Emergence of Donor Control in Patchy Predator–Prey Systems

J. CH. POGGIALE

Centre d'Océanologie de Marseille,
Université d'Aix-Marseille II,
13288 Marseille cedex 9,
France

E-mail: poggiale@com.univ-mrs.fr

J. MICHALSKI

Department of Mathematics,
Swiss Federal Institute of Technology,
1015 Lausanne,
Switzerland

R. ARDITI

Institut National Agronomique Paris-Grignon,
75231 Paris cedex 05,
France

Ecologie des Populations et Communautés,
URA 2154, Bâtiment 362,
Université Paris-Sud XI,
91405 Orsay cedex,
France

We study a general predator–prey system in a spatially heterogeneous environment. The predation process, which occurs on a behavioural time-scale, is much faster than the other processes (reproduction, natural mortality and migrations) occurring on the population dynamics time-scale. We show that, taking account of this difference in time-scales, and assuming that the prey have a refuge, the dynamics of the system on a slow time-scale become donor-controlled. Even though predators may control the prey density locally and on a behavioural fast time-scale, nevertheless, both globally and on a slow time-scale, the prey dynamics are independent of predator density: the presence of predators generates a constant prey mortality. In other words, in heterogeneous environments, the prey population dynamics depend in a switch-like manner on the presence or absence of predators, not on their actual density.

© *1998 Society for Mathematical Biology*

1. INTRODUCTION

The majority of predator–prey dynamic models considered in the ecological literature have the following general form:

$$\frac{dN}{dT} = f(N)N - g(N, P)P, \tag{1a}$$

$$\frac{dP}{dt} = eg(N, P)P - \mu P, \tag{1b}$$

where N and P are, respectively, the densities of prey and predators, $F(N)$ is the prey growth rate in the absence of predation and μ is the food-independent predator mortality rate, which is assumed to be a constant. The trophic function $g(N, P)$ (also called the functional response) is the amount of prey eaten per predator in a unit of time. In the predator equation (1b), $eg(N, P)$ describes the per capita predator production (the numerical response), where e is the trophic efficiency. The form of (1) assumes that the growth rate of predators is proportional to the predators' impact on the prey growth rate. Although not all models make this hypothesis (e.g., Getz[8] 1991[8] Berryman[4]*et al.*[4] 1995[4] see also Michalski[18]*t al.*[17]1997)[7] we will consider a model of type (1).

In most predator–prey models the functional response is assumed to be a function of the prey density only (prey-dependent predation): $g(N, P) = g(N)$. This reflects the assumption that the predator density does not have any direct effect on the instantaneous individual feeding rate. One of the consequences of this assumption is that the equilibrium prey density [determined directly from the equilibrium solution of the predator equation (1b)] does not depend on the prey growth function, $f(N)$. In particular, changes in the prey-carrying capacity do not affect the prey equilibrium density—they are only followed by changes in the predator equilibrium density. Thus, in such systems, the predators control the prey density and the dynamics of the lower trophic level depend entirely on trophic activities of the higher trophic level. A well-known predator–prey model of this kind is that with the Holling type II functional response. Models with prey-dependent trophic function $g(N)$, generalized to trophic chains of several trophic levels, demonstrate top–down trophic cascades: the top predator trophic level can directly depress the level below it; such depression then cascades indirectly as the lower trophic level is released from consumptive pressure and increases in biomass. The trophic cascades lie at the foundations of the 'green world' hypothesis (Hairston[9]*et al.*[9] 1960)[9] and it has been suggested that they occurred in several aquatic systems [see Strong[19]1992)[19]for review]. However, top–down trophic cascades are not common in nature: the large majority of them have algae at the base, and most are in isolated, discrete freshwater habitats, streams and mesotrophic lakes (Strong[19]1992)[19]

The alternative to top–down control is bottom–up (or donor) control. The term 'donor control' was first used by Pimm[18]1982)[18]in relation to predator–prey systems

in which the donor (prey) controls the density of the recipient population (predator) but not the reverse. Mathematically, this means that the prey equation does not depend explicitly on predator density. In such systems, equilibrium prey density directly follows changes in the prey-carrying capacity, which is in contrast to systems dominated by top–down control, such as models with prey-dependent predation. A classical example of donor control is a predator–prey system in which the predators kill mainly prey individuals likely to die because of other reasons, including old age and starvation (Pimm,[1]1982).[8]

In the ecological literature, the term donor control has been extended to a very wide meaning. In general, donor control refers to direct or indirect dependence of community structure (abundance, distribution, diversity) on factors producing variation at lower trophic levels (Menge,[1]1992).[6] In this paper, however, we restrict ourselves to the following mathematical definition of donor control: a predator–prey system is donor-controlled if the prey equation does not depend explicitly on the predator density.

Arditi and Ginzburg[1](1989)[1]argued that the trophic function should be calculated on the slow time-scale of the population dynamics at which predator–prey models of type (1) operate—not on the fast behavioural time-scale. The trophic function on the slow time-scale may be completely different from the behavioural response on the fast time-scale (Hanski,[10]1991).[9] In this paper, we demonstrate rigorously how, on the slow time-scale, apparent donor control emerges from a detailed model with spatial heterogeneity and two time-scales. In particular, we show that, when predators are very effective and the prey can take refuge, the global predator–prey system on the slow time-scale exhibits donor-controlled dynamics. Then, the presence of the predators has a dramatic depressing effect on the prey abundance but the prey dynamics are apparently independent of the predator dynamics. In heterogeneous environments, the slow time-scale prey dynamics strongly depend only on the presence or absence of very effective predators, not on their actual density.

