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## POPULATION CONSEQUENCES OF CONSTITUTIVE AND INDUCIBLE PLANT RESISTANCE: HERBIVORE SPATIAL SPREAD

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*Abstract.*—Little attention has been paid to the impact that constitutive and inducible plant resistance traits will have on herbivore spatial dynamics. We investigate mathematical models in which herbivore demographic rates and movement rates respond to host plant quality, which in turn is determined by constitutive and inducible resistance. Models with and without induced resistance yield the same analytic expression for the asymptotic speed at which a herbivore population will spread through an initially uninduced plant population, suggesting that induced resistance will have no effect on the rate of invasion of herbivores that respond to plant resistance on small spatial scales. In contrast, constitutive resistance will influence the speed of an invasion. If herbivore movement is quite sensitive to plant quality, an increase in constitutive resistance can actually accelerate the rate of herbivore spread even while it reduces the herbivore's intrinsic rate of increase. In other scenarios, the rate of invasion attains a maximum at intermediate levels of constitutive resistance. These results argue that our view of plant resistance should be broadened to include herbivore movement if we are to understand fully the implications of differences in resistance for the dynamics of herbivore populations in natural and managed settings.

Individual plants vary in their suitability as food for herbivores, either because of relatively immutable differences in resistance traits (so-called constitutive resistance) or because of phenotypic differences induced by herbivore feeding damage (so-called inducible resistance). Numerous experiments have shown that constitutive and inducible resistance can alter demographically important rates of herbivore birth, growth, and survival (see reviews in Schultz 1988; Karban and Myers 1989; Harvell 1990; Tallamy and Raupp 1991; Kennedy and Barbour 1992), and theoretical studies have argued that the dynamics of herbivore populations may be shaped by plant resistance traits (Haukioja 1980; Rhodes 1983; Edelman-Keshet 1986; Edelman-Keshet and Rausher 1989; Adler and Karban 1994). However, the vast majority of past studies have focused on the consequences of plant resistance for the temporal dynamics of herbivores while ignoring spatial dynamics (but see Morris and Kareiva 1991; Wilhoit 1991; Lewis 1994). Because herbivores can respond to plant resistance by moving away from low-quality plants (e.g., Bernstein 1984; Harrison and Karban 1986; Power

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1991), constitutive and inducible resistance could also affect a herbivore's spatial dynamics. Recent years have witnessed a broad-scale ecological awareness of the potential importance of the spatial dimension in the dynamics of populations (Kareiva 1990; Holmes et al. 1994). One particular focus of this work has been to understand the factors that determine the rate of spread of invading organisms, such as introduced agricultural pests (Andow et al. 1990*a*, 1990*b*) or insect pathogens (Dwyer 1994). However, studies of the population consequences of plant resistance and of the spatial spread of populations have remained largely unconnected.

Our goal in this article is to link these two questions to build a better understanding of how constitutive and inducible plant resistance affect the density of herbivores in both time and space. This desire is motivated by the realization that if herbivores are mobile, then plant resistance traits need not have equivalent effects on the temporal and spatial dynamics of herbivore populations. One might naively expect that the faster that individuals are added to a herbivore population (temporal dynamics), the faster the population will spread (spatial dynamics). However, if individual herbivores disperse more rapidly from more resistant plants, then an increase in the average level of constitutive resistance in the plant population (e.g., by the introduction of novel resistance alleles through natural or artificial means) might increase the rate of herbivore population spread even as it reduces the fecundity and survival of individual herbivores. We seek to go beyond this intuitive notion by developing a more quantitative understanding of when plant resistance will have similar versus opposite effects on herbivore population growth and spatial spread.

Our approach is to use reaction-diffusion theory, a methodology that includes explicit consideration of movement behavior. In our formulation, plants vary in quality because of both constitutive and inducible resistance traits, and plant quality exerts two independent influences on herbivores: it affects demographically important rates of birth and death, and it alters the movement behaviors of individual herbivores. We analyze a simple model that tracks a population of herbivores and the quality of their host plants in continuously varying time and space, and we address the following questions: How do constitutive and inducible plant resistance influence the net rate of herbivore population growth in a spatially explicit context? How do constitutive and inducible resistance affect the rate of herbivore spatial spread, and under what conditions will the effect of resistance on spatial dynamics differ from its effects on population growth?

#### A SIMPLE MODEL OF PLANT RESISTANCE AND QUALITY-DEPENDENT HERBIVORE MOVEMENT

The simplest model of induced resistance requires two dynamic variables, one to represent herbivore density and one to represent the quality of the host plants. We follow the lead of Edelstein-Keshet (1986; see also Edelstein-Keshet and Rausher 1989; Morris and Kareiva 1991; Lewis 1994) and represent host-plant quality by a dimensionless variable  $q$ . While seemingly abstract, the variable  $q$  has a number of advantages over alternative representations of plant resistance.

Although one might argue that it would be preferable to model the level of a secondary chemical that changes after herbivore damage, the precise relationship between the concentration of particular secondary chemicals and the response of herbivores is unknown for most plant-herbivore systems. In most cases, the levels of an array of compounds change as a result of herbivory, and we do not know which of these changes (or combinations of changes) are actually responsible for altered herbivore performance. Instead, we combine all of the host plant changes that accompany herbivory into a single index that can be measured by means of bioassay experiments (Edelstein-Keshet 1986).

Because  $q$  would be estimated by measuring the responses of herbivores to plants that differ in quality, the simplest approach is to assume that  $q$  is linearly related to either the intrinsic rate of increase or the movement rate of the herbivore. We have chosen to define  $q$  in terms of herbivore movement, both because an investigation of movement is the primary goal of this article and because we expect that bioassay experiments that measure the movement rate of herbivores can be performed more quickly and easily than bioassays that require the measurement of demographic rates, at least for moderately mobile herbivores. Specifically, we assume that herbivores move at random with respect to direction but at a rate (represented by the diffusivity  $D\{q\}$  in the model) that scales linearly with  $q$ . Moreover, to give an intuitive meaning to different values of plant quality, we constrain  $q$  to vary on a scale from 0 to 1, so that herbivores move at their maximum rate when they are in the vicinity of the lowest-quality plants (i.e.,  $q = 0$ ) and at their minimum rate near the highest-quality plants ( $q = 1$ ). However, we allow the demographic rates of herbivores to respond in a variety of ways (not necessarily linearly) to our index of plant quality that is measured on the basis of herbivore movement.

