Extrinsic mortality and senescence: a guide for the perplexed

de Vries, Charlotte[1,2,*]; Galipaud, Matthias[1]; Kokko, Hanna[1,3,4]

[1] Department of Evolutionary Biology and Environmental Studies, University of Zurich, Zurich, Switzerland

[2] Department of Biological and Environmental Science University of Jyväskylä, Jyväskylä, Finland

[3] Konrad Lorenz Institute of Ethology University of Veterinary Medicine Vienna, Austria

[4] Faculty of Biological and Environmental Sciences University of Helsinki, Helsinki, Finland

*Corresponding author

Correspondence: charlotte.c.devries@jyu.fi

Abstract

Do environments or species traits that lower the mortality of individuals create selection for delaying senescence? Reading the literature creates an impression that mathematically oriented biologists cannot agree on the validity of George Williams' prediction (who claimed 'yes'). The abundance of models and opinions may bewilder those that are new to the field. Here we provide heuristics as well as simple models that outline when the Williams prediction holds, why there is a 'null model' where extrinsic mortality does not matter, and why it is also possible to expect the opposite of William's prediction, where increased extrinsic mortality favours slower senescence. While most existing theory focuses on interpreting differences in selection gradients, we hope to offer intuition by quantifying how much delaying the 'placement' of an offspring into the population reduces its expected contribution to the gene pool of the future. Our first example shows why the null result arises and why the null can stop being valid in models that consider population regulation. Thereafter, a model with 10 different choices for density dependence shows that high extrinsic mortality has the power to favour either slow or fast life histories on the fast-slow continuum. The latter case occurs when increasing density harms juvenile production and/or their survival. An interesting implication, so far untested, is that empirical studies finding support for Williams-like patterns could suggest that density regulation often impacts the production and/or survival of juveniles, as opposed to the survival of older individuals.

Keywords: Senescence, Life-History Evolution, Trade-Offs

Introduction

"It is not the case that additional mortality automatically favours the evolution of senescence."

Caswell and Shyu, 2017

"Reports of the death of extrinsic mortality moulding senescence have been greatly exaggerated."

Jack da Silva, 2018

"Williams' intuition about extrinsic mortality is irrelevant"

Moorad et al. 2020b

The above quotes lay bare a rather odd state of affairs: more than six decades after Williams (1957) presented his argument for the relationship between adult mortality rates and the evolution of senescence, mathematically trained biologists still cannot seem to agree on what patterns theory actually predicts. Williams' seminal work argued that populations experiencing different rates of mortality (as adults) should senesce at different rates (Gaillard & Lemaître 2017). The intuitive message is that if life is bound to be short 'anyway' (due to, e.g., high predation risk), it makes little sense to invest in a robust body able to resist 'wearing out' for a long time (Medawar 1952, Williams 1957).

William's work has since been interpreted to mean that an increase in age-independent extrinsic mortality — typically defined as the result of hazards from the environment which are constant throughout life (Koopman et al. 2015, see Moorad et al. 2020a for definitional issues) — should select for faster senescence (Da Silva 2018, Dańko et al. 2018, André and Rousset 2020). Others have argued against this idea, stating that age-independent extrinsic mortality cannot affect the evolution of senescence (Gadgil and Bossert 1970, Taylor et al. 1974, Abrams 1993, Caswell 2007, Caswell and Shyu 2017, Wensink et al. 2017, Moorad et al. 2019). Recent work, while aiming to clarify, has simultaneously led to a large number of different models and opinions, which as a whole may be confusing to those that are new to the field (André and Rousset 2020, Dańko et al. 2017,2018, and the debate started by Moorad et al. 2019 and continued in Day & Abrams 2020, da Silva 2020, Moorad et al. 2020a,b). Here our aim is to explain what happens in models of senescence in relation to extrinsic mortality, and outline when the prediction made by Williams holds and why it is also possible to state an expectation of it not holding.

In the following, we call, for the sake of conciseness, the prediction that age-independent extrinsic mortality does not impact senescence 'the null result'. The null result can be interpreted to mean that 'Williams was wrong', but it is useful to distinguish the null from an even stronger way for a prediction to disagree with the Williams hypothesis: it is logically possible that higher extrinsic mortality associates with *slower* (not faster) senescence (Abrams 1993). Thus, we have a range of potential results which we, for brevity, call 'Williams' (is right), 'null', and 'anti-Williams'.

The null result is typically explained using selection gradients in an approach that derives the strength of selection for a trait that is assumed to improve a vital rate (e.g. survival), and asks whether selection differs between organisms where age-independent extrinsic mortality rates are *a priori* high or low (Caswell 2007, Caswell & Shyu 2017). The result can be summarized as additional age-independent mortality having, perhaps surprisingly, no effect on selection gradients in the absence of density-dependence, or in the presence of density-dependence that impacts survival of all ages equally (Caswell 2007). An alternative explanation of the null result, as well as deviations from the null-result, can be found in the appendix of Day & Abrams' (2020) where they use growth rate optimization to quantify the effect of an increased extrinsic mortality under different kinds of density-dependence. Here we hope to provide an intuitive explanation for the null result by focusing instead explicitly on the time that a newborn is placed into a population. Delaying the 'placement' of an offspring into the population reduces its expected contribution to the gene pool of the future — but only if a population is growing.

We first show the utility of this approach with a simple example that shows why the null result arises and why the null can stop being valid in models that consider population regulation (as opposed to unlimited exponential growth). This model is intentionally kept simple and analytically tractable, e.g., we assume no tradeoffs between reproduction and survival. We thereafter introduce such tradeoffs by linking the ideas of fast and slow life histories (Stearns 1989, Promislow and Harvey 1990) with Gompertz-Makeham survival curves (Gompertz 1825, Makeham 1860, Missov and Lenart 2013), together with a total of ten different styles of density regulation.

The advantage of a simulation approach is that it allows linking senescence to the 'understudied territory' identified by Moorad et al. (2019): what happens when a population does not stabilize to zero growth but fluctuates, so that there are years (or, more generally, time steps) with increasing and others with decreasing population sizes (see also Caswell & Shyu 2017)? Fluctuations in population abundance due to continually occurring stochastic fluctuations in the vital rates are a common way to model such situations (Tuljapurkar 2013, Caswell & Shyu 2017), but populations might also fluctuate due to events that occur less often

and cause large mortality in a pulsed manner, a scenario that we include. These events may impose age-dependent or stage-dependent mortality. A population may be regulated via these events if they happen more often at high density (e.g. a disease spreads), and the population may then spend much of its time growing towards high density rather than remaining near an equilibrium (in other words, transient dynamics become important). In this case predictions based on selection gradients might not apply (Capdevila et al. 2020), since their calculation requires demographic stability or small stochastic and age-independent fluctuations around a demographic equilibrium (Caswell and Shyu 2017).

Our results provide several examples yielding intuition as to why regulation that operates via fecundity or recruitment can be expected to have a different impact on senescence than regulation that operates via declining survival (across all ages) with increasing population densities. While such results do not overturn previous insights already gained (Abrams 1993, Caswell and Shyu 2017, Wensink et al. 2017, Dańko et al. 2017,2018, and other papers cited above), we hope that our presentation will make the issues more heuristically transparent.

An example free of tradeoffs: why does the null result arise?

Being able to fly is often quoted as an example of reduced extrinsic mortality (Austad and Fischer 1991, Holmes and Austad 1994, Healy et al. 2014). Although this is clearly not the only reason for e.g. bat lifespans exceeding those of similarly sized rodents (for complexities, e.g., hibernation, see Wilkinson and Adams 2019), we take the dichotomy 'volant or not' as a way to conceptualize extrinsic mortality differences in our first, trade-off-free model. We ask whether a bat, assumed to experience relatively low extrinsic mortality, will be selected more strongly to delay senescence than a similar-sized non-volant organism, such as a mouse. Note that ignoring trade-offs means that we are in this first exercise not interested in the fact that litter sizes are smaller in bats than in rodents; we wish to consider the effect of mortality in isolation of everything else. Reproductive effort and its potential trade-offs with senescence will be considered in the second part of our paper (see also the appendix of Day & Abrams 2020 for an analytical example with trade-offs).

We further simplify the situation (away from real life, but helpful for heuristic understanding) by assuming a finite lifespan that does not permit more than one or two breeding attempts. Both the bat and the mouse have two competing types that differ in their rates of senescence in a simple and dramatic fashion: a 'fast-senescer' can only breed once and always dies thereafter, while a 'slow-senescer' can breed up to two times (we also include survival up to each breeding event). Clearly, both mice and bats will benefit from adding an extra breeding attempt to their lifespan, if all else is equal (i.e. in the absence of any trade-offs). The sign of

selection is therefore clear, and our aim here is to compare its relative strength for the two species. If bats benefit much more from the extra breeding attempt than mice, then selection on bats to reduce senescence is stronger and the result is in line with Williams' hypothesis.

Each mouse individual survives with probability s_M from birth to first reproduction, and slow-senescing mice additionally have the same probability of surviving after their first breeding to reach their second attempt. For bats, the rules are the same, but the survival probabilities equal s_B. Since we assume all else is equal, we assign the same fecundity F to mice and to bats. F also does not change between the first and the second breeding attempt. Since there are already many analytical results available (e.g. Day & Abrams 2020), and our aim is to aid intuition maximally, we will make use of a single numerical example where s_B=3 s_M, i.e., bat survival is three times that of mice, and we show results assuming 20% survival in mice, 60% in bats (Table 1 gives an overview of bat and mice life-history parameters).

The lifetime reproductive success (LRS) of slow-senescing bats is increased by 4.8/3 = 1.6 relative to the fast-senescing bats, i.e. an improvement of 60%. The LRS of slow-senescing mice is increased by 1.2/1 = 1.2 relative to fast-senescing mice, i.e. an improvement of 20%. It is not a coincidence that 60% and 20% are identical to the survival values we assigned to the two species since $\frac{\mathrm{LRS_{slow}}}{\mathrm{LRS_{fast}}} = \frac{sF+s^2F}{sF} = 1+s$, thus s is a direct measure of the expected improvement over the baseline. Since the improvement in LRS of bats was a factor three times the improvement in LRS of the mice when gaining the ability to breed twice, one might be tempted to conclude that bats are selected to reap the benefits of a long life much more strongly than mice, based on the extrinsic mortality argument ($s_B > s_M$).

