species may be regarded relatively as fire-facilitating or fire-retarding.

Apparent disasters, like fire, may be advantageous to some species relative to their interspecific competitors[17]. Pest outbreaks by generalized insects that consume several competing species may be advantageous to trees whose seeds and seedlings can dominate post-outbreak regeneration. In such a case, natural selection may have favoured herbivore-facilitating traits among some species when they reach a given age or size or when they face displacement by competitors. Where improved nutritional status of tree foliage is associated with pest outbreaks, as has been shown for balsam fir (*Abies balsamea*) and its pest, the spruce budworm (*Choristoneura fumiferana*)[18], the outcome of the outbreak may be advantageous to the offspring of adult balsam fir trees when its white spruce (*Picea glauca*) competitors are destroyed as well as the firs, and the seedling balsam firs are thus released to grow in full sunlight[17]. Given such processes, then forest management must include the possibility that some tree species may facilitate the agents of destruction which are the targets of control.

We thank the Florida Division of Forestry for assistance with the experimental fires.

Received 12 May, accepted 18 September 1981.

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Epidemic models used to explain biogeographical distribution limits

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It is an accepted principle in biogeography that the distributions of organisms are primarily controlled by the climate[1–3]; thus it is widely supposed that the geographical distribution limits of plants can be explained in terms of their physiological responses to particular climatic factors. However, such responses have rarely been demonstrated at natural distribution limits[3]; those responses that are reported generally have such small effects on survival that it is hard to explain how control could really be exercised by them. Those seeking explanations of distribution limits have, however, largely neglected the impermanence of the colonies constituting them. We suggest here that it is necessary to consider not only the effects of the climate on the performance of plants within existing sites but also the impact that these primary effects may have on the invasion of new sites. Epidemic models of the invasion process result in threshold theorems which explain how small changes in plant performance could result in large changes in the probability of new colonies appearing.

The difficulty in explaining distribution limits has been largely concealed by poorly founded statements about their cause[2,3]. Correlations between distribution limits and the isolines of synoptic climatic variables have been extensively described[4–8] but these cannot be attributed with causative significance[3,9,10], and general studies on the responses of plants to climate have rarely focused on variations as small as those that occur across natural distribution limits[7,11,12]. Physiological explanations for the limits of a few perennial plants have nevertheless been proposed[3,12]. Surprisingly, failure to detect responses has been most marked among annual ruderals, which supposedly respond to the climate alone at their limits[13]. When introduced beyond their limits, many produce copious seed and persist locally. British examples which we have studied are *Lactuca serriola*, *Kickxia spuria* and *Lathyrus nissolia*; others are *Mercurialis annua*, *Kickxia elatine* and *Melampyrum arvense*[3]. Similar observations have been reported for sedentary marine animals[14]. Present concepts of distribution-limit control implicitly require that any limit-controlling responses to the climate—whether in establishment, growth or reproduction—should ultimately reduce fecundity so that continued regeneration is impossible. In the plants mentioned above, this does not seem to be the case, which leads us to believe that their limits are controlled by the small reductions in fecundity which may with difficulty be detected in some of them. However no mechanism for this has ever been proposed. Moreover many plants are abundant at their limits[13], which are therefore abrupt relative to the climatic gradients that presumably control them, and this too is unexplained.

Little is known about the precise patterns shown by colonies of plants at their distribution limits but it is at least certain that they are constantly changing, even when the position of the limit is not. Distribution limits are abstractions from real patterns of colonies in the field and thus conceal their dynamic nature. It is therefore seldom noted that distribution limits might be controlled by climatic effects on the establishment of new colonies.

The spread of weeds has been likened to the spread of infectious diseases[15,16], but the mathematical theory developed in connection with this[17,18] has only been used by botanists to describe the dispersal patterns of plant propagules[16] (although a related, population-dynamic model has been discussed[19]). There is, however, no reason why the general deterministic epidemic model[17], which gives rise to the epidemic threshold theorem of Kermack and McKendrick[17,20], should not be used to describe the infection of new sites.

