Spread dynamics of invasive species

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Species invasions are a principal component of global change, causing large losses in biodiversity as well as economic damage. Invasion theory attempts to understand and predict invasion success and patterns of spread. However, there is no consensus regarding which species or community attributes enhance invader success or explain spread dynamics. Experimental and theoretical studies suggest that regulation of spread dynamics is possible; however, the conditions for its existence have not yet been empirically demonstrated. If invasion spread is a regulated process, the structure that accounts for this regulation will be a main determinant of invasion dynamics. Here we explore the existence of regulation underlying changes in the rate of new site colonization. We employ concepts and analytical tools from the study of abundance dynamics and show that spread dynamics are, in fact, regulated processes and that the regulation structure is notably consistent among invasions occurring in widely different contexts. We base our conclusions on the analysis of the spread dynamics of 30 species invasions, including birds, amphibians, fish, invertebrates, plants, and a virus, all of which exhibited similar regulation structures. In contrast to current beliefs that species invasions are idiosyncratic phenomena, here we provide evidence that general patterns do indeed exist.

conservation | invasion | population dynamics | range expansion | regulation

Species invasions are a principal component of global change, given that they can cause habitat degradation, extinction of native flora and fauna, changes in ecosystem functioning, and facilitation of subsequent invasions that reinforce the aforementioned damage (1). The magnitude of the problem is such that invasive species threaten almost 60% of the species listed in the U.S. Endangered Species Act (1). Large economic impacts are also associated with many invasive species, which can provoke agricultural losses, disrupt ecosystem services, and lead to disease proliferation (2). Consequently, a prime objective of invasion theory is to understand and predict invasion success and patterns of spread (2). Since the early works of Fisher to the present (e.g., refs. 2–8) invasion theory has principally focused on identifying common features shared among invaders to explain their successful establishment outside their native ranges. In this regard, many alternative approaches have been explored, including theoretical models (reviewed in ref. 3), experimental studies (9), and the search for statistical associations between invasion success and attributes of the invading species or the invaded community (10). Despite the great advances achieved thus far, many aspects of real invasion dynamics continue to be poorly understood. In particular, commonalities among invaders have remained elusive, and idiosyncratic explanations of invasions have been more emphasized (11–13). Unfortunately, this lack of generality is of little use for control and management purposes, and a more profound understanding of the spreading process is urgently needed to develop wide-ranging strategies for coping with the impacts caused by biological invasions (2).

Although specific, taxa-independent characteristics that facilitate successful establishment and expansion have not been unambiguously identified, the invasion process itself has been observed to roughly follow the same sequence of stages regardless of the taxonomic identity of the invader: (*i*) an initial establishment phase with low spread, (*ii*) an expansion phase marked by increasing spread rates, and (*iii*) a saturation phase when spread rates reach a plateau (4). During the expansion phase, successful invaders present unregulated exponential growth in abundance until space becomes saturated (4). However, before saturation, invasion advancement can be weakened by biological constraints. This weakening may occur for several reasons, e.g., (*i*) individuals must grow to maturity at newly invaded sites before generating propagules for further invasion; (*ii*) Allee effects may constrain growth at low abundances (9, 14); and (*iii*) time delays associated with the production and dispersal of propagules potentially impose limits on totally unregulated spread (15, 16). The existence of a reproductive lag, such as that recently reported for *Spartina alterniflora*, illustrates a biological constraint that determines the observed pattern of invasion (17, 18). This type of lag might account for the existence of regulation mechanisms operating during spread; however, conditions for its existence have not yet been empirically demonstrated. Regulation of spread may arise as a consequence of the mechanisms mentioned above (e.g., reproductive lag), or other mechanisms that are yet to be explored, as long as the mechanism acts to establish a relationship between current and future spreading rates (19, 20). However, despite the fact that the scenario for regulation of spread dynamics is theoretically possible, conditions for its existence have not yet been empirically demonstrated.

