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Quantifying the capacity for contemporary trait changes to drive intermittent predator–prey cycles

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Abstract

A large and growing body of theory has demonstrated how the presence of trait variation in prey or predator populations may affect the amplitude and phase of predator–prey cycles. Less attention has been given to so-called intermittent cycles, in which predator–prey oscillations recurrently disappear and re-appear, despite such dynamics being observed in empirical systems and modeling studies. A comprehensive understanding of the conditions under which trait changes may drive intermittent predator–prey dynamics, as well as their potential ecological implications, is therefore missing. Here we provide a first systematic analysis of the eco-evolutionary conditions that may give rise to intermittent predator–prey cycles, investigating 16 models that incorporate different types of trait variation within prey, predators, or both. Our results show that intermittent dynamics often arise through predator–prey coevolution, but only very rarely when only one trophic level can adapt. Additionally, the frequency of intermittent cycles depends on the source of trait variation (genetic variation or phenotypic plasticity) and the genetic architecture (Mendelian or quantitative traits), with intermittency occurring most commonly through Mendelian evolution, and very rarely through phenotypic plasticity. Further analysis identified three necessary conditions for when trait variation can drive intermittent cycles. First, the intrinsic stability of the predator–prey system must depend on the traits of prey, predators, or both. Second, there must be a mechanism causing the recurrent alternation between stable and unstable states, leading to a "trait" cycle superimposed on the population dynamics. Finally, these trait dynamics must be significantly slower than the predator–prey cycles. We show how these conditions explain all the abovementioned patterns. We further show an important unexpected consequence of these necessary conditions: they are most easily met when intraspecific trait variation is at high risk of being lost. As trait diversity is positively associated with ecosystem functioning, this can have potentially severe negative consequences. This novel result highlights the importance of identifying and understanding intermittent cycles in theoretical studies and natural

systems. The new approach for detecting and quantifying intermittency we develop here will be instrumental in enabling future study.

KEYWORDS
eco-evolutionary feedbacks, ecosystem functioning, intraspecific trait variation, population cycles, predator–prey dynamics, trait dynamics

INTRODUCTION

Predator–prey interactions form the fundamental components of natural food webs, and understanding how their interaction affects the population dynamics of both species has been the subject of extensive theoretical and empirical study. One of the most classic results in predator–prey dynamics is the emergence of ongoing population cycles in predator and prey abundances (Elton & Nicholson, 1942; Lotka, 1925; Volterra, 1928). The mechanisms determining the presence (or absence) and amplitude of these predator–prey cycles are very well understood theoretically (Rosenzweig & MacArthur, 1963; see Box 1) and have been confirmed experimentally (Blasius et al., 2020; Fussmann et al., 2000). Ecological theory predicts the emergence of regular cycles with a constant amplitude, and a constant phase lag (i.e., how long the peak in predator abundance is delayed compared with the peak in prey abundance) of a quarter of the cycle period (Bulmer, 1976).

Real predator–prey cycles observed in the field or in experimental settings clearly do not always strictly resemble these theoretical predictions. Even under constant environmental conditions, they are generally not as regular as theory would predict, and may show variability in the amplitude, phase lag, or both (Blasius et al., 2020). Some degree of deviation can simply be the result of stochastic variability; the presence of strong deviations from theoretical predictions, however, may indicate the presence of processes present in real systems that are not accounted for in classic theoretical models, such as more prey or pathogens (Raatz et al., 2018; Singh et al., 2004).

In recent years, intraspecific trait variation and consequent trait adaptation has received much attention as a potential explanation for striking divergences from regular predator–prey cycles. Particular focus has been on traits that mediate the strength of the predator–prey interaction: defense traits in prey (Becks et al., 2010; Yoshida et al., 2003, 2007), counterdefense (or "offense") traits in predators (Cortez & Patel, 2017), or both together (Cortez, 2018; Coutinho et al., 2016; Frickel et al., 2016; Mougi, 2012; van Velzen & Gaedke, 2017). Such traits tend to be major determinants of fitness and are therefore typically under strong selective pressure; as a consequence, they provide some of the most striking documented examples of rapid evolution (Frickel et al., 2016; Hairston et al., 1999; Yoshida et al., 2003). The direction and strength of selection of these traits depends on the current predator and prey abundances, and changes in these traits can affect those population abundances in return, resulting in a mutual eco-evolutionary feedback between trait and population dynamics (Fussmann et al., 2007). Such eco-evolutionary feedbacks can leave strong signatures in the resulting predator–prey dynamics, which may be used as a "smoking gun" to infer the presence of rapid evolution (Cortez, 2018; Hiltunen et al., 2014). Examples include cryptic predator–prey cycles, in which the predator exhibits large-amplitude cycles while the prey population remains nearly constant (Yoshida et al., 2007), antiphase cycles in which the predator peaks follow prey peaks with a delay of half the cycle period instead of a quarter (Becks et al., 2012; Yoshida et al., 2003), and reversed cycles in which predator peaks precede prey peaks, rather than vice versa (Cortez & Weitz, 2014; van Velzen & Gaedke, 2018). A prerequisite for these patterns is that adaptation must be fast enough that it is roughly on the same timescale as the predator–prey population dynamics.

The examples cited above all involve predator–prey dynamics that are distinctly different from what purely ecological theory would predict; however, they are all still "regular" cycles in the sense that they have a fixed period length and amplitude. This contrasts with a type of predator–prey dynamics that has received very limited theoretical attention: intermittent cycles, which are characterized by "interruptions" in which predator–prey cycles are strongly dampened or disappear entirely, after which they re-establish themselves (Figure 1). Such dynamics have been found in the field (Figure 1a–c; Ecke et al., 2017, Krebs et al., 2013, Wegge & Rolstad, 2018), but also in highly controlled chemostat experiments with fixed environmental conditions (Figure 1d–f; Blasius et al., 2020, McCauley et al., 1999, Wei et al., 2011). These dynamics are striking because they appear to indicate recurrent switching between the presence and absence of predator–prey cycles, whereas established ecological theory predicts that predator–prey cycles are either present or absent (see Box 1).

BOX 1 Theory behind stable and unstable predator–prey dynamics

The presence or absence of ongoing cycles in population in a predator–prey system can be explained by the dominant type of self-regulation in the two interacting species. Negative self-regulation, in which an increase in the population of one species decreases its own net per capita growth rate, has a stabilizing effect on the dynamics. Conversely, positive self-regulation, in which an increase in the population increases its own growth rate, is destabilizing. The balance of stabilizing and destabilizing effects present in the system determines both whether ongoing cycles emerge and their amplitude.

In the most basic predator–prey model, the Lotka–Volterra predator–prey equations (Lotka, 1925; Volterra, 1928), no stabilizing or destabilizing effects are present at all; consequently, this model exhibits neutral cycles that are neither dampened nor amplified over time. However, the combination of exponential growth and a type I functional response makes this model highly unrealistic. The simplest realistic model that is commonly used is the Rosenzweig–MacArthur model (Rosenzweig & MacArthur, 1963):

$$\begin{aligned}\frac{dN}{dt} &= \left(r\left(1-\frac{N}{K}\right)-\frac{aP}{1+ahN}\right)N\\ \frac{dP}{dt} &= \left(e\frac{aN}{1+ahN}-d\right)P\end{aligned} \tag{B1.1}$$

Here, the prey (N) grow logistically without predators, with an intrinsic growth rate r and carrying capacity K. Predation follows a Holling type II functional response (Holling, 1965) with attack rate a and handling time h. Finally, the predator (P) has a conversion efficiency e with which captured prey are converted into predator biomass, and a constant mortality rate d.

In Equation (B1.1), the equation for prey dynamics contains both a negative and a positive self-regulation term; the predator equation still contains neither, as its per capita growth rate is not dependent on its own abundance. Stability therefore depends entirely on the dominant type of self-regulation in the prey (see also Appendix S4: Section S4). Logistic growth is the negative self-regulation term: as prey abundance increases toward the carrying capacity it slows its own growth down. The strength of this negative self-regulation, and the resulting stabilizing effect, is determined by the carrying capacity K: the lower the K value, the stronger the stabilizing effect.

The positive self-regulation is contained within the type II functional response. The total predation rate increases with prey abundance, but the per capita predation rate actually decreases. Therefore, an increase in prey abundance gives rise to a "safety in numbers" effect, lowering mortality and resulting in a positive effect on itself. This positive self-regulation has the potential to destabilize the dynamics and result in predator–prey cycles. Here, the strength of the destabilization is determined by the curvature of the functional response, with a stronger curvature (higher a and h) being more strongly destabilizing.

The relative strengths of these stabilizing and destabilizing effects are determined by the parameter values for K, a, h, e, and d (with r being the only parameter that has no effect at all), and this determines the dynamic behavior of the predator–prey system. If the stabilizing effect outweighs the destabilizing effect, the system will converge to a stable equilibrium. If the destabilizing effect is stronger, ongoing predator–prey cycles will emerge, with an amplitude determined by how much stronger is the destabilizing effect.

Obviously, the components in Equation (B1.1) can be modified in many ways, some of which will cause new stabilizing or destabilizing effects to appear. For example, a density-dependent mortality rate for the predator has a stabilizing effect on the dynamics, as it constitutes an additional form of negative self-regulation. But regardless of how the baseline equations are extended and modified, the theory behind stability always remains the same: it depends on the relative strengths of all stabilizing and destabilizing effects present in the system.

The interpretation of observed intermittent patterns in predator–prey cycles depends on how pronounced are the interruptions. Brief interruptions, after which cycles re-establish themselves almost immediately, are generally (and probably correctly) attributed to stochastic effects (Blasius et al., 2020). When cycles are interrupted over a

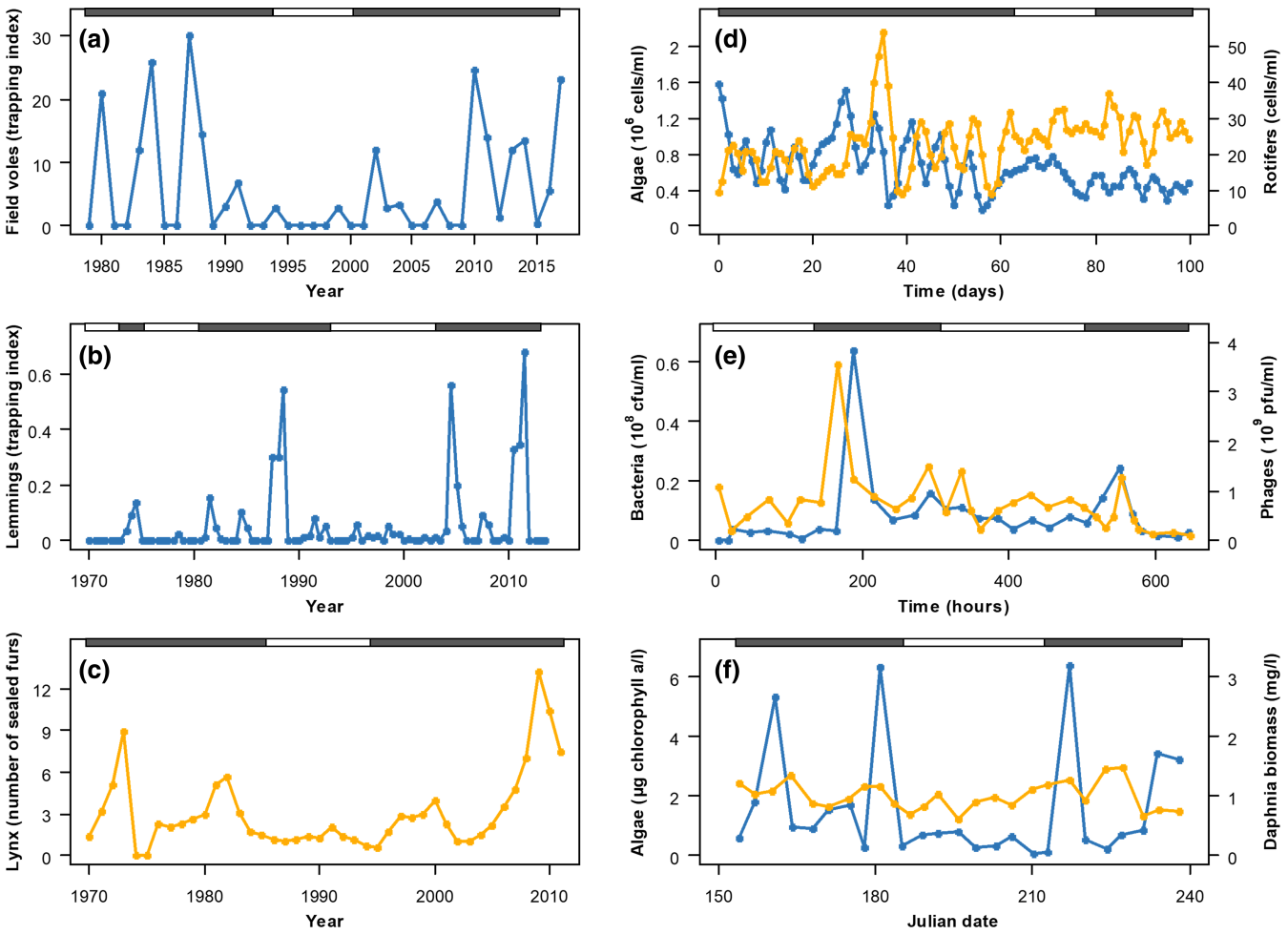

FIGURE 1 Examples of intermittent population dynamics observed in empirical systems. Prey abundances are shown in blue, predator abundances in orange. Bars above the time series indicate the presence (filled rectangles) or absence/near absence (open rectangles) of population cycles. (a) Abundance of field voles in southeast Norway (data from Wegge & Rolstad, 2018; figure 1a). (b) Abundance of lemmings in northern Sweden (data from Ecke et al., 2017; figure 1d). (c) Lynx abundance in Alaska (data from Krebs et al., 2013; figure 2). Although only the prey are pictured in (a) and (b) and only the predators in (c), population cycles in these three species are widely considered to be caused by predator–prey interactions (Oli, 2019; Elton & Nicholson, 1942). (d) Experimental predator–prey dynamics with an algal prey and rotifer predators (data from Blasius et al., 2020, experiment C4). (e) Experimental eco-evolutionary predator–prey dynamics of *Vibrio cholerae* and its bacteriophage (data from Wei et al., 2011; figure 3c). Note that the first major predator peak precedes the prey peak ($t \approx 170$); this is an example of reversed predator–prey cycles driven by bacteria–phage coevolution (van Velzen & Gaedke, 2018). (f) Experimental predator–prey dynamics with algal prey and *Daphnia pulex* as predators (data from McCauley et al., 1999; figure 1d). Note that predator population cycles appear disconnected from those in the prey; this is an example of cryptic cycles (Yoshida et al., 2007), in this case driven by stage structure in *Daphnia* (McCauley et al., 1999). But what causes the population cycles to disappear and re-appear in the algal prey is not well understood

longer period of time, however, a mechanistic explanation is required. One possibility is a combination of stochasticity and bistability: the predator–prey system has two attractors, a stable equilibrium and a limit cycle, and switches between them at random intervals as a result of stochasticity (Ives et al., 2008; Figure 2a). A second possibility is that some aspect of the environment is changing, thereby changing the stability of predator–prey dynamics. For example, environmental changes in temperature or light availability can certainly affect the stability of predator–prey dynamics and, when these changes happen recurrently, they can drive intermittent cycles (Scheffer et al., 1997, Nelson et al., 2013; Figure 2b), for example in a seasonal pattern in which cycles appear in summer and disappear in winter. The remaining question is whether such proposed mechanisms may explain observations of intermittent cycles in natural systems. Long-term dampening of the three-year population cycles of small rodents in Europe started in the 1990s, and has received much attention in studies attempting to link the disappearance of pronounced cycles to climate change (Cornulier et al., 2013; Ims

