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MODÈLES DYNAMIQUES EN BIOLOGIE, R. ARDITI (DIR.)
DYNAMICAL MODELS IN BIOLOGY, R. ARDITI (ED.)

Ecology of Individuals and Predator-Prey Dynamics

BY

CARLOS BERNSTEIN[1]

Abstract.– BERNSTEIN C., 1990. Ecology of individuals and predator-prey dynamics. *In*: Dynamical Models in Biology, R. Arditi (ed.). *Mém. Soc. vaud. Sc. nat. 18.3*: 195-212.
In this work I discuss the influence that individual differences could have on predator-prey and host-parasitoid dynamics and how they can be included in models. The first section considers the differences between hosts in survival probability and in vulnerability to parasitoid attacks. The strength of intraspecific competition (i.e. whether competition is «contest» or «scramble», a consequence of how animals share their resources) can have an important influence, expanding or contracting the boundaries of stability. Individual differences in susceptibility to parasitoid attacks can contribute to the stabilizing influence of spatial heterogeneity in the distribution of these attacks. The second part discusses the influence of individual learning and decisions on the spatial distribution of predators and parasitoids and of their attacks. The relative speeds of the consumption and learning processes can determine whether a population approaches the Ideal Free Distribution (IFD). Slow depletion can lead to a distribution near the IFD and to spatially density-dependent prey mortality. Fast depletion can make it difficult for the population to approach the IFD. Prey mortality becomes then, inversely density dependent.
Models assuming individual learning converge towards the IFD faster than «omniscient» versions, in which predators use the environment average in prey availability and migrate only to patches with capture rate larger than their current one.

Résumé.– BERNSTEIN C., 1990. Ecologie des individus et dynamique des systèmes proie-prédateur. *In*: Modèles dynamiques en biologie, R. Arditi (dir.). *Mém. Soc. vaud. Sc. nat. 18.3*: 195-212.
Ce travail discute l'influence que peuvent avoir sur la dynamique des systèmes proie-prédateur et hôte-parasitoïde les différences entre les individus et comment elles peuvent être incluses dans les modèles.

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La première partie concerne l'influence des différences de probabilité de survie et de vulnérabilité aux attaques par les parasitoïdes, entre les hôtes. Les différents types de compétition intraspécifique («mêlée» ou «combat», conséquence du partage égalitaire ou non des ressources) peuvent avoir une forte influence en augmentant ou en diminuant les domaines de stabilité. Les différences entre les individus, dans la susceptibilité aux attaques par les parasitoïdes, peuvent contribuer aux effets stabilisateurs de l'hétérogénéité dans la distribution des attaques.
La seconde partie discute l'influence de l'apprentissage et des décisions individuelles sur la distribution spatiale des prédateurs et parasitoïdes et de leurs attaques. Les résultats montrent que le fait qu'une population de prédateurs s'approche de la Distribution Idéale et Libre (DIL) peut dépendre du rapport des vitesses de consommation des proies et d'apprentissage. Une consommation lente peut conduire la population à une distribution proche de la DIL et à une mortalité des proies dépendante de la densité dans le sens spatial. Une consommation rapide peut rendre très difficile pour une population de s'approcher de la DIL. La mortalité des proies devient alors inversement dépendante de la densité.
Un modèle qui suppose un apprentissage individuel peut converger vers la DIL plus rapidement que certains modèles qui supposent que les prédateurs connaissent parfaitement la disponibilité moyenne des proies dans l'environnement et qu'ils agissent en conséquence.

INTRODUCTION

Many of the current ideas in ecological modelling can be traced to the classical works of LOTKA (1925), VOLTERRA (1928) and NICHOLSON and BAILEY (1935). In the field of predator-prey and host-parasitoid systems, concepts and models have been enriched by the addition of some realistic features such as spatial heterogeneity (HASSELL and MAY 1973, 1974, MURDOCH and OATEN 1975, MURDOCH 1977, MAY 1978, BEDDINGTON *et al.* 1978), interference between searching individuals (ROGERS and HASSELL 1974, HASSELL and MAY 1973, FREE *et al.* 1977), variable sex ratios (HASSELL *et al.* 1983) and differences in the timing of density dependent processes (MAY *et al.* 1981).

In a pioneering study, BAILEY *et al.* (1962) analyzed the influence that individual differences in vulnerability to parasitoid attacks could have on the dynamics of host-parasitoid systems. Thereafter, this subject has received little attention and most models still treat both populations as formed by identical individuals. Here I will discuss the influence that individual differences could have on predator-prey and host-parasitoid dynamics and how they can be included in models.

In the first section I will discuss the influence that individual differences between hosts in survival probabilities and in vulnerability to parasitoid attacks could have on the dynamics of host-parasitoid systems.

The theme of the second part is the influence of individual learning and decisions on the spatial distribution of predators and parasitoids and of their attacks.