The topic of population regulation has been the focus of an historical debate in ecology (density-dependent versus density-independent population dynamics), which has generated a strong theoretical framework, with a clear definition of regulation, and tools for its detection and analysis (19–24). Population regulation has been defined as the existence of a negative feedback in population abundance with an internal equilibrium condition (20, 24), that is, a negative slope for the relationship between the state variable (e.g., density and spread) and its rate of change, and a 0 rate of change for some value of the state variable. Different types of feedback structures are illustrated in Fig. 1 and explained in more detail elsewhere (see refs. 19, 20, and 24). In this article we assess the existence of these conditions and the functional structure of regulation in the spreading process. The detection of regulation and its structure is in practice estimated by means of a plot that relates the observed rate of change in the variable with the variable itself; following previous terminology, we will refer to these graphs as *R*-functions (20). This approach further allows for the discrimination of unregulated processes (e.g., exponential growth and random walks) from regulated ones (Fig. 1). For example, the *R*-function of deterministic exponential growth is a straight line with a 0 slope (Fig. 1*A*), and a random walk shows a cloud of points with a 0 slope and 0 intercept (Fig. 1*B*). In contrast, the *R*-function of a regulated

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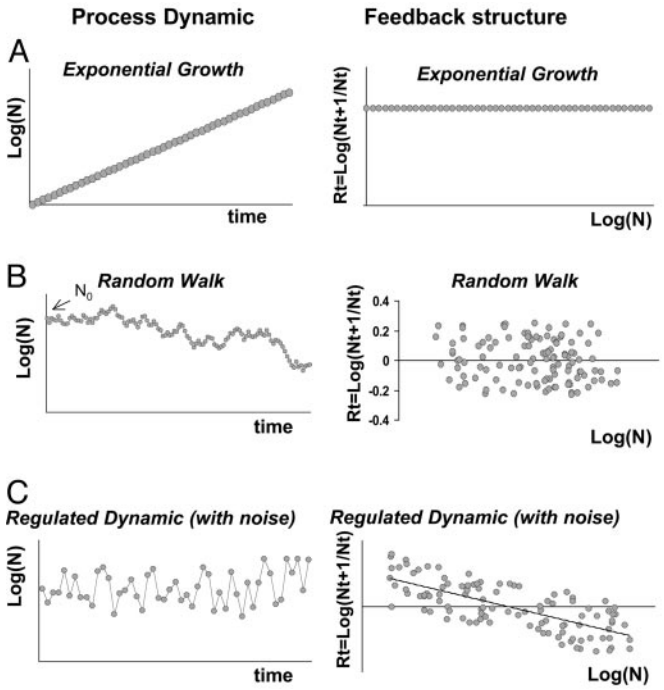

Fig. 1. Schematic representation of different dynamic processes (*Left*) and their associated feedback structures (i.e., *R*-functions) (*Right*). Unregulated processes such as exponential growth (*A*) and random walk (*B*) do not present a negative feedback structure that stabilizes the system at some equilibrium value, as is observed in a regulated process (*C*). Dynamics were generated from the following processes: exponential, $\log(N_{t+1}) = R + \log(N_t)$, where R is constant; random walk, $\log(N_{t+1}) = \log(N_t) + R_t$, where R is a set of random numbers taken from a normal distribution; and a regulated process, $N_{t+1} = N_t + R_t$, where $R_t = a - b\,N_t + e_t$, where e_t is a set of random numbers taken from a normal distribution.

process presents a negative slope that crosses the x axis (20), implying the existence of an equilibrium value in abundance (i.e., carrying capacity).

It should be highlighted that the observed dynamic depends not only on the equilibrium point, but also on the form of the regulation structure (R-function) and the effects of external variables that displace the population from equilibrium. Thus, populations with the same equilibrium value but different R-functions could exhibit different temporal dynamics and respond differently to external perturbations (see refs. 19 and 20). Thus, to understand a dynamic process it is important to analyze the existence of regulation as well as characterize the underlying R-function. Although this approach is currently used to analyze population abundance dynamics, here we use it to analyze the dynamics of new site colonization by invasive species. However, whereas population dynamics uses variations in growth rate to infer the density dependency, in invasion dynamics changes in spread rate represent "spread dependency."