In the next section, we present the biological rationale that underlies the assumptions of our model. Then in Section 3, we propose a model that fulfils these assumptions. The model contains large and small terms in order to take into account the different time-scales on which different processes take place. The feeding rate on the fast behavioural time-scale is assumed to depend on the local prey density alone. In Section 4 we use perturbation theory to demonstrate that, on the slow time-scale, the dynamics of predators depend on that of the prey and that the prey dynamics become apparently independent of predator dynamics. In Section 5 we show by example how an increase in predation strength can change the behaviour of the predator–prey dynamics from those determined by top–down effects to those controlled from the bottom. In Section 6 we generalize our results of Section 4 by showing that, under a weak condition on the behavioural feeding rate, prey refuges and strong predation always lead to apparent donor control on the slow time-scale. Section 7 is devoted to a comparison between donor control and ratio-dependence in the context of this paper. We discuss our results in the final section.

2. BIOLOGICAL RATIONALE OF THE MODEL

In previous papers (Auger and Poggiale[3] 1996[3] Michalski[1]*et al*[1,7] 1997)[7] we presented a method of obtaining population-level dynamic models from models at a detailed, behavioural level of description. In Michalski[1]*et al*.[1](1997)[1] we considered a multi-patch predator–prey model, the dynamics of which contained two time-scales: a fast one, associated with migrations between patches, and a slow one, on which interactions, reproduction and mortality occur. The methods of perturbation theory enabled us to show how the details of individual behaviour on the fast time-scale determine the population dynamics on the slow time-scale. We showed how the aggregated functional response on the slow time-scale emerged from the more detailed description on the fast time-scale. In the present paper, we use the same methods, but we consider a different hypothesis concerning the time-scales: the fast process is assumed to be predation rather than migration. Indeed, empirical studies on fragmented populations demonstrate that within-patch processes often occur faster than inter-patch dispersal.

Nature is not homogeneous. Abiotic and biotic factors form complex structures that modify in a non-uniform way the space in which species live. Spatial heterogeneity in predator–prey systems means that predation pressure varies from on place to another. There are places where predation is strong, places where it is weaker, and places where it is practically non-existent. Therefore we will study a predator–prey system in a multi-patch environment in which each patch is supposed to be homogeneous. Arditi and Saïah[2](1992)[2] demonstrated experimentally that in spatially homogeneous systems, one might expect prey-dependent predation. Therefore, we assume that the feeding rate within a given patch depends only on the local prey density.

Commonly, when predators are placed together with their prey species in a homogeneous space, their dynamics bring the prey density to a very low value. As a result, the prey becomes extinct, followed closely by the predator. Such dynamics have been frequently observed in laboratory experiments with a homogeneous environment (Gause[7] 1934[7] Huffaker[13]1958[13]Luckinbill[14]1973[14]1974)[15] When a predator species causes the extinction of its prey in a homogeneous environment, the predation must be very intense. Therefore, we will consider that predation within a given patch is very strong and much more important than all other processes. A refuge patch, where the prey are free from predation, will allow the coexistence of the two species.

3. THE MODEL

We study a predator–prey system in a multi-patch environment. For the sake of simplicity we consider two patches only: one accessible to both prey and predators (patch 1) and the other one being a refuge for the prey (patch 2). Each patch is

supposed to be homogeneous. We assume that the feeding rate in patch 1 depends only on the local prey abundance. We assume that predation in patch 1 is stronger than all other processes. Consequently, in this patch, the prey abundance quickly tends towards zero. This does not mean that all the prey disappear—only those accessible to predators. The prey refuge (patch 2) constitutes a reservoir of prey. We suppose that the prey migrate between the two patches randomly. The growth of prey in each patch in the absence of predators is assumed to be logistic. The numerical response is taken to be proportional to the feeding rate.

All these assumptions can be summarized in five points:

(1) There are two patches, one refuge patch for prey and one patch for both predators and prey, the prey can migrate between these patches.
(2) Predation is the strongest process.
(3) In the predation patch, the feeding rate is prey-dependent.
(4) In each patch the growth of prey is logistic in the absence of predators.
(5) The numerical response of predators is proportional to the feeding rate.

The following model fulfils all these assumptions:

$$\frac{dN_1}{d\tau} = -\frac{AN_1 P}{1 + CN_1} + \varepsilon \left(rN_1 \left(1 - \frac{N_1}{K_1} \right) + m_{12}N_2 - m_{21}N_1 \right), \tag{2a}$$

$$\frac{dN_2}{d\tau} = \varepsilon \left(rN_2 \left(1 - \frac{N_2}{K_2} \right) + m_{21}N_1 - m_{12}N_2 \right), \tag{2b}$$

$$\frac{dP}{d\tau} = P \left(\frac{eAN_1}{1 + CN_1} - \varepsilon\mu \right), \tag{2c}$$

where N_i is the prey abundance on patch i, P is the predator abundance, m_{ij} is the migration rate from patch j to patch i, e is the conversion efficiency, μ is the predator mortality rate, and τ is the time. The local feeding rate in patch 1 is given by Holling's 'disc equation' (Holling, 1959)[2]

$$g(N_1) = \frac{AN_1}{1 + CN_1}, \tag{3}$$

and depends only on the local prey abundance. In (2), all parameters (except ε) are of the order of 1. $\varepsilon \ll 1$ is a small dimensionless parameter and its presence in (2) shows explicitly that predation is much stronger than other processes. Note that the mortality rate of the predators is assumed to be a slow process. Otherwise, as the abundance of accessible prey quickly reached a small value, a mortality rate of the order of 1 would be too strong and would lead to the extinction of the predators. As we will show later, the system (2) can have positive equilibria for both prey and predator densities. Note that in (2) the conversion efficiency is of the order of 1; however, it should not exceed 1 in order to assure the conservation

of mass. Nevertheless, it can be much smaller than 1 and, in that case, this would be technically equivalent to putting ε in front of the r.h.s. of (2c). In that case the predator dynamics would rapidly become slower than the prey dynamics, but our results concerning the overall dynamics on the slow time-scale would remain unchanged.

