Received: 3 June 2019 | Revised: 4 September 2019 | Accepted: 6 September 2019

DOI: 10.1111/jeb.13543

RESEARCH PAPER

JOURNAL OF Evolutionary Biology eseb WILEY

The joint evolution of lifespan and self-fertilization

Thomas Lesaffre | Sylvain Billiard

CNRS UMR 8198 - Evo-Eco-Paleo, Université de Lille, Villeneuve d'Ascq, France

Correspondence
Thomas Lesaffre, CNRS UMR 8198 - Evo-Eco-Paleo, Université de Lille, F-59655 Villeneuve d'Ascq, France.
Email: thomas.lesaffre@univ-lille.fr

Funding information
European Research Council, Grant/Award Number: 648321

Abstract

In Angiosperms, there exists a strong association between mating system and lifespan. Most self-fertilizing species are short-lived, and most predominant or obligate outcrossers are long-lived. This association is generally explained by the influence of lifespan on the evolution of the mating system, considering lifespan as fixed. Yet, lifespan can itself evolve, and the mating system may as well influence the evolution of lifespan, as is suggested by joint evolutionary shifts of lifespan and mating system between sister species. In this paper, we build modifier models to study the joint evolution of self-fertilization and lifespan, including both juvenile and adult inbreeding depression. We show that provided that inbreeding depression affects adult survival, self-fertilization is expected to promote evolution towards shorter lifespan, and that the range of conditions under which selfing can evolve rapidly shrinks as lifespan increases. We study the effects of inbreeding depression affecting various steps in the life cycle and discuss how extrinsic mortality conditions are expected to affect evolutionary associations. In particular, we show that selfers may sometimes remain short-lived even in a very stable habitat, as a strategy to avoid the deleterious effects of inbreeding.

KEYWORDS
coevolution, generations overlap, inbreeding depression, life history, mating system

1 | INTRODUCTION

In Angiosperms, strong associations exist between mating systems and other life-history traits, such as dispersal (Auld & Rubio de Casas, 2012), allocation to male versus female functions (Brunet, 1992) or lifespan. Despite significant empirical work (Barrett & Harder, 1996; Munoz, Violle, & Cheptou, 2016; Scofield & Schultz, 2006), the latter association has received limited attention from the theoretical standpoint. Indeed, while it has long been recognized that most self-fertilizing species are short-lived and most predominant or obligate outcrossers are long-lived (Barrett & Harder, 1996; Duminil, Hardy, & Petit, 2009; Munoz et al., 2016; Stebbins, 1950), relatively few theoretical arguments have been advanced to explain this association.

One of the most prevalent arguments to explain the evolution of self-fertilization is reproductive assurance: contrary to an outcrosser, a selfer is able to reproduce in the absence of pollinators or compatible mating partners, which grants it an advantage when pollination is inefficient (Eckert, Samis, & Dart, 2006). Stebbins (1950) proposed that this advantage may be weaker in perennials compared to annuals, since perennials get more than one try at reproducing, and therefore may be less impacted by poor pollination during a given mating season. Later, Lloyd (1992) suggested that self-fertilization, because it ensures reproduction, could also cause the consumption of resources that could have been more advantageously allocated to post-breeding survival or future outcrossed reproduction in perennials, yielding an additional cost to self-fertilization is such species (between-seasons seed discounting). Morgan, Schoen, and Bataillon (1997) investigated the validity of these arguments by developing a phenotypic model and concluded that the association between annuality and

selfing is more likely to be due to between-seasons seed discounting, rather than reproductive assurance. They also showed the importance of the repeated effect of adult inbreeding depression for the maintenance of outcrossing in perennials. Empirical evidence also suggests that inbreeding depression is overall higher in perennials than in annuals (Angeloni, Ouborg, & Leimu, 2011), which could constitute an additional barrier to the evolution of self-fertilization in perennials.

These arguments focus on the consequences of perenniality for the evolution of self-fertilization, considering lifespan as a fixed characteristic. Yet, lifespans evolve in nature (Stearns, 1992), and joint evolutionary shifts of lifespan and mating system have been documented. Indeed, the transition to self-fertilization is often associated with significant morphological changes, such as vegetative size and flower size reduction (the selfing syndrome, Sicard & Lenhard, 2011), and lifespan shortening compared to outcrossing relatives (Ehrlen & Lehtila, 2002). For example, *Arabidopsis thaliana* is a highly selfing annual that recently differentiated from its self-incompatible and perennial relatives *A. lyrata* and *A. halleri* (Clauss & Mitchell-Olds, 2006). Furthermore, studying pairs of sister species across 15 families, Grossenbacher, Briscoe Runquist, Goldberg, and Brandvain (2015) found numerous joint shifts towards selfing and annuality from outcrossing, perennial ancestors in genera such as *Mimulus* or *Medicago* and very few shifts to longer lifespans in association with selfing. In fact, the only such shifts they found were observed in the *Oenothera* genus, where segregation and recombination are suppressed when reproducing by self-fertilization, which implies that selfing individuals are effectively reproducing clonally (Johnson, Smith, & Rausher, 2009).

These examples show that joint shifts of mating system and lifespan almost always occur in the same direction, from outcrossing more long-lived ancestors towards self-fertilizing derived species with a shorter lifespan. In such situations, lifespan shortening could have allowed for the evolution of self-fertilization. Alternatively, the transition to self-fertilization could have induced evolution towards a shorter lifespan. This possibility has seldom been investigated. Indeed, classical studies of the evolution of lifespan predict that it should be fine-tuned to best fit the extrinsic mortality conditions experienced by the considered population, through optimal allocation of resources to reproduction, growth or survival (e.g. Cichoń, 1997; Schaffer, 1974) and senescence (e.g. Silvertown, Franco, & Perez-Ishiwara, 2001), but rarely consider the evolution of lifespan in interaction with other traits. From a theoretical standpoint, the only study that, to our knowledge, has investigated the influence of the mating system on lifespan evolution is that of Zhang (2000), who developed a phenotypic model for the joint evolution of reproductive effort and sex allocation in partially self-fertilizing hermaphrodites. Assuming a survival versus reproduction trade-off (Stearns, 1992), they reached the conclusion that reproductive effort increases (and lifespan decreases) when the selfing rate increases through greater allocation to the female function, provided that inbreeding depression is weak ($\delta < \frac{1}{2}$), female reproduction is very costly, and juvenile survivorship is constrained within a narrow range of values. Importantly however, Zhang (2000) assumed inbreeding depression to only affect the survival of juveniles to maturity, although inbreeding depression typically occurs over all stages of the life cycle (Husband & Schemske, 1996).

In summary, on the one hand, the influence of lifespan on the evolution of the mating system has been studied, considering lifespan as a fixed characteristic (Morgan et al., 1997). On the other hand, the potential influence of the mating system on the evolution of lifespan has only been scarcely investigated, assuming no inbreeding depression occurred in adults (Zhang, 2000). Finally, the question of the joint evolution of lifespan and mating system has never been tackled.

In this paper, we build modifier models (Kirkpatrick, Johnson, & Barton, 2002) to investigate the joint evolution of lifespan and selfing in a population with overlapping generations, including inbreeding depression affecting various steps in individuals' life cycle as fixed parameters. Following previous authors, we model the evolution of lifespan through that of reproductive effort, assuming a survival versus reproduction trade-off for which we consider various shapes (i.e. convex, linear or concave Stearns, 1992). We incorporate extrinsic mortality as a constant parameter (Schaffer, 1974; Zhang, 2000). We first study the evolution of each trait separately, taking the other as fixed, and incorporate inbreeding depression affecting juvenile and adult survival. In each case, we obtain accurate analytical approximations for the evolutionary stable strategies. We show that self-fertilization is expected to favour evolution towards shorter lifespans when inbreeding depression affects adult survival. Conversely, we show that the range of inbreeding depression under which selfing can evolve rapidly shrinks as lifespan increases, in agreement with previous work (Morgan et al., 1997). Then, using individual-centred simulations along with our previous analytical approximations, we study the joint evolution of lifespan and selfing. We study the effects of inbreeding depression affecting various steps in the life cycle and discuss how extrinsic mortality conditions are expected to affect evolutionary associations. In particular, we show that selfers may sometimes remain short-lived even in a very stable habitat, as a strategy to avoid the deleterious effects of inbreeding.

2 | METHODS

2.1 | Outline of the model

2.1.1 | Life cycle and demographic assumptions

We consider a very large population made of partially selfing hermaphrodites, which are assumed to be diploid. We assume the population stays at carrying capacity. This implies that juveniles may only settle in replacement of recently deceased adults (Figure 1). Once settled, juveniles reach maturity before the next mating event.

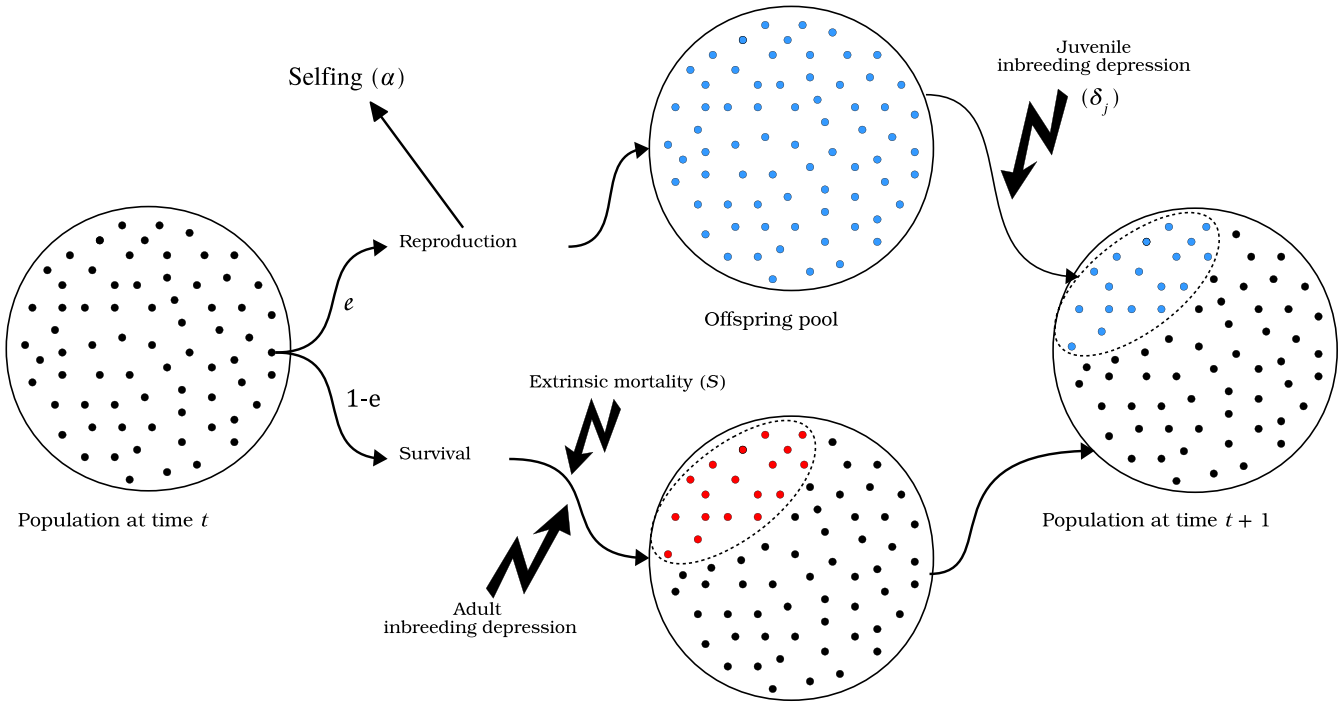

FIGURE 1 Schematic representation of the life cycle and demography assumed in the model. Established individuals allocate a fraction e of their resources to reproduction, and the remaining $1-e$ to survival. Juveniles replace deceased adults. Red dots depict deceased adults, and blue dots depict juveniles

