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A different model to explain delayed germination

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ABSTRACT

Goal: To provide an alternative to the usual bet-hedging explanation for delayed germination, one that takes account of known facts about germination in stable, fine-grained environments.

Context: Small patches with local environmental conditions (microhabitats) such that seedlings can establish themselves are customarily called safe sites.

Key assumptions: We focus on a single species. Its safe sites become available randomly. Seeds that germinate outside safe sites all die as seedlings. All seeds are equal, i.e. their probability of dying over the year and probabilities to germinate when the right season is there do not depend on their age or any other aspect of their individual history. Moreover, we make the standard assumption of ESS theory that the population is genetically homogeneous but for the occasional mutant 'testing the ESS'. There is a trade-off between the germination probability in safe sites and the probability not to germinate outside safe sites. For germination strategies close to the ESS, the environment does not fluctuate.

Procedure: Start with a simple population model, in which the yearly seed survival and the fraction of the area covered by safe sites are fixed quantities. For this model, derive an optimization principle that finds the Evolutionarily Steady Strategy vector consisting of the probabilities to germinate in safe sites and elsewhere. Using this optimization principle, analyse the effect of various trade-offs using Levins' fitness set technique. Analyse how the results extend to ESSs for general life histories and community dynamics subject only to the key assumptions.

Conclusion: Seeds in safe sites should not all germinate on the first opportunity if the relationship between the probability to germinate in safe sites and the probability to germinate elsewhere is accelerating and has a sufficiently steep slope at the highest germination probabilities.

Keywords: delayed germination, Levins' fitness set, life stages, optimization principle.

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INTRODUCTION

The phenomenon that not all seeds germinate directly when conditions are suitable but that some delay germination for one to several years is usually referred to as just 'delayed germination'. Starting with the work of Dan Cohen (1966, 1967), the standard explanation in the theoretical literature for this phenomenon has been bet-hedging in time-varying environments (Bulmer, 1984; Ellner, 1985a, 1985b, 1986; Brown and Venable, 1986, 1991; Cohen and Levin, 1987; Klinkhamer *et al.*, 1987; Venable and Brown, 1988; Venable, 1989; Rees, 1994; Easterling and Ellner, 2000; Aikio *et al.*, 2002; Mathias and Kisdi, 2002; Tielbörger and Valleriani, 2005; Valleriani, 2005, 2006; Valleriani and Tielbörger, 2006).

Bet-hedging models assume that environmental conditions vary in time but at each instant in time conditions are the same for all plants. Despite this uniformity, plants are predicted to develop adaptations that prevent all of their seeds from germinating during the same growing season. Bet-hedging strategies may be realized as a 'coin-flipping mechanism' by the seed or by parents producing several types of seeds with different germination propensities.

We are more familiar with a different scenario. In our experience, seeds germinate mainly as a reaction to local disturbances, and hence seeds may be expected to adapt this reaction to maximize their chances of becoming successful reproductive adults. Seeds of foxglove (*Digitalis purpurea*) can survive in the soil for many years only to germinate after a disturbance, for instance due to a tree fall (van Baalen, 2004). Our own field studies with spear thistle (*Cirsium vulgare*) and houndstongue (*Cynoglossum officinale*) also suggest that disturbances may dramatically increase germination (Klinkhamer and De Jong, 1988). For surveys of possible mechanisms, see Baskin and Baskin (1998, in particular Chapter 4) and Fenner and Thompson (2005), and for a quick summary with a view towards ecology, see Rees (1997). Recently, Jankowska-Blaszczuk and Daws (2007) showed how high red to far red ratios can influence germination propensities. Vandelook *et al.* (2008) showed that local temperature fluctuations as well light and nitrate availability can stimulate germination (the authors refer to 'gap-detection signals'). An entirely different type of mechanism may involve germination inhibitors in the endocarp that first have to leach away, with the leaching process depending on local conditions. Hu *et al.* (2008) have shown that in addition other layers of the fruit may be involved in more indirect ways.

The difference is, of course, that we work primarily in the Netherlands, which has a rather humid and stable temperate climate and fine-scaled vegetation structures, whereas Dan Cohen's outlook was shaped by the dry climate of Israel where the favourable circumstances for germination arise only after sporadic rainfalls that affect relatively large areas.

In this paper, we describe a model that covers the fine-scaled temperate outlook. We start by considering the simplest scenario of annual plants with only seedling interactions. This interaction is idealized. We divide the world into places where seedlings cannot survive and so-called safe sites, small patches where they can survive (cf. Skellam, 1951; Pielou, 1975; De Jong *et al.*, 1987; Geritz *et al.*, 1988). In these safe sites seedlings compete, so that at most a few survive. Otherwise, the plants do not interact. Safe sites become available unpredictably in space and in time. Seeds can distinguish between the two possible microhabitats, but only within a certain margin of error. Hence, increasing the germination probability in a safe site also increases the germination probability in the remainder of the habitat. As the strategy variable, we choose the germination probability in safe sites. We rigged this eco-evolutionary model such that it satisfies an optimization principle (De Jong *et al.*, 1987; Mylius and

Diekmann, 1995; Metz *et al.*, 2008a), allowing for an easy calculation of the Evolutionarily Steady germination fraction. [We interpret the second symbol in 'ESS' as 'Steady' instead of the commonly used 'Stable'; see, for example, Metz (2008, p. 1605) for an explanation: only ESSs in the subset of so-called Continuously Stable Strategies are indeed evolutionarily stable in the standard interpretation of the term stable.] The existence of an optimization principle guarantees that the corresponding ESSs are, moreover, evolutionarily attracting (Continuously Stable).