One may raise an objection concerning equation (2a): if the predator abundance, P, is very small, then the predation term in (2a) can be smaller than other terms and thus the assumption 2) would not be fulfilled. Indeed, in our model we require that $AP/(1 + CN_1)$ be of the order of 1. This means that, if N_1 remains in a given closed interval (which can be large if ε is small), then the predator abundance must be of order of $1/A$. The results presented in the next section remain valid as long as this condition is satisfied. This is not a very restrictive condition. Indeed, we will see that the system (2) has a positive attractive equilibrium and it suffices that the predator equilibrium abundance is at least of the order of $1/A$ and that assumption 2 is fulfilled.

4. FROM TOP–DOWN TO BOTTOM–UP

In patch 1, predation is very strong [see equation (2a)]. As a result, the prey abundance in patch 1 rapidly reaches a small value and ceases to be an independent variable. It becomes a function of the other two independent variables: $N_1 = N_1(N_2, P)$, which can be described in terms of the asymptotic expansion by (see Appendix for a more formal derivation of the results of this section):

$$N_1 = \varepsilon \omega_1(N_2, P) + O(\varepsilon^2). \tag{4}$$

One can neglect the rapidly disappearing transient dynamics that occur for large N_1, by substituting (4) into (2) to obtain

$$\frac{dN_1}{d\tau} = \varepsilon(-A\omega_1 P + m_{12}N_2) + O(\varepsilon^2), \tag{5a}$$

$$\frac{dN_2}{d\tau} = \varepsilon\left(rN_2\left(1 - \frac{N_2}{K_2}\right) - m_{12}N_2\right) + O(\varepsilon^2), \tag{5b}$$

$$\frac{dP}{d\tau} = \varepsilon P(eA\omega_1 - \mu) + O(\varepsilon^2). \tag{5c}$$

From (4), (5b) and (5c) we have

$$\frac{dN_1}{d\tau} = \frac{\partial N_1}{\partial P}\frac{dP}{d\tau} + \frac{\partial N_1}{\partial N_2}\frac{dN_2}{d\tau} = O(\varepsilon^2). \tag{6}$$

Comparing the terms of order of ε in (5a) and (6) we obtain

$$\omega_1 = \frac{m_{12}N_2}{AP}. \tag{7}$$

Substituting (7) into (5) and summing up the two prey equations we obtain the aggregated model that describes the dynamics of the system (2) on the slow time-scale:

$$\frac{dN}{dt} = rN\left(1 - \frac{N}{K_2}\right) - m_{12}N + O(\varepsilon), \tag{8a}$$

$$\frac{dP}{dt} = em_{12}N - \mu P + O(\varepsilon), \tag{8b}$$

where $N = N_1 + N_2$ is the total prey abundance and $t = \varepsilon\tau$ defines the new slow time-scale, and $O(\varepsilon)$ denotes terms of order of ε.

The system (8) is structurally stable for $\varepsilon = 0$, i.e., its dynamic behaviour does not change qualitatively if the system is slightly perturbed. Therefore, in (8) we can neglect the terms of order of ε and the qualitative behaviour of the system remains unchanged. The resulting model is:

$$\frac{dN}{dt} = rN\left(1 - \frac{N}{K_2}\right) - m_{12}N, \tag{9a}$$

$$\frac{dP}{dt} = em_{12}N - \mu P. \tag{9b}$$

The solutions of the initial model (2) are rapidly close (with errors of order of ε) to the solutions of (9) (see Appendix). This means that, on the slow time-scale, the dynamics of system (9) approximate that of the full initial system (2). Indeed, Fig. 1 shows that if ε is small enough, the trajectories of the full system converge rapidly towards those of the aggregated systems.

From (9a) we see that the prey dynamics are independent from predator abundance. The dynamics of predators depend, however, on prey abundance. This is a characteristic feature of donor control: the predators have no effect on the prey, whereas they themselves depend on prey availability. On the detailed level of description, the behavioural, fast time-scale feeding rate (3) was prey-dependent and thus predators controlled locally the prey abundance (top–down control). When we look, however, at the slow time-scale dynamics on the population level, bottom–up control appears. Thus, when we pass from local to global and from short time-scale to long time-scale, we pass from top–down to bottom–up control.

