} J(P_+) < 0$, showing that P_+ is a stable node. These two latter equilibria merge at $\mathbf{x}_c = (1/3, 1/2)$ in a saddle-node bifurcation when $\varepsilon = \varepsilon_c = 1/3$, ε_c being the bifurcation value.

This simplified model allows an analytical characterization of the lag-induced basin reorganization through the τ -dependent spectrum of nontrivial equilibria. The equilibria of eqs. (2) are independent of τ , however, the basins are τ -dependent because the basin boundary is an invariant manifold in the (history) phase space of the DDE. The separatrix dividing the basins of the origin and the persistent states, given by the stable manifold of the saddle, persists for $\tau > 0$ as the stable manifold $W^s(P_-; \tau)$ in the history space, and its geometry varies with τ . To see this explicitly, linearizing eq. (2) around any nontrivial equilibrium $P_* = (x^*, y^*)$, *i.e.*, P_- , gives the linear DDE of the form

$$\frac{d\mathbf{u}}{dt}(t) = A_0 \mathbf{u}(t) + A_1 \mathbf{u}(t - \tau),$$

whose eigenvalues λ satisfy

$$F(\lambda; \tau) \equiv (\lambda - a)(\lambda - d) - K e^{-\lambda\tau} = 0, \quad (3)$$

with $a = y^*(1 - 2x^*) - \varepsilon$, $d = -(x^* + \varepsilon)$, and

$$K = x^*(1 - x^*)(1 - y^*) = \varepsilon x^*(1 - x^*)/(x^* + \varepsilon).$$

Hence, the spectrum depends on τ through the factor $e^{-\lambda\tau}$; differentiating (3) yields

$$\frac{d\lambda}{d\tau} = -\frac{\partial_{\tau} F}{\partial_{\lambda} F} = -\frac{K \lambda e^{-\lambda\tau}}{2\lambda - (a + d) + K \tau e^{-\lambda\tau}},$$

a value that is generically nonzero, showing that the (un)stable eigendirections of the saddle P_- vary with τ . Because the basin boundary is precisely $W^s(P_-; \tau)$, this implies that changing τ reshapes the basins of attraction of P_0 and P_+ (as also quantified numerically by the τ -dependent separatrix in Fig. 4).

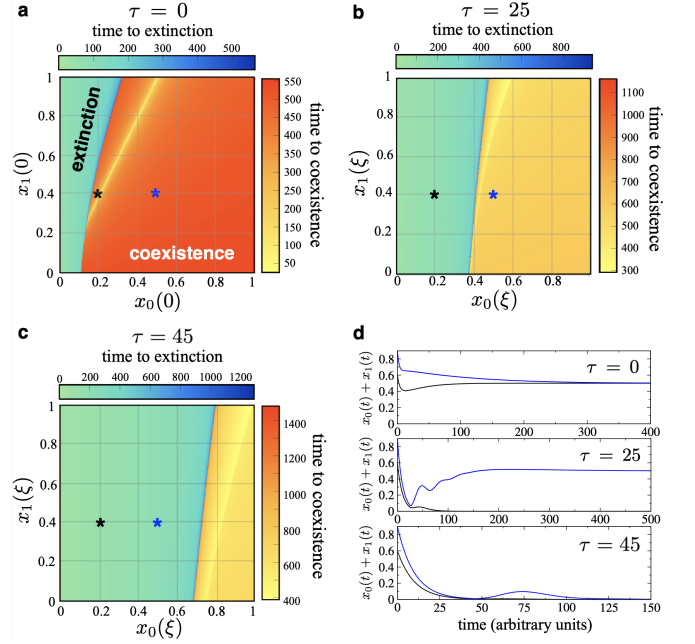


FIG. 3. (a-c) Basins of attraction of eqs. (1) with $p(0) = 0$, $\gamma = 0.5$, $\mu = 0.2$, $r = 0.7$, $k = 0.6$ and $\varepsilon_{0,1,p} = 0.1$. For increasing $\tau = 0$, $\tau = 25$, and $\tau = 45$, we show convergence times to extinction (blue gradient) or persistence (orange gradient) for $\xi \in [-\tau, 0]$. Increasing τ enlarges the extinction basin without changing mutation or fitness traits. (d) Time series of $x_0(t) + x_1(t)$ obtained from the history initial conditions $(x_0(\xi), x_1(\xi)) = (0.2, 0.4)$ (black) and $(x_0(\xi), x_1(\xi)) = (0.5, 0.4)$ (blue).

