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Investing for survival of rare severe stresses in heterogeneous environments

Dan Cohen[1]* and Marc Mangel[2]

[1]*Department of Evolution, Systematics and Ecology, The Silberman Institute of Life Sciences, The Hebrew University of Jerusalem, Jerusalem 91904, Israel and* [2]*Department of Environmental Studies, University of California, Santa Cruz, CA 95064, USA*

ABSTRACT

We model the ESS investment of limiting resources for survival of rare severe stresses, with an emphasis on investment by trees in mechanical strength for survival of storm stresses. The basic model includes the effects of the fitness benefits of increased survival, the cost in reproduction, the effects of the distributions and timing of the stress in the population, and of stress-independent mortality. The ESS investment in survival of unsynchronized stresses increases if stress-independent mortality decreases, and if the cost of the resistance decreases. The ESS investment increases, and then decreases, when the probability of extreme stresses increases. The fitness of each individual increases if the allocation of resources for resisting stress is optimally adapted to its local stress probability distribution. A Bayesian model is constructed for updating the estimate of the local stress probability distribution, which each individual can get from the exposure to sub-lethal stresses during its life. This estimate can then be used for the ESS investment. The results are discussed and applied to a wider class of organisms, stresses and resistance mechanisms.

Keywords: Bayesian updating, ESS survival, exponential distribution, rare stress, storm damage, threshold stress, trees.

INTRODUCTION

The allocation of limiting resources between reproduction and survival is a fundamental problem in the evolution of life-history characteristics of most plants and animals (e.g. Stearns, 1976, 1992; Sarukhan and Dirzo, 1984). Usually, mortality or damage caused by environmental stresses or by predation can be reduced or avoided by greater investment in resistance or defence mechanisms – for example, in chemical and mechanical defences of plants against herbivory (Karban and Baldwin, 1997) – and in mechanical support against wind damage (Coutts and Grace, 1995). However, such investment in resistance usually results in reduced growth rate or reproduction. Long-term evolution is expected, therefore, to lead to an investment allocation strategy that maximizes the net gain in long-term lifetime fitness. In general, therefore, a higher investment in resistance is expected if the cost in reproduction of the resistance decreases.

* Author to whom all correspondence should be addressed. e-mail: dancohen@vms.huji.ac.il

In addition, a higher stress-independent mortality is expected to decrease the benefit of investing resources in survival against rare stresses, because the benefit will affect a smaller fraction of surviving individuals. The optimal investment in surviving stresses is expected, therefore, to decrease with increasing stress-independent mortality for the same cost of investment.

The optimal investment in resisting stress may be a decreasing or an increasing function of the intensity of the stress or the effectiveness of the investment, depending on whether they increase or decrease the marginal benefit of investment in resistance relative to the marginal increase of the cost. This is a general property of optimal allocation models in economics (e.g. Varian, 1984) and in ecology (e.g. Givnish, 1986).

As different individuals of the same species may be exposed to different environmental conditions with different probability distributions of stress or of the cost of defence, fitness could be increased considerably if the allocation of resources to resisting stress by each individual were adapted to its local conditions. An environmentally induced phenotypic increase of investment in resistance as a response to stress or to correlated signals has been reported, including for wind (Coutts and Grace, 1995) and herbivory (Karban and Baldwin, 1997).

A basic difficulty with the induced allocation of resources to defence or resistance is that it must depend on limited and unreliable information that each individual can have about the probability distribution of stresses in its immediate environment. This is especially true for resistance against rare extreme lethal events that occur infrequently in the lifetime of the individuals, because direct learning by experience is inherently impossible in such cases. The optimal induced investment in resistance has to take into account the inherent uncertainty of this information.

In this paper, we construct a simple model for the evolutionarily stable investment in resistance against environmental stresses. We provide an expression for the optimal investment as a function of the probability distribution of the stress and of the reproduction cost function of the resistance. We then analyse and discuss the problem of the optimal use of environmental signals for inducing and regulating the resistance investment, and propose some partial solutions.

THE BASIC MODEL

We consider the investment in mechanical strength by trees against the mechanical stress and damage caused by wind and snow. In this system, both the mechanical investment and the damage can be observed and measured easily, and there are many reported observations and measurements of the effects of wind intensity and other local conditions on wind damage in trees (e.g. Foster, 1988; Quine, 1988, 1995; Foster and Boose, 1995). For example, an average of 0.2–0.4% mean destruction per annum was observed in New Zealand soft wood plantations, with a large variation between sites. The worst damage was caused by sudden increases in exposure (Somerville, 1995). Also, many trees modify their mechanical strength and structure in response to local wind exposure and other environmental factors (Mattheck, 1991; Stokes *et al.*, 1995; Telewski, 1995). The relative investment in mechanical strength in trees increases, and their wind survival decreases, as trees increase in size and age (Nielsen, 1995; Telewski, 1995).

The results of this model are quite general, however, and can be applied to a much wider class of organisms and resistance mechanisms against other rare stresses. Analogous

examples are mechanical support in a wide range of non-arboreal plants, including aquatic plants, and the mechanical strength of skeletal parts of animals. Predation and herbivory risks and defences can also be included in the most general extension of the model.

Most interesting are the predicted effects of the probability distribution of stress, and of the effectiveness of investment, on the optimal investment in resistance. An increased effectiveness can confer the same resistance at a lower level of investment, and thus may be expected to reduce the optimal investment. On the other hand, an increased effectiveness increases the average survival and, in this case, will be expected to increase the optimal investment. The opposite effects are caused by an increase or decrease of the stress intensities. An increased stress intensity may select for a higher optimal investment in resistance, if the benefit increment is higher than the cost increment, or for a lower optimal investment in resistance if the opposite occurs. In our model, we show that both effects can occur at different ranges of the parameters of the model.