However, this conclusion is premature, and this illustrates a key argument in the debate. In the absence of density regulation, the superior survival of bats compared with mice also makes their population grow much faster than that of mice – in our example, their growth rate is precisely threefold (Table 1). This result applies for any positive value of F: the terms containing F in the calculation of the growth rate λ are identical for bats and for mice. It does, however, require that bat fecundity does not differ from mouse fecundity, which is simply a reminder that we are focusing here on the effect of extrinsic mortality alone, and leave fecundity considerations for later.

An important point to note here is that LRS is only a valid fitness measure if density-dependence acts on fecundity of individuals of all ages equally (Mylius and Dieckmann, 1995). In the absence of any density dependence, populations will be growing exponentially and the population with the fastest growth rate will dominate. In general, invasion fitness is the only reliable fitness measure (see Kokko 2021 for a review about population fitness), but under

some circumstances invasion fitness simplifies to a familiar life history measure such as the population growth rate, or the life-time reproductive success (see discussion in Mylius and Dieckmann, 1995). Intuitively, if two individuals both have the same LRS but one produces its offspring earlier, these (and their descendants) form a larger proportion of the future gene pool in a growing population. To quantify precisely how important it is to reproduce early in a fast growing population, we calculate the relative contribution to the total population at some later time *t* of an early produced offspring and a late produced offspring in both the mouse and the bat population.

This point is easiest to make with populations of slow senescers in both bats and mice, simply because contrasting fast senescers only would not allow us to specify how an individual's fitness accrues from early and late produced offspring. Also, for there to be any late-produced offspring, we focus on an initial parent assumed to be among the lucky ones who survive to breed twice. The offspring themselves are examples of slow-senescing life histories with the appropriate survival rates. These initial founding offspring, of which we consider 1 each (early and late produced) in both species, are placed into a population that is growing exponentially at the appropriate species-specific rate (Figure 1, with growth rates from Table 1). The differing timing of offspring placement into the population is graphically illustrated as an earlier and a later star symbol in Figure 1), and since the populations of both mice and bats are assumed to grow, the late-produced offspring form a smaller proportion of the population at the time of production than the early-produced offspring. This initial disadvantage has consequences ever after. Measured at a later time point, the proportion of the population that descends from the early-produced offspring is far larger than the proportion descending from the late-produced offspring in the (well-surviving and hence fast growing) bat population. This difference also exists in the mouse population, but it is much less extreme in this species (the widths of the two 'stripes' denoting lineages show only moderate differences in Figure 1a, and strong differences in Figure 1b).

These differences can be quantified. The descendants of an early-produced offspring, $N_{\mathrm{B,early}}$, as well as a late-produced offspring, $N_{\mathrm{B,late}}$, will eventually reach a stable proportion of young and 'old' (namely second year) individuals, forming two lineages that both grow at a rate identical to the population growth rate (Caswell 2001). It follows that the lineage arising from the early-produced offspring, measured at some time *t* after both lineages have been initiated, is larger than the lineage arising from the population of descendants of the late-born offspring, by a factor of λ_{B}. That is,

$$N_{\mathrm{B,early}}(t) = \lambda_B N_{\mathrm{B,late}}(t). \qquad (1a)$$

Likewise for the descendants of the early and late offspring of a focal mouse individual,

$$N_{\mathrm{M,early}}(t) = \lambda_M N_{\mathrm{M,late}}(t). \qquad (1b)$$

If we divide both sides by the total population (this total contains additionally all other descendants from this or other parents), we obtain the proportion of the total population at time *t* that represents descendants of early and late offspring, respectively. The proportion of the population descended from the original bat parent's early offspring is larger than that of her late offspring by a factor λ_{B}, and for mice, this is λ_{M}. Since $\lambda_{\mathrm{M}} = \frac{1}{3}\lambda_{\mathrm{B}}$ (Table 1), the early produced bats are worth three times more (relative to their later produced siblings) than early produced mice (relative to theirs). In other words, in a population that is growing at a threefold rate compared with another, the importance of reproducing early is also elevated by the exact same factor (threefold). This is analogous to investing money into a growing economy: the faster the growth, the better off are those who were able to invest early; the penalty (discounting) of late investments is visible in Figure 1 as the trumpet shaped pale stripes (descendant lineages) being more unequal in height for the bat than for the mouse.

Therefore, we have a situation where on the one hand it appears more 'profitable' to have the ability to breed twice if chance often permits this longevity to really materialize (in the bats), but on the other hand, this very ability allows populations to grow fast, and this means that late-produced offspring are, to borrow an economic term, discounted (much less valuable). The cancelling out occurs, in other words, because one could argue both 'for' and 'against' one of the species being the one selected more strongly to survive to breed twice. The argument for those who root for the bats: clearly selection to have a robust enough body that can breed twice can only pay off if extrinsic circumstances allow this to be materialized, and they do so three times more often for bats than for mice. The counterargument, favouring the idea that mouse populations should instead be selected more strongly: in bats, late-produced offspring are a particularly poor investment, as the good survival of all individuals means that a late-produced young forms a much smaller proportion of the gene pool than an early-produced one. In the mice, this penalty is only a third of what it was in the bats. The truth is that neither bat nor mice experience stronger selection to breed twice: the factors (3 and 1/3) cancel out.

Mathematically, note that the relative increase in LRS due to the second offspring for bats was given by $\frac{s_B^2 F}{s_B F} = s_B$. If we divide this by the growth rate of the population of slow-senescing bats, λ_B, to account for their reduced value compared to early offspring, we obtain $\frac{s_B}{\lambda_B} = \frac{0.2}{1.17}$ = 0.17. Similarly, for mice the increase in LRS due to the second offspring weighted by their reduced value is given by $\frac{s_M}{\lambda_M}$= 0.17. For both mice and bats fitness this term is exactly equal to the increase in the population growth rate relative to the old population growth rate, as can be seen from the Euler-Lotka equation, $\lambda_{slow} = sF(1 + \frac{s}{\lambda_{slow}})$. The growth rate increases by 17% due to the possibility of second set of offspring, and therefore selection to become a slow senescer that can breed again is the same in bats and mice. It is worth re-emphasizing that the example works with other s_B : s_M ratios too (this can be seen by dividing the expressions in the last row of Table 1 with those on the penultimate row; the values of s_B and s_M simply cancel out, and the values $\lambda_{slow}/\lambda_{fast}$ become identical for the two species.

To conclude, even though being able to delay senescence until after the 2nd breeding attempt (instead of dying after the 1st) benefits bat LRS much more than mice if surviving to breed is more likely for bats, LRS fails as a predictor of selection because it does not take into account that late-produced offspring are also less valuable than the early-produced ones — and this decline in value is much faster for the species that, by virtue of its high survival, has faster population growth. Since we assumed that higher survival directly translates into a higher growth rate, the 'penalty' of placing offspring late into the population is far greater for bats than for mice. These two effects (better improvement in LRS, and the larger penalty) cancel each other out exactly. The outcome is the 'null result': selection for slow life-histories (against senescence) is equally strong in the bat and the mouse population.

This result can also be confirmed by comparing population growth rates of entire populations of fast-senescers versus slow-senescers. Calculating the population growth rate improvement of slow-senescing bats and mice relative to their fast-senescing competitors yields the same answer for both species: both improvements are 17% (with data from Table 1, note that 1.17 / 1 = 3.51 / 3). Since population growth rate is the correct fitness proxy for exponentially growing or declining populations (Charlesworth, 1994; Mylius and Diekmann, 1995; Caswell, 2001), not the LRS, this section has confirmed that age-independent extrinsic mortality does not affect the relative benefit of reduced senescence for species experiencing different levels of extrinsic mortality, *in the absence of density-dependence.*

Beyond the null: what cancels out under density dependence, what does not?

Above, we intentionally considered an unrealistic comparison, to be able to show what happens if survival is the only difference between two populations. Real bat populations do not show threefold growth compared with mice, and neither can sustain exponential growth forever. Intuition (to some at least) suggests that the slow-senescing bats can begin to truly reap the benefits of a long life if density dependence makes the 'penalty' of having to discount the value of late-produced offspring less severe. Why? If the population does not in reality expand as fast as predicted by density-independent growth rules "5 offspring per year and 60% survival for all who aren't scheduled to die of old age yet", then the trumpet shapes of Figure 1 do not expand as fast as they did before; mathematically, slower growth means that the value of late-placed offspring is not devalued as strongly compared to the early-placed ones, and as a whole density-dependence offers a potential for a smaller penalty for a lineage of descendants appearing late into a population. If population growth ceases altogether, the penalty vanishes as well. In other words: *if we assume that slow bats can reach old age just as often as they did in the density-independent case,* and now their late offspring are not nearly as bad investments as they were under unlimited population growth, then selection is now much freer to reward slow life histories (Figure 2 illustrates the idea graphically).

This intuition can be correct, but it comes with a strong caveat: the *if* clause in the previous sentence. The argument relies on the assumption that bats really can reach old age just like they did under unlimited growth. The crux of the issue is that population growth cannot be reduced 'just like that'; that is, while keeping all vital rates unchanged. Something has to change for the growth to be lower. Perhaps fewer young are born, or perhaps some are never born because their potential mothers had already died. There are many possibilities, and this matters.

If slowed down population growth is achieved by making reproduction somewhat harder for everyone, then it is indeed possible that the chances that a slow-senescing bat reaches old age remain the same ($s_B^2 = 0.36$ in our example) across all densities (Abrams 1993, Day & Abrams 2020). In this situation, the slowing down and eventual stabilization of population growth can begin favouring delaying senescence in those organisms that are relatively likely to reach old age in the first place (i.e. bats as opposed to mice in our example). Slow-senescing mice, too, enjoy some of this advantage, but only 4% of them do, because high extrinsic mortality ($s_M^2 = 0.04$) means most (96%) do not live to enjoy their intrinsic ability to breed twice.

But, importantly, slowing down (the tendency of $r = \ln(\lambda)$ to decline towards 0) can also be achieved via other mechanisms. High densities could, for various ecological reasons, make it

very hard for older bats (or mice) to survive while the fecundity of survivors is left intact. Now it is quite hard to be convinced that those who *in principle* have good prospects for reaching old age (bats, as opposed to mice, in our example) would also *in practice* achieve this benefit. If density regulation effectively prevents slow-senescing types from translating their intrinsic survival ability to actual survival (and subsequent reproduction), selection will be blind to their slow-senescing phenotypes.