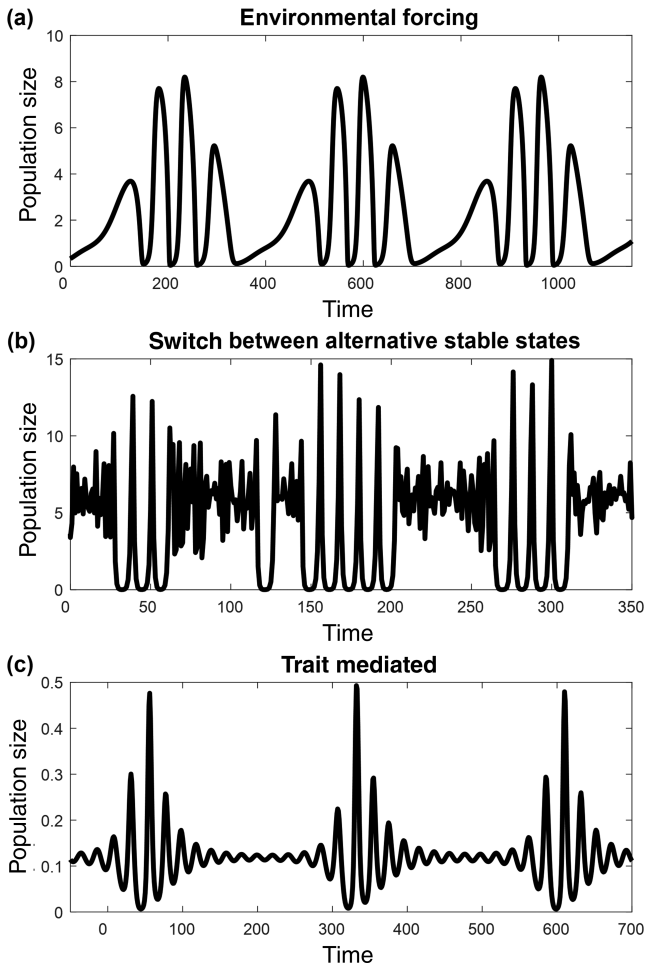

FIGURE 2 Mechanisms that may drive intermittency in predator–prey cycles; dynamics in (a, b) were generated by simulating published models, whereas those in (c) were generated by one of our own models (V-M; see Figure 3a). (a) Environmental noise causes switching between a stable equilibrium and a large-amplitude limit cycle in a bistable system (Ives et al., 2008; figure 2b, with adjusted levels of noise). (b) Seasonal forcing causes shifts between cyclic and stable dynamics (Scheffer et al., 1997; figure 9d). (c) Temporal variation in a predator trait causes switching between stable and unstable predator–prey dynamics (this paper, prey dynamics in Figure 7a)

et al., 2008). However, although changes in global temperature appeared to explain the dampening of the vole population cycles in the past, they cannot explain the re-establishment of high-amplitude population cycles in the more recent vole dynamics (Brommer et al., 2010; Wegge & Rolstad, 2018), because global warming has not reversed direction. Although this does not necessarily rule out environmental forcing as an explanation, it does raises the question of what alternative mechanism may underlie intermittent cycles.

As defense and offense traits can affect the stability of predator–prey dynamics (Cortez, 2018), it stands to reason that they may also drive intermittent cycles. Yet this possibility has not received much attention from theoretical ecologists (but see Bengfort et al., 2017, Yamamichi et al., 2011), and is generally not considered as an explanation for empirically observed intermittent dynamics at all. It seems clear to us that these two patterns are connected: the lack of focused theoretical study means that eco-evolutionary feedbacks are not on the radar of empirical ecologists as a potential explanation for intermittent cycles; conversely, as empiricists rely on stochasticity or environmental forcing as an explanation, intermittent cycles are generally not on the radar of theorists as something that merits investigation. An additional difficulty is the fact that detecting intermittent cycles requires a longer time series than detecting for example antiphase or cryptic cycles, which presents no difficulty for theoretical work but may hinder empirical study.

Despite the lack of focused theoretical study, intermittent cycles have been observed in numerous predator–prey models under a seemingly wide variety of scenarios (Abrams et al., 2003; Abrams & Shen, 1989; Coutinho et al., 2016; Klauschies & Gaedke, 2020; Mougi, 2012; Mougi & Nishimura, 2007; van Velzen & Gaedke, 2017; Yamamichi et al., 2011). These studies mostly involve rapid trait changes, indicating that eco-evolutionary feedbacks can indeed be a driver of intermittent cycles. Some of the abovementioned models incorporate trait variation as two discrete genotypes or phenotypes (i.e., "Mendelian" traits) within the prey (Yamamichi et al., 2011), predators (Klauschies & Gaedke, 2020), or both (Abrams & Shen, 1989; Mougi, 2012; Mougi & Nishimura, 2007), in which evolution refers to the shift in genotype frequencies as selection favors one over the other. Others assume a continuous (i.e., "quantitative") trait evolving in the direction of higher fitness (Coutinho et al., 2016; van Velzen & Gaedke, 2017). Finally, some studies have investigated phenotypically plastic traits (Mougi, 2012) or a combination of evolution and phenotypic plasticity (Yamamichi et al., 2011). Therefore, we have no shortage of potential explanations that involve trait changes interacting with predator–prey population dynamics; the difficulty is not in proving that trait changes can be responsible for generating intermittent cycles, but in gaining a comprehensive picture from the above set of disparate and potentially idiosyncratic results.

Our aim here is to provide a first systematic theoretical investigation of the potential of trait variation, and consequent trait adaptation, to cause intermittent cycles in simple predator–prey systems. We investigated a total of 16 predator–prey models with trait adaptation on different trophic levels (in the prey, predators, or both), different types of trait variation (Mendelian or quantitative

traits) and different mechanisms for adaptation (evolution or phenotypic plasticity), to quantify which of these scenarios, if any, are likely to result in intermittent predator–prey cycles. As there is no established method for detecting the presence of intermittent cycles in predator–prey dynamics, we developed a new approach for detecting intermittency, combining Fourier analysis with a moving window approach. We further developed a quantitative measure, the "intermittency index" I, to determine not only the presence but also the degree of intermittency.

Our analysis shows that intermittent cycles arise at a substantial frequency in eight of our 16 predator–prey models. It further reveals some very clear patterns: intermittent cycles were most frequently caused by adaptation on both trophic levels (as opposed to adaptation only in the prey or only in the predator); by adaptation through Mendelian rather than quantitative traits; and by adaptation through evolution rather than phenotypic plasticity. Further analysis revealed that all these patterns arose from the same necessary condition: trait adaptation must occur on a significantly slower timescale than the ecological predator–prey cycles. This requirement sets intermittent cycles apart from other signatures of rapid adaptation, such as cryptic cycles or antiphase cycles, which all require that adaptation must occur on the same timescale as the predator–prey cycles.

Perhaps most importantly, in all models with Mendelian evolution exhibiting intermittent cycles, we found a strong link between intermittency and fragility of trait variation: intermittent cycles occur in parameter ranges in which at least one of the genotypes is at high risk of extinction. Therefore, when intermittent cycles are driven by trait dynamics, their presence may indicate that the currently present trait variation is at risk of being lost.

BACKGROUND: THE ORIGINS OF PREDATOR–PREY CYCLES AND INTERMITTENT CYCLES

The term "intermittent cycles" describes a type of predator–prey dynamics that shows temporal variability in stability, that is, recurrent switching between dampening and amplification of predator–prey cycles. To understand what mechanisms might cause such dynamics, it is helpful to start with classic ecological theory explaining what causes the presence or absence of predator–prey cycles (Box 1). The short version is that various types of self-regulation are typically present in a predator–prey interaction, with either a stabilizing or a destabilizing effect on the dynamics. When stabilizing effects outweigh destabilizing ones, the oscillations will become dampened over time and converge to a stable equilibrium; conversely, when destabilizing effects outweigh stabilizing ones, small oscillations tend to become amplified over time until they reach a stable limit cycle (Box 1). If populations fall to a very low abundance during their limit cycle, demographic stochasticity may result in extinction ("paradox of enrichment"; Rosenzweig, 1971).

This straightforward theory therefore explains under what conditions the long-term behavior of a simple predator–prey system is likely to be characterized by the presence or absence of ongoing population cycles: for any given combination of parameter values (see Box 1), theory predicts either a stable equilibrium, or a stable limit cycle with a fixed amplitude. This prediction should generally hold true if the parameter values of the system, and therefore the strengths of stabilizing and destabilizing effects, are constant. If one or several relevant parameters of the system are not constant, however, the result is temporal variability in their stabilizing and/or destabilizing effects. This temporal variability may be a result of environmental forcing, but it may also arise as a result of trait dynamics, for example when changes in prey defense cause changes in the predator's attack rate. If this variability is strong enough, the system may switch between stable and unstable dynamics, that is, intermittent cycles.

From the above, we can deduce two conditions that are necessary for intermittent cycles to arise:

1. The temporal variability in parameter(s) must be strong enough for stabilizing and destabilizing effects to switch dominance, resulting in the recurrent dampening and amplification of population oscillations.
2. This switching in dominance must occur on a timescale that is significantly slower than the "ecological" predator–prey cycles. Intermittent cycles can only occur through this mechanism if there is time for several predator–prey cycles to occur before they are dampened.

Put together, intermittent cycles may be defined as the superimposition of two cyclic dynamics: a fast "ecological" cycle that results from the predator–prey interaction, and a slower cycle that determines the amplitude of these population cycles. Our focus here is on the role of intrinsic feedbacks within the predator–prey system in generating this slow "amplitude" cycle, and thereby intermittent cycles. With "feedbacks" we mean specifically feedbacks between trait changes and changes in species abundances, caused by trait variation (and consequent trait adaptation) in one or both trophic levels.

Most of the scenarios we investigate involve traits that have been previously studied in the context of eco-evolutionary feedbacks in predator–prey systems, and affect parameters that have a major effect on the stability of the dynamics (attack rate, handling time, and predator mortality rate). These comprise scenarios I, III, V, and VI described in the "*Models investigated*" section below. The last two scenarios, II and IV, are generalized versions of the inducible defense model studied by Yamamichi et al. (2011) and the inducible offense model studied by Yamamichi and Letten (2021), respectively, which showed that temporal variability in the induction rate could result in intermittent cycles.

Our first goal was to investigate which of these scenarios could generate intermittent cycles, and to quantify how likely they are to occur for each scenario. Our second goal, after these scenarios had been identified, was to investigate in more detail exactly when and how intermittent cycles were generated by these scenarios, and what set them apart from scenarios that did not cause intermittent cycles.

METHODS

Models investigated

We studied 16 predator–prey models comprising seven different trait variation scenarios (see Figure 3a): two scenarios with trait variation in the prey (scenarios I and II), three with trait variation in the predators (scenarios III, IV, and V), and two with trait variation in both (scenarios VIa and VIb). In addition to the across-scenario comparison, we aimed to clarify the effect of the type of trait (Mendelian or quantitative) and the mechanism for adaptation (evolution or phenotypic plasticity). Previous studies have used three different approaches for modeling trait adaptation (see Figure 3b, and more detailed descriptions in Box 2):

Mendelian-trait evolution (M) corresponds to the co-occurrence of two genotypes with different trait values in the population, whose frequencies change in response to selection (Box 2, Equation B2.1; Yoshida et al., 2003). This trait variation is the type used in the scenario descriptions below.

