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A different model to explain delayed germination

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Contents

A different model to explain delayed germination[1]

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[1] This paper is dedicated to Tom Vincent, to honour his life and work. We unfortunately only met in passing. However, studying his work has been enriching and enjoyable.

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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 optimisation principle satisfied by the Evolutionarily Steady Strategy vector consisting of the probabilities to germinate in safe sites and elsewhere. Using this optimisation 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 relation 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.

Key words: delayed germination, optimisation principle, Levins' fitness set, life stages

1 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. The standard explanation in the theoretical literature for this phenomenon, starting with the work of Dan Cohen (1966, 1967) through the work of Michael Bulmer (1984), Steve Ellner (1985a,b, 1986), Joel Brown and Larry Venable (1986, 1991; and in opposite order 1988), Dan Cohen and Si Levin (1987), ourselves with John Val (Klinkhamer et al., 1987), Mark Rees (1994), Larry Venable (1989), Andrea Mathias and Éva Kisdi (2002), Michael Easterling and Steve Ellner (2002), Sami Aikio, Esa Ranta, Veijo Kaitala and Per Lundberg (2002) to Angelo Valleriani, on his own (2005, 2006) and in collaboration with Katja Tielbörger (2006; and in opposite order 2005), is as a bet hedging strategy in time-varying environments.

Bet-hedging models assume that environmental conditions vary in time but at each point in time are the same for all plants. Despite this uniformity, plants are predicted to develop adaptations that make that in a single germination window not all of their seeds germinate. Bet-hedging strategies may be realised by some coin flipping mechanism of the seed or by parents producing more types of seeds with different germination characteristics.

We are more familiar with a different scenario. In our experience seeds germinate mainly as reaction to local disturbances, and hence selection may be expected to improve this reaction in order to let them end up as reproductive adults as often as possible. 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 Rees (1997). Recent references are by Jankowska-Blaszczuk and Daws (2007), who analysed how high red : far red ratios can stimulate local germination propensity, and by Vandelook et al. (2008), who 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 again is through 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 good circumstances for germination arise only after rainfalls that affect relatively large areas.

In this paper we describe a model covering the fine-scaled temperate outlook. We start with considering the simplest possible scenario, annuals with only seedling interaction. This interaction is idealised in that the world is divided up in places where seedlings cannot and so-called safe sites, small patches where they can survive (c.f. 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 come 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 strategy variable we choose the germination probability in safe sites. We rigged this eco-evolutionary model such that it satisfies an optimisation 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 e.g. Metz (2008) p. 1605 for an explanation: only ESSes 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 optimisation principle guarantees that the corresponding ESSes are moreover evolutionarily attracting (Continuously Stable).

In principle, the existence of an optimisation principle excludes a plethora of potential evolutionary phenomena. However, for our purpose, to wit 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 removed without changing the results. We cap off with a discussion of the experimental options for testing the theory.

2 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 is 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 is just over:

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 till 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

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

with, when there are no mutants around yet,

$$P = h\,\Phi(GX), \qquad (2)$$

$\Phi(GX)$ the expected number of surviving seedlings in a safe site if the average density of germinating seeds in a site is GX. In words, the seeds of next year, the density of which is denoted as X', will consist of this year's seeds that neither germinate nor succumb plus the surviving new seeds from this year. P equals the density of safe sites times 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 the area of all safe sites is the same,

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

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 ingredients:

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 and decelerates, 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 linearised recurrence for the mutant at the pure resident 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 will 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

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

Apparently evolution maximises

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

3 Calculating the Evolutionarily Steady Strategy

The optimisation principle M increases in G and decreases in 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 possible, time honoured (see e.g. De Jong and Klinkhamer, 2005), relation between F and G

being

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

For this trade-off, with G^* the Evolutionarily Steady value of G,

$$\text{for } \beta > 1 \text{ and } \alpha(\beta-1) > \frac{1-S}{(1-k)S}: \qquad 0 < G^* = \left(\frac{1-S}{S\alpha(1-k)(\beta-1)}\right)^{1/\beta} < 1,$$
$$\text{else:} \qquad G^* = 1. \tag{16}$$

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. Else, seeds in safe sites should just germinate, independent of the cost that this incurs through seeds also germinating elsewhere.