INDIVIDUAL DIFFERENCES AND PREDATOR-PREY DYNAMICS

A General Framework

Many host-parasitoid models for discrete generations (e.g. formed by univoltine populations in temperate climates) (NICHOLSON and BAILEY 1935, HASSELL and MAY 1973, BEDDINGTON *et al.* 1975, MAY *et al.* 1981, MAY and HASSELL 1981, BERNSTEIN 1986, 1987) can be expressed in the general form

$$N_{t+1} = \lambda N_t f(N_t) p(N_t, P_t) \qquad (1)$$
$$P_{t+1} = N_t g(N_t)[1 - p(N_t, P_t)]$$

that corresponds to the case where parasitism acts first followed by density-dependent mortality of both parasitized and unparasitized hosts (MAY *et al.* 1981). N is the density of the host and P that of the parasitoid in successive generations t and $t+1$, λ is the host finite rate of increase $[\lambda = \exp(r)]$, $p(N_t, P_t)$ is the fraction of host evading parasitism, $f(N_t)$ is the density-dependent survival rate of non parasitized hosts and $g(N_t)$ that of parasitized ones, before the emergence of the parasitoid (and hence represents the survival of the parasitoids).

The simplest version of eq. (1) was analyzed by NICHOLSON and BAILEY (1935) in their classical work. They left aside density dependent processes and hence $f(N_t) = g(N_t) = 1$. They also considered that the parasitoids distribute their attacks strictly at random ($p(N_t, P_t)$ becomes then the zero term of the Poisson distribution). It is well known that this model is unstable and produces oscillations of growing amplitude in both populations. The difference between the behaviour of this model and the persistence of real host-parasitoid systems fruitfully guided the work of students of host-parasitoid dynamics for more than 50 years.

The heterogeneous distribution of parasitoids in the environment may result in a heterogeneous distribution of their attacks. One of the most significant developments of the theory of predator-prey and host-parasitoid interactions has been to realize that the heterogeneous distribution of host mortality (whether dependent on host density or not) can have a strong stabilizing influence. For instance, on phenomenological grounds (it fits many data sets), MAY (1978) proposed the negative binomial distribution to describe the aggregation of parasitoids attacks. In this case, $p(N_t, P_t)$ becomes

$$p(N_t, P_t) = (1 + aP_t/k)^{-k}\ , \qquad (2)$$

where k determines the degree of clumping in the attacks by the parasitoids[1]. As k decreases the distribution of attacks becomes more contagious.

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[1]Even if, on logical grounds, the fraction of hosts evading parasitism, $p(N_t, P_t)$,

At the limit $k \to \infty$ the negative binomial distribution becomes Poisson and the attacks are distributed completely at random. As shown by MAY (1978), eq. (1) with $f(N_t) = g(N_t) = 1$, and including eq. (2), is stable for $k < 1$. A similar conclusion (stability for $k < 1$) is attained for a model (MAY *et al.* 1981) incorporating (2) and in which density-dependent host survival is represented by a logistic function $[f(N_t) = g(N_t) = \exp(-dN_t)]$.

These models correspond to a general perception of predator-prey or host-parasitoid systems as formed by tightly interacting populations in which the persistence would be the consequence of regulation. This view is challenged by different authors (MURDOCH *et al.* 1984, 1985, REEVE and MURDOCH 1985, STRONG 1988) on the grounds that neither stability nor sufficiently strong spatial differences in host mortality are common features of successful biological control. According to this view, predator-prey or host-parasitoid systems would be always locally unstable and the persistence in larger areas would be the consequence of random extinction in some places and recolonization in others.

MURDOCH *et al.* (1984), REEVE and MURDOCH (1985) devised two methods to measure the strength of spatially heterogeneous, density-independent host mortality based on MAY's (1978) model. The methods allow determination of whether k is lower than 1 in natural host-parasitoid systems. They used these methods in two studies of successful biological control of scale insects. In both, they found values of k larger than 1 and so concluded that spatial heterogeneity does not play a key role in the stability of either system. Here I will argue that one important weakness in the work of MURDOCH *et al.* is that it takes into account neither the host individual differences in competition ability nor the differences in vulnerability to parasitoid attack.

Contest and Scramble Competition in Single Population Models

It has been shown (BEGON 1984, ŁOMNICKI 1988) that in many populations some individuals have a strong competitive ability and obtain a disproportionate share of the resources. When the density of the population is too big in comparison with the resources available, the individuals with larger competitive ability monopolize the resources (their share of the resources is little affected by the presence of individuals of lower competitive ability). These individuals survive and reproduce. The others fail to do so. In this way the growth of the population is more or less adjusted to the availability of the key resource (fig. 1a).

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should depend both on host and parasitoid densities, in most models it becomes a function of parasitoid density alone, as a consequence of mathematical simplifications.