However, the donor control in the system considered here is only apparent: if the predators are reduced, by external factors, to a very small abundance or removed, the assumption of strong predation is no longer valid. In this case, the prey population will increase to a value determined by the carrying capacities of both patches, K_1 and K_2, and may be much higher than in the presence of predators. Thus, an efficient predator species can suppress the abundance of its prey by confining them to refuges but, as less and less prey are available outside the refuges, predators have an ever-decreasing effect on the prey population. Donor control dynamics described by (9) rapidly beings to dominate.
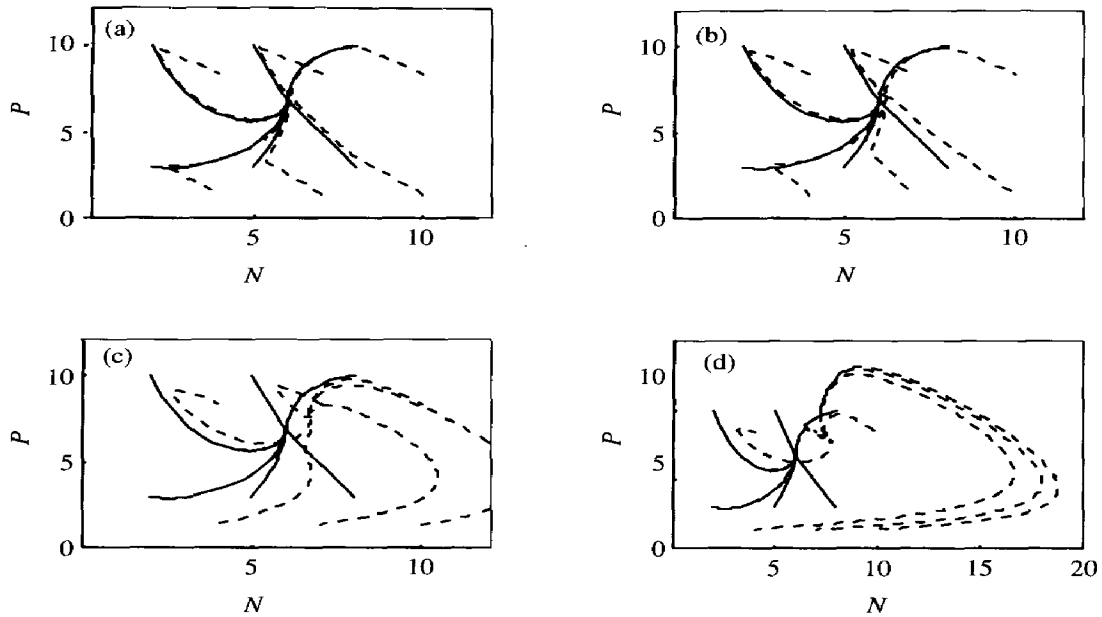
Figure 1. Phase portraits of the complete system (2) (dashed lines) and corresponding ones of the aggregated system (9) (solid lines). The values of the parameters are: $r = 2$, $K_1 = 5$, $K_2 = 12$, $m_{12} = m_{21} = 1$, $A = 1$, $C = 1$, $\mu = 0.7$. (a) $\varepsilon = 0.05$, (b) $\varepsilon = 0.1$, (c) $\varepsilon = 0.3$, (d) $\varepsilon = 0.5$. One sees that, (a) if ε is small the trajectories of the complete system are rapidly very close to trajectories of the aggregated system, i.e., the approximation is good after a very short transient phase. When $\varepsilon = 0.5$ (d) the approximation is very bad. (b) and (c) show phase portraits for intermediate values of ε.

The system (9) has a stable equilibrium given by:

$$N^* = K_2 \left(1 - \frac{m_{12}}{r} \right),$$

(10a)

$$P^* = \frac{em_{12}}{\mu} N^*.$$

(10b)

This equilibrium is positive if the prey initial growth rate r is larger than the migration rate to patch 1. If the equilibrium value of the predator abundance is at least of the order of $1/A$ then, for sufficiently high initial predator abundance, this abundance remains in the application domain of our method. If, however, the equilibrium predator abundance (10b) is much smaller than $1/A$, then the assumption of strong predation in patch 1 is not fulfilled and the system (2) cannot be aggregated to give the donor-controlled system (9).

Note that, even if the local behavioural feeding rate (3) saturated for high local prey densities N_1, the system (9) does not contain any saturation term for predators. This is due to the fact that, since predation in patch 1 is very strong, the predators rapidly deplete their available resources. The remaining prey, while being able to support (via slow migrations) a non-zero abundance of predators, do not suffice to saturate them. The behavioural feeding rate (3) saturates for high N_1 but due to

the strong predation on patch 1, N_1 rapidly decreases to a very small value and the saturation term, CN_1 in the r.h.s. of (3), can be neglected.

Note also that non parameter of the behavioural feeding rate (3) enters explicitly into the aggregated system (9). This suggests that the details of predation on the fast time-scale are unimportant for dynamics on the slow time-scale. We explore this suggestion in Section 6, after an example illustrating the theoretical results of this section.

5. EXAMPLE

In order to illustrate the theoretical results of Section 4, we consider a predator–prey system described by (2), where we abandon the parameter ε and attribute numerical values to the remaining parameters:

$$\frac{dN_1}{dt} = \frac{AN_1 P}{1 + CN_1} + rN_1 \left(1 - \frac{N_1}{K_1}\right) + m_{12}N_2 - m_{21}N_1, \tag{11a}$$

$$\frac{dN_2}{dt} = rN_2 \left(1 - \frac{N_2}{K_2}\right) + m_{21}N_1 - m_{12}N_2, \tag{11b}$$

$$\frac{dP}{dt} = P \left(\frac{eAN_1}{1 + CN_1} - \mu\right), \tag{11c}$$

with $A = 0.25, C = 0.05, e = 0.25, \mu = 0.1, r = 1$ and $m_{12} = m_{21} = 0.1$. Suppose now that patch 1 occupies 90% of the total space, the remaining 10% being the prey refuge (patch 2). This can be represented in our model by the carrying capacity of patch 1 being equal to 90% of the total carrying capacity: $K_1 = 0.9K$, $K_2 = 0.1K$, where K is the carrying capacity of the total space.

Figure 2 shows an example of the predator–prey dynamics in a system in which the carrying capacity changes periodically and abruptly between $K = 5$ and $K = 4$. In the absence of predators, the prey abundance would simply vary between 5 and 4. If predators are present and predation is weak ($A = 0.25$) then the mean prey abundance and the predator abundance follow a pattern characteristic of top–down controlled systems: the prey abundance remains almost constant (apart from short transients) and the variations of the primary productivity are followed by the predator abundance only (Fig. 2a). If predation is strong ($A = 1$, equivalent to decreasing ε four times), the same predator–prey system shows dynamics which can be clearly identified as donor-controlled: the variations of the predator abundance follow those of the prey (Fig. 2b).