We assume that adults keep the same fecundity and survival probability throughout their lives (i.e. no age-specific effects). Established individuals allocate a fraction e of their resources to reproduction and the remaining fraction $1-e$ to post-breeding survival. Consequently, sexually mature individuals have a certain probability of survival between mating events (say, flowering seasons) and generations may overlap: the more resources an individual allocates to reproduction, the larger its reproductive output, but the lower its chances of survival until the next mating event. During each mating event, individuals reproduce by self-fertilization in a proportion α and by random mating otherwise. Selfed offspring suffer from inbreeding depression (Charlesworth & Charlesworth, 1987) differently depending on the considered stage. As juveniles, they suffer from juvenile inbreeding depression, denoted δ_j, which decreases their probability of survival to maturity. If they reach maturity, they suffer from adult inbreeding depression, denoted δ_a, which decreases their survival probability between mating events. Denoting $S_o(e)$ the survival probability between two mating events of an outcrossed individual as a function of its reproductive effort, that of a selfed individual, $S_s(e)$, is therefore given by $S_s(e)=S_o(e)\times(1-\delta_a)$. In simulations, we also considered the case where selfed adults suffer from inbreeding depression on fecundity, which diminishes selfed individuals' contribution to the gamete pool by a proportion δ_f (Appendix 5).

Whether lifetime inbreeding depression, that is the decrease in lifetime fitness of selfed individuals relative to the outcrossed, varies with life expectancy depends on the life stages we assume inbreeding depression to affect. When inbreeding depression affects juvenile survival or fecundity, lifetime inbreeding depression is unaffected by life expectancy. On the contrary, when inbreeding depression affects adult survival, lifetime inbreeding depression increases with life expectancy (Appendix 1). Indeed, in the latter case, selfed individuals have less opportunities to reproduce, while in the former, selfed individuals have the same life expectancy as the outcrossed.

2.1.2 | Genetic assumptions

We assume that individuals' selfing rate and reproductive effort are each entirely determined by a single biallelic modifier locus. In each case, we consider the population to be initially fixed with one allele (the resident) and introduce a rare mutant allele which has a small effect on its bearer's phenotype; that is, we assume weak selection. We then follow the change in frequency of this mutant when it is rare, and look for situations where no mutant can increase in frequency, that is evolutionarily stable strategies (ESS, Maynard Smith & Price, 1973).

2.2 | Analytical methods

For each model, we obtained analytical predictions for the evolutionary stable strategies, using the theoretical framework introduced by Barton and Turelli (1991) and generalized by Kirkpatrick et al. (2002). Only a summary of the results is given in the main text, and detailed recursions can be found in Appendixes 1, 2 and 3.

2.3 | Numerical analyses and simulations methods

All programs used in the present study are available on GitHub (https://github.com/Thomas-Lesaffre/M2_project).

2.3.1 | Numerical analyses

The analytical results with approximations we obtain when studying the evolution of reproductive effort and selfing separately are compared with that of exact numerical analyses. For each model, the exact recursions, that is tracking genotypic frequencies (rather than allelic frequencies and genetic associations) with no approximations, are run for 10^6 generations. A rare mutant with a small effect ($p_m = 10^{-4}$, $\epsilon = 0.01$) is introduced at $t = 0$, and the resulting frequency of the mutant is compared to its initial one. When the mutant increases in frequency, the mutant allele is taken as resident, and recursions are run again introducing a new mutant, until mutants no longer increase in frequency or the analysis hits a bound (i.e. 0 or 1). As these analyses were only conducted for validation purpose, outputs are presented in Appendix 4. Our results showed that the approximations we obtain are very close to the numerically obtained ESS, when considering the evolution of reproductive effort and selfing separately.

2.3.2 | Individual-centred simulations

To study the joint evolution of lifespan and selfing, we performed individual-centred C++ simulations, incorporating inbreeding depression affecting juvenile (δ_j) and adult survival (δ_a). In Appendix 5, we also consider the influence of inbreeding depression affecting fecundity (δ_f). In simulations, individuals follow the same life cycle as described above (Figure 1), and their selfing rate and reproductive effort are each determined by one modifier locus, which are allowed to mutate in both directions, following a uniform distribution in $[\alpha_0 - d, \alpha_0 + d]$ and $[e_0 - d, e_0 + d]$, where α_0 and e_0 are the parent's selfing rate and reproductive effort, respectively, with $d = 10^{-1}$ and a mutation rate $U_m = 10^{-2}$. Free recombination ($r = \frac{1}{2}$) is assumed between the two loci.

3 | RESULTS

Throughout the following sections, we will need to track the proportion of selfed individuals in the population (Θ), which stays close to its equilibrium value in the absence of mutants (Θ^*) when mutants at modifier loci are rare and only weakly deviate from the resident strategy. As shown in Appendix 1, this equilibrium value is given by.

$$\Theta^* = \frac{\alpha\left(1-\delta_j\right)\left(1-S_o(e)\right)}{\alpha\left(1-\delta_j\right)\left(1-S_o(e)\right)+\left(1-\alpha\right)\delta_a S_o(e)}. \quad (1)$$

Equation (1) is a decreasing function of $S_o(e)$; that is, s selfed individuals in the population is decreased by overlapping generations. We show in Appendix 1 that this is due to the repeated effect of adult inbreeding depression on post-breeding survival.

3.1 | Evolution of lifespan in a partially selfing population

In this section, we analyse a model for the evolution of lifespan under partial selfing through the evolution of reproductive effort, considering the selfing rate α as a parameter and assuming inbreeding depression affects juvenile and adult survival. We assume that the reproductive effort of a given individual is entirely determined by its genotype at a single biallelic modifier locus. Alleles *M* and *m*, which we assume to be codominant, code for a reproductive effort $e = e_0$ and $e = e_0 + \epsilon$ ($\epsilon \ll 1$), respectively. Furthermore, following Zhang (2000), we assume that the survival probability between two mating events of an outcrossed individual as a function of its reproductive effort *e* is given by.

$$S_o(e) = S\left(1-e^x\right), \quad (2)$$

where s is the maximal survival probability, that is a measure of extrinsic mortality, and *x* controls the shape of the survival versus reproduction trade-off. We use this function form because it is flexible and allows for the consideration of a variety of trade-off shapes. The detailed recursions are given in Appendix 2. In brief, we follow the variation of three variables, namely the frequency of allele *m* (p_m), which is assumed to be a very rare mutant with a small effect ($\epsilon \ll 1$), the deviation in homozygosity at the modifier locus as compared to the panmictic expectation ($D_{m,m}$) and the proportion of selfed individuals in the population (Θ), which is assumed to remain close to its equilibrium value Θ^*. We look for evolutionarily stable strategies (ESS, Maynard Smith & Price, 1973), that is situations where no mutant allele *m* may invade the population and replace the resident allele *M*.

3.1.1 | Allelic frequencies change

To leading order in ϵ, we can express the change in frequency of allele *m* between two timesteps (Δp_m) as.

$$\Delta p_m = \frac{\epsilon}{2e_0}\left(p_m\left(1-p_m\right)+D_{m,m}\right)V + o(\epsilon^2), \quad (3)$$

with $V = 1 - S\left(1-\Theta\delta_a\right)\left(1+\left(x-1\right)e_0^x\right)$. Using a separation of timescales approximation (Kirkpatrick et al., 2002), that is assuming the deviation in homozygosity at the modifier rapidly equilibrates in comparison with allelic frequencies, we obtain a quasi-equilibrium value by solving $\Delta D_{m,m} = 0$ for $D_{m,m}$ (where $\Delta D_{\{m,m\}}$ is the change in excess in homozygotes at the modifier between two timesteps). Since $D_{m,m} \geq 0$ provided that $\epsilon \ll 1$, the first term in Equation (3) is always of the sign of ϵ, and only *V* matters for the determination of the equilibrium.

3.1.2 | Evolutionarily stable strategy

In order to determine the equilibrium reproductive effort e^*, we plug Equation (1) into V, and solve $V = 0$ for e_0, which yields.

$$e^* = \left[\frac{\mathcal{B} + \sqrt{(-\mathcal{B})^2 + 4(1-\mathcal{S})(x-1)(1-\delta_a)[1-\mathcal{S}\mathcal{S}(1-\delta_a)](1-\alpha\delta_j)^2}}{2\mathcal{S}(x-1)(1-\delta_a)(1-\alpha\delta_j)} \right]^{\frac{1}{x}}, \quad (4)$$

with $\mathcal{B} = (1-\mathcal{S})(2-x)(1-\alpha\delta_j) - \delta_a\left[(1-\mathcal{S}(2-x))(1-\alpha\delta_j) - x\alpha(1-\delta_j)\right]$.

For this equilibrium to be stable, that is for Equation (4) to be an ESS, it is required that.

$$\frac{\partial V}{\partial e} = x(1-x)e^{x-1}\mathcal{S}(1-\Theta\delta_a) < 0. \quad (5)$$

One can see that when the survival versus reproduction trade-off is convex or linear ($x \leq 1$), condition (5) is not fulfilled, which implies that $\Delta p_m \geq 0$. Thus, alleles increasing their bearer's reproductive effort are always favoured, and only annuality ($e^* = 1$) can evolve. On the other hand, when the trade-off is concave ($x > 1$), $\frac{\partial V}{\partial e} < 0$, Equation (4) is an ESS and perennial strategies can be maintained. This result is classical and has bsseen reached by numerous authors before (e.g. Bell, 1980; Schaffer, 1974; Zhang, 2000). In addition, Equation (4) is a decreasing function of $\mathcal{S}$. That is, higher extrinsic mortality favours higher reproductive efforts: allocating resources to survival becomes less and less advantageous as extrinsic mortality increases (i.e. as $\mathcal{S}$ decreases), because individuals are more likely to die independently of the resources they spend on survival. Hence, our model also predicts the classical observation that annuals are more likely to evolve in more disturbed habitats, where extrinsic mortality is higher.

