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Senescence and the Evolution of Life History and Evolvability

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Contents

Abstract

Organismal senescence appears paradoxical, as to the individual, it is a detrimental process, reducing its fitness. Nonetheless, it is widespread across the tree of life. One way to resolve this is by saying that individuals must die to give way to newer generations in order for evolution to occur. Senescence would thus be the mechanism by which this is achieved, making it inevitable. However, there are species that senesce at very different rates, some even appearing to be immortal. The question remains why species senesce at different rates and how different lifespans evolved.

Many theories exist on the evolution of senescence, ranging from mutation accumulation, antagonistic pleiotropy, and the disposable soma theory to the theory that senescence is beneficial by increasing evolvability and that it evolved through group selection. We propose that senescence evolved as a consequence of the evolution of a faster life history when there are trade-offs between life history traits.

To test this as a proof of principle, I used individual-based simulations in Wolfram Mathematica. I modelled five different life history scenarios where trade-offs were implemented as constraints on the reproductive output. Two scenarios had strong trade-offs, which meant that an increase in lifespan could not achieve more offspring. Two scenarios had intermediate trade-offs, where decreasing the lifespan also decreased the offspring number while increasing lifespan lead to more offspring. The fifth scenario had weak trade-offs. Increasing the lifespan also increased the offspring number, more so than for the two intermediate scenarios.

The simulations resulted in different outcomes for the age at death, depending on the scenario. While for the first two scenarios with strong trade-offs the age at death as well as reproductive onset always decreased, for the scenario with weak trade-offs, only age at reproductive onset decreased while age at death increased, thereby maximizing offspring number. The intermediate scenarios either showed a decrease in age at death or an increase, depending on the choice of parameters. However, the more offspring the individuals could potentially produce, the more age at death decreased. The reason for the decrease in age at death and reproductive onset was a direct contribution to fitness as well as an increase in evolvability, which allowed the selected trait x to evolve faster.

The simulations show that a faster life history evolves and increases evolvability of a population. When trade-offs between life history traits exist, such as between lifespan and offspring number, a faster life history also comes with a shortening of the lifespan. Indeed, many examples of such life history trade-offs are known. Therefore, it is possible that the various rates of senescence and the diversity of lifespans are a consequence of different life history strategies in distinct species.

German Abstract (Zusammenfassung)

Die Seneszenz von Organismen erscheint paradox, da sie die Fitness des Individuums mindert. Dennoch ist Seneszenz im Tierreich weit verbreitet, denn damit Evolution stattfinden kann, müssen Individuen sterben, um der nächsten Generation Platz zu machen. Arten können jedoch sehr unterschiedlich schnell altern. Es bleibt die Frage, warum Seneszenz so verschieden abläuft und wie sich unterschiedliche Lebensdauern entwickelt haben.

Theorien zur Evolution von Seneszenz reichen von Mutationsakkumulation, antagonistischer Pleiotropie und der „Disposable Soma“ Theorie bis hin zur Theorie, dass Seneszenz die Evolutionsfähigkeit steigert und sich durch Gruppenselektion entwickelt hat. Wir schlagen vor, dass sich Seneszenz als Folge der Evolution eines schnelleren Lebenszyklus entwickelt hat, wenn es Kompromisse zwischen Lebenszyklusmerkmalen gibt.

Um die Plausibilität dieser Idee zu überprüfen, habe ich individuenbasierte Simulationen in Wolfram Mathematica durchgeführt. Ich habe fünf verschiedene Lebenszyklusstrategien modelliert, in denen Kompromisse als Einschränkungen der Fortpflanzungsleistung implementiert wurden. Zwei Szenarien wiesen starke Kompromisse auf, was bedeutete, dass die Nachkommenzahl für alle gleich war. Zwei weitere Szenarien wiesen mittlere Kompromisse auf, während das fünfte Szenario schwache Kompromisse aufwies. In diesen Fällen führte eine Verkürzung der Lebensdauer zu einer verringerrung der Nachkommenzahl, während eine Verlängerung sie erhöhte. Diese Änderungen waren jedoch stärker bei dem Szenario mit starken Kompromissen.

Die Simulationen ergaben je nach Szenario unterschiedliche Ergebnisse für das Sterbealter. Während bei den Szenarien mit starken Kompromissen sowohl das Sterbealter als auch das Alter bei Erreichen der Fortpflanzungsphase stets abnahmen, sank beim Szenario mit schwachen Kompromissen nur der Beginn der Fortpflanzungsphase, während das Sterbealter anstieg. Die mittleren Szenarien zeigten je nach Wahl der Parameter entweder eine Abnahme oder eine Zunahme des Sterbealters. Je mehr Nachkommen die Individuen potenziell zeugen konnten, desto stärker sank jedoch das Sterbealter. Der Grund für die Abnahme des Sterbealters und des Beginns der Fortpflanzungsphase war ein direkter Beitrag zur Fitness sowie eine Zunahme der Evolutionsfähigkeit, wodurch die Evolution des Merkmals x schneller ablaufen konnte.

Die Simulationen zeigen, dass sich ein schnellerer Lebenszyklus entwickelt und dabei die Evolutionsfähigkeit einer Population erhöht. Liegen Kompromisse zwischen verschiedenen Merkmalen des Lebenszyklus wie Lebensdauer und Nachkommenzahl vor, so geht ein schnellerer Lebenszyklus auch mit einer Verkürzung der Lebensdauer einher. Viele Beispiele für solche Kompromisse im Lebenszyklus sind bekannt. Die Vielfalt der Alterungsraten und Lebensdauern in der Natur könnte somit eine Folge unterschiedlicher Lebenszyklusstrategien zwischen verschiedenen Arten sein.

Introduction

Different species exhibit a great variety of life strategies to ensure the propagation of their genes in the next generation. While some have a very short and fast lifecycle, others can take years to reproduce but in exchange they often live longer. The longest lifespan among vertebrates has been estimated for Greenland sharks, which can live up to several centuries (Nielsen et al., 2016), but the record holders in the animal kingdom are invertebrates such as sponges or cnidarians that can even live up to several thousand years (Jochum et al., 2012; Jones et al., 2014). On the other end of the spectrum are mayflies, where the adults usually live for only a few hours, making them one of the shortest-living animals (Leonard, 2026). This great diversity that we see in lifespans between species brings up the question how senescence, which influences the length of the lifespan, has evolved across the tree of life.

Life History Traits

Life history traits describe the timing and allocation of resources in processes such as growth, maintenance, and reproduction. They include, but are not limited to, traits such as neonate size, litter size, age at reproductive maturity, and lifespan. Since they affect the survival and reproductive success of an individual, they also directly contribute to fitness. To maximise one's fitness, all life history traits would ideally be optimised simultaneously as well. Thus, an individual that lives longer would also reproduce more often. However, this is neither feasible due to the restricted amount of energy and resources available to each individual, nor is it really observed in nature, where we often find that there are constraints on different life history traits (Fabian & Flatt, 2012; Flatt, 2020), so-called life history trade-offs. Specifically, there is a negative correlation between early reproduction and lifespan. Within a population or species, those individuals who have more offspring at an early age tend to reproduce less at a later age and live shorter. This implies that there is a cost of reproduction that does not allow individuals to live and at the same time reproduce endlessly.