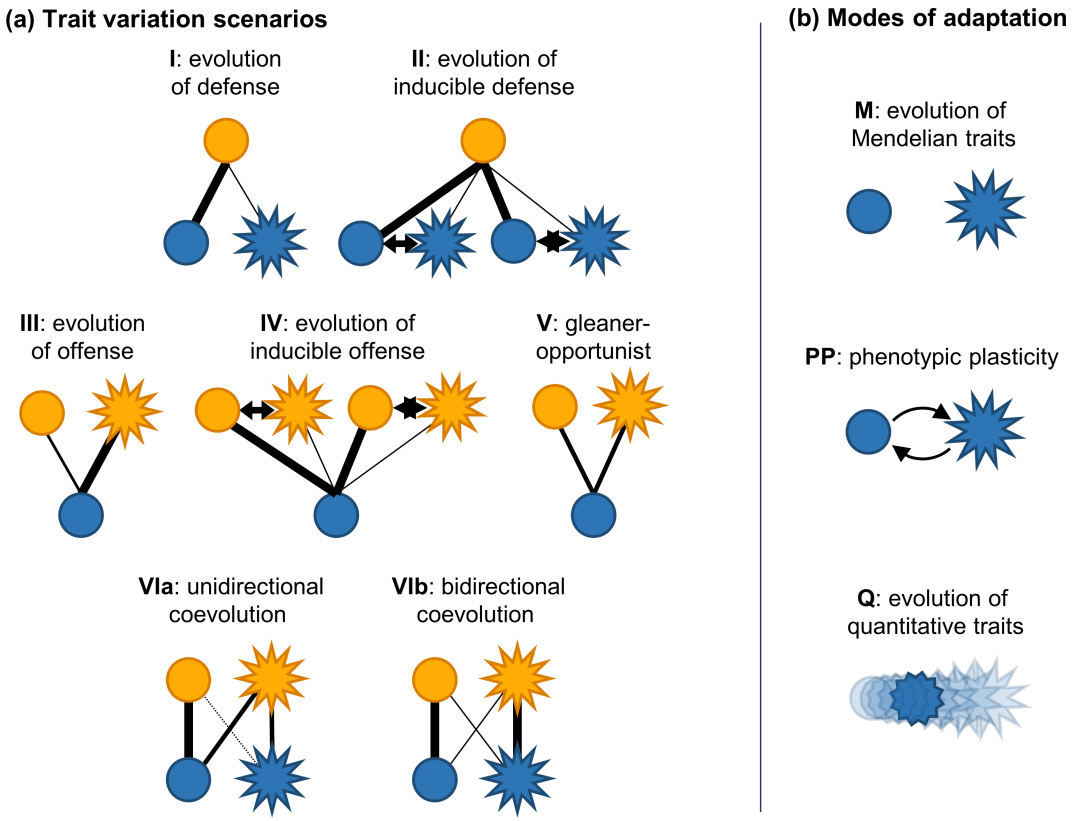

FIGURE 3 (a, b) Schematic representation of the seven adaptive scenarios and the three modes of adaptation investigated. In (a), pictures represent the models with Mendelian-trait evolution; prey genotypes are pictured in blue, and predator genotypes in orange. Solid lines indicate feeding links, with the thickness of the lines reflecting their strength (i.e., the predation rate). In scenarios II and IV, the arrows represent phenotypically plastic switching between phenotypes expressed by the same genotype, with the thickness of the arrows representing the speed of switching between two phenotypes expressed by the same genotype (i.e., the speed of phenotypic plasticity). Equations for all models are given in Appendix S1

BOX 2 Approaches to modeling trait changes

Trait dynamics can be represented mathematically in different ways, depending on assumptions on the type of trait (discrete vs. continuous) and the mechanism for trait changes (evolution vs. phenotypic plasticity). The way trait dynamics are modeled can affect the speed of trait changes, which may in turn affect the stability of predator–prey dynamics; for example, rapid inducible defenses have been found to stabilize predator–prey cycles more strongly than the rapid evolution of defense (Cortez, 2011; Yamamichi et al., 2011). To investigate how the mode of adaptation may affect the likelihood of intermittent cycles, we compared three commonly used approaches for representing trait changes, which we describe here.

The three main modes of adaptation

Evolution of Mendelian (discrete) traits (M)

This approach represents changes in a trait that takes two discrete values, for example when prey can be undefended or defended, but not partially defended. Such traits are typically determined by a single gene (locus), for which the expression of two different alleles give rise to two phenotypes with different trait values. The dynamics of the prey and/or predator population are here modeled by separate equations representing each genotype:

$$\begin{aligned}\frac{dN_i}{dt} &= F_{N_i}\left(\vec{N},\vec{P}\right)N_i \\ \frac{dP_i}{dt} &= F_{P_i}\left(\vec{N},\vec{P}\right)P_i\end{aligned} \tag{B2.1}$$

where N_i and P_i represent the abundances of the prey and predator genotypes, respectively. In models in which trait variation is present on only one trophic level (I-M–V-M), the other trophic level is represented by a single equation (see Appendix S1 for details). F_{N_i} and F_{P_i} represent the Malthusian fitness (i.e., the per capita net growth rates) of genotypes N_i and P_i, respectively (cf. Equation B1.1 in Box 1). The fitness functions generally depend on the abundances of all prey and predator genotypes, as indicated by the vectors $\vec{N}$ and $\vec{P}$. Trait changes occur through shifts in the frequencies of the two genotypes in the total population, and depend on the fitness difference between them. The above equations assume perfect inheritance of traits that arise from asexual reproduction; they are therefore commonly used to describe selection on predation-related traits in asexually reproducing species such as phytoplankton or zooplankton (e.g., Becks et al., 2010; Yoshida et al., 2003).

Phenotypic plasticity (PP)

This approach represents populations in which individuals can express two distinct phenotypes, depending on current conditions. In predator–prey systems, this is most commonly applied to inducible defenses (Cortez, 2011; Vos et al., 2004; Yamamichi et al., 2019), but it can also represent inducible predator traits (Mougi, 2012), and can be implemented by modifying the components in Equation (B2.1):

$$\begin{aligned}\frac{dN_i}{dt} &= F_{N_i}\left(\vec{N},\vec{P}\right)N_i - \chi^N_{ij}N_i + \chi^N_{ji}N_j \quad (i \neq j) \\ \frac{dP_i}{dt} &= F_{P_i}\left(\vec{N},\vec{P}\right)P_i - \chi^P_{ij}P_i + \chi^P_{ji}P_j \quad (i \neq j)\end{aligned} \tag{B2.2}$$

χ^y_x are switching functions (Yamamichi et al., 2019) and represent the probability that an individual currently expressing one phenotype will switch to the other one. We typically assume here that these switching

rates are a function of the fitness difference between the two phenotypes, so individuals are more likely to switch from the low-fitness to the high-fitness phenotype ("hybrid model" in Yamamichi et al., 2019; see Appendix S1 for details). Trait changes driven by switching therefore amplify those already occurring through selection, making trait dynamics overall more rapid than in the "M" models. The maxima of the switching rates are given by the parameter $\chi_{\max}$, which therefore determines the speed of trait changes. Models in which trait variation is present on only one trophic level (I-PP, III-PP and V-PP) again represent the other trophic level as a single equation, so the switching rates do not apply there.

Evolution of quantitative (continuous) traits (Q)

This approach applies to quantitative traits, which are determined by the action of many genes, for example body size or speed of locomotion. Individuals within the population will all have slightly different trait values, making up the trait distribution of the population. The trait dynamics follows changes in the population average of the trait, caused by fitness differences between individuals with different trait values. The combined biomass and trait dynamics are represented as follows:

$$\begin{aligned}\frac{dN}{dt} &= F_N(N,P,u,v)N \\ \frac{dP}{dt} &= F_P(N,P,u,v)P \\ \frac{du}{dt} &= G_N \cdot V_N \cdot \left.\frac{\partial F_N}{\partial \hat{u}}\right|_{\hat{u}=u} \\ \frac{dv}{dt} &= G_P \cdot V_P \cdot \left.\frac{\partial F_P}{\partial \hat{v}}\right|_{\hat{v}=v}\end{aligned} \tag{B2.3}$$

where the first two equations describe the change in the population sizes, and the last two equations the change in the average trait values (u for the prey trait, v for the predator trait). The speed of trait changes is determined by three factors: the heritability of the trait (G_N and G_P), the amount of additive genetic variation (V_N and V_P), and the fitness gradient or selection gradient (the last term in each equation). In the fitness gradient, $\hat{u}$ and $\hat{v}$ denote the trait values of an individual, whereas u and v represent the population average values. In models in which trait variation is present on one trophic level (I-Q, III-Q, and V-Q), the parameter G_x for the other trophic level is set to zero.

This approach assumes that the trait distribution is symmetric and unimodal, and remains so under the action of selection (Abrams, 2001). This was first shown to hold under random mating, weak selection, and many loci having additive effects on the trait (Lande 1976, Felsenstein 1979); later it was shown that it also holds under strong selection (Turelli and Barton 1994). This approach has been widely used for modeling the contemporary evolution of quantitative traits (Abrams, 2001; Cortez, 2018; Mougi, 2010; Schreiber et al., 2011; Vasseur et al., 2011).

Evolution of phenotypic plasticity: Combining two approaches

Two scenarios (II and IV) can be viewed as a combination of two of the above approaches ("M" and "PP"). These models describe evolutionary change in the speed of phenotypic plasticity, which is modeled using the Mendelian approach (Equation B2.1): one genotype is fast-switching whereas the other is slow-switching, and their frequencies change according to their fitness differences. Trait changes also occur within each genotype, as individuals can switch between two phenotypes; these changes are modeled using the "PP" approach (Equation B2.2). See Appendix S1: Equations (S4) and (S8) for the full equations of these models (II-M and IV-M).

Phenotypic plasticity (PP) corresponds to a population with two discrete phenotypes, in which individuals have the ability to switch between them based on a cue (e.g., predator density for inducible defenses) or based on the relative fitness of the two phenotypes, which is more likely to be adaptive (Yamamichi et al., 2019). This allows the population to respond rapidly to changing conditions (Box 2, Equation B2.2).

Quantitative trait evolution (Q) corresponds to the presence of a continuous, quantitative trait in the population. The average population trait shifts in response to selection, with the direction and speed of this shift determined by the fitness gradient (Box 2, Equation B2.3; Abrams, 2001).

To determine how the type of trait variation and adaptation affects the likelihood for intermittent cycles, we investigated all three models for the seven scenarios, whenever this was possible. For three of the scenarios (II, IV, and V), the quantitative trait evolution model was not feasible (see Appendix S1 for details); additionally, the PP model made no ecological sense for scenarios II and IV. Therefore, investigation of these scenarios was limited to the "M" model (for scenarios II and IV) and the "M" and "PP" models (for scenario V). For the remaining four scenarios all three models were used, bringing the total number of models we investigated to 16. Mathematical details of all models, as well as the investigated parameter ranges, can be found in the Supplementary Material, Appendix S1.

Scenario I: Defense traits (prey)

This scenario describes variability in a prey trait that determines its vulnerability to predation. This can cover a wide range of defense traits such as increased running or swimming speed to escape predation, investment into defensive armor, or changes in size or shape that allow prey to escape gape-limited predators (Becks et al., 2010; Bro-Jørgensen, 2013; Scharf et al., 2000). A trade-off between defense and growth is assumed, with defended genotypes growing and/or reproducing more slowly than undefended ones. This trade-off has been shown to result in an eco-evolutionary feedback between predator and defense dynamics (Becks et al., 2010; Yoshida et al., 2003), as defended genotypes are selectively favored when predation pressure is high, and disfavored when predation pressure is low. Therefore, when the predators are at high densities, the average level of defense in the prey population increases; this causes predator densities to fall, resulting in turn in a decline in defense, after which the cycle starts again. This feedback can cause strong variability in the predator's attack rate, which is high when the prey population is dominated by undefended genotypes, and low when the prey population is dominated by defended ones.

Scenario II: Evolution of inducible defenses (prey)

This scenario is a modified version of the model studied by Yamamichi et al. (2011), for which they reported intermittent cycles. The original model consisted of a phenotypically plastic prey, with inducible defenses enabling switching between undefended and defended phenotypes based on predator density, and a non-plastic prey with two genotypes, also undefended and defended. The plastic prey was assumed to have reduced growth rates for both phenotypes, compared with those of the non-plastic prey. This model resulted in a direct feedback between ecological dynamics (i.e., the presence or absence of cycles) and selection on the prey. When predator–prey cycles occurred, the plastic prey had a strong advantage over the non-plastic one, as it was able to react rapidly to changes in predation pressure. However, inducible defenses are strongly stabilizing, and so a dominance of phenotypically plastic prey dampened the predator–prey cycles. This removed the advantage of the plastic prey, allowing the non-plastic prey to increase; but as it rose to dominance it induced predator–prey cycles, in turn giving the selective advantage back to the plastic prey (Yamamichi et al., 2011).

Here we studied a slightly modified version of this model: rather than assuming a strict division between a plastic and a non-plastic prey, we assumed that both prey have some degree of plasticity, but that they differ strongly in their induction speeds. Therefore, prey are subdivided into "fast-switching" and "slow-switching"; "slow-switching" in principle also includes a switching rate of zero, making this scenario a more general version of the previously published model. As in Yamamichi et al. (2011) we assumed that the fast-switching prey had a lower growth rate as a result of the cost of plasticity.

Scenario III: Offense traits (predators)

Scenario III is the mirror image of scenario I: here it is the predator population that has variability in a trait determining predation success. Predators with an "offensive" strategy have a higher attack rate than those with a "non-offensive" or "normal" strategy, for example as a result of higher activity levels or faster running speed (Bro-Jørgensen, 2013; Kiørboe, 2011). This increase in predation success is assumed to come at the cost of an

increased mortality rate, for example as a result of high activity levels increasing its risk to be captured by its own predators or increasing its energy expenditure (Kiørboe, 2011). The dynamics of such an offense trait can interact with prey dynamics, in exactly the same way that defense dynamics interact with those of the predators: low-offense strategies are selectively favored when prey are rare or very abundant, and disfavored for intermediate prey abundance (see Supplementary Material, Appendix S1 for a detailed explanation).

Scenario IV: Evolution of inducible offenses (predators)

Scenario IV is the mirror image of scenario II, applied to inducible offenses rather than inducible defenses. A version of this scenario was shown by Yamamichi and Letten (2021) to produce intermittent cycles, by the same mechanism as in scenario II: a non-plastic predator destabilized the system, thereby giving the advantage to a phenotypically plastic predator, which in turn stabilized the dynamics and allowed the non-plastic predator to increase again. As in scenario II, we investigated a more general version of this scenario, consisting of two plastic predators that differed in their switching rate between "offensive" and "non-offensive" phenotypes. We assumed the "fast-switching" predator carries a cost for its higher level of plasticity, which is modeled as an increased mortality rate for both of its phenotypes (cf. scenario II).

Scenario V. Gleaner–opportunist trade-off (predators)

This scenario is related to, but certainly not identical with, scenario III: in this case also, predators differ in their attack rates; but here, a higher attack rate comes at the cost of a longer handling time. This results in a trade-off: the predator with a high attack rate ("gleaner") has the advantage when prey are scarce, whereas the other predator has the advantage when prey are abundant ("opportunist"). A second consequence of this trade-off is that the opportunist tends to have a competitive advantage over the gleaner when the prey population exhibits cycles, whereas the reverse is true when the prey population is constant. At the same time, the opportunist tends to stabilize the predator–prey dynamics, whereas the gleaner tends to induce predator–prey cycles; therefore, each predator modifies the prey dynamics in such a way that its competitor gains the advantage (cf. scenario II). This can enable coexistence between the predators (Armstrong & McGehee, 1980) and, more relevantly in the context of this study, drive intermittent cycles (Abrams et al., 2003; Klauschies & Gaedke, 2020).

Scenario VI: Defense and offense traits (both)

Here, prey defense traits and predator offense traits together determine how successful the predator is at capturing this prey. In addition to the feedbacks between trait and population dynamics described above for scenarios I and III, there is now also the potential for feedbacks between the dynamics of prey and predator traits: whether defense is selected for or against depends on the current offense strategy of the predator, and vice versa.

This scenario can be subdivided into two subscenarios VIa and VIb. These represent the two major ways in which the combined defense and offense traits determine predation success, often called unidirectional and bidirectional traits (Abrams, 2000).