PART I: A UNIFORM ENVIRONMENT

We begin by considering non-synchronized, individual random stresses based on the following assumptions:

1. We consider a population of a single species of trees extending over a large area of a uniform environment with a constant density of equivalent habitable sites.
2. To simplify the presentation, we assume that stresses that affect survival hit individual trees randomly and independently at an intensity I with a probability density function $f(I)$, which is the same over the whole area in all years, after seed production for that year (e.g. in the winter).
3. There is an additional stress-independent mortality probability m for any one tree in any one year.
4. All vacant sites are occupied the following year by seeds that are uniformly dispersed over the whole area and germinate the next year without dormancy. The probability of occupying a site by any one seed is inversely proportional to the number of seeds at that site (i.e. according to the Lottery Model). For simplicity, we ignore the details of the age structure and assume a steady-state distribution of the population.
5. The probability that a tree exposed to stress I and investing a fraction R of its resources in resistance survives the stress, $S(I,R)$, is a decreasing function of I and an increasing function of R. We assume no stress damage in addition to mortality.
6. Seed production is reduced by a factor $1 - C(R)$, where $C(R)$ is the relative cost function in seed production of the investment in resistance.

These are reasonable simplifying assumptions that allow us to construct analytical models for the ESS fraction R^* of resources invested by trees in the survival of wind stress.

The growth rate of a rare type

The evolutionarily stable strategy (ESS) investment fraction R^* is derived by analysing the stability conditions for a large population in which all individuals invest evolutionarily stable R^* in resistance against the increase of a rare type with R_i investment.

A rare type i ($N_i \ll N$) will change its density N_i by a constant growth factor r_i in any one year. The numbers of the rare type i in year $t+1$ are related to those in year t by

$$N_i(t+1) = N_i(t)(1-m)E\{S(I, R_i)\} + (N(t)[1-(1-m)E\{S(I, R^*)\}] + N_i(t)[1-(1-m)E\{S(I, R_i)\}]) \frac{N_i(t)(1-C(R_i))}{N_i(t)(1-C(R_i)) + N(t)(1-C(R^*))} \quad (1a)$$

where the first term on the right-hand side of (1a) is the mean survival of the rare type and the second term is its mean occupation of vacant sites.

Defining the annual growth factor $r_i = [N_i(t+1)]/[N_i(t)]$, and assuming that $N_i(t) \ll N(t)$, we get the simplified expression for the growth of the rare type:

$$r_i = (1-m)E\{S(I, R_i)\} + [1-(1-m)E\{S(I, R^*)\}]\frac{1-C(R_i)}{1-C(R^*)} \quad (1b)$$

To find the conditions for the evolutionarily stable investment in resistance R^*, we differentiate r_i with respect to R_i and set $R_i = R^*$, which gives

$$\frac{\partial r_i}{\partial R_i} = (1-m)\frac{\partial E(S(I, R_i))}{\partial R_i} - \frac{1-(1-m)E(S(I, R))}{1-C(R^*)}\frac{\mathrm{d}C}{\mathrm{d}R_i}[R_i = R^*] \quad (2a)$$

There is an ESS $R^* = 0$ if the right-hand side of (2a) is negative at $R_i = 0$, that is, when

$$\frac{\partial E(S(I, R_i))}{\partial R_i} - \frac{1-(1-m)E(S(I, R))}{(1-m)}\frac{\mathrm{d}C}{\mathrm{d}R_i}\frac{1}{1-C(R^*)}[R_i = R^* = 0.0] < 0 \quad (2b)$$

For ESS $R^* > 0$, we set the right-hand side of (2a) equal to 0, and $R_i = R^*$, to obtain an implicit equation for the ESS $R^* > 0$:

$$\frac{\partial E(S(I, R))}{\partial R} = \frac{1-(1-m)E(S(I, R^*))}{(1-m)}\frac{\mathrm{d}C}{\mathrm{d}R}\frac{1}{1-C(R^*)}[R_i = R^*] \quad (3)$$

The general solution for the ESS R^* depends on the cost function, the survival function at any level of stress, the probability distribution of the stress, and the non-stress mortality m. Even at this general level of analysis, it can already be seen that $R^* = 0$ becomes more likely and $R^* > 0$ decreases as the mortality coefficient m increases. $R^* = 0$ is also more likely when the derivative of the cost function increases and the derivative of expected survival decreases at $R = 0$.

In the following sections, we derive the ESS R^* for several representative simplifying assumptions about the characteristic functions of the model.

A threshold survival function

Let us assume a threshold survival function such that

$$S(I, R) = \begin{cases} 1 & \text{if } I < AR \\ 0 & \text{otherwise} \end{cases} \quad (4)$$

where A is a coefficient of effectiveness of the investment in resisting the stress. A threshold survival function is a reasonable assumption for the damage caused by uprooting or breaking of trees by extreme wind stress. The mean annual expected survival in the population is

$$E\{S(I, R)\} = \int_0^{AR} f(I)\mathrm{d}I = F(AR) \tag{5}$$

where $f(I)$ is the annual probability density function of the wind stress I, and $F(I)$ is the cumulative probability distribution of I. The implicit equation for the ESS $R^* > 0$ (equation 3) then becomes:

$$(1-m)A\,f(AR^*) = \frac{1-(1-m)F(AR^*)}{1-C(R^*)}\frac{\mathrm{d}C}{\mathrm{d}R}\,[R_i = R^*] \tag{6}$$

For simplicity, we replace R^* by R and rearrange equation (6) as

$$\frac{\mathrm{d}C}{\mathrm{d}R}\frac{1}{1-C(R)} = \frac{(1-m)Af(AR)}{1-(1-m)F(AR)} \tag{7}$$

We call the left-hand side of (7) the cost derivative function and the right-hand side of (7) the benefit derivative function. We can also use equation (7) to determine when $R^* = 0$. In particular, if $C(0) = 0$, and if $\mathrm{d}C/\mathrm{d}R_{[R=0]}$ exceeds the right-hand side of (7) at $R = 0$, then $R^* = 0$. Equation (7) is the implicit solution for ESS $R^* > 0$.

