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Influence of time delay and nonlinear diffusion on herbivore outbreak

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ABSTRACT

Herbivore outbreaks, a major form of natural disturbance in many ecosystems, often have devastating impacts on their food plants. Understanding those factors permitting herbivore outbreaks to occur is a long-standing issue in conventional studies of plant-herbivore interactions. These studies are largely concerned with the relative importance of intrinsic biological factors and extrinsic environmental variations in determining the degree of herbivore outbreaks. In this paper, we illustrated that how the time delay associated with plant defense responses to herbivore attacks and the spatial diffusion of herbivore jointly promote outbreaks of herbivore population. Using a reaction-diffusion model, we showed that there exists a threshold of time delay in plant-herbivore interactions; when time delay is below the threshold value, there is no herbivore outbreak. However, when time delay is above the threshold value, periodic outbreak of herbivore emerges. Furthermore, the results confirm that during the outbreak period, plants display much lower density than its normal level but higher in the inter-outbreak periods. Our results are supported by empirical findings.

1. Introduction

Herbivory is an important feeding process. By feeding on different plant parts or materials, herbivores can affect plant growth, transfers of nutrients to the soil surface, and habitat and resource conditions for other organisms (see the review of [58] and references therein). These effects are mostly viewed as beneficial outcomes of plant-herbivore interactions as it exerts positive effects for the maintenance and conservation of plant population structure and composition during non-outbreak periods. However, periodic outbreaks of herbivores can denude or kill plants over many square kilometers and led to changes in plant community structure and composition. This capacity to alter community structure allows herbivores to act as biotic agents of disturbance [69]. Unlike abiotic disturbances, herbivore outbreaks have continuous impact on plant population and hold selectivity for the feeder plant species. As a result, a core issue of herbivory is that a threshold at which it shifts from a normally acting process to a disturbance in terms of intensity, scale and frequency similar to those of fire, storm, drought or flood. In temperate forest, for example, at a normal level of herbivory, defoliating insects consume 5–15 percents of foliage production but it may reach to 100 percents during outbreak periods [50,39,9].

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Although many herbivore populations exhibit outbreak behavior, the main causes of these phenomena remain unclear [2]. Thus, determining which factors regulate or induce outbreak in herbivore populations continues to be an important issue in the filed of both ecological and agricultural research [66,8]. The previous work revealed that herbivore outbreak was caused by interactions with enemies [62,30,41,40,13], inducible defences or physiological stress [14,6,37,65,71,32,67], the case that herbivore population's parents and grandparents experienced in preceding generations [17], environmental forcing [3,15,26] and resource-limited [1,2]. The aim of this paper is to give another mechanism for the outbreak of the herbivore.

While plant-herbivore interactions exhibit cyclic population dynamics, the spatiotemporal outbreak patterns of herbivore population are often explained by those underlying causes of population synchrony in which peak phases of herbivore population cycle are inversely correlated with that of crash phases of plant population cycle. A suite of studies have attempted to link these periodic outbreak patterns with that of the large-scale processes such as climatic variation [46,48]. The other approaches focused on the analysis of the rate of spread of herbivores, seeking traveling wave solutions in the model of plant-herbivore interactions. In this case that rate of spread often depends on distributions and dispersal ranges of both populations [60,32]. Consequently, both spatial and temporal aspects play very important roles in the plant-herbivore population dynamics, which can not be ignored.

On the other hand, biological systems with time delays have been of considerable interest. It means the time between immature and mature [18], the time of pregnancy of the mother [31] and so on [16,49,38]. The interaction of plant and herbivore share some common features with prey-predator model. So delay widely exists in the dynamics of plant-herbivore interaction [10,51]. For example, the delay may arise between herbivore damage and deployment of inducible defenses. Moreover, theoretical analysis pointed out that time lags in reproduction influencing population dynamics [5,57,53,68].

Underwood et al. firstly demonstrated that time delays in inducible defenses and non-linear dispersal can lead to spatial instabilities [67]. However, it was a simple simulation study and thus, studies of the population consequences of time delay and the spatial spread of populations have remained largely unconnected. As a result, in the present paper, our aim is to build a better understanding of how time delay affects the dynamic behavior of herbivores in both space and time, including spatial pattern and herbivore fluctuation. We will analysis a reaction-diffusion model by addressing the following questions: (1) How do time delay influence herbivore cycles; (2) How do time delay and nonlinear diffusion term have effect on the spatial spread of the herbivore?

The paper is organized as follows. In Section 2, we use a reaction-diffusion model with delay to describe the interaction of plant and herbivore. In Section 3, by using both mathematical analysis and numerical simulation, we show that herbivore outbreak can be induced by time delay. Moreover, we show the relationship between wavelength and time delay. Finally, conclusions and discussions are presented in Section 4.