Different species have adopted different life history strategies. Often, these are categorized along a fast-slow continuum. Species such as the house mouse have fast life histories. They have short life cycles, but produce many offspring early in life with little investment. On the other hand, species with slow life histories (for example, humpback whales) have fewer and later offspring but with higher investment (Jeschke et al., 2019; Stott et al., 2024).

Evolution of Evolvability

Evolvability describes the ability of a population to evolve, i.e., to adapt to selective pressures. Wagner (1981) has already shown theoretically that evolvability can change by becoming coupled to another trait under selection. We propose that certain life history traits, namely, the age at onset of reproduction (reproductive maturity) and the age at death or cessation of reproduction, can influence the evolvability of a population by decreasing the generation time and age at reproduction. In this thesis, evolvability is evaluated by the magnitude of change in a trait mean per unit of time. When a lineage reproduces earlier, it will produce more offspring and more variation in a given amount of time than another lineage where the individuals reproduce more slowly. Thus, the first lineage will have an advantage compared to the second because it can adapt faster and will have a higher fitness gain (see **Fig. 1**). The age at reproduction is itself a component of fitness here according to the Malthusian model. Additionally, (Mitteroecker, 2025) has shown in individual-based simulations that the age at reproductive onset becomes statistically coupled to another trait under selection. Individuals who have both a beneficial trait value as well as an early reproduction will have the highest rate of reproduction. Therefore, by producing offspring with a similar combination of alleles and phenotypes, the association between early reproduction and beneficial trait value is strengthened in the population. This will lead to an additional decrease in the age at reproductive onset, even though it is not directly under selection itself.

We hypothesise that also the age at reproductive halt (and thus death) would become coupled to the trait by increasing the evolvability. This is because if individuals reproduce more than once, then not only the onset of reproduction matters, but also the mean age when an individual reproduces over its entire reproductive lifespan. The earlier all of the offspring are born, not just the first one, the more evolvable the population becomes. The way to influence the mean age of reproduction is to either shift the onset of reproduction earlier or the end of the reproductive phase.

Importantly, what actually increases evolvability is the cessation of reproduction, and not the death of the individual. However, once an individual no longer reproduces, its reproductive value goes down to zero since it will no longer contribute to the next generation. Thus, there will be no selective pressure to keep such individuals alive for longer (Hamilton, 1966). Reproductive success and senescence indeed often go hand in hand (Croft et al., 2015), and few species live long beyond their reproductive age. This is true only for species where the parents in no way contribute to the survival and reproductive success of their offspring after giving

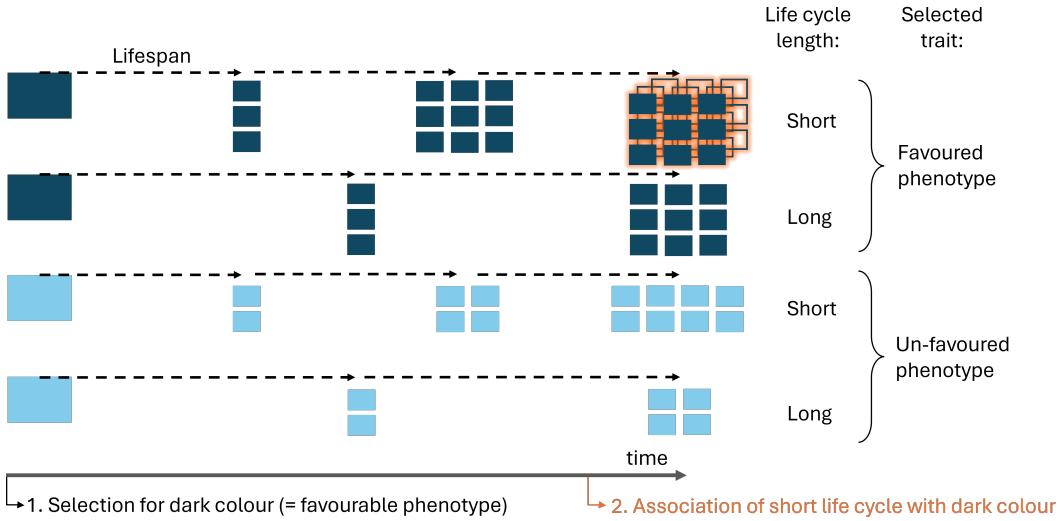

Fig. 1: Faster life cycles increase the population turnover, thereby accelerating the response to selection (= higher evolvability) and enhancing the overall fitness of the corresponding lineages.
Individuals are depicted as blue rectangles, with darker shades being favoured by selection (higher reproductive output: three vs. two offspring). In addition, lineages with shorter life cycles produce more generations per time, amplifying both the spread of advantageous phenotypes and overall reproductive output. This results in an increased co-occurance of these traits, further accelerating evolutionary change. This graphic best corresponds to Scenarios 1 and 2 (see **Methods** section).

birth to them (Roper et al., 2023). This assumption is, however, not always met, particularly in mammals, who often live in social groups. Indeed, menopause, the survival of the mother past her age of reproductive halting, can be found across various groups (Croft et al., 2015; Winkler & Goncalves, 2023). Here, age at death is delayed, possibly due to the higher inclusive fitness when the mother stays to support her offspring.

In this thesis, I will nonetheless consider the halting of reproduction and age at death as the same event, since in most species it is connected.

Senescence

Biological ageing, also referred to as senescence or organismal senescence, is a biological process that is almost ubiquitous in animals and other multicellular species. Even some unicellular organisms and prokaryotes exhibit certain signs of ageing (Steinkraus et al., 2008; Stewart et al., 2005). In an environment where resources are limited and where individuals compete against each other to represent their genes in the next generation, it is clear that everyone must die eventually, otherwise evolution would not be possible. This by itself, however, does not explain why we

see a gradual decline in physiological capabilities with age as well as an increasing mortality, as described for humans by the Gompertz law (Hansen & Strulik, 2025). It also does not explain why lifespans and the rate of ageing vary so strongly across the tree of life (Jones et al., 2014; Shefferson et al., 2017), or why some species do not seem to be exhibiting any form of ageing (negligible senescence) or even show "negative senescence" (Jones et al., 2014; Yun, 2021). Although the environment has a strong influence on the lifespan, there are also genetic, heritable contributions (Pettay et al., 2005; Shenhar et al., 2026). Thus, evolution can act on this trait too.

While it is difficult to define ageing, particularly across phyla or kingdoms, there are some hallmarks that are widely associated with it, particularly in mammals (López-Otín et al., 2023). These include primary hallmarks, such as increased genomic instability or shortening of telomeres, antagonistic hallmarks, e.g. cellular senescence, which have both beneficial as well as deleterious effects depending on the context, and integrative hallmarks, e.g. stem cell exhaustion, which result from an accumulation of changes in the other hallmarks. The definition of the hallmarks relies mostly on a molecular or cellular view. From an evolutionary perspective, ageing is usually referred to as senescence and it is viewed as a reduction in the fitness gain of an organism with age. This can happen through an increase in mortality with age, or a decrease of reproductive success. In this thesis, I will be using the word senescence to refer to biological ageing from this point onwards, since I am modelling the evolution of ageing. To represent ageing, I use the time of death, which coincides with the cessation of reproduction. Ageing on lower levels (e.g., cellular or molecular levels) is not directly included in the model, though of course they contribute to this more systemic phenotype.