We therefore define for a species the following: x, the number of susceptible sites in an area (unoccupied sites in which the species could grow); y, the number of infective sites (occupied sites from which it disseminates seeds); β, an infection rate dependent on the numbers of seeds produced in infective sites, efficiency of seed dispersal and the probability of establishment in susceptible sites; and γ, a removal rate dependent on the length of time for which infective sites remain occupied. Following the general deterministic epidemic model[17], the rate at which susceptible sites become infective sites is given by

$$\frac{dy}{dt} = \beta xy - \gamma y$$

For the plant to spread, dy/dt must be positive, which requires $x \geqslant \gamma/\beta$. The relative removal rate γ/β therefore gives a threshold number of susceptible sites that must be exceeded if a small inoculum of the plant is to spread. Similar threshold theorems arise in epidemic models of all kinds, including stochastic and spatial ones[18,21,22]. These models may be applied to plants that occupy discrete sites and have observable rates of colonization and removal. They are largely valuable as strategic models (*sensu* May[23]) and their parameters may be difficult to estimate, although we believe this may be possible for some plants.

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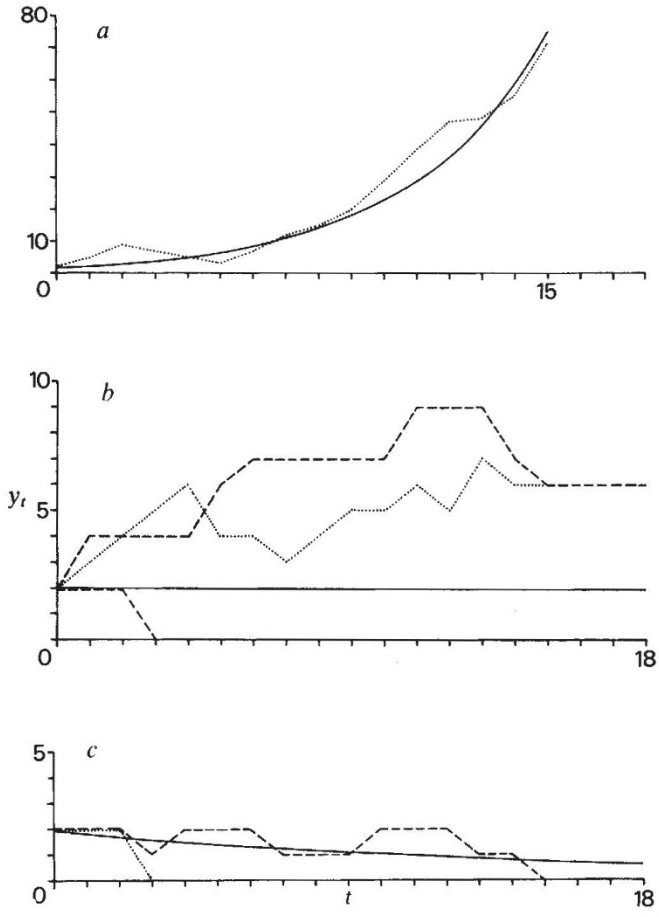