2. The model

Since that long time series of the density of both plant and herbivore is needed, it is difficult to identify the causes of the fluctuations empirically [35,1]. Thus, it may provide useful information by constructing mathematical models to explain the phenomenon observation in the real world.

Here, we introduce a simple reaction-diffusion model to analyze the effect of time delays in the induction of plant defenses on the outbreak and spatial distribution of herbivores in a closed, one-dimensional landscape. We model the level that inducible defenses have reached in each location at each time, which is dependent on herbivore density and the level of already induced defenses. We do not explicitly model changes in plant biomass since such changes are potentially uncorrelated with the effects of induction in empirical plant-herbivore systems [29]. Induced defenses increase in response to herbivore densities according to a saturating function such that there is a maximum amount α by which induction can change between t and $t+\Delta t$; the shape of the saturation curve is set by θ. Induction also exhibits self-limitation, tuned by β, which reduces the potential for increased induction in response to new damage. To reflect delays in the deployment of inducible defenses, we set induction changes at time t dependent on herbivore densities at $t-\tau$ time steps previously. Induction decays at a constant rate δ, reflecting the breakdown of inducible defenses and plant repair [68]. We assume that the herbivore population grows logistically in the absence of induced defenses and suffers a linear increase in mortality when defenses are activated. Furthermore, we assume that herbivores move away from areas of high induced defenses according to Fokker–Planck nonlinear diffusion, which is dependent on the density of both induction levels and herbivores.

On the basis of the above assumptions, we arrive at the following equations:

$$\frac{\partial I(x,t)}{\partial t} = (\alpha - \beta I(x,t))\frac{H^{\theta}(x,t-\tau)}{b^{\theta}+H^{\theta}(x,t-\tau)} - \delta I(x,t), \tag{1.a}$$

$$\frac{\partial H}{\partial t} = rH(x,t)\left[1-\frac{H(x,t)}{K}\right] - mI(x,t)H(x,t) + \frac{\partial^2}{\partial x^2}[(D_0+\chi I(x,t))H(x,t)], \tag{1.b}$$

where $I(x,t)$ and $H(x,t)$ represent induced defense and herbivore density in both space and time. The biological meanings of the parameters are given in Table 1.

Table 1
Parameter used in model (1).

Symbol	Comments
α	maximum induction rate per herbivore
β	per-unit reduction of induction rate by self-limitation
δ	per-unit induction decay rate
b	half-maximum for herbivore effectiveness of damage
θ	herbivore damage effectiveness shape tuning parameter
r	intrinsic rate of herbivore population growth
K	herbivore carry-capacity in the absence of inducible defenses
m	mortality rate by induction
D_0	baseline diffusion rate
χ	sensitivity of diffusion rate to induction
τ	time delay between herbivore damage and deployment of inducible defenses

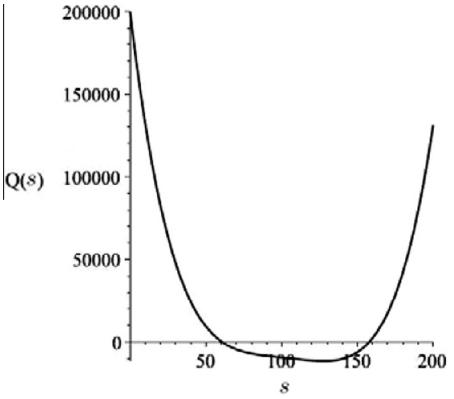

Fig. 1. Plot of $Q(s)$ as a function of s, and the expression of $Q(s)$ is in Eq. (2). Note that there are two pure imaginary solutions of $Q(s)$, which have no biological meanings.

3. Main results

In this section, we set that $\alpha = 200, \beta = 1, \delta = 0.75, b = 5, \theta = 3, r = 1, K = 10, m = 0.01, D_0 = 0.1, \chi = 0.1$, and to see what will occur as the time delay increases. The first step in analyzing the model is to determine the stationary point of the non-spatial model obtained by setting space derivatives equal to zero. It is, naturally, the dynamics in the biologically meaningful region $I \geqslant 0, H \geqslant 0$ that are of interest. By considering the nullclines $F(I,H) = 0$, $G(I,H) = 0$ (see from Appendix A), and the intersection of these curves in phase space, we have a stationary point $(0,0)$ which is a saddle,[1] and (I^*, H^*). Additionally, I^* is the solution of

$$Q(s) = 0.00175s^4 - 0.725s^3 + 112.5s^2 - 7843.75s + 200000 \tag{2}$$

and $H^* = 10 - 0.1I^*$. It can be see from Fig. 1 that there are positive stationary point. One is $(60,4)$, which is locally stable, and the other one does not exist by reason of the negative of H^*. Thus, from the biological point of view, we are interested to study the stability behavior of the interior equilibrium point $(60,4)$.