Theories on the Evolution of Senescence

Theories on the evolution of senescence can broadly be put into two main categories. Some theories ascribe senescence to an inevitable accumulation of mutations as an organism ages. Here, senescence is viewed as a deleterious process, which results from physical or chemical constraints. The second group of theories sees senescence as a beneficial process, because it increases the evolvability of the species and gives space to the next generation. In this second group of theories, it is mostly theorized that senescence comes about through group selection. I will describe the most important existing theories in more detail below.

Medawar (1952) first described senescence as the effect of an accumulation of mutations that act in late age. The underlying idea is that, even if we assume that the individuals of a population are potentially immortal and there is no senescence,

there is a certain probability, that any one individual dies. This can be due to predation, diseases, or an accident like falling off a tree or being hit by lightning. Because of this, the number of older individuals in a population will on average always be fewer than the number of younger individuals, simply because they have had more possibilities to die by chance. Since selection works more efficiently the more individuals there are, as their contribution to the next generation is also larger, deleterious mutations that only have an effect in late age will not be removed from the population as efficiently. Consequently, such mutations will accumulate, causing the age-associated decline.

This idea was extended by Williams (1957), proposing that antagonistic mutations can additionally accumulate, due to the reduced selection pressure with age. Antagonistic mutations are beneficial at a young age and deleterious in late age. Even if the early beneficial effect is very small, it can counterbalance the negative effect of the mutations as long as that occurs sufficiently late in life.

This theory is supported by the long lifespan exhibited by cave-dwelling or arboreal species, which are better protected from predators and are therefore less likely to die early (Healy et al., 2014; Moorad & Promislow, 2010; Shattuck & Williams, 2010; Voituron et al., 2011). Due to the lower extrinsic mortality, age-dependent deleterious mutations only act later in life, delaying also the development of senescence. Experiments in fruit flies have further shown that by exposing flies to a high adult mortality treatment, the flies evolved an earlier high intrinsic mortality compared to flies with low adult mortality (Stearns et al., 2000). Further evidence for the antagonistic mutation theory comes from alleles that have an antagonistic effect depending on age. Such is for example APOE-ε4, which has an early beneficial effect by increasing fecundity, but it also increases the risk for dementia and cardiovascular diseases later in life (Trumble et al., 2023). Cellular senescence is sometimes mentioned as an example for early benefit by preventing cancer, but negative effects in late age due to reduced regenerative capabilities. It is, however, disputed whether the beneficial and deleterious effects are indeed separable by age so that it could support the antagonistic mutation theory (Giaimo & d'Adda Di Fagagna, 2012). Other more recent studies have also dismissed the basal assumption that selection strength decreases with age as being wrong (Giaimo & Traulsen, 2022).

Another theory that sees senescence as a deleterious process is the disposable soma theory by Kirkwood (1977). In this theory, senescence results from a compromise of energy and resource allocation. An individual needs to distribute its resources between the germline, responsible for reproduction, and the soma, which maintains the organism but is indirectly also required for reproduction. When an

organism invests more into reproduction, thereby increasing its fitness defined as the number if offspring, there will not be enough energy left for its own maintenance. Investing more into the soma, however, would have the effect of reducing fitness, since fewer offspring could be produced. The theory is connected to the mutation accumulation theory in that it states that the soma will be maintained as long as the cumulative probability of death from external sources is low. Once the probability of having died by a certain age becomes sufficiently large, there is no reason to invest energy into maintenance beyond that point. Thus, only as much energy for maintenance of the soma will be expanded and the rest is invested into the germline.

The second group of theories, that senescence is an advantageous process (Goldsmith, 2017), is based on findings that show that senescence is programmed and that lifespan can actually be extended by interfering with those processes (Pamplona et al., 2023). One example is caloric restriction, which prolongs lifespan in different organisms ranging from yeast through flies to mice by interfering with the insulin/IGF-1 pathway (Fontana et al., 2010). Pamplona et al. (2023) see support for this theory through several pathways that contribute to earlier senescence and through the wide phylogenetic distribution of these. Also in support of a programmed senescence is that some species seem to be lacking it, such as the hydra (Jones et al., 2014), which cannot be explained by the non-adaptive theories. Although Goldsmith (2017) suggested that senescence evolves because it increases the evolvability of a population, the evolutionary mechanism proposed has mostly been group selection, the functioning of which is strongly disputed and likely only occurs in very special circumstances.

A recent study has shown through simulations that senescence can evolve by being beneficial and selected for without requiring group selection. However, this model still requires kin selection and a specific spatial distribution of individuals in the population to work (Szilágyi et al., 2023).

We propose that senescence evolved as the implementation of a faster life history, meaning, that individuals complete their life cycle faster by starting and ending the reproductive phase earlier. The faster life cycle itself would evolve because it increases the evolvability of a population and contributes to fitness (see **Fig. 1**). To show this, individual-based simulations were done, where different life strategies with different levels of trade-offs and constraints between the life history traits were compared. Trade-offs were included between age at reproductive maturity (onset of reproduction), number of reproductive events, and lifespan (age at death) to see under which conditions senescence would evolve, demonstrated by a forward shift of age at death.

Since evolvability describes the ability to evolve, one way to think about it is as the rate of adaptive evolutionary change. To see how changes in the life cycle affect evolvability, the simulated populations were under directional selection, where the rate of change in the selected trait was used to qualitatively estimate evolvability.

Methods

General Structure of the Simulations

The individual-based simulations were written in Wolfram Mathematica (v12.1). Five different scenarios were considered (see **Fig. 2**) under directional selection, each one both for asexually and sexually reproducing populations:

1. Strong life history trade-offs:

   ➢ Age at death and reproductive onset are completely coupled due to the constraint of the reproductive lifespan R (time between reproductive onset and death) being the same for all individuals. As the reproductive interval r (time that needs to pass between two consecutive possible reproductive events) is invariable as well, the number of offspring is not affected by a shift of the reproductive lifespan.

2. Strong life history trade-offs:

   ➢ Age at reproductive onset and age at death can vary independently of one another. However, the reproductive interval scales with the reproductive lifespan. Therefore, an increase in reproductive lifespan R does not change the ratio $\frac{R}{r}$, thus keeping the number of offspring unchanged.

3. Intermediate life history trade-offs:

   ➢ Age at reproductive onset changes in the same direction as age at death, meaning, if age at death increases, so does age at reproductive onset. However, age at reproductive onset only shifts by half the distance of the change in age at death. E.g., if age at death decreases by 0.2, age at reproductive onset decreases by only 0.1. Thus, the reproductive lifespan can vary while the reproductive interval remains the same. This allows more offspring to be born when death occurs later.

4. Intermediate life history trade-offs:

   ➢ Age at reproductive onset and age at death can vary independently of one another. When the reproductive lifespan changes, the reproductive interval changes in the same direction. However, the reproductive interval only scales with half the change in the reproductive lifespan. E.g., if the reproductive lifespan decreases by 10 %, the reproductive interval

decreases by only 5 %. This allows more offspring to be born when age at death goes up and increases the reproductive lifespan.

5. Weak life history trade-offs:

   ➢ Age at reproductive onset and age at death can vary independently of one another. The only constraint is that the reproductive interval is invariant. As the age at death increases, the reproductive lifespan is increased as well and thus also the number of offspring.