Assuming $x > 1$, that is a concave trade-off, differentiating Equation (4) with respect to α yields.

$$\frac{\partial e^*}{\partial \alpha} = \frac{\delta_a(1-\delta_j)e^*}{(1-\alpha\delta_j)\sqrt{(-\mathcal{B})^2 + 4(1-P)(x-1)(1-\delta_a)[1-\mathcal{S}(1-\delta_a)](1-\alpha\delta_j)^2}} \geq 0. \quad (6)$$

Equation (6) shows that the ESS reproductive effort is an increasing function of the selfing rate α, as long as some selfed juveniles survive to maturity ($\delta_j \neq 1$), and adults suffer from inbreeding depression ($\delta_a \neq 0$). In other words, through its impact on adult survival, self-fertilization favours evolution towards higher reproductive efforts, and hence shorter lifespans. This effect becomes stronger as adult inbreeding depression increases, because allocating resources to survival becomes less beneficial in selfed adults, and as juvenile inbreeding depression decreases, because it reduces the proportion of selfed individuals entering the adult population. Therefore, we show that self-fertilization favours evolution of shorter lifespans.

3.2 | Influence of lifespan on the evolution of self-fertilization

In this section, we study the influence of lifespan on the evolution of self-fertilization assuming inbreeding depression only affects juvenile and adult survival, taking reproductive effort, that is lifespan, as a fixed parameter. We assume that the selfing rate of a given parent is entirely determined by a single biallelic locus. Alleles M and m, which are assumed to be codominant, code for selfing rates α_0 and $\alpha_0 + a$, respectively. Allele m is assumed to be a rare mutant with a weak effect ($a \ll 1$). In this section, we do not need to make any assumption regarding the shape of the survival versus reproduction trade-off. Hence, the general $\mathcal{S}_o(e)$ notation will be used. Recursions are given in Appendix 3. As in the previous section, we follow three variables: the frequency of the mutant allele (p_m), the deviation in homozygosity at the modifier locus as compared to the panmictic expectation ($D_{m,m}$) and the proportion of selfed individuals in the population (Θ), which is assumed to remain close to its equilibrium value (Θ^*, Equation (1) when the mutant is rare. We look for the conditions under which allele m invades the population.

3.2.1 | Allelic frequencies change

Plugging Equation (1) into the allelic frequencies change (Δp_m) yields, to leading order a,

$$\Delta p_m = -\frac{a(1-\alpha_0\delta_j)D_{m,m}(1-\mathcal{S}_o(e))}{2\left[\mathcal{S}_o(e)(1-\alpha_0)\delta_a + (1-\mathcal{S}_o(e))(1-\alpha_0\delta_j)\right]^2} \times T + o\left(a^2\right) \quad (7)$$

with $T = \left[1-\mathcal{S}_o(e)(1+\delta_a)\right]\left[\mathcal{S}_o(e)\delta_a - (1-\mathcal{S}_o(e))(1-2\delta_j)\right]$.

Using the same arguments as in the previous section, we show in Appendix 3 that for all α, $D_{m,m} > 0$. Hence, the first term in Equation (7) is always negative, and only T matters for the determination of the equilibrium. Since T does not depend on the selfing rate, there are only two possible situations: either $T > 0$ and full outcrossing ($\alpha = 0$) is favoured, or $T < 0$ and full selfing evolves ($\alpha = 1$), similar to the findings of previous authors (e.g. Lande & Schemske, 1985). Solving $T < 0$ for δ_j, we have.

$$T < 0 \Leftrightarrow \delta_j < \frac{1-\mathcal{S}_o(e)(1+\delta_a)}{2(1-\mathcal{S}_o(e))}, \quad (8)$$

which simplifies to $\delta_j < \frac{1}{2}$ in the annual case ($\mathcal{S}_o(e) = 0$). Otherwise, this threshold is a decreasing function of $\mathcal{S}_o(e)$ when $\delta_a > 0$. Hence, we find that the range of conditions under which self-fertilization can evolve in a population decreases when lifespan increases, provided that inbreeding depression affects adult survival, in agreement with previous results (Morgan et al., 1997). This result implies that even very weak adult inbreeding depression is sufficient to prevent evolution of self-fertilization in long-lived species. Equation (8) was validated using numerical analyses, which were

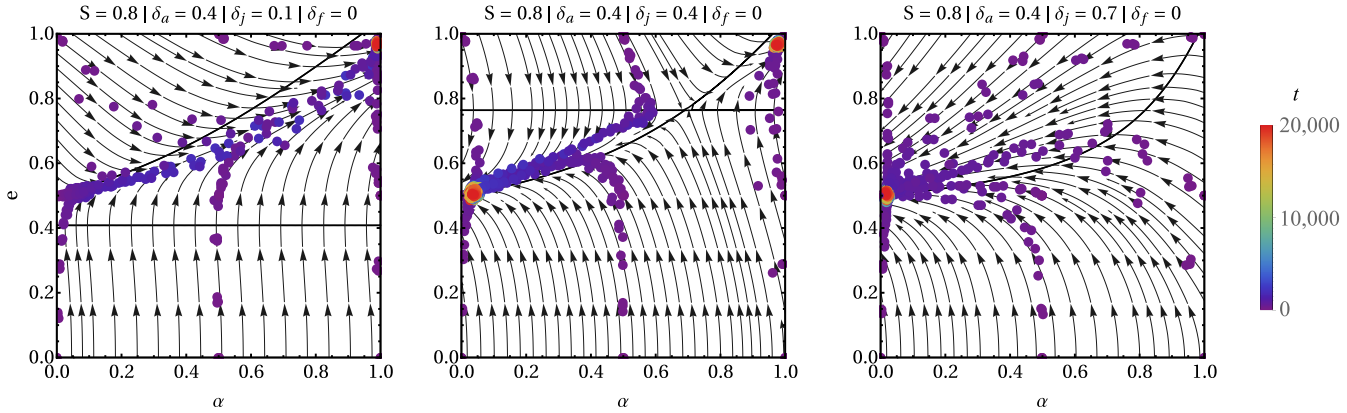

FIGURE 2 Phase diagrams highlighting the three kinds of behaviour that can arise from the coevolutionary dynamics of lifespan and selfing. Reproductive effort (e) is plotted against selfing rate (α). Solid lines depict isoclines, and arrows indicate how the joint evolution behaves. Points depict simulation results and are coloured with respect to time

found to be in very good agreement with the analytical prediction (Appendix 4).

3.3 | Joint evolution of lifespan and self-fertilization

In this final section, we study the joint evolution of lifespan and selfing. Figure 2 highlights the different situations that arise when considering the joint evolution of lifespan and selfing. The threshold reproductive effort above which selfing can evolve ($\bar{e}$) can be obtained by setting $S_o(e) = S\left(1-e^x\right)$ in Equation (8), and solving $T < 0$ for e. It is given by.

$$\bar{e} = \left(\frac{S(1+\delta_a)+2\delta_j(1-S)-1}{S(1+\delta_a-2\delta_j)}\right)^{\frac{1}{x}}. \quad (9)$$

Overall, the transition to selfing is always associated with increased reproductive effort, that is reduced lifespan. Depending on the inbreeding depression and extrinsic mortality conditions, one of three things can happen: (1) selfing is always favoured (Figure 2a), and reproductive effort evolves to.

$$e^*_{\text{self}} = \left(\frac{1-S(1-\delta_a)}{S(1-\delta_a)(x-1)}\right)^{\frac{1}{x}}, \quad (10)$$

(2) outcrossing is always maintained (Figure 2c), and reproductive effort stabilizes at.

$$e^*_{\text{out}} = \left(\frac{1-S}{S(x-1)}\right)^{\frac{1}{x}}, \quad (11)$$

(3) either of the two stable equilibria ($\alpha = 0$ and $\alpha = 1$) can be reached, depending on initial conditions (Figure 2b). This occurs when adult inbreeding depression is high enough, provided that $\delta_j < \frac{1}{2}$ (Figure 3). Selfers then stabilize at a high reproductive effort, which allows them to escape the deleterious effects of adult inbreeding depression. In other words, in situations where extrinsic mortality conditions would allow for the evolution of longer lifespans, selfers could remain short-lived solely owing to the deleterious effects of inbreeding.

Figure 3 summarizes the evolutionary stable outcomes of the joint evolution of lifespan and selfing in various inbreeding depression and extrinsic mortality conditions. Simulation results are in good agreement with analytical predictions, although small deviations are observed in Figures 2 and 3 for high and low selfing rates and reproductive efforts. These deviations are a consequence of the value constraints applied to the traits to keep them within bounds. Indeed, as the trait value (i.e. selfing rate or reproductive effort) approaches a bound of its definition domain (i.e. 0 or 1 on the [0,1] interval), it will tend to mutate slightly away from it, as all mutations exceeding the bound will be cut back to the bound value (e.g. a mutation at the reproductive effort modifier coding for a reproductive effort $e > 1$ will be cut to $e = 1$)

In Appendix 5, we also consider the effect of inbreeding depression affecting fecundity (δ_f). We show that δ_f or δ_j have the same effect: they reduce the range of conditions under which the full selfing strategy can evolve. The mechanisms underlying these similar behaviours are, however, slightly different. Indeed, whereas both cases they are caused by a reduction of the participation of selfed individuals to reproduction, δ_j decreases the proportion of selfed individuals in the population. On the other hand, δ_f reduces the contribution of selfed adults to reproduction by reducing their contribution to the gamete pool.

4 | DISCUSSION

We built modifier models to investigate the joint evolution of lifespan and mating system. To the best of our knowledge, this is the first time modifier models are used to study the evolution of life-history traits in a population with overlapping generations.