We consider a herbivore population spreading in one spatial dimension, with  $x$  denoting position along the axis of spread. This scenario could apply to an insect pest that moves primarily along the rows in an agricultural field. Alternatively, the spatial axis could depict a perpendicular transect extended through the front of a large herbivore population invading from the edge of a two-dimensional plant population. We assume that the rates of change over time of the density of mobile herbivores,  $m$ , and of plant quality  $q$  are governed by the equations

$$\frac{\partial m}{\partial t} = r\{q\}m - \frac{m^2}{K} + \frac{\partial^2}{\partial x^2}(D\{q\}m) \quad (1a)$$

and

$$\frac{\partial q}{\partial t} = \mu q \left[ 1 - \frac{(q + \eta\{m\})}{\hat{q}} \right]. \quad (1b)$$

In equation (1a), we have assumed that plant quality influences the herbivore's intrinsic rate of increase,  $r\{q\}$ , but not the herbivore carrying capacity,  $K$ , both for simplicity and because  $K$  does not affect the ultimate rate of spatial spread (see eq. [3] below). The form of the diffusion term that we have adopted is ap-

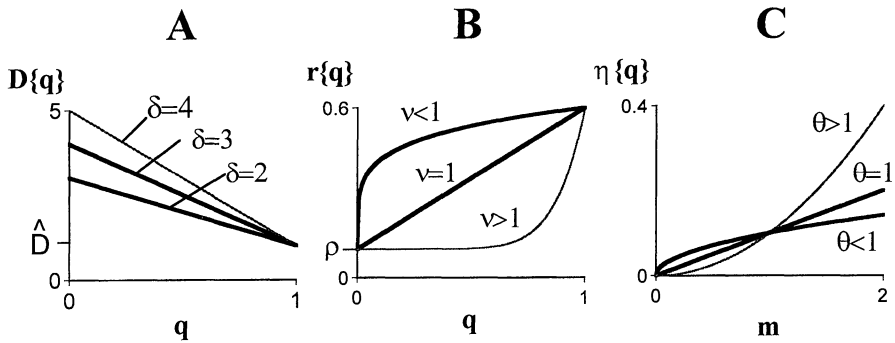


FIG. 1.—Possible responses of a herbivore's intrinsic rate of increase  $r\{q\}$  and diffusivity  $D\{q\}$  to plant quality,  $q$ , and possible responses of plant quality to herbivore density,  $\eta\{q\}$ .

appropriate for herbivores that base their rate of movement only on the quality of the plant on which they are currently located, not on the quality of neighboring plants (for alternative forms that make different assumptions about movement, see Okubo 1980, pp. 82–86; Gueron and Liron 1989; Lewis 1994). The functional form used in equation (1b) is appropriate if herbivory induces a reduction in plant quality, but quality recovers after herbivory is discontinued (as is true, e.g., of soybeans, *Glycine max*, damaged by Mexican bean beetles, *Epilachna varivestis*; N. Underwood, unpublished data). Specifically,  $\mu$  is the proportional rate at which plant quality recovers from low values, the function  $\eta\{m\}$  is the rate at which herbivory induces a reduction in plant quality, and  $\hat{q}$  is the equilibrium plant quality in the absence of herbivores (where we require that  $0 \leq \hat{q} \leq 1$ ). We use  $\eta\{m\}$  as a measure of the strength of inducible resistance (hence if  $\eta\{m\} = 0$  for all values of  $m$ , then plants lack an induced response to herbivory), and we use  $\hat{q}$  to represent constitutive plant quality (note that plants with high constitutive resistance have low  $\hat{q}$ ). Neglecting the spatial derivative, the model in equation (1) is formally similar to a modified Lotka-Volterra predator-prey equation in which logistically growing herbivores “consume” the logistically growing plant quality, but the “consumption” of quality influences only the herbivore's intrinsic rate of increase.

We have deliberately included general functions for the rates of herbivore population growth and movement ( $r\{q\}$  and  $D\{q\}$ ) and of induction ( $\eta\{m\}$ ) to allow us to calculate an expression for the rate of herbivore spread that does not depend on the exact details of induced resistance or the response of herbivores. Before making this calculation, we invest the model with specific functional forms to demonstrate the range of biologically realistic processes that the model can encompass. Under the assumption that herbivore diffusivity scales linearly with  $q$ ,  $D\{q\}$  takes the form  $\hat{D} + \delta(1 - q)$ , with a maximum  $\hat{D} + \delta$  at  $q = 0$  and a minimum at  $q = 1$  (fig. 1A). However, the intrinsic rate of herbivore increase,  $r\{q\}$  need not scale linearly with the index of plant quality that we have defined arbitrarily on the basis of movement. A reasonable functional form that allows for a nonlinear relationship between  $r\{q\}$  and  $q$  is  $r\{q\} = \rho + \gamma q^v$ ,

where  $\rho$  is the per capita herbivore population growth rate on the lowest-quality plants ( $q = 0$ ), and  $\gamma$  and  $\nu$  determine how  $r\{q\}$  changes with  $q$  (fig. 1B). If  $\nu = 1$ , then  $r\{q\}$  increases linearly at a rate  $\gamma$  as plant quality is increased. To understand the implications of values of  $\nu \neq 1$ , consider how herbivore movement  $D\{q\}$  and intrinsic rate of increase  $r\{q\}$  change as plant quality is reduced from its maximum value of  $q = 1$  (fig. 1A, B). Values of  $\nu < 1$  can be thought of as representing "skittish" herbivores; in this case, herbivore movement increases consistently as plant quality declines (fig. 1A), but there is initially little change in the population growth rate (fig. 1B). Only when plant quality reaches low values does population growth show a steep decline. Skittish herbivores that move away from damaged plants before they experience any significant demographic consequences of induced resistance could be acting to avoid natural enemies that use damage as a cue to find prey (Faeth 1985; Turlings et al. 1990). At the other extreme, herbivores with values of  $\nu > 1$  might be labeled "long-suffering," because they do not leave host plants rapidly even though they experience sharply reduced population growth on all but the highest-quality plants. A reluctance to emigrate in the face of induced resistance may simply reflect poor dispersal ability, or it may be a strategic response to a low likelihood of finding higher-quality food if host plant density is typically low.

The rate of induction  $\eta\{m\}$  may also be a nonlinear function (fig. 1C). For example, if herbivores interfere with one another's feeding at high density (so that the rate of damage is not strictly proportional to herbivore density) or if there is a maximum rate at which plants can muster an induced response, the rate at which quality declines may approach an asymptote as herbivore density increases. Alternatively, herbivores may act synergistically, so that  $q$  declines in a faster-than-linear fashion as  $m$  increases. A function that allows both these possibilities is  $\eta\{m\} = \hat{\eta}m^\theta$ , where  $\hat{\eta}$  and  $\theta$  control the initial rate and nonlinearity of induction, respectively.