For the first two scenarios, where there is a strong trade-off, the possible number of reproductive events and thus offspring is constant. One cannot increase the number of offspring by extending ones lifespan. This is different from the fifth scenario, where the age at reproductive maturity and the age at death are completely independent. Due to a constant interval between two reproductive events, a longer reproductive lifespan also allows more offspring to be produced. The third and fourth scenarios also allow more reproductive events with an extended lifespan. However, for a given reproductive lifespan, the number of additional reproductive events is lower than for the fifth scenario.

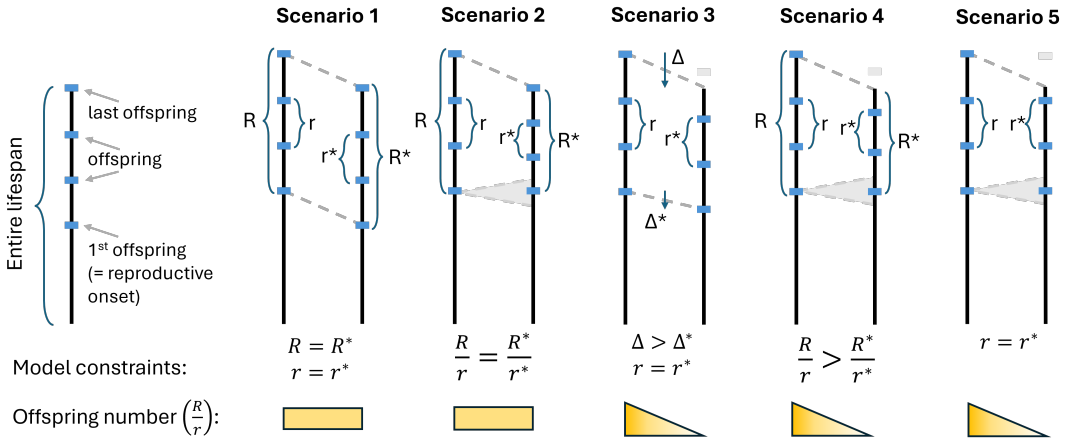

Fig. 2: Five alternative life history scenarios and how their model-specific constraints affect reproductive dynamics when age at death is reduced.
The reproductive lifespan R (or R^* after reduction) is defined as the time between age at reproductive onset (first possible reproduction) and age at death. Reproductions occur at discrete time points in an individual's life, separated by the reproductive interval r (or r^* after adjustment). Across scenarios, reductions in age at death alter the reproductive lifespan and/or reproductive interval depending on the imposed trade-off. For Scenario 3, a reduction in age at death by for example $\Delta = 0.2$ leads to a correlated but attenuated reduction in age at reproductive onset ($\Delta^* = 0.1$). For Scenario 4, a reduction in the reproductive lifespan by for example 10 % leads to a 5 % reduction in the reproductive interval.

The simulations were run for 100 time units, which corresponds to 100 generations with the initial parameters. After every 0.1 units of time, the population was evaluated. First, all individuals who fulfilled the selection conditions would reproduce with a certain probability depending on their selected trait value. After that, individuals who have reached their age at death were removed from the population. When necessary, further individuals were randomly removed to keep the population size at 5000.

Assumptions

In the simulations, we assumed that all the traits (selected phenotype x, age at reproductive onset, age at death) were quantitative traits and followed a normal distribution. Further, the traits had a heritability lower than one. This was implemented by separately tracking the genetic and environmental contributions of the trait. For the sexually reproducing populations, we assumed a hermaphroditic population with random mating among those individuals who were permitted to reproduce at the evaluated timepoint. We did not assume any type of population structure or kin selection, and we did not model interactions between the individuals beyond the reproductions in the sexually reproducing populations.

Starting Population

The simulated populations started with 5000 individuals. The age of each individual could be calculated from its time of birth $N(\mu = -0.5, \sigma^2 = 1.69)$. Further, there were three traits, each with a separately modelled genetic and an environmental component: trait $x = g_x + e_x$ with the genetic component $g_x \sim N(1, \sigma^2_{x,g})$ and the environmental component $e_x \sim N(0, \sigma^2_{x,e})$; age of reproductive onset with $g_{reprod} = N(1, \sigma^2_{reprod,g})$, $e_{reprod} = N(0, \sigma^2_{reprod,e})$; and age at death with $g_{death} = N(2, \sigma^2_{death,g})$, $e_{death} = N(0, \sigma^2_{death,e})$. The reproductive lifespan was the time between the onset of reproduction and death of the individual. During this time, individuals reproduced on several occasions, when a certain time (the reproductive interval) had passed since the last reproduction event.

Selection Types

The population was experiencing two selection pressures, which were indirectly antagonistic. One selective pressure was favouring a larger trait x value. The fitness, or probability to reproduce, was a linear function of the trait x. The linear relationship

was computed in the following way:

$$p_i = s\frac{(x_i - \overline{x})}{\sigma_x} + p_r$$

Here, p_i was the probability that the individual i will reproduce at the evaluated timepoint. The trait value x_i of the individual i was made up of a genetic component ($g_{x,i}$), which was inherited from the parent, and an environmental component ($e_{x,i}$). To account for mutations and recombination, the genetic component was drawn from a normal distribution centered around the parental genetic component. The environmental component was added to the inherited value and was also drawn from a normal distribution. We computed the z-score for each individual regarding the trait x: only the difference between the trait value and the mean trait value in the population $\overline{x}$ was considered, and we normalized the difference with the standard deviation of the population, σ_x. To determine the probability with which each individual should reproduce, we scaled the z-transformed mean deviation with the selection strength s. This scaling corresponds to the strength of selection, because the larger the variation in a trait, the more efficient selection is (Price, 1972). Thus, modifying the spread of the distribution changes the efficiency of selection. Finally, we shifted the individual reproduction probability by p_r, the average probability of reproductive success at a single reproduction event, so that the probabilities of reproduction mostly fell within the interval [0,1]. If selection strength was chosen too high or too low relative to the spread of the trait, individuals on the edge of the distribution fell outside of the range from zero to one. This lead to a non-linear function for the probability to reproduce, as Mathematica set p_i to 0 then.

The second type of selection was on the age at reproductive onset. Reproduction could not start before an individual has fully developed to reproductive maturity, and the time it took to develop could not become arbitrarily short. Since too early reproduction is harmful to both the parent and the offspring (Hill & Kaplan, 1999; Stearns, 2000; Stevens-Simon et al., 2002), there was a selective pressure against very early reproduction. This selection did not permit individuals aged below a minimal critical threshold (r_{min}) to reproduce at all. Above the minimal threshold, but below a second threshold ($r_{healthy}$) where reproduction was possible but with reduced efficiency, the probability to reproduce depended linearly on the individual's age:

$$q_i = \frac{a_i - r_{min}}{r_{healthy} - r_{min}},$$

where q_i was the probability of individual i to reproduce, based on its age a_i alone at the time of evaluation.

Individuals who were above the age where reproduction could be harmful could reproduce without constraints, *i.e.*, their probability to reproduce based on age alone was 1.

To determine which individuals were able to reproduce at the time of evaluation, they needed to fulfill the following conditions:

- An individual must have reached its age of reproductive onset (reproductive maturity reached).
- All following reproductions could only occur at timepoints that were multiples of the reproduction interval; the reproduction interval describes how much time needed to pass since the previous reproduction (e.g., time between fertilization and giving birth).
- Based on the selective forces, an individual needed to be eligible. Eligibility was determined by calculating the total probability to reproduce based on the selection pressures. Since the selective pressures were independent of each other, the probabilities were multiplied. An individual reproduced based on a pseudorandom choice (resembling a Bernoulli trial) with the probability of success, i.e. reproduction, being the total probability to reproduce: $p_i q_i$.