In this article we look for evidence of regulation and its structure in the invasion process for a broad spectrum of taxa, encompassing a total of 30 well recorded cases of invasion that include birds, amphibians, fish, invertebrates, plants, and a virus. Our aim was to look for common features among invasions within a framework not previously explored, putting emphasis on the spreading process itself rather than on the invaders' biology or community aspects.

Methods

Empirical Databases. We used time-series data of the number of new sites invaded by a species per unit of time from well recorded invasion processes of 30 taxonomically distinct organisms (see Fig. 2). Databases included surveys made at different spatial scales. Taxa included 7 bird species taken from the Breeding Bird Survey Database (25) measured at the scale of routes (≈24.5 linear miles), 4 amphibian species, 6 invertebrates, 2 fish, and 10 plant species (including both aquatic and terrestrial plants). In these cases data were measured at the county level. Officials at the U.S. Geological Survey Nonindigenous Aquatic Species information resource graciously provided us with these databases. We also included the extraordinarily well documented epidemic of foot-and-mouth virus, which occurred in the United Kingdom during 2001 and was monitored by the Department for Environment, Food and Rural Affairs of the United Kingdom (DEFRA-UK) at the level of individual farms. These data are available upon request from DEFRA-UK. All of the time series were analyzed on an annual basis, with the exception of the frogs *Bufo marinus* and *Xenopus laevis* (analyzed on a biannual basis), the zebra mussel *Dreissena polymorpha* (analyzed on a monthly basis), and the foot-and-mouth virus (analyzed on a daily basis). Because our interest was focused on spread dynamics we concentrated on the periods of invasions corresponding to expansion phases.

Detection of Spread Regulation Structure in Empirical Databases. For each organism we calculated the per capita rate of invasion (S) as the mean number of new sites invaded (N) in a given time (t) from previously occupied sites, as $S_t = N_{t+1}/\Sigma_{t-5}^{t} N_i$. The variable S_t was log-transformed to remove the positive feedback inherent to ecological dynamics. The window of five time units was used to avoid underestimating S_t, given that invaded sites may become isolated (in the sense that they are no longer part of the leading front, because sites within the dispersal potential are already occupied). When long invasion time series are analyzed, this effect can strongly influence the estimated per capita invasion rate. For this reason, we assumed that, after five time steps, "infected" sites had invaded all possible "susceptible" sites. To estimate the parameters of the R-function for each invasion, we fitted the nonlinear model $R_t = A - CS_t^Q$, where A is a constant corresponding to the maximum R value that can be achieved, Q is a coefficient of curvature, and C is the slope when $Q = 1$. This function can be linear ($Q = 1$), concave ($Q < 1$), or convex ($Q > 1$). This model was preferred because it is flexible enough to detect a wide range of functional forms. When the parameter Q was not different from 1, the model became the linear equation $R_t = A - CS_t$. If invasion advancement is regulated, R must be 0 for some value of S and have a negative slope at this point; the magnitude of this slope and the general form of the R-function will account for the observed spread dynamic (refs. 19, 20, and 24; see Fig. 1).

Results and Discussion

Regulation functions for diverse taxa are presented in Fig. 2. Linear feedback structures with slope values in the range of $[-0.5, -1]$ were the most common patterns observed (Fig. 2). Estimated parameters, their statistical significance, and explained variance from the complete model are included within each plot. All fits were statistically significant ($P < 0.05$). The linear negative feedback observed in most cases indicates the existence of strong regulation, and the values of the slopes suggest an asymptotic approach toward an equilibrium rate of spread. This observed regulation means that, when stochastic effects cause spread to deviate from its equilibrium, this equilibrium is rapidly restored without damping oscillations (see ref. 19 for details). Moreover, in cases where the slope is closer to -1, deviations are compensated in a single time step. This type of regulation represents the strongest kind of regulation that can be observed. It should be noted that in population abundance dynamics this regulation structure without time lags is referred