We next investigate the resulting dynamics numerically [24]. Figure 4 illustrates the dynamics of the simplified system (2) through phase portraits for two values of the degradation rate ε at $\tau = 0$. For low ε [panel (a)], the stable coexistence equilibrium P_+ lies far from the saddle P_- , whose stable manifold defines the separatrix between extinction and persistence. The basin of attraction of the extinction state P_0 is shown in blue. Superimposed are numerically computed separatrices for increasing replication time lags ($\tau = 1, 5, 10, 25, 50$), which progressively shift toward larger x , indicating a marked expansion of the extinction basin along the genome-density direction. A similar lag-induced reorganization is observed for larger ε [panel (b)], where both the saddle and P_+ approach each other and the extinction basin again expands with τ . Under biologically relevant initial histories, $x(\xi) \ll 1$ and $y(\xi) = 0$ for $\xi \in [-\tau, 0]$, increasing replication time lags, therefore, bias trajectories toward extinction, consistently with the full model given by eqs. (1).

Finally, we quantify the expansion of the extinction basin induced by the replication time lag τ . For different ε , the lagged system was integrated from a 150×150 uniform grid in $(x, y) \in [0, 1] \times [0, 1]$, and trajectories were classified by convergence to extinction or persistence. The fractions of the extinction basin across this

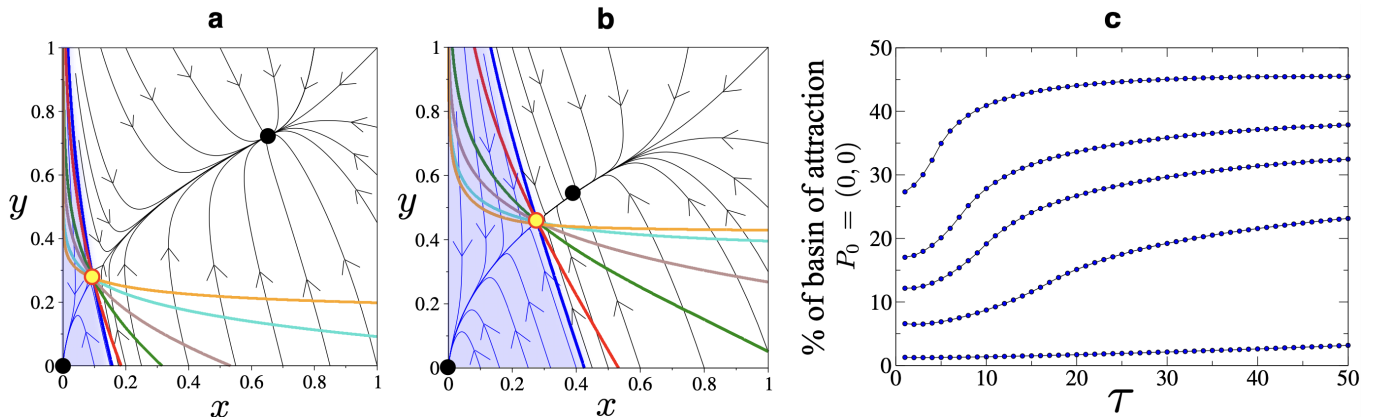


FIG. 4. Phase portraits of Eqs. (2) for $\tau = 0$ with $\varepsilon = 0.25$ (a) and $\varepsilon = 0.33$ (b). The system is bistable and trajectories converge to P_0 or P_+ depending on the initial conditions; the basin of P_0 is shown in transparent blue (black circles denote attractors; yellow circle denotes the saddle point P_-). Overlapped, we display the separatrix of the two attractors for different time lags, with: $\tau = 0$ (blue curve); $\tau = 1$ (red curve); $\tau = 5$ (green curve); $\tau = 10$ (brown curve); $\tau = 25$ (turquoise curve); and $\tau = 50$ (orange curve). (c) Basin size of P_0 versus τ for $\varepsilon = 0.15, 0.25, 0.29, 0.31$, and 0.33 (from bottom to top).

grid are shown in Fig. 4 for $\varepsilon = 0.15, 0.25, 0.29, 0.31$, and 0.33 (from bottom to top). In all cases, increasing τ rapidly enlarges the extinction basin, with the widening becoming more pronounced as $\varepsilon \rightarrow \varepsilon_c$ (see Appendix B).

IV. DISCUSSION

In this work, we investigated how explicit replication time lags affect the dynamics and persistence of replicating populations in a quasispecies-derived model with explicit polymerase dynamics. Motivated by the inherently time-dependent nature of viral replication and by experimental evidence showing that polymerase-targeting antivirals can reduce genome amplification [25], we introduced a minimal delay differential equation model coupling master and mutant defective genomes to the functional availability of the viral RNA-dependent RNA polymerase (RdRp). Unlike classical quasispecies transitions, which are typically driven by changes in mutation rates or replication-fitness asymmetries [2, 4–6, 8], our results show that extinction can emerge solely from increasing replication delays, even when the fraction of defective genomes generated and the topography of the fitness landscape remain fixed. By explicitly incorporating delayed feedback between genome abundance, RdRp production, and functional polymerase availability, the model reveals that replication timing acts as an independent dynamical control parameter capable of destabilizing persistence and driving populations toward collapse.

