Commun Nonlinear Sci Numer Simulat 19 (2014) 1507–1518

Contents lists available at ScienceDirect

Commun Nonlinear Sci Numer Simulat

journal homepage: www.elsevier.com/locate/cnsns

Influence of time delay and nonlinear diffusion on herbivore outbreak

Gui-Quan Sun [a,b,c,*], Amit Chakraborty [c], Quan-Xing Liu [c], Zhen Jin [a,b], Kurt E. Anderson [d], Bai-Lian Li [c]

[a] Complex Sciences Center, Shanxi University, Taiyuan 030006, PR China
[b] School of Mathematical Sciences, Shanxi University, Taiyuan 030006, PR China
[c] Ecological Complexity and Modeling Laboratory, Department of Botany and Plant Sciences, University of California, Riverside, CA 92521-0124, USA
[d] Department of Biology, University of California, Riverside, CA 92521, USA

ARTICLE INFO

Article history:
Received 9 February 2013
Received in revised form 20 August 2013
Accepted 16 September 2013
Available online 26 September 2013

Keywords:
Herbivore-plant
Time delay
Spatial diffusion
Outbreak
Synchrony

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].

* Corresponding author at: Complex Sciences Center, Shanxi University, Taiyuan 030006, PR China.
E-mail addresses: gquansun@126.com (G.-Q. Sun), amitc@ucr.edu (A. Chakraborty), q.liu@nioo.knaw.nl (Q.-X. Liu), jinzhn@263.net (Z. Jin), kurta@ucr.edu (K.E. Anderson), bai-lian.li@ucr.edu (B.-L. Li).

1007-5704/$ - see front matter Crown Copyright © 2013 Published by Elsevier B.V. All rights reserved.
http://dx.doi.org/10.1016/j.cnsns.2013.09.016

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.