From the stability analysis in Appendix B and direct calculations, we know that when $\tau > \tau_c = 2.54980875$, Hopf bifurcation of the no-spatial model emerges. On the other hand, the positive equilibrium $(60,4)$ is stable when $\tau < \tau_c$. This is illustrated by the numerical simulation in Fig. 2 (with initial data $(70,2)$). Further, when delay passes through the critical value τ_c, the positive equilibrium $(60,4)$ loses its stability and the system goes into oscillations, see Fig. 3.

Now, we want to see the effect of time delay when spatial terms are added. In Fig. 4, the dispersal relation,[2] which can be obtained from Eq. (B.9) in Appendix B, is given when there is no time delay. It is easy to find that, when there is no time delay, there is no spatial pattern.

[1] The local stability of the stationary point can be checked by Eq. (A.3) in Appendix A.

[2] The dispersion relation can determine how time oscillations $e^{\lambda t}$ are linked to spatial oscillations $e^{i\kappa \vec{r}}$ (here, $\vec{r} = x$ (y) or $\vec{r} = (x,y)$. The dispersion relation can be described by the characteristic value-λ, with respect to wave number κ.

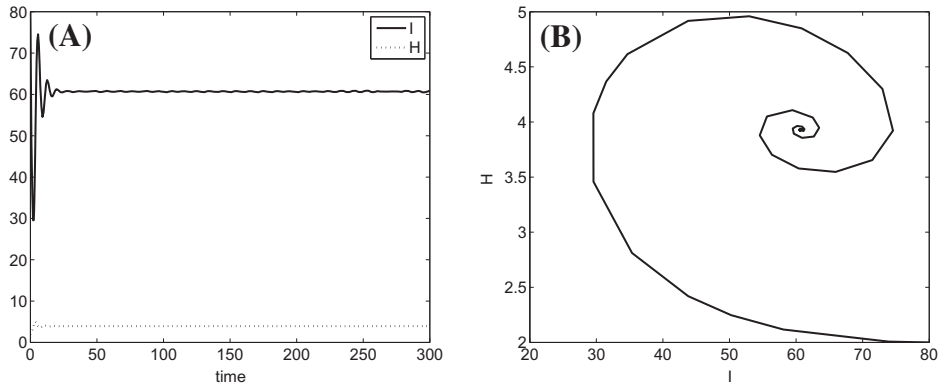

Fig. 2. When $\tau = 1 < \tau_c$, the positive equilibrium $(60, 4)$ is stable. Parameter values are used as: $\alpha = 200, \beta = 1, \delta = 0.75, b = 5, \theta = 3, r = 1$, $K = 10, m = 0.01, D_0 = 0$ and $\chi = 0$. (A) Time series of the induced defense and herbivore; (B) Phase diagram of induced defense and herbivore.

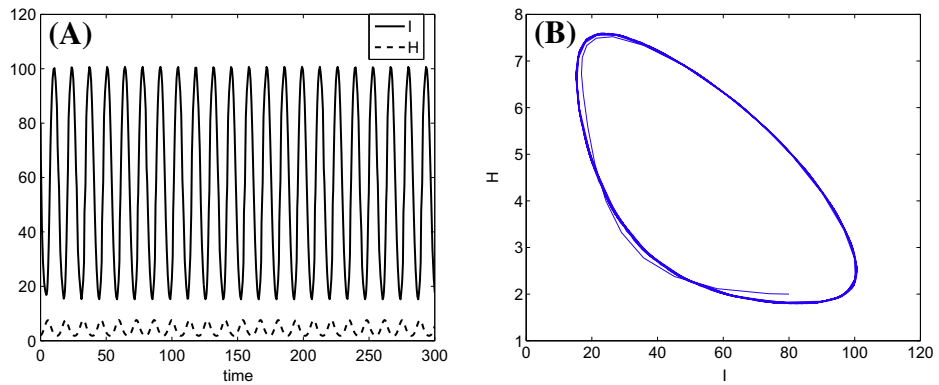

Fig. 3. When $\tau = 4 > \tau_c$, Hopf bifurcation occurs and leads periodic solution. Parameter values are used as: $\alpha = 200, \beta = 1, \delta = 0.75, b = 5, \theta = 3, r = 1$, $K = 10, m = 0.01, D_0 = 0$ and $\chi = 0$. (A) Time series of the induced defense and herbivore; (B) Phase diagram of induced defense and herbivore.

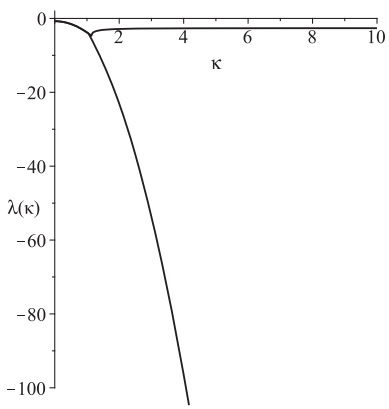

Fig. 4. Dispersal relation of the model (1) without time delay. Parameter values are used as: $\alpha = 200, \beta = 1, \delta = 0.75, b = 5, \theta = 3, r = 1, K = 10, m = 0.01$, $D_0 = 0.1, \chi = 0.1$ and $\tau = 0$. Note that the characteristic value is less than zero, which means there is no pattern.