An important point concerns the interpretation of the model parameters in realistic viral systems. Lag-induced extinction is not controlled by τ alone, but by its magnitude relative to the characteristic timescales of amplification and loss. In this sense, genome degradation is essential for the mechanism: if genomes and polymerase were indefinitely stable, increasing τ would mainly delay

replication rather than cause extinction. Biologically, ε should therefore be interpreted as an effective loss rate including RNA and protein turnover, dilution, degradation, and, depending on the system, innate-immune-mediated clearance or RNA silencing. Consequently, the critical lag for extinction is system-dependent. Viruses with low effective decay rates would require larger delays to enter the extinction regime, whereas treatments or cellular conditions that reduce RNA/protein stability or slow RdRp synthesis, maturation, localization, or replication-complex formation would shift the system closer to the lag-induced extinction threshold.

In realistic viral systems, the delay parameter τ should be interpreted as an effective timescale combining several processes, including RdRp translation, folding, maturation, localization to replication complexes, and the switch from translation to replication of the positive-strand RNA. Under antiviral treatment, τ may also include delays caused by polymerase inhibition or drug binding. Thus, the present model is not intended to provide quantitative extinction thresholds for specific virus-drug systems, but rather to identify the dynamical regime in which lag-induced extinction can occur. In particular, extinction arises when the availability of functional polymerase is delayed beyond the timescale over which viral genomes and replication machinery remain productively available. Connecting this regime to specific viral systems would require independent estimates of RNA and protein lifetimes, RdRp synthesis and maturation times, replication-complex formation, strand-copying kinetics, and drug-binding parameters.

Thus, the model should be viewed as a mechanistic framework for organizing experimentally measurable timescales, rather than as a fully parameterized description of any single viral infection.

Several experimental studies suggest that viral extinction can be strongly influenced by externally modulating

the availability of the replication machinery. In particular, antiviral inhibitors targeting viral polymerases reduce effective replication and viral load, and can synergize with mutagenic treatments to drive RNA virus populations to extinction [25, 26]. While these works were interpreted within mutation- and fitness-based frameworks, they are also consistent with a dynamical scenario in which limiting RdRp availability introduces effective temporal constraints on replication, providing empirical motivation for lag-driven extinction mechanisms.

The lag-induced transition identified here differs fundamentally from classical error-threshold or lethal-mutagenesis mechanisms. In conventional quasispecies models, extinction thresholds arise from changes in mutation rates or effective replication fitness. In contrast, the transition studied here emerges from delayed feedback and temporal desynchronization between genome replication and RdRp availability. Mathematically, the explicit delay converts the system into a delay differential equation with memory, thereby increasing the effective dimensionality of the dynamics. Consequently, the extinction threshold cannot generally be reduced to a static renormalization of replication rates. As mentioned, the biological relevance of the lag-induced extinction mechanism depends not only on the delay parameter τ itself, but also on its relation to the characteristic amplification and loss timescales of the viral system.

Although motivated by RNA virus replication, the mechanism described here is generic to autocatalytic or cross-catalytic systems, *i.e.*, hypercycles, with lagged catalytic activity. From a dynamical-systems perspective, time lags are known to reshape basins of attraction and alter attractor selection without changing instantaneous control parameters [27–29]. In this framework, the extinction reported here corresponds to a time-lag-driven reorganization of basins, whereby increasing lags redirect trajectories toward extinction. Therefore, similar timing-driven transitions may arise in prebiotic replicators, synthetic autocatalytic networks, and other enzyme-limited evolutionary systems. Replication timing thus emerges as a fundamental and previously unrecognized control dimension of quasispecies dynamics, with implications for evolutionary theory and antiviral intervention.

Mechanisms leading to collapse in complex ecological systems can emerge from a wide range of dynamical processes, including structural instabilities, stochastic perturbations, transient dynamics, and network interactions. Within a broader context, tipping phenomena represent a particularly important class of mechanisms behind critical transitions. The most prominent mecha-

nism is bifurcation-induced tipping (B-tipping), typically associated with a saddle-node bifurcation. Other major mechanisms include rate-induced tipping (R-tipping), noise-induced tipping (N-tipping), shock-induced tipping (S-tipping), anomalous-noise tipping (A-tipping), phase-dependent tipping (P-tipping), and tipping at the end of long transients (LT-tipping) [30]. In addition to these mechanisms, stochastic bifurcation tipping (SB-tipping) [31, 32], which constitutes a distinct mechanism of N-tipping, and social tipping (SO-tipping) [33] have been recently introduced.