In principle, the existence of an optimization principle excludes a plethora of potential evolutionary phenomena. However, for our purpose, namely showing that a particular evolutionary explanation can work, this simplification is relatively harmless. [See the introduction of Metz *et al.* (2008b) for a more detailed discussion of when using simplifications of this type can be considered methodologically sound.]

After having tackled the simple model, we investigate which ecological restrictions of the simple model can be relaxed without changing the results. We end with a discussion of the experimental options for testing the theory.

A SIMPLE ECO-EVOLUTIONARY MODEL

In following the ecological scenario described in the previous section, we assume that both the dispersal distances and the area in which the population lives are so large that the use of a deterministic model is warranted. In that case, the scenario translates into simple recurrences for the seed densities of a resident population and of a potentially invading mutant population. We shall use the following notation (when there is a need to distinguish between residents and mutants, resident parameters will be in UPPER CASE and mutant ones in lower case), based on a census of seeds just before germination and of plants at a time when seedling competition has just ended:

- h: density of safe sites available at germination time;
- a: average area of such a safe site;
- k: fraction of total area covered by those safe sites (= ha);
- S: yearly survival of seeds in the soil;
- Y: seed production of a mature plant;
- U: survival of newborn seeds until their first germination opportunity;
- G,g: probability that a seed in a safe site germinates;
- F,f: probability that a seed outside a safe site germinates;
- X,x: overall seed density prior to germination;
- P,p: density of mature plants (i.e. plants that survived seedling competition).

The quantities h to U are supposed to be constant parameters. With this notation, the recurrence for the resident population becomes

$$X' = S[1 - kG - (1 - k)F]X + UYP, \tag{1}$$

with, when there are no mutants around yet,

$$P = h\Phi(GX). \tag{2}$$

$\Phi(GX)$ is the expected number of surviving seedlings in a safe site if the average density of germinating seeds in a site is GX. In other words, next year's seeds (the density of which is denoted as X') will consist of this year's seeds that neither germinate nor die plus the

surviving new seeds from this year. P equals the density of safe sites multiplied by this year's average number of survivors of seedling competition in a site. If in each site exactly one out of any positive number of seedlings remains, seeds are distributed randomly over space, and all safe sites have the same area:

$$\Phi(Z) = (1 - e^{-aZ}). \tag{3}$$

Now suppose that a mutant is introduced. The mutant seed density satisfies

$$x' = S[1 - kg - (1 - k) f]X + UYp, \tag{4}$$

where p is calculated by multiplying the following expressions:

expected number of surviving seedlings in a safe site: $\Phi(GX + gx)$,

probability that a randomly chosen seed is of the mutant type: $gx/(GX + gx)$.

A similar argument applies to the resident in a combined mutant–resident population, giving

$$P = h\Psi(GX + gx)GX, \quad p = h\Psi(GX + gx)gx, \tag{5}$$

with

$$\Psi(Z) := \Phi(Z)/Z. \tag{6}$$

If Φ increases at a decelerating rate, as is the case in the example, then the pure resident recurrence has a unique globally attracting internal equilibrium if and only if

$$S[1 - kG - (1 - k)F] + UYh\Psi(0)G > 1 \quad \text{and} \quad \lim_{Z \to \infty} \Psi'(Z) < 1. \tag{7}$$

We shall from now on make the assumption that the former is the case. The internal equilibrium satisfies

$$1 = S[1 - kG - (1 - k)F] + UYh\Psi(GX^*)G. \tag{8}$$

Therefore,

$$UYh\Psi(GX^*) = [1 - S(1 - kG - (1 - k)F)]/G. \tag{9}$$

Whether a mutant can invade or not has to be judged from the linearized recurrence for the mutant population with the resident population at its equilibrium:

$$\begin{aligned} x' &= S[1 - kg - (1 - k) f]x + UYh\Psi(GX^*)gx \\ &= \{S[1 - kg - (1 - k) f] + (g/G)[1 - S(1 - kG - (1 - k)F)]\}x. \end{aligned} \tag{10}$$

A mutant can invade if

$$S[1 - kg - (1 - k) f] + (g/G)[1 - S(1 - kG - (1 - k)F)] > 1, \tag{11}$$

or equivalently

$$g/\{1 - S[1 - kg - (1 - k) f]\} > G/\{1 - S[1 - kG - (1 - k)F]\}, \tag{12}$$

and only if

$$g/\{1 - S[1 - kg - (1 - k) f]\} \geq G/\{1 - S[1 - kG - (1 - k)F]\}. \tag{13}$$