Although mixed mating is common in flowering plants (Munoz et al., 2016), we found only two stable mating systems:

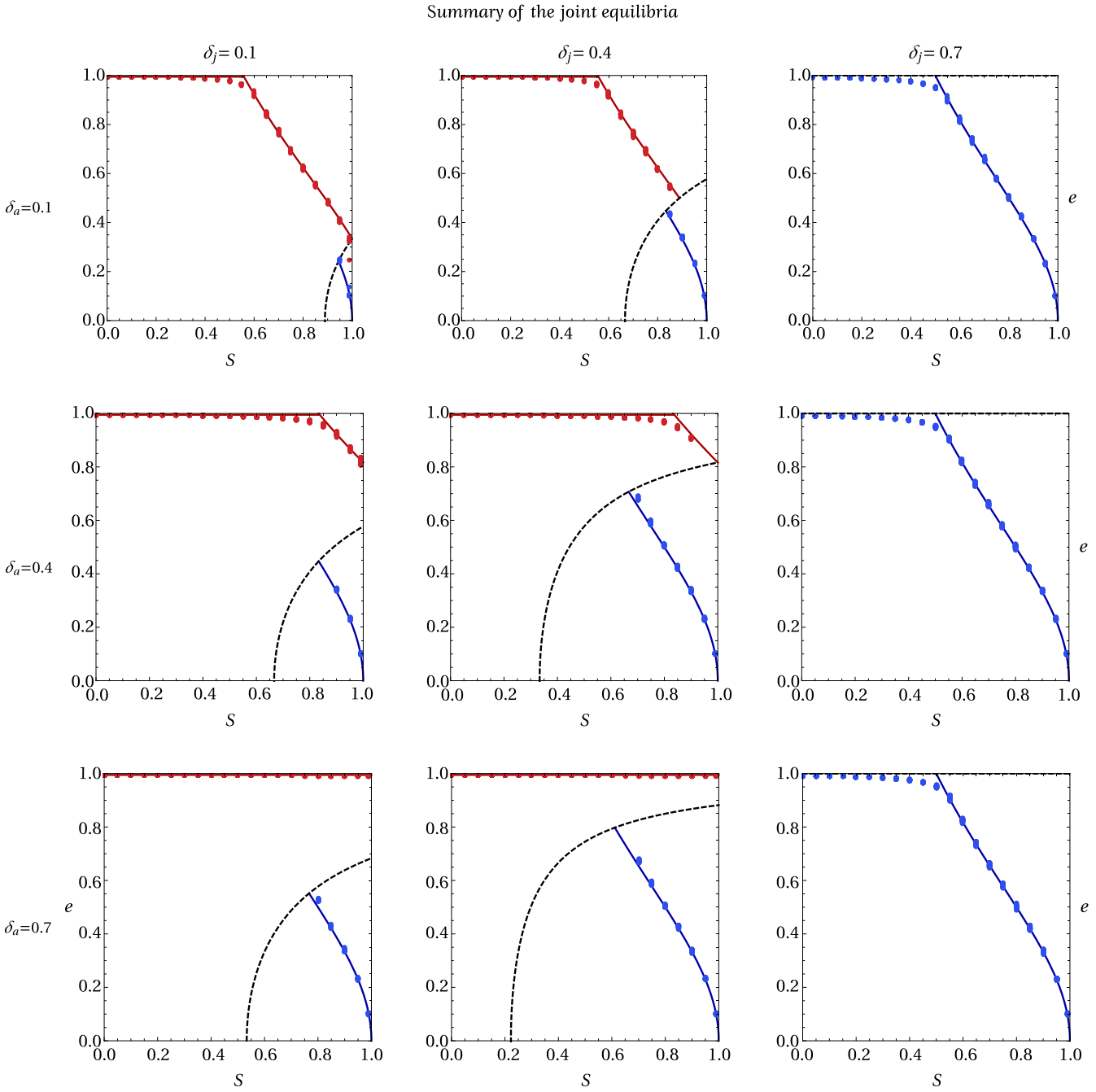

FIGURE 3 Joint evolutionary equilibria. All results presented here assume x = 2. Y-axis is the ESS reproductive effort (e^*), and X-axis is extrinsic mortality (S). The lines correspond to analytical results, whereas dots depict simulations results. The red ones corresponds to the equilibrium reproductive effort when α = 1 is reached, whereas the blue line corresponds to the equilibrium reproductive effort when α = 0 is reached. The dashed black line represents the threshold lifetime inbreeding depression under which outcrossing can be maintained

full outcrossing or full selfing, similar to previous authors (e.g. Charlesworth, Morgan, & Charlesworth, 1990; Lande & Schemske, 1985). This discrepancy between theoretical predictions and empirical observations has given rise to multiple theoretical investigations. All of which proposed complications of earlier models which allow mixed mating to be maintained as an evolutionarily stable strategy (Goodwillie, Kalisz, & Eckert, 2005; Barrett & Harder, 2017}, but go beyond the scope of the present paper. The role of lifespan in the maintenance of mixed mating may, however, represent a track worth following.

In agreement with Morgan et al. (1997), we found that increasing lifespan considerably reduces the range of conditions under which self-fertilization can evolve when inbreeding depression affects adult survival. This effect is due to the fact that life expectancy (and therefore lifetime reproductive success, that is fitness in our model) scales exponentially with survival probability. Indeed, when

survival probability in the population is higher, its reduction among selfed individuals due to inbreeding depression is more consequential for fitness than it is in more short-lived species, which leads to higher lifetime inbreeding depression and therefore greater selection against selfing. Furthermore, we found inbreeding depression on juvenile survival and fecundity (Appendix 5) to affect the coevolutionary dynamics similarly, as they both reduced the range of conditions under which the full selfing strategy can be reached. This is because decreasing the contribution of selfed parents to the production of offspring by reducing the amount of gametes they produce (fecundity) or diminishing their frequency among the parents (juvenile survival) is equivalent in our model. Conversely, we showed that self-fertilization is expected to cause evolution towards shorter lifespans due to inbreeding depression affecting adult survival, even under the low inbreeding depression conditions required for selfing to evolve in long-lived species. In addition, we showed that self-fertilizing species could remain short-lived even in a very stable habitat, as a strategy to avoid the deleterious effects of inbreeding. Finally, we showed that long-lived selfers can only emerge under very weak adult inbreeding depression and should be very rare. This is corroborated by the work of Munoz et al. (2016), who found that only 2\% of woody perennials are obligatory selfers.

The only previous study of the influence of the mating system on the evolution of lifespan is that of Zhang (2000). Whereas our main conclusions are in line with theirs, they also differ in a number of ways. Zhang (2000). concluded that self-fertilization could induce evolution of shorter lifespans through increased allocation to female reproduction, without any role for adult inbreeding depression. Zhang (2000)'s results and ours are not mutually exclusive, since they rely on different mechanisms. However, we expect our prediction to apply more generally. Indeed, whereas our predictions only require inbreeding depression to affect adult survival, which seems reasonable, as inbreeding depression commonly occurs over all stages of life (Husband & Schemske, 1996), Zhang (2000)'s prediction requires that female reproduction is sufficiently costly, that inbreeding depression remains low and that juvenile survivorship is constrained within a narrow range of values. Importantly, Zhang (2000)'s approach and ours also differ widely in the methods used, as we assume that juveniles are produced in large excess with respect to the available resources, so that the population reaches its carrying capacity before each flowering seasons and the population size is kept constant, whereas Zhang (2000) modelled adult survival as an additive term (see Equation 3 in their study), which is only valid in the case of an exponentially growing population, where juvenile recruitment is not limited by resource availability (Kozłowski, 1993; Stearns, 1992). Small founding populations, which cannot produce enough offspring to saturate their habitat over the course of one mating season, may exhibit such rapid growth. However, no population grows forever as resources sooner or later become limiting. Since nonannual species, and particularly long-lived species such as trees, tend to inhabit more stable environments (Petit & Hampe, 2006; Schaffer, 1974), we expect relatively stable populations to be more widespread than exponentially growing populations, which is a necessarily transient state, and therefore would also expect our approach to hold greater generality than that of Zhang (2000).

Overall, both theoretical and empirical results indicate that predominantly selfing species are almost always short-lived, while predominantly outcrossing ones are mostly long-lived, but the causative mechanisms underlying this correlation still require theoretical and empirical work to be unravelled. One role of models such as the present study is to provide putative ecological and genetic conditions under which a given association may emerge. Given these results, we propose that lifespan could act as a confounding factor when considering the joint evolution of self-fertilization with other traits. For example, lifespan shortening following the transition to self-fertilization could affect other traits, thereby contributing to the emergence of the selfing syndrome, rather than self-fertilization per se alone. Indeed, as a shorter lifespan implies that individuals have to complete their life cycle more rapidly, they could grow smaller flowers regardless of an adaptation to more efficient selfing (Sicard & Lenhard, 2011). Accounting for lifespan could also shed new light on some long-standing evolutionary questions related to the evolution of mating systems. For instance, lifespan could influence the joint evolution of dispersal and self-fertilization in two ways. First, increased lifespan induces higher local relatedness (Duputié & Massol, 2013), and perennials tend to occupy more saturated, competitive environments. Hence, kin competition should be greater in more long-lived species, and long-distance dispersal could be favoured as a mean to avoid it. Since perennials outcross more than annuals, this could generate an indirect association between long-distance dispersal and outcrossing through lifespan. Second, the repeated effect of adult inbreeding depression considerably increases the detrimental effects of mating among relatives in perennials. Long-distance dispersal and outcrossing could thus be favoured jointly in such species as an inbreeding avoidance strategy (Auld & Rubio de Cases, 2012).

Throughout this work, we assumed fixed inbreeding depression. Inbreeding depression is generally thought to be caused by recessive deleterious mutations segregating at low frequencies in populations (Charlesworth & Willis, 2009). The population genetics of such mutations in populations with overlapping generations are poorly understood theoretically. Evidence from large-scale meta-analyses suggest that the measured magnitude of inbreeding depression increases as species' lifespan increases (Angeloni et al., 2011; Duminil et al., 2009). However, it is unclear whether this pattern is due to mating system differences between long-lived and short-lived species (Munoz et al., 2016), or to lifespan. Additionally, empirical studies of inbreeding depression in perennials are rather scarce, and rarely span over several years, let alone individuals' entire lives (Husband & Schemske, 1996). Consequently, the quantities measured in such studies are not likely to depict inbreeding depression in its classic definition, that is the lifetime fitness decrease in selfed individuals as compared to outcrossed ones (Charlesworth & Charlesworth, 1987), but rather

its magnitude at a given timestep or stage. Therefore, estimates in short-lived and long-lived species may not be readily comparable, and higher inbreeding depression levels reported in perennials could in part reflect measurement biases (Angeloni et al., 2011). Lifespan may also interact with the mutation load in nontrivial ways. On the one hand, longer lifespan may increase selection against deleterious mutations, because of the increased number of opportunities for selection to occur, thereby leading to lower levels of inbreeding depression through better purging (Morgan, 2001). On the other hand, more long-lived species may endure significantly more mitotic mutations throughout their lives owing to their overall larger stature, which could result in an increase in inbreeding depression as plants do not have a separated germline (Scofield & Schultz, 2006). Furthermore, based on theoretical arguments (Abu Awad, Billiard, & Tran, 2016), it was proposed that perennials may experience reduced purging of mutations affecting juvenile fitness, as some of these mutations would remain as neutral mutations among adults, and be recurrently reintroduced in the population through reproduction.

Importantly, these various predictions stem from vastly different approaches, and remain mostly verbal. In particular, the rare models that considered populations with overlapping generations vary considerably in terms of the life stage they assume deleterious mutations to affect. Indeed, whereas some authors assume they only act on juvenile survival or gamete production (Abu Awad et al., 2016), others assume they affect their bearers' survival throughout their lives (Morgan, 2001). As for somatic mutations (Scofield & Schultz, 2006), although it is clear such mutations occur and can be inherited in large-statured plants such as oaks (*Quercus robur*, Plomion et al., 2018), the population-level consequences of this mechanism were never investigated theoretically. Besides, no study has yet considered the interaction between separate loads affecting different life stages, although these situations are likely to occur since differential purging has been reported between life stages (Angeloni et al., 2011). Finally, every theoretical study so far has assumed individuals' fecundities and survivorship to not depend upon their age, which is a strong simplifying assumption (Franco & Silvertown, 1996; Petit & Hampe, 2006). In the future, significant insight for the evolution of mating systems and life histories is to be gained through investigations of the dynamics of mutation loads in perennials.