Scenario VIa: Unidirectional traits

In this scenario, the prey have the same defense trait as in scenario I and face the same trade-off; therefore, prey can be undefended and fast-growing, or defended and slow-growing. The predator varies in its offense strategy, which can be described as a selectivity–efficiency or generalist–specialist trade-off. Selective predators can feed very effectively on undefended prey, but are unable or almost unable to capture defended prey; conversely, non-selective predators can capture all prey, but this comes at the cost of a reduced attack rate. This scenario leads to a feedback loop between the two traits: when the predator population is dominated by selective predators, selection favors defended prey over undefended ones; but as the prey population becomes dominated by defended prey, the non-selective predator performs better than the selective one and increases. This, in turn, causes selection on defense to reverse and, as the prey population becomes dominated by undefended phenotypes, the selective predator takes over in the predator population again.

Scenario VIb: Bidirectional traits

In this scenario, a predator is effective at capturing a certain prey when prey and predator traits match. Therefore, prey that are defended against one predator strategy are highly vulnerable to another, and each predator is effective at consuming prey that correspond to its trait, but less effective or unable to capture others. This can lead to a similar trait–trait feedback as in the unidirectional case (scenario VIa): whenever one predator phenotype becomes dominant in the population, prey that are defended against this predator are selected for; as they rise in dominance, predators that can utilize this prey will

follow, causing selection on the prey to reverse, in turn followed by selection on the predators, and so on. This continuing back and forth has been shown to result in intermittent cycles (Bengfort et al., 2017).

Intermittent cycles have already been shown in modeling studies for scenarios II, IV, V, VIa, and VIb (II: Yamamichi et al., 2011; IV: Yamamichi & Letten, 2021; V: Abrams et al., 2003; Klauschies & Gaedke, 2020; VIa: Coutinho et al., 2016; Mougi, 2010, 2012; Mougi & Nishimura, 2008, van Velzen & Gaedke, 2017; VIb: Bengfort et al., 2017; Jones et al., 2009; Khibnik & Kondrashov, 1997), but their prevalence has not been quantified. Scenarios I and III have not been previously linked to intermittent cycles, but such a link is plausible. By conducting a systematic quantitative comparison between the above scenarios, we aimed to determine what type(s) of trait variation have the greatest potential for resulting in intermittent cycles.

Simulation approach

To quantify the incidence of intermittent cycles across the 16 investigated models, we ran 10,000 simulations per model with randomized initial conditions and randomly chosen parameter values (see Appendix S1 for details on the investigated parameter ranges). Simulations were run for 60,000 time steps; all calculations were based on the last 30,000 time steps of the generated dynamics. Simulations that resulted in any extinctions (defined as any species or genotype being below an extinction threshold of 10^{-20} at the end of the simulation) were discarded before analysis, because their dynamics no longer represented the scenario being investigated. The discarded results were not considered part of the data set of 10,000 simulations, and new parameter values were chosen. Wolfram Mathematica 10 was used for all simulations.

Models that were found to result in intermittent cycles at a significant frequency (≥5% of simulations resulting in intermittent cycles) were subsequently studied in more detail to gain a comprehensive understanding of the conditions under which intermittent cycles arose.

Detecting intermittent cycles

Intermittent cycles are characterized by the presence of two timescales in the dynamics: a fast timescale on which predator–prey oscillations occur, and a slower timescale on which the amplitude of these predator–prey oscillations change (see Figure 2). In all models, the dynamics used for this analysis were those of the total prey and total predator populations; for models with discrete genotypes (M) or phenotypes (PP), the biomasses per trophic level were summed up before analysis. Detection of both timescales in the simulated dynamics can be done with Fourier analysis. However, as changes in the amplitude of oscillations did not show up as a single peak on the Fourier spectrum at the corresponding frequency (see Appendix S2 for a detailed explanation), detecting the presence of intermittent cycles therefore required a more sophisticated approach, which we describe below.

Time series decomposition: Detecting the fast and slow timescales

To detect intermittent cycles, we started by deconstructing the original dynamics (OD) into three components: mean dynamics (MD), ecological cycles derived from rescaled dynamics (RD), and the amplitude dynamics (AD) (see Figure 4). The latter two were then used to quantify intermittency (see below).

Mean dynamics (MD)

Over the course of the slow cycle, the average biomass of prey or predators may change (Figure 4, upper panels); if the slow cycle corresponds to changes in the attack rate, for example, this will affect prey and predator biomasses as well as the stability of dynamics. This can confound the Fourier analysis on the biomass dynamics (see Appendix S2 for a more detailed explanation), making de-trending necessary. The MD were derived using a moving window approach, in which mean biomass was calculated as the average biomass over the window. The width of the window was set to be twice the average distance between biomass peaks in the OD.

Rescaled dynamics (RD)

In the next step, the RD were generated by subtracting the MD from the OD. This removed any effect of trait changes on the average biomass; all dynamics in this time series therefore arose solely from ecological predator–prey cycles, yielding a clean signal in the Fourier spectrum. Fourier analysis of the RD was done to determine the average power and frequency of this "fast" cycle.

Amplitude dynamics (AD)

These were derived using the moving window approach on the RD, this time calculating the standard deviation of the biomass within this window. The width of the window was the same as for the MD. The Fourier spectrum of the AD was used for determining the power and frequency of the "slow" cycle.

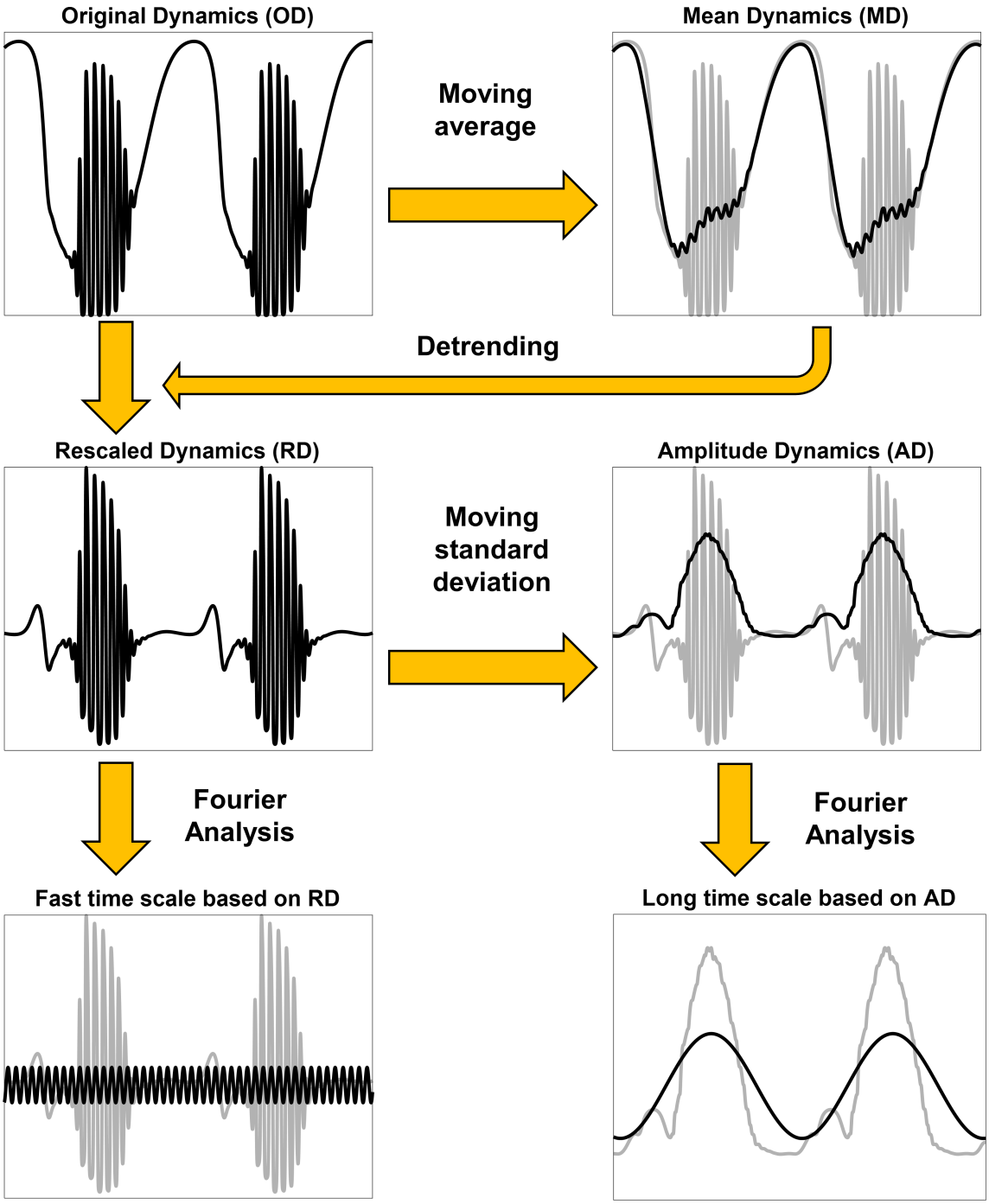

FIGURE 4 Roadmap for detecting intermittency in a time series. The first step is de-trending the original dynamics (OD) by subtracting the long-term average, resulting in rescaled dynamics (RD) which ideally contains only the ecological predator–prey cycles. The moving standard deviation of these RD yields the amplitude dynamics (AD). With Fourier analysis, the amplitudes and frequencies of RD and AD are extracted, and are used in the calculation of the intermittency index (see Equation 1). The above example yields an intermittency index of $I = 0.84$

Quantifying intermittency

Intermittent cycles were present when the Fourier spectra of RD and AD (see "*Time series decomposition: Detecting the fast and slow timescales*") both contained a substantial peak, indicating that there were biomass oscillations on the fast timescale and variability in the amplitude on the slow timescale. Further oscillations in the temporal average of population abundances on the slow timescale may be present as well, but their presence or absence is unrelated to the question of whether the predator–prey cycles count as "intermittent"; therefore,

the Fourier spectrum of MD was not used for detecting or quantifying intermittency.

We developed the intermittency index I as a quantitative measure, using the information from the Fourier analysis: the frequency and power of the highest peak ("dominant frequency"; see Appendix S2) of the fast dynamics in RD (F_{RD} and P_{RD}, respectively), and the frequency and power of the slow dynamics in AD (F_{AD} and P_{AD}):

$$I = \frac{2P_{RD}P_{AD}}{P_{RD}^2 + P_{AD}^2} \cdot \left(1 - \frac{2F_{RD}F_{AD}}{F_{RD}^2 + F_{AD}^2}\right) \quad (1)$$

This index was composed of two parts, both of which had a minimum of zero and a theoretical maximum of 1. Therefore, I is a number between 0 and 1, where $I = 0$ indicates completely regular cycles, and $I > 0$ some degree of intermittency; higher values imply the dynamics are more strongly intermittent. The two halves of the index reflect the two necessary components for the presence of intermittency: there must be substantial variability in the amplitude of the predator–prey cycles (measured by P_{AD} relative to P_{RD}); and this variability must occur on a timescale significantly slower than the predator–prey cycles (measured by F_{AD} relative to F_{RD}). For details on how P_{AD}, P_{RD}, F_{AD}, and F_{RD} affected the value of I, see Appendix S3: Figure S1.

The left part of the definition of I uses the power of the highest peaks in the Fourier spectra of RD and AD. If only the fast timescale is present ($P_{AD} = 0$), this part is zero, and therefore $I = 0$. When both timescales are present ($P_{RD} > 0$ and $P_{AD} > 0$), this part will have a positive value, with a maximum of 1 when $P_{RD} = P_{AD}$. This indicated that the fast and slow timescales were equally strongly contributing to the total dynamics, corresponding to a high degree of intermittency.

The right part of the definition of I uses the frequencies of the fast and slow timescales, with a minimum of 0 when the timescales are the same (indicating no intermittency at all), and increasing toward a theoretical maximum of 1 with an increasing difference in the timescales. This theoretical maximum will in practice never be reached, as it would require the slow timescale to be infinitely slower than the fast timescale.

As we are measuring intermittency in predator–prey dynamics, the power and frequency values in the above equation always represent the average values calculated for prey and predator dynamics. Dynamics were classified as "intermittent" when $I > 0.2$; this threshold value implies that P_{AD} and P_{RD} do not differ by more than an order of magnitude, and F_{AD} is at least twice as slow as F_{RD} (see Appendix S3 for details). The use of a different threshold value only has a minor effect on the results (Appendix S3: Figure S2).

Application to empirical and experimental data

The index I as defined above in Equation (1) is based on Fourier analysis, which means that it requires very long time series data to give a reliable result. This is not an issue for analyzing model dynamics, but empirical data sets may not be long enough for this. In particular, even relatively long data sets (such as the dynamics shown in Figure 1) will not be long enough to detect the frequency of the AD with spectral analysis. To apply the intermittency index to empirical data, the results of Fourier analysis were approximated by fitting a sine wave function to the data:

$$W(t) = \boldsymbol{A}\sin(2\pi\boldsymbol{\omega} \cdot t + \boldsymbol{\varphi}) + \boldsymbol{c} \quad (2)$$

This function was fitted to the RD to get the fast timescale, and to the AD to get the slow timescale; the four parameters were estimated using the MATLAB function *fitnlm()*. The estimated amplitudes (A_{RD} and A_{AD}) and frequencies (ω_{RD} and ω_{AD}) were then used to calculate I (Equation 1).

RESULTS

Quantitative analysis

The 16 investigated models showed a wide variation in the frequency with which they exhibited intermittent cycles, ranging from 0% to slightly more than 60% of the investigated parameter space (Figure 5). Of the seven scenarios, five showed intermittent cycles (II, IV, V, VIa, and VIb) whereas the remaining two (I and III) did not. Although

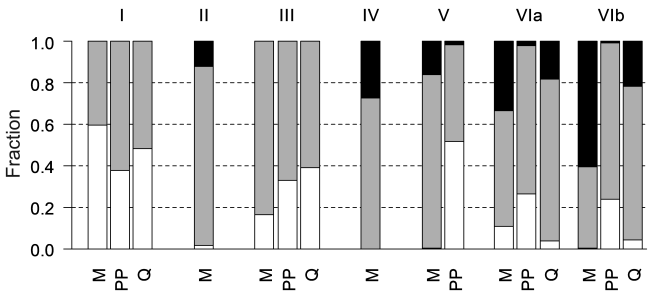

FIGURE 5 Results of the quantitative analysis of all 16 investigated models; bars represent frequencies of outcomes found across 10,000 replicate simulation runs. Colors denote the three possible outcomes: stasis (standard deviation of prey and predator dynamics both $<10^{-3}$; white), regular cycles (SD $> 10^{-3}$ and $I < 0.2$; gray), and intermittent cycles (SD $> 10^{-3}$ and $I > 0.2$; black). The Roman numerals I–VIb represent the seven scenarios (see Figure 3); the mode of adaptation is given on the x-axis (M: evolution of Mendelian traits; PP: phenotypic plasticity; Q: evolution of quantitative traits)

the scenarios that exhibited intermittent cycles always did so for all modes of adaptation, the actual frequency strongly depended on the mode of adaptation: evolution of Mendelian traits (M) resulted in the highest frequency of intermittent cycles, followed by evolution of quantitative traits (Q). Adaptation through PP always resulted in a very low frequency of intermittent cycles (Figure 5).