Inheritance

For all traits, a genetic component, which was inherited by the offspring, as well as an environmental component were modelled. The environmental component followed a normal distribution and was not inherited but was computed independently for every individual at birth.

For the asexually reproducing populations, in each generation, the offspring inherited the genetic components from the parent. For the sexually reproducing populations, they inherited the mean parental value. In both population types, a certain amount of variation, which was drawn from a normal distribution, was added to the inherited genetic components. This variation represents variation occurring due to mutations, and, in the case of the sexually reproducing populations, recombination.

By including the environmental component, we could model traits with a heritability below 1. The narrow-sense heritability describes the ratio of the additive genetic variance to the total phenotypic variance: $h^2 = \frac{G}{P}$. While in theory the heritability can take on any value between 0 and 1, most traits have heritabilities between 0.2 and 0.8 (Mousseau & Roff, 1987; Pazokitoroudi et al., 2021; Roff & Mousseau, 1987; Visscher et al., 2008) and a recent study determined the heritability of lifespan to be 0.5 (Shenhar et al., 2026).

The heritability influences the efficiency of selection. As described by the Breeder's equation: $R = h^2 S$, the response to selection can be increased either by increasing the selection strength S, or the heritability h^2 (Falconer & Mackay, 1996).

Simulation Parameters

The simulations contained the following parameters:

- Mean trait value, $\overline{x}$, in starting population
  - This value was always set to 1. It was arbitrarily picked, as the precise value was irrelevant to the simulations, since only the relative values between individuals mattered in the selection process.
- *genetictraitvar* $\sqrt{V_{g_x}} = \sigma_{x,g}$
  - Standard deviation of the normal distribution from which the genetic component of the trait x in the starting population was drawn. The exact value by itself was also arbitrary, as long as it was a lot smaller than the mean value of the trait, so that no individual was assigned a negative trait value. The larger this value is, the stronger is the effect of selection because there is a larger variation of trait values in the population (Price, 1972).
- *envtraitvar* $\sqrt{V_{e_x}} = \sigma_{x,e}$
  - Standard deviation of the normal distribution from which the environmental component of the trait x was drawn and which was added to the genetic component. The absolute environmental contribution to variation remained constant over generations. The value was chosen so as to achieve a certain heritability for the trait x:

$$\sqrt{V_{e_x}} = \sqrt{\frac{V_{g_x}}{h^2} - V_{g_x}}$$

- *traitmutvar*
  - Standard deviation of the normal distribution from which the genetic variation of the trait x was drawn and then added to the inherited genetic component in each generation. It was chosen so that a roughly constant genetic variance was maintained. Therefore also a constant ratio of genetic and environmental variance and thus heritability was kept

across time. It represents the newly created variation through mutations and recombination.

- Mean age of reproductive onset in the starting population (*startingreprodonset* for Scenarios 3 and 4)
  - This value is arbitrarily chosen to be 1.
- *geneticagevar*
  - Standard deviation of the normal distribution from which the genetic component of the age at reproductive onset in the starting population was drawn. Same rules applied as to *genetictraitvar*.
- *envagevar*
  - Standard deviation of the normal distribution from which the environmental component of the age at reproductive onset was drawn and then added to the genetic component. Same rules applied as to *envtraitvar*.
- *agemutvar*
  - Standard deviation of the normal distribution from which the genetic variation in the age at reproductive onset was drawn and then added to the inherited genetic component in each generation. Same rules applied as to *traitmutvar*.
- *reprodlifespan* (*defaultreprodlifespan* for Scenario 3; *startingreprodlifespan* for Scenario 4)
  - The reproductive lifespan was only used in the simulations of Scenario 1 and 3 where the age at death was (variably) coupled to the age at reproductive onset, thus death occurred at a fixed or determined time after reproduction started. The value was chosen so that several reproductive events could occur in an individuals reproductive lifespan. In all scenarios, the value was chosen to be 1. For Scenario 4, this parameter was not used to couple age at reproductive onset and death, but to calculate the individual reproductive interval, as it was coupled to the individual reproductive lifespan.
- Mean age of death in the starting population

  - This parameter was used only in the Scenarios 2, 4 and 5. It was arbitrarily set to 2.

- *geneticlifespanvar*

  - Standard deviation of the normal distribution from which the genetic component of age at death in the starting population was drawn. Only used for Scenarios 2, 4 and 5, where the age at death was not coupled to the reproductive onset. Same rules applied as to *genetictraitvar*.

- *envlifespanvar*

  - Standard deviation of the normal distribution from which the environmental component of age at death was drawn and then added to the genetic component. Only used for Scenarios 2, 4 and 5. Same rules applied as to *envtraitvar*.

- *lifespanmutvar*

  - Standard deviation of the normal distribution from which the genetic variation in age at death was drawn and then added to the inherited genetic component in each generation. Only used for Scenarios 2, 4 and 5. Same rules applied as to *traitmutvar*.

- *reprodinterval* (*defaultreprodinterval* for Scenario 4)

  - This parameter describes the time that needs to pass between two potential reproductive events. It was predefined only in the Scenarios 1, 3, and 5. It was chosen arbitrarily, but should be small enough to permit multiple reproductions during the reproductive lifespan. It should not be smaller than the time steps when the population is being evaluated ($\Delta t = 0.1$).
  - For Scenarios 2 and 4, the interval between two reproductive events was computed individually, depending on the individual's reproductive lifespan, to accommodate a certain number of reproductive events (see *offspringnumber*).

- *offspringnumber* (n_o)

  - This parameter was used only for the Scenario 2. It counted the number of possible reproductive events for an individual. It was chosen arbitrarily, while considering that it should be more than 1 but not so high that an

individual could reproduce at almost every evaluated timepoint ($\Delta t = 0.1$), since that would not have permitted a shortening of the reproductive lifespan.

- *probreprod* (p_r)
  - Probability of any one individual to reproduce at any single reproductive event, before considering selection. It describes the mean success of reproduction. The probability was chosen from the interval (0;1), though it should not be too close to the edges, so that there are no individual probabilities p_i below 0 or above 1, once the reproductive distribution after considering selection comes on top of this.
- *selstrength* (s)
  - This was the standard deviation of the distribution of individual probabilities to reproduce, p_i, after considering also selection. It should be less than $\frac{1}{3}\min(p_r, 1-p_r)$ so that it is unlikely that probabilities < 0 occur. If the distribution is too wide, individuals on the edge of the distribution fall outside of the range from 0 to 1. When that happens, Mathematica assigns those individuals a probability of 0, which means that the selection gradient will no longer be entirely linear, but will be partially resembling truncation selection.
- *h2trait*, *h2reprodonset*, *h2deathage*
  - These are the heritabilities of the trait x under selection, the age at reproductive onset and the age at death, respectively. The heritabilities are chosen arbitrarily, but within a reasonable range, typically $0.2 < h^2 < 0.8$ (see Mousseau and Roff, 1987; Pazokitoroudi et al., 2021; Roff and Mousseau, 1987; Shenhar et al., 2026; Visscher et al., 2008).
- *noreprod* (r_{min})
  - At this age or lower, reproduction is not possible.
  - In all simulations, the value is arbitrarily defined as 0.3, but changing the value does not influence the direction of changes just the lower boundary of age at reproductive onset and age at death.
- *minagehealthyreprod* ($r_{healthy}$)
  - Age below which reproductive success is reduced.