The exponential stress function

Next, we specify the probability distribution of the stress by assuming that it is exponentially distributed with parameter $L = 1/I_m$, so that

$$P_r\{i < I < i + d_i\} = L\,\mathrm{e}^{-L_i}d_i = 1/I_m\mathrm{e}^{-I/I_m} \tag{8}$$

where L is 1/(mean $I = I_m$). The exponential probability distribution function of wind stress is a good approximation for the higher end of the observed and measured distributions of wind speed and stress (e.g. Hanna *et al.*, 1995). The cumulative probability distribution then becomes $F(I) = 1 - \mathrm{e}^{-I/I_m}$. In this case, the condition (7) for ESS $R^* > 0$ becomes

$$\frac{\mathrm{d}C}{\mathrm{d}R}\frac{1}{1-C(R)} = \frac{(1-m)(A/I_m)\,\mathrm{e}^{-(A/I_m)R}}{m+(1-m)\,\mathrm{e}^{-(A/I_m)R}} \tag{9}$$

If $C(0) = 0$, the condition that ESS $R^* = 0$ is now

$$\frac{\mathrm{d}C}{\mathrm{d}R_{|R=0}} \geq (1-m)A/I_m \tag{10}$$

The condition for ESS $R^* = 0$ is satisfied over a smaller range of parameter values when the effectiveness coefficient A increases and the mean stress I_m decreases, and when the mortality coefficient m decreases. The benefit derivative function – that is, the right-hand side of (9) – is a decreasing function of R for $m > 0$, and decreases more strongly as m increases. Thus, $R^* > 0$ always decreases when m increases (Fig. 1c). In equations (9) and (10), and in all the subsequent equations, the effectiveness ratio A/I_m can be considered as *one* variable, which scales the effectiveness coefficient A in units of the mean stress I_m.

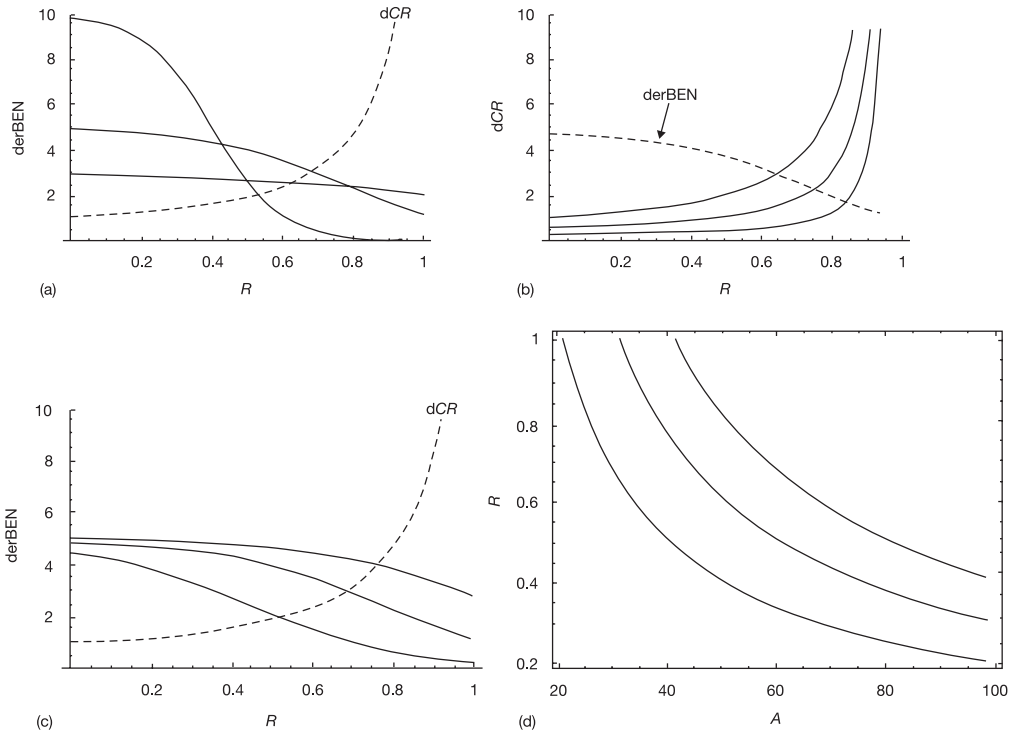

Fig. 1. The effects of the parameters of the model on the benefit derivative and the cost derivative as functions of R, as derived in equation (13). (a) The effects of the scaled effectiveness $A/I_m = 3, 5, 10$ ($I_m = 10$; $A = 30, 50, 100$) on the benefit derivative derBEN, plotted on the y axis as a function of R, and on ESS R^*. $m = 0.02$. The benefit derivative is an increasing function of A/I_m at low levels of R and a decreasing function of A/I_m at high levels of R. The cost derivative dCRk, with $k = 1$, is also plotted (dashed line). (b) The effects of the cost coefficient k on the cost derivative dCRk as a function of R, and on R^*. $k = 0.2, 0.5, 1.0$. The benefit derivative derBEN for $A/I_m = 1$ is also plotted (dashed line). (c) The effect of the mortality coefficient m on the benefit derivative derBEN and on R^*: $I_m = 10$, $A = 50$, $m = 0.005, 0.02, 0.1$. The cost derivative dCR, with $k = 1$, is also plotted (dashed line). In (a)–(c), ESS R^* is given by the intersections between the benefit derivative functions and the cost derivative functions. (d) A phase plane plot of the zero isocline of the derivative with respect to A of the benefit derivative function, as a function of A and R. The three curves give the zero solution in the two-dimensional space of R and A with $I_m = 10$, for $m = 0.005$, 0.02 and 0.1. For each curve, the area below and left of the line represents the positive domain, where the benefit derivative function is an increasing function of A.

The effect of the effectiveness ratio A/I_m on R^*

The right-hand side of equation (9) (i.e. the benefit derivative function) is an increasing function of the effectiveness ratio A/I_m at low levels of R, less than a crossover point R_b, and a decreasing function of A/I_m at high levels of $R > R_b$. R_b is the zero solution of the derivative of the benefit derivative function with respect to A/I_m. The crossover point R_b is a decreasing function of A/I_m, and is also a decreasing function of m (see Fig. 1d). The ESS R^* is thus an increasing function of mean stress if R^* is greater than R_b, or a decreasing

function of mean stress if R^* is less than R_b in different habitats. Thus, the ESS $R^* > 0$ always increases and then decreases as a function of increasing effectiveness ratio A/I_m, with an intermediate maximum for any particular cost and mortality parameters. The effects of A and I_m on R^* are, therefore, always in opposite directions.