Strikingly, all six models in scenarios VIa and VIb, incorporating trait adaptation on both trophic levels, showed intermittent cycles, whereas they were found in only four of the 10 models with adaptation only in the prey or only in the predators (scenarios I–V). Additionally, the models with trait adaptation on both trophic levels showed the highest frequencies of intermittent cycles: the M-variants of scenarios II, IV, and V had relatively few intermittent cycles (12%, 27%, and 16%, respectively) compared with those of scenarios VIa and VIb (48% and 60%, respectively).

Overall, these results indicated that intermittent cycles were far more likely to be driven by trait changes on both trophic levels than by trait changes on a single trophic level; but they also clearly showed that this was not a necessary condition. In the next section, we will take a closer look at the necessary conditions for intermittent cycles, and how they explain the results of the quantitative analysis.

Explaining differences between models: What is needed for intermittent cycles?

The results of the quantitative analysis give rise to two questions:

1. Why are intermittent cycles never found in two of the seven scenarios?
2. Why is the incidence of intermittent cycles so strongly dependent on the type of trait adaptation within each scenario?

Both questions can be answered by considering the conditions that are necessary for intermittent cycles to arise.

Necessary condition 1: Trait changes must result in recurrent switching between stable and unstable dynamics

Intermittent cycles arise from temporal variation in the stability of the predator–prey dynamics. Trait adaptation in itself is not sufficient to ensure this; the change in the trait(s) must also be pronounced enough for the system to switch between stable and unstable dynamics. Therefore, one possible explanation is that scenarios I and III do not support the required magnitude of trait changes, whereas the other four scenarios do. However, it can easily be shown that this is not the case: both scenarios I and III can result in pronounced trait changes (Figure 6a,c), and these trait changes can certainly result in switching between stability and instability (Appendix S4: Figures S3a and S4a), yet they do not result in intermittent cycles (Figure 6a,c), as the system never remains stable long enough for the existing cycles to become dampened. In addition, in scenarios II, IV, and V, trait changes that are sometimes relatively minor can result in intermittent cycles (Figures 6b,d and 7a). This is particularly pronounced in scenario II, in which the slow-switching prey remain strongly dominant throughout the simulation (Figure 6b), but the dynamics are stabilized as soon as the fast-switching prey are noticeable present in the population (Appendix S4: Figure S3b). Therefore, the first necessary condition is met in all scenarios, and is not an explanation for why intermittent cycles are not found in scenarios I and III.

Necessary condition 2: Trait dynamics must be slower than ecological dynamics

The second necessary condition is that recurrent switching between unstable and stable dynamics must occur on a slower timescale than the predator–prey cycles. This is necessary for two reasons: first, because several predator–prey cycles have to occur in between the "interruptions"; and second, because the interruptions have to last long enough for the existing cycles to be dampened entirely or almost entirely. This second condition is not met in scenarios I and III (Figure 6a,c) whereas it is met in scenarios II, IV, and V (Figures 6b,d and 7a), as well as in scenarios VIa and VIb (Figure 7b,c). Below we will discuss the models with trait variation on one trophic level (scenarios I–V) and on both trophic levels (VIa and VIb) separately, as the necessary delay in the trait cycle is achieved in different ways in these two cases.

Trait changes on a single trophic level

When trait variation is only present on one trophic level, the potential for a trait cycle that is significantly slower than the ecological cycles is quite restricted. On the one hand, trait changes must be driven by the ecological population dynamics; but on the other hand, they cannot be driven by the changes in population density caused by the predator–prey interaction. This is the critical distinction between the scenarios in which intermittent cycles are not possible (I and III) and those for which they are possible

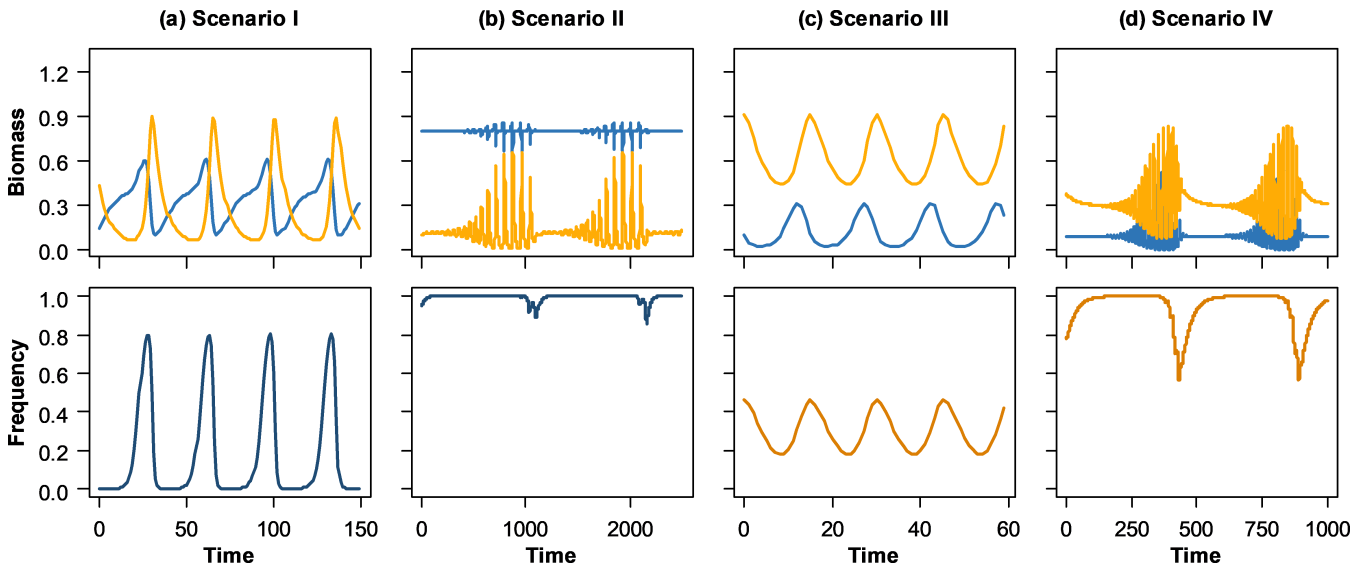

FIGURE 6 Examples of time series of scenarios I–IV (see section "*Methods*" and Figure 3 for details). The mode of trait adaptation is evolution of Mendelian traits in all cases. Upper panels: biomass dynamics of prey (in blue) and predators (in orange). Lower panels: Trait dynamics (i.e., the change in frequencies of the discrete genotypes). Frequencies of prey genotypes are shown in dark blue (a, b) and of predator genotypes in dark orange (c, d). In all cases, a higher frequency is associated with instability of the dynamics (see Appendix S4: Figures S3 and S4: in (a), it represents the frequency of undefended genotypes; in (b), the frequency of slow-switching prey; in (c), the frequency of the offensive predator; and in (d), the frequency of the slow-switching predator. For details on the models and the parameter values used, see Appendix S1

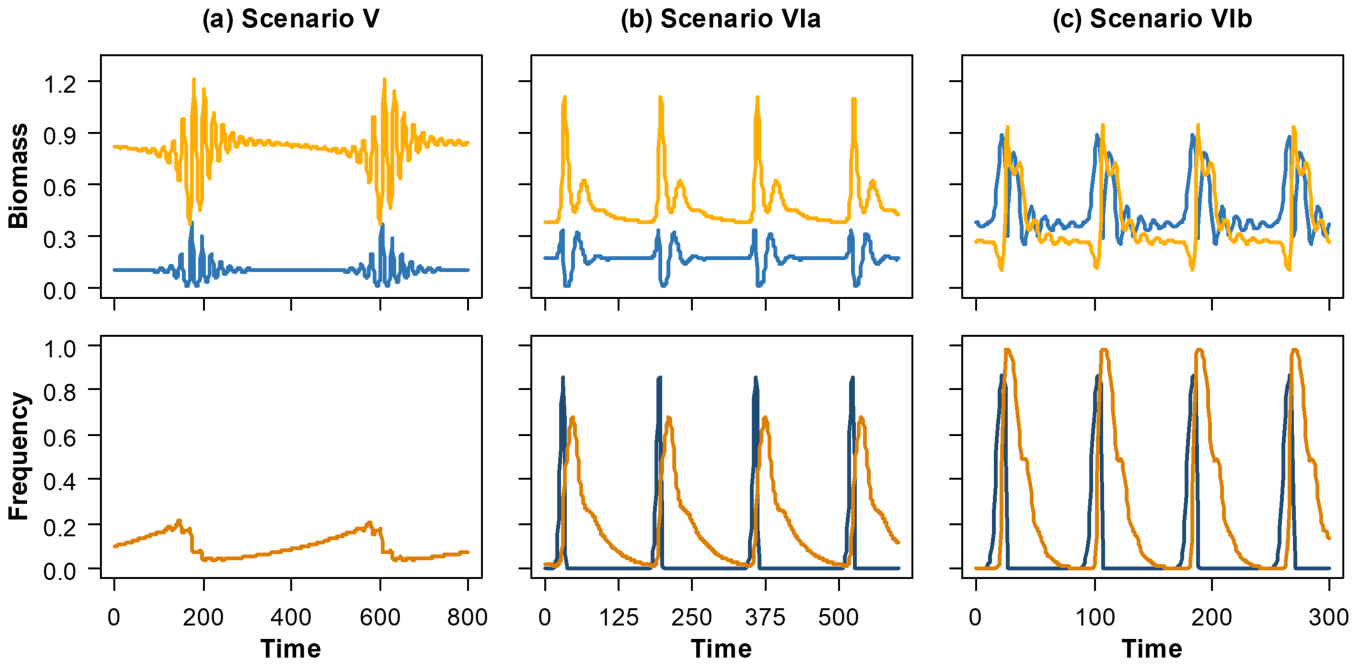

FIGURE 7 Examples of time series of scenarios V, VIa, and VIb (see Figure 3 and section "*Methods*" for details). The mode of trait adaptation is evolution of Mendelian traits in all cases. Upper panels: Biomass dynamics of prey (in blue) and predators (in orange). Lower panels: Trait dynamics (i.e., the change in frequencies of the discrete genotypes). Frequencies of prey genotypes are shown in dark blue (in [b], frequency of undefended prey; in [c], frequency of prey N_1), and of predator genotypes in dark orange (in [a], frequency of the gleaner; in [b], frequency of selective predators; in [c], frequency of predator P_1). For details on the models and parameter values used see Appendix S1

(II, IV, and V). In scenario I, the prey trait confers defense against predators, resulting in selection for defended phenotypes when predator density is high, and selection for undefended phenotypes when predator density is low (Figure 6a). Similarly, in scenario III, selection favors high-offense predator phenotypes when prey density is high, and low-offense predator phenotypes when prey density is low (Figure 6c). In both cases, this can result in pronounced recurring trait changes; but these trait changes will always occur on the same timescale as the predator–prey cycles, because the changes in predator abundance (in scenario I) or in prey abundance (in scenario III) that result from the predator–prey interaction directly cause the trait changes (Figure 6a,c).

We can therefore specify the second necessary condition somewhat further: for intermittent cycles to arise, the trait changes must respond to something other than the ups and downs of the ecological predator–prey cycles. At the same time, however, when trait changes occur on only one trophic level, this must be in response to the predator–prey cycles (at least when environmental forcing is absent). The resolution to this apparent contradiction is that trait changes must be driven by the presence or absence, or the amplitude, of predator–prey cycles: one genotype has the advantage during large-amplitude cycles, whereas the other is promoted when cycles are small or absent. But this is not sufficient to generate intermittent cycles: the other necessary part is that the genotype that has the selective advantage when the dynamics are cycling must stabilize the dynamics, whereas the genotype that has the advantage when cycles are absent must induce cycles. If any of these steps are missing, there will be either no recurrent trait changes or they do not result in intermittent cycles.

Scenarios II, IV, and V all support this entire set of conditions. In scenario II, the fast-switching prey is promoted by pronounced cycles in predator density, but it stabilizes the dynamics; and the slow-switching prey has the advantage when predator density is stable, but induces cycles. The same mechanism generates intermittent cycles in scenario IV, but with fast-switching and slow-switching predators. Similarly, in scenario V, the opportunist predator is favored when prey density is cycling, but stabilizes those cycles when it is dominant, whereas the gleaner predator benefits from stable prey densities, but will induce cycles. Therefore, both scenarios support this complex feedback cycle that generates intermittent cycles when trait changes only occur on one trophic level; but even then, the frequency with which this happens is low compared with the scenarios in which trait changes occur on both trophic levels (Figure 5), reflecting the restrictive nature of the conditions that must be met.

Trait changes on both trophic levels

Although a disconnect between the timescales of the ecological dynamics and trait dynamics is difficult to achieve when trait dynamics only occur on one trophic level, the potential is far greater when trait changes occur on both trophic levels. Here, the most common situation is that prey and predator traits change in response to each other, rather than in response to the population dynamics (Figure 7b,c). For example, in scenario VIb, each predator genotype has a preferred prey genotype; selection on the prey trait is therefore mainly dependent on which predator genotype is dominant, rather than on general predator density, and the same holds true for selection on the predator trait. This results in a pattern, found in both scenarios VIa and VIb, in which prey and predator traits "chase" each other back and forth on a timescale that is often slower than the predator–prey cycles (Figure 7b,c). Interruptions in the predator–prey cycles are now mainly caused by periods in which the predator trait is slow in catching up with the prey trait, resulting in protracted periods of time when the predators are poorly adapted to the prey (Figure 7b,c, lower panels). Intermittent cycles are promoted by longer periods of maladaptation, as this provides two necessary ingredients: a slow trait cycle that ensures the decoupling from the predator–prey cycles; and enough time for the existing cycles to become (almost) entirely dampened.