– In all simulations, the value is arbitrarily defined as 0.5, but changing the value does not influence the direction of changes just the lower boundary of age at reproductive onset and age at death.

Reference Simulations

In the main simulations, the trait x was under selection, while the age at reproductive onset and death were free to change within the constraints imposed by the respective scenario. As a control to show that the effects observed in the simulations are stronger than expected without correlation and synergistic effects, reference simulations were included under two different conditions. The first condition served as a reference for the trait x. Here, changes in the trait x were modelled under directional selection while keeping the age at reproductive onset and death constant across individuals as well as across time. This meant that all individuals reached reproductive maturity at the age of 1 unit of time and died at the age of 2. The second condition provided the references for the age at reproductive onset and death. The changes in these traits were modelled while they were free to change within the constraints of the respective scenario, however, there was no selection on the trait x, which was exposed only to drift.

Results

The simulations from the different scenarios resulted in trajectories along a continuous spectrum, both for asexually and sexually reproducing populations (see **Fig. 3**). Differences were mostly in the magnitude and rate of change for the traits.

For asexually reproducing populations, the scenarios with strong life history trade-offs (Scenarios 1 and 2) showed a strong decrease in both age at reproductive onset and age at death (see **Fig. 4**, see **Table 1** for parameters). Initially, the decrease was fast, but it plateaued once the age at reproductive onset approached the value of 0.5. This shift to earlier reproduction was present to almost the same degree in the reference simulation as well, where there was no selection on the trait x. The selected trait x, on the other hand, showed a much stronger increase in the main simulation than in its reference simulation, where the trait was under selection but the age at reproductive onset and death were kept constant over time.

For the two scenarios with intermediate trade-offs between the life history traits (Scenarios 3 and 4), the results were also intermediate (see **Fig. 5**). Whether age at reproductive onset and age at death increased or decreased, and how strong the change would be, depended on the choice of parameters (**Fig. 6**). With a higher number of potential offspring, age at death was more likely to decrease. For Scenario 3, the coupling of age at reproductive onset and age at death meant that the reproductive onset also decreased in those cases, however, it increased if age at death increased. For Scenario 4, the age at death usually increased initially, and only decreased later. It also did not decrease as often or as strongly as in Scenario 3. The age at reproductive onset, however, always decreased and it did so stronger than how the age at death increased.

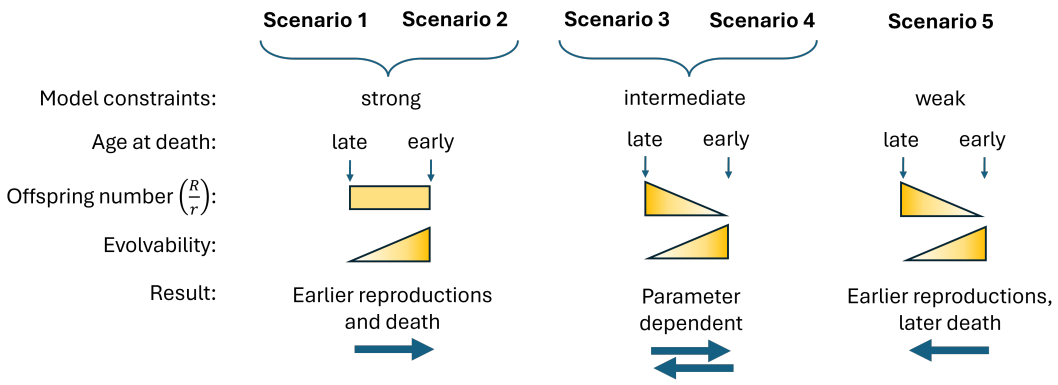

Fig. 3: Summary of simulation results.
The results suggest that lifespan is not an independent target of selection, but an emergent property of life history–dependent constraints and evolvability.

Parameter name	Asexual population	Sexual population
traitmutvar	0.021	0.08
geneticstraitvar	0.12	0.12
h2trait	0.7	0.7
agemutvar	0.06	0.08
geneticagevar	0.4	0.12
h2reprod	0.7	0.7
startingreprodonset	1	1
reprodlifespan	1	1
defaultreprodlifespan	1	1
startingreprodlifespan	1	1
lifespanmutvar	0.06	0.08
geneticlifespanvar	0.4	0.12
h2deathage	0.7	0.7
reprodinterval	0.3	0.3
defaultreprodinterval	0.3	0.3
offspringnumber	4	4
probreprod	0.5 (0.3, 0.4, 0.6)*	0.5 (0.3, 0.4, 0.7)*
selstrength	0.06	0.06
noreprod	0.3	0.3
minagehealthyreprod	0.5	0.5

Table 1: Parameters for simulations.
For all scenarios of one population type (asexually or sexually reproducing), the same parameter values were used. Differences between the asexually and sexually reproducing populations are highlighted in orange. The *probreprod* values labelled with an asterisk were used only for the different cases of Scenarios 3 and 4 depicted in **Fig. 6** and **Fig. 11**.

As in the first two scenarios, the references for the age at reproductive onset and age at death changed in the same direction as in the main simulation. The difference between the main and the reference simulations was largest, when the age at death increased, in which case the reference age at death would increase a lot stronger. The behaviour of the selected trait x in the main simulation compared to the reference also depended on the behaviour of the two life history traits. For Scenario 3, the trait would increase stronger than the reference when the age at reproductive onset and death decreased. An increase of the age at reproductive onset and death, however, caused the trait x to increase slower than the reference. For Scenario 4, the trait x always increased stronger than the reference, however, the difference was never as large as for Scenarios 1 or 2.