ACKNOWLEDGMENTS

This preprint has been reviewed and recommended by Peer Community In Evolutionary Biology (https://dx.doi.org/10.24072/pci.evolbiol.100070). We thank Thomas Bataillon, along with two anonymous reviewers, for taking the time to consider our manuscript, and ultimately recommending our work. Their comments greatly improved the quality of the manuscript. We also thank Sylvain Glémin, Florence Débarre, Diala Abu Awad, Vincent Castric, Louis Mahé and Denis Roze for helpful discussions and comments on the manuscript. This work was funded by the European Research Council (NOVEL project, grant #648321). The authors also thank the Région Hauts-de-France, and the Ministère de l'Enseignement Supérieur et de la Recherche (CPER Climibio), and the European Fund for Regional Economic Development for their financial support.

ORCID

Thomas Lesaffre https://orcid.org/0000-0002-0815-259X

Sylvain Billiard https://orcid.org/0000-0003-2135-3246

REFERENCES

Abu Awad, D., Billiard, S., & Tran, V. C. (2016). Perenniality induces high inbreeding depression in self-fertilising species. *Theoretical Population Biology*, *112*, 43–51.

Angeloni, F., Ouborg, N., & Leimu, R. (2011). Meta-analysis on the association of population size and life history with inbreeding depression in plants. *Biological Conservation*, *144*, 35–43.

Auld, J., & Rubio de Casas, R. (2012). The correlated evolution of dispersal and matingsystem traits. *Evolutionary Biology*, *40*, 185–193.

Barrett, S. C., & Harder, L. D. (1996). The comparative biology of pollination and mating in flowering plants. *Philosophical Transactions of the Royal Society of London B*, *351*, 1271–1280.

Barrett, S., & Harder, L. (2017). The ecology of mating and its evolutionary consequences in seed plants. *Annual Review of Ecology, Evolution, and Systematics*, *48*, 135–157.

Barton, N., & Turelli, M. (1991). Natural and sexual selection on many loci. *Genetics*, *127*, 229–255.

Bell, G. (1980). The costs of reproduction and their consequences. *The American Naturalist*, *116*, 45–76.

Brunet, J. (1992). Sex allocation in hermaphroditic plants. *Trends in Ecology & Evolution*, *7*, 79–84.

Charlesworth, D., & Charlesworth, B. (1987). Inbreeding depression and its evolutionary consequences. *Annual Review of Ecology and Systematics*, *18*, 237–268.

Charlesworth, D., Morgan, M., & Charlesworth, B. (1990). Inbreeding depression, genetic load, and the evolution of outcrossing rates in a multilocus system with no linkage. *Evolution*, *44*, 1469–1489.

Charlesworth, D., & Willis, J. (2009). The genetics of inbreeding depression. *Nature Reviews Genetics*, *10*, 783–796.

Cichon, M. (1997). Evolution of longevity through optimal resource allocation. *Proceedings of the Royal Society of London B*, *264*, 1383–1388.

Clauss, M., & Mitchell-Olds, T. (2006). Population genetic structure of *arabidopsis lyrata* in europe. *Molecular Ecology*, *15*, 2753–2766.

Duminil, J., Hardy, O., & Petit, R. (2009). Plant traits correlated with generation time directly affect inbreeding depression and mating system and indirectly genetic structure. *BMC Evolutionary Biology*, *9*, 177.

Duputié, A., & Massol, F. (2013). An empiricist's guide to theoretical predictions on the evolution of dispersal. *Interface Focus*, *40*, 20130028.

Eckert, C., Samis, K., & Dart, S. (2006). Reproductive assurance and the evolution of uniparental reproduction in flowering plants. In L. Harder & S. Barrett (Eds.), *Ecology and Evolution of Flowers* (pp. 183–203). Oxford, UK: Oxford University Press.

Ehrlén, J., & Lehtilä, K. (2002). How perennial are perennial plants? *Oikos*, *98*, 1070–1072. https://doi.org/10.1034/j.1600-0706.2002.980212.x

Fisher, R. (1941). Average excess and average effect of a gene substitution. *Annals of Human Genetics*, *11*, 53–63.

Franco, M., & Silvertown, J. (1996). Life-history variation in plants: An exploration of the fast-slow continuum hypothesis. *Philosophical Transactions of the Royal Society of London B*, *351*, 1341–1348.

Goodwillie, C., Kalisz, S., & Eckert, C. (2005). The evolutionary enigma of mixed mating systems in plants: Occurence, theoretical explanations,

and empirical evidence. *Annual Review of Ecology, Evolution, and Systematics*, *36*, 47–79.
Grossenbacher, D., Briscoe Runquist, R., Goldberg, E., & Brandvain, Y. (2015). Geographic range size is predicted by plant mating system. *Ecology Letters*, *18*, 706–713.
Husband, B., & Schemske, D. W. (1996). Evolution of the magnitude and timing of inbreeding depression in plants. *Evolution*, *50*, 54–70.
Johnson, M., Smith, S., & Rausher, M. (2009). Effects of plant sex on range distributions and allocation to reproduction. *New Phytologist*, *186*, 769–779.
Kirkpatrick, M., Johnson, T., & Barton, N. (2002). General models of multilocus evolution. *Genetics*, *161*, 1727–1750.
Kozłowski, J. (1993). Measuring fitness in life-history studies. *Trends in Ecology & Evolution*, *8*, 84–85.
Lande, R., & Schemske, D. (1985). The evolution of self-fertilization and inbreeding depression in plants. I. Genetic Models. *Evolution*, *39*, 24–40.
Lloyd, D. (1992). Self- and cross-fertilization in plants. II. the selection of self-fertilization. *International Journal of Plant Sciences*, *153*, 370–380.
Maynard Smith, J., & Price, G. (1973). The logic of animal conflict. *Nature*, *246*, 15–18.
Morgan, M. (2001). Consequences of life history for inbreeding depression and mating system evolution in plants. *Proceedings of the Royal Society of London B*, *268*, 1817–1824.
Morgan, M., Schoen, D., & Bataillon, T. (1997). The evolution of self-fertilization in perennials. *The American Naturalist*, *150*, 618–638.
Munoz, F., Violle, C., & Cheptou, P.-O. (2016). CSR ecological strategies and plant mating systems: Outcrossing increases with competitiveness but stress-tolerance is related to mixed mating. *Oikos*, *125*, 1296–1303.
Petit, R., & Hampe, A. (2006). Some evolutionary consequences of being a tree. *Annual Review of Ecology, Evolution, and Systematics*, *37*, 187–214.
Plomion, C., Aury, J.-M., Amselem, J., Leroy, T., Murat, F., Duplessis, S., … Salse, J. (2018). Oak genome reveals facets of long lifespan. *Nature Plants*, *4*, 440–452.
Schaffer, W. (1974). Optimal reproductive effort in fluctuating environments. *The American Naturalist*, *108*, 783–790.
Scofield, D., & Schultz, S. (2006). Mitosis, stature and evolution of plant mating systems: low and high plants. *Proceedings of the Royal Society of London B*, *273*, 275–282.
Sicard, A., & Lenhard, M. (2011). The selfing syndrome: A model for studying the genetic and evolutionary basis of morphological adaptation in plants. *Annals of Botany*, *107*, 1433–1443.
Silvertown, J., Franco, M., & Perez-Ishiwara, R. (2001). Evolution of senescence in iteroparous perennial plants. *Evolutionary Ecology Research*, *3*, 393–412.
Stearns, S. (1992). *The evolution of life histories*. Oxford, UK: Oxford University Press.
Stebbins, G. (1950). *Variation and evolution in plants*. New York, NY: Columbia University Press.
Zhang, D. Y. (2000). Resource allocation and the evolution of self-fertilization in plants. *The American Naturalist*, *155*, 187–199.

How to cite this article: Lesaffre T, Billiard S. The joint evolution of lifespan and self-fertilization. *J Evol Biol*. 2020;33:41–56. https://doi.org/10.1111/jeb.13543

APPENDIX 1

Proportion of selfed individuals and lifetime inbreeding depression

In this section, we detail the derivation of the equilibrium proportion of selfed individuals in the population and show that its decrease when longevity increases is attributable to stronger selection against inbred individuals.

Proportion of selfed individuals

Following reproduction, adult individuals survive with probability $S_o(e)$ if they were produced by outcrossing, and $(1-\delta_a)\,S_o(e)$ if they were selfed. Therefore, denoting Θ the proportion of selfed individuals in the population at a given timestep, the proportion of adult individuals surviving between two mating events ($\overline{S}$) is given by

$$\overline{S} = S_0(e)[\Theta(1-\delta_a)+1-\Theta], \tag{A1}$$

and the proportion of selfed individuals among them, Θ^s is

$$\Theta^s = \frac{\Theta\,(1-\delta_a)}{\Theta\,(1-\delta_a)+1-\Theta} \tag{A2}$$

Among the juveniles settling in the population at a given mating event, the proportion of selfed individuals (Θ^j) given the selfing rate α can be expressed as

$$\Theta^j = \frac{\alpha(1-\delta_j)}{\alpha(1-\delta_j)+1-\alpha}. \tag{A3}$$

Hence, the proportion of selfed individuals at the next timestep (Θ') is

$$\Theta' = \overline{S}\,\Theta^s + \left(1-\overline{S}\right)\Theta^j, \tag{A4}$$

and, solving $\Delta\Theta = \Theta' - \Theta = 0$, we obtain the equilibrium proportion of selfed individuals in the population, Θ^*:

$$\Theta^* = \frac{\alpha\,(1-\delta_j)\,(1-S_o(e))}{S_o(e)\,(1-\alpha)\,\delta_a + (1-S_o(e))\,(1-\alpha\delta_j)}. \tag{A5}$$

Lifetime inbreeding depression

In our model, inbreeding depression only affects survival, in juveniles and in adults. Thus, the decrease in fitness selfed individuals suffer from in comparison with the outcrossed throughout their lives, and not only between two mating events (*i.e.* lifetime inbreeding depression) can be expressed in terms of life expectancies. The life expectancies for selfed ($\mathcal{L}_s$) and outcrossed ($\mathcal{L}_0$) individuals, respectively, are given by

$$\mathcal{L}_s = \frac{1-\delta_j}{1-S_o(e)\,(1-\delta_a)} \text{ and, } \mathcal{L}_o = \frac{1}{1-S_o(e)}, \tag{A6}$$

and lifetime inbreeding depression can be expsressed as

$$\overline{\delta}=1-\frac{\mathcal{L}_s}{\mathcal{L}_o}=1-\frac{\left(1-\mathcal{S}_o(e)\right)\left(1-\delta_j\right)}{1-\mathcal{S}_o(e)\left(1-\delta_a\right)}. \tag{A7}$$