The necessity of slow adaptation and, in particular, slow adaptation in the predator compared with the prey, also explains why within the same scenario the different modes of adaptation show such strikingly different shares of intermittency (Figure 5). In quantitative trait (Q) models, the speed of adaptation is linked to the heritabilities of the traits, which are not necessarily equal and therefore modeled as independent parameters. Intermittent cycles are rarely found when the predators can adapt more rapidly than the prey (Figure 8a), so that the potential for intermittent cycles is roughly half that of their Mendelian-trait counterparts (Figure 5). Finally, PP results in rapid trait changes by its very nature. This can only result in intermittent cycles when the switching rates are very low, resulting in a very small frequency of intermittent cycles (Figures 5 and 8b).

Linking intermittent cycles to demographic rates

The results of the previous sections show that intermittent cycles can arise from the presence of contemporary trait adaptation; if so, it is most likely if trait adaptation is occurring on both trophic levels. At the same time, even for the models that most frequently generate intermittent cycles,

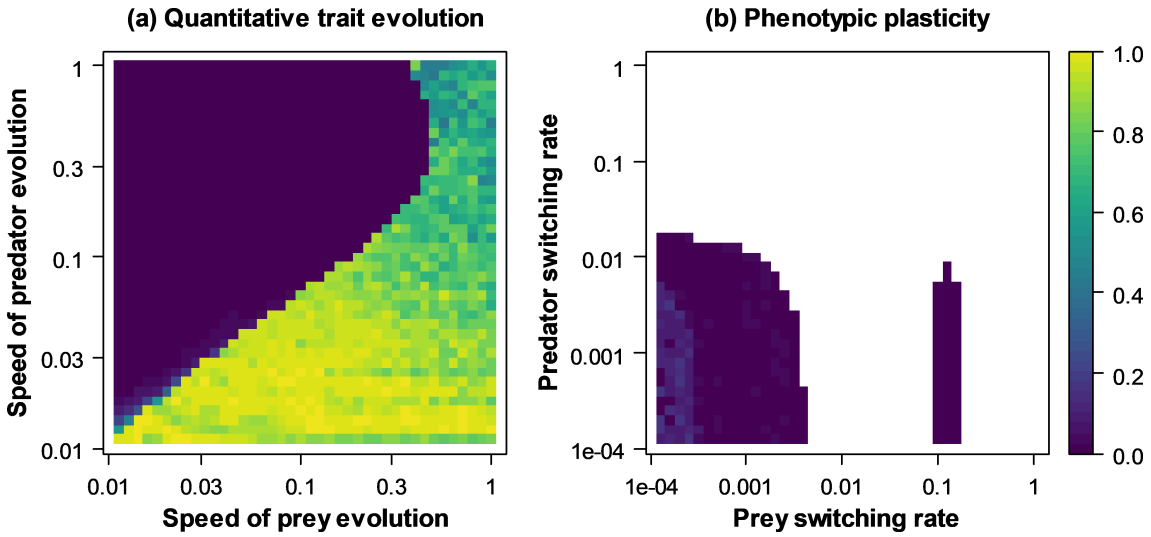

FIGURE 8 Impact of the speed of adaptation in predators and prey on intermittency in scenario VIb (bidirectional co-adaptation), for the two modes of adaptation in which the speed of adaptation can be independently varied. (a) Coevolution of quantitative traits (Q), varying the heritability parameters G_N and G_P. (b) Mutual phenotypic plasticity (PP), varying the parameters χ_N and χ_P (see Appendix S1 for details). Colors denote the index value, as shown in the legend on the right; in (b), white areas denote a stable equilibrium

this outcome is far from universal (Figures 5 and 9). To gain more detailed insights into what regions of parameter space are likely to yield intermittent cycles, we investigated the models in which intermittent cycles were found more systematically. In the following, we focus on the models for evolution of Mendelian traits, because this mode of adaptation could be investigated for all scenarios, and because it is associated with the highest probability of intermittent cycles. Because intermittent cycles arising from trait changes occurring on one or two trophic levels (scenarios II, IV, and V vs. VIa and VIb) are generated by different mechanisms, we will again treat these two cases separately.

Trait changes on one trophic level

The complex set of conditions that are necessary for intermittent cycles to arise in this case have been outlined in the previous section. But even though all these conditions are necessary, they are not sufficient, as is evident from the fact that intermittent cycles only occur for part of the parameter space in scenarios II, IV, and V (Figure 9a,d,g). There is an additional requirement: stabilization of the dynamics must be strong enough to dampen the predator–prey cycles sufficiently. This means that intermittent cycles are found when the stabilizing genotype can increase very rapidly in abundance and therefore quickly becomes dominant, or when it stabilizes the dynamics very strongly and rapidly; they are most likely when both of these conditions are true (see Appendix S5).

In scenarios II and IV, this means that intermittent cycles are promoted by the two prey (in scenario II) or predators (scenario IV) differing strongly in their switching rates (Figure 9a,d), as this is the region where the fast-switching (i.e., stabilizing) genotype can increase most rapidly and stabilizes the dynamics most strongly (Appendix S5: Figures S1a–c and S2a–c). In scenario V, the region for intermittency is located at the upper edge of the coexistence region (Figure 9g), adjacent to the exclusion boundary of the (destabilizing) gleaner. This is the region of parameter space where the (stabilizing) opportunist can increase rapidly, whereas the gleaner can only increase very slowly as a result of its mortality rate being close to the maximum it can endure before being excluded (Appendix S5: Figure S3b,e). This combination allows the opportunist to remain dominant long enough to stabilize the dynamics (see Figure 7a). Therefore, in this scenario, the presence of intermittent cycles points to a certain fragility of trait diversity: a minor change in environmental conditions (specifically, a change resulting in increased mortality for the gleaner) would result in exclusion of the gleaner.

Trait changes on both trophic levels

In scenarios VIa and VIb, the parameter regions where intermittent cycles occur reflect the requirement that the predator trait must be slow in catching up with the prey trait (see Figure 7b,c). In both scenarios, this means that intermittent cycles are found in two distinct regions in parameter space; in each, one of the predators can

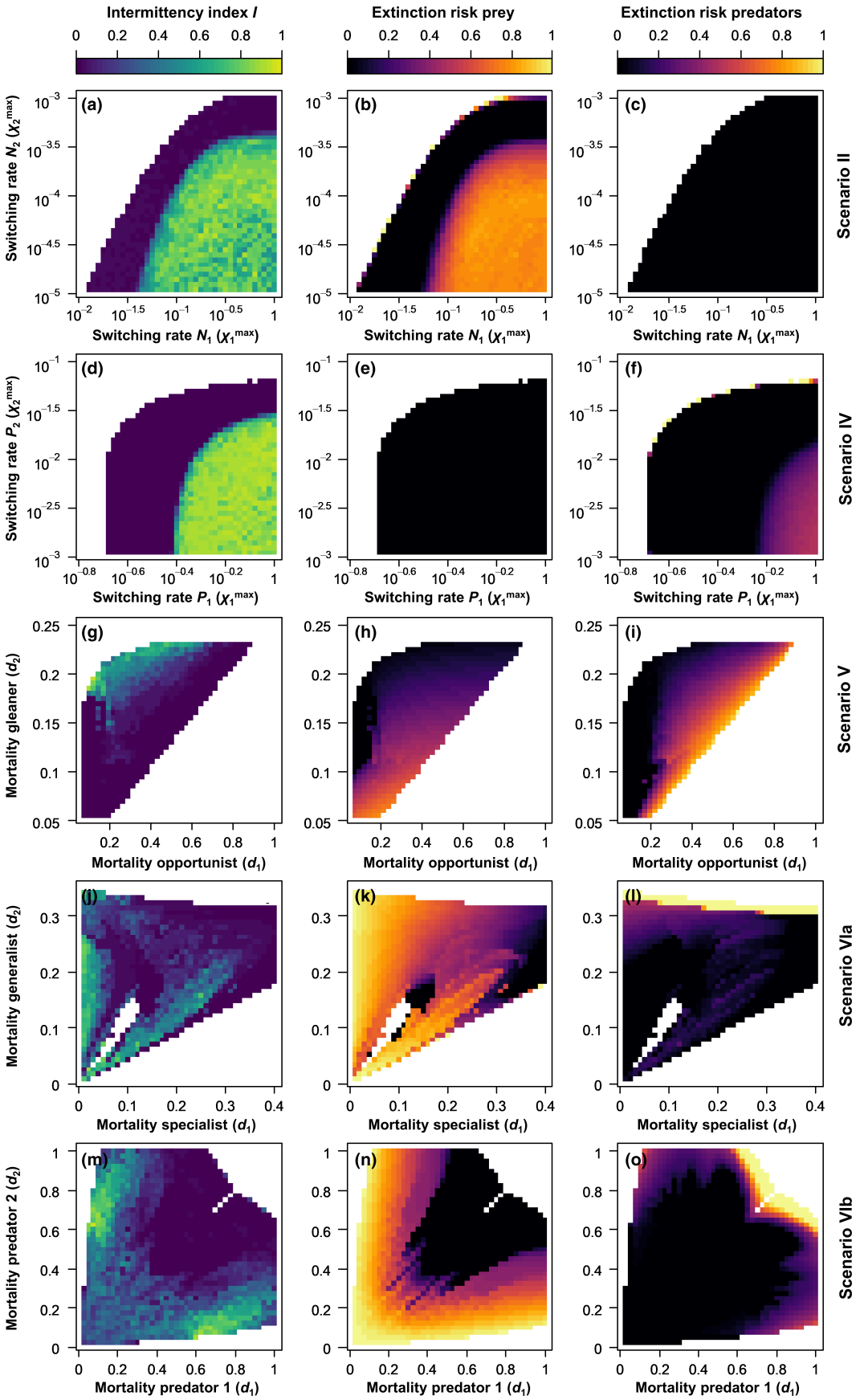

FIGURE 9 Legend on next page.

increase quickly and the other only slowly (see Appendix S5: Figures S6–S8).

In scenario VIa, these regions correspond to a mortality rate for the specialist predator that is either very high or very low (Figure 9j). In the former case, the specialist predator had difficulty increasing in frequency as a result of its high mortality rate; in the latter case, the generalist predator could only displace the specialist slowly as a result of the specialist's very low mortality rate. In scenario VIb, the strongest intermittent cycles are found when one of the predators has a very high mortality rate, whereas that of the other predator is low (Figure 9m). As in scenario V, three of these four regions in VIa and VIb are located next to the exclusion boundary of one of the predators (Figure 9j,m), as those predators will have low (but positive) invasion fitness when their mortality rate is close to the maximum that they can withstand. This indicates that, here too, intermittent cycles show a link to fragility of trait diversity.

Risk of diversity loss under intermittent cycles

Our results indicate (see previous section) that intermittent cycles may be a signal for the fragility of diversity maintenance, because of their tendency to occur near exclusion boundaries (Figure 9g,j,m). This link between intermittency and risk of trait loss can be quantified in scenarios V and VIb, showing a strong negative correlation between the index value and the distance from the exclusion boundaries (Spearman rank correlation: $r = 0.65$ for scenario V, $r = 0.87$ for scenario VIb; see Appendix S6: Figure S2). But there is an even more striking pattern pointing to a link with fragility: if trait changes occur in the prey, either by itself or in combination with the predators (i.e., scenarios II, VIa, and VIb), intermittent cycles are consistently and strongly associated with a high exclusion risk of at least one of the prey genotypes (Figure 9b,k,n). The risk of exclusion in this case is measured as the fraction of time that at least one genotype has a biomass below a threshold value of 10^{-3}, putting it at risk of stochastic extinction. In the parameter regions where intermittent cycles are found, this number is very high: up to 75% in scenario II, and up to nearly 100% in scenarios VIa and VIb (Figure 9b,k,n). Regions where cycles are regular are not generally associated with high risk of trait loss in the prey. The correlation between intermittency and prey risk of trait loss is indeed strong in all three scenarios (II: $r = 0.71$, VIa: $r = 0.62$, VIb: $r = 0.85$; see Appendix S6: Figure S3). The same pattern does not hold for the predators: although there are sometimes regions where their extinction risk is substantial, they are typically small and show no correlation with intermittency (Figure 9, right column).

In all three scenarios (II, VIa and VIb) this pattern can be explained by the fact that intermittent cycles require extended periods of dominance of one of the prey genotypes. In scenario II, the "slow-switching" prey destabilizes the dynamics only very weakly, whereas the "fast-switching" prey stabilizes quite strongly (Appendix S5: Figure S1a,d). Therefore, the emergence of predator–prey cycles requires a near-complete dominance of the slow-switching prey for a substantial period of time, because the dynamics of the entire system are stabilized as soon as the fast-switching prey is noticeably present in the population (see Figure 6b for an example of these dynamics). In scenarios VIa and VIb, the pattern arises due to the causal link between intermittency and a delay in trait changes in the predators, resulting in a protracted period of time when the predator is poorly adapted to the prey (see "*Explaining differences between models*" for a full explanation). During this period of predator maladaptation, one of the prey genotypes is always dominating the population, whereas the other is at extremely low biomass (see Figure 7b,c), putting it at high risk of stochastic extinction. These periods of extreme dominance of one prey genotype often constitute the majority of the total time, only punctuated by brief periods when the prey biomasses are more equal (Figure 7b,c), explaining the very high risk of losing trait variation faced by the prey in these scenarios (Figure 9k,n).

DISCUSSION

Intermittent predator–prey cycles have previously not received much attention from either ecological theorists or from empiricists, even though they have been demonstrated to occur in both models (Bengfort et al., 2017; Klauschies & Gaedke, 2020; Mougi, 2012; Yamamichi et al., 2011) and empirical systems (Blasius et al., 2020;

FIGURE 9 (a–o) Parameter ranges resulting in intermittent cycles for the five scenarios in which intermittency is found (see Figures 3 and 5). Left column: The intermittency index I; colors denote the index value, as shown in the legend at the top. The other two columns show the extinction risk on the prey level (middle column) and the predator level (right column); colors denote the fraction of the total time when at least one of the genotypes had a biomass below the threshold value of 10^{-3}. In all panels, white areas indicate extinction of at least one genotype

Wegge & Rolstad, 2018). Without a comprehensive investigation of how common they might be, the relevance of intermittent cycles to ecologists has so far been unclear. In this study we provide the first systematic investigation of the potential for contemporary intraspecific trait changes in prey, predators, or both to drive endogenous intermittent dynamics in predator–prey cycles.