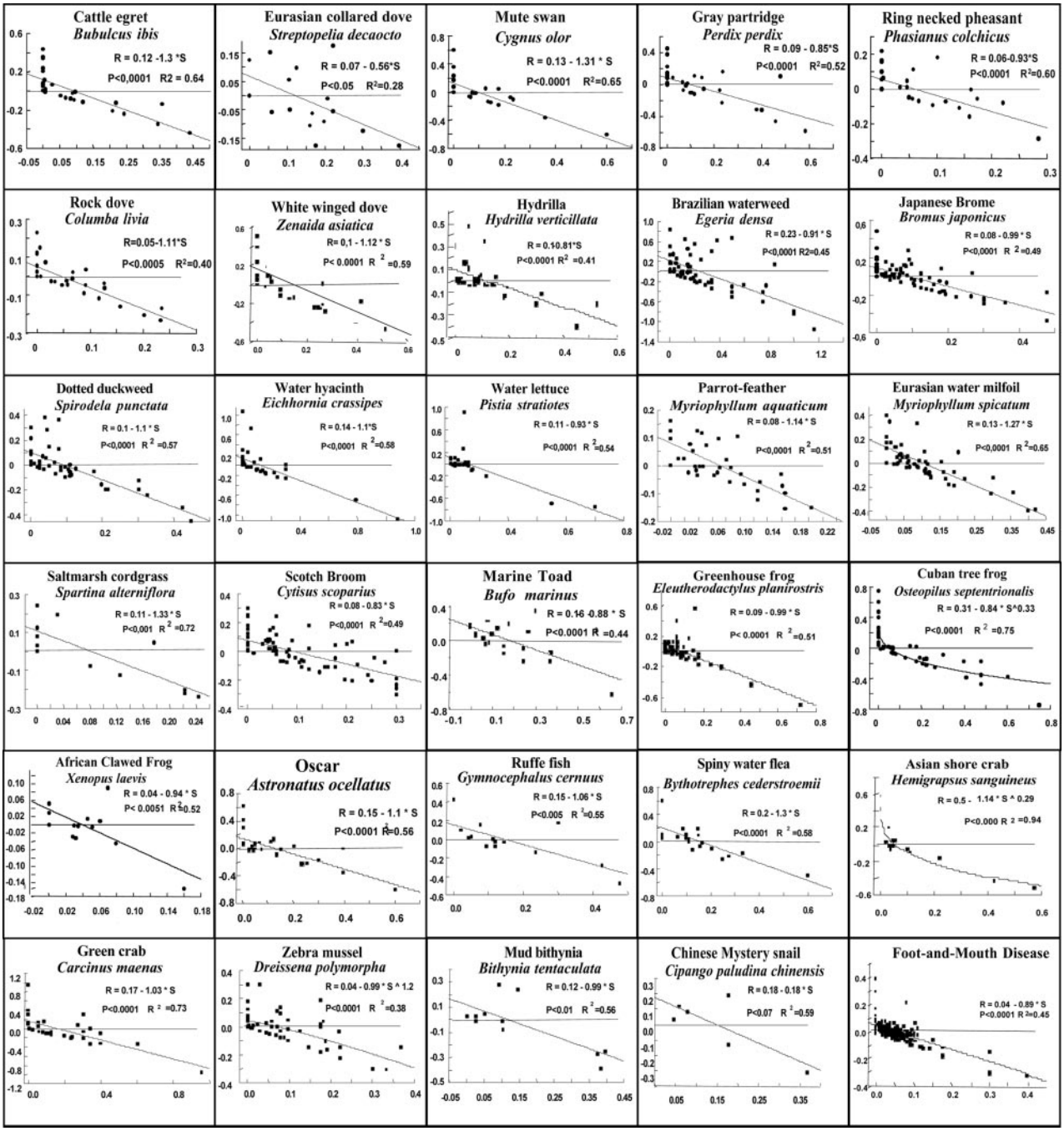

Fig. 2. Observed *R*-functions for the 30 taxonomically distinct organisms analyzed in this study. In all plots, the *y* axis is the rate of change in *S* (i.e., R_t), and the *x* axis is *S*, as in Fig. 1. Estimated parameters, their statistical significance, and explained variance from the complete model are indicated within each plot. All cases indicate strong regulation of spread. Only two cases were best described by nonlinear *R*-functions (far right column, rows four and five).

to as first-order dynamics and is typically the result of competition among individuals for a limited resource (20). A similar process could be invoked for spread dynamics, although the precise mechanisms operating on this process still need to be established.