The reason for this behaviour is clear: on one hand, when predation is weak, then the prey are everywhere and the total prey abundance is determined mainly by that in patch 1 (because it occupies 90% of the total space), where they are controlled by the predators. Therefore, the overall dynamics are similar to those regulated by
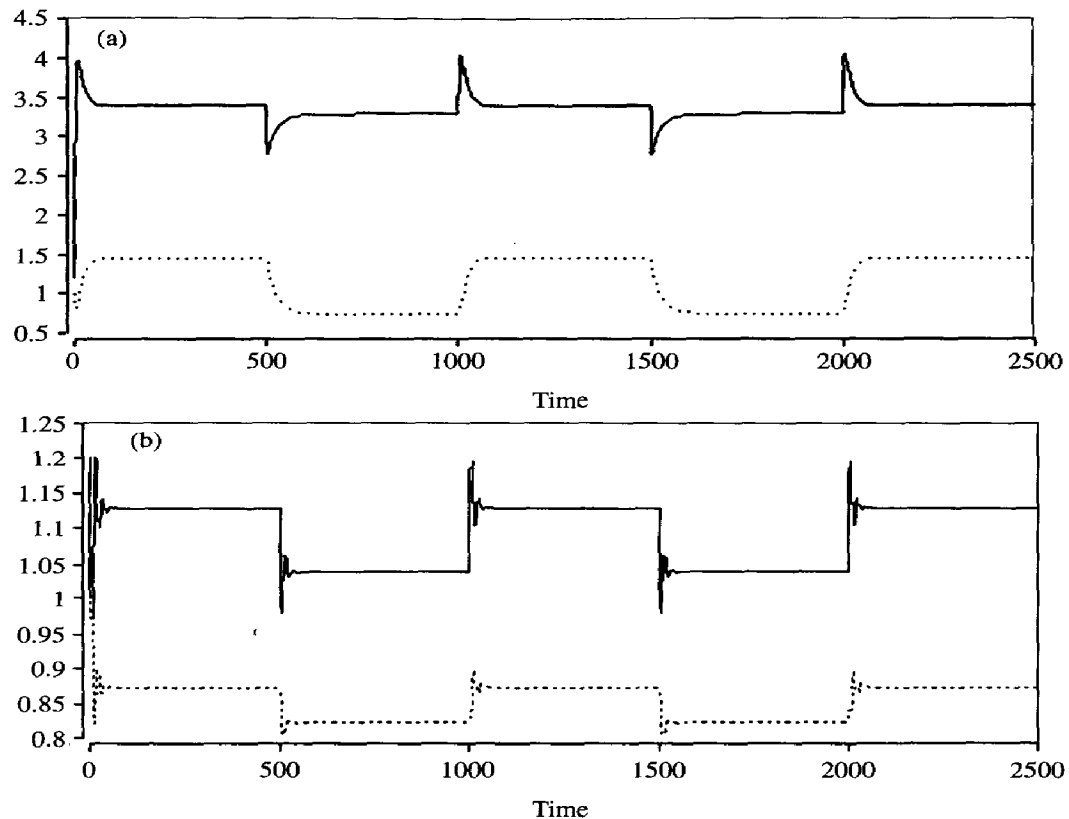
Figure 2. The densities of the prey (solid line) and the predator (dashed line) of the system described by (11). The primary productivity (K) jumps abruptly every 500 time units between $K = 5$ and $K = 4$. (a) $A = 0.25$ (weak predation). The pattern is characteristic of a system with top–down effects: the prey density remains almost constant and the variations of the primary productivity are followed by the predator density only. (b) $A = 1$ (strong predation). The pattern is characteristic of a donor-controlled system: the variations of the predator density follow those of the prey.

top–down effects. On the other hand, when predation is strong, there are almost no prey in patch 1 and the total prey abundance is determined mainly by that in patch 2, where predators have no effect. Thus, in this case, the prey abundance is apparently not affected by the predators; the latter, however, depend on the slow migration rate of the prey out of patch 2, controlled by the prey density in that patch. Therefore, strong predation results in overall dynamics which are apparently donor-controlled.

6. GENERALIZATION

In this section we relax the assumptions 3–5 from Section 3 and show that the space heterogeneity and strong predation are sufficient conditions for donor control to occur. As before, we consider a system on two patches.

Let the behavioural feeding rate on patch 1 (the predation patch) by any function of N_1 and P that monotonically increases with the prey density and satisfies

$$g(0, P) = 0, \tag{12a}$$

$$\left.\frac{\partial g(N_1, P)}{\partial N_1}\right|_{N_1=0} = O(1), \tag{12b}$$

i.e., the feeding rate $g(N_1, P)$ is very sensitive to changes in the prey density when prey are rare. In the previous sections we assumed that the numerical response of predators was proportional to the feeding rate; now it may be any increasing function of it,

$$H(N, P) = H(g(N, P)), \tag{13}$$

that satisfies $H(0) = 0$. The prey growth rate in the absence of predators may be of any form. The only condition is that it should be self-limiting in order to avoid an explosion of the prey population which may invalidate the assumptions of the model: if the prey density becomes too high, then the migration terms may become larger than the predation term.

As before, we summarize these conditions in five points, the first two of which remain unchanged:

(1′) There are two patches, one refuge patch for prey and one patch for both predators and prey, the prey can migrate between these patches.