3.1. Spatial pattern

In the following part, numerical solutions for the system (1) with both time delay and spatial terms in the one-dimensional space will be presented. In practice, the reaction-diffusion system is solved in a discrete domain. The space between the lattice points is defined by the lattice constant Δx. In the discrete system the Laplacian describing diffusion is calculated by using finite differences, i.e., the derivatives are approximated by differences over Δx. For $\Delta x \to 0$, the differences approach

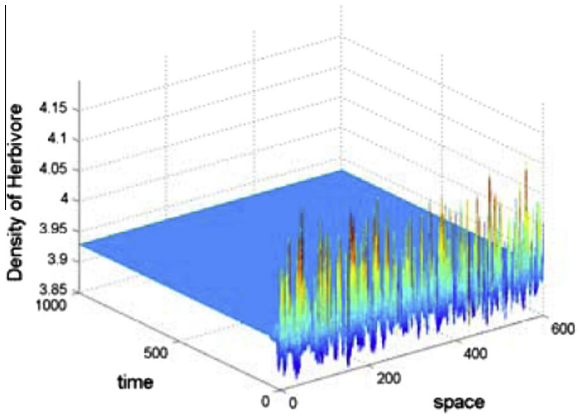

Fig. 5. Model solutions with $\alpha = 200, \beta = 1,\ \delta = 0.75, b = 5, \theta = 3, r = 1, K = 10, m = 0.01,\ D_0 = 0.1, \chi = 0.1,$ and $\tau = 1$. The initial condition is that $I(x,0) = 60$, and $H(x,0) = 4 + 10^{-7}(x - 300)$.

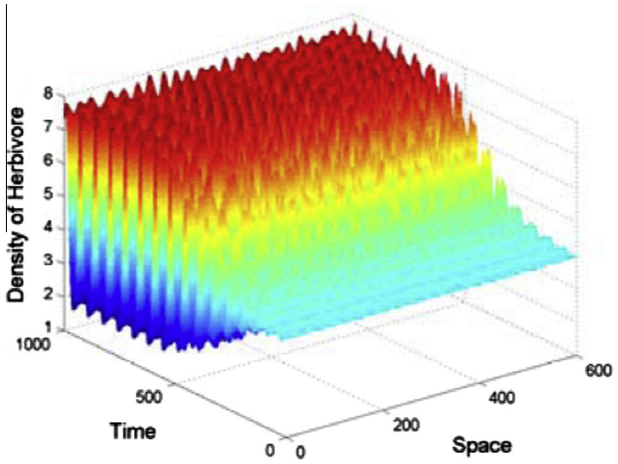

Fig. 6. Model solutions with $\alpha = 200, \beta = 1, \delta = 0.75, b = 5, \theta = 3, r = 1, K = 10, m = 0.01, D_0 = 0.1, \chi = 0.1,$ and $\tau = 4$. The initial condition is the same as in Fig. 5.

the derivatives. The time evolution is also discrete, i.e., the time goes in steps of Δt. The time evolution can be solved by using the Euler method, which means approximating the value of the concentration at the next time step on the basis of the change rate of the concentration at the previous time step. In our calculations, the parameters values were taken to be $\Delta x = 1$, $\Delta t = 0.01$ and the space was discretized to 1×600 lattices on the one-dimensional space. The periodic boundary condition was used in the simulation. We also find that when we vary the number of mesh points such as 1×1000 and 1×300, the results of system (1) have the same dynamical behaviors. At the same time, it was checked that a further decrease of the step values did not lead to any significant modification of the results.

In Fig. 5, we show the solution with respect to the space and time with $\tau < \tau_c$. One can see that the solutions are stable and converge to constant. However, as time delay increases as the case $\tau > \tau_c$, the model (1) has period solutions which is shown in Fig. 6. As the time delay further increases, the amplitude of the oscillations becomes larger, see in Fig. 7. In a word, time delay plays a constructive role in the pattern formation of the herbivore, i.e., induce periodic pattern.

3.2. Synchronization and outbreak

Synchronization is a fundamental phenomenon arising in many biological and physical contexts for which there are two or more coupled oscillating systems. In the classical sense, and dating back at least to Huygens in the 17th century [27], synchronization has been understood as the mutual adjustment of periodic oscillators and the frequency locking that results because of their (often weakly) coupled interaction [63]. Synchrony has also been observed among populations of sympatric species that are not directly linked through trophic interactions, such as herbivorous forest insects [42,25,45,55,33] and allopatric large herbivores [54].