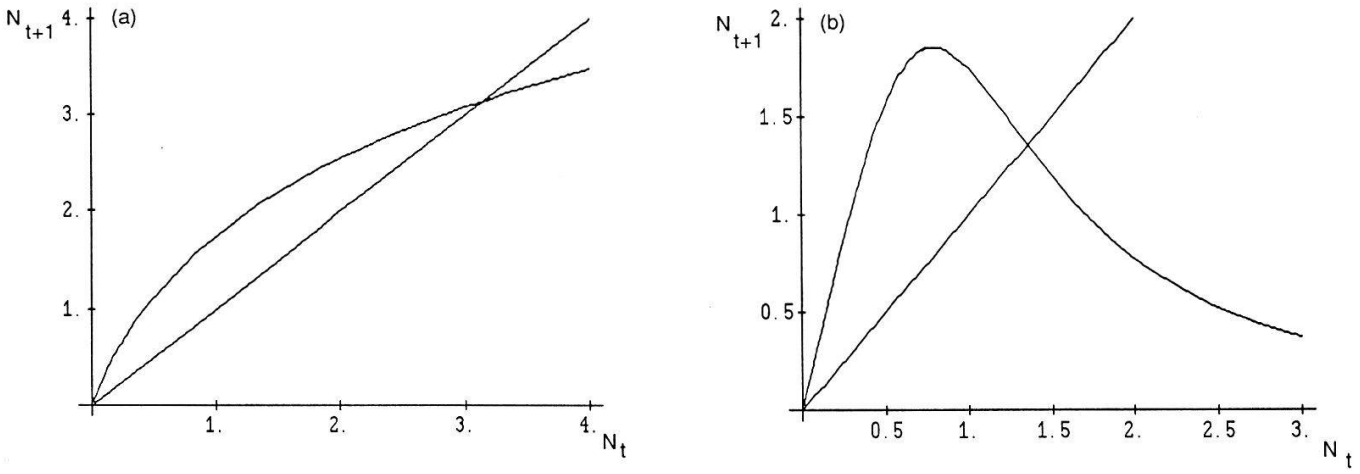

Figure 1.– The influence of differences in competitive ability on population growth. Number of individuals in one generation N_{t+1} as a function of the number on the previous one, N_t. (a) «Contest» competition: after an initial increase, the curve levels off for high population densities. (b) «Scramble» competition: after the initial increase, for high population densities, the curve decreases more or less steeply. The straight line has a slope of unity and shows the equilibrium density ($N_t = N_{t+1}$). The stability of these single population models depends on the first derivative of the growth curve at the equilibrium point.

This is usually known as «contest» competition[1]. In other populations the individual differences in competitive ability are not that large and the share of the resources is relatively egalitarian. In this case («scramble competition»), when the number of individuals is too high in relation to resource availability, all individuals get an insufficient share of the resources and die or do not succeed to reproduce. This produces a decrease in the number of individuals in the next generation (fig. 1b).

Different single population models have been proposed to represent the position of a population in the contest-scramble spectrum. Two that fit well many data sets and fulfil the logical requirements for a growth function (BELLOWS 1981) are the MAYNARD SMITH and SLATKIN (1973) model

$$N_{t+1} = \frac{\lambda N_t}{1 + aN_t^b}$$

[1]Other processes such as preemption of resources by first arrived animals can also lead to «contest» competition.

and the generalized logistic or θ-Ricker curve (POMERANTZ *et al.* 1980, THOMAS *et al.* 1980)

$$N_{t+1} = \lambda N_t \exp\left[-r(N_t/K)^{\theta}\right] ,$$

where b and θ determine the severity of density-dependence. Increasing values of b or θ imply movement along the contest-scramble competition towards more severe scramble.

The θ-Ricker curve has some weakness: the distinction between contest and scramble competition is not as sharp as in the MAYNARD SMITH and SLATKIN (1973) model, but many alternative models can be easily derived from it (BELLOWS 1981). This is also true for host-parasitoid models incorporating that function.

Contest and Scramble Competition in Predator-Prey Models

Even if density-dependence has not been totally neglected as a factor promoting stability in predator-prey and host-parasitoid models (BEDDINGTON *et al.* 1975, 1978, MAY *et al.* 1981), it has always been rigidly represented without reference to the position of the populations in the contest-scramble spectrum.

BERNSTEIN (1986) incorporated the differences in the strength of intraspecific competition into eq. (1) [$p(N_t, P_t)$ being represented as in eq. (2)] by using the same representation as in the θ-curve, by making $f(N_t) = g(N_t) = \exp[-r(N_t/K)^{\theta}]$.

The stability of this model has been explored by means of neighbourhood stability analysis. The aim of biological control is to produce a displacement of the density of the pest population to a new equilibrium well below its carrying capacity (very low q values, $q = N^*/K$, where N^* is the equilibrium after the introduction of the natural enemy and K the one before the introduction). It is interesting then, to include q among the relevant parameters, when studying the stability of a host-parasitoid model. The results, presented in fig. 2 in terms of r, θ and q, show that the differences in the strength of intraspecific competition can have a strong influence on the stability of host-parasitoid systems. For low and realistic values of θ (POMERANTZ *et al.* 1980) (i.e. contest competition) the model becomes stable for low (but not extreme) values of q. High θ values (scramble competition) produce a strong contraction of the stability boundaries. As shown in fig. 2 the interaction between contest competition and heterogeneity in the distribution of parasitoid attacks (lower k values) leads to even larger stability boundaries.