Equation (A7) is an increasing function of $\mathcal{S}_o(e)$; that is, selection against selfed individuals grows stronger as longevity increases. This is due to the repeated effect of adult inbreeding depression on post-bsssreeding survival. In the annual case ($\mathcal{S}_o(e)=0$), Equation (A7) reduces to $\overline{\delta}=\delta_j$. Interestingly, Equation (A7) relates to Θ^* (Equation A5) in the following way:

$$\Theta^*=\frac{\alpha\left(1-\overline{\delta}\right)}{\alpha\left(1-\overline{\delta}\right)+1-\alpha}. \tag{A8}$$

Therefore, the equilibrium proportion of selfed individuals in the population decreases when generations overlap (i.e. $\mathcal{S}_o(e)$) increases, due to stronger selection against selfed individuals owing to the repeated effect of adult inbreeding depression on adult survival. \ nolinenumbers

APPENDIX 2

Recursions for the evolution of reproductive effort in a partially selfing population

In this section, we detail the mathematical derivations leading to the ESS reproductive effort expression we obtain. The selfing rate is fixed, and reproductive effort is controlled by a single modifier locus. To describe the changes happening in the population between two timesteps, we use the theoretical framework first described by Barton and Turelli (1991) and generalized by Kirkpatrick et al. (2002). We follow the variation of three variables, namely the frequency (p_m) of the mutant allele, which is assumed to be a rare with a small effect (ε) on its bearer's reproductive effort, the excess in homozygosity at the modifier locus ($D_{m,m}$) and the proportion of selfed individuals in the population (Θ).

We define the indicator variables X_m and $\dot{X}_m$, corresponding to the two allelic positions of the modifier locus located on the paternally and maternally inherited chromosomes, respectively. These variables can take two values, 1 if the mutant allele (m) is present at the considered position, and 0 otherwise. Since there is no maternal or paternal effect on the expression of the alleles at the modifier, we have

$$\mathbb{E}[X_m]=\mathbb{E}[\dot{X}_m]=p_m. \tag{A9}$$

Using these indicator variables, we may define the centred variables ζ_m and $\dot{\zeta}_m$ as

$$\zeta_m=X_m-p_m \text{ and, } \dot{\zeta}_m=\dot{X}_m-p_m \text{ so that } \mathbb{E}\left[\zeta_m\right]=\mathbb{E}\left[\dot{\zeta}_m\right]=0. \tag{10}$$

These variables allow us to define the excess in homozygotes in the population at the modifier as compared to the panmictic expectation ($D_{m,m}$) as

$$D_{m,m}=\mathbb{E}[\zeta_{m,m}]=\mathbb{E}[X_m\dot{X}_m]-p_m^2, \tag{A11}$$

where $\zeta_{m,m}=\zeta_m\times\dot{\zeta}_m$.

Reproduction

Using indicator variables, the reproductive effort of a given individual, e, can be expressed as follows

$$e=e_0+\frac{\varepsilon}{2}\left(X_m+\dot{X}_m\right)=e_0+\varepsilon\, p_m+\frac{\varepsilon}{2}\left(\zeta_m+\dot{\zeta}_m\right). \tag{A12}$$

The average reproductive effort is thus

$$\overline{e}=\mathbb{E}[e]=e_0+\varepsilon\, p_m, \tag{A13}$$

which yields, to leading order in ϵ, the relative contribution of a given individual to reproduction ($\tilde{e}$) during a given mating event

$$\tilde{e}=\frac{e}{\overline{e}}\approx 1+\hat{\varepsilon}_j\left(\zeta_m+\dot{\zeta}_m\right), \text{with } \hat{\varepsilon}_j=\frac{\varepsilon}{2e_0}. \tag{A14}$$

Frequency of the mutant

Using Equation (A14), the frequency of the mutant allele (p^j_m) among juveniles following reproduction is

$$p^j_m=\mathbb{E}\left[\tilde{e}\,\frac{X_m+\dot{X}_m}{2}\right]=\frac{1}{2}\mathbb{E}\left[\tilde{e}\,\left(\zeta_m+\dot{\zeta}_m+2p_m\right)\right]=p_m+\hat{\varepsilon}_j(p_mq_m+D_{m,m}), \tag{A15}$$

with $q_m = 1 - p_m$, and the change in frequency of the mutant among juveniles is therefore

$$\Delta^j p_m=\hat{\varepsilon}_j\left(p_mq_m+D_{m,m}\right). \tag{A16}$$

Excess in homozygotes

The change in homozygosity at the modifier in juveniles can be divided into two phases, selection and syngamy. The excess in homozygotes at the modifier following selection, $D^{j\cdot}_{m,m}$, is given by

$$D^{j\cdot}_{m,m}=\mathbb{E}\left[\left(X^j_m-p^j_m\right)\left(\dot{X}^j_m-p^j_m\right)\right], \tag{A17}$$

where X^j_m and $\dot{X}^j_m$ are indicator variables defined among juveniles following selection (so that $\mathbb{E}\left[X^j_m\right]=\mathbb{E}\left[\dot{X}^j_m\right]=p^j_m$). Nothing that $p^j_m=p_m+\Delta^j p_m$, and expressing Equation (A17) in terms ssof ζ-variables, we have

$$D^{j\cdot}_{m,m}=\mathbb{E}\left[\tilde{e}\,\zeta_{m,m}-\Delta^j p_m\left(\tilde{e}\,\zeta_m\right)-\Delta^j p_m\left(\tilde{e}\,\dot{\zeta}_m\right)+\left(\Delta^j p_m\right)^2\right]=\mathbb{E}\left[\tilde{e}\,\zeta_{m,m}\right]+\left(\Delta^j p_m\right)^2\approx D_{m,m}\left(1+2\hat{\varepsilon}_j\right) \tag{A18}$$

to leading order in ϵ and assuming p_m is small. During syngamy, homozygosity is generated by inbreeding. In our model, we assumed partial selfing. Therefore, the excess in homozygotes among juveniles, after accounting for juvenile inbreeding depression, is given by

$$D^j_{m,m}=\Theta^j\mathbb{E}\left[\frac{1}{4}\zeta^{j2}_m+\frac{1}{2}\zeta^j_{m,m}+\frac{1}{4}\dot{\zeta}^{j2}_m\right]=\frac{\Theta^j}{2}\left(D^{j\cdot}_{m,m}+p^j_mq^j_m\right)\approx\frac{\Theta^j}{2}\left(D_{m,m}\left(1+3\hat{\varepsilon}_j\right)+p_m\left(1+\hat{\varepsilon}_j\right)\right), \tag{A19}$$

where $\zeta_m^j = X_m^j - p_m^j$ (resp. $\dot{\zeta}_m^j = \dot{X}_m^j - p_m^j$) is the centred variable defined for the paternally (resp. maternally) inherited chromosome among juveniles following selection, that is taking the frequency of the mutant among juveniles following selection (p_m^j) as reference value (Kirkpatrick et al., 2002).

Survival

We denote by $\mathcal{S}$ the probability of survival osf a parent taken at random in the parental population. Using our indicator variables, and given the proportion Θ of selfed individuals in the population, the probability of survival is

$$\mathcal{S} = \Theta\left(1-\delta_a\right)S_o(e) + (1-\Theta)S_o(e) = S\left[1-\left(e_0 + \frac{\varepsilon}{2}\left(X_m + \dot{X}_m\right)\right)^x\right]\left(\Theta\left(1-\delta_a\right)+1-\Theta\right). \tag{A20}$$

Substituting indicator variables with ζ-variables and assuming that ε and p_m are small, we obtain, to leading order,

$$\mathcal{S} = \mathcal{S}\left(1-\Theta\,\delta_a\right)\left[1-e_0^x - \frac{x\,\varepsilon\, e_0^{x-1}}{2}\left(\zeta_m + \dot{\zeta}_m\right)\right] + o\left(\varepsilon^2\right). \tag{A21}$$

Thus, the mean survival probability is

$$\overline{\mathcal{S}} = \mathbb{E}\left[\mathcal{S}\right] \approx \mathcal{S}\left(1-\Theta\,\delta_a\right)\left[1-e_0^x\right], \tag{A22}$$

and the relative survival probability ($\widetilde{\mathcal{S}}$) simplifies to

$$\widetilde{\mathcal{S}} = \frac{\mathcal{S}}{\overline{\mathcal{S}}} \approx 1 - \hat{\varepsilon}_s\left(\zeta_m + \dot{\zeta}_m\right), \text{with } \hat{\varepsilon}_s = \frac{x\,\varepsilon\, e_0^{x-1}}{2\left(1-e_0^x\right)}. \tag{A23}$$

Frequency of the mutant

Using Equation (A14), the frequency of the mutant (p_m^j) among the surviving parents is

$$p_m^s = \mathbb{E}\left[\widetilde{\mathcal{S}}\,\frac{X_m + X_m}{2}\right] \approx p_m - \hat{\varepsilon}_s\left(p_m q_m + D_{m,m}\right), \tag{A24}$$

and the variation in frequency of the mutant owing to selection among parents is therefore given bys

$$\Delta^s p_m = -\hat{\varepsilon}_s\left(p_m q_m + D_{m,m}\right) \tag{A25}$$

Excess in homozygotes

Since only selection acts at this stage, the excess in homozygotes at the modifier locus among the parents ($D_{m,m}^s$) is given by

$$D_{m,m}^s = \mathbb{E}\left[\left(X_m^s - p_m^s\right)\left(\dot{X}_m^s - p_m^s\right)\right]. \tag{A26}$$

Noting that $p_m^s = p_m + \Delta^s p_m$, and expanding Equation (A26), we have

$$\begin{aligned} D_{m,m}^s &= \mathbb{E}\left[\widetilde{\mathcal{S}}\,\zeta_{m,m} - \Delta^s p_m\left(\mathcal{S}\,\zeta_m\right) - \Delta^s p_m\left(\widetilde{\mathcal{S}}\,\dot{\zeta}_m\right) + \left(\Delta^s p_m\right)^2\right] \\ &= \mathbb{E}\left[\widetilde{\mathcal{S}}\,\zeta_{m,m}\right] + \left(\Delta^s p_m\right)^2 \approx D_{m,m}\left(1-2\hat{\varepsilon}_s\right). \end{aligned} \tag{A27}$$

Next timestep

From our previously derived expressions, the frequency of the mutant (p'_m), the excess in homozygotes ($D'_{m,m}$) and the proportion of selfed individuals in the population (Θ') in the next timestep is

$$\begin{cases} p'_m = \overline{\mathcal{S}}\,p_m^s + (1-\overline{\mathcal{S}})p_m^j \\ D'_{m,m} = \overline{\mathcal{S}}\,D_{m,m}^s + (1-\overline{\mathcal{S}})D_{m,m}^j \\ \Theta' = \overline{\mathcal{S}}\,\Theta^s + (1-\overline{\mathcal{S}})\Theta^j. \end{cases}$$

In order to determine the *evolutionary stable strategy* (ESS, Maynard Smith & Price, 1973) for the population given the parameters of the model, one has to determine the value of e_0 for which no mutant allele (m) may invade the population and replace the resident (M). To do so, we will consider the change in frequency of the mutant allele over one timestep (Δp_m), which can be expressed as

$$\Delta p_m \approx \left[\left(1-\overline{\mathcal{S}}\right)\hat{\varepsilon}_j - \overline{\mathcal{S}}\,\hat{\varepsilon}_s\right]\left(p_m q_m + D_{m,m}\right) = \hat{\varepsilon}_j\left(p_m q_m + D_{m,m}\right)V, \tag{A28}$$

with $V = 1-\mathcal{S}\left(1-\Theta\delta_a\right)\left(1+\left(x-1\right)e_0^x\right)$.