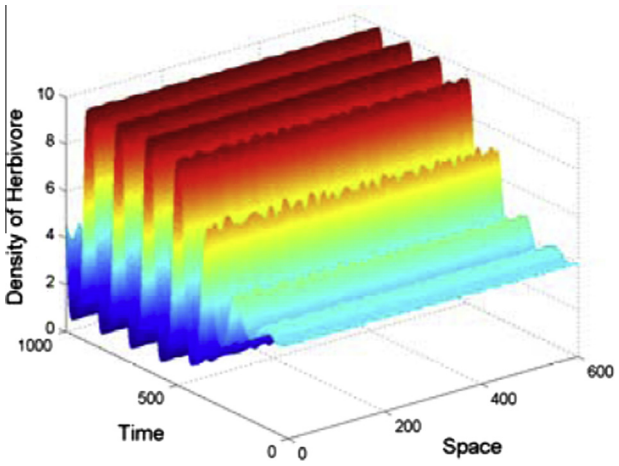

Fig. 7. Model solutions with $\alpha = 200, \beta = 1, \delta = 0.75, b = 5, \theta = 3, r = 1, K = 10, m = 0.01, D_0 = 0.1, \chi = 0.1$, and $\tau = 10$. The initial condition is the same as in Fig. 5.

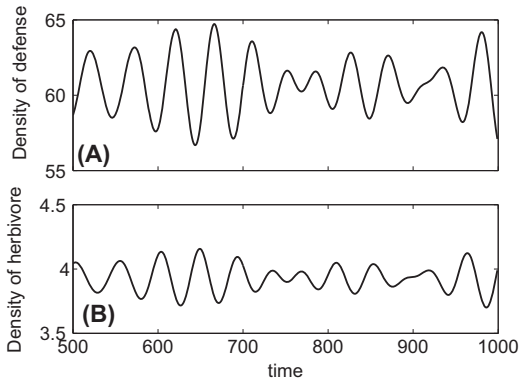

Fig. 8. Synchrony of the induced defense and the herbivore with $\tau = 2.5$. Parameter values are used as: $\alpha = 200, \beta = 1, \delta = 0.75, b = 5, \theta = 3, r = 1, K = 10, m = 0.01, D_0 = 0.1$ and $\chi = 0.1$.

In Figs. 8–10, we show the time series of the induced defense and herbivore as time delay increases. One can see from these three figures, the induced defense and the herbivore are anti-phase synchrony. When the time delay is smaller than but very close to the critical value, both the two population exhibit fluctuation behavior. When it is becoming larger than the critical value, periodic solutions with fixed period emerge, see Figs. 9 and 10.

To well explain the cause of herbivore outbreak, we plot the time series of the two population when τ is less than τ_c in Fig. 11. When τ is small, the density of induced defense and herbivore converge to a constant for a long time. In other words, there is no outbreak for small τ. As seen from Fig. 8, when τ is larger than the critical values, fluctuation of herbivore is shown. As the time delay further increases, the oscillation aptitude and period becomes larger by comparing with Fig. 9 and 10. We can conclude that herbivore outbreak is induced by time delay. Moreover, Figs. 8–10 show that the density of induced defense is very low in the outbreak period, but high in the inter-outbreak period, which confirm the previous argument [34].

3.3. Wavelength

Investigating the relationship between spatial patterns in population densities and environmental heterogeneity is crucial to the understanding of population dynamics and for the management of species in communities. Consequently, determining the wavelength for the spatial pattern is a key issue in the field of spatial ecology [61,70]. In other words, we can see the distribution of the population in the spatial space directly by calculating the wavelength.

In order to see the effect of time delay, we set τ as a parameter, and show the corresponding wavelength as it varies in Fig. 12. It can be found that the time delay increase the wavelength of the spatial pattern, and wavelength reaches the maximum values when $\tau \approx 3.5$. However, when τ further increases, the wavelength is a decreasing function of it. From the

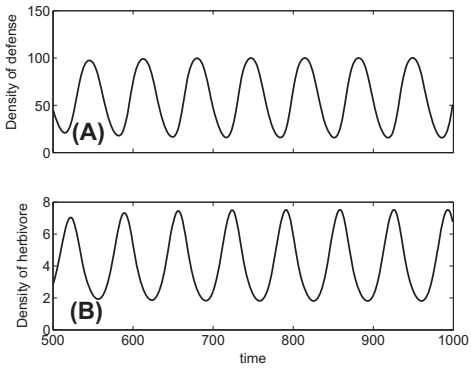

Fig. 9. Synchrony of the induced defense and the herbivore, and the parameters are the same as in Fig. 6.