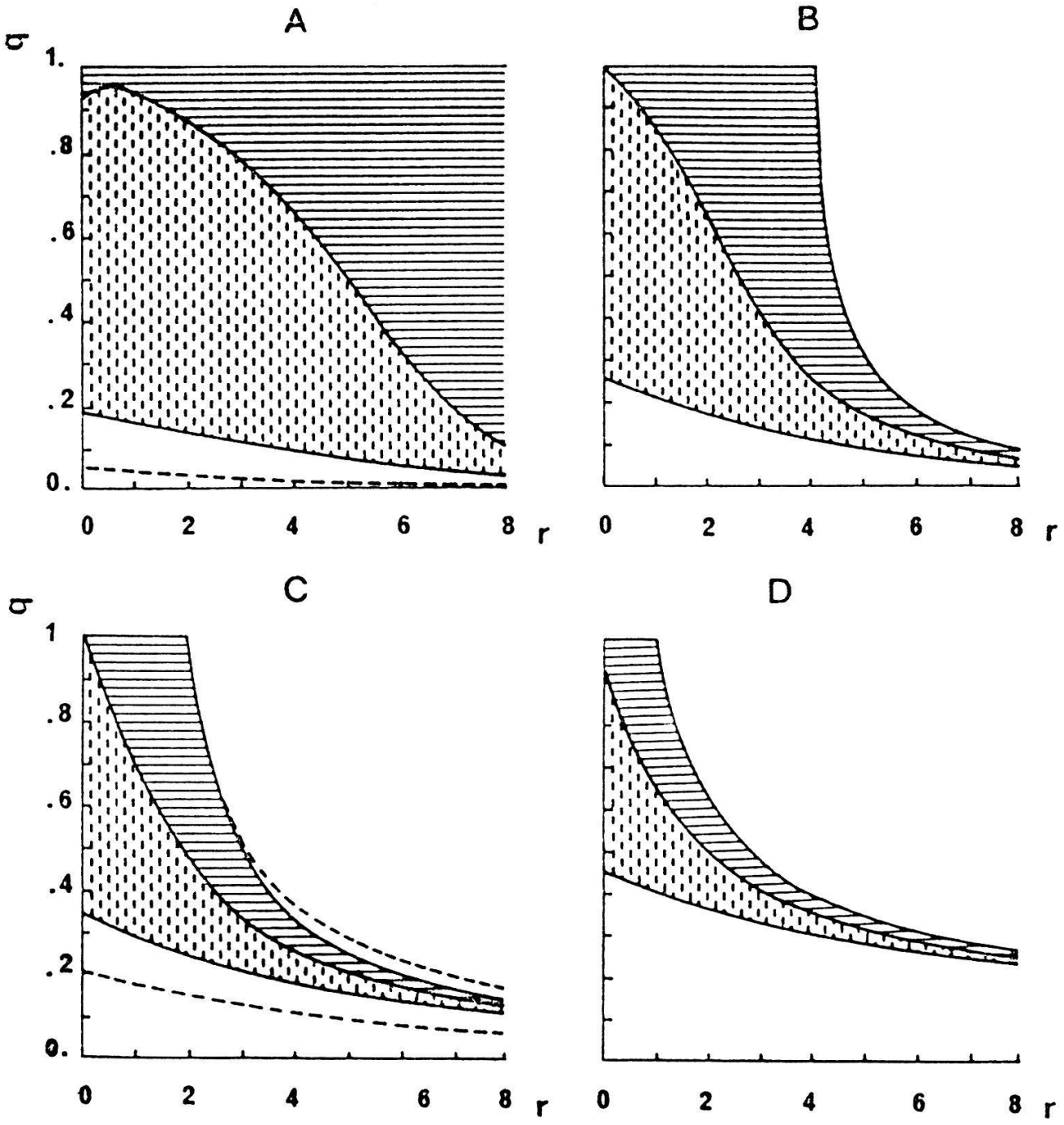

Figure 2.– Differences in competitive ability and host-parasitoid dynamics. Local stability boundaries of the BERNSTEIN (1986) model in terms of the depression of the prey equilibrium ($q = N^*/K$) and the prey rate of increase (r). The boundaries shown correspond to the following parameter values (A) $\theta = 0.2$, (B) $\theta = 0.5$, (C) $\theta = 1$ and (D) $\theta = 2$. The dotted area denotes the region of oscillatory damping and the horizontally hatched area that of exponential damping for $k \rightarrow \infty$ (in eq. (2), see text). The broken lines in (A) and (C) show the stability boundaries for a moderate aggregagtion of parasitoid attacks.

Differences in Host Vulnerability to Parasitoid Attacks

Different processes can result in differences in vulnerability between hosts

a–genetic differences (BOULÉTREAU and WAJNBERG 1986) sometimes related to their behaviour (CARTON and DAVID 1985),

b–partial asynchrony between the populations that can lead to the unavailability for parasitism of a fraction of the host population (MÜNSTER-SWENDSEN and NACHMAN 1978);

c–differences in age and size among hosts in the susceptible stages, as parasitoids show different degrees of selectivity in this respect (DRANKS 1975, VINSON 1976, PRINCE 1976, VAN ALPHEN and DRIJVER 1982, VAN ALPHEN and THUNNISSEN 1980);

d–rejection (by encapsulation) of the parasitoids by a fraction of the hosts (SALT 1970, VINSON and IWANTSCH 1980, CARTON and BOULÉTREAU 1985).

MURDOCH *et al.* (1984) and CHESSON and MURDOCH (1986) pointed out that the negative binomial distribution of parasitoid attacks can be derived by assuming that parasitoids are gamma-distributed among spatially discrete patches and that searching by parasitoids within patches is random. If u_t is the number of parasitoids in the vicinity of each host and $s(u_t)$ the probability density function of u_t, then the average or expected proportion of unparasitized hosts $p(N_t, P_t)$ is given by

$$p(N_t, P_t) = \int_0^\infty \exp(-a\, u_t) s(u) du \; .$$

When $s(u)$ is gamma-distributed, $p(N_t, P_t)$ becomes the zero term of the binomial distribution.