Our results show that intermittent cycles can indeed occur under contemporary trait adaptation, although this is of course not the only mechanism that may drive them. This is in line with previous theoretical studies that showed intermittent cycles driven by trait changes (scenario II: Yamamichi et al., 2011; scenario IV: Yamamichi & Letten, 2021; scenario V: Klauschies & Gaedke, 2020; scenario VIa: Coutinho et al., 2016, van Velzen & Gaedke, 2017 scenario VIb: Bengfort et al., 2017); but the comprehensive nature of our investigation gives rise to some new insights. When intermittent cycles are driven by trait changes, this is far more likely to be a result of evolution than of PP; they are most likely to be caused by evolutionary changes on both trophic levels and they are more likely to have arisen from the evolution of Mendelian traits than quantitative traits. Moreover, when they are driven by contemporary coevolution between predators and prey, they are strongly promoted by the predators having difficulty adapting in response to trait changes in the prey, resulting in protracted periods when the predator is maladapted. Finally, there is a strong link between intermittency and the fragility of trait variation; so although intermittent cycles may be driven by the presence of trait variation, they also indicate that this trait variation, and the resulting adaptive ability, is at very high risk of being lost.

Although we covered a comprehensive range of models, it should be noted that this study was not intended as an exhaustive investigation of all conceivable mechanisms that might generate intermittent cycles, as this would clearly not be feasible. We specifically limited our investigations to predator–prey models incorporating mechanisms for adaptive trait changes in prey, predators, or both; the model equations themselves contain many assumptions (logistic growth in the prey, a type II functional response of the predators [except scenario V], and specific trade-off structures and trade-off shapes, in addition to the assumptions on trait dynamics outlined in Box 2). Other models incorporating trait adaptation in different ways or with different traits or trade-offs can certainly be devised, and this would almost certainly have an effect on the incidence of intermittent cycles (Figure 5). To prevent this from biasing our results, we kept the above model assumptions as identical as possible across all models we investigated, but we did not systematically investigate the effect of the assumptions themselves. However, the specifics of the model equations are not nearly as relevant as the necessary conditions for intermittency that we can derive from their results, as these point to general principles underlying intermittent cycles. An illustrative example is the model we used for scenario II (evolution of inducible defense), which was based on a model developed and studied by Yamamichi et al. (2011), but differs from this original model in some ways. First, the original model assumed that only one prey was phenotypically plastic, whereas we assumed in our model that both prey are plastic, but differ in their speed of plasticity. Second, the original model assumed intergenerational plasticity, whereas we assumed intragenerational (reversible) plasticity. Third, the original model was a chemostat model with explicit resource dynamics, whereas we assume prey self-limitation through logistic growth. Yet despite these seemingly significant differences, the dynamics produced by our model II-M (Figure 6b) are highly similar to those arising from the original model (Yamamichi et al., 2011); most critically, both models generate intermittent cycles through the same mechanism, described in the "*Methods*" section (description of scenario II), pointing to the presence of general underlying principles that should hold for other models as well.

Similarly, seasonal forcing may result in intermittent cycles (Nelson et al., 2013; Scheffer et al., 1997), but only under the same conditions that we demonstrated for intermittent cycles driven by trait changes: the seasonal variation in environmental factors must result in parameter changes (e.g., in growth or attack rates) that are pronounced enough to cause the dynamics to shift recurrently between stable and unstable dynamics; this seasonal cycle must be significantly slower than the intrinsic period of the predator–prey cycles (Figure 2a). Similarly, intermittent cycles can be caused by stochasticity-induced switches between stable and unstable dynamics in a bistable system (Ives et al., 2008), but whether or not this happens will critically depend on the strength of environmental noise. If the level of noise is too low, the system will remain on one of the two attractors, preventing the emergence of intermittent cycles; conversely, very high levels of environmental noise will cause rapid switching between stable and unstable states, preventing the dynamics to be clearly dampened through remaining on the stable attractor for a sufficiently long time. Intermittent cycles therefore require an intermediate level of noise (Figure 2b). In summary, regardless of what mechanism, or combination of mechanisms, is the driver behind intermittent cycles, we expect the same necessary conditions to apply.

Explaining intermittent predator–prey cycles

We identified two necessary conditions for trait changes to drive intermittency: first, that any trait changes must result in a switch between stable and unstable states; and second, that this switch must occur on a timescale that is significantly slower than the predator–prey cycles. Although the first condition can be met in all the models we investigated, the second condition is far more restrictive. It means that, although trait adaptation must be fast, it must not be too fast. This makes it very difficult for intermittency to be caused by phenotypically plastic trait changes, as these are typically very rapid, often occurring within a single generation. Conversely, even very rapid evolution will require several generations of selection before a significant change in the average population trait occurs. This is particularly the case when previous selective pressure has driven one of the genotypes down to very low frequency, so that it may remain nearly absent for a substantial amount of time even after it becomes selectively favored, slowing down the trait dynamics and thereby promoting intermittent cycles. This agrees well with intermittent cycles found in previous models with Mendelian traits (Abrams et al., 2003; Bengfort et al., 2017; Jones et al., 2009; Yamamichi et al., 2011), which were invariably linked with at least one genotype (or species, depending on the interpretation) falling to near extinction. The same pattern was observed in a model with phenotypically plastic defense and offense traits (Mougi, 2012), which appears to contradict our conclusion that PP is too rapid to produce intermittent cycles. However, in this model, the speed of trait changes depends explicitly on the phenotype frequencies, so that the trait dynamics are slowed down when one of the phenotypes falls to low frequency; it is again this slowing down that generates intermittent cycles (Mougi, 2012). In nature, we may therefore expect to see intermittent cycles in systems in which the main mode of adaptation is through rapid evolution in both prey and predators; this especially includes small organisms with fast generation times and large population sizes (DeLong et al., 2016). In contrast, larger animals will often show fast behavioral plasticity, for example hiding behavior in prey or switching behavior in predators, which would be likely to prohibit intermittent cycles.

The second condition also explains why trait changes on only one trophic level are unlikely to result in intermittent cycles. This requires some further conditions to be met: the fitness of genotypes with different traits depends on the presence or the amplitude of population cycles on the other trophic level; the genotype that has the selective advantage when the dynamics are cycling must stabilize the dynamics, whereas the genotype that has the advantage when cycles are absent must in turn induce cycles. Although some scenarios for trait adaptation do support this full set of conditions (scenarios II, IV, and V, previously demonstrated by Abrams et al., 2003, Klauschies & Gaedke, 2020, Yamamichi et al., 2011, and Yamamichi & Letten, 2021), our analysis shows that, even in these scenarios, intermittent cycles occur only at a fairly low frequency. All other models we investigated with trait adaptation only in the prey or only in the predators never yielded intermittent cycles at all, supporting the conclusion that trait adaptation on one trophic level is far less likely to be the mechanism underlying intermittency.

The difference between scenarios III and V is striking, given their strong similarity: both feature predators with different functional responses and different mortality rates, and the only distinction is that their handling times are identical in scenario III. Furthermore, coexistence of the predator genotypes is enabled by the same mechanism as in the classic gleaner–opportunist trade-off (Abrams & Holt, 2002; Armstrong & McGehee, 1980; Yamamichi & Letten, 2021). Closer examination shows that it is not strictly impossible for model III-M to generate intermittent cycles, but it is extremely unlikely (the incidence of intermittent cycles in III-M is 0.24%; for models III-PP and III-Q, the incidence is 0%). It is likely that the answer lies, at least partly, in the very narrow potential for predator coexistence when they have the same handling times (Abrams & Holt, 2002; Xiao & Fussmann, 2013), mainly allowing coexistence of predator genotypes that are very similar in their response to prey dynamics. This in turn makes it nearly impossible for the two predators to have a sufficiently different effect on prey dynamics to generate intermittent cycles. Another possibility lies in the different trait dynamics generated by these models: in the classic gleaner–opportunist trade-off, the predator with the higher attack rate is the "gleaner" and is favored when prey abundance is low (Abrams & Holt, 2002); the reverse appears to be true in the "offense evolution" scenario, in which predators with a higher attack rate are favored when prey abundance is high (Figure 6c). However, deriving a full explanation for this pattern is beyond the scope of this study.

The intermittent cycles found by Blasius et al. (2020), shown in Figure 1d, were hypothesized to be caused by stochastic effects. Our analysis indicates that this interpretation is most likely to be correct: whereas it is possible that undetected variation in edibility (i.e., defense) was present or arose through mutation in the algal prey, this is highly unlikely to be the case for their rotifer predators, which were all genetically identical at the start of the experiment. Therefore, any trait variation would have corresponded to scenario I in our investigation (see Figure 3), which does not yield intermittent cycles. In contrast, the intermittency

in the bacteria–phage dynamics of Wei et al. (2011), shown in Figure 1e, probably results from coevolution: this system contains two bacteriophages, and bacteria that are resistant to one are susceptible to the other, corresponding to scenario VIb.

In summary, coevolution between predators and prey is a more likely scenario for intermittent cycles than evolution on a single trophic level. At the same time, however, they mainly arise when predators evolve more slowly than their prey, either because heritable genetic variation is lower in the predators than in the prey (for quantitative traits), or because one of the predator genotypes has difficulty "invading" the population (for Mendelian traits). Another factor affecting the relative speed of evolution in prey and predators may be size differences between prey and predators: as predators are typically larger than their prey (Brose et al., 2006), they have smaller population sizes and longer generation times, and therefore generally evolve more slowly than their prey. But, regardless of how slow predator evolution is achieved, it is clear that intermittency is closely linked to maladaptation in the predators: this allows the prey to temporarily escape predation, resulting in the interruption and temporary disappearance of the predator–prey cycles. In addition, the presence of slow predator adaptation enables the predator to eventually overcome this maladaptation, driving the re-emergence of cycles.

Intermittent cycles are associated with risk of diversity loss

Our results show a consistent link between intermittency and fragility of trait variation, which can be subdivided into two distinct types; we further show that these are linked to different trophic levels. In the predators, intraspecific trait variation is at risk due to structural fragility, that is, the presence of intermittent cycles indicates proximity to an exclusion boundary for one of the predator genotypes (Figure 9g,j,m). This implies that a small change in environmental conditions may push the system over the exclusion boundary, resulting in the loss of trait variation. Therefore, the dynamics indicate that even a small change in the environment that increases mortality rates, for example as a result of increases in metabolic rates caused by global warming, will quickly push the system past a critical threshold where trait variation will collapse. Intermittent cycles are indicative of this type of fragility because they mainly occur when at least one of the predator genotypes takes a long time to increase after selective pressure changes in its favor. This implies that this predator genotype already has great difficulty recovering from low densities, and only a small amount of added mortality will push it beyond the point at which it can recover at all.

In the prey, conversely, intermittent cycles are associated with stochastic fragility: they occur when at least one of the prey genotypes spends a substantial fraction of time at extremely low densities. This indicates a high risk of extinction as a result of demographic stochasticity, and because of the discrete nature of living organisms (i.e., extremely low densities mean that, realistically, at some point there are zero individuals left). Therefore, trait-driven intermittent cycles are an indicator that trait variation is at high risk to be lost, regardless of whether they result from trait variation in the prey, the predators, or both. Moreover, loss of trait variation on one trophic level may also subsequently result in the loss of trait variation on the other. In the bidirectional coevolution model, for example, the loss of one of the prey genotypes will typically quickly be followed by the loss of the predator genotype that preferentially feeds on this prey. Therefore, just as species diversity in prey may cascade upwards to promote diversity in their predators (Brodersen et al., 2018), the loss of trait variation may cause an upwards cascade of collapsing trait variation on higher trophic levels.

The risk of losing intraspecific trait variation is obviously different from the risk of losing a species, but knowing whether the collapse of trait variation is imminent is still highly relevant. The well documented positive relationship between biodiversity and ecosystem functioning (Cardinale et al., 2012) does not require that biodiversity has to be in the form of species diversity; in fact, the main driver of this positive relationship is functional trait diversity rather than species richness (Cadotte et al., 2011; Gagic et al., 2015). Therefore, intraspecific trait variation can be just as important as species diversity for maintaining ecosystem functioning, and the loss of intraspecific trait variation may be just as devastating as species loss. Although it is also true that the presence of maladapted genotypes in the population lowers overall fitness, and the species may therefore benefit from the loss of such genotypes, this should only hold true if maladapted genotypes are expected to remain maladapted. This is not the case in all our models showing intermittent cycles, in which selective pressures recurrently change and each genotype goes through periods of being selectively favored and disfavored (Figures 6 and 7). The presence of currently maladapted traits may therefore prevent strong population declines, or even extinctions, when the selection regime changes. It has been shown that intraspecific trait variation in predation-related traits can allow populations to adapt to temporary changes in environmental conditions (pulse perturbations, for example storms, droughts or floods), thereby buffering population declines, lowering the risk of extinctions and allowing a faster recovery to the preperturbation state (Raatz et al., 2019). Loss of

intraspecific trait variation can therefore result in heightened extinction risk for the species in response to environmental stochasticity. As the frequency of extreme weather events is increasing due to climate change (Coumou & Rahmstorf, 2012; Ummenhofer & Meehl, 2017), the relevance of this type of extinction risk will also continue to increase, and the maintenance of intraspecific trait variation is likely to become more and more critical for a species' long-term survival.

Finally, although we have here interpreted the different prey and predators in Mendelian-trait models as genotypes of the same species, this is not the only valid interpretation of these scenarios. Mathematically, the dynamics of different genotypes and those of different species are indistinguishable; these models can therefore just as well describe the dynamics of different prey and predator species within the same community. Under this interpretation, loss of trait variation is identical to loss of species diversity. Although it generally makes little sense to analyze the total dynamics of multiple prey or multiple predator species in search for intermittency, the dynamics of individual prey or predator types (whether interpreted as genotypes or species) will show intermittent dynamics as well (Appendix S4: Figures S1 and S2). The common pattern is that one of the types within each trophic level exhibits intermittent cycles, depending on the density of the other, which shows a pattern of smooth increase and decline at the frequency of the slow "trait" cycle (Appendix S4: Figures S1 and S2). Interestingly, the type exhibiting intermittent cycles is not necessarily the one vulnerable to extinction (Appendix S4: Figure S2). This last result obviously makes the application to conservation efforts difficult: when intermittency is found, it will not be immediately clear whether it signals the impending loss of intraspecific genetic diversity or of species diversity; if it indicates the latter, the species at risk may be a different one entirely. Intermittent cycles can therefore be seen as an indicator that there is extinction risk present, but are not specific enough to indicate which species in particular need to be protected; this can only be found out by further investigations.

The link between intermittency in predator–prey cycles and extinction risk may suggest that intermittent cycles driven by trait dynamics should not be commonly found in the wild. However, mutations may allow lost genotypes to re-establish, especially in species with fast generation times and large population sizes. Another potential mechanism for maintaining trait diversity under high extinction risk is metacommunity dynamics, as local extinctions may be followed by immigration of the lost genotype as long as it was still present regionally (Leibold et al., 2004). Therefore, local processes may interact with regional processes in driving persistent intermittent predator–prey cycles.