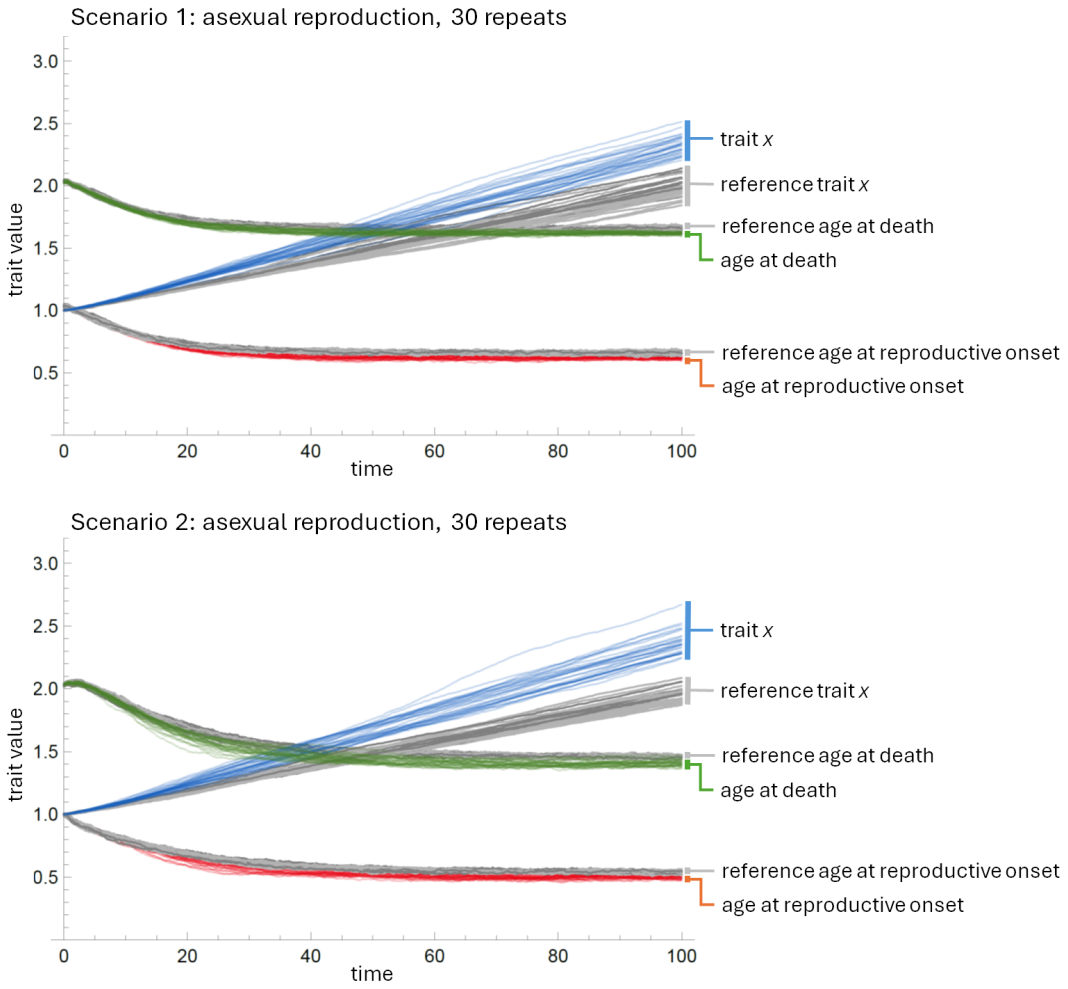

Fig. 4: Trajectories of the selected trait x, age at reproductive onset, and age at death for 30 replicates of the scenarios with strong trade-offs for asexually reproducing populations.
Each plot shows 30 independent simulation replicates for Scenario 1 (top) and Scenario 2 (bottom). The x-axis depicts units of time, while the y-axis depicts trait values (arbitrary unit). Both age variables are expressed in the same arbitrary time units used for the x-axis. Trajectories represent population mean values (N = 5000) over 100 time units. The population trajectories are shown under the three conditions for each scenario: (1) selection + flexible life history (blue/red/green) - directional selection acts on trait x (blue), while age at reproductive onset (red) and age at death (green) are free to evolve, (2) selection + fixed life history (grey, trait reference) - directional selection acts on trait x (reference for trait x, grey), while age at reproductive onset and age at death (not shown) are fixed at ages 1 and 2, respectively, (3) no selection + flexible life history (grey, life history reference) - trait x is not under selection (not shown, fluctuates around starting value), while age at reproductive onset (reference for reproductive onset, grey) and age at death (reference for age at death, grey) evolve freely.

Finally, the scenario with weak life history trade-offs (Scenario 5) was at the other end of the continuous spectrum. Here, the age at reproductive onset always went decreased, rarely even going below 0 (see **Fig. 7**). The age at reproductive onset

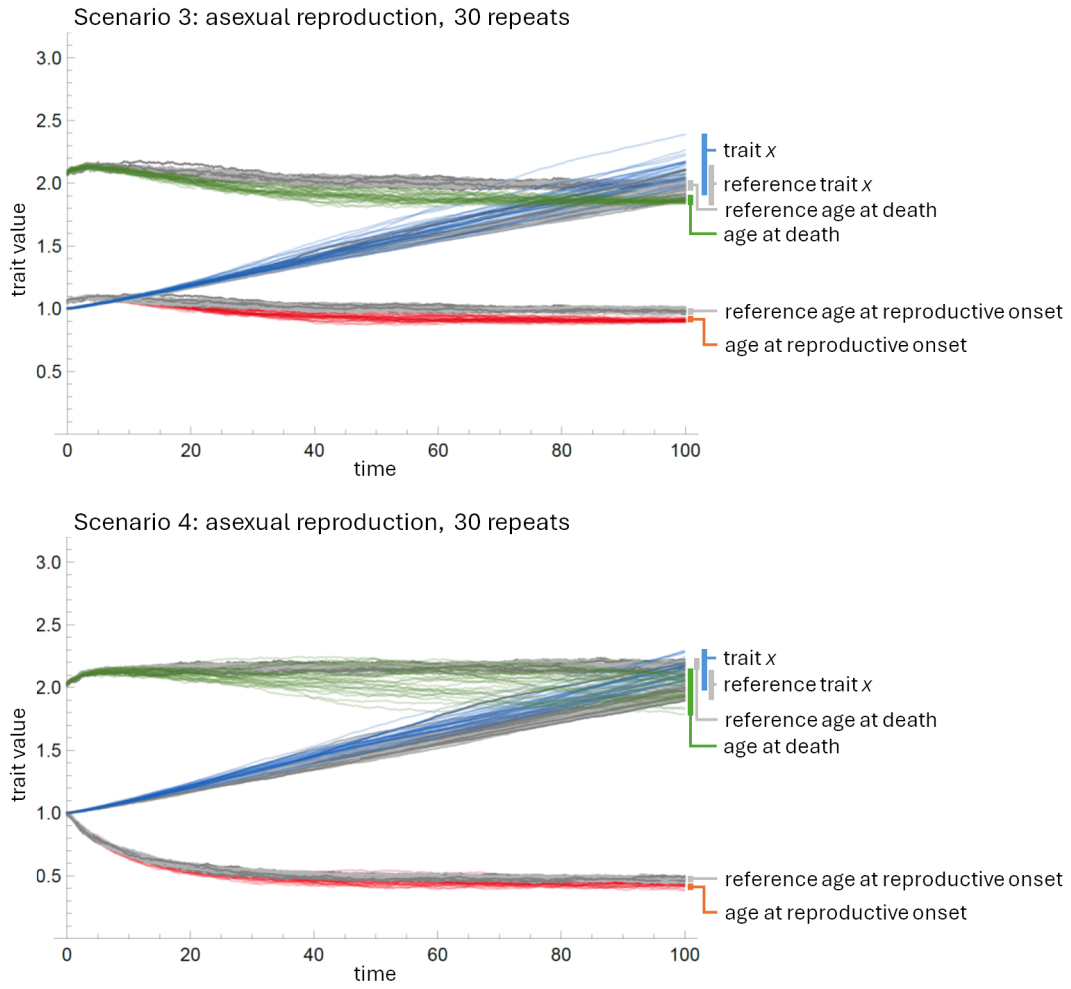

Fig. 5: Trajectories of the selected trait x, age at reproductive onset, and age at death for 30 replicates of the scenarios with intermediate trade-offs for asexually reproducing populations.
Each plot shows 30 independent simulation replicates for Scenario 3 (top) and Scenario 4 (bottom). All else is the same as for **Fig. 4**.

refers to the genetically determined age from when it becomes theoretically possible to reproduce, it is not necessarily the actual age when an individual reproduces, as there is a further constraint that does not allow too early reproduction. Unlike in the other scenarios, the age at death always increased, though it also slowed down over time. While the reference for the age at reproductive onset always behaved like in the main simulation, the reference for the age at death increased stronger than age at death with selection. Similarly to Scenario 4, the trait x did increase faster than its reference with constant age at reproductive onset and death, but the difference was smaller than for Scenarios 1 and 2.