BAILEY *et al.* (1962) studied the complementary situation. If a, the risk of being parasitized, varies among hosts and $r(a)$ specifies the distribution of a, then the expected fraction of the population escaping parasitism is

$$p(N_t, P_t) = \int_0^\infty \exp(-a\, u_t) r(a) da \; .$$

Differences in parasitoid distribution and host vulnerability are likely to combine to a greater or lesser extent in many, if not all, host-parasitoid systems. If $s(u)$ and $r(a)$ are independent (an acceptable assumption), a general expression for $p(N_t, P_t)$ could be

$$p(N_t, P_t) = \iint_0^\infty \exp(-a\, u_t) s(u) r(a) du \; da \; . \qquad (3)$$

Ideally, both $s(u)$ and $r(a)$ should be taken to be gamma, or another equally flexible distribution, but the mathematics become rapidly too cumbersome. Nevertheless, an interesting insight can be obtained if $s(u)$ is

taken as gamma-distributed and $r(a)$ as uniform. The interest of this particular $r(a)$ distribution is that it cannot stabilize host-parasitoid systems on its own. In this case, in the absence of any form of intraspecific competition, the condition for stability for a model incorporating eq. (3) into eq. (1) is $k < 2$ and not $k < 1$ as in the MAY (1978) model. This shows that even small differences in individual vulnerability to parasitoid attacks can have a clear influence on the stability of host-parasitoid systems.

SPATIAL DISTRIBUTION OF PREDATORS

Given the multiplicity of distribution patterns of predator attacks found in nature (LESSELLS 1985, STILING 1987, WALDE and MURDOCH 1988) and the influence that this distribution can have on the dynamics of predator-prey systems, it is interesting to enquire about the ecological mechanisms that lead to these different patterns. Optimality models are powerful tools to study this problem at the individual level and to try to link individual behaviour to population distribution.

Imagine a heterogeneously distributed resource and «omniscient» consumers (i.e. having a perfect and continuously updated knowledge of the distribution of prey availability). The consumers are assumed to be «free» to move to the patch that gives them the highest intake rate and «ideal» in the sense of having all the same ability to compete and consume. Under these conditions, individuals experiencing a lower feeding rate as a consequence of the number of consumers and the density of the resource in their present patch should move to a more profitable patch. Animals would move from one patch to another until an equilibrium is reached in which all individuals get the same rate of gain. The equilibrium condition is known as the «Ideal Free Distribution» (IFD) (FRETWELL and LUCAS 1970).

SUTHERLAND (1983) applied this idea to predators (or parasitoids) that attack a non-depleting population of prey (or hosts). He considered that in each patch the attacks by predators (or parasitoids) followed a type II functional response (HOLLING 1959). Predators are assumed to interfere with each other so that the larger the predator density, the lower their searching efficiency (HASSELL and VARLEY 1969, HASSELL and MAY 1973, HASSELL 1978). Combining these assumptions with the requirement that, at equilibrium, the number of prey captured by each predator must be the same in all patches, SUTHERLAND's model makes it possible to calculate the distribution of predators and of their attacks as a function of the number of prey per patch. This model predicts that for normal values of predator interference there will be more predators per prey at high prey density and that prey mortality from predation will be density-dependent. At the highest levels of predator interference mortality becomes density-independent. No

«domed» (i.e. with a maximum for intermediate prey densities) or inversely density-dependent distributions are generated by this model.

LESSELLS (1985) reviewed 49 studies of spatial distribution of attacks by insect parasitoids and found that, in contradiction with SUTHERLAND's predictions, host mortality was inversely density-dependent in 15 cases. Two cases of domed density-dependence were also detected.

The Distribution of Learning Predators

The IFD model has at least three shortcomings

1–it contains the unrealistic assumption of the predators being omniscient about prey availability;

2–it is unable to generate the different patterns of predator distribution found in nature;

3–it assumes an equilibrium distribution but no proof is given that it would be attained.

The aim of a new model (BERNSTEIN *et al.* 1988) has been to incorporate realistic assumptions of animal learning to the general framework of the IFD model. This simulation model considers the environment as divided in a number of cells. A generation is divided in a number of discrete time units (simulation cycles). At each simulation cycle and in each cell, predators attack the prey and a relative capture rate (q = number of prey ingested/maximum ingestion capacity) is calculated. Predators are assumed to attack the prey following a type II functional response and to interfere among them.

Each predator is assumed to leave its patch if q in that patch falls below γ, its «personal» estimate of the mean value of q in all patches of the environment. γ is updated at each time step as a weighted mean between the capture rate experienced in the past and that experienced in the current simulation step. The updating algorithm is a linear operator (BUSH and MORSTELLER 1955, KACELNIK and KREBS 1985)

$$\gamma_{t+1} = \alpha q_t + (1 - \alpha)\gamma_t \qquad 0 \leq \alpha \leq 1 \tag{4}$$

where γ_t is the value of γ at time t and α is the «memory factor». All migrating predators are pooled and distributed at random between patches.

To enquire about the dependence of animals achieving the IFD on the assumption of omniscience, BERNSTEIN *et al.* (1988) compared the results of their model with the predictions of SUTHERLAND (1983). By definition, at the IFD capture rate per individual is independent of local prey density. The number of predators in any given patch as a function of the number of prey in that patch, and the average capture rate per predator are predicted by the SUTHERLAND (1983) model. Any of these different predictions could

be used to test the convergence of the modelled predators to the IFD.