Hence, in this model, natural selection favours the strategy that maximizes

$$M(G, F) := G/\{1 - S[1 - kG - (1 - k)F]\}. \tag{14}$$

CALCULATING THE EVOLUTIONARILY STEADY STRATEGY

The optimization principle M increases with G and decreases with F. So, if there are no further constraints, G will increase to its maximal value 1, and F will decrease to its minimal value 0. In reality, increasing G will no doubt also increase F. One time-honoured (see, for example, De Jong and Klinkhamer, 2005), special choice for the relation between F and G is

$$F \geq \alpha G^{\beta}, \text{ with } \alpha \leq 1. \tag{15}$$

For this relation, with G^* the Evolutionarily Steady value of G,

$$\text{for } \beta > 1 \text{ and } \alpha(\beta - 1) > \frac{1 - S}{(1 - k)S}: \qquad 0 < G^* = \left(\frac{1 - S}{S\alpha(1 - k)(\beta - 1)}\right)^{1/\beta} < 1 ,$$

$$\text{otherwise:} \qquad G^* = 1. \tag{16}$$

In other words, under the first conditions, even in safe sites only a fraction of the seeds should germinate, since increasing the germination fraction beyond this optimal value would lead to too great a cost from inopportunely germinating seeds in deadly territory. Otherwise, seeds in safe sites should all germinate, independent of the costs incurred from seeds inappropriately germinating elsewhere.

A more general analysis is possible using Levins' idea of fitness sets (Levins, 1962, 1968) [see Rueffler *et al.* (2004) for an extension to cases without optimization principle]. In such an approach, the axes correspond to life-history parameters in which the evolutionarily maximized quantity increases. Moreover, it helps when the contour lines of the optimization principle are straight lines. This leads naturally to the choice of G and $1 - F$ for our coordinate system, leading to the following formula for the fitness contours:

$$1 - F = \frac{1 - kS + (kS - M^{-1})G}{(1 - k)S} . \tag{17}$$

We denote the trade-off as $1 - F \leq T(G)$. Since

$$\frac{1 - kS}{(1 - k)S} > 1, \tag{18}$$

and any reasonable trade-off satisfies $T(0) = 1$, the Levins style pictures will look like Fig. 1a when the ESS corresponds to an intermediate germination fraction. When the fitness set is concave instead of convex, seeds should always germinate. For a convex fitness set, complete germination will be ES when (see Fig. 1b)

$$T(1) - \frac{\mathrm{d}T}{\mathrm{d}G}(1) < \frac{1 - kS}{(1 - k)S} \tag{19}$$

(with the physiological and ecological parameters collected on different sides of the inequality sign).

For the case of $F \geq \alpha G^{\beta}$, with $\alpha \leq 1$, condition (19) translates into $\alpha(\beta - 1) > \frac{1 - S}{(1 - k)S}$, as found before.

To interpret criterion (19), observe that at $G = 1$ at the ecological equilibrium X^*, the pay-off, in seeds next year, of germinating when in a safe site equals $UYh\Psi(GX^*) =$

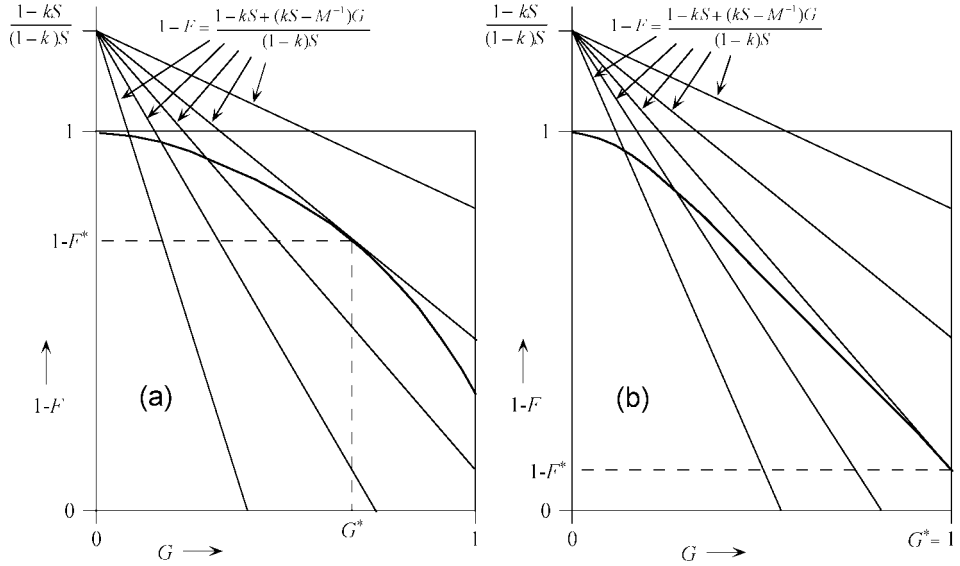

Fig. 1. Two possible convex fitness sets together with a sample of contour lines of the optimization criterion. The left configuration leads to an internal ESS, the right one to a boundary ESS.