This can explain why the 'null' result sometimes happens even when density dependence is included (e.g. Caswell and Shyu 2017). Typically, in these cases, a range of extrinsic mortality values are compared between hypothetical populations, but each population is also forced to have zero growth ($r = 0$). If the condition $r = 0$ is achieved by adjusting mortality rates at all ages equally, then, effectively, the initial elevation of extrinsic mortality (for those populations in the comparison who were supposed to have high extrinsic mortality) is removed again from the model by density-dependent adjustments of the mortality itself. Some people argue that this is a fundamental and exciting proof that helps us understand why extrinsic mortality cannot matter (Moorad et al. 2019); Moorad et al. 2020a make their preference for including the total effects of a mortality adjustment more explicit still. Others might reason that this particular exercise is somewhat pointless, as it assumes that underlying variations in extrinsic mortality will not be visible in the mortality schedules experienced by individuals at equilibrium. Phrased in the context of our example, they would never be measurable as real bats having lower mortality than real mice.

Because this example is important, we repeat the message in the context of an experiment. While our example is hypothetical, it is inspired by experimentally imposed high and low adult mortality regimes in *Drosophila* populations (Stearns et al. 2000). Imagine that an empiricist is applying random (age-independent) mortality to the population in the high-mortality treatment, but ends up realizing that the remaining individuals respond with improved survival, so that total mortality (considering both the treatment and its subsequent effect) remains unchanged. Any measures of senescence rates remain unchanged as well. Did the researcher recover a deep insight, confirming Moorad et al's message? Or will she instead respond by stating "my experiment didn't work - it remained uninformative because the manipulation failed to produce an actual difference in the mortality actually experienced by the population, making the subsequent finding that senescence didn't change a trivial one"? We leave it to the reader to form their own opinions about this matter, as we believe both viewpoints have their merits. It is of interest to note that Moorad et al. 2020a identify a difference in Day & Abrams' (2020) thinking compared to theirs based on whether the label 'extrinsic mortality' is applied

before or after various consequences, such as those in our hypothetical experiment, have been allowed to act on the population.

To sum up, by now, we have achieved some intuition as to why it is important to identify who precisely fails to survive, or fails to be born, when increasing densities reduce population growth. The key question is: can a slow-senescing phenotype reap the benefits of its long life across all relevant population densities, or are its survival prospects themselves affected by density? If survival of older individuals is left relatively intact and so is the value of late-placed young (due to the population no longer growing so fast), then we can expect the Williams prediction to hold. If the slow-senescer, on the other hand, itself suffers from density increases, we may enter the realm of the null, or even an anti-Williams region (see Abrams 1993 for examples), if the survival of old slow-senescers is disproportionately targeted by density regulation.

Ten case studies of slow and fast life histories

To make our thought experiment above as simple to follow as possible, we focused on an 'all else being equal' comparison where the two species did not differ in fecundity and there were no trade-offs: an ability to delay senescence required no lowering of reproductive effort. We next turn to examples that are considerably more realistic than the above comparison between hypothetical species that only differ in one respect (survival) and cannot ever breed more than twice. We now sacrifice analytical tractability to achieve three goals: (i) we consider a wide variety of density-dependent scenarios; (ii) we link senescence to the ideas of fast and slow life histories (which is argued to underlie e.g. the mammal-bird dichotomy in senescence rates, Jones et al. 2008, relationships between senescence and latitude across bird species, Møller 2007, all the way to within-species patterns, Cayuela et al. 2020), taking into account that a slow senescence rate may involve 'accepting' lower fecundity; and (iii) we see if the intuition remains robust in non-equilibrium situations.

We explore 10 different kinds of density regulation, of which nine are organized in a 3×3 setup (Table 2) and an additional one (density dependence acting on recruitment probability) added for the reason that this form of population regulation is often discussed in territorial species (Newton, 1992; Sæther et al. 2002; López-Sepulcre and Kokko, 2005; Krüger et al. 2012; Grant et al. 2017). In the 3×3 scheme, we have three examples each of density-dependence acting on (1) survival in an age-independent manner, (2) on adult survival (neither the number of juveniles nor adults impacts juvenile survival) or (3) on fecundity (noting also that fecundity regulation in this case is mathematically indistinguishable from newborns dying, or having trouble recruiting into the population; see Discussion). Each of these is investigated in three

different ways: density dependence may be absent for a while until it acts in a pulsed ('catastrophic') manner via sudden decreases in the vital rates either (a) deterministically above a certain density or (b) stochastically, or (c) density-dependence may exert a continuously increasing pressure on the relevant vital rate. The additional scenario of density dependence acting via recruitment limitation is closest to the case that combines (3) with (c). Obviously, the ten scenarios we consider do not represent an exhaustive list of all (infinitely many) possibilities, but are helpful for highlighting what is common and what is different between fecundity and survival regulation.

We implement the same type of trade-off between fecundity and survival in all ten scenarios, where we contrast the success of a 'fast' life history with one 'slow' one that does not senesce at all. In the above, trade-off-free section, the slow type always had an advantage, but now we switch to a trade-off: absence of senescence can only be achieved by 'accepting' a lower fecundity than that achieved by the fast-senescer. The 'fast' type has thus superior fecundity but also experiences senescence according to the Gompertz-Makeham model, where mortality has an intrinsic component that increases exponentially with age (Gompertz 1825, Makeham 1860, Missov and Lenart 2013). For simplicity, we only consider survival senescence, and fecundities do not depend on the age of the reproducing individual (while they depend on population density in 3 out of the 10 scenarios). We use subscript 0 to denote slow (using the mnemonic that 'no senescence' is indicated with a 0), and 1 denotes the fast type.

We describe here what we call the 'standard procedure' (Figure 4), which are the steps that are shared among all our regulation scenarios; Table 2 then describes what differs between each scenario.

Each step begins with a census of all individuals, whose ages are integers 1, 2, … with no upper limit (in practice, we worked with an upper limit of 200, without ever observing a significant number of individuals reaching this age). The life cycle continues with reproduction, where slow and fast individuals' fecundities relate to each other as a *per capita* fecundity ratio $F_0 : F_1$. In the standard scenario, this is achieved simply by letting slow types produce F_0 offspring, while fast types produce F_1. In case of fecundity regulation (3 of the 10 cases), the fecundities need to respond to density; we then interpret F_0 and F_1 as maximal fecundities in the absence of competitors, and use realized fecundities when letting strategies compete: realized fecundities are αF_0 and αF_1 where $\alpha < 1$ takes smaller values with increasing density (Table 2).

Next in the life cycle, survival is applied deterministically, such that a proportion of individuals remain to be part of the next census (Figure 4). Survival for slow life histories equals

$$s_0 = e^{-\mu}, \qquad (2)$$

where the subscript 0 refers to slow, μ is extrinsic mortality (interpreted as a constant hazard, which means that 1 year is survived with $e^{-\mu t}$ where t = 1, hence $e^{-\mu}$), and there is no age-dependency since slow individuals do not senesce. Note that the 'no age-dependency' statement applies to the standard procedure; density-dependent adjustments may mean that survival is adjusted (multiplication with a factor α) for some age classes $s_0(i)$ but not others (Table 2).

Fast individuals' survival is age-dependent to begin with (even in the standard procedure; age-dependency may become additionally modified by density dependence). In the standard procedure, we model senescence of fast individuals using the commonly used Gompertz-Makeham model of mortality which assumes mortality has a constant age-independent component μ and a component that increases exponentially with age i,

$$\mu_{\mathrm{GM}}(i) = \mu + \frac{1}{d} e^{\frac{(i-a)}{d}}. \quad (3)$$

It follows that the probability that a newborn reaches age 1 (and becomes part of its first census) is $s_1(1) = P_1(1) = e^{-\mu + e^{-\frac{a}{d}} - e^{\frac{1-a}{d}}}$. Here $s_1(1)$ denotes survival over 1 unit of time from 0 to 1, which here is the same as $P_1(1)$, the proportion of individuals still alive at age 1 (the 1 in brackets denotes age, the subscripted 1 indicates this applies to the fast strategy). For the case of newborns these are the same value ($s_1(1) = P_1(1)$). For later ages, they are not. Generally

$$P_1(i) = e^{-\mu i + e^{-\frac{a}{d}} - e^{\frac{i-a}{d}}}. \quad (4)$$

Since in our notation $s_1(i)$ captures survival from $i-1$ to i, it equals $P_1(i)/P_1(i-1)$, which yields

$$s_1(i) = e^{-\mu + e^{\frac{i-1-a}{d}} - e^{\frac{i-a}{d}}}. \quad (5)$$

In the absence of extrinsic mortality ($\mu = 0$), senescence is the only cause of death, and under this (unlikely) scenario the parameter a gives the modal age of death. In the presence of extrinsic mortality, a alone no longer translates into the modal age of death; across all values of $\mu \geq 0$, a is better interpreted as the age at which senescence acts strongly to limit lifespan (Figure 3) — loosely put, it measures how long an individual is 'built for'. The parameter d impacts the variance in lifespan: at low d values most individuals die around the same age, at higher d values there is more variation in the age at death. As before (with s_0), the values $s_1(i)$ can be further modified by density dependence (Table 2).

Clearly, we do not claim that nature offers only two life history options available for a population to choose from, or that a completely non-senescing phenotype is within the range of evolvable possibilities for many organisms (but see Roper et al. 2021 for a recent discussion on the topic). We focus on the simple contrast between an ageing high-fecundity and a non-ageing low-fecundity strategy because it serves our general aim of improving intuition about why density dependence has its known effects on the general applicability of the null result. For each of the ten types of population regulation (density dependence), we report the outcome of competition between the slow and the fast type for a range of values of extrinsic mortality μ (which acts on both types equally).

Whatever the value of μ, the outcome obviously depends on just how much lower the fecundity of the slow type is (the ratio F_0:F_1). Intuition suggests that there is always some intermediate value where the fates of the two strategies switch. At the one extreme, if $F_0 = 0$, the lack of senescence of slow individuals cannot help them in competition against F_1 individuals, as the former are infertile; while at the other extreme, where $F_0 = F_1$, slow individuals have a longer lifespan with no cost in fecundity, and the slow life history is guaranteed to take over. In between, there is a value of F_0:F_1 where selection switches from favouring fast to favouring slow. Therefore we show all results in the form of an answer to the following question: what is the lowest fecundity (F_0) that allows the slow strategy to outcompete the fast strategy (with fecundity F_1)? And, how does this threshold depend on extrinsic mortality? If it increases with μ, then the 'Williams' prediction holds: low-μ conditions make it easy for slow life histories to evolve, even if building a robust body (high a) means sacrificing fecundity by a lot.