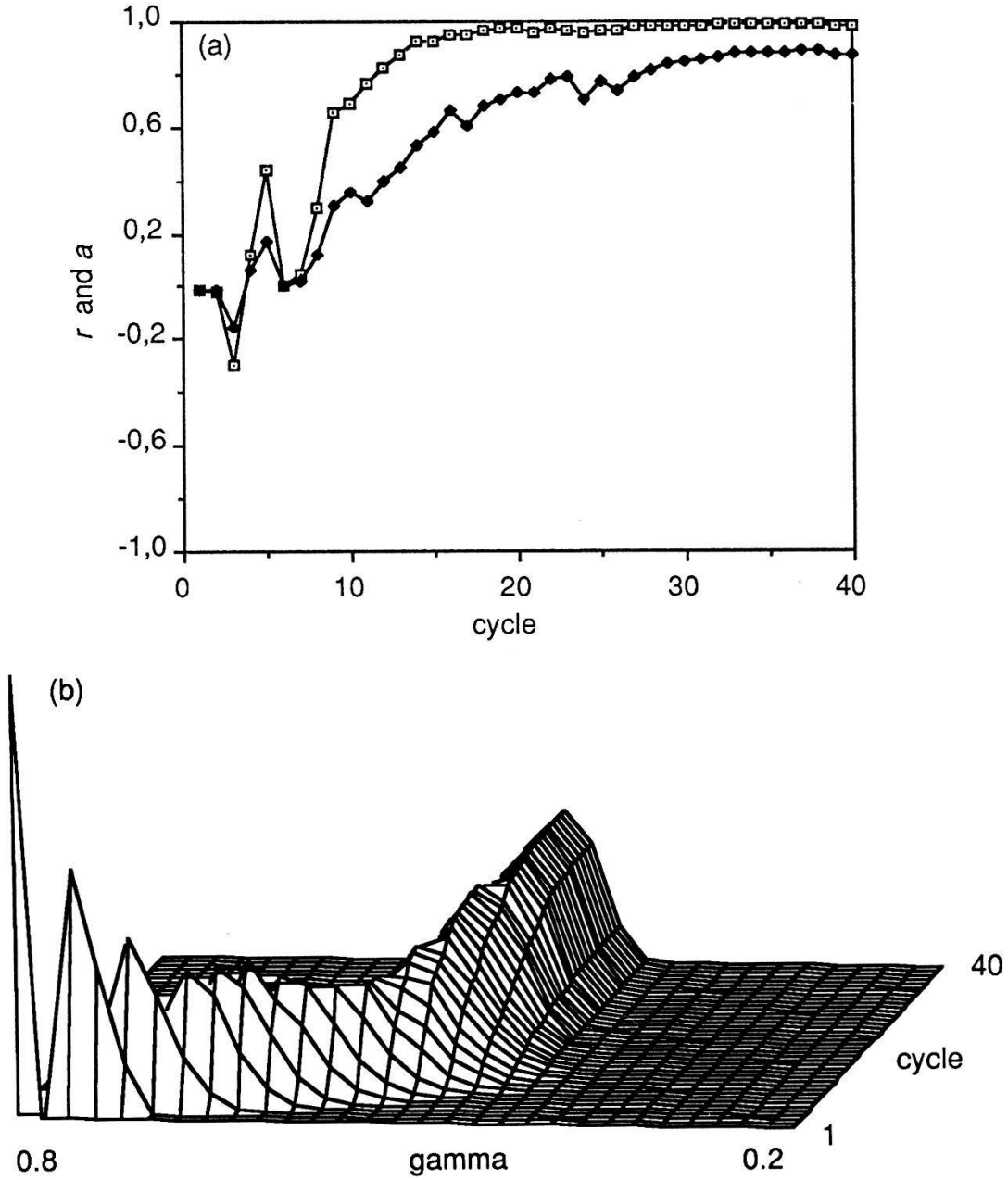

Figure 3.– The spatial distribution of learning predators (BERNSTEIN *et al.* 1988). (a) Correlation (open symbols) and regression coefficients (closed symbols) between the results of the simulations and the predictions of the ideal free distribution for the number of predators in each patch as a function of the number of prey. The distributions generated by the model are compared with the equilibrium predictions of the IFD at each simulation cycle. Correlation and regression coefficients of 1 indicate a perfect fit to the IFD. (b) Frequency distribution of γ values among predators as a function of simulation cycle.

BERNSTEIN *et al.* (1988) start by analyzing the behaviour of the model when prey density is unaffected by predator consumption (prey are supposed to be replaced as they are consumed). A typical run of the model is

illustrated in fig. 3a. After about 30 simulation steps the system reaches a form of dynamic equilibrium in which, although some predators still move from one step to the next, the population parameters are rather stable.

The first and most important observation is that the model, although based on individual («molecular») decision rules, equilibrates close to the predictions of the IFD. This result is a strong demonstration that in an environment with many patches and in which individual predators follow rate-maximizing foraging decisions based on learning, the population can reach an IFD. In other words, it is not necessary to assume that predators are omniscient.

Figure 3b shows the time course of changes in the frequency distribution of predators among the different γ values. All individuals start with the same γ value. For each individual, this value changes with time according to the particular sequence of patches it has experienced. The distribution finishes with mean and mode values near the true γ value for the environment has a whole.

In further developments, depletion was incorporated by subtracting from each patch the prey consumed in each simulation cycle. When consumption of prey is slow the distribution of predators is close to that in the non-depleting environment and fits the IFD. As the rate of consumption of prey per predator increases, the data depart progressively from the IFD but at a final accumulated mortality of about 50 this departure is still not large. Figure 4 shows the effect of a high foraging efficiency of predators (final consumption of prey was 88.5%). In this case, the fit to the IFD is poor and decreases as time goes on. Two phenomena contribute to this result: firstly, as prey are consumed differentially between patches, between-patch variability in prey density changes, and the speed of this process depends on predators efficiency; secondly, there is a complex effect of the relation between the rates of learning and depletion.