$1 - S[1 - k - (1 - k)F] = 1 - (1 - k)(1 - T(1))S$ (see equation 9). With this in mind, criterion (19) can be rewritten as

$$1 - (1 - k)T(1)S > S\left(k + (1 - k)\frac{-\mathrm{d}T}{\mathrm{d}G}(1)\right). \tag{20}$$

That is, at $G = 1$ for seeds in safe sites, the average pay-off of germinating is larger than the marginal pay-off of not germinating. This marginal pay-off has to be calculated for an average seed, not for the seed under consideration. It is composed of two terms depending on whether a seed finds itself in a safe site, which is the case with probability k, or elsewhere. The term $-\mathrm{d}T/\mathrm{d}G$ equals the marginal change in $1 - F$, the probability that a seed outside a safe site does not germinate and hence does not succumb in the germination window. After adding, the result is multiplied by S to arrive at the seeds of next year.

In general, (9) tells us that the equilibrium pay-off of germinating is $Uyh\Psi(GX^*) = \{1 - S[1 - kG - (1 - k)F]\}/G = \{1 - S[(1 - k)T(G) + k(1 - G)]\}/G$. Internal ESSs satisfy (cf. Fig. 1a)

$$T(G^*) = \frac{1 - kS + (kS - M^{-1})G^*}{(1 - k)S} \quad \text{and} \quad \frac{\mathrm{d}T}{\mathrm{d}G}(G^*) = -\frac{kS - M^{-1}}{(1 - k)S}, \tag{21}$$

which can be rewritten as

$$\frac{1 - S((1 - k)T(G^*) + k(1 - G^*))}{G^*} = S\left(k + (1 - k)\frac{-\mathrm{d}T}{\mathrm{d}G}(G^*)\right). \tag{22}$$

That is, at internal ESSs, for seeds in safe sites the average pay-off of germinating is equal to the marginal pay-off of not germinating.

In all cases, evolution minimizes the pay-off of germinating in a safe site in accordance with Result 3 in Mylius and Diekmann (1995) and Proposition 3.2 in Metz *et al.* (2008a): When the relevant environmental variables, in this case GX, affect the expected lifetime reproductive output of an individual in a monotone one-dimensional manner (through the function Ψ), the feedback loop through the environment means that evolution causes the organisms to end up in the worst possible world.

REMOVING THE ECOLOGICAL SIMPLIFICATIONS

To see which of the ecological simplifications made earlier are essential for reaching the conclusions of the previous section, we rely on the following general argument [expounded in Example 6.2 of Metz *et al.* (2008a)]. 'Let the life history consist of a number of subsequent stages. Call a stage reproductive if reproduction is possible during, or before as well as after that stage, and all preceding stages "pre-reproductive". If there is no overlap between the sets of pre-reproductive stages affected by, respectively, the strategy vector $\mathbf{Q}$ and the environment $\mathbf{E}$, and the reproductive stages are affected by at most one of those two variables, the average lifetime offspring production can be expressed as $R_0(\mathbf{Q}, \mathbf{E}) = \phi(\mathbf{E})$ $R_0(\mathbf{Q}, \mathbf{E}_0)$, $\mathbf{E}_0$ some arbitrary reference environment. Therefore, evolution within those confines maximizes $R_0(\cdot, \mathbf{E}_0)$'. This suggests considering life histories of the form depicted in Fig. 2.

The essential characteristic of the life histories shown in Fig. 2 is their decomposability into two stages between which no information is transferred, as all seedlings are equal, and so are all newly produced seeds. Within the two stages, the seeds and plants are differentiated in for example (age, depth in soil)- and (age, size above ground, size below ground)-classes, respectively. Moreover, one can decompose the environmental influences on an average individual plant into three components: (1) influences on the seeds, to be represented by a vector $\mathbf{E}_b$ (consisting of, for example, seed predation pressures and fungal and bacterial attack rates at different depths; $_b$ from below ground); (2) influences on the seedlings, including their own average initial density in safe sites Z, to be represented by a vector $\mathbf{E}_s$; and (3) influences after the seedling stage, to be represented by a vector $\mathbf{E}_a$ (capturing all direct and indirect competitive influences within the community through shading, nutrient depletion, and changing predation pressures; $_a$ from above ground). Since the birth of a safe site necessarily coincides with the demise or a state change of one or more

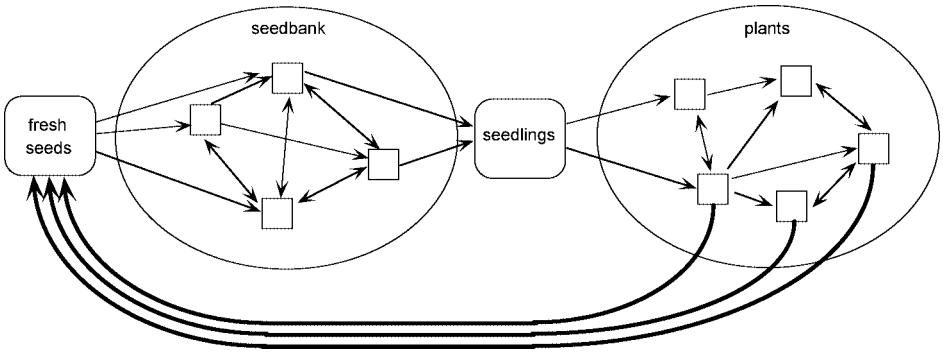

Fig. 2. A generalized plant life history with the restriction that all seeds are equal initially, as are all seedlings.