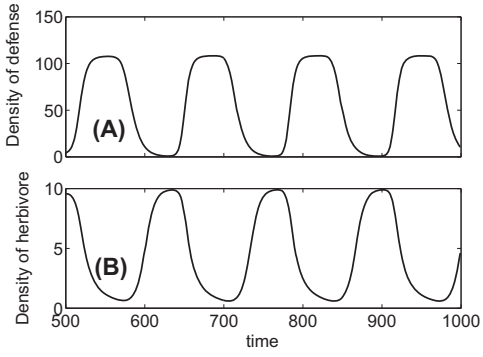

Fig. 10. Synchrony of the induced defense and the herbivore and the parameters are the same as in Fig. 7.

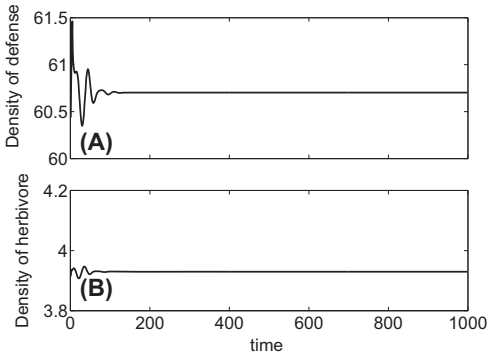

Fig. 11. Time series of the induced defense and the herbivore with $\tau = 1$. Parameter values are used as: $\alpha = 200$, $\beta = 1, \delta = 0.75, b = 5, \theta = 3, r = 1, K = 10$, $m = 0.01, D_0 = 0.1$ and $\chi = 0.1$.

biological point of view, the increase of time delay can largely affect the distance between one peak distribution and the adjacent one.

4. Discussion and conclusion

In this paper, we present a reaction-diffusion model described by interaction of plant and herbivore with delay. The results showed that the time delay can induce the herbivore outbreak (cf. Figs. 8–10). Moreover, herbivore outbreak in our model is characterized by high density of induced defense during the inter-outbreak period, which is consistent with the experimental data [34,36,56,59]. In addition to that, we show that time delay may have great effect on the wavelength of the spatial pattern (cf. Fig. 12). We can see from the figure that there exist a threshold value on time delay that the

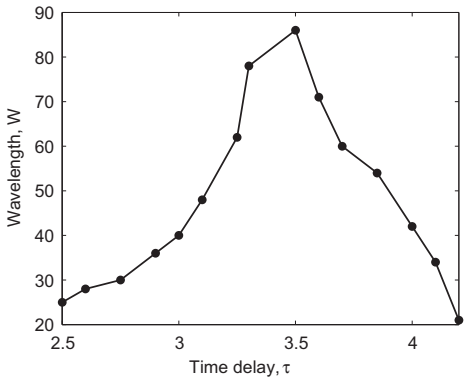

Fig. 12. An illustration of the variation in pattern wavelength with different τ.Parameter values are used as: $\alpha = 200$, $\beta = 1, \delta = 0.75, b = 5, \theta = 3, r = 1, K = 10$, $m = 0.01, D_0 = 0.1$ and $\chi = 0.1$.

wavelength reaches its maximum value. In summary, our results identify some biological features of plant-herbivore dynamics that determines whether the outbreak occurs or not and suggest the further investigation should take into account the effects of both time delay and spatial diffusion.

These above-noted model results are consistent with the empirical findings in birch forest regions, where defoliating insect outbreaks are one of the most common disturbances. Along the Scandinavian mountain chain, some birch forest areas are usually experienced with periodic, synchronous peaks of Geometrid defoliators Epirrita (Oporinia) autumnata and Operophetera spp. [64]. Their outbreak cycles are characterized by large amplitude with 1000 to 100,000 fold differences between population lows and peaks [24,28,7], which is clearly larger than in the well known vole cycles with 25–250 fold differences [21]. It has been observed that although the period of the cycle is remarkably stable, the peak densities show large variation even within a single site (see Neuvonen et al., 2001 and references therein). Detailed investigation of these patterns revealed that the time delay associated with inducible plant defenses, which refers to decreasing foliage quality in the years following insect attacks, is one of the major factors behind such cyclic outbreak patterns [24,22,23,47].

An interesting question of interest is whether nonlinear diffusion terms have effect on the herbivore outbreak. To address this issue, we performed extensive computer simulations for different parameter values of D and χ (in total, more than 50 parameter sets were examined). We found that the results nonlinear diffusion just influence the density not the outbreak (for the sake of brevity, we do not provide separated figures).