Comparison with other mechanisms for intermittent cycles

Other mechanisms than recurrent trait changes may drive intermittent cycles (Figure 2a,b; Ives et al., 2008; Nelson et al., 2013; Scheffer et al., 1997). For example, intermittent cycles might be the result of dampened predator–prey oscillations that are often restored through pulse perturbations on a more or less regular basis. Furthermore, although the long-term dynamics of predator–prey systems may be associated with regular cycles, their transient dynamics may be intermittent (Abrams et al., 2003). As recent studies have emphasized that transient dynamics may be very important in the wild (Hastings et al., 2018), this may point to an even greater importance of intermittent cycles in nature. Because of its broad applicability, the approach we develop here for detecting intermittency cannot distinguish between different mechanisms, as it only measures to what degree intermittency is present. For instance, intermittent cycles caused by environmental forcing may in fact look highly similar to those caused by trait changes (Figure 2b,c), because both types of intermittency are caused by temporal changes in one or more parameters that are associated with the overall stability of the system. The main distinction is that, for environmental forcing, the period of the amplitude changes in the population cycles will be highly correlated with the period of the external driver that underlies the environmental forcing (e.g., cycles appearing and disappearing in a clearly seasonal pattern). Trait-driven intermittency, conversely, will not show a consistent association with environmental factors; although this does not rule out the presence of an, as yet unidentified, environmental driver, it may still serve as an indication that trait changes may be the causal factor. Switches between alternative stable states (Ives et al., 2008) will similarly be generally uncorrelated with environmental factors but, as this mechanism is driven by stochasticity, changes in cycle amplitude do not show any periodicity (Figure 2a), in which this is the case for seasonal forcing (Figure 2b) or trait changes (Figures 2c, 6 and 7). But these are merely guidelines; intermittent cycles may indicate the presence of any of the above mechanisms, or a combination of them (Taylor et al., 2013), and the final identification of the underlying mechanism will require specific investigation.

Future directions

Application to empirical data

In this study, we demonstrated the potential for intraspecific trait changes to drive intermittent predator–prey

cycles. One of our major aims was to bring intermittent cycles to the attention of the broader ecological community as a dynamic for which to look out. To enable this, we developed a new methodology for detecting and quantifying intermittency in population dynamics that is readily available for future investigations into intermittent cycles. Although most empirical time series data will not be long enough for Fourier analysis to yield accurate results, fitting a sine wave to the RD (third row of Figure 10) and AD (bottom row of Figure 10) can serve as an alternative approach for deriving the amplitudes and frequencies of the two timescales. This method is potentially less accurate than Fourier analysis, but has the advantage of being applicable to shorter time series (Figure 10).

As an example of how this approach may be applied, we used it on three empirical "prey" time series (the field vole *Microtus agrestis*, Figure 10a; the snowshoe hare *Lepus americanus*, Figure 10b, and the green alga *Monoraphidium minutum*, Figure 10c). The abundance data of field voles show clear intermittency, as indicated by an index value of $I = 0.80$ (Figure 10a); this corresponds very closely with studies finding a complete or near-complete dampening of their population cycles in the 1990s, followed by the re-emergence of high-amplitude cycles in the 2000s (Brommer et al., 2010; Wegge & Rolstad, 2018). The snowshoe hare data show a slightly more ambiguous pattern: there appears to be a gradual dampening of population cycles between 1860 and 1910, after which the amplitude starts to increase again (Figure 10b, bottom panel). The data set is not long enough to confirm whether this increase continued, forming a genuine intermittent pattern (with incomplete rather than complete dampening), instead of a general trend of

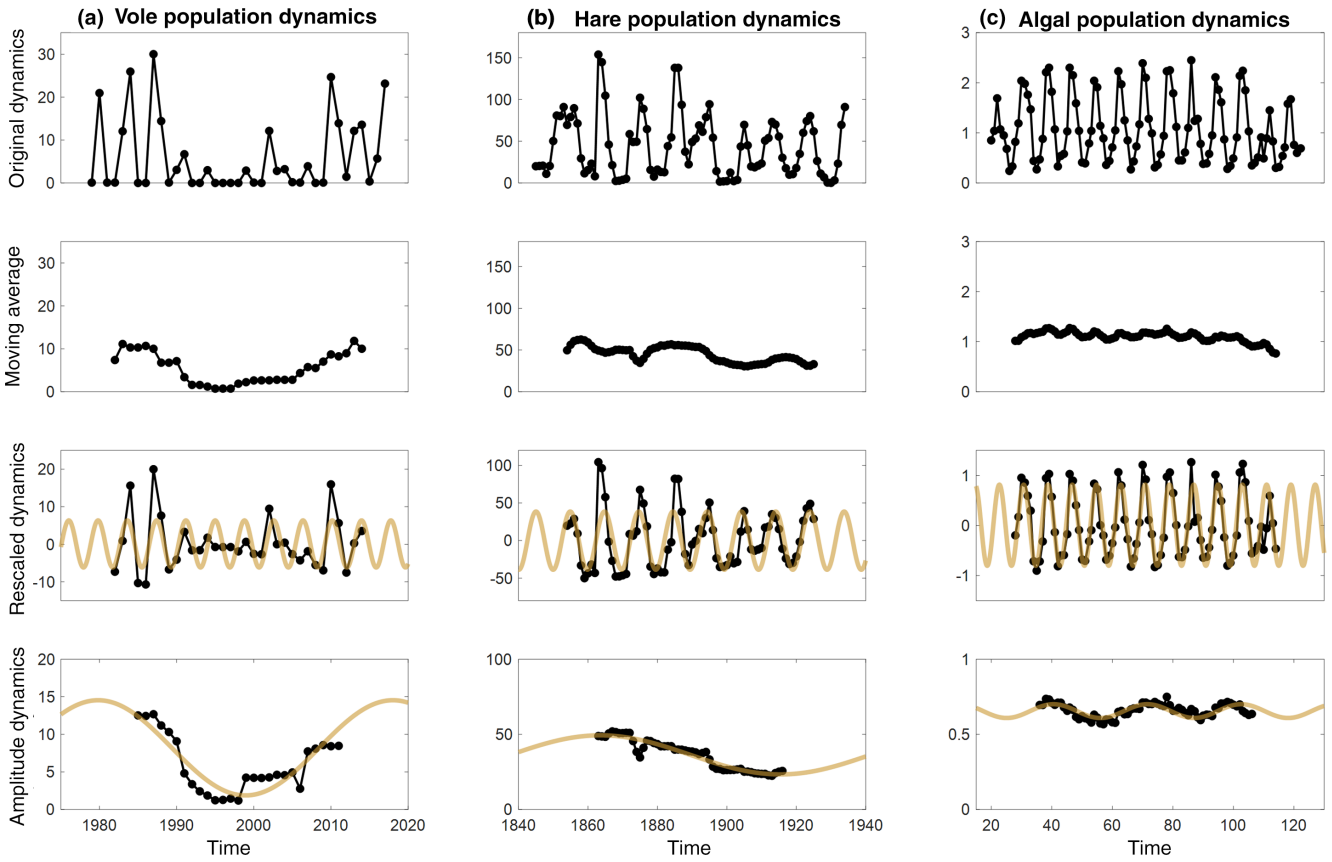

FIGURE 10 Application of the intermittency index to empirical data, using three time series of prey abundances. (a) Field vole trapping indices (data from figure 1a in Wegge & Rolstad, 2018), showing dampening and later re-establishment of high-amplitude population cycles. (b) Fur trade data for snowshoe hare (in thousands; data from Elton & Nicholson, 1942), showing a general dampening of population cycles, with a possible increase again after 1910. (c) Algal population dynamics (10^6 cells/ml; data from Blasius et al., 2020, experiment C8), showing very regular cycles. The orange lines in the third and fourth rows show the sine wave functions fitted to the rescaled dynamics (RD) and amplitude dynamics (AD), respectively. From these fitted curves, the intermittency index I can be calculated (see sections "*Methods*", "*Application to empirical and experimental data*"). (a) $A_{RD} = 6.27$, $\omega_{RD} = 0.264$, $A_{AD} = 6.34$, $\omega_{AD} = 0.026$; $I = 0.80$. (b) $A_{RD} = 38.72$, $\omega_{RD} = 0.101$, $A_{AD} = 13.01$, $\omega_{AD} = 0.01$; $I = 0.49$. (c) $A_{RD} = 0.81$, $\omega_{RD} = 0.125$, $A_{AD} = 0.046$, $\omega_{AD} = 0.032$; $I = 0.058$

dampening population cycles for this species. The curve fitting assumes the former, giving an index value of $I = 0.49$. Finally, the algal dynamics (Figure 10c) are highly regular and, accordingly, these dynamics yield an index value of $I = 0.058$: there is a slight degree of variability in the amplitude, as will be found in all empirical data sets, but the index value is well below our threshold for intermittency.

It should be noted here that the above calculations must be interpreted with great care. In the example of the hare dynamics (Figure 10b) in particular, the fit of the AD to a sine wave is unlikely to stand up to scrutiny, as the four-parameter sine wave fit would be rejected in favor of a linear regression model. A proper development of a statistical procedure for detecting intermittency in an empirical time series is beyond the scope of this paper, and we limit ourselves here to presenting a conceptual development of the type of analysis that could be done with empirical data.

Nevertheless, the strong intermittency ($I = 0.80$) found in the dynamics of field voles may be an indicator for contemporary trait changes; if so, these trait changes may be intraspecific through evolution (i.e., the "genotype" interpretation) or interspecific through changes in species composition within a trophic level (the "species" interpretation). In this particular case, the latter appears to be the more likely explanation. Latitudinal gradients in vole cycle amplitude within Fennoscandia, with higher-amplitude cycles found in the north, have been linked to a stronger dominance of specialist predators in this region (Hanski et al., 2001). The same reasoning holds true for temporal patterns: a shift toward dominance of generalist predators would dampen cycles, whereas recovery of the specialists would lead to their re-emergence. This exact pattern was demonstrated for voles in Finland (Korpela et al., 2014), linked specifically to the relative abundances of two small mustelid predators, the highly specialized least weasel (*Mustela nivalis nivalis*) and the somewhat more generalist stoat (*Mustela erminea*). Dominance of the stoat corresponded to periods when cycles disappeared, whereas their re-emergence was linked to increasing relative abundances of the least weasel (Korpela et al., 2014). This trait change scenario shows close correspondence with scenario VIa in our model analysis, which was one of the most likely trait change scenarios to drive intermittent cycles (Figure 5).

If the above interpretation of intermittency in vole population dynamics is correct, this has implications for potential extinction risk: of the voles themselves, of their specialist predators, or of other small rodent species competing with voles. The disappearance of vole cycles does appear to be linked with a strong decline in their overall abundance (Figure 10a, top panel; Gouveia et al., 2015; Korpela et al., 2013; Wegge & Rolstad, 2018), which would point to the extinction risk of the voles themselves. Their specialist predators would also be at risk, especially the least weasel that depends almost entirely on voles as prey. Finally, the above information implies that conservation efforts for protecting the voles might reduce the extinction risk of the voles themselves and of their specialist predators.

Further theory development

Although our results enable a comprehensive understanding of how and when intermittent cycles can arise because of trait variation, there are still some interesting open questions for future theoretical study. In our models we have strictly limited ourselves to considering simple bi-trophic systems, whereas in nature such predator–prey pairs will be embedded in a larger food web. It is unclear how the presence of other species (e.g., top predators or competitors) affects the degree of intermittency exhibited by the population dynamics of the focal predator–prey pair. Additionally, to keep our analysis feasible, we also assumed a strict separation of scenarios and modes of adaptation. For example, in the coevolution models we always assumed identical modes of adaptation in prey and predators. In nature, different modes of adaptation may be found on different trophic levels (Yamamichi et al., 2019); such asymmetric adaptive scenarios are rarely studied theoretically (but see Bengfort et al., 2017, Yamamichi & Ellner, 2016), but may be particularly interesting in the context of intermittent cycles. Similarly, different trait scenarios may be combined, for example by including a gleaner–opportunist trade-off within a coevolution scenario. Finally, trait adaptation may co-occur with other mechanisms for intermittent cycles such as seasonal forcing, and their interplay can give rise to complex dynamics (Taylor et al., 2013). Investigating how different mechanisms may combine, and how their joint dynamics differ from those generated by each mechanism in isolation, could be a next step in providing a more comprehensive predictive theory on intermittent cycles in natural systems.

Although we show that the presence of intermittent cycles can arise from contemporary trait changes, our method does not offer a way to distinguish between the different scenarios that could be causing them, or between different mechanisms. Further analysis may identify features of intermittent cycles that are distinct between different mechanisms or different scenarios, allowing a more specific interpretation of observed intermittent cycles. The phase lag between prey and predator dynamics may be one such feature: preliminary results suggest that antiphase predator–prey cycles can be found

in scenario II, but may be less likely in the other scenarios (Appendix S4: Figure S6). Whether this will hold up a general pattern will be interesting to investigate further. The shape of the amplitude cycle may be another feature: for example, when cycles re-appear, does their amplitude increase gradually (Figure 6b) or do large-amplitude cycles appear very suddenly (Figure 7b)? Is the overall dynamic one of long periods of large-amplitude cycles punctuated by brief interruptions (Figure 6b), or long periods of near-stasis punctuated by brief bouts of large-amplitude cycles (Figure 7a)? Do the mean biomasses also change, or do they largely stay constant? The identification of the correct features, as well as the development of ways to detect them, will be helpful both in gaining a deeper fundamental understanding of intermittent cycles and in the interpretation of intermittency in empirical time series.

CONCLUSIONS

Although some recent studies have suggested that intermittent predator–prey cycles may be caused by contemporary prey or predator adaptation (Bengfort et al., 2017; Yamamichi et al., 2011), this idea has received little wider attention. A major reason for this is a lack of focused theoretical study: without this, it remains unclear what types of changes in prey or predator traits might cause intermittency, how likely they are to do so, and under what conditions this is expected to happen.

Here we show the potential for contemporary trait changes to drive intermittent cycles across a wide range of adaptive scenarios, and show that predator–prey coevolution is far more likely to drive intermittent cycles than adaptation in prey or predators alone. We further show a strong link between intermittency and fragility of trait variation; the risk of impending loss of intraspecific trait diversity may be of particular interest, as this is otherwise very difficult to notice and may have severe negative consequences. We therefore argue that intermittent cycles should receive more attention in general, and the novel method we developed here provides all the tools necessary for future investigations.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

DATA AVAILABILITY STATEMENT

Data are from previous publications and these publications have been cited appropriately here.

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