Substituting these functional forms into equation (1) yields the model

$$\frac{\partial m}{\partial t} = (\rho + \gamma q^\nu)m - \frac{m^2}{K} + D\{q\} \frac{\partial^2 m}{\partial x^2} - 2\delta \frac{\partial q}{\partial x} \frac{\partial m}{\partial x} - \delta m \frac{\partial^2 q}{\partial x^2} \quad (2a)$$

and

$$\frac{\partial q}{\partial t} = \mu q \left[ 1 - \frac{(q + \hat{\eta}m^\theta)}{\hat{q}} \right]. \quad (2b)$$

Note that the dependence of herbivore diffusivity on plant quality introduces an advection term,  $-2\delta(\partial q/\partial x)(\partial m/\partial x)$ , into the equation for herbivore density. Thus, even though the direction of motion of individual herbivores is random, populations of herbivores whose rate of movement is quality dependent will move toward higher-quality plants at a rate that is proportional to the local gradient in plant quality,  $\partial q/\partial x$ . The tendency for herbivores to slow their rate of movement in the neighborhood of high-quality plants is a form of area-restricted search, a simple behavioral mechanism that has been shown to allow chemotactic bacteria to locate nutrient-rich patches (Keller and Segel 1971) and insect

predators to aggregate effectively at outbreaks of their prey (Kareiva and Odell 1987). More sophisticated modifications of dispersal in response to plant quality are possible, such as tactical orientation toward long-distance visual or chemical cues, which would alter the exact form of equation (2a) (Morris and Kareiva 1991). Because we believe that local cues will play a dominant role in the response of many herbivores (particularly insects) to plant quality, we focus on area-restricted search in this article.

To guide our exploration of the spatial model, we first consider the spatially uniform case in which  $m$  and  $q$  take on constant values for all values of  $x$  (obtained by setting the spatial derivatives to 0). To make a broad statement about the dynamic consequences of induced and constitutive resistance, we return to the general functions for herbivore increase and induction in equation (1) ( $r\{q\}$  and  $\eta\{m\}$ , respectively), and we make three biologically realistic assumptions about their properties: the intrinsic rate of herbivore increase is a monotonically increasing function of plant quality ( $[\partial r\{q\}/\partial q] > 0$ ); the rate at which herbivores reduce plant quality is a monotonically increasing function of herbivore density ( $[\partial \eta\{m\}/\partial m] > 0$ ); and in the absence of herbivores, there is no induced decline in plant quality ( $\eta\{0\} = 0$ ). The model in equation (1) has three equilibria: a trivial one in which both herbivores and plant quality are 0, an equilibrium at which herbivores are absent, and an internal equilibrium with nonzero herbivore density. In appendix A, we show that if the three assumptions hold, the first two equilibria are always unstable and the internal equilibrium is always stable. Hence, the herbivore-plant quality system described by a spatially homogeneous version of equation (1) will approach the internal equilibrium from all biologically realistic initial conditions ( $m, q > 0$ ); sustained cycles of herbivore density and plant quality are not possible in this model.

Turning now to the spatial model (eq. [2]), we find that numerical solution demonstrates that a herbivore population initially located at  $x = 0$  will invade a population of plants with initial quality  $\hat{q}$  in a traveling wave (fig. 2B; for all numerical solutions, spatial derivatives were approximated using the Crank-Nicolson method described in Crank 1975). Behind the wave, herbivore density and plant quality approach the internal equilibrium of the spatially homogeneous model. As the process of invasion progresses, the shape of the wave converges on a fixed form, the familiar traveling wave observed in the well-known Fisher equation (Fisher 1937).

To examine the spatial consequences of plant resistance, we calculate the velocity of the traveling herbivore wave, defined as the speed with which the position of a given density on the wave moves through space. In appendix B, we subject the general model (eq. [1]) to a standard analysis (Murray 1989, chap. 11) to calculate  $v\{\hat{q}\}$ , the asymptotic speed of the advancing wave:

$$v\{\hat{q}\} = 2 \sqrt{r\{\hat{q}\}D\{\hat{q}\}}. \quad (3)$$

Equation (3) is our main result, and it raises several important conclusions about herbivore spatial spread that we examine in the following section.

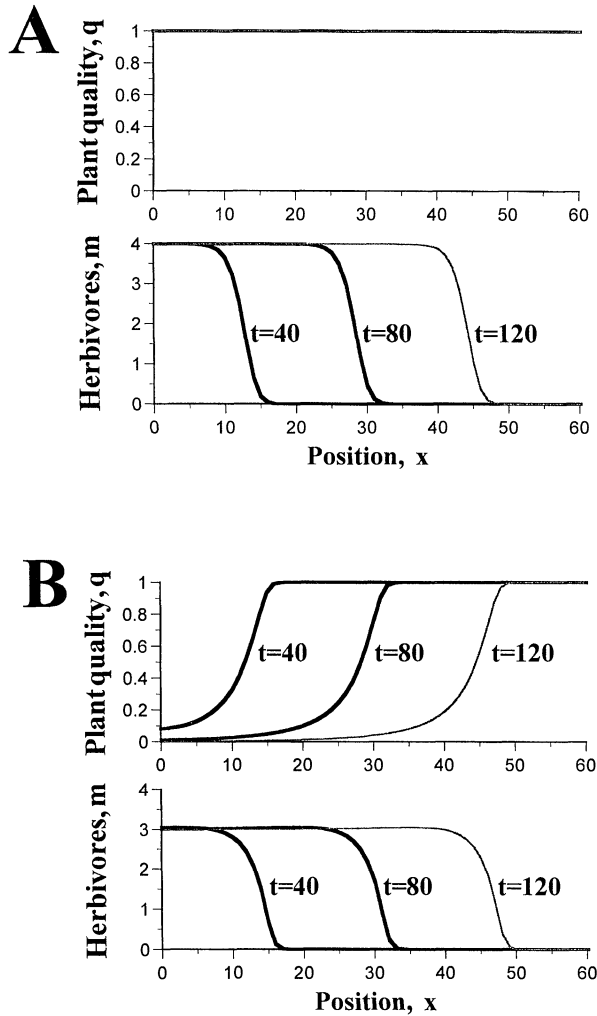


FIG. 2.—An example of induced resistance not altering the speed of a herbivore invasion according to equation (2). *A*, The changing spatial distribution of herbivores (*lower panel*) in the absence of induced resistance (constant plant quality landscape shown in upper panel). *B*, In the presence of induced resistance, plant quality declines behind the advancing herbivore front, but the rate of spread is the same as that in *A*. In both cases, the analytical wave speed is 0.4; numerically calculated wave speeds for  $100 \leq t \leq 120$  are (A) 0.394 and (B) 0.406. Parameter values are as follows:  $\rho = 0.3$ ,  $\gamma = \hat{D} = 0.1$ ,  $\delta = \theta = v = \hat{q} = 1$ ,  $\mu = 0.2$ ,  $K = 10$ , and  $\hat{\eta} = 0$  (A) or 0.4 (B). Initial conditions are as follows:  $m = 1$  at  $x = 0$ , and  $m = 0$  elsewhere;  $q = 1$  for all  $x$ . Boundary conditions are reflecting at  $x = 0$ , absorbing at  $x = 100$ .