Only two of the species analyzed were best described by markedly nonlinear *R*-functions (the Cuban tree frog *Osteopilus septentrionalis* and the Asian shore crab *Hemigrapsus sanguineus*). Interestingly, the parameters observed for these *R*-functions imply the existence of eruptive invasion dynamics. These dynamics originate from three attributes of their *R*-function. First, the function is very concave and the equilibrium point is very near to the origin. Second, the slope to the left of the equilibrium point is very steep. Third, the slope to the right of the equilibrium point is shallow. These features imply that negative deviations from equilibrium are largely overcompensated, forcing spread rate to very high values. In contrast, equilibrium is recovered at a slow rate over several time steps after positive deviations. This asymmetry in response to deviations from equilibrium can produce a huge explosion in advancement followed by periods of invasion fade-out tending toward equilibrium. Without knowledge of the regulation structure,

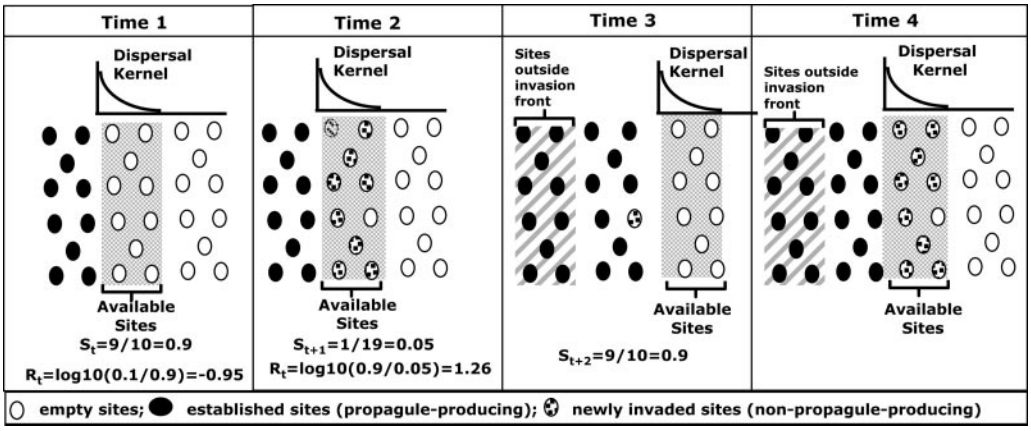

Fig. 3. Schematic of the proposed mechanism for spread regulation. The shaded area represents sites within the dispersal kernel. The semishaded area represents sites that are outside of the invasion front, because no empty sites are available within the dispersal kernel of these sites. The dispersal kernel depends on the age of invaded sites, because newly invaded sites do not produce propagules and, thus, do not contribute to the kernel. At $t = 1$ only those sites that are within the invader's dispersal kernel are truly available for colonization. If a large proportion of available sites are colonized (i.e., there is a large number of newly colonized sites at $t = 2$, and, thus, S_t is high), few empty sites can be colonized in the next time step (i.e., there are few newly colonized sites at $t = 3$), and so spread is reduced at $t = 2$. This dynamic is observed in the value of R_t, which is negative at $t = 1$, indicating a deceleration in spread. At $t = 3$ a large number of empty sites are again available for colonization, and a large number of new invasions are observed at $t = 4$, resulting in a high value of S_{t+2}. Consequently, R_{t+1} is now positive, indicating acceleration in spread. This sequence is repeated throughout the invasion process, producing ''sawtooth'' dynamics in invasion spread, which are also called linear first-order dynamics. This dynamic is detected in the *R*-function as a negative slope that is 0 for some value of spread, indicating the existence of regulated dynamics. Regulation occurs as a result of two mechanisms: (*i*) ''competition'' for empty sites, reinforced by (*ii*) the time lag between invasion and propagule production.