The extinction mechanism identified here can be interpreted within a broader framework of tipping phenomena in nonequilibrium dynamical systems, where gradual changes in control parameters induce qualitative shifts between dynamical regimes. In our case, the control parameter is the time lag τ , whose increase destabilizes persistence and redirects trajectories toward extinction through delayed feedback and temporal desynchronization. Building on this framework, we argue that lag-time-induced extinction should be regarded as a distinct tipping mechanism, even though it is not associated with a classical discontinuous bifurcation. Instead, the increase of the critical delay parameter alone is sufficient to destabilize an otherwise stable persistence state and induce a qualitative dynamical transition toward collapse. In this sense, we refer to the critical transition described here as a lag-induced, or τ -tipping, mechanism.

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APPENDIX

A. Mechanisms causing lags in genome replication

Multiple mechanisms can generate substantial time lags between genome translation and the appearance of a functional RdRp in RNA viruses. In many positive-sense RNA viruses, RdRp is produced as part of a polypro-

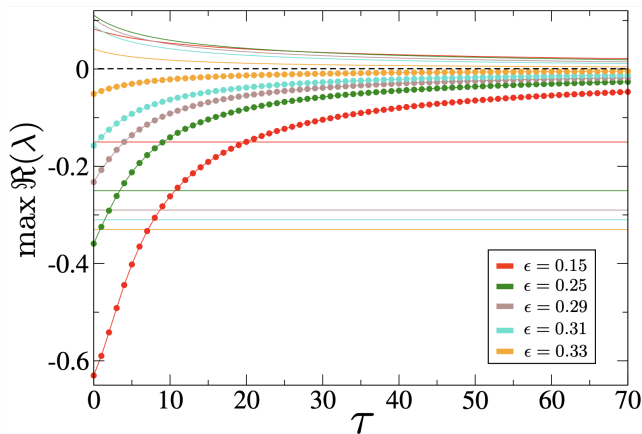


FIG. 5. Lag-dependent spectral stability of the equilibria of eqs. (2) for five values of $\varepsilon < \varepsilon_c$. Shown is the rightmost real part eigenvalue, $\max \Re(\lambda)$. The equilibrium P_0 is τ -independent and stable (horizontal lines); P_- is unstable for all τ , with its instability weakening at larger lags (positive curves); P_+ remains stable over the full delay range (dotted curves), with its attractive strength also weakening.

tein that requires co- and post-translational proteolytic processing before becoming active, as in picornaviruses [34] and flaviviruses [35]. Additional delays arise from folding kinetics and chaperone-assisted maturation [36], which are required for proper RdRp conformation and activity. The function of RdRp is often further dependent on post-translational modifications [37] and assembly into multi-protein replication complexes, frequently associated with host's proteins [38] and virus-induced membrane compartments [39], introducing spatial and temporal separation between synthesis and activity. In

several viruses, RdRp relocalization and cooperative activation introduce additional delays before effective replication can begin, even after translation is complete [39]. Finally, because the viral RNA serves simultaneously as mRNA and replication template, ribosome occupancy can transiently block RdRp access, generating an additional functional delay [34, 40]; strand switching between positive and negative RNAs further contributes to temporal separation between translation and replication [34].

B. Lag-dependent spectral stability

We numerically analyzed the lag-dependent spectral stability of P_0 , P_+ , and P_- of eqs. (2) by solving the characteristic eq. (3). For each equilibrium and each value of τ , we computed the rightmost characteristic root $\max \Re(\lambda)$, which provides a direct indicator of the local dominant spectral stability. Figure 5 summarizes the results for representative values of the degradation rate ε . The extinction equilibrium $P_0 = (0, 0)$ is characterized by a double eigenvalue $\lambda = -\varepsilon$ and therefore remains locally asymptotically stable for all τ . The persistence equilibrium P_+ remains spectrally stable for all lags, with $\max \Re(\lambda) < 0$ and decreasing attraction as τ increases. In contrast, the saddle P_- retains a positive real eigenvalue for all τ , with weakening instability at larger delays. Thus, increasing τ induces no local bifurcations of P_0 or P_+ for the parameter values explored here; instead, it continuously deforms the stable manifold $W^s(P_-; \tau)$ forming the basin boundary. Temporal lags therefore reorganize basins of attraction without changing the fixed-point structure, providing the mechanism for lag-induced extinction identified in this work.