A more general analysis is possible using Levins's idea of fitness sets (Levins, 1962, 1969; see Rueffler et al., 2004, for an extension to cases without optimisation principle). According to tradition, in such an approach the axes should correspond to life history parameters in which the evolutionarily maximised quantity increases. Moreover, it helps when the contour lines of the optimisation principle are straight lines. This naturally leads to the choice of G and $1-F$, with as formula for the fitness contours

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

The trade-off we denote 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 Figure 1a in cases where seeds should not all germinate. When the fitness set is concave instead of convex, seeds should always germinate, and the same is the case when for a convex fitness sets (see Figure 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).

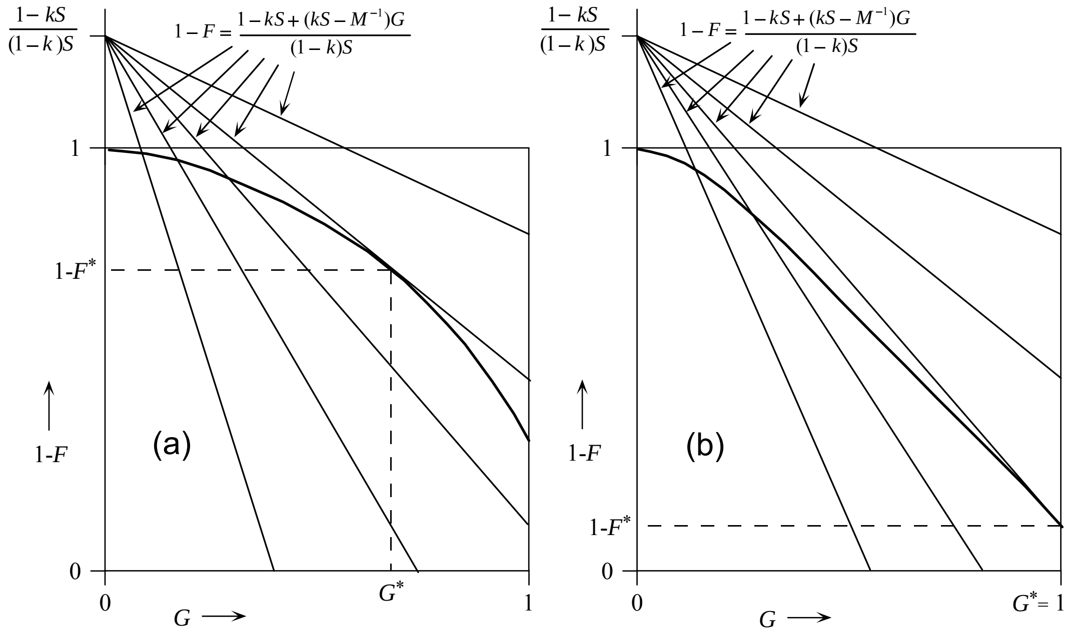

Figure 1: Two possible convex fitness sets together with a sample of contour lines of the optimisation criterion. The left configuration leads to an internal ESS, the right one to a boundary ESS.

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

To interpret the criterion (19), observe that at $G = 1$ at the population dynamical equilibrium X^* the pay-off, in seeds next year, of germinating when in a safe site equals $UYh\Psi(GX^*) = 1\text{-}S[1\text{-}k\text{-}(1\text{-}k)F] = 1\text{-}(1\text{-}k)(1\text{-}T(1))S$; see (9). With this in mind the criterion (19) can be rewritten as

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

In words: 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 $-\text{d}T/\text{d}G$ 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 with S in order to get the seeds of next year.

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

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

which can be rewritten as

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

In words: at internal ESSes 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 minimises 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 makes that evolution lets the organisms end up in the worst possible world.

4 Removing the ecological simplifications

To see which of the ecological simplifications made earlier are essential for reaching the conclusions from the previous section, we fall back 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 maximises $R_0(\cdot,\mathbf{E}_0)$.” This suggests considering life histories of the form depicted in Figure 2.