(2′) Predation is the strongest process.

(3′) In the predation patch, the feeding rate $g(N_1, P)$ is zero for $N_1 = 0$ and is very sensitive to changes in the prey density when the prey are rare.

(4′) In each patch the growth of prey is self-limiting.

(5′) The numerical response of predators is an arbitrary function of the feeding rate satisfying $H(0) = 0$.

The following model satisfies these conditions:

$$\frac{dN_1}{d\tau} = -g(N_1, P)P + \varepsilon(f_1(N_1)N_1 + m_{12}N_2 - m_{21}N_1), \tag{14a}$$

$$\frac{dN_2}{d\tau} = \varepsilon(f_2(N_2)N_2 + m_{21}N_1 - m_{12}N_2), \tag{14b}$$

$$\frac{dP}{d\tau} = P(H(g(N_1, P)) - \varepsilon\mu). \tag{14c}$$

Due to strong predation, the prey density in patch 1 rapidly decreases to a small value given by

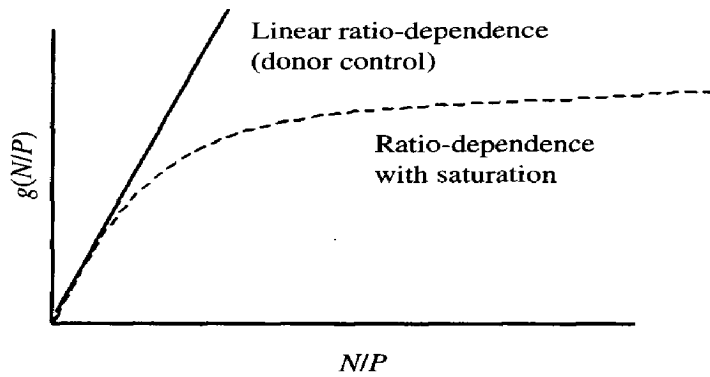$$N_1 = \varepsilon\omega_1 + O(\varepsilon^2), \tag{15}$$

Figure 3. Comparison of the donor control functional response (solid line) (21) with a more general saturating ratio-dependent functional response (dashed line) (Arditi and Ginzburg,[1] 1989}.

with

$$\omega_1 = \frac{m_{12}N_2}{A(P)P},\tag{16}$$

where $A(P)$ is a function of predator density and is defined by

$$A(P) = \left.\frac{\partial g(N_1, P)}{\partial N_1}\right|_{N_1=0}.\tag{17}$$

Substituting (15) into (14) is equivalent to neglecting the fast transient dynamics and retaining only terms that are important for the population dynamics on the slow time-scale. Proceeding in a similar way as in Section 4, we obtain the following predator–prey model on the slow time-scale:

$$\frac{dN}{dt} = f_2(N)N - m_{12}N + O(\varepsilon),\tag{18a}$$

$$\frac{dP}{dt} = em_{12}N - \mu P + O(\varepsilon),\tag{18b}$$

where

$$e = \left.\frac{\partial H}{\partial g}\right|_{g=0},\tag{19}$$

$f_2(N)N$ is the prey growth rate in patch 2, and $t = \varepsilon\tau$ defines the slow time-scale.

Note that the model (18) obtained with assumptions 1'–5' has the same form as the model (8) obtained with the more restricting conditions 1–5. This confirms the hypothesis formulated at the end of Section 4 that the detailed form of the behavioural feeding rate on the fast time-scale is not important for the population dynamics on the slow time-scale.

7. DONOR CONTROL AND RATIO-DEPENDENCE

There is a link between donor control, as described in the previous sections, and the ratio-dependent functional response in the sense of Arditi and Ginzburg (1989). In order to identify the functional response, the aggregated, slow predator–prey model given by (9) [or more generally by (18)] must be brought to the form of equation (1). This shows that the functional response is

$$g(N, P) = m_{12} \frac{N}{P} \tag{20}$$

This function is ratio-dependent, i.e., it depends on the ratio of prey to predators: $g = g(N/P)$. Indeed, Fig. 3 shows that as long as saturation effects do not operate (as in this paper), any ratio-dependent functional response can be approximated by

$$g\left(\frac{N}{P}\right) \approx \alpha \frac{N}{P}, \left(\text{for } \frac{N}{P} \text{ small}\right) \tag{21}$$

where α is the slope at the origin. If the ratio N/P is high, then the theorem used in this paper does not apply and donor control cannot be predicted. One may expect that, in such a case, predators are saturated and predation cannot be 'strong' (i.e., much more important than other processes). Combining the two complementary cases of low N/P and high N/P, the full picture of a type II ratio-dependent functional response emerges (Fig. 3).

8. DISCUSSION

The behavioural feeding rate is an individual-level process, while predator–prey models are concerned with population-level processes. The two kinds of processes may occur at different spatial and temporal scales. The time-scale of population dynamics may not be comparable to the time-scale of the behaviour of individuals. In this paper, we have studied a detailed predator–prey system with processes occurring on two different time-scales in a patchy environment. Predation took place on the fast (behavioural) time-scale; other processes (predator mortality, prey growth, migrations) were assumed to be slow. We used perturbation theory to show that, on the slow time-scale, a model with donor control describes the population-level dynamics of the predator–prey system. Donor control emerged from a mechanism that combined spatial heterogeneity (prey refuges) and strong predation.