The present paper provides some new insights into the spatial ecology of interaction of plant and herbivore, but it also leaves many questions open for the future investigations. We would like to emphasize two relevant topics where may be the main subjects of the further research. Firstly, in the model (1), we consider the case that only herbivores diffuse in the space. However, for some plants their weeds also can move in the space caused by purely environmental factors such as wind. In that situation, the model we employ may be changed as

$$\frac{\partial I(x,t)}{\partial t} = (\alpha - \beta I(x,t))\frac{H^{\theta}(x,t-\tau)}{b^{\theta} + H^{\theta}(x,t-\tau)} - \delta I(x,t) + D_I \frac{\partial^2}{\partial x^2} I(x,t), \tag{3.a}$$

$$\frac{\partial H}{\partial t} = rH(x,t)\left[1 - \frac{H(x,t)}{K}\right] - mI(x,t)H(x,t) + \frac{\partial^2}{\partial x^2}[(D_0 + \chi I(x,t))H(x,t)], \tag{3.b}$$

where D_I is the diffusion coefficient of the plant. Secondly, we found that periodic wave solution in the model (1) (cf. Figs. 6 and 7). However, previous work revealed that traveling wave of herbivore may exist by modeling the interaction of plant and herbivore [44]. Thus, we need to check whether model (1) have traveling wave solution, especially how time delay influence such solution.

It also should be noted that, in our model simulations, we have considered all the parameters as constants. However, herbivore populations are sometimes considered to be strongly influenced by abiotic factors such as weather and climatic conditions [3,43,11,12,19]. As a result, all the parameters can show temporal and spatial variations: indeed, some can be both stochastic and show significant seasonal variations [20,52,4]. We hope that our efforts will provide a new starting point for the analysis of more detailed models to understand the outbreak in plant-herbivore dynamics.

Acknowledgments

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Appendix A. Linear stability of the no-spatial model

For sake of simplicity, we rewrite the system (1) as following:

$$\frac{\partial I}{\partial t} = F(I,H), \tag{A.1a}$$

$$\frac{\partial H}{\partial t} = G(I,H) + \frac{\partial^2 P}{\partial x^2}. \tag{A.1b}$$

Denote the constant equilibrium solution as (I^*, H^*), i.e.,

$$F(I^*,H^*) = 0, \quad G(I^*,H^*) = 0. \tag{A.2}$$

We are aim to look for the conditions so that (I^*, H^*) is stable for the no-spatial version of (1) and is unstable for the spatial version of (1). We always assume that (I^*, H^*) is linearly stable with respect to perturbation of I and H, then the eigenvalues of the Jacobian

$$J = \begin{pmatrix} \frac{\partial F}{\partial I} & \frac{\partial F}{\partial H} \\ \frac{\partial G}{\partial I} & \frac{\partial G}{\partial H} \end{pmatrix} \triangleq \begin{pmatrix} a_{11} & a_{12} \\ a_{21} & a_{22} \end{pmatrix}$$

at (I^*, H^*) must have negative real parts, which is equivalent to

$$\mathtt{tr}(J) = a_{11} + a_{22} < 0, \quad \mathtt{Det}(J) = a_{11}a_{22} - a_{12}a_{21} > 0. \tag{A.3}$$

Appendix B. Dispersal relation

The main goal is to study the instability for the system (1) with the nonlinear diffusion. To deal with the nonlinear terms, we use the method of transformation variable. Define the new variables as $P = P(I,H)$. Then we have the following system with three equations:

$$\frac{\partial I}{\partial t} = F(I,H), \tag{B.1a}$$

$$\frac{\partial H}{\partial t} = G(I,H) + \frac{\partial^2 P}{\partial x^2}, \tag{B.1b}$$

$$P = P(I,H). \tag{B.1c}$$

It is easy to see that the instability of (I^*, H^*) in the system (1) is equivalent to that of (I^*, H^*, P^*) in the system (B.1) where $P^* = P(I^*, H^*)$. Let $u = I - I^*, h = H - H^*$ and $p = P - P^*$ be a spatial perturbation at the equilibrium (I^*, H^*, P^*), then linearizing the reaction-diffusion system (B.1) at (I^*, H^*, P^*) yields

$$\frac{\partial u}{\partial t} = a_{11}u(x,t) + a_{12}h(x,t-\tau), \tag{B.2a}$$

$$\frac{\partial h}{\partial t} = a_{21}u(x,t) + a_{22}h(x,t) + \frac{\partial^2 p}{\partial x^2}, \tag{B.2b}$$

$$p(x,t) = a_{31}u(x,t) + a_{32}h(x,t), \tag{B.2c}$$

where

$$a_{31} = \frac{\partial P(I,H)}{\partial I}|_{(I^*,H^*)}, \qquad a_{32} = \frac{\partial P(I,H)}{\partial H}|_{(I^*,H^*)}. \tag{B.3}$$

In order to examine the linear stability of (I^*, H^*, P^*) of system (B.1), we linearize the dynamic model (B.2) around the spatially homogeneous fixed point $(0,0,0)$ for small space- and time-dependent fluctuations and expand them in Fourier space:

$$(u,h,p)^T = (A_1, A_2, A_3)^T e^{\lambda t + i\kappa r}, \tag{B.4}$$

which yields

$$A_1\lambda e^{\lambda t + i\kappa r} = (A_1 a_{11} + A_2 a_{12} e^{-\lambda\tau}) e^{\lambda t + i\kappa r}, \tag{B.5a}$$

$$(A_2\lambda + A_3\kappa^2)e^{\lambda t+i\kappa r} = (A_1a_{21} + A_2a_{22})e^{\lambda t+i\kappa r}, \tag{B.5b}$$

$$A_3e^{\lambda t+i\kappa r} = (A_1a_{31} + A_2a_{32})e^{\lambda t+i\kappa r}. \tag{B.5c}$$