IMPLICATIONS OF THE SIMPLE MODEL FOR HERBIVORE POPULATION  
GROWTH AND SPREAD

We now investigate how the details of a herbivore's responses to plant quality influence population dynamics on a changing-quality landscape. According to equation (3), the eventual speed of an advancing herbivore wave is governed by a simple expression involving the herbivore's rate of increase and diffusivity. It is instructive to compare the wave speed in the plant quality model with the asymptotic speed  $v_F = 2\sqrt{r_c D_c}$  predicted by Fisher's equation, which is obtained by replacing the quality-dependent rates of herbivore population growth  $r\{q\}$  and dispersal  $D\{q\}$  in equation (1a) by the quality-independent constants  $r_c$  and  $D_c$ , respectively. As in Fisher's equation, the speed of an advancing herbivore front in equation (1) depends only on the rate at which herbivores increase from low density and the rate of random movement, not on the herbivore carrying capacity. Moreover, the asymptotic wave speed for the quality-dependent model depends only on the level of constitutive plant resistance at the leading edge of the wave,  $\hat{q}$ , not on the quality of plants behind the wave or the rate at which quality changes either intrinsically ( $\mu$ ) or in response to herbivory ( $\eta\{m\}$ ).

Because plant quality enters equation (3) only through the level of constitutive resistance  $\hat{q}$ , the induction of resistance will not alter the wave's velocity through either a reduction in the herbivore's intrinsic rate of increase or an increase in its mobility. In figure 2, we show numerical solutions of equation (2) in which induced resistance is absent (A) or present (B). The numerically calculated wave speeds are nearly identical for the two cases, and by  $t = 100$ , they have already approached to within a few percentage points the asymptotic wave speed given by equation (3). The fact that the rate of herbivore spread is not affected by induced resistance is a consequence of a well-known feature of reaction-diffusion models. The asymptotic wave speed depends on the rates of "reaction" and diffusion at the leading edge of the wave, where herbivore density is low and consequently induction of plant resistance is negligible. Thus, the wave speed given by equation (3) is influenced by the assumption of local movement that is built into the use of simple diffusion to represent herbivore dispersal (see Discussion).

Although induced plant resistance behind a spreading front of herbivores does not influence the speed of spread, it does influence the rate of herbivore population growth. In comparing figure 2A and B, note that although the two waves converge on the same speed, the population attains a higher equilibrium density behind the wave in the absence of induced resistance (fig. 2A); thus, the net rate of population growth integrated over the entire spatial domain will be greater. This will be true as long as induced resistance affects the intrinsic rate of herbivore increase  $r\{q\}$  (i.e., as long as  $\gamma \neq 0$ ); inducible resistance that affects only a herbivore's motility  $D\{q\}$  will influence the transient dynamics at the initiation of an invasion but will not alter the long-term rate of population growth (verified by additional numerical simulations, which are not shown). To summarize, the simple model predicts that population growth will be influenced by the

TABLE 1

A SUMMARY OF THE EFFECTS OF INDUCIBLE AND CONSTITUTIVE PLANT RESISTANCE ON HERBIVORE POPULATION GROWTH AND SPATIAL SPREAD, ACCORDING TO EQUATION (1)

RESPONSES OF INDIVIDUAL HERBIVORES TO PLANT RESISTANCE:	LONG-TERM RESPONSES OF HERBIVORE POPULATION TO PLANT RESISTANCE	
	Population Growth	Rate of Spatial Spread
Inducible resistance (IR):		
Demographic rates respond to IR, but movement rates do not	Proportional to demographic effect	None
Movement rates respond to IR, but demographic rates do not	None	None
Constitutive resistance (CR):		
Weak demographic response but strong movement response	Weak	Spread fastest at high levels of CR
Demographic and movement responses similar in magnitude	Proportional to demographic effect	Spread fastest at intermedi- ate levels of CR
Strong demographic response but weak movement response	Strong	Spread fastest at low levels of CR

demographic consequences of induced resistance but not by its dispersal consequences, and the ultimate speed of population spread will be completely unaffected by induced resistance (table 1).

In contrast to induced resistance, constitutive resistance (through its effect on  $\hat{q}$  in front of the advancing wave of herbivores) does affect the asymptotic wave speed. The fact that the ultimate rate of spread depends on both demographic rates and rates of movement at the wave front calls forth an interesting paradox. Decreasing the constitutive level of plant quality will reduce the herbivore's intrinsic rate of increase  $r\{q\}$  but increase the rate of random movement  $D\{q\}$ . Whether altering constitutive resistance will accelerate or decelerate herbivore spread depends on a dynamic tension between these two critical rates. To illustrate the effect of changing  $\hat{q}$  on the speed of herbivore spread, we substituted some of the  $r\{q\}$  and  $D\{q\}$  curves from figure 1 into equation (3) (fig. 3). If  $r\{q\}$  is more sensitive to  $\hat{q}$  than is  $D\{q\}$ , then reducing  $\hat{q}$  (i.e., increasing constitutive resistance) will decrease monotonically the speed of the wave (fig. 3A). Conversely, if herbivore movement is more sensitive to plant quality than is the rate of population growth, reducing plant quality in front of the wave will actually increase the rate of herbivore spread (fig. 3C). In intermediate situations in which the sensitivities of population growth and movement to plant quality are similar, the herbivore population will spread most rapidly at intermediate values of  $\hat{q}$  (fig. 3B).

We illustrate in figure 4 a numerical example in which inducible resistance is absent ( $\eta\{m\} = 0$ ) and the herbivore's rate of spread is highest at intermediate levels of constitutive resistance. Note that, as was true for inducible resistance