Fig. 1 The population growth of a hypothetical plant simulated in an experiment which allowed control of infection rate (β). The experiment was performed in a 24 × 8 m area of cliff-top grassland at Peartree Point, South Devon. Susceptible sites for the plant were taken as those actually occupied by rosettes of *Plantago coronopus*. In each trial two rosettes were initially selected as occupied (infective) sites. From each, a specified number of coloured disks (mean diameter 9 cm) were thrown at random and rosette-centres on which disks landed were taken as being newly occupied sites. This procedure was carried out repeatedly, the total number of occupied sites being noted at each generation, that is, after each disk-throwing. Occupied sites were allowed to remain occupied for only three generations. Variation in the seed production of the plant was simulated by altering the number of disks thrown from each occupied site in different trials of the experiment. Broken lines show observed total numbers of occupied sites in successive generations for trials in which 10 (*a*), 5 (*b*) and 4 (*c*) disks were thrown from each occupied site. On completion of the trials the actual number of rosettes in the area was determined (number of occupied 10×10 cm squares = 1,704). At generation t, the number of susceptible sites remaining (x_t) is given by $x_t = x_{t-1} - \beta w_{t-1} x_{t-1} + \gamma y_{t-1}$ where β is the probability of a particular disk landing on a particular rosette; w_{t-1} is the total number of disks throws at generation $t-1$; γ is the discrete-time removal rate for occupied sites; and y_{t-1} is the number of occupied sites at $t-1$. w_{t-1} can be rewritten as λy_{t-1} where λ is the number of disks throws from each occupied site. Similarly, the number of occupied sites at generation t is given by $y_t = y_{t-1} + \beta\lambda y_{t-1} x_{t-1} - \gamma y_{t-1}$. Non-extinction of the hypothetical plant requires $y \geq y_{t-1}$ so that a threshold condition for its survival is given by $x \geq \gamma/\lambda\beta$. The discrete-time removal rate γ can be estimated as $\gamma = 1 = e^{-\hat{\gamma}} = 0.2834$, where $\hat{\gamma}$ (continuous-time removal rate) is 1/3. The infection rate β can be estimated from the area of disk relative to the total area ($\beta = 1/30{,}181$). Substituting these estimates in $x \geq \gamma/\lambda\beta$, the threshold should have occurred at $\sim\lambda = 5$. Unbroken lines show expected values of y_t for successive generations. It can be seen that $\lambda = 10$ gives a good fit to the expected curve; $\lambda = 5$ seems to be about the threshold; extinction occurred in one trial and not in two. All trials with $\lambda < 5$ led to extinction.

Along a climatic gradient, the number of susceptible sites for a plant could decline if its vegetational niche became less frequent; the infection rate could decline if its fecundity, dispersal or establishment were impaired; and the removal rate could increase if it were eliminated more quickly by vegetational change. Within a plant's limit, the density of susceptible sites must exceed the relative removal rate. Towards the limit, changes in the climate might have small effects on the plant and its niche, which might increasingly alter the parameters of the threshold equation but the condition for spread ($x \geq \beta/\gamma$) would continue to be satisfied. Ultimately, however, further small changes might so alter the parameters that this condition was not met, and there the plant would reach its limit. In this way climatic gradients could produce abrupt plant distribution limits even though the physiological responses elicited might appear too small to explain them. This idea is illustrated by an experiment (Fig. 1) in which we simulated the effect of reducing the seed output of a hypothetical plant. The results agree closely with the predictions of a discrete-time model as to the course of the plant's spread and the seed output necessary for its survival.

Most plants occur as casuals beyond their main areas of distribution and, to explain this, existing concepts of distribution-limit control require that casuals should only occur where conditions are especially favourable; but there is no evidence for this. In epidemic models susceptible sites clearly can exist beyond the limit, provided that their density nowhere exceeds the relative removal rate, and it is not surprising that plants should sometimes establish and perform well in them. It also follows that if the density of susceptible sites in an area near to but beyond a plant's distribution limit were artificially increased so as to exceed its relative removal rate, then the position of its climatic equilibrium distribution limit should change without any change in the climate or in the plant's biology. This has been deduced theoretically[19] and agrees with the observation that the sudden spread of some long-indigenous ruderals in Britain in the 1930s was related to the extensive building of arterial roads[24]. Another consideration is that as removal rates in perennials are obviously much lower than in annual ruderals, the density of susceptible sites required for their spread is therefore also lower, so that they should approach closer to the point where growth of the individual plant is prevented by climate. This would explain why responses to the climate should be harder to observe in annuals at their limits.

Although we have been concerned with the use of epidemic models to explain geographical distribution limits, they might also be used to explain small-scale limits, for example, those determined by local edaphic factors. The success of the simulation experiment (Fig. 1) reinforces this view.

We thank Dr R. M. Anderson for advice and assistance in formulating the model shown in Fig. 1.

Received 19 February; accepted 10 August 1981.

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