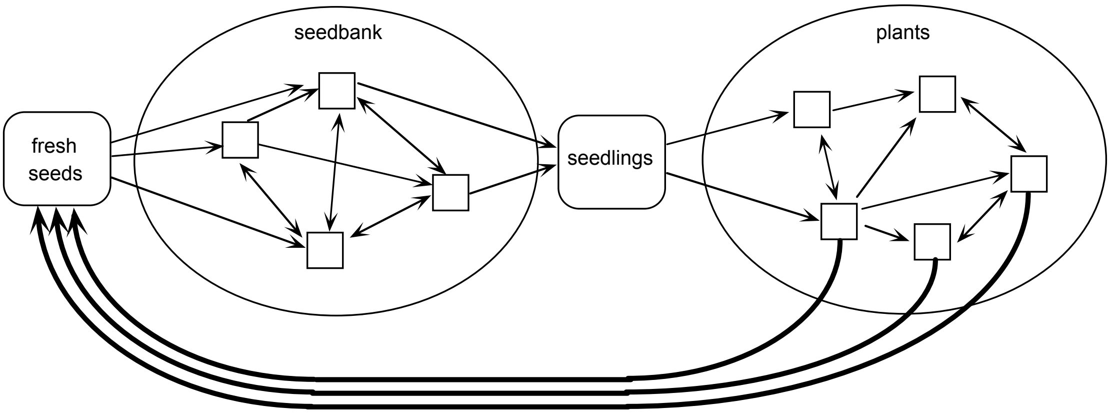

Figure 2: A generalised plant life history with the restriction that all seeds are initially equal, and so are all seedlings.

The essential characteristic of the life histories symbolically represented by Figure 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 e.g. (age, depth in soil)-respectively (age, size above ground, size below ground)-classes. 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 $\mathbf{E}_\mathrm{b}$ (consisting of e.g. seed predation pressures and fungal and bacterial attack rates at different depths; $_\mathrm{b}$ from belowground), (2) influences on the seedlings, including their own average initial density in safe sites Z, to be represented by a vector $\mathbf{E}_\mathrm{s}$, and (3) influences after the seedling stage, to be represented by a vector $\mathbf{E}_\mathrm{a}$ (capturing all direct and indirect competitive influences within the community through shading, nutrient depletion and changing predation pressures; $_\mathrm{a}$ from aboveground). Since the birth of a safe site necessarily coincides with the demise or a state change of one or more plants in the community it may also be assumed that $\mathbf{E}_\mathrm{a}$ 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,
$\mathbf{U}(\mathbf{E}_\mathrm{b})$: state distribution of a new seed just prior to germination time,
$\mathbf{S}(\mathbf{E}_\mathrm{b})$: matrix of survival and state transition probabilities of seeds,
$h(\mathbf{E}_\mathrm{a})$: density of safe sites available at germination time,
$k(\mathbf{E}_\mathrm{a})$: 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,
$\Psi(\mathbf{E}_\mathrm{s})$: average number of seedlings in a safe site that survive seedling competition divided by the average density of novel seedlings in safe sites,
$\mathbf{J}(\mathbf{E}_\mathrm{a})$: state distribution of young plants that have survived seedling competition,
$\mathbf{A}(\mathbf{E}_\mathrm{a})$: matrix of survival and state transition probabilities of plants,
$\mathbf{Y}(\mathbf{E}_\mathrm{a})$: 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 **U**, will in general be defective (i.e., has total mass smaller 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 **J**, will probably be concentrated on but a single plant state: small juvenile. **J** by definition has full mass as the probabilities of seedling death are all accounted for by $\Psi(\mathbf{E}_\mathrm{s})$.