plants in the community, it may also be assumed that $\mathbf{E}_a$ determines the fraction of the area covered by safe sites. Therefore, for a full description of the eco-evolutionary model, the following quantities and functions are needed:

- $\mathbf{X}$, $\mathbf{x}$: vector of densities of seeds in different states;
- $\mathbf{P}$, $\mathbf{p}$: vector of densities of plants in different states;
- $\mathbf{U}(\mathbf{E}_b)$: state distribution of a new seed just prior to germination time;
- $\mathbf{S}(\mathbf{E}_b)$: matrix of survival and state transition probabilities of seeds;
- $h(\mathbf{E}_a)$: density of safe sites available at germination time;
- $k(\mathbf{E}_a)$: fraction of total area covered by those safe sites;
- $\mathbf{G}$, $\mathbf{g}$: state-dependent probabilities that a seed in a safe site germinates;
- $\mathbf{F}$, $\mathbf{f}$: state-dependent probabilities that a seed outside a safe site germinates;
- $\Psi(\mathbf{E}_s)$: average number of seedlings in a safe site that survive seedling competition divided by the average density of novel seedlings in safe sites;
- $\mathbf{J}(\mathbf{E}_a)$: state distribution of young plants that have survived seedling competition;
- $\mathbf{A}(\mathbf{E}_a)$: matrix of survival and state transition probabilities of plants;
- $\mathbf{Y}(\mathbf{E}_a)$: seed production by plants in different plant states.

Note that the probability distribution of the state of a newborn seed at the next germination time, encoded in the vector $\mathbf{U}$, will in general sum to less than one due to seed mortality. Note also that in most concrete instances, the probability distribution of plant states after seedling competition, encoded in the vector $\mathbf{J}$, will probably be concentrated on but a single plant state: small juvenile. $\mathbf{J}$ by definition sums to one, as the probabilities of seedling death are all accounted for by $\Psi(\mathbf{E}_s)$.

The resident population state satisfies the following recurrences

$$\begin{aligned}\mathbf{X}' &= \mathbf{S}(\mathbf{E}_b)(\mathbf{I} - k(\mathbf{E}_a)\mathrm{diag}(\mathbf{G}) - (1 - k(\mathbf{E}_a))\mathrm{diag}(\mathbf{F}))\mathbf{X} + \mathbf{U}(\mathbf{E}_b)\mathbf{Y}^{\mathrm{T}}(\mathbf{E}_a)\mathbf{P},\\ \mathbf{P}' &= \mathbf{A}(\mathbf{E}_a)\mathbf{P} + \mathbf{J}(\mathbf{E}_a)h(\mathbf{E}_a)\Psi(\mathbf{E}_s)\mathbf{G}^{\mathrm{T}}\mathbf{X},\end{aligned} \tag{23}$$

with

$$\mathrm{diag}\begin{pmatrix} v_1 \\ \vdots \\ v_k \end{pmatrix} := \begin{pmatrix} v_1 & 0 & \cdots & 0 \\ 0 & \ddots & \ddots & \vdots \\ \vdots & \ddots & \ddots & 0 \\ 0 & \cdots & 0 & v_k \end{pmatrix} \text{ and } \begin{pmatrix} v_1 \\ \vdots \\ v_k \end{pmatrix}^{\mathrm{T}} := (v_1 \quad \cdots \quad v_k)\,.$$

A verbal rendering of these equations mimics that for the scalar case. (By default the earlier assumption that the resident population dynamics converge to an equilibrium remains in place.) By the same token, the state of the mutant population satisfies

$$\begin{aligned}\mathbf{x}' &= \mathbf{S}(\mathbf{E}_b)(\mathbf{I} - k(\mathbf{E}_a)\mathrm{diag}(\mathbf{g}) - (1 - k(\mathbf{E}_a))\mathrm{diag}(\mathbf{f}))\mathbf{x} + \mathbf{U}(\mathbf{E}_b)\mathbf{Y}^{\mathrm{T}}(\mathbf{E}_a)\mathbf{p},\\ \mathbf{p}' &= \mathbf{A}(\mathbf{E}_a)\mathbf{p} + \mathbf{J}(\mathbf{E}_a)h(\mathbf{E}_a)\Psi(\mathbf{E}_s)\mathbf{g}^{\mathrm{T}}\mathbf{x}.\end{aligned} \tag{24}$$

These equations should be combined with equations for the remainder of the community to determine ($\mathbf{E}_a$, $\mathbf{E}_s$, $\mathbf{E}_b$). As it turns out, this model is still a bit too general to allow the ES germination strategy to be determined from an optimization principle. However, it is only by considering more general models that it is possible to delineate the crucial assumptions underlying the results from the previous section.