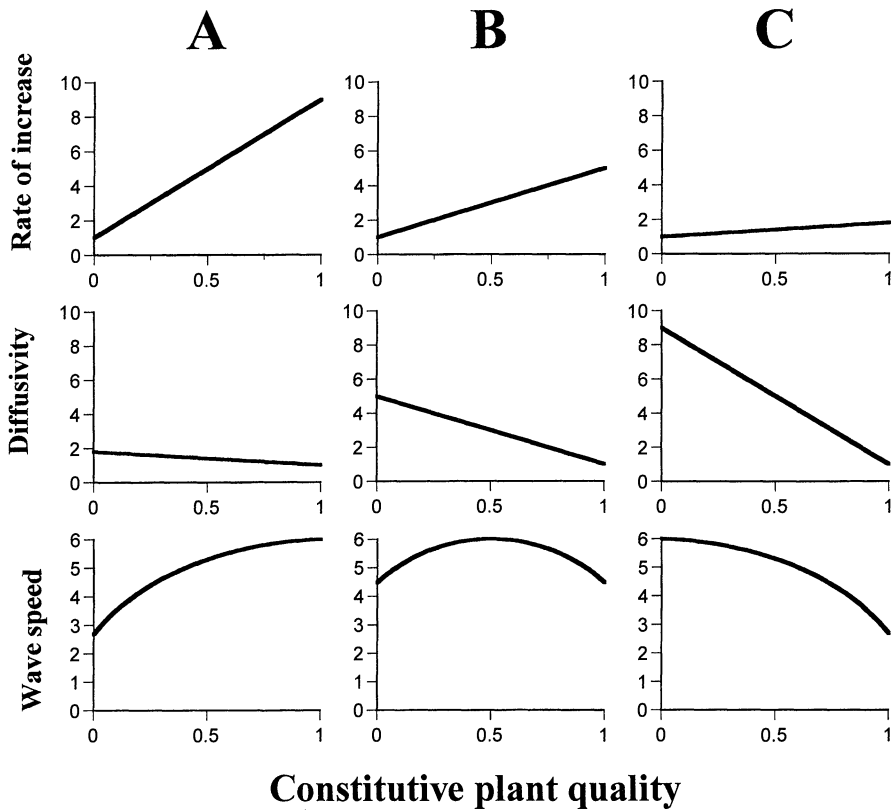


FIG. 3.—Combinations of the intrinsic rate of herbivore increase and diffusivity at the wave front ( $r\{\hat{q}\}$  and  $D\{\hat{q}\}$ , respectively) that lead to a maximum asymptotic wave speed  $v\{\hat{q}\}$  at (A) high, (B) intermediate, or (C) low levels of constitutive plant quality,  $\hat{q}$  (an inverse measure of constitutive resistance).

(fig. 2), high levels of constitutive resistance (i.e., low  $\hat{q}$ ) suppress the equilibrium herbivore density behind the wave front and thus reduce the net rate of population growth (table 1). Transient dynamics before the establishment of the stable wave form are more prolonged when  $\hat{q}$  is low (fig. 4A). Nevertheless, even for low  $\hat{q}$ , the asymptotic wave speed predicted by equation (3) is approached rapidly.

If the intrinsic rate of increase is a nonlinear function of  $\hat{q}$ , the relationship between the speed of invasion and plant quality in front of the wave can be quite complex (fig. 5). In particular, “long-suffering” herbivores may exhibit a wave speed that first increases, then decreases, and then increases again as  $\hat{q}$  is reduced from 1 to 0. This result highlights the fact that we must understand both demography and movement in detail if we are to predict how constitutive plant resistance will influence the spatial dynamics of herbivore populations.

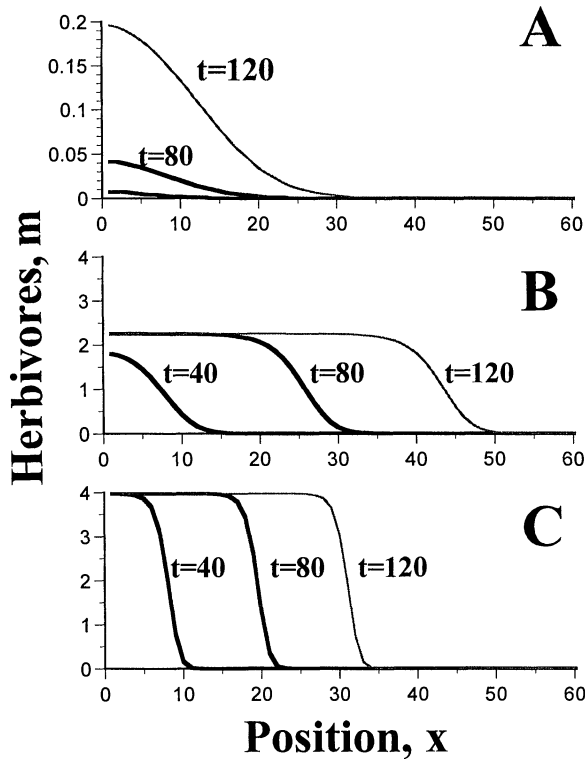


FIG. 4.—An intermediate level of constitutive resistance giving rise to the fastest herbivore spread. When a herbivore's intrinsic rate of increase and motility have similar sensitivities to plant quality (see fig. 3B), spread is slower when constitutive resistance is (A) high ( $\hat{q} = 0.01$ ) or (C) low ( $\hat{q} = 0.99$ ) than when it is (B) moderate ( $\hat{q} = 0.5$ ). In all cases illustrated, the numerically calculated wave speed is within 2% of the asymptotic speed (eq. [3]) for  $100 \leq t \leq 120$ . Other parameter values are as follows:  $\rho = \hat{D} = 0.05$ ,  $\gamma = \delta = 0.35$ ,  $\theta = v = 1$ ,  $\mu = 0.2$ ,  $K = 10$ , and  $\hat{\eta} = 0$ . Initial conditions are as follows:  $m = 0.1$  at  $x = 0$ , and  $m = 0$  elsewhere;  $q = \hat{q}$  for all  $x$ . Boundary conditions are like those in figure 2.

#### DISCUSSION

The effects of constitutive and inducible plant resistance on herbivore demographic rates have been amply demonstrated in previous studies (see contributions in Tallamy and Raupp 1991). Yet, by focusing attention on the temporal dynamics of herbivore populations, ecologists have invested too little effort in exploring the notion that resistance traits can exert different effects on herbivore spatial spread. Two results from our analyses substantiate this notion. First, induced resistance may have no influence on a herbivore's spatial spread even though it decreases the rate of population growth. Second, strong constitutive resistance can simultaneously reduce herbivore population growth and hasten the rate of spatial spread. In the following paragraphs, we explore these two results

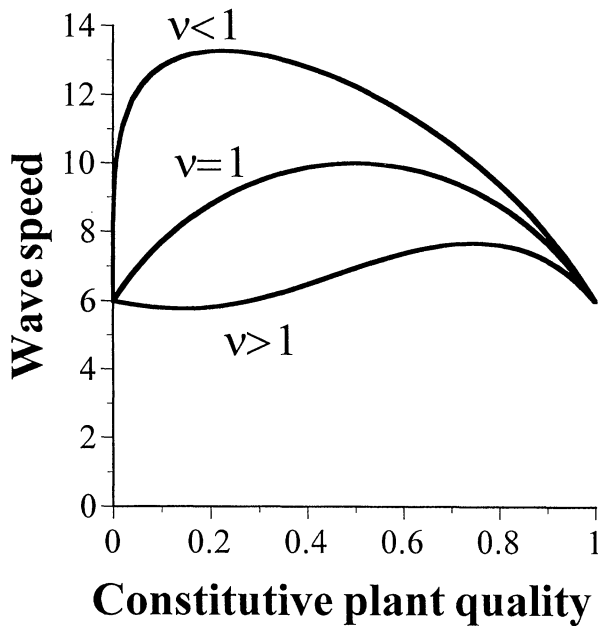


FIG. 5.—Asymptotic wave speed  $v\{\hat{q}\}$  versus constitutive plant quality  $\hat{q}$  for (top to bottom): “skittish” herbivores ( $v = 0.3$ ), neutral herbivores ( $v = 1$ ), and “long-suffering” herbivores ( $v = 2.5$ ) (see the text for an explanation). Other parameters are  $\rho = \hat{D} = 1$ , and  $\gamma = \delta = 8$ .

in more detail and then discuss their implications for the population dynamics of herbivores in natural and agricultural systems.