The results of the 10 different regulation styles are clearly categorizable in three rows (Figure 5). When density-dependence causes all individuals to suffer diminished survival, extrinsic mortality has no effect on the threshold value, i.e. the 'null' result holds (Figures 5: 1A-C) — and it does so regardless of whether we chose a 'pulsed' type of regulation acting occasionally (cases 1A,B) or one of a more continuous nature (case 1C). When juveniles are shielded from the negative effects of density, however (ecologically, such a result might arise if their niche differs from that of the adults, and the adult niche is the limiting one), then an increase in extrinsic mortality makes it easier for the slow strategy to invade (the threshold reduces), and we find an anti-Williams pattern (Figure 5: 2A-C). Finally, when density-dependence acts on fecundity or on juvenile recruitment, an increase in extrinsic mortality makes it harder for the slow strategy to invade (Figure 5: 3A-D), in line with Williams' hypothesis and predictions made by later models (Abrams 1993).

Discussion

Williams' hypothesis has triggered lively debates among theoreticians for decades. Previous work has generally focused on selection gradients. Our approach offers an alternative analysis, focusing on an intuitive explanation by considering the relative importance of placing offspring into a population earlier rather than later. Our results do not contradict earlier work (e.g. Hamilton 1966, Charlesworth 1993, Wensink et al. 2017, Dańko et al. 2018, Day & Abrams 2020), but we hope that our examples make it easier to grasp why age-independent extrinsic mortality does not affect the evolution of senescence in the absence of density-regulation, or in the presence of density-regulation that depresses survival to an equivalent degree across all individuals.

Also, our results are fully in line with earlier findings (Abrams 1991, Day & Abrams 2020) that emphasize that whenever density dependence 'hurts juveniles' (either by making it difficult for adults to produce them in the first place, or making their survival or recruitment low), then Williams' prediction is likely to hold). The general pattern that density-dependence acting on juvenile production or recruitment leads to a Williams result is strikingly consistent, and given that there is rather broad empirical support for Williams-type patterns across species (e.g. Ricklefs 2008), it may be seen as indirect evidence that population regulation often operates via this mode. Note that we obtained the same general pattern using a cancer-inspired survival curve from Kokko & Hochberg (2015) instead of the Gompertz-Makeham curve as well (code and figures at https://doi.org/10.5281/zenodo.6705180).

To understand why density regulation affecting fecundity leads to a Williams-like result, it is useful to go back to our first, trade-off-free result: the comparison of bats and mice. There, we showed that the intuition behind the Williams prediction is that the benefits of a potentially long life (little senescence) can only materialize if the organism also avoids all other causes of death that do not directly relate to senescence. Populations grow, and growth must become limited at some point; the crucial question is where in the life cycle the effects are 'felt'. One possibility is that juvenile production suffers. This allows the slow-senescing type to keep reaping the benefits of its robust body even when population regulation is acting. In a typical assumption set (like ours), it survives just as well as under low density, and while its fecundity now suffers, this effect is felt by parents of all ages, and does not translate into a reduction in relative productivity of old parents (the crucial tacit assumption here is that juveniles feel the negative effects of density equally across all ages of their *parents*). The intuition that increased extrinsic mortality rate reduces the benefit of a long life is therefore correct *when regulation acts on fecundity*, and increased mortality increases the threshold fecundity needed by the slow strategy to win (figures 3A-3D).

Our 10 examples, that themselves are based on a specific comparison where a no-senescing type competes with a Gompertz-Makeham-type senescer, obviously do not constitute proof that deviant patterns could never be found, should one consider other comparisons. Our approach provided its simple graphical contrasts (flat, decreasing and increasing curves in Figure 5 corresponding to null, anti-Williams and Williams, respectively) by pitting a senescing type against an ideal type that is immune to senescence. The biological interpretation of the latter is somewhat challenging in some cases, especially our setting 2A where only oldest age classes are removed at high density. The idealized type of a slow life history here combines the assumption of intrinsically age-independent survival (in the sense of eq. 1) with an inability to withstand high density situations that applies from a certain age onwards; the physiological interpretation of such a case is challenging. A general point, however, remains: given that different population regulation modes definitely exist (e.g., Drury & Dwyer 2005, Sæther et al. 2016, Dánko et al. 2017, Lee et al. 2021), variance in senescence (and lifespan) among taxa cannot be solely attributed to differences in extrinsic mortality.

Because our results show that comparative predictions ideally require an understanding of causes of shorter lifespans in one population compared with another, as well as general information on the mode of population regulation, it may be premature to make statements about individual case studies. It is nevertheless interesting that e.g. predation has been shown to impact senescence either positively or negatively. Insular populations of opossums are under lower predation pressure and senesce at a lower rate compared to mainland populations (Austad 1993). Reznick et al. (2004) on the other hand showed that guppy populations subject to higher predation rates senesced at lower rates than populations under lower predation risk. The latter authors speculate about possible mechanisms explaining this anti-Williams type pattern, but data is still lacking to show possible density-dependent effects on older age classes. More generally, empirical studies of the effect of extrinsic mortality on senescence usually lack evaluations of density-dependent effects on vital rates (but see Stearns et al. 2000), hindering interpretations about causal factors behind observed patterns. There is also indirect evidence for the effect of predation on senescence; patterns of senescence are compared among species with different modes of life, under the assumption that the ability to fly or to live underground decrease exposure to predation (Austad and Fischer 1991, Holmes and Austad 1994, Healy et al. 2014).

Since our focus was on making the theory easy to understand, we do not claim that our study encompasses all the mechanisms by which extrinsic mortality affects senescence. It is interesting that our results, in line with earlier theory (Abrams 1991, André and Rousset 2020, Day & Abrams 2020), emphasize the importance of understanding population regulation, while in experimental (Stearns et al. 2000) and observational (e.g., desiccating ponds) data, high mortality or high risks of habitat disappearance are often stated to lead to faster life histories (*Daphnia:* Dudycha and Tessier 1999, killifish: Tozzini et al. 2013). This may appear to be at odds with our predictions, as desiccation typically kills adults and the next generation hatches from eggs once the water returns. Similarly, grasshoppers living at higher altitudes are subject to higher risks of freezing episodes and accordingly show faster life-histories and earlier senescence compared to populations at lower altitudes (Tatar et al. 1997). Note, however, that abiotic causes behind mass mortality do not involve a causal link from high density to mortality, a link that is incorporated in density-dependent senescence models (like ours). In other words, although *Daphnia* populations are more dense just before a desiccation event than when the hatching first began, and there may be more grasshoppers late in the season than early, this is correlation, not causation: an abundance of *Daphnia* does not cause ponds to dry and winter does not happen because grasshoppers became abundant. Ephemeral habitats therefore require models of their own; one possibility that our models did not address is a timescale where ephemeral habitats may cut individual lives short before maturity is reached. Speeding up maturation time may be an adaptive response in such situations, with effects felt throughout the life cycle.

Models that include processes not included by us may highlight other reasons for finding specific patterns. Anti-Williams patterns may, for example, be found when explicitly considering condition-dependence impacting susceptibility to extrinsic mortality (the definition of 'extrinsic' is then subtly different: it ceases to be 'unavoidable' as an organism's traits now influence its susceptibility to it). In brief, when being frail or senescent increases an organism's susceptibility to extrinsic mortality, high extrinsic mortality leads to stronger selection on slow senescence (Abrams 1993, Williams & Day 2003). Fitting this pattern, salmon populations senesce at lower rates when predation rates by bears are high and directed towards senescing individuals specifically (Carlsson et al. 2007). Selection for heat resistance is associated with increase in lifespan in *Caenorhabditis elegans*, such that populations under higher temperature-related mortality risks also senesce at slower rates (Chen and Maklakov 2012). We have also chosen to model trade-offs (or lack thereof) in a stylized way, leaving subtleties such as the difficulty of optimizing function simultaneously for early and late life (Maklakov & Chapman 2019) for later studies. For additional viewpoints see e.g. the system reliability approach (Gavrilov & Gavrilova 2001, Laird & Sherratt 2009, 2010a,b) as well as selection that relates to the possibility of

565 indeterminate growth (Vaupel et al. 2004, Caswell & Salguero-Gómez 2013, Purchase et al.
566 2022). While the multitude of factors listed above suggest that wide diversity in senescence
567 patterns and lifespans (Jones et al. 2014) is the expectation, we hope that our conceptual
568 examples help to see why a specific feature of life cycles – the diversity in modes of population
569 regulation — continue to play a very important role.

570 Data, scripts and codes availability

571 Matlab scripts are online: https://doi.org/10.5281/zenodo.6705180.

572 Conflict of interest disclosure

573 The authors declare that they comply with the PCI rule of having no financial conflicts of
574 interest in relation to the content of the article. In addition, the authors declare that they have
575 no non-financial conflict of interest with the content of this article.

576 Funding

577 This research was supported by Swiss National Science Foundation grant number
578 310030B_182836 (awarded to Hanna Kokko). CdV was also supported by an Academy of
579 Finland grant (no. 340130, awarded to Jussi Lehtonen).

580 References

581 Abrams, P. A. (1991). The fitness costs of senescence: The evolutionary importance of events in
582 early adult life. *Evolutionary Ecology*, *5*(4): 343–360.

583 Abrams, P. A. (1993). Does Increased Mortality Favor the Evolution of More Rapid Senescence?
584 *Evolution*, *47*(3): 877–887.

585 André, J-B, and Rousset, F. Does extrinsic mortality accelerate the pace of life? A bare-bones
586 approach. *Evolution and Human Behavior 41.6* (2020): 486-492.

587 Austad, S. N. (1993). Retarded senescence in an insular population of Virginia opossums
588 (Didelphis virginiana). *Journal of Zoology*, 229(4), 695-708.

589 Austad, S. N., & Fischer, K. E. (1991). Mammalian aging, metabolism, and ecology: Evidence
590 from the bats and marsupials. *Journals of Gerontology*, *46*(2): 47–53.