At the resident equilibrium, the average number of offspring over a resident's lifetime equals 1. The calculation of this average lifetime offspring number can be broken down into a number of steps. First, we calculate the average number of full seasonal cycles (measured between end-of-seedling-competition time points) that a survivor from the seedling stage lives through during its lifetime, split up according to the state the plant was in at the end-of-seedling-competition moments. From the general Markov chain results in Kemeny and Snell (1960), it follows that these numbers are given by the vector $(\mathbf{I} - \mathbf{A}(\mathbf{E}^*_{a,G,F}))^{-1}\,\mathbf{J}(\mathbf{E}^*_{a,G,F})$, with $(\mathbf{E}^*_{a,G,F},\mathbf{E}^*_{s,G,F},\mathbf{E}^*_{b,G,F})$ the equilibrium environment, to be determined from the full community dynamical equations for the resident strategy $(\mathbf{G}, \mathbf{F})$. Hence, the average number of seeds that a plant that just germinated in a safe site will produce over its lifetime is $\mathbf{Y}^T(\mathbf{E}^*_{a,G,F})(\mathbf{I} - \mathbf{A}(\mathbf{E}^*_{a,G,F}))^{-1}\,\mathbf{J}(\mathbf{E}^*_{a,G,F})\Psi(\mathbf{E}^*_{s,G,F})$. Similarly, the average number of germination moments that a seed experiences while in various seed states equals $(\mathbf{I} - \mathbf{S}(\mathbf{E}^*_{b,G,F})(\mathbf{I} - k(\mathbf{E}^*_{a,G,F})\mathrm{diag}(\mathbf{G}) - (1 - k(\mathbf{E}^*_{a,G,F}))\mathrm{diag}(\mathbf{F})))^{-1}\,\mathbf{U}(\mathbf{E}^*_{b,G,F})$. Therefore, the probability of a seed germinating in a safe site instead of dying or germinating elsewhere equals $\mathbf{G}^T(\mathbf{I} - \mathbf{S}(\mathbf{E}^*_{b,G,F})(\mathbf{I} - k(\mathbf{E}^*_{a,G,F})\mathrm{diag}(\mathbf{G}) - (1 - k(\mathbf{E}^*_{a,G,F}))\mathrm{diag}(\mathbf{F})))^{-1}\,\mathbf{U}(\mathbf{E}^*_{b,G,F})$. Multiplying these two numbers gives

$$\begin{aligned} 1 = R_0(\mathbf{G},\mathbf{F};\mathbf{E}^*_{a,G,F},\mathbf{E}^*_{s,G,F},\mathbf{E}^*_{b,G,F}) = \mathbf{Y}^T(\mathbf{E}^*_{a,G,F})(\mathbf{I} - \mathbf{A}(\mathbf{E}^*_{a,G,F})^{-1}\mathbf{J}(\mathbf{E}^*_{a,G,F})\Psi(\mathbf{E}^*_{s,G,F}) \times \\ \mathbf{G}^T(\mathbf{I} - \mathbf{S}(\mathbf{E}^*_{b,G,F})(\mathbf{I} - k(\mathbf{E}^*_{a,G,F})\mathrm{diag}(\mathbf{G}) - (1 - k(\mathbf{E}^*_{a,G,F}))\mathrm{diag}(\mathbf{F})))^{-1}\,\mathbf{U}(\mathbf{E}^*_{b,G,F}). \end{aligned} \tag{25}$$

Hence,

$$\Psi(\mathbf{E}^*_{s,G,F}) = \begin{pmatrix} \mathbf{Y}^T(\mathbf{E}^*_{a,G,F})(\mathbf{I} - \mathbf{A}(\mathbf{E}^*_{a,G,F})^{-1}\mathbf{J}(\mathbf{E}^*_{a,G,F}) \times \\ \mathbf{G}^T(\mathbf{I} - \mathbf{S}(\mathbf{E}^*_{b,G,F})(\mathbf{I} - k(\mathbf{E}^*_{a,G,F})\mathrm{diag}(\mathbf{G}) - (1 - k(\mathbf{E}^*_{a,G,F}))\mathrm{diag}(\mathbf{F})))^{-1}\,\mathbf{U}(\mathbf{E}^*_{b,G,F}) \end{pmatrix}^{-1}. \tag{26}$$

Similarly, we find for the average lifetime offspring production of a mutant, after substituting the expression for $\Psi(\mathbf{E}^*_{s,G,F})$ and cancelling terms that appear in both numerator and denominator,

$$R_0(\mathbf{g},\mathbf{f};\mathbf{E}^*_{a,G,F},\mathbf{E}^*_{s,G,F},\mathbf{E}^*_{b,G,F}) = \frac{\mathbf{g}^T(\mathbf{I} - \mathbf{S}(\mathbf{E}^*_{b,G,F})(\mathbf{I} - k(\mathbf{E}^*_{a,G,F})\mathrm{diag}(\mathbf{g}) - (1 - k(\mathbf{E}^*_{a,G,F}))\mathrm{diag}(\mathbf{f})))^{-1}\,\mathbf{U}(\mathbf{E}^*_{b,G,F})}{\mathbf{G}^T(\mathbf{I} - \mathbf{S}(\mathbf{E}^*_{b,G,F})(\mathbf{I} - k(\mathbf{E}^*_{a,G,F})\mathrm{diag}(\mathbf{G}) - (1 - k(\mathbf{E}^*_{a,G,F}))\mathrm{diag}(\mathbf{F})))^{-1}\,\mathbf{U}(\mathbf{E}^*_{b,G,F})}. \tag{27}$$