Strong predation is defined as occurring when predators deplete rapidly (almost) all available resources. In a homogeneous environment, strong predation either drives the prey to extinction (deterministically) or leads to large oscillations of the prey and predator populations (limit cycle) that will also result in (stochastic) extinction of the prey. In a heterogeneous environment, however, if the prey can find

refuges, the same strong predation leads to donor control. This suggests immediately where to look for donor control. If predators and their prey exhibit unstable dynamics after spatial heterogeneity has been removed (e.g., in laboratory micro- or mesocosms or in field manipulations), this is evidence for strong predation. Then, according to our theoretical results, one may expect that, in the natural environment where they coexist, donor control might predominate. Thus, strong predation together with slow migrations may be considered as a sufficient condition for donor control to occur.

Efficient predators control the abundance of their prey by eating almost all of the prey outside the refuges. However, as available prey quickly become depleted, predator dynamics become dependent on the slow flow of prey from refuges. Thus, when a very efficient predator species is added to a system where its prey has refuges, the prey abundance will decline very quickly, as almost no prey that are not hidden can survive. Then, predators no longer have any impact on the prey density but their own population becomes limited by prey availability. It is clear that if a very efficient predator, which confines the prey to its refuge, disappears for some reason, a dramatic increase of the prey density would occur (in the system from the example of Section 5, almost five-fold): the prey would be abundant in the whole available space. Conversely, the arrival of a very efficient predator can result in a dramatic decrease of prey density, but, after a short transient period, the dynamics of the predator–prey system will be as if donor control is the prevailing force in the system. This is quite a different idea of donor control from that of predators eating prey that are bound to die anyway. Our model shows that there is no contradiction between the ability of predators (or parasitoids, used in biological control) to depress strongly prey density and donor control. Indeed, food webs that contain parasitoids (which are very different in killing their hosts) are under a large degree of donor control (Hawkins,[11] 1992).[1] When predators are very efficient, top-down effects can be observed on a short time-scale. On a long time-scale (if they prey and the predators are still there), bottom–up effects predominate.

ACKNOWLEDGEMENTS

APPENDIX

Here we use the Centre Manifold Theorem (Fenichel,[6] 1971;[6] Chow,[5] 1994)[5] to find a model for the aggregated variables, $N = N_1 + N_2$ and P, the dynamics of which

approximate that of the system (2) on the slow time-scale. The full system we study here is

$$\frac{dN_1}{d\tau} = -\frac{AN_1 P}{1 + CN_1} + \varepsilon \left(rN_1 \left(1 - \frac{N_1}{K_1} \right) + m_{12}N_2 - m_{21}N_1 \right),$$ (A1a)

$$\frac{dN_2}{d\tau} = \varepsilon \left(rN_2 \left(1 - \frac{N_2}{K_2} \right) + m_{21}N_1 - m_{12}N_2 \right),$$ (A1b)

$$\frac{dP}{d\tau} = P \left(e\frac{AN_1}{1 + CN_1} - \varepsilon\mu \right),$$ (A1c)

$$\frac{d\varepsilon}{d\tau} = 0,$$ (A1d)

where we added the trivial equation (A1d) for the small constant ε in order to apply the Centre Manifold Theorem directly.

Note that $x = eN_1 + P$ is a slowly changing variable since its time derivative is of the order of ε. The system (A1) in the new variables, $(N_1, N_2, x, \varepsilon)$, becomes:

$$\frac{dN_1}{d\tau} = -\frac{AN_1(x - eN_1)}{1 + CN_1} + \varepsilon \left(rN_1 \left(1 - \frac{N_1}{K_1} \right) + m_{12}N_2 - m_{21}N_1 \right),$$ (A2a)

$$\frac{dN_2}{d\tau} = \varepsilon \left(rN_2 \left(1 - \frac{N_2}{K_2} \right) + m_{21}N_1 - m_{12}N_2 \right),$$ (A2b)

$$\frac{dx}{d\tau} = \varepsilon \left(e \left(rN_1 \left(1 - \frac{N_1}{K_1} \right) + m_{12}N_2 - m_{21}N_1 \right) - \mu P \right),$$ (A2c)

$$\frac{d\varepsilon}{d\tau} = 0.$$ (A2d)

Clearly, for each value of N_2 and x, the vector $(N_1, N_2, x, \varepsilon) = (0, N_2, x, 0)$ is an equilibrium solution of the system (A2). The linearization of (A2) near this equilibrium is given by the following Jacobian:

$$L(0, N_2, x, 0) = \begin{pmatrix} -Ax & * & * & * \\ 0 & 0 & * & * \\ 0 & 0 & 0 & * \\ 0 & 0 & 0 & 0 \end{pmatrix},$$ (A3)

where the asterisks stand for non-zero terms whose exact form is not important for the subsequent analysis.

The Jacobian (A3) has two eigenvalues: the first one, $-Ax$, is associated with the space of N_1; the second eigenvalue is zero with multiplicity 3. If N_1 remains in a given closed and bounded interval I then, according to the Centre Manifold

Theorem, for sufficiently small ε, there exists a manifold W (called the centre manifold), which is the graph of a map:

$$(N_2, x, \varepsilon) \rightarrow N_1(N_2, x, \varepsilon) \in I, \tag{A4}$$

such that:

(i) $(N_2, x, 0) \rightarrow 0$, for each x and N_2;

(ii) W is invariant under the flow defined by (A2) and is attractive;

(iii) W is tangent to the eigenspace associated with the zero eigenvalue.