591 Capdevila, P., Stott, I., Beger, M., & Salguero-Gómez, R. (2020). Towards a comparative
592 framework of demographic resilience. Trends in Ecology & Evolution, 35(9), 776-786.

593 Carlson, S. M., Hilborn, R., Hendry, A. P., & Quinn, T. P. (2007). Predation by bears drives
594 senescence in natural populations of salmon. *PloS one*, 2(12), e1286.

595 Caswell, H. (2007). Extrinsic mortality and the evolution of senescence. *Trends in Ecology &*
596 *Evolution*, *22*(4): 173–174.

597 Caswell, H., & Shyu, E. (2017). Senescence, Selection Gradients and Mortality pp. 56–82 in R. P.
598 Shefferson, O. R. Jones, & R. Salguero-Gomez (Eds.), *The Evolution of Senescence in the Tree of*
599 *Life*. Cambridge University Press. 2017

600 Caswell, H., 2001. *Matrix Population Models: Construction, Analysis and Interpretation* (2nd
601 ed.). Sinauer Associates, Sunderland, Massachusetts

602 Cayuela, H., Lemaitre, J.-F., Bonnaire, E., Pichenot, J. & Schmidt, B.R. 2020. Population position
603 along the fast-slow life-history continuum predicts intraspecific variation in actuarial
604 senescence. J. Anim. Ecol. 89:1069-1079.

605 Charlesworth, B. (1993). Evolutionary mechanisms of senescence. *Genetica*, 91(1-3): 11-19.

606 Charlesworth, B., 1994. *Evolution in age-structured populations* (Vol. 2). Cambridge: Cambridge
607 University Press.

608 Chen, H. Y., & Maklakov, A. A. (2012). Longer life span evolves under high rates of condition-
609 dependent mortality. *Current biology*, 22(22): 2140-2143.

610 da Silva, J. (2018). Reports of the Death of Extrinsic Mortality Moulding Senescence Have Been
611 Greatly Exaggerated. *Evolutionary Biology*, *45*(2): 140–143.

612 da Silva, J. 2020. Williams' intuition about extrinsic mortality was correct. Trends Ecol. Evol.
613 35:378-379.

614 Dańko, M. J., Burger, O., & Kozłowski, J. (2017). Density-dependence interacts with extrinsic
615 mortality in shaping life histories. *PloS one* 12(10): e0186661.

616 Dańko, M. J., Burger, O., Argasiński, K., & Kozłowski, J. (2018). Extrinsic Mortality Can Shape
617 Life-History Traits, Including Senescence. *Evolutionary Biology*, *45*(4): 395–404.

618 Day, T., & Abrams, P. A. (2020). Density dependence, senescence, and Williams' hypothesis.
619 *Trends in Ecology & Evolution*, 35(4): 300-302.

620 de Roos, A.M. and Persson, L., 2013. *Population and community ecology of ontogenetic*
621 *development* (Vol. 59). Princeton University Press.

622 Doebeli, M., Ispolatov, Y., & Simon, B. (2017). Point of view: Towards a mechanistic foundation
623 of evolutionary theory. *Elife*, 6: e23804.

624 Dudycha, J. L., & Tessier, A. J. (1999). Natural genetic variation of life span, reproduction, and
625 juvenile growth in Daphnia. *Evolution*, 53(6): 1744-1756.

626 Drury, K.L. and Dwyer, G., 2005. Combining stochastic models with experiments to understand
627 the dynamics of monarch butterfly colonization. *The American Naturalist*, 166(6), pp.731-750.

628 Gadgil, M., & Bossert, W. H. (1970). Life historical consequences of natural selection. *The*
629 *American Naturalist*, *104*: 1–24.

630 Gaillard, J. M., & Lemaître, J. F. (2017). The Williams' legacy: A critical reappraisal of his nine
631 predictions about the evolution of senescence. *Evolution*, 71(12): 2768-2785.

632 Gavrilov, L.A. & Gavrilova, N.S. (2001). The reliability theory of aging and longevity. J. theor.
633 Biol. 213:527-545.

634 Gompertz, B. (1825). On the nature of the function expressive of the law of human mortality,
635 and on a new mode of determining the value of life contingencies. *Philosophical transactions of*
636 *the Royal Society of London*, (115): 513-583.

637 Grant, J.W.A., Laura K. Weir, L.K. and Steingrímsson, S. Ó. "Territory size decreases minimally
638 with increasing food abundance in stream salmonids: Implications for population regulation."
639 *Journal of Animal Ecology* 86.6 (2017): 1308-1316.

640 Hamilton, W. D. (1966). The moulding of senescence by natural selection. *Journal of theoretical*
641 *biology*, 12(1): 12-45.

642 Healy, K., Guillerme, T., Finlay, S., Kane, A., Kelly, S.B.A., McClean, D., Kelly, D.J., Donohue, I.,
643 Jackson, A.L. & Cooper, N. (2014). Ecology and mode-of-life explain lifespan variation in birds
644 outand mammals. *Proc. R. Soc. B.:* 28120140298

645 Holmes, D. J., & Austad, S. N. (1994). Fly now, die later: life-history correlates of gliding and
646 flying in mammals. *Journal of Mammalogy*, *75*(1): 224–226.

647 Jones, OR, Gaillard, JM, Tuljapurkar, S, Alho, JS, Armitage, KB, Becker, PH, Bize, P, Brommer, J,
648 Charmantier, A, Charpentier, M, Clutton-Brock, T, Dobson, FS, Festa-Bianchet, M, Gustafsson, L,
649 Jensen, H, Jones, CG, Lillandt, BG, McCleery, R, Merilä, J, Neuhaus, P, Nicoll, MAC, Norris, K, Oli,
650 MK, Pemberton, J, Pietiainen, H, Ringsby, TH, Roulin, A, Sæther, BE, Setchell, JM, Sheldon, BC,

651 Thompson, PM, Weimerskirch, H, Wickings, EJ, Coulson, T. 2008. Senescence rates are
652 determined by ranking on the fast-slow life-history continuum. *Ecol. Lett.* 11:664-673.

653 Jones, O.R., Scheuerlein, A., Salguero-Gómez, R., Camarda, C.G., Schaible, R., Casper, B.B.,
654 Dahlgren, J.P., Ehrlén, J., Garcia, M.B., Menges, E.S., Quintana-Ascensio, P.F., Caswell, H.,
655 Baudisch, A. & Vaupel, J.W. 2014. Diversity of ageing across the tree of life. *Nature* 505:169-
656 172.

657 Kokko, H. and Hochberg, M.E., 2015. Towards cancer-aware life-history modelling. *Philosophical*
658 *Transactions of the Royal Society B: Biological Sciences*, 370(1673), p.20140234.

659 Krüger, O., Chakarov, N., Nielsen, J.T., Looft, V., Grünkorn, T., Struwe-Juhl, B. & Møller, A.P.
660 2012. Population regulation by habitat heterogeneity or individual adjustment? J. Anim. Ecol.
661 81:330-340.

662 Laird, R.A. & Sherratt, T.N. 2009. The evolution of senescence through decelerating selection
663 for system reliability. J. Evol. Biol. 22:974-982.

664 Laird, R.A. & Sherratt, T.N. 2010a. The evolution of senescence in multi-component systems.
665 BioSystems 99:130-139.

666 Laird, R.A. & Sherratt, T.N. 2010b. The economics of evolution: Henry Ford and the Model T.
667 Oikos 119:3-9.

668 Lee, E.D., Kempes, C.P. & West, G.B. 2021. Growth, death, and resource competition in sessile
669 organisms. Proc. Natl. Acad. Sci. USA 118:e2020424118.

670 López-Sepulcre, A., and Kokko, H. Territorial defense, territory size, and population regulation.
671 *The American Naturalist* 166.3 (2005): 317-329.

672 Makeham, W. M. (1860). On the law of mortality and the construction of annuity tables. *Journal*
673 *of the Institute of Actuaries*, 8(6)" 301-310.

674 Maklakov, A.A. and Chapman, T., 2019. Evolution of ageing as a tangle of trade-offs: energy
675 versus function. *Proceedings of the Royal Society B*, 286(1911), p.20191604.

676 Medawar, P.B. (1952). An Unsolved Problem in Biology. H.K. Lewis, London.

677 Mylius, S. D., & Diekmann, O. (1995). On evolutionarily stable life histories, optimization and
678 the need to be specific about density dependence. *Oikos*, 218-224.

679 Missov, T. I., & Lenart, A. (2013). Gompertz–Makeham life expectancies: Expressions and
680 applications. *Theoretical Population Biology* 90: 29-35.

681 Møller, A.P. 2007. Senescence in relation to latitude and migration in birds. J. Evol. Biol. 20:750-
682 757.

683 Moorad, J., Promislow, D. & Silvertown, J. 2020a. George C. Williams' problematic model of
684 selection and senescence: Time to move on. *Trends Ecol. Evol*. 35:303-305.

685 Moorad, J., Promislow, D. & Silvertown, J. 2020b. Williams' intuition about extrinsic mortality is
686 irrelevant. *Trends Ecol. Evol.* 35:379.

687 Moorad, J., Promislow, D., & Silvertown, J. (2019). Evolutionary Ecology of Senescence and a
688 Reassessment of Williams' 'Extrinsic Mortality' Hypothesis. *Trends Ecol. Evol. 34*(6): 519–530.

689 Newton, I. (1992). Experiments on the limitation of bird numbers by territorial behaviour.
690 *Biological Reviews*, 67(2): 129-173.

691 Promislow, D. E., & Harvey, P. H. (1990). Living fast and dying young: A comparative analysis of
692 life-history variation among mammals. *Journal of Zoology*, 220(3): 417-437.

693 Purchase, C.F., Rooke, A.C., Gaudry, M.J., Treberg, J.R., Mittell, E.A., Morrissey, M.B. & Rennie,
694 M.D. 2022. A synthesis of senescence predictions for indeterminate growth, and support from
695 multiple tests in wild lake trout. Proc. R. Soc. B 289:20212146.

696 Reznick, D. N., Bryant, M. J., Roff, D., Ghalambor, C. K., & Ghalambor, D. E. (2004). Effect of
697 extrinsic mortality on the evolution of senescence in guppies. *Nature*, 431(7012): 1095-1099.

698 Ricklefs, R.E. (2008). The evolution of senescence from a comparative perspective. *Funct. Ecol.*,
699 22:379-392.

700 Roper, M., Capdevila, P. & Salguero-Gómez, R. Senescence: why and where selection gradients
701 might not decline with age. *Proceedings of the Royal Society B* 288.1955 (2021): 20210851.