Hence, ESSs can be determined by optimizing $(\mathbf{G}, \mathbf{F})$ in

$$\tilde{M}(\mathbf{G},\mathbf{F};k,\mathbf{S},\mathbf{U}) := \mathbf{G}^T(\mathbf{I} - \mathbf{S}(\mathbf{I} - k\,\mathrm{diag}(\mathbf{G}) - (1 - k)\mathrm{diag}(\mathbf{F})))^{-1}\,\mathbf{U} \tag{28}$$

dependent on $(k,\mathbf{S},\mathbf{U})$, and solving the community dynamical equilibrium equations together with $(\mathbf{G},\mathbf{F}) = (\mathbf{G},\mathbf{F})_{\mathrm{opt}}(k(\mathbf{E}_{a,G,F}),\mathbf{S}(\mathbf{E}_{b,G,F}),\mathbf{U}(\mathbf{E}_{b,G,F}))$.

If and only if $\mathbf{G}$ and $\mathbf{F}$ do not influence the equilibrium values of the seed state transition and survival probabilities and the fraction of the area covered by safe sites, $\mathbf{S}(\mathbf{E}^*_{b,G,F}) = \bar{\mathbf{S}}$, $\mathbf{U}(\mathbf{E}^*_{b,G,F}) = \bar{\mathbf{U}}$ and $k(\mathbf{E}^*_{a,G,F}) = \bar{k}$, the function $(\mathbf{G},\mathbf{F}) \to \tilde{M}(\mathbf{G},\mathbf{F};\bar{k},\bar{\mathbf{S}},\bar{\mathbf{U}})$ is an evolutionary optimization principle, in accordance with the general result described in Example 6.2 of Metz *et al.* (2008a).

In the special case that all seeds are equal, $\tilde{M}(\mathbf{G},\mathbf{F};k,\mathbf{S},\mathbf{U})$ reduces to $M(G,F;k,S) = G/\{1 - S[1 - kG - (1 - k)F]\}$ and the graphical representations from the previous section apply also for the ESS, with the modification that $S(\mathbf{E}^*_{b,G,F})$ and $k(\mathbf{E}^*_{a,G,F})$ are no longer

constants, but depend on the community dynamical equilibrium for that value of (G, F). In particular, also in this more general case, only a fraction of the seeds in safe sites should germinate when the fitness set $0 \leq 1 - F \leq T(G) \leq 1$ is convex and at $G = 1$ the average pay-off of germinating for seeds in safe sites is smaller than the marginal pay-off of not germinating, with the minor complication that these pay-offs are not *a priori* specified parameters but depend on the community dynamics through $k(\mathbf{E}^*_{\mathbf{a},1,T(1)})$ and $S(\mathbf{E}^*_{\mathbf{b},1,T(1)})$. However, since in applications (assuming boldly that these will actually occur) it will rarely be possible to estimate all parameters of a mechanistic model predicting k and S, so that these quantities will in all probability be estimated directly from field data, the latter will have greater conceptual than practical significance.

The theory from the previous section applies without modification, if and only if the seeds are all equal and the equilibrium seed survival $S(\mathbf{E}^*_{\mathbf{b},G,F})$ and the equilibrium fraction of the area covered by safe sites $k(\mathbf{E}^*_{\mathbf{a},G,F})$ are not influenced by G or F.

EXPERIMENTAL TESTS?

Despite clear-cut mathematical results, there is still the problem how well they relate to biological reality. First, it may be that seeds in the right season and given seemingly extremely favourable conditions just always germinate. For instance, in our experience the fresh seeds of common mullein (*Verbascum thapsus*), a plant species with a well-developed and long-lived seed bank, without exception germinate in light-exposed Petri-dishes. If in the field not all seeds germinate in apparent safe sites, this may be due to such extreme germination conditions never occurring in nature, or to a fraction of the seeds from the field observations actually not being in safe sites. Which scenario applies can only be inferred by comparing germination results from laboratory experiments with results from patches with optimal and poor conditions for seedling survival that are experimentally created in the field.

It may also be that the dichotomy between safe sites and deadly remaining territory is an oversimplification, and that the non-germinating seeds, although located in safe sites, may still be in a weaker position as competitors, since, for example, they have to spend more energy to push their cotyledons up from a greater depth. The reaction to light shown by many seeds provides an instrument to respond to such a situation. Indeed, if light was the only signal, it might be technically impossible for a seed to show the best possible reaction to depth due to the fast fading of the signal. This would also explain why seeds use more than one cue to base their decisions on. Note that when seeds are thus differentiated according to soil depth, the assumption that all seeds are equal no longer holds. In the previous section, we wrote down a model that in principle can handle such complicated mechanisms, but only to show what structural properties of the model lead to the existence of an optimization principle. As a tool for making concrete predictions, such a model will soon contain too many parameters, so that judging whether seeds indeed play a strategy close to an ESS will unavoidably be thwarted by experimental noise. Evolution uses larger sample sizes than any experimenter, so one may expect it to draw finer lines than humans ever can.