The resident population state satisfies the following recurrences

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

with

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

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

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

These equations should be combined with equations for the remainder of the community to determine ($\mathbf{E}_\mathrm{a}$,$\mathbf{E}_\mathrm{s}$,$\mathbf{E}_\mathrm{b}$). As it turns out, this model is still a bit too general to allow the ES germination strategy to be determined from an optimisation 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 lifetime offspring number of a resident 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 & Snell (1960) it follows that these numbers are given by the vector $(\mathbf{I}-\mathbf{A}(\mathbf{E}^*_{\mathrm{a},\mathbf{G},\mathbf{F}}))^{-1}\mathbf{J}(\mathbf{E}^*_{\mathrm{a},\mathbf{G},\mathbf{F}})$, with $(\mathbf{E}^*_{\mathrm{a},\mathbf{G},\mathbf{F}},\mathbf{E}^*_{\mathrm{s},\mathbf{G},\mathbf{F}},\mathbf{E}^*_{\mathrm{b},\mathbf{G},\mathbf{F}})$ the equilibrium environment, to be determined from the full community dynamical equations for the resident strategy (**G**,**F**). Hence, the average number of seeds that a plant that just germinated in a safe site will produce over its lifetime is $\mathbf{Y}^{\mathrm{T}}(\mathbf{E}^*_{\mathrm{a},\mathbf{G},\mathbf{F}})(\mathbf{I}-\mathbf{A}(\mathbf{E}^*_{\mathrm{a},\mathbf{G},\mathbf{F}}))^{-1}\mathbf{J}(\mathbf{E}^*_{\mathrm{a},\mathbf{G},\mathbf{F}})\Psi(\mathbf{E}^*_{\mathrm{s},\mathbf{G},\mathbf{F}})$. Similarly, the average number of germination moments that a seed experiences while in various seed states equals $\left(\mathbf{I}-\mathbf{S}(\mathbf{E}^*_{\mathrm{b},\mathbf{G},\mathbf{F}})\left(\mathbf{I}-k(\mathbf{E}^*_{\mathrm{a},\mathbf{G},\mathbf{F}})\mathrm{diag}(\mathbf{G})-(1-k(\mathbf{E}^*_{\mathrm{a},\mathbf{G},\mathbf{F}}))\mathrm{diag}(\mathbf{F})\right)\right)^{-1}\mathbf{U}(\mathbf{E}^*_{\mathrm{b},\mathbf{G},\mathbf{F}})$. Therefore, the probability of a seed germinating in a safe site instead of dying or germinating elsewhere equals $\mathbf{G}^{\mathrm{T}}\left(\mathbf{I}-\mathbf{S}(\mathbf{E}^*_{\mathrm{b},\mathbf{G},\mathbf{F}})\left(\mathbf{I}-k(\mathbf{E}^*_{\mathrm{a},\mathbf{G},\mathbf{F}})\mathrm{diag}(\mathbf{G})-(1-k(\mathbf{E}^*_{\mathrm{a},\mathbf{G},\mathbf{F}}))\mathrm{diag}(\mathbf{F})\right)\right)^{-1}\mathbf{U}(\mathbf{E}^*_{\mathrm{b},\mathbf{G},\mathbf{F}})$. Multiplying these two numbers gives

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

Hence,

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

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

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

Hence, ESSes can be determined by optimising (**G**,**F**) in

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

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

If and only if **G** and **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, $\mathbf{S}(\mathbf{E}^*_{\mathrm{b},\mathbf{G},\mathbf{F}}) = \bar{\mathbf{S}}$, $\mathbf{U}(\mathbf{E}^*_{\mathrm{b},\mathbf{G},\mathbf{F}}) = \bar{\mathbf{U}}$ and $k(\mathbf{E}^*_{\mathrm{a},\mathbf{G},\mathbf{F}}) = \bar{k}$, the function $(\mathbf{G},\mathbf{F}) \mapsto \tilde{M}(\mathbf{G},\mathbf{F};\bar{k},\bar{\mathbf{S}},\bar{\mathbf{U}})$ is an evolutionary optimisation 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 $M(G,F;k,S) = G\big/\left\{1-S\left[1-kG-(1-k)F\right]\right\}$ and the graphical considerations from the previous section apply also for the ESS, with the modification that $S(\mathbf{E}^*_{\mathrm{b},G,F})$ and $k(\mathbf{E}^*_{\mathrm{a},G,F})$ 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 $0 \le 1 - F \le T(G) \le 1$ 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 $k(\mathbf{E}^*_{\mathrm{a},1,T(1)})$ and $S(\mathbf{E}^*_{\mathrm{b},1,T(1)})$. However, since in applications (assuming boldly that these will once 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 difference has greater conceptual than practical significance.