Figure 5 shows the behaviour of the model in terms of the spatial distribution of prey mortality. Figure 5a shows a clear density-dependent mortality in the conditions of slow prey consumption and good fit to the IFD. In the case of high prey consumption and poor fit to the IFD (as in fig. 4) prey mortality is inversely density-dependent (fig. 5b).

The Distribution of «Omniscient» Predators

As I discussed above, some previous models of the response of predators to patchily distributed prey had assumed that predators were able to assess the quality of all patches instantaneously. BERNSTEIN *et al.* (1988) modified

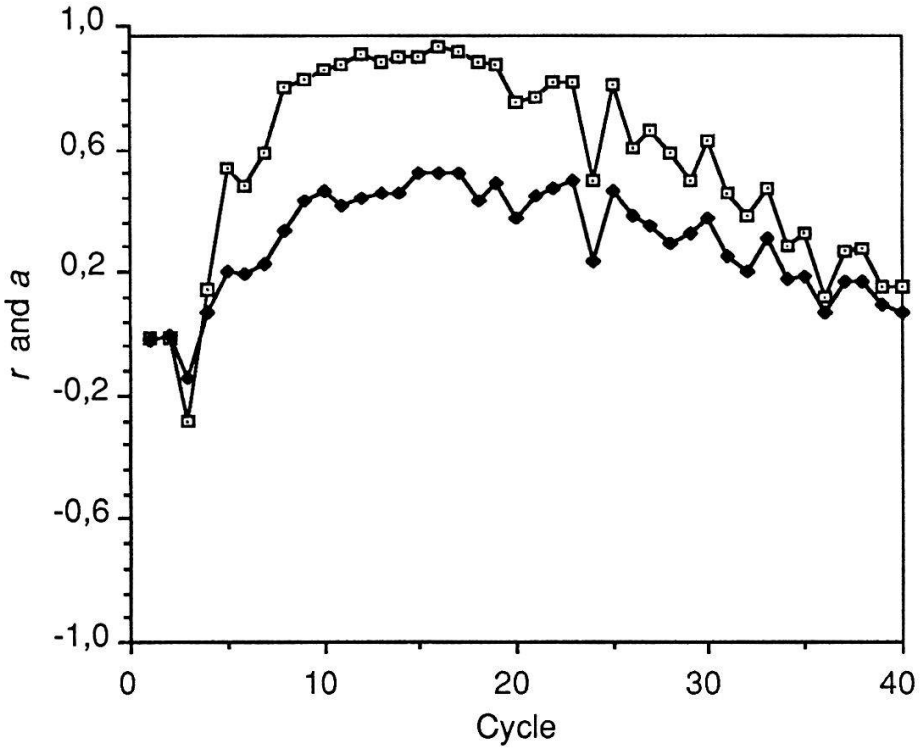

Figure 4.– The effect of rapid depletion. As in fig. 3a but with a high capture rate and without replacement of consumed prey.

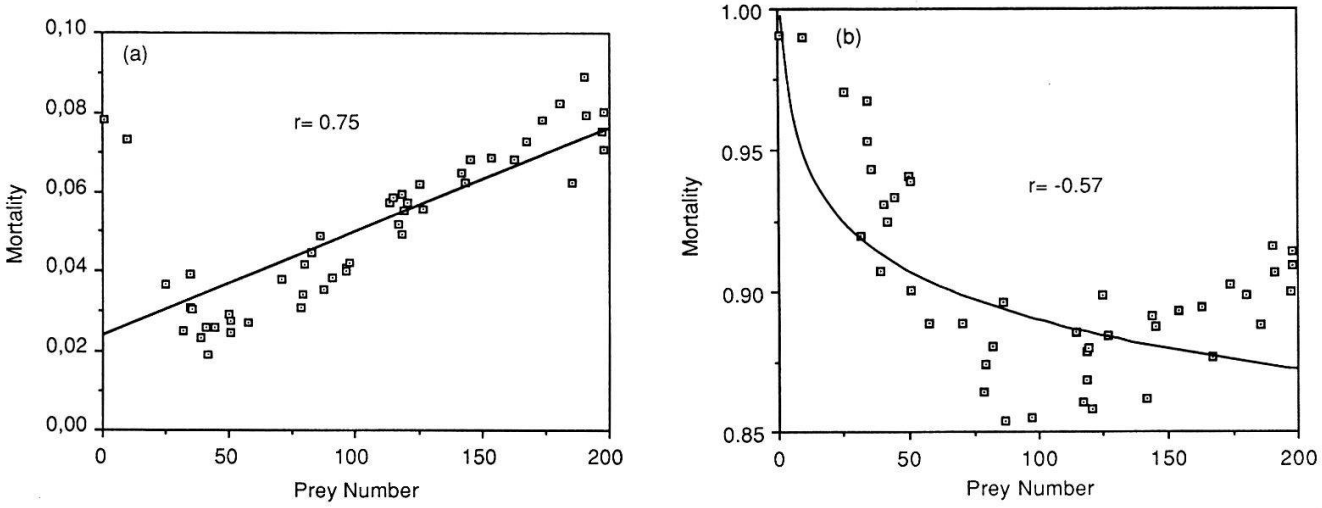

Figure 5.– Learning predators and density-dependent prey mortality. Spatial distribution of cumulative prey mortality after 40 stimulation steps. (a) Under conditions of slow prey consumption, prey mortality is density dependent. (b) When consumption is fast, prey mortality becomes inversely density-dependent.

their model in order to examine the implications of this assumption. The change consisted in replacing the learning mechanism by the rule that all predators were permanently «informed» of the exact value of the average capture rate in the environment. Predators still leave their patch when average overall capture rate is higher than that experienced in the current patch, but now they know both the environment and the patch in which they are. Predators also know which patches are above the average q and thus migrants are randomly assigned within the set of patches with q higher than average.