such dynamics may appear to be unregulated, but, in fact, it is the regulation structure itself that produces this phenomenology in invasion. This is a nontrivial result for management purposes because an invasion that is occurring close to equilibrium, if moved to an inferior rate in an attempt to control spread, will eventually respond with an overcompensation leading to a very high rate of spread. In the same way, stochastic perturbations can also lead to explosive spread dynamics.

Real populations are typically observed near equilibrium (20), making observations of spread values far from equilibrium very unlikely. Thus, our empirical findings of mainly linear spread dependencies do not exclude the possible existence of more complex underlying regulation structures (e.g., Allee effects, metastability; see ref. 20). This complex regulation could explain, for example, cases of spontaneous collapse when invading populations are pushed below the Allee threshold (4, 26). In the same way, populations can experience sudden explosions when pushed above this threshold (27). However, it is often difficult to completely reconstruct complex regulation structure from available databases for at least two reasons: (*i*) the state variable is rarely observed near unstable equilibria (20), and (*ii*) invasion events are frequently not documented until they enter a conspicuous expansion phase that calls the attention of interested parties (18). In fact, we made a theoretical analysis of invasions running in a lattice model, finding that different levels of nonlinearity in regulation structure are expected in real invasions.

Our analysis suggests that regulation in invasion advancement is a widespread phenomenon in nature and, more importantly, that the regulation structure is strikingly consistent among invasions. The spread of invading birds, amphibians, invertebrates, fish, plants, and a virus all exhibited similar negative feedback structures that stabilized their rate of spread. The invasion events analyzed here represent data collected from different temporal scales (i.e., biannual, annual, monthly, daily; corresponding to the specific biology of each species) and spatial scales (i.e., counties, routes, farms) for widely distinct taxonomic organisms inhabiting different environments, employing different dispersal mechanisms, and embedded in different communities and biological interactions. Despite these differences, most species presented an invasion pattern reminiscent of a first-order dynamic, which is expected under competition for a limiting resource (20). The colonization of accessible sites represents the depletion of limited resources, because these sites are not available for new invasions (16). New sites will be further colonized only after the population locally grows at invaded sites and starts to produce new propagules that can sustain the wave of advancement (Fig. 3) (15, 16). In other words, there is a nonreproductive leading front that delays spread advancement (28). In addition, first-order dynamics in invader spread are congruent with the enemy-release hypothesis (29, 30), where feedback structure does not involve predators or competitors that regulate invasion spread (14, 31–33), given that this kind of feedback produces higher-order dynamics (e.g., cycles; see refs. 31 and 32).

Because the shape of the feedback structure will be affected by interactions between invasive species and reactive or independent environmental variables, it is (in principle) possible to deduce all of these components from the analysis of its regulation structure. Furthermore, untangling the effects of environmental variables on spread requires first identifying its regulation structure and second understanding how this structure interacts with the climatic signal (19). If simple statistical associations are used, without any knowledge of the underlying regulating structure of the process, one may arrive at spurious conclusions. Nevertheless, there is a well developed framework for analyzing the effects of climate on population dynamics (19), and such an approach could greatly improve our understanding of the connection between environmental factors and invasions.

A principal aim in invasion biology is to find general patterns that would permit researchers and managers to predict the outcome of species introductions. Nevertheless, invasions continue to be considered idiosyncratic phenomena (11–13). This study suggests that general patterns do exist. The recognition of invasion spread as a regulated process and the notable consistency in observed *R*-functions open up an additional link between bioinvasions and population dynamics theory and thus the possibility of using the wide array of tools and concepts already

available to better understand patterns of invasion, its underlying mechanisms, and the role played by the environment.

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