Despite the intuitively appealing notion that movement of individuals away from damaged plants could allow herbivores to circumvent the impact of induced resistance on population growth, our models show that this is not necessarily true for herbivores that disperse in a diffusive manner. The growth rate of an invading population is determined by both the wave speed and the equilibrium herbivore density behind the wave. For herbivores that rapidly adjust their movement to local plant quality, the wave speed is affected only by movement at the front, so that the increased rates of movement behind the front have no effect on invasion speed. The equilibrium herbivore density behind the front is also unaffected by movement rates. As a result, changes in movement rates with induced changes in resistance have no effect on population growth. These conclusions about the inability of damage-induced changes in movement to alter rates of population growth and spread will apply to herbivores whose dispersal can be adequately described as a diffusive process, which implies that movement is composed of a series of relatively short jumps whose frequency or length depends on plant quality. We believe that the relevance of these conclusions is strengthened by the observation that diffusion serves as a good descrip-

tion of dispersal for a diverse assemblage of insect herbivores (Kareiva 1983; Rudd and Gandour 1985; Andow et al. 1990*a*, 1990*b*; Plant and Cunningham 1991). Also, we emphasize that our model formulation allows for fairly complex herbivore movement behavior, ranging from simple diffusion to area-restricted search. However, we recognize that for many herbivores, dispersal is better viewed as a blend of local jumps (so-called trivial movement) and long-distance migration. The models presented above may still be useful for predicting the rate of spread of such herbivores on spatial scales sufficiently large (e.g., the regional expansion of agricultural pests or of phytophagous insects introduced for biological control of weeds) that all dispersal can be effectively treated as trivial movement. For studying smaller-scale processes, models that allow herbivores to respond to induced resistance by altering their entire spectrum of move lengths (the so-called dispersal kernel) would be a valuable extension of the models presented here.

Because the model in equation (1) yields a simple analytic expression for the asymptotic wave speed  $v\{\hat{q}\} = 2\sqrt{r\{\hat{q}\}D\{\hat{q}\}}$ , it provides a general result for exploring how constitutive resistance will influence the speed of a herbivore invasion. We have shown that elevating the levels of constitutive resistance (i.e., decreasing  $\hat{q}$ ) can either increase or decrease the rate of invasion, depending on the details of how the herbivore's rates of increase and movement respond to plant quality. If the increase in movement and decrease in growth as plant quality is reduced are of similar magnitude, the speed of invasion may reach a maximum at intermediate levels of constitutive resistance. This result should sound a note of caution for empirical studies of herbivore spread; if only a limited range of resistance is explored, we might falsely conclude that increasing constitutive resistance will always increase or always decrease the rate of spread. Finally, we point out that the rate of spread will depend on the level of constitutive resistance even if the herbivore's intrinsic rate of increase is independent of plant quality (i.e., if  $r\{q\} = r_c$ , a constant), as long as herbivores alter their movement in response to constitutive resistance. Hence, commonly conducted studies that measure only the demographic effects of plant traits on herbivores confined to a single plant may be missing a potentially important population consequence of plant resistance.

The above conclusions regarding constitutive and inducible resistance have potentially important implications for the dynamics of herbivores feeding on natural and managed plant populations. In natural plant-herbivore systems, the rate of spread of seasonally migrating or irruptive insects (e.g., monarch butterflies or locusts) may depend in part on the average levels of constitutive resistance in the plant populations through which the insects disperse. We caution that our models omit an important feature of natural plant populations, namely, variation among individual plants in their levels of constitutive resistance and their propensity to change as a result of herbivore damage. Shigesada et al. (1986) have shown that spatial heterogeneity in demographic and movement rates may have profound effects on the speed of an invasion; accurate prediction of herbivore spread in natural systems may require a second generation of models in which resistance is spatially heterogeneous (see Morris and Kareiva 1991).

Because of the assumption of uniform plant populations, the models presented here may have the greatest relevance for agricultural systems, which often experience invasions of pest herbivores that colonize the margins of agricultural fields (e.g., see French et al. 1993). In agriculturally important plants, humans have long manipulated plant resistance traits with an eye toward reducing pest numbers; most have been attempts to modify constitutive resistance (for an ecologically minded review, see Kennedy and Barbour 1992). However, some recent studies have argued that agronomists might take advantage of induced resistance to "immunize" crop plants, either by inoculating them with relatively benign herbivores (Karban and English-Loeb 1990; Karban et al. 1991) or pathogens (Kuc and Caruso 1977) or by manipulating chemical cues responsible for induction (Hare 1983; Kogan and Paxton 1983; Fischer et al. 1990). It is worth considering the consequences of both approaches for the spread and the growth of pest populations. If a pest's movement rate is more sensitive to plant quality than are its rates of birth, survival, and growth, then reducing the quality of crop plants by manipulating constitutive resistance may have the undesirable effect of hastening the spread of pest herbivores (fig. 3C). Because the induction of resistance through immunization has the effect of reducing plant quality in front of an advancing pest invasion, it may similarly exacerbate pest spread. As a result, farmers may be less able to suppress pests with localized methods (e.g., local release of biocontrol agents or restricted spraying of insecticides), pest damage and insect-vectored diseases may be spread throughout more of the crop, and the spread of resistance genes that allow the pest to overcome crop resistance may accelerate. If insects respond to plant quality as in figure 3C, then it might be better to forgo immunization but instead select crop varieties that respond with a strong induced resistance once they are damaged. This response might reduce pest density behind the wave without increasing the speed of the invasion.