Since $e^{\lambda t+i\kappa r}\neq 0$, (B.5) is equivalent to the following linear algebraic equations

$$\begin{pmatrix} \lambda - a_{11} & -a_{12}e^{-\lambda\tau} & 0 \\ -a_{21} & \lambda - a_{22} & \kappa^2 \\ -a_{31} & -a_{32} & 1 \end{pmatrix}\begin{pmatrix} A_1 \\ A_2 \\ A_3 \end{pmatrix} = \begin{pmatrix} 0 \\ 0 \\ 0 \end{pmatrix}. \tag{B.6}$$

Nontrivial solutions to the above equations (B.6) exist if

$$\mathrm{Det}\begin{pmatrix} \lambda - a_{11} & -a_{12}e^{-\lambda\tau} & 0 \\ -a_{21} & \lambda - a_{22} & \kappa^2 \\ -a_{31} & -a_{32} & 1 \end{pmatrix} = 0, \tag{B.7}$$

which equals that

$$\mathrm{Det}\begin{pmatrix} \lambda - a_{11} & -a_{12}e^{-\lambda\tau} \\ -a_{21} + a_{31}\kappa^2 & \lambda - a_{22} + a_{32}\kappa^2 \end{pmatrix} = 0. \tag{B.8}$$

Note that (B.8) can be solved, yielding the characteristic polynomial of the original problem:

$$\lambda^2 - (a_{11} + a_{22} - a_{32}\kappa^2)\lambda + a_{11}a_{22} - a_{12}a_{21}e^{-\lambda\tau} - a_{11}a_{32}\kappa^2 + a_{12}a_{31}e^{-\lambda\tau}\kappa^2 = 0. \tag{B.9}$$

Appendix C. Critical value of time delay

We firstly pay attention to the model without spatial terms. Thus, the corresponding characteristic equation is that:

$$\lambda^2 - (a_{11} + a_{22})\lambda + a_{11}a_{22} - a_{12}a_{21}e^{-\lambda\tau} = 0. \tag{C.1}$$

We know that $iw(w > 0)$ is a root of Eq. (C.1) if and only if w satisfies the following equation:

$$-w^2 - (a_{11} + a_{22})iw + a_{11}a_{22} - a_{12}a_{21}(\cos w\tau - i\sin w\tau) = 0. \tag{C.2}$$

Separating the real and imaginary parts, one can have

$$(a_{11} + a_{22})w = a_{12}a_{21}\sin w\tau, \tag{C.3a}$$

$$-w^2 + a_{11}a_{22} = a_{12}a_{21}\cos w\tau. \tag{C.3b}$$

By squaring and adding the two parts of Eq. (C.3), it follows that

$$w^4 + (a_{11}^2 + a_{22}^2)w^2 + a_{11}^2a_{22}^2 - a_{12}^2a_{21}^2 = 0. \tag{C.4}$$

Suppose that $a_{11}a_{22} > a_{12}a_{21}$, then (C.4) have the solution:

$$w^2 = \frac{-(a_{11}^2 + a_{22}^2) + \sqrt{(a_{11}^2 + a_{22}^2)^2 - 4(a_{11}^2a_{22}^2 - a_{12}^2a_{21}^2)}}{2} \triangleq \eta. \tag{C.5}$$

If we define

$$\tau_j = \frac{1}{\sqrt{\eta}}\left[\arccos\frac{-\eta + a_{11}a_{22}}{a_{12}a_{21}} + 2j\pi\right], \quad j = 0, 1, 2, \cdots, n, \tag{C.6}$$

then Eq. (C.1) with $\tau = \tau_j$ has a pair of purely imaginary roots $\pm iw$, and it is easy to see that $\tau_0 < \tau_1 < \cdots < \tau_n$.

It can be found that (C.1) that

$$\left(\frac{d\lambda}{dt}\right)^{-1} = \frac{a_{11} + a_{22} - 2\lambda}{a_{12}a_{21}\lambda e^{-\lambda\tau}} - \frac{\tau}{\lambda} \tag{C.7}$$

and thus

$$\mathrm{Re}\left(\frac{d\lambda}{dt}\right)^{-1}|_{\tau=\tau_j} = \frac{a_{11}^2 + a_{22}^2 + 2w^2}{a_{12}^2a_{21}^2} > 0. \tag{C.8}$$

In other words, for the model (1) without spatial terms, the Hopf bifurcation will occur at $\tau = \tau_j$, and period solution will emerge when $\tau > \tau_j$.

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