The crossover point between any two particular levels of effectiveness ratio A/I_m, R_{b2}, can be obtained by numerically solving equation (11). Because it is only the ratio A/I_m that enters into the equations, we set $A = 1$. For simplicity we let $1/I_m = L$, and for definiteness assume that $L_1 > L_2$. The two benefit derivative functions then intersect at the value of R_{b2} such that

$$\frac{L_1 \, e^{-(L_1 AR)}}{m + (1-m) \, e^{-(L_1 AR)}} = \frac{L_2 \, e^{-(L_2 AR)}}{m + (1-m) \, e^{-(L_2 AR)}} \tag{11}$$

R_{b2} is an increasing function of the difference $L_1 - L_2$ for any given L_2, and is a decreasing function of the mortality coefficient m (see Figs 1a and b).

The cost function

Plausible simple assumptions for the relative cost function $C(R)$ are that $C(0) = 0$, it is monotonically increasing, and becomes 1 at high levels of $R = 1$. We choose $C(R) = 1 - (1 - R)^k$, where k determines the initial slope and curvature of the cost function. The cost derivative function is

$$\frac{dC}{dR} \frac{1}{1 - C(R)} = \frac{k}{1 - R} \tag{12}$$

This cost function satisfies the reasonable assumption that, in general, the relative loss of reproductive output is an increasing function of the investment in survival. In this case, the derivative of the relative cost is an increasing function of R and k.

Combining this cost derivative function with the ESS condition (9) gives the following solution for the ESS $R^* > 0$:

$$\frac{k}{1 - R} = \frac{(1-m) A/I_m \, e^{-A/I_m R}}{m + (1-m) \, e^{-A/I_m R}} \tag{13}$$

with ESS $R^* = 0$ if $k > (1 - m)A/I_m$.

Note that if $m = 0$, the benefit derivative is constant at A/I_m, and (13) can be solved analytically for the ESS value of R^*, which is

$$R^* = 1 - \frac{k}{A/I_m} \tag{14}$$

In this case, ESS $R^* = 0$ if $k > A/I_m$.

This result is a special case of the change of the crossover between increasing and decreasing R^* as a function of A/I_m and m. With $m = 0$, R_b is infinitely high, so that the ESS investment always increases with increasing A and with decreasing mean intensity of stress I_m. We interpret it as follows: When the only source of mortality is the rare but powerful stress, the contribution of stress survival to future reproduction is enhanced, so that it is advantageous to invest even more to 'assure' survival when mean stress decreases.

Numerical results

Figures 1, 2 and 3 provide graphical illustrations of the effects of the parameters of the basic model on the benefit and cost derivatives, and on the resulting solutions for ESS R^* and for the average ESS survival of stress.

R is the fraction of resources invested in resistance, so the range of R is $0 \leq R \leq 1$. We chose a representative range of mean wind speed between 0 and 20 $m \cdot s^{-1}$, which is typical of natural mean wind speeds in exposed conditions (e.g. Hanna *et al.*, 1995). The range of the effectiveness coefficient A was chosen to include values both above and below I_m, mostly between 5 and 50. We chose a natural range of stress-independent annual mortality coefficients m between 0.005 and 0.1 per year, with 0.02 as the standard reference. We chose values of the cost coefficient k between 1 and 0.3, to represent varying costs and non-linearities.

Figure 1a illustrates the benefit derivative function. As noted above, the benefit derivative function increases with increasing A/I_m at low R below the crossover point, and decreases with increasing A/I_m above the crossover point, as illustrated in Fig. 1d. This is in contrast to the effect of the mortality coefficient m, which always decreases the benefit derivative function, as shown in Fig. 1c. Numerical examples of the cost derivative function are illustrated in Fig. 1b.

The solutions for ESS R^* were obtained by solving numerically equation (13), using the FindRoot function in the Mathematica 3.0 program by Wolfram Research. The number of iterations was varied between 50 and 100, and the damping factor between 1 and 0.1, to obtain efficient convergence.

ESS R^* is an increasing function of mean stress I_m at low levels of I_m and a decreasing function of I_m at high levels of I_m (Fig. 2a). The effect of A is similar in the opposite direction, because the effect depends only on the ratio A/I_m. The range of k has a large effect on R^* (Fig. 2b), in comparison with the effects of the range of m (Fig. 2c).

Figures 3a, b and c illustrate the effects of the parameters of the model on the ESS average survival of stress when the ESS R^* is invested:

$$\text{average ESS survival of stress} = 1 - \exp(-A/I_m\, R^*)$$

Increasing A greatly increases ESS average survival because it scales the mean stress (Fig. 3a). Decreasing the cost coefficient k has a similar large effect (Fig. 3b). Increasing m has a relatively small effect on the ESS average survival, because m has a smaller effect on R^* (Fig. 3c). A set of reference curves of unoptimized average stress survival at increasing constant levels of R between 0.1 and 1.0 are plotted in Fig. 3d.

PART II: OPTIMAL INVESTMENT IN A HETEROGENEOUS ENVIRONMENT

We now assume that the environment includes patches of habitats with different probability distributions of stress damage, either because of a different exposure to strong winds, or because the ground support changes the damage probability of any given wind stress. We also assume that the seeds produced in any one-habitat patch are uniformly dispersed over a large enough area that includes all the habitats in their representative proportions. We denote the fraction of the total habitat that is type j by p_j and the probability of surviving a stress of intensity I in habitat j when the investment is R by $S_j(I, R)$.