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#### APPENDIX A

##### STABILITY ANALYSIS OF THE SPATIALLY UNIFORM MODEL

This appendix presents the stability analysis for the spatially homogeneous version of equation (1). Setting the spatial derivatives to 0, we obtain the set of ordinary differential equations:

$$\frac{dm}{dt} = r\{q\}m - \frac{m^2}{K} \quad (\text{A1a})$$

and

$$\frac{dq}{dt} = \mu q \left[ 1 - \frac{(q + \eta\{m\})}{\hat{q}} \right]. \tag{A1b}$$

Equation (A1) has three equilibria: a trivial one at  $(m^*, q^*) = (0, 0)$ ; a herbivore-free equilibrium  $(m^*, q^*) = (0, \hat{q})$ ; and an internal equilibrium  $(m^*, q^*) = (\bar{m}, \bar{q})$ , where  $\bar{m} = r\{\bar{q}\}K$  and  $\bar{q} = \hat{q} - \eta\{\bar{m}\}$ . The Jacobian matrix for the system of equations is

$$\mathbf{J} = \begin{bmatrix} r\{q^*\} - \frac{2m^*}{K} & m^* r'\{q^*\} \\ -\frac{\mu q^*}{\hat{q}} \eta'\{m^*\} & \mu - \frac{2\mu q^*}{\hat{q}} - \frac{\mu \eta\{m^*\}}{\hat{q}} \end{bmatrix},$$

where

$$r'\{q^*\} \equiv \left. \frac{\partial r\{q\}}{\partial q} \right|_{q=q^*}$$

and

$$\eta'\{m^*\} \equiv \left. \frac{\partial \eta\{m\}}{\partial m} \right|_{m=m^*}.$$

At the trivial equilibrium,  $\det(\mathbf{J} - \lambda \mathbf{I})$  has two positive roots (where we have made use of the assumption that  $\eta\{0\} = 0$ ); hence, the trivial equilibrium is an unstable node. The equilibrium at which herbivores are absent is a saddle point, because the characteristic equation has one positive and one negative root:  $\lambda_1 = r\{\hat{q}\}$ ,  $\lambda_2 = -\mu$ . At the internal equilibrium, we use the fact that  $r\{\bar{q}\} = \bar{m}/K$  and  $\eta\{\bar{m}\} = \hat{q} - \bar{q}$  to simplify the Jacobian:

$$\mathbf{J} = \begin{bmatrix} \frac{-\bar{m}}{K} & \bar{m} r'\{\bar{q}\} \\ -\frac{\mu \bar{q}}{\hat{q}} \eta'\{\bar{m}\} & \frac{-\mu \bar{q}}{\hat{q}} \end{bmatrix}.$$

The trace and determinant of  $\mathbf{J}$  are given by

$$\text{Tr } \mathbf{J} = -\frac{\bar{m}}{K} - \frac{\mu \bar{q}}{\hat{q}}$$

and

$$\det \mathbf{J} = \frac{\mu \bar{m} \bar{q}}{\hat{q}} \left( \frac{1}{K} + r'\{\bar{q}\} \eta'\{\bar{m}\} \right).$$

Under the assumption that  $r'\{q\} > 0$  and  $\eta'\{m\} > 0$ ,  $\det \mathbf{J} > 0$ , and because  $\text{Tr } \mathbf{J} < 0$ , the internal equilibrium is a stable node (Murray 1989). Note that  $\text{Tr } \mathbf{J}$  and  $\det \mathbf{J}$  do not change sign if we remove herbivore self-regulation by setting  $K = \infty$ . We conclude that the internal equilibrium in the spatially homogeneous model will be stable whether or not herbivore population growth is density dependent.



## APPENDIX B

PLAUSIBILITY ARGUMENTS FOR THE EXISTENCE OF TRAVELING WAVES  
AND WAVE SPEED CALCULATION

In this appendix, we show that traveling waves are topologically plausible for equation (1), and we calculate the asymptotic wave speed. We use only the three assumptions outlined in the text (i.e.,  $\partial r/\partial q > 0$ ,  $\partial \eta/\partial m > 0$ , and  $\eta\{0\} = 0$ ). We first define a moving frame of reference by setting  $m(x, t) = m(\xi)$  and  $q(x, t) = q(\xi)$ , where  $\xi = x + vt$  is the position along a wave traveling with velocity  $v$ . Making this change of variables and defining  $z \equiv dm/d\xi$ , we obtain the system of three ordinary differential equations (where primes denote differentiation with respect to  $\xi$ ):

$$m' = z,$$

$$q' = \frac{\mu}{v}q - \frac{\mu}{v\hat{q}}q^2 - \frac{\mu}{v\hat{q}}\eta\{m\}q,$$

and

$$z' = \frac{1}{D\{q\}} \left( vz - r\{q\}m + \frac{m^2}{K} + 2\delta zq \left( \frac{\mu}{v} - \frac{\mu}{v\hat{q}}q - \frac{\mu}{v\hat{q}}\eta\{m\} \right) \right. \\ \left. + \delta mq \left( \frac{\mu}{v} - \frac{\mu}{v\hat{q}}q - \frac{\mu}{v\hat{q}}\eta\{m\} \right)^2 \right. \\ \left. - \frac{\delta\mu}{v\hat{q}}mq^2 \left( \frac{\mu}{v} - \frac{\mu}{v\hat{q}}q - \frac{\mu}{v\hat{q}}\eta\{m\} \right) - \frac{\delta\mu}{v\hat{q}}mqz \frac{\partial \eta}{\partial m} \right). \quad (\text{B1})$$

To establish that traveling waves are topologically plausible, we show that conditions exist for trajectories that emerge from the critical point  $(m, q, z) = (0, \hat{q}, 0)$  far in advance of the wave ( $\xi \rightarrow \infty$ ) to join the critical point  $(m, q, z) = (\bar{m}, \bar{q}, 0)$  behind the wave. In doing so, we obtain an expression for the wave speed  $v$ .

For the wave to be biologically realistic,  $m$  must not assume negative values; hence, the linearized system near  $(0, \hat{q}, 0)$  must not show oscillatory solutions. The Jacobian matrix of the system evaluated at  $(0, \hat{q}, 0)$  is

$$\begin{bmatrix} 0 & 0 & 1 \\ -\frac{\mu}{v} \frac{\partial \eta}{\partial m} \Big|_{m=0} & -\frac{\mu}{v} & 0 \\ -\frac{r\{\hat{q}\}}{D\{\hat{q}\}} & 0 & \frac{v}{D\{\hat{q}\}} \end{bmatrix}.$$

For solutions to be nonoscillatory near the critical point, the roots of the characteristic equation

$$-\lambda \left( -\frac{\mu}{v} - \lambda \right) \left( \frac{v}{D\{\hat{q}\}} - \lambda \right) - \frac{r\{\hat{q}\}}{D\{\hat{q}\}} \left( -\frac{\mu}{v} - \lambda \right) = 0$$

must be real. Eliminating the root  $\lambda_1 = -\mu/v$  leaves the other two roots:

$$\lambda_{2,3} = \frac{v}{2D\{\hat{q}\}} \pm \frac{1}{2} \sqrt{\frac{v^2}{D^2\{\hat{q}\}} - \frac{4r\{\hat{q}\}}{D\{\hat{q}\}}}.$$