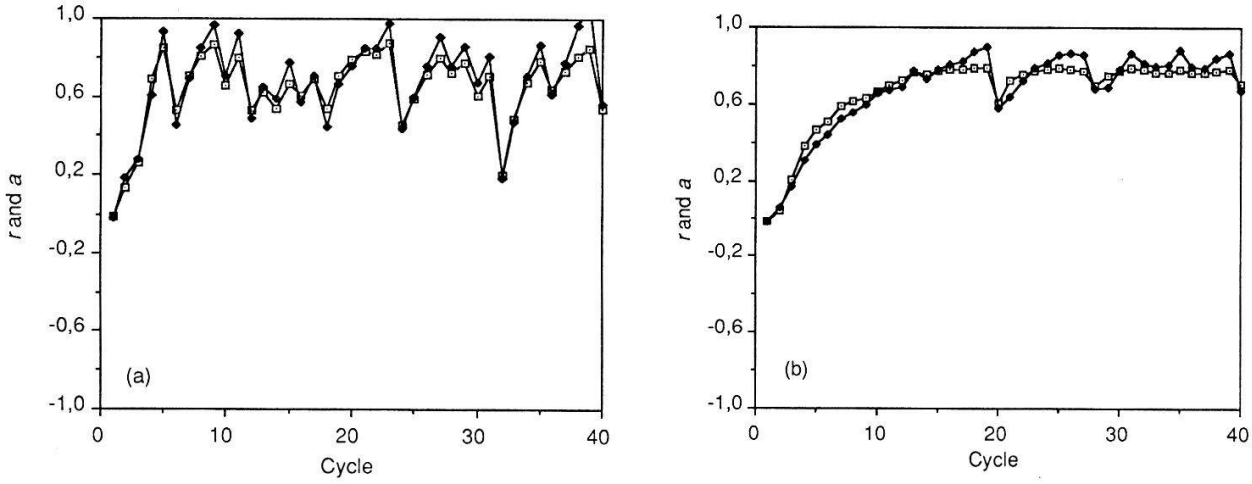

Figure 6.– The spatial distribution of «omniscient» predators. Behaviour of BERNSTEIN *et al.* (1988) modified model (under the same conditions as fig. 3; see text). (a) All predators experiencing a capture rate lower than the average are allowed to migrate; (b) only a fraction of 0.4 are allowed to migrate.

Figure 6a shows the behaviour of this model in the same conditions as in fig. 3. The distribution of predators is much farther away from the IFD than the «learner» model. One possible reason for this rather surprising result is that, although the predators are modelled as having good information about the opportunities in patches other than their current patch, they do not take into account the fact that all other predators have the same information. This property may affect the system because all predators may move at once to the best patches, and thus turn them into very bad patches in the next time step. This property may be seen as an artefact of the simulation procedure, as it depends on all predators being equally informed and taking migrating decisions in synchrony. Figure 6b shows the result of reducing the effect of synchronous migration by allowing only a fraction (40% in this case) of all the possible migrants to migrate in each time step. Although the value of 40% was chosen after several runs because it gave the best

fit to the IFD, the fit to the IFD is still below that of the learner model. These results strongly suggest that, perhaps counter-intuitively, individual differences in past foraging experience could make it more likely for a population to converge to the IFD. This is so because some individuals, as a consequence of the availability of prey in the particular patches they had traversed, would have a relatively low estimate of γ and would «accept» to settle longer in patches of lower food availability, thus smoothing out the process of converging to equilibrium.

CONCLUSIONS

I have reviewed here a few examples of the influence of individual differences on the behaviour of predator-prey and host-parasitoid systems. Although scanty, these examples illustrate well that ignoring these differences can lead to misleading conclusions. In the cases analyzed here, larger differences seem to promote the «good behaviour» or stability of the populations. This needs not always be the case. PARKER and SUTHERLAND (1986) have shown that differences in susceptibility to interference can preclude the population from reaching the IFD. (In fact, as they show in such a case, this is not the equilibrium distribution.)

An interesting point related to the results presented here is that of the interaction between stabilizing processes. Other examples of interaction between processes are given by BAILEY *et al.* (1962), HASSELL and MAY (1973) and HASSELL *et al.* (1983). Although in all these examples stabilizing processes combine to produce stronger stability, no mathematical demonstration seems to have been given that it must be the case, and any counter-example would be most interesting.

As progressively more realistic descriptions of the differences between individuals are incorporated, models seem to grow too fast in complexity. The excellent work of DE JONG (1979) on the influence of spatial heterogeneity on the stability of single population models shows that in many cases this complexity can be collapsed to expressions that are simple and easy to work with.

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