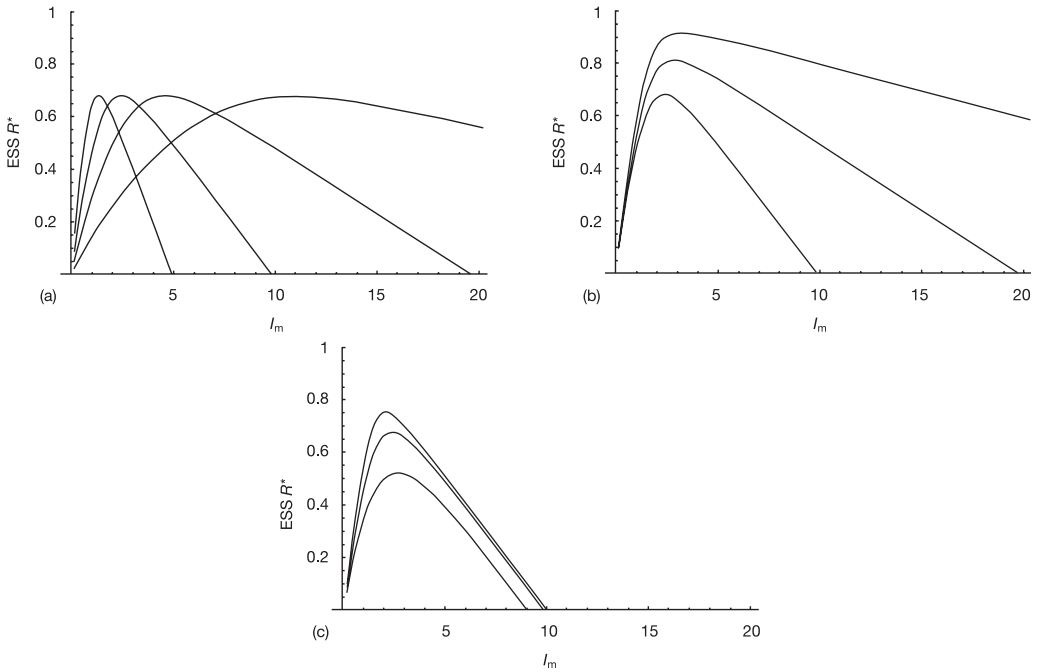

Fig. 2. The ESS investment in resistance R^*. (a) R^* as a function of I_m for different levels of effectiveness $A = 5, 10, 20, 50$; $I_m = 10$, $m = 0.02$, $k = 1.0$. Note that A is scaled by I_m. (b) R^* as a function of I_m for different levels of the cost coefficient $k = 0.2, 0.5, 1.0$; $A = 10$, $m = 0.02$. (c) R^* as a function of I_m for different levels of the mortality coefficient $m = 0.005, 0.02, 0.1$; $A = 10$, $k = 1.0$.

The model

Because of the uniform dispersing and mixing of the seeds every year, we can deal with the growth rate of the total number of the rare type in all habitats. Indicating habitat type by j, the generalization of (1b) is

$$r_i = (1 - m) \sum_j P_j E(S_j(I, R_i)) + \frac{1 - C(R_i)}{1 - C(R^*)} [1 - (1 - m) \sum_j P_j E\{S_j(I, R^*)\}] \tag{15}$$

Without discrimination by the trees between the different distributions of stress in the different habitats where they actually grow, there is an ESS R^* that is the weighted optimal strategy over all the habitats. In the general case, the ESS can be found in the manner described above. If we make the same specific assumptions as in Part I – that is, threshold survival, exponentially distributed stresses, the power cost function, and assume two habitats (with $p_1 = p$ and $p_2 = 1 - p$) – the analogue of equation (13) for the ESS R^* is

$$\frac{k}{1 - R} = \frac{(1 - m)A[pL_1 \, e^{-L_1 AR} + (1 - p)L_2 \, e^{-L_2 AR}]}{1 - (1 - m)[pL_1 \, e^{-L_1 AR} + (1 - p)L_2 \, e^{-L_2 AR}]} \tag{16}$$