For  $\lambda_{2,3}$  to be real, we require that  $(v^2/D^2\{\hat{q}\}) - (4r\{\hat{q}\}/D\{\hat{q}\}) > 0$ , from which we obtain an expression for the minimum wave speed (see eq. [3] in the text):

$$v = 2 \sqrt{r\{\hat{q}\}D\{\hat{q}\}}. \tag{B2}$$

Next, we show that it is topologically plausible for trajectories from the critical point  $(0, \hat{q}, 0)$  to proceed to the critical point  $(\bar{m}, \bar{q}, 0)$ . The critical point  $(0, \hat{q}, 0)$  has two eigenvalues with positive real part and one eigenvalue with negative real part, with the two positive eigenvalues associated with two eigenvectors that span the unstable manifold. Perhaps the simplest topology of the  $m$ - $q$ - $z$  space that would allow traveling wave trajectories to link the two critical points in a heteroclinic orbit would be one in which the unstable manifold of  $(0, \hat{q}, 0)$  becomes the stable manifold of  $(\bar{m}, \bar{q}, 0)$ . For this to be the case,  $(\bar{m}, \bar{q}, 0)$  must have two (and only two) stable eigenvectors, and hence two eigenvalues with negative real part and one with positive real part (Kuznetsov 1995). We now show that, barring cases with purely imaginary, zero, or identical eigenvalues, the critical point  $(\bar{m}, \bar{q}, 0)$  always has two negative eigenvalues and one positive eigenvalue. The characteristic equation evaluated at  $(\bar{m}, \bar{q}, 0)$  is

$$f\{\lambda\} = \lambda^3 + \left(\frac{\mu\bar{q}}{v\hat{q}} - C\right)\lambda^2 - \left(\frac{\mu\bar{q}}{v\hat{q}}C + A\right)\lambda + \frac{\mu\bar{q}}{v\hat{q}}\left(B\frac{\partial\eta}{\partial m}\Big|_{m=\bar{m}} - A\right) = 0, \tag{B3}$$

where

$$A = \frac{1}{D\{\bar{q}\}}\left(r\{\bar{q}\} + \frac{\delta\mu^2}{v^2\hat{q}^2}\bar{m}\bar{q}^2\frac{\partial\eta}{\partial m}\Big|_{m=\bar{m}}\right),$$

$$B = \frac{1}{D\{\bar{q}\}}\left(-\bar{m}\frac{\partial r}{\partial q}\Big|_{q=\hat{q}} + \frac{\delta\mu^2}{v^2\hat{q}^2}\bar{m}\bar{q}^2\right),$$

and

$$C = \frac{1}{D\{\bar{q}\}}\left(v - \frac{\delta\mu}{v\hat{q}}\bar{m}\bar{q}\frac{\partial\eta}{\partial m}\Big|_{m=\bar{m}}\right).$$

Because  $f\{\pm\infty\} = \pm\infty$ ,  $f\{\lambda\}$  looks like one of the curves depicted in figure B1. For the required number of positive and negative eigenvalues,  $f\{\lambda\}$  must resemble figure B1B. The fact that

$$f\{0\} = -\frac{\mu\bar{q}}{v\hat{q}D\{\bar{q}\}}\left(r\{\bar{q}\} + \bar{m}\frac{\partial r}{\partial q}\Big|_{q=\hat{q}} + \bar{m}\bar{q}^2\frac{\partial\eta}{\partial m}\Big|_{m=\bar{m}}\right) \tag{B4}$$

is always negative for biologically realistic scenarios rules out the possibility that zero or two eigenvalues have positive real parts (fig. B1A, C). Finally, the fact that

$$\frac{\partial f\{0\}}{\partial\lambda} = -\frac{1}{D\{\bar{q}\}}\left(r\{\bar{q}\} + \frac{\mu\bar{q}}{\hat{q}}\right)$$

is always negative precludes the possibility of three eigenvalues with positive real parts (fig. B1D). Hence, the scenario of figure B1B is the only possibility when all three eigenvalues of the critical point  $(\bar{m}, \bar{q}, 0)$  have nonzero real parts, thus establishing the plausibility of one particular topology that allows traveling wave solutions.

We have not been able to rule out other plausible topologies, including the possibility that orbits join the critical points  $(0, 0, 0)$  and  $(0, \hat{q}, 0)$  or that trajectories from  $(0, \hat{q}, 0)$  join a closed loop, such as a limit cycle, around  $(\bar{m}, \bar{q}, 0)$ . Given the large number of different conditions under which such loops can occur in three-dimensional vector spaces (Kuznetsov 1995), excluding such trajectories is beyond the scope of this article. Instead, we simply

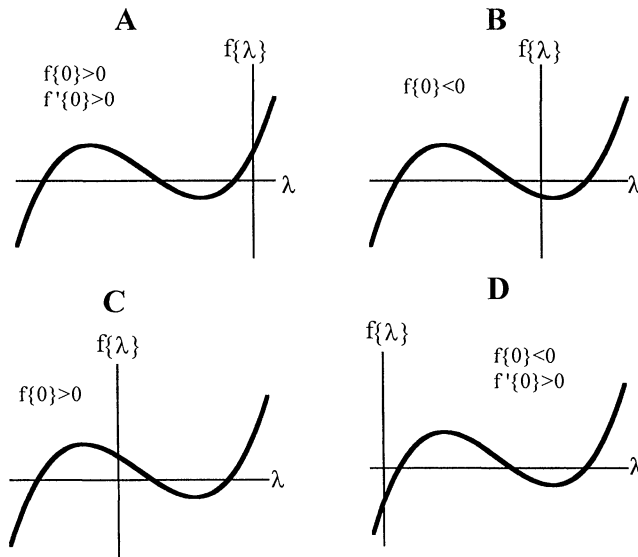


FIG. B1.—Possible shapes of the function  $f\{\lambda\}$  in the characteristic equation (eq. [B3]) that yield (A) zero, (B) one, (C) two, or (D) three eigenvalues with positive real parts.

note that, in our simulations, we have never observed oscillations in trajectories that approach  $(\bar{m}, \bar{q}, 0)$  from  $(0, \hat{q}, 0)$ , which suggests that all such trajectories are asymptotically stable near  $(\bar{m}, \bar{q}, 0)$ . Moreover, the impossibility of oscillations around the internal equilibrium of the spatially homogeneous model (app. A) suggests that they may also be precluded near  $(\bar{m}, \bar{q}, 0)$  in the spatial model.

Strictly speaking, traveling waves in Fisher's equation do not approach the analytical wave speed  $v_F = 2\sqrt{r_c D_c}$  for all initial conditions (Mollison 1977; Murray 1989), and we suspect that the same is true for waves in equation (1). Proving that waves traveling at the minimum speed given in equation (B2) are the only stable waves for a given set of initial conditions is extremely difficult (Murray 1989). The initial condition of the greatest biological interest, however, is a point release of herbivores into a plant population of spatially constant quality, and for such initial conditions, our numerical simulations always approached the minimum speed.

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