This means that the dynamics defined by (A2) are rapidly close to its restriction on W, which is obtained by replacing the variable N_1 by a function $N_1(N_2, x, \varepsilon)$. The conclusion (iii) allows us to compute the asymptotic expansion of $N_1(N_2, x, \varepsilon)$ for small ε:

$$N_1(N_2, x, \varepsilon) = \varepsilon \omega_1(N_2, x) + O(\varepsilon^2). \tag{A5}$$

Substituting (A5) into (A2a) we obtain

$$\frac{dN_1}{d\tau} = \varepsilon(-Ax\omega_1(N_2, x) + m_{12}N_2) + O(\varepsilon^2). \tag{A6}$$

On the other hand, from (A5a), (A2b) and (A2c) we have

$$\frac{dN_1}{d\tau} = \frac{\partial N_1}{\partial N_2}\frac{dN_2}{d\tau} + \frac{\partial N_1}{\partial x}\frac{dx}{d\tau} = O(\varepsilon^2). \tag{A7}$$

Identifying in (A6) and (A7) the terms of the same order in ε, we obtain:

$$\omega_1 = \omega_1(N_2, x) = \frac{m_{12}N_2}{Ax}. \tag{A8}$$

The restriction of (A2) on W can now be described by:

$$\frac{dN_2}{dt} = rN_2\left(1 - \frac{N_2}{K_2}\right) - m_{12}N_2 + O(\varepsilon), \tag{A9a}$$

$$\frac{dP}{dt} = P\left(e\frac{m_{12}N_2}{x} - \mu\right) + O(\varepsilon), \tag{A9b}$$

where $t = \varepsilon\tau$ defines the time on the slow time-scale.

Note that N_1, the density of accessible prey in patch 1, is quickly close to zero. Hence the variable x is quickly close to P:

$$x = P + eN_1 = P + O(\varepsilon). \tag{A10}$$

Consequently, one obtains

$$\omega_1 = \frac{m_{12}N_2}{AP} + O(\varepsilon), \tag{A11}$$

and the equation (A9b) becomes:

$$\frac{dP}{dt} = P\left(e^{\frac{m_{12}N_2}{P}} - \mu\right) + O(\varepsilon). \tag{A12}$$

Since $dN_1/d\tau$ is of order of ε [see equation (A6)], the dynamics of the total prey density, $N = N_1 + N_2$, is described by:

$$\frac{dN}{dt} = \frac{dN_1}{dt} + \frac{dN_2}{dt} = rN_2\left(1 - \frac{N_2}{K_2}\right) - m_{12}N_2 + O(\varepsilon). \tag{A13}$$

Substituting $N_2 = N - (\varepsilon\omega_1 + O(\varepsilon))$ into (A12) and (A13) we obtain the predator–prey model on the slow time-scale:

$$\frac{dN}{dt} = rN\left(1 - \frac{N}{K_2}\right) - m_{12}N + O(\varepsilon). \tag{A14a}$$

$$\frac{dP}{dt} = em_{12}N - \mu P + O(\varepsilon). \tag{A14b}$$

Small perturbations of a structurally stable system do not change the qualitative behaviour of its dynamics. The system (A14) is structurally stable for $\varepsilon = 0$, therefore one can neglect the small perturbation terms $O(\varepsilon)$, resulting in an error of the order ε.

REFERENCES

Arditi, R. and L. R. Ginzburg (1989). Coupling in predator–prey dynamics: ratio-dependence. *J. Theor. Biol.* **139**, 311–326.

Arditi, R. and H. Saïah (1992). Empirical evidence of the role of heterogeneity in ratio-dependent consumption. *Ecology* **73**, 1544–1551.

Auger, P. M. and J. C. Poggiale (1996). Emergence of population growth models: fast migrations and slow growth. *J. Theor. Biol.* **182**, 99–108.

Berryman, A. A., J. Michalski, A. P. Gutierrez, and R. Arditi (1995). Logistic theory of food web dynamics. *Ecology* **76**, 336–343.

Chow, S. N., C. Lie, and D. Wang (1994). *Normal Forms and Bifurcation of Planar Vector Fields*, Cambridge: Cambridge University Press.

Fenichel, N. (1971). Persistence and smoothness of invariant manifolds for flows. *Indiana Univ. Math. J.* **21**, 193–226.

Gause, G F. (1934). *The Struggle for Existence*, New York: Hafner Publishing Co.

Getz, W. M. (1991). A unified approach to multispecies modeling. *Nat. Res. Mod.* **5**, 393–421.

Hairston, N., F. Smith, and L. Slobodkin (1960). Community structure, population control and competition. *Am. Nat.* **94**, 421–425.

Hanski, I. (1991). The functional response of predators: worries about scale. *Trends Ecol. Evol.* **6**, 141–142.

Hawkins, B. A. (1992). Parasitoid–host food webs and donor control. *Oikos* **65**, 159–162.

Holling, C. S. (1959). Some characteristics of simple types of predation and parasitism. *Can. Entom.* **91**, 385–398.

Huffaker, C. B. (1958). Experimental studies on predation: dispersion factors and predator–prey oscillations. *Hilgardia* **27**, 343–383.

Luckinbill, L. S. (1973). Coexistence in laboratory populations of *Paramecium aurelia* and its predator *Didinium nasutum*. *Ecology* **54**, 1320–1327.

Luckinbill, L. S. (1974). The effect of space and enrichment on a predator–prey system. *Ecology* **55**, 1142–1147.

Menge, B. A. (1992). Community regulation: under what conditions are bottom–up factors important on rocky shores? *Ecology* **73**, 755–765.

Michalski, J., J.-Ch. Poggiale, R. Arditi, and P. M. Auger (1997). Macroscopic dynamic effects of migrations in patchy predator–prey systems. *J. Theor. Biol.* **185**, 459–474.

Pimm, S. L. (1982). *Food Webs*, London and New York: Chapman and Hall.

Strong, D. R. (1992). Are tropic cascades all wet? Differentiation and donor-control in specious ecosystems. *Ecology* **73**, 747–754.