702 Sæther, B.E., Grotan, V., Engen, S., Coulson, T., Grant, P.R., Visser, M.E., Brommer, J.E., Grant,
703 R.B., Gustafsson, L., Hatchwell, B.J., Jerstad, K., Karell, P., Pietiäinen, H., Roulin, A., Røstad, O.W.
704 & Weimerskirch, H. 2016. Demographic routes to variability and regulation in bird populations.
705 Nature Comm. 7:12001.

706 Sæther, B. E., Engen, S., & Matthysen, E. (2002). Demographic characteristics and population
707 dynamical patterns of solitary birds. *Science*, 295(5562): 2070-2073.

Stearns, S. C. (1989). Trade-offs in life-history evolution. *Functional ecology*, 3(3): 259-268.

Stearns, S. C., Ackermann, M., Doebeli, M., & Kaiser, M. (2000). Experimental evolution of aging, growth, and reproduction in fruitflies. *Proceedings of the National Academy of Sciences,* 97(7): 3309-3313.

Tatar, M., Gray, D.W. and Carey, J.R., 1997. Altitudinal variation for senescence in Melanoplus grasshoppers. *Oecologia*, 111(3), pp.357-364.

Taylor, H. M., Gourley, R. S., Lawrence, C. E., & Kaplan, R. S. (1974). Natural Selection of Life History Attributes: An Analytical Approach. *Theoretical Population Biology*, *5*:104–122.

Tozzini, E. T., Dorn, A., Ng'oma, E., Polačik, M., Blažek, R., Reichwald, K., ... & Cellerino, A. (2013). Parallel evolution of senescence in annual fishes in response to extrinsic mortality. *BMC evolutionary biology*, 13(1): 1-12.

Tuljapurkar, S. *Population dynamics in variable environments.* Vol. 85. Springer Science & Business Media, 2013.

Vaupel, J.W., Baudisch, A., Dölling, M., Roach, D.A. & Gampe, J. 2004. The case for negative senescence. Theor. Pop. Biol. 65:339-351.

Wensink, M. J., Caswell, H., & Baudisch, A. (2017). The Rarity of Survival to Old Age Does Not Drive the Evolution of Senescence. *Evolutionary Biology*, *44*(1): 5–10.

Wilkinson, G. S., & Adams, D. M. (2019). Recurrent evolution of extreme longevity in bats. *Biology letters*, 15(4): 20180860.

Williams, G. C. (1957). Pleiotropy, Natural Selection, and the Evolution of Senescence Author. *Evolution*, *11*(4): 398–411.

Williams, P. D., & Day, T. (2003). Antagonistic pleiotropy, mortality source interactions, and the evolutionary theory of senescence. *Evolution*, 57(7): 1478-1488.

Table 1: List of variables and their values used in the trade-off free example. To calculate λ, start from the Euler-Lotka equation for a species with ω age classes,

$$\sum_{a=1}^{\omega} \lambda^{-a} l(a) b(a) = 1,$$

where $l(a)$ is the proportion of individuals surviving until age a, and $b(a)$ is the mean reproductive output of these survivors. For example, for the slow senescing mouse the Euler-Lotka equation becomes

$$\frac{1}{\lambda} s_M F + \frac{1}{\lambda^2} s_M^2 F = 1.$$

Solving for λ, gives the equation in the table.

				
	Variable name, or expression	Numerical example	Variable name, or expression	Numerical example
Fertility	F	5	F	5
Survival (to breed, or to breed again after breeding once)	s_{M}	0.2	s_{B}	0.6
Lifetime reproductive success				
Fast scenescers	$s_{\mathrm{M}}F$	1	$s_{\mathrm{B}}F$	3
Slow scenescers	$s_{\mathrm{M}}F$+s_{M}^2F	1.2	$s_{\mathrm{B}}F + s_{\mathrm{B}}^2F$	4.8
Growth rate (λ)				
Fast scenescers	$s_{\mathrm{M}}F$	1	$s_{\mathrm{B}}F$	3
Slow senescers	$\frac{1}{2}s_{\mathrm{M}}(F+\sqrt{F^2+4F})$	1.17	$\frac{1}{2}s_{\mathrm{B}}(F+\sqrt{F^2+4F})$	3.51

Table 2: Description of each of the ten scenarios used to create Figure 5.

Scenario	Description
1A	*Exponential growth & marked excess mortality when carrying capacity is exceeded.* Standard procedure (see main text) except in generations where the pre-breeding census yields $\sum_i N_0(i) + \sum_i N_1(i) > K$, where $N_0(i)$ and $N_1(i)$ are the number of slow-senescing and fast-senescing individuals in age class i, and *K* is a predefined carrying capacity. These high-density census events lead to the pre-breeding population experiencing 90% mortality across all ages of both life-history strategies (slow and fast). Thereafter, breeding and survival proceed normally (standard procedure) among the survivors. This type of regulation features exponential growth with a 'resetting' to small population sizes at regular intervals, with the cull impacting all ages equally.
1B	*Excess mortality events become more common at high density.* Like (1A), but resetting the population to small sizes occurs in a stochastic manner. Whether the 90% pre-breeding mortality occurs is decided probabilistically at each census. The probability *p* that 90% pre-breeding mortality is applied increases with density and happens with certainty if density K is reached or exceeded: $p = \begin{cases} \frac{\sum_i N_0(i) + \sum_i N_1(i)}{K} & \text{if } \sum_i N_0(i) + \sum_i N_1(i) < K \\ 1 & \text{if } \sum_i N_0(i) + \sum_i N_1(i) \geq K \end{cases}$
1C	*Continuous decline of survival with density.* At each census, we compute the value of a density-dependent factor $\alpha = \begin{cases} 1 - \frac{\sum_i N_0(i) + \sum_i N_1(i)}{K} & \text{if } \sum_i N_0(i) + \sum_i N_1(i) < K \\ 0 & \text{if } \sum_i N_0(i) + \sum_i N_1(i) \geq K \end{cases}$. The population follows the standard procedure as defined in the main text, but modified such that every survival value is multiplied by α. Note that this multiplication is also applied to juveniles born in the census year, who did not yet contribute to the census.
2A	*High density removes all old individuals.* The standard procedure is applied except when the pre-breeding census reveals a total population size above *K*, i.e. $\sum_i N_0(i) + \sum_i N_1(i) > K$. If the threshold is exceeded, all individuals above a certain age j, irrespective of being type 0 or 1, die before breeding begins. The value of *j* is chosen as the largest possible age threshold (leading to the smallest possible number of age classes removed) that yields $\sum_{i=1}^{j} N_0(i) + \sum_{i=1}^{j} N_1(i) < K$, i.e. brings the pre-breeding population back to < *K*: If some individuals from age class *j* need to be removed, everyone in that age class is removed.

	Afterwards, the remaining population reproduce and survive as normal (standard procedure).
2B	*High density leads stochastically to an event of removing all old individuals*. The removal event described in (2A) occurs probabilistically, with a probability that behaves like *p* in (1B).
2C	*Continuous decline of adult survival with density.* Like (1C), but only survival of parents is negatively impacted by high density. The production and survival of newborns of the current year is unaffected, as their parents' survival is only negatively impacted after breeding occurred (Figure 4).
3A	*Crowding stops reproduction entirely.* If the census yields $\sum_i N_0(i) + \sum_i N_1(i) > K$, there is no reproduction in the given year. Extant individuals survive according to the standard procedure.
3B	*High density leads stochastically to an event of reproductive failure*. As in 3A but now a no-reproduction year occurs with probability *p*, defined as in 1A.
3C	*Continuous decline of fecundity with density.* At each census, we compute α as in 1C, but it is now applied to fecundities. Multiplication with α, when performed both for F_0 and F_1, keeps the ratio F_0:F_1 intact.
3D	*Territoriality.* The standard procedure is applied in all other respects than juvenile survival. Adults die, which implies that there are $K - (\sum_i N_0(i)s_0(i) + \sum_i N_1(i)\, s_1(i))$ vacancies available once the population is proceeding towards the new census; survived juveniles are recruited to vacant territories, and should there not be sufficiently many vacancies available, the rest die.

Table 3: Classification of the ten scenarios according to the type of density-dependence and according to which vital rates are affected by the density-dependence.

Density-dependence acts on	**Pulsed DD**		**Continuous DD**	
	Deterministic	Stochastic	Deterministic	Competition for territories
Survival in an age-independent way	1A	1B	1C	
Adult survival in an age-dependent way	2A	2B	2C	
Recruitment, fecundity, or newborn survival	3A	3B	3C	3D