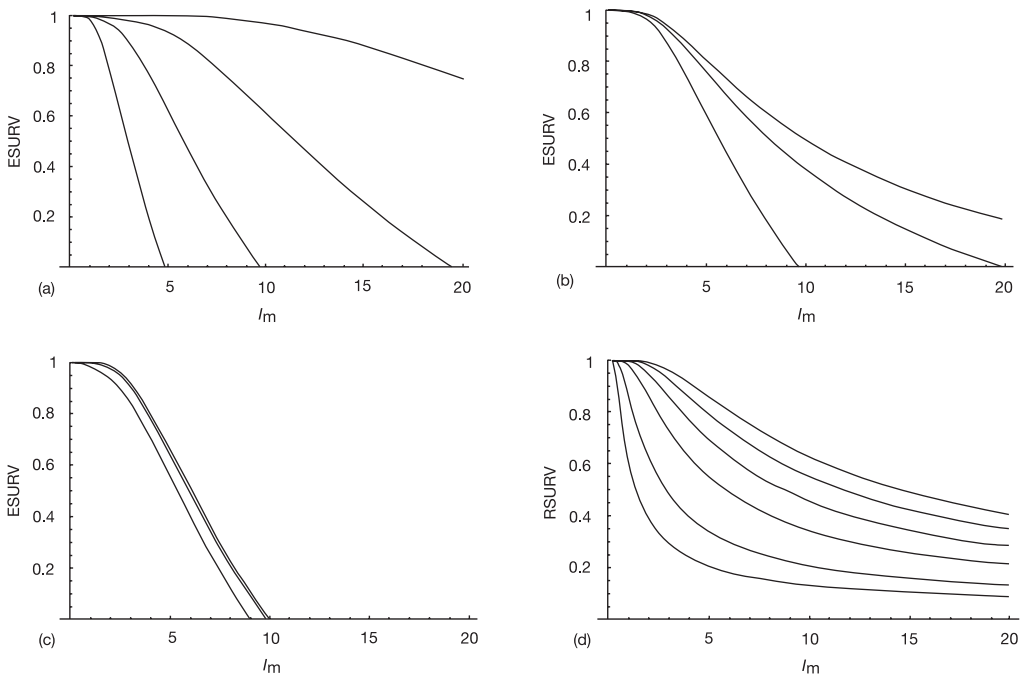

Fig. 3. The average ESS survival of wind stress with $R = R^*$, ESURV, as a function of the mean stress I_m. $E(S(I, R^*)) = 1.0 - \exp(-A/I_m R^*)$. (a) The effect of the effectiveness coefficient A on average ESS survival of stress: $A = 5, 10, 20, 50$; $m = 0.02$, $k = 1.0$. (b) The effect of the cost coefficient k on average ESS survival of stress: $k = 0.3, 0.5, 1.0$; $A = 10$, $m = 0.02$. (c) The effect of the mortality coefficient m on average ESS survival of stress: $m = 0.005, 0.02, 0.1$; $A = 10$, $k = 1.0$. (d) The unoptimized reference curves of the average survival of wind stress as a function of I_m, at different constant levels of $R = 0.1$, 0.2, 0.4, 0.6, 0.8, 1.0; $A = 10$.

In this case, the condition for the ESS $R^* = 0$ is

$$k > (1 - m)A[pL_1 + (1 - p)L_2] \tag{17}$$

which is determined by the harmonic mean of I_m in the two habitats.

The ESS R^* can be calculated by numerical solution of equation (16) for any levels of mean stress in the habitats and their proportions in the area. We used equation (16) to calculate specific numerical examples of ESS R^* and of ESS survival of stress in two habitats as a function of their mean stress and of the proportion p of habitat 1 with the lower mean stress in the environment. The results are plotted in Figs 4a and b.

As discussed in the previous section, the ESS R^* in each of the two habitats alone may be an increasing or decreasing function of I_m. ESS R^* decreases with mean stress below the intersections of the benefit derivative functions at the two habitats and increases above the intersections of the benefit derivative functions at the two habitats.

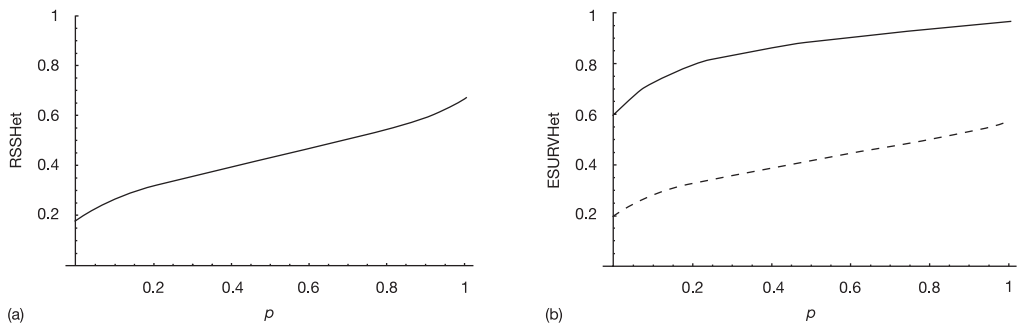

Fig. 4. (a) The ESS investment in resistance R^* by a mixed population in a heterogeneous environment with two habitats, RSSHet, as a function of changing the fraction p of habitat 1 with the lower mean stress: $I_m(1) = 2$, $I_m(2) = 8$, $A = 10$, $m = 0.02$, $k = 1.0$. (b) The ESS average survival of wind stress, ESURVHet, in habitat 1 (continuous line) and in habitat 2 (dashed line) as a function of the fraction p of habitat 1 in the environment.

The informational problem and learning by sampling

Clearly, a better investment strategy for all the trees would be to sense in some way in which habitat they grow, and invest the optimal amount for the characteristic stress probability distribution in each habitat. For simplicity, we consider only two habitats, but our formulation can be used in general.

Individual trees may obtain information about their habitat type in many different ways. Here we analyse the most direct way of obtaining information: by learning from the experience of previous stresses. It is reasonable to assume that, below the threshold, wind stresses can be perceived by trees during their lifetime, which could then act as signals that convey information about the stress probability distribution at each particular location. These signals are then used to regulate the allocation of resources to investment in mechanical strength.

The informational problem is thus for each tree to experience a series of stresses and to estimate, given these data, the probability that the tree is located in habitat 1 or habitat 2. We do this by Bayesian analysis (Hilborn and Mangel, 1997). Assume that the current level of investment is R. The tree can survive any stress less than AR, so we set a threshold stress $I_{\text{th}} = AR$. Consequently,

$$P_{\text{r}}\{\text{tree experiences and survives a stress of intensity } I\} = \frac{L \exp[-LI]}{1 - \exp[-LI_{\text{th}}]} \tag{18}$$

Given that a tree has survived an experienced stress I, we compute the posterior probability that it is in habitat 1 according to:

$$P_{\text{r}}\{\text{tree is in habitat 1, given it experiences and survives a stress of intensity } I\} =$$

$$\frac{P_{\text{r}}\{\text{in habitat 1 and observe and survive } I\}}{P_{\text{r}}\{\text{observe and survive } I\}} \tag{19}$$

If we call $P(p, I, I_{th})$ this updated value, we have

$$P(p,I,I_{th}) = \frac{(pL_1 \exp[-L_1 I])/(1 - \exp[-L_1 I_{th}])}{(pL_1 \exp[-L_1 I])/(1 - \exp[-L_1 I_{th}]) + ((1-p)L_2 \exp[-L_2 I])/(1 - \exp[-L_2 I_{th}])} \quad (20)$$

Equation (20) is the Bayesian updating result, which can be used sequentially and repeated (over years) as additional data are collected. In this updating, the threshold survival value imposes a truncation mechanism that includes in the sampling set only the below-threshold values for surviving trees.

It is reasonable to assume that individual trees have evolved to perceive such below-threshold wind stresses during their lifetime, and to use analogous developmental and physiological mechanisms for regulating and updating the ESS allocation of resources to investment in mechanical strength.