Separation of timescales approximation

When selection is weak (ϵ is small), we may assume that the excess in homozygotes in the population reaches a value close to equilibrium much faster than the allelic frequencies. It is obtained by solving

$$D'_{m,m} - D_{m,m} = 0$$

for $D_{m,m}$. Thus, assuming the mutant is rare (p_m is of order ϵ) we have

$$D_{m,m}^* = \frac{\Theta^j p_m}{2-\Theta^j}\left(1 - \frac{4\overline{\mathcal{S}}\hat{\varepsilon}_s - 3\Theta^j\left(1-\overline{\mathcal{S}}\right)\hat{\varepsilon}_j}{\left(1-\overline{\mathcal{S}}\right)\left(2-\Theta^j\right)}\right) \approx \frac{\Theta^j}{2-\Theta^j}p_m. \tag{A29}$$

Since we have $D_{m,m}^* \geq 0$ (Equation(A29)), the first term in Equation (A28) is always positive, and only V matters for the determination of the equilibrium.

Evolutionarily Stable Reproductive Effort

Injecting Equation (A5) into Equation (A28) and solving $\Delta\, p_m = 0$ for e_0 yield the following ESS for the reproductive effort (e^*):

$$e^* = \left[\frac{\mathcal{B} + \sqrt{(-\mathcal{B})^2 + 4\left(1-\mathcal{S}\right)\left(x-1\right)\left(1-\delta_a\right)\left[1-\delta_a\left(1-\mathcal{S}\right)\right]\left(1-\alpha\delta_j\right)^2}}{2\mathcal{S}\left(x-1\right)\left(1-\delta_a\right)\left(1-\alpha\delta_j\right)}\right]^{\frac{1}{x}} \tag{A30}$$

with $\mathcal{B} = \left(1-\mathcal{S}\right)\left(2-x\right)\left(1-\alpha\delta_j\right) + \delta_a\left[\alpha x\left(1-\delta_j\right) - \left[1-\mathcal{S}\left(2-x\right)\right]\left(1-\alpha\delta_j\right)\right]$.

APPENDIX 3

Recursions for the evolution of self-fertilization in a perennial population

In this section, we detail the mathematical derivations for the evolution of self-fertilization in a perennial population. Individuals reproduce by self-fertilization at a rate given by their genotype at a single biallelic modifier locus and by random mating otherwise. At the modifier, alleles *M* and *m* are assumed to be codominant, and code for selfing rates α_0 and $\alpha_0 + a$, respectively. Allele *m* is assumed to be a rare mutant with a weak effect (i.e. *a* is small). The theoretical framework we use is the same as in Appendix 2.

Reproduction

Let us split the population into two groups: the selfed and the outcrossed individuals. The frequency of allele *m* in each of these groups is denoted $p_{m,s}$ and $p_{m,o}$, respectively, so that its frequency in the whole population is given by

$$p_m = \Theta p_{m,s} + (1-\Theta) p_{m,o} \tag{A31}$$

where Θ is the proportion of selfed individuals in the population.

Frequency of the mutant among the selfed

The contribution of a given parent to the production of selfed offspring, α, is

$$\alpha = \alpha_0 + 2\times\frac{a}{2}\left(X_m + \dot{X}_m\right) = \alpha_0 + a\left(X_m + \dot{X}_m\right), \tag{A32}$$

owing to the fact that increasing its selfing rate by $\frac{a}{2}$ induces a twofold transmission advantage in comparison with others (Fisher, 1941). Injecting ζ-variables into Equation (A32), we obtain the following expression for the relative contribution of a given parent to the production of offspring by self-fertilization:

$$\tilde{\alpha} = \frac{\alpha}{\overline{\alpha}} = \frac{\alpha_0 + ap_m + a\left(\zeta_m + \dot{\zeta}_m\right)}{\alpha_0 + ap_m} = 1 + \frac{a}{\alpha_0 + ap_m}\left(\zeta_m + \dot{\zeta}_m\right). \tag{A33}$$

Thus, the frequency of allele *m* among the selfed offspring ($p^j_{m,s}$) is given by

$$p^j_{m,s} = \mathbb{E}\left[\tilde{\alpha}\frac{X_m + X_m}{2}\right] = p_m + \frac{a}{\alpha_0 + ap_m}(p_m(1-p_m) + D_{m,m}). \tag{A34}$$

Frequency of the mutant among the outcrossed

The contribution of a given parent to the production of outcrossed offspring, *o*, is

$$o = 1 - \alpha_0 - \frac{a}{2}\left(X_m + \dot{X}_m\right), \tag{A35}$$

which yields the following expression for its relative contribution ($\tilde{O}$):

$$\tilde{o} = 1 - \frac{a}{2(1-\alpha_0 - ap_m)}\left(\zeta_m + \dot{\zeta}_m\right). \tag{A36}$$

Hence, the frequency of allele *sm* among the outcrossed offspring ($p^j_{m,o}$) is

$$p^j_{m,o} = \mathbb{E}\left[\tilde{o}\frac{X_m + \dot{X}_m}{2}\right] = p_m - \frac{a}{2(1-\alpha_0 - ap_m)}(p_m(1-p_m) + D_{m,m}). \tag{A37}$$

Excess in homozygotes

Considering the whole offspring pool, we may neglect the effect of the mutant on the selfing rate. Thus, we may express the excess in homozygotes at the modifier among juveniles as

$$D^j_{m,m} \approx \frac{\Theta^j}{2}(p_m(1-p_m) + D_{m,m}) \tag{A38}$$

where Θ^j is the proportion of selfed individuals among the juveniles settling in the population (Equation (A3)).

Survival

Among the parents, allelic frequencies among the selfed and among the outcrossed do not vary, because there is no direct selection acting on the modifier among them. Therefore,

$$p^s_{m,o} = p_{m,o} \text{ and } p^s_{m,s} = p_{m,s}. \tag{A39}$$

Moreover, we have

$$D^s_{m,m} = D_{m,m}. \tag{A40}$$

Selfed individuals are, however, counterselected due to adult inbreeding depression. Thus, their proportion varies according to Equation (A2).

Next timestep

In the next timestep, assuming the proportion of selfed individuals remains close to its equilibrium value (Θ^*, Equation (A5)), the frequency of the mutant (p'_m) and the excess in homozygotes ($D'_{m,m}$) are given by

$$\begin{cases} p'_m = \overline{S}\, p^s_m + (1-\overline{S})\, p^j_m \\ D'_{m,m} = \overline{S}\, D^s_{m,m} + (1-\overline{S}) D^j_{m,m} \end{cases}, \tag{A41}$$

where $\overline{S} = S_o(e)\left[\Theta^*(1-\delta_a) + 1 - \Theta^*\right]$ is the proportion of parents surviving until the next timestep. Using these recursions, we may express the change in allelic frequencies at the modifier (Δp_m) as

$$\Delta p_m = -\frac{a(1-\alpha_0\delta_j) D_{m,m}(1-S_o(e))}{2\left[S_o(e)(1-\alpha_0)\delta_a + (1-S_o(e))(1-\alpha_0\delta_j)\right]^2}\times T \tag{A42}$$

with $T = \left[1 - S_o(e)(1+\delta_a)\right]\left[S_o(e)\delta_a - (1-S_o(e))(1-2\delta_j)\right]$.

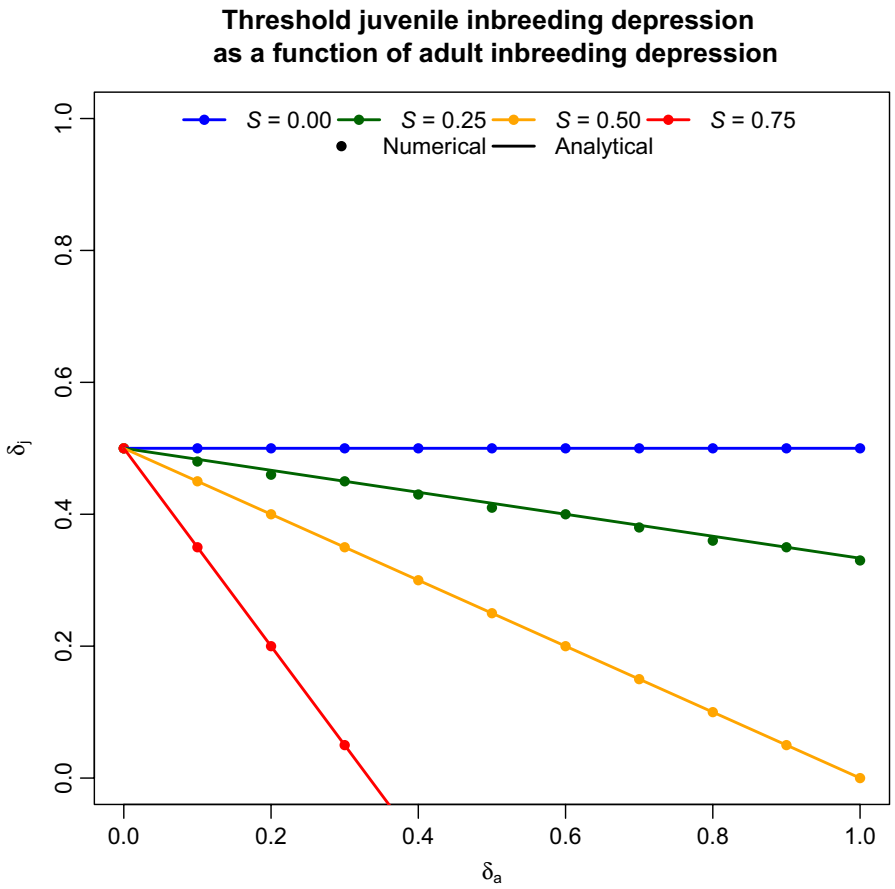

FIGURE A1 Numerical analysis results for the evolution of self-fertilization. Threshold juvenile ID is presented as a function of adult ID, for various survival probabilities ($S_o(e) = 0\,0.25\,;0.5\,;0.75$)

Separation of timescales approximations

Assuming homozygosity equilibrates quickly in comparison with allelic frequencies at the modifier, we obtain a quasi-equilibrium expression (Kirkpatrick et al., 2002) for the excess in homozygotes at the modifier ($D^*_{m,m}$) by solving $D'_{m,m} - D_{m,m} = 0$,

$$D^*_{m,m} = p_m\left(1-p_m\right)\frac{\alpha_0\left(1-\delta_j\right)}{2-\alpha_0\left(1+\delta_j\right)} \geq 0. \tag{A43}$$

Using Equation (A43), one can see that the first term in Equation (A42) is always negative, and only T matters for the determination of the equilibria. Because T does not depend on α_{0s}, there is only two situations: either $T > 0$ and full outcrossing is maintained ($\alpha^* = 0$), or $T < 0$ and full selfing is favoured ($\alpha^* = 1$). Hence, by solving $T < 0$, we obtain the following threshold for the evolution of self-fertilization:

$$\delta_j < \frac{1-\left(1+\delta_a\right)S_o(e)}{2\left(1-S_o(e)\right)} \tag{A44}$$

APPENDIX 4
Numerical analyses

In this section, numerical analysis results are presented along with our analytical predictions.