Figure 1

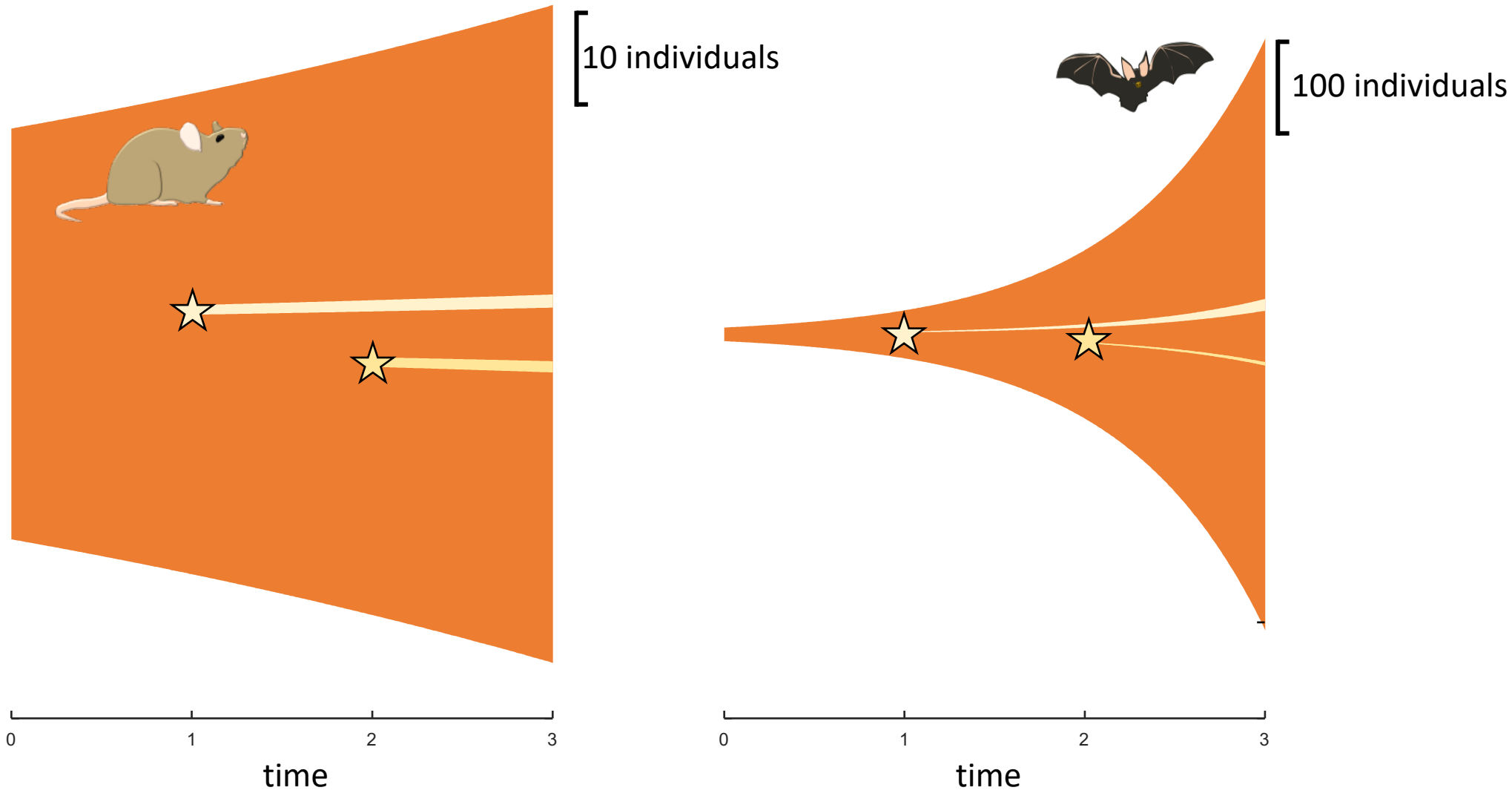


Figure 1. A visualization of why the bat, with its threefold growth rate, is penalized more for a delay in placing offspring into a growing population. For purely aesthetic reasons, population growth is depicted over continuous time ($r = \ln(\lambda)$). Values of r correspond to λ values given in Table 1. Populations are assumed to consist of slow senescers (Table 1) and stars depict the placement of one offspring at time $t = 1$ or $t = 2$ into a growing population. Both species consist of 50 individuals at time $t = 1$ (shown at a different scale as indicated, to fit the entire growth into picture, as bats, with their higher survival, increase their numbers much faster than mice. For both species, the lineage (pale stripes) that starts with an offspring placed into the population at $t = 2$ is thinner than the lineage that had its start at $t = 1$, but this difference is much more marked if population growth is (b) fast than if it is (a) slow. In (b) both stripes appear narrow, because the vertical scale has to differ between (a) and (b) to allow the entire bat population to be depicted.

Figure 2.

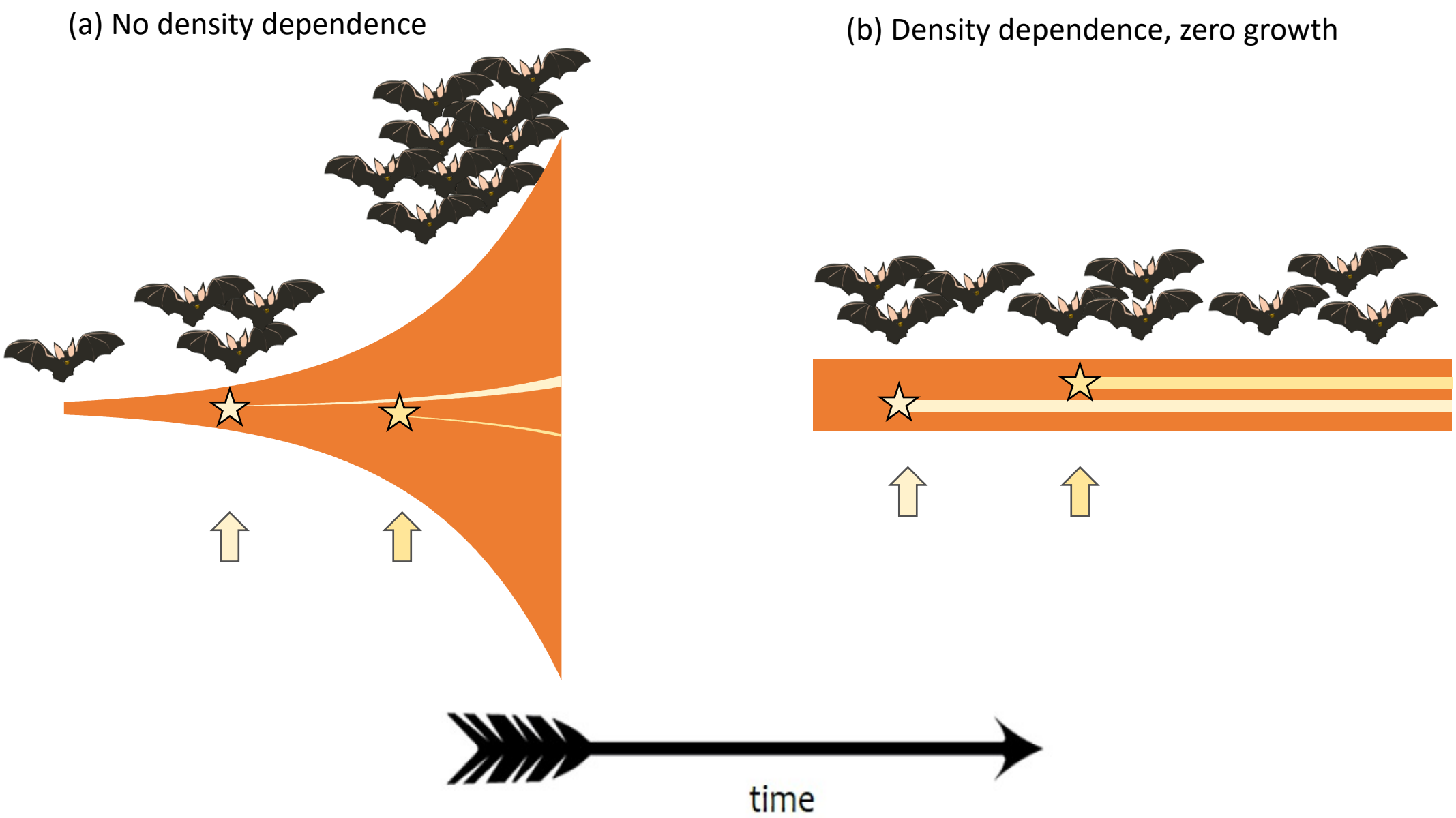

(a) No density dependence
(b) Density dependence, zero growth
time

Figure 2. A visualization of the difference between placing offspring early or late in an exponentially growing population (on the left) or in a stable population (on the right). In a growing population, the late placed offspring will be a smaller fraction of the future population than the early placed offspring. In a stationary population, there is no such discounting of early *vs.* late placed offspring.