To illustrate these ideas, consider trees with $A = 10$, $k = 1$, $m = 0.02$, and two habitats with $L_1 = 0.5$ and $L_2 = 0.1$. Assume that, *a priori*, the two habitats occupy the same area; we then set $p = 0.5$ and find that the corresponding level of ESS investment is $R^* = 0.38$, as determined by the solution of equation (16) using Newton's method (Press *et al.*, 1986). Now, a tree is either in habitat 1 or habitat 2 and experiences stresses drawn from the truncated exponential distribution given by equation (18). Updating, and thus determination of the new level of investment by trees in each habitat, is done using equation (20). Even though the stresses that the trees experience may vary considerably (Figs 5a, b), over a 20 year interval the Bayesian procedure leads to fairly accurate determination of whether a tree is in habitat 1 or habitat 2, and a consequent separation of the level of optimal investment in the two habitats (Fig. 5c).

DISCUSSION

Our model makes plausible specific assumptions about the structure and parameters of the contributing functions, especially of a threshold survival function and an exponential distribution of wind stress. In this respect, the results and predictions of the model are probably fairly general. The assumptions of independently distributed exposure to stress of individual trees, and of a uniform distribution of dispersing seeds, are less general, and may have important effects on the ESS investment. The opposite extreme of assuming the destruction of the whole population is also unrealistic, because without a seed bank the whole population will go extinct. We are now investigating other versions of the model, which analyse the additional effects of investing in recovery from damage by regeneration or resprouting, and of a viable seed bank in the soil.

The model predicts that short-lived and successional trees will be much more likely to suffer wind mortality than long-lived climax species. This prediction is highly consistent with the observed distribution of storm damage to different tree species (e.g. Foster, 1988; Foster and Boose, 1995; Dyer and Baird, 1997). Short-lived early succession tree species, such as *Pinus* sp. or *Populus tremuloides*, suffer much greater mortality than long-lived climax trees.

The model predicts that an increasing cost coefficient will decrease the investment in resistance and the average stress survival. Reproductive costs are very difficult to measure or even to estimate, so there is no effective way to test these predictions. However, a large fraction of the total resources of trees is typically invested in the construction and maintenance of their mechanical support. This suggests that the relative cost in reduced

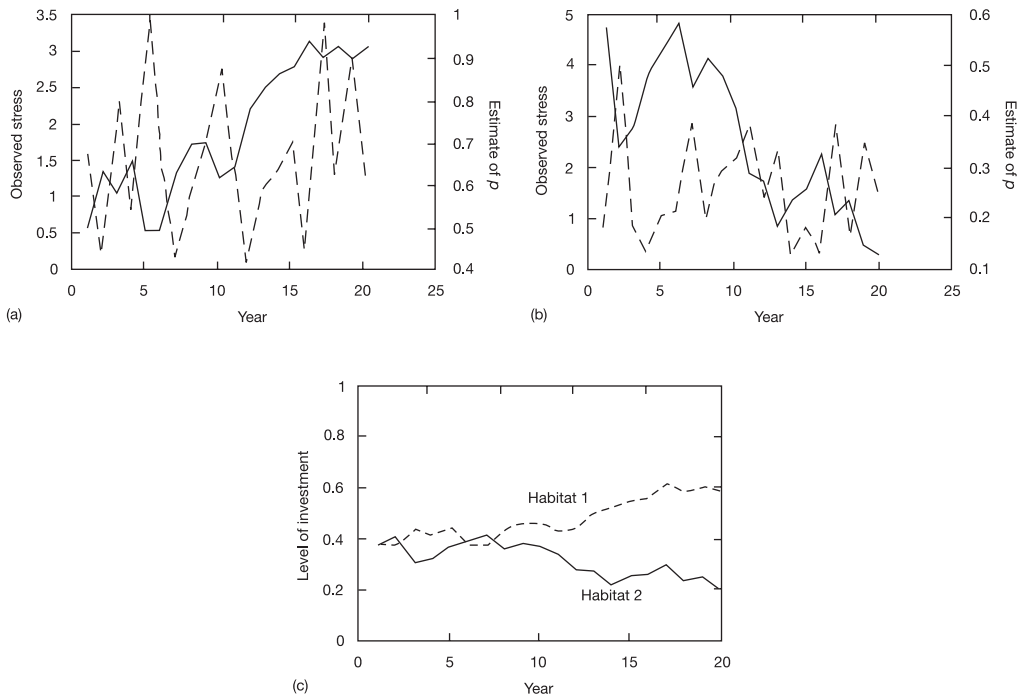

Fig. 5. The effects of experiencing stresses over 20 years on updating the estimates of the probabilities of trees in heterogeneous environments for being in habitat 1 or in habitat 2. The Bayesian updating is implemented by a recursive application of equation (20) to a sequence of 20 random truncated samplings of wind stresses from the distributions in the two habitats. As an example, we consider trees with $A = 10$, $k = 1$, $m = 0.02$, with I_{m1} in habitat $1 = 2.0$ and I_{m2} in habitat $2 = 10$. We assume that, *a priori*, the two habitats occupy the same area; so we set the initial probability for being in habitat $1 = p = 0.5$ and find that the corresponding initial ESS investment is $R^* = 0.38$ by solving equation (16). (a) The stresses experienced (dotted line) and estimate of p (solid line) for a tree in habitat 1. (b) The same for a tree in habitat 2. Note the difference in scale. (c) The separation in the levels of optimal investment in resistance for the trees in habitat 1 (dashed line) and in habitat 2 (solid line), as calculated by equation (16) with the updated estimate p of the probability for being in h_1 in each habitat.

seed production of investment in resistance is not very high for moderate levels of investment (i.e. the relative cost coefficient k is considerably less than 1).

The most interesting, but less obvious, results of the model are those that predict an increasing and then decreasing dependence of ESS R^* on the ratio between the effectiveness coefficient A and the mean wind speed I_m over a range of the other parameters (see Figs 2a, b, c). These results are caused by the effects of A/I_m on the benefit derivative function (see Fig. 1d). Although this is a widely known property of optimal allocation models in economics (e.g. Varian, 1984) and in ecology (e.g. Givnish, 1986), we did not expect it to be so pronounced in the optimal investment by trees in wind resistance.