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

5 Experimental tests?

Although the mathematical results are pretty clear-cut, there is still the problem how well they relate to biological reality. First of all, 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 yet 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 is the case 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 too gross an oversimplification, and that the non-germinating seeds, although located in safe sites, may still be in a lesser position as competitors, for example since 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. If indeed light were the only signal it may well 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 clue 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 good. In the previous section we wrote down a model that in principle can handle such more complicated mechanisms, but only in order to show what structural properties of the model lead to the existence of an optimisation 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 of 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.

Finally, for many species the dichotomy between safe sites and deadly remaining territory will be an oversimplification in a different respect, with the degree of safety being more gradual, requiring our model to be extended in a different way. In our view the dichotomy appears not too unreasonable a first approximation for at least a fair number of species, although in any specific case this remains up to the field biologist concerned to decide.

But possibly for the sharp dichotomy between safe sites and deadly remaining territory, the ecological ingredients of the general model introduced from the previous section are pretty indisputable. This is less so for the physiological ingredients introduced one section earlier. In order to comply with the traditions of Levin's 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 realised germination propensities of seeds that genetically differ, 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 germinating. 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 into a trade-off curve, 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.

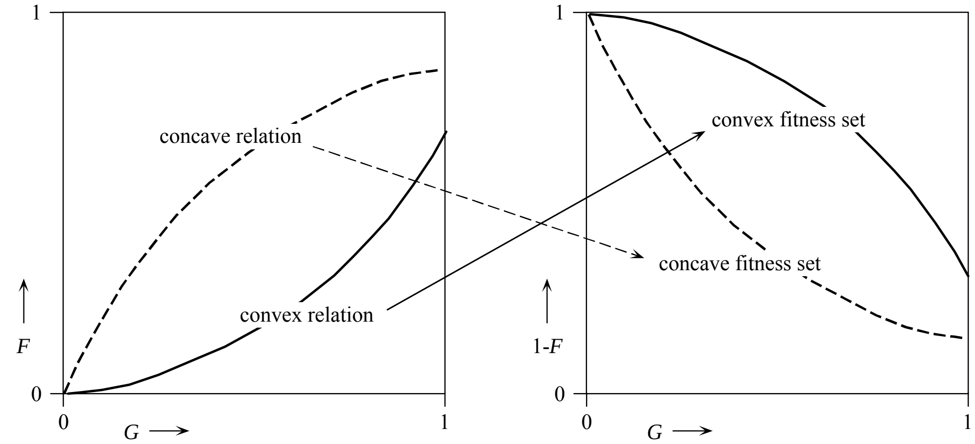

Figure 3: Connection between the relation between *G* and *F* and the corresponding fitness set. According to standard mathematical terminology when the relation between *G* and *F* is convex the trade-off between *G* and 1-*F* is concave and the fitness set is convex. Similarly, when the relation between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

6 Discussion

So far the theoretical research community has primarily kept refining ever more the model of Cohen (1966) for the evolution of delayed germination. However, alternative explanatory schemes, such as kin selection in viscous populations (Kobayashi and Yamamura, 2002; see also Venable and Lawlor, 1980; Westoby, 1981; Ellner and Shmida, 1981; Ellner, 1986), also should be considered. The potential explanation proposed in this note aims at reflecting more of the results of a large body of empirically oriented 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 in such more fortunate circumstances has to be paid for by a risk to germinate when there is no chance that the resulting seedling will ever be successful. Making no further assumptions than that all seeds are equal, leaving open all other details of the plant life cycle, we showed 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. Remains the, unfortunately awesomely difficult, empirical question how common such trade-offs are in the real world.

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