Figure 3

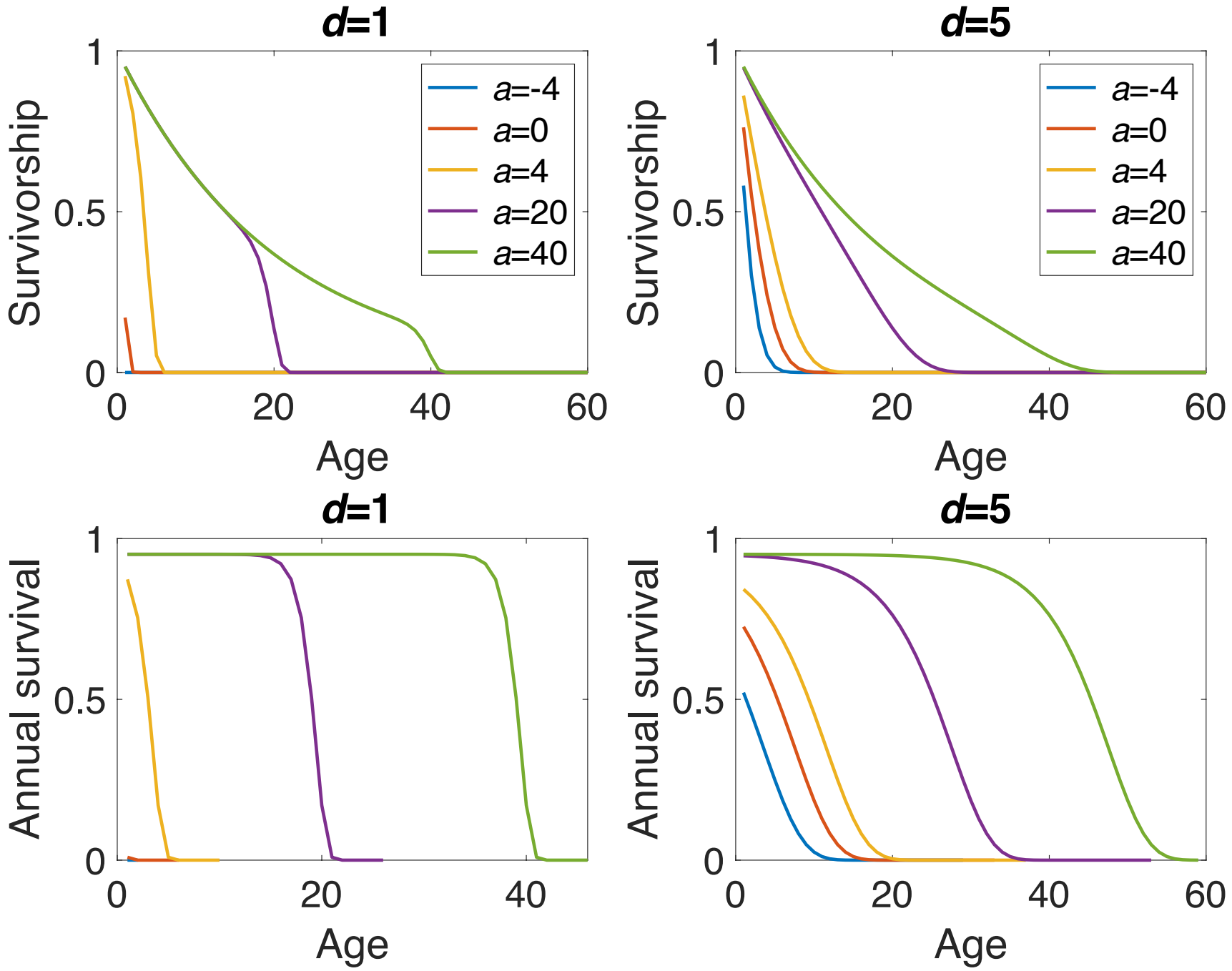
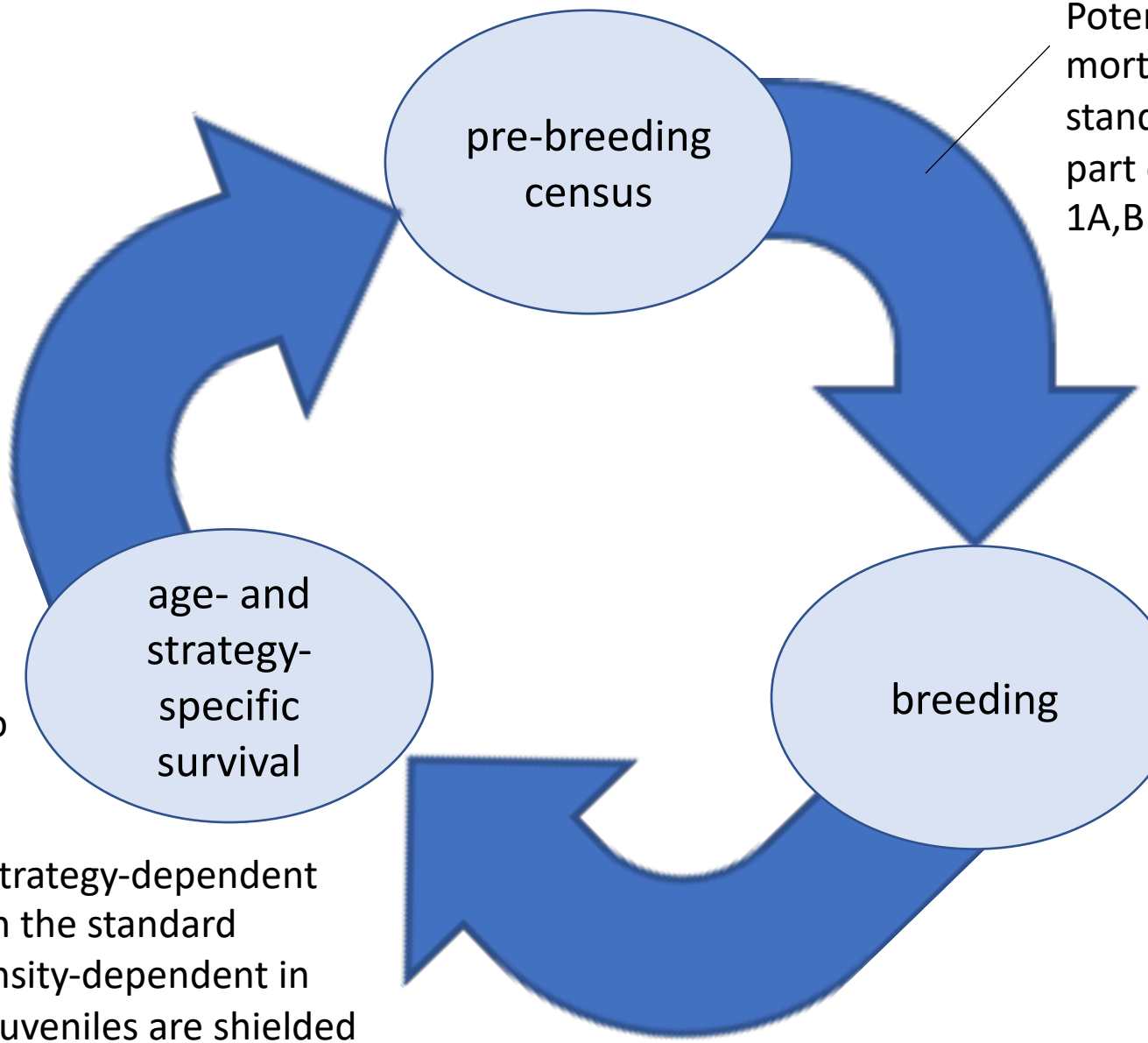

d=1
d=5
Survivorship
Annual survival
Age
a=-4
a=0
a=4
a=20
a=40
0
0.5
1
0
20
40
60

Figure 3. (a,b) Gompertz-Makeham survivorship function (the probability that an individual is still alive) and (c, d) age-dependent survival for example combinations of *a* and *d* as indicated. All examples use $\mu = 0.05$.



Potential pre-breeding mortality - not part of the standard procedure but part of regulation types 1A,B and 2A,B.

Fecundity is constant in the standard procedure but responds to density in regulation types 3A-C.

All survivors age by 1 unit. For juveniles this means becoming 1 year old and also maturing.

Survival values are age-and strategy-dependent but not density-dependent in the standard procedure. They become density-dependent in regulation types 1C and 2C; juveniles are shielded from being impacted in 2C; only newborns are impacted in 3D.

Figure 4: The standard procedure, describing a life cycle used to create the 10 different modes of density regulation. Note that there is no implication that completing each of the three arrows takes equally much time: in reality, census is immediately before breeding, to allow mortality rates the appropriately long time to apply before the next year. Generations are overlapping, therefore at each point the population will consist of individuals of different integer ages. Details about regulation are briefly summarized next to the loop; for full see details in Table 2.

Figure 5

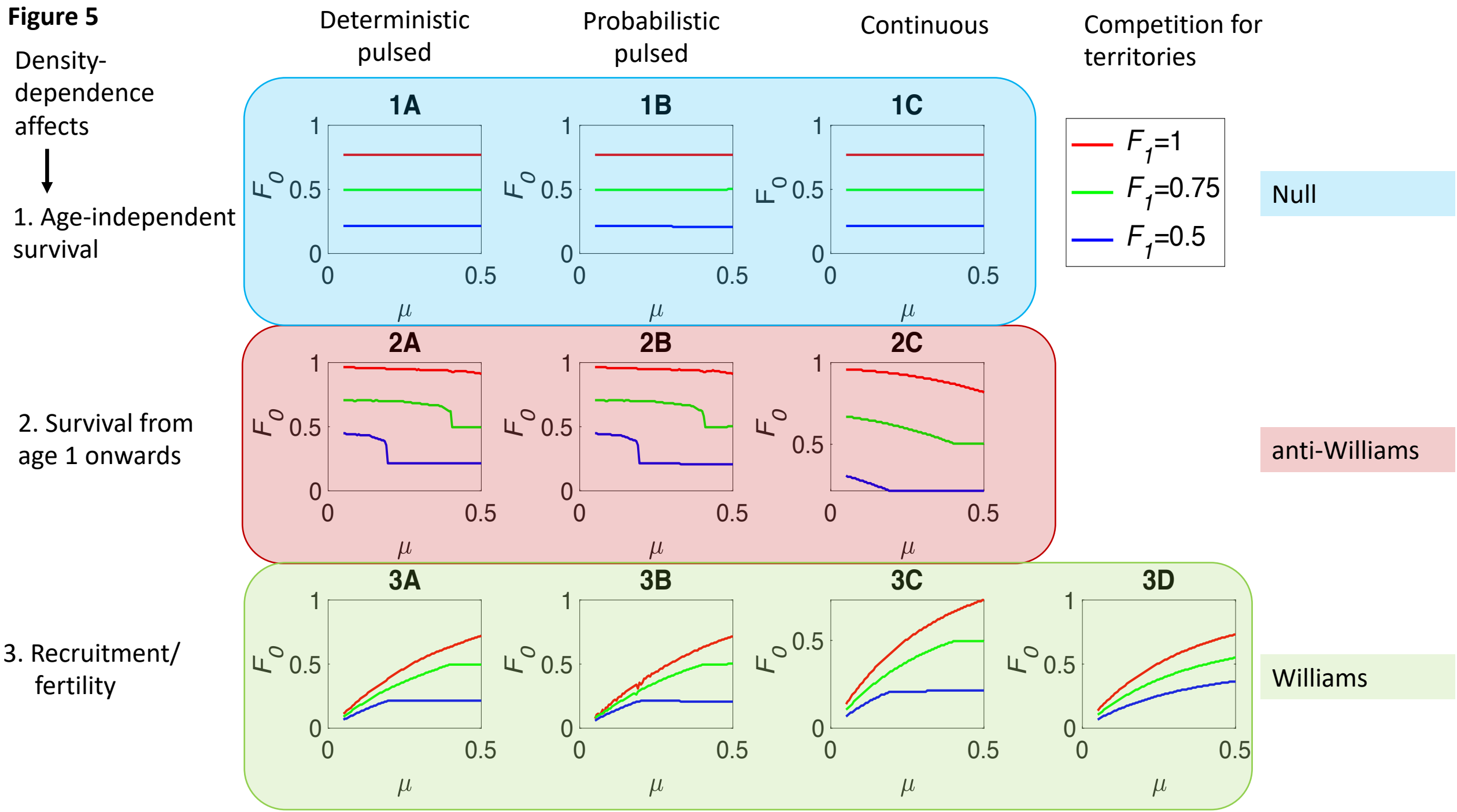

Deterministic pulsed
Probabilistic pulsed
Continuous
Competition for territories
Density-dependence affects
1. Age-independent survival
2. Survival from age 1 onwards
3. Recruitment/ fertility
1A
1B
1C
2A
2B
2C
3A
3B
3C
3D
F_0
μ
F_1=1
F_1=0.75
F_1=0.5
Null
anti-Williams
Williams

Figure 5. How much fertility does a non-senescing (slow) strategy need to beat the senescing (fast) strategy? The lines indicate a threshold fecundity F_0 for the slow life history: above this threshold slow types win, below this threshold the fast strategy wins. Parameters used for the Gompertz-Makeham (equation 3): *a*=4, *d*=1, *K*=100 000.