The intuitive conventional wisdom of most evolutionary ecologists and of forest ecologists in particular (e.g. Telewski, 1995), predicts an increasing optimal investment in defence or resistance with an increasing probability of higher stress. This would apply in general also to other types of stresses, and not just to investment in wind resistance. Typical

observations and measurements of investment of resources for defence or resistance against stresses report an *increased* level of investment with increasing levels of wind stress (e.g. Ennos, 1995; Nielsen, 1995; Stokes *et al.*, 1995; Telewski, 1995) and for herbivory defence (e.g. Karban and Baldwin, 1997). Trees or saplings that are exposed to higher wind stress or are artificially shaken produce more mechanical support tissues, whereas protected or artificially supported trees produce less.

The results of our model may in part be explained by the assumption that, above the threshold, lethal wind damage only occurs with a moderate to low probability. On average, this provides long intervals for producing more seeds by trees that invest less in resistance.

The predictions of our model on the effects of increasing mean wind stress on the ESS investment in resistance are consistent with the observations over the lower range of mean wind stress. Our model predicts that, as mean wind stress increases, the increased ESS investment in resistance will overcome the increased stress up to some point close to the maximal ESS investment (e.g. compare Fig. 3a with Fig. 3d). This means that the average natural optimal survival of wind stress in trees is predicted to remain high over a wide range of mean wind stress up to some critical level, above which it is predicted to decrease very sharply. This prediction is consistent with the observation that the proportion of trees killed by tornados was similar in locations with very different wind exposures within the forest stands (Dyer and Baird, 1997). Apparently, trees exposed to a higher wind stress for a long time had invested sufficiently in stronger mechanical support to survive to the same extent the higher wind stress.

Support for the predictions of the model of decreased optimal survival over the higher range of mean wind stress is provided by the observations that tree species with wind-resistant tall trunks do not grow at sites with very high wind stresses. Depending on the values of the parameters, our model predicts that there is a level of mean wind stress above which the ESS is not to invest in resisting wind stress (i.e. a tree life form is not optimal; see Figs 3a and b). Trees could probably survive and grow in such high wind stress if they invest a sufficiently high fraction of their resources in resistance. However, under these conditions, the marginal benefit presumably becomes less than the marginal cost.

Our model is consistent with the observed allocation of mechanical support tissues within a tree to those parts that experience the largest stress caused by deformation or strain (e.g. Ennos, 1995), because this is the expected optimal within-tree allocation for any given total investment. Increased deformation by mechanical stress is known to be the mechanism that causes both the overall and the local adaptive increased production of mechanical support tissue in trees in response to wind stress (Mattheck, 1991; Telewski, 1995). Increased production of the plant hormone ethylene, probably induced by mechano-receptors in cell walls, is probably one of the immediate causes of the physiological responses (Telewski, 1995). Typical growth responses to increased mechanical shaking or wind stress are increased stem diameter, decreased elongation, and decreased elongation of upper branches. These findings are entirely consistent with the predictions of our model. Analogous with this is the increased synthesis of defence chemicals by plants in response to herbivore damage (e.g. Karban and Baldwin, 1997).

However, our model predicts in addition that the increased investment in defence would be a cumulative response to a number of exposures to rare stresses over a long time. This prediction is consistent with the finding that trees are generally ‘over-designed’ in response to the mechanical stresses to which they had been exposed in the past, and that rare

acute sub-threshold wind stresses are very effective in inducing an increased investment in mechanical support (e.g. Telewski, 1995).

Early strong herbivory damage in annual plants sometimes induces a persistent long-term increased investment in chemical and physical defences (e.g. Karban and Baldwin, 1997), which is consistent with the predictions of our model. It has been shown that early leaf damage by chewing insects induced persistent high concentrations of defence chemicals, while the same amount of leaf removal by cutting did not, suggesting that specific herbivory signals are necessary for the induction of the herbivory defence (Agrawal, 1998). This finding is consistent with the process of information updating in our model. Early cutting of leaves is not correlated with future insect attacks, whereas actual insect chewing is.

The mechanisms which provide the persistent reinforcement for the long-term response of plants to a sequence of rare stresses are not known. However, high levels of ethylene have been found to be associated with both short-term and long-term responses to wind stress in trees. Applications of Ethrel, which releases ethylene, simulate the effects of wind stress (Telewski, 1995). Persistent deformation caused by an acute stress has been suggested as a possible mechanism for the persistent responses. For example, a long-term storage of the effects of physical stimuli for the formation of epidermal meristems has been reported in the hypocotyl of plantlets of flax. The responses were delayed for many days if the stimulations were combined with high concentrations of calcium. The response to the stimulus could occur much later when calcium was depleted (Verdus, 1997).

Much lower wind stress mortality has been observed in smaller younger trees in a wide range of tree species (e.g. Foster, 1988; Foster and Boose, 1995; Nielsen, 1995; Dyer and Baird, 1997). This is usually explained by the combined effects of increased exposure and increased mechanical susceptibility of trees to wind damage as a tree increases in height.

We suggest the following explanations for these effects of age and size:

1. Stress-independent mortality may increase in older trees, which selects for a lower optimal investment in stress resistance. The same argument applies when trees have a limited reproductive life-span.
2. Smaller trees are mechanically stronger for the same amount of investment – that is, their effectiveness coefficient A is higher – which may lead to a combination of lower ESS investment and a higher average ESS survival (Figs 2a, 3a).
3. Competition for light between mature trees provides a selective advantage for trees that grow taller and thus are exposed to higher wind stress, at the same time increasing the seed production cost of investment in resistance. This explanation is consistent with the observations that naturally isolated trees, or trees growing at lower densities, without or with less competition for light, are less likely to be damaged by wind stress, in spite of their more exposed situation (e.g. Nielsen, 1995; Dyer and Baird, 1997).
4. In heterogeneous environments, young trees may be optimally over-protected for some years until they have accumulated sufficient experience of their local stress distribution and have adjusted their investment accordingly.

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