Data supporting the assumption that all seeds are equal may be found in Figure 4/11 in Harper (1977), Figure 7.4 in Baskin and Baskin (1998), and Figure 5.8 in Silvertown and Charlesworth (2001). Figure 7.7 in Rees (1997) shows one case supporting the assumption and three graphs for cases where it fails to hold true.

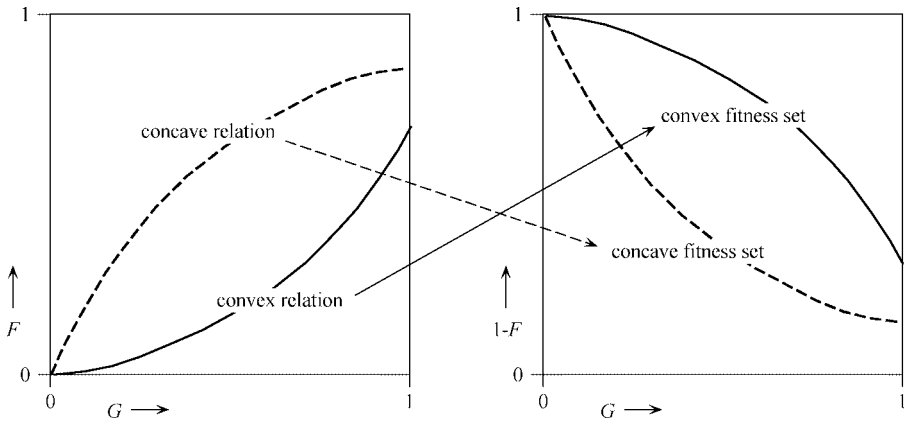

Fig. 3. Connection between the relation between G and F and the corresponding fitness set. According to standard mathematical terminology, when the relation between G and F is convex, the trade-off between G and $1 - F$ is concave and the fitness set is convex. Similarly, when the relation between G and F is concave, the trade-off between G and $1 - F$ is convex and the fitness set is concave.

Finally, for many species, the dichotomy between safe sites and deadly remaining territory may be an oversimplification and that instead the degree of safety changes more gradually rather than in a dichotomous fashion. In our view, however, the dichotomy probably is a good first approximation for at least a fair number of species, although in any specific case this remains up to the field biologist to decide.

Save for the sharp dichotomy between safe sites and deadly remaining territory, the ecological ingredients of our general model seem pretty indisputable. This is less so for the physiological ingredients introduced one section earlier. To comply with the traditions of Levins' method, we moreover had to represent the trade-off in a non-intuitive way. Figure 3 shows what the notions of convex and concave fitness sets mean in terms of the relation between G and F: a convex fitness set corresponds to a convex relation between G and F (and a concave trade-off between G and $1 - F$). Somehow, intuitively a concave relation between G and F feels more natural. However, the only proof of the pudding is in the eating. One possible set of observations that would allow judging the shape of the trade-off would be to compare the realized germination propensities of seeds that differ genetically, for example, since they come from areas with different values of k or S, (1) in patches overgrown by natural vegetation, (2) in artificially cleaned patches, and (3) in the laboratory under circumstances most favourable to germination. A final approach could be to try to develop a model that translates known facts about germination-inducing stimuli and the behaviour of those stimuli in patches that clearly act as safe sites and patches that do not. Perhaps a trade-off curve could be constructed using the data reviewed in Chapter 4 of Baskin and Baskin (1998) and Fenner and Thompson (2005) and gleaned from more recent literature. (Germination physiology is a thriving topic. See the Introduction for a sample of recent references.)

The unfortunate upshot is that attempts at testing the theoretical results of this note will probably require major research efforts.

DISCUSSION

So far, the theoretical research community has tried to explain delayed germination mainly by refining over and over again Cohen's (1966) model. However, alternative explanatory schemes, such as kin selection in viscous populations (Kobayashi and Yamamura, 2002; see also Venable and Lawlor, 1980; Ellner and Shmida, 1981; Westoby, 1981; Ellner, 1986), should also be considered. The explanation proposed in this note reflects the results of a large body of empirical literature from temperate regions (Harper, 1977; Fenner, 1985; Rees, 1997; Baskin and Baskin, 1998; Silvertown and Charlesworth, 2001; Fenner and Thompson, 2005). It turns out that under the envisioned scenarios, evolution may in principle also lead to germination probabilities smaller than one, even if we consider only seeds that happen to be in the best possible circumstances for producing a functioning adult plant. The reason is that too large a willingness to germinate under such fortunate circumstances has to be paid for by a risk of germinating when there is no chance of seedling success. Making no further assumptions than that all seeds are equal, leaving open all other details of the plant life cycle, we have shown that only a fraction of the seeds in safe sites should germinate when the relation between the probability to germinate in safe sites and elsewhere is convex and has a sufficiently steep slope at the highest germination probabilities. What remains is the – unfortunately awesomely difficult – empirical question of how common such trade-offs are in the real world.

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