For the evolution of self-fertilization, the analysis was conducted starting from $\alpha_0 = 0$ and introducing mutants increasing the selfing rate. Good agreement was found between our analytical predictions and numerical analyses. Figure A1 shows some inbreeding depression threshold values obtained for various longevity situations. Either full selfing or strict outcrossing always evolved.

For the evolution of reproductive effort, two types of analyses were conducted: starting from $e_0 = 0$ and introducing mutants increasing reproductive effort (upwards analysis, presented in orange), and starting from $e_0 = 1$ and introducing mutants decreasing reproductive effort (downwards analysis, presented in blue). Part of the results are presented in Figure A2. Discrepancies between upwards and downwards analyses were observed in situations involving high adult inbreeding depression and low extrinsic mortality for intermediate selfing rates (Figure A2b). Such situations are never reached in the context of the joint evolution of lifespan and selfing, because inbreeding depression is too high for selfing to evolve and intermediate selfing rates are never evolutionarily stable. Besides, good agreement was found between analytical predictions and numerical analyses in most cases, especially for selfing rates close to 0 or 1 (Figure A2b), which are the states that matter for the joint evolution of lifespan and selfing. Therefore, we considered our analytical prediction to satisfactorily agree with numerical analyses.

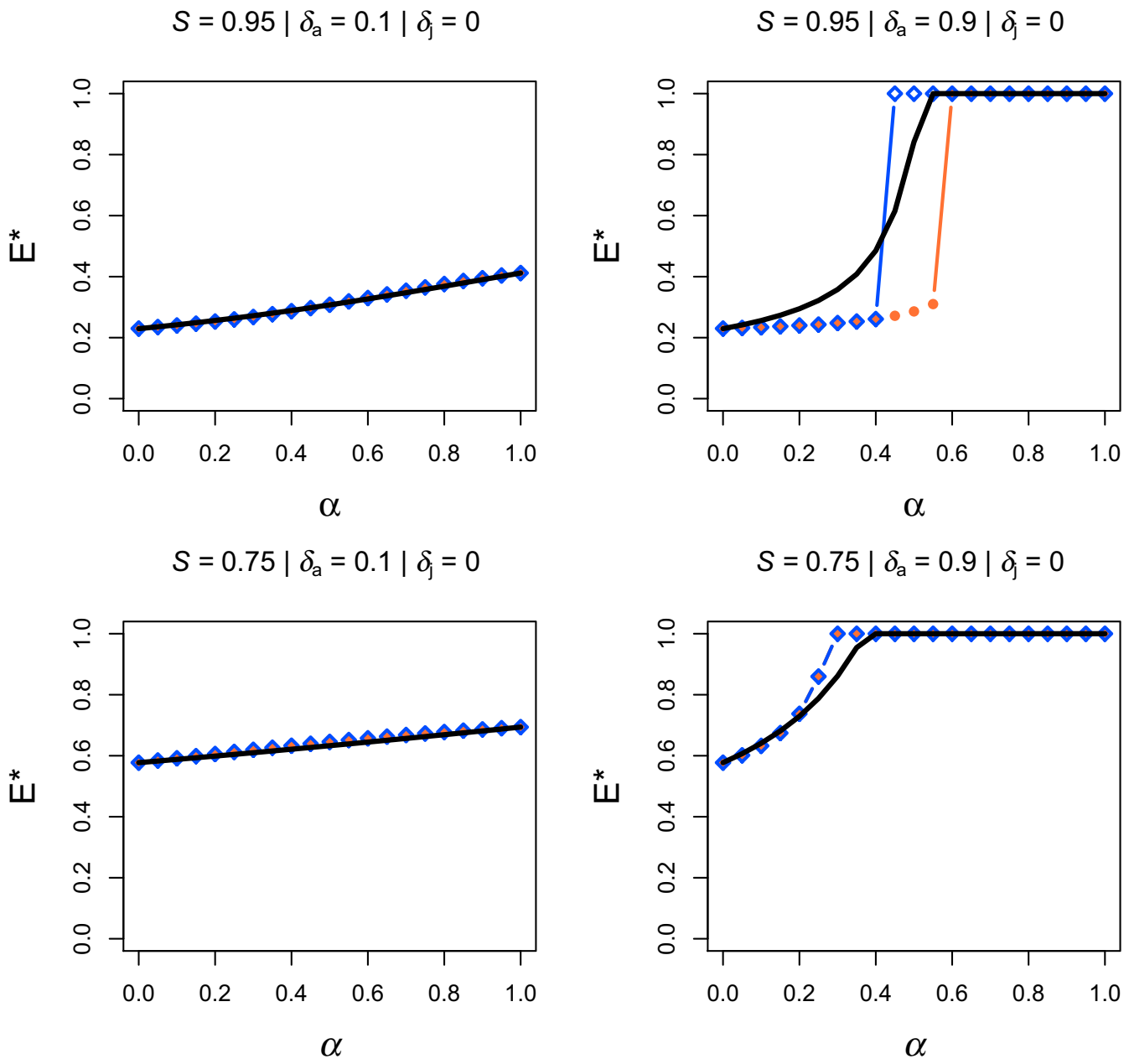

FIGURE A2 Numerical analysis results for the evolution of reproductive effort. ESS reproductive effort is presented as a function of the selfing rate, in low (S = 0.95, a and b) and mild (S = 0.75, c and d) extrinsic mortality conditions, with low (Δ_a = 0.1, a and c) and high (Δ_a = 0.9, b and d) adult inbreeding depression. Numerical analyses are presented in orange (*upwards*) and blue (*downwards*). Analytical predictions are presented in black

APPENDIX 5

INbreeding depression affecting fecundity

In this appendix, we investigate the effect of inbreeding depression affecting fecundity, that is the contribution of selfed individuals to the gamete pool, on the coevolutionary dynamics. In Figure A3, we show the equilibria reached by simulations with different starting points, when setting $\delta_j = 0$ and $\delta_f = 0.1; 0.4; 0.7$ with δ_a = 0.4 and S = 0.8. Solid lines represent the analytical expectations in the absence of inbreeding depression on fecundity ($\delta_f = 0$), and with $\delta_j = 0.1; 0.4; 0.7$.

Simulations results are well predicted by these analytical expectations, which shows that increasing δ_f or δ_j has the same effect: it only reduces the range of conditions under which the full selfing strategy can evolve. The mechanisms underlying these similar behaviours are, however, slightly different. Indeed, whereas in both cases they are caused by a reduction of the participation of selfed individuals to reproduction, δ_j decreases the proportion of selfed individuals in the population. On the other hand, δ_f reduces the contribution of selfed adults to reproduction by reducing their contribution to the gamete pool.

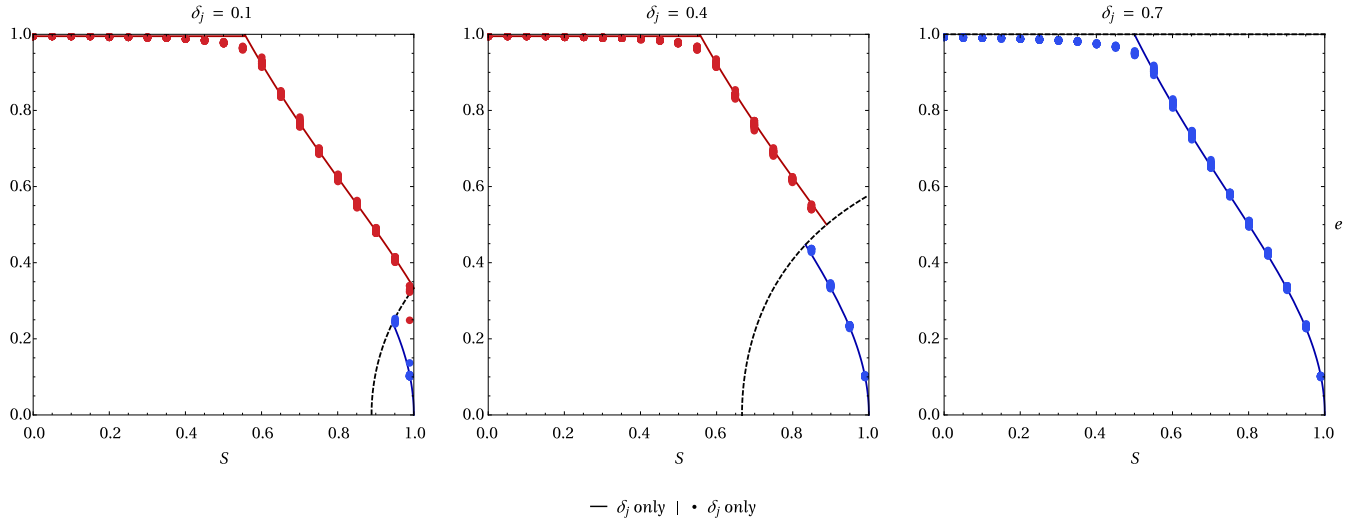

FIGURE A3 All results presented here assume $\Delta_a = 0.1$ and $x = 2$. Y-axis is the ESS reproductive effort (e^*), and X-axis is extrinsic mortality (S). The red line corresponds to the equilibrium reproductive effort when $\alpha = 1$ is reached. The blue line corresponds to the equilibrium reproductive effort when $\alpha = 0$ is reached. The dashed black line represents the threshold lifetime inbreeding depression under which outcrossing can be maintained. Dots depict the results of individual-centred simulations. When red dots mean $\alpha = 1$ is reached, whereas blue dots mean $\alpha = 0$ is reached