For all five scenarios, the correlation between the selected trait x and the age at death (or age at reproductive onset) was usually negative, even when the age

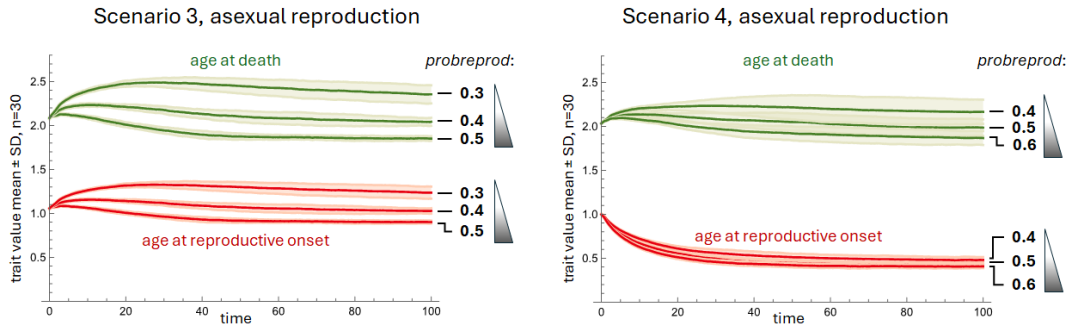

Fig. 6: Age at reproductive onset and death depending on the mean probability of successful reproduction for Scenarios 3 and 4 for asexually reproducing populations. Simulations for Scenarios 3 (left) and 4 (right) were repeated with varying values for *probreprod*: 0.3, 0.4, and 0.5, or 0.4, 0.5, and 0.6, respectively. For each scenario and parameter set, the means and standard deviations were calculated over the 30 replicates. The x-axis depicts units of time, while the y-axis depicts the trait values (equivalent units as for the x-axis). For each scenario, the plot shows the age at reproductive onset (red) and death (green) under all three parameter conditions.

at death increased (see **Fig. 8**). The magnitude of the correlation was larger, the bigger the difference between the age at death in the main simulation with selection and the reference without selection was. In the rare cases when the reference of the age at death (or reproductive onset) was lower than the main simulation, the correlation was positive for those sections.

The exact behaviour of the trait trajectories was determined by the choice of parameters. However, the tendencies remained and it was mostly just a shift along

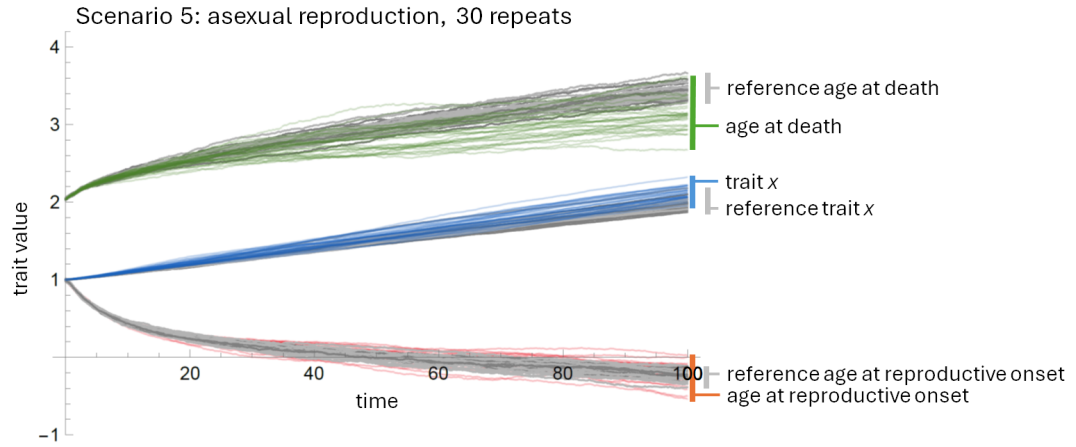

Fig. 7: Trajectories of the selected trait x, age at reproductive onset, and age at death for 30 replicates of the Scenario 5 with weak trade-offs for asexually reproducing populations.
The plot shows 30 independent simulation replicates for the Scenario 5. The y-axis scaling differs from the previous plots, all else is the same as for **Fig. 4**.

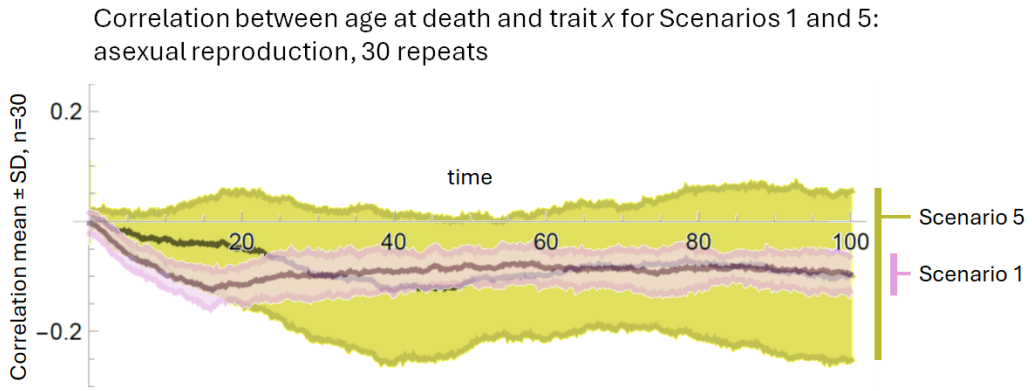

Fig. 8: Average correlation of the selected trait x with the age at death over 30 replicates of the Scenario 1 and 5 simulations for asexually reproducing populations. The correlation between trait x and age at death was computed at every time point ($\Delta t = 0.1$) individually using the distributions of the two traits in the population at that time. The correlation coefficients were then averaged over the 30 replicates. The means and standard deviations of the correlations are depicted for Scenarios 1 (plum and pink) and 5 (olive and yellow). The remaining scenarios are not shown, since their means closely resemble the two depicted means and their standard deviations are within the range of that of the two depicted scenarios.

the spectrum or a change in the signal strength. Increasing the genetic variance of the traits, the heritability, the selection strength or the number of offspring typically all lead to a stronger decrease in the age at reproductive onset and death, and a stronger increase in the selected trait x.

The simulations of sexually reproducing populations gave very similar results to those of the asexually reproducing population. Again, the scenarios with strong life history trade-offs (Scenarios 1 and 2), always showed a strong decrease in age at reproductive onset and death, while the selected trait x increased stronger than the reference (see **Fig. 9**). With intermediate life history trade-offs (Scenarios 3 and 4), age at reproductive onset and death could either increase or decrease, depending on the exact choice of parameters (see **Fig. 10** and **Fig. 11**). Finally, the simulations with no life history trade-offs (Scenario 5) again showed a decreasing age at reproductive onset, but an increasing age at death (see **Fig. 12**). The trait x, however, still increased faster then its reference.

Interestingly, for all the scenarios with the sexually reproducing population, the correlation between the trait x and the age at death (or reproductive onset), was almost zero (see **Fig. 13**). This was reflected also by the life history trajectories from the main simulations not differing from the reference simulations.

While the asexually and sexually reproducing populations show the same tendencies for trait evolution, there are small differences between them. For one, the sexually reproducing populations required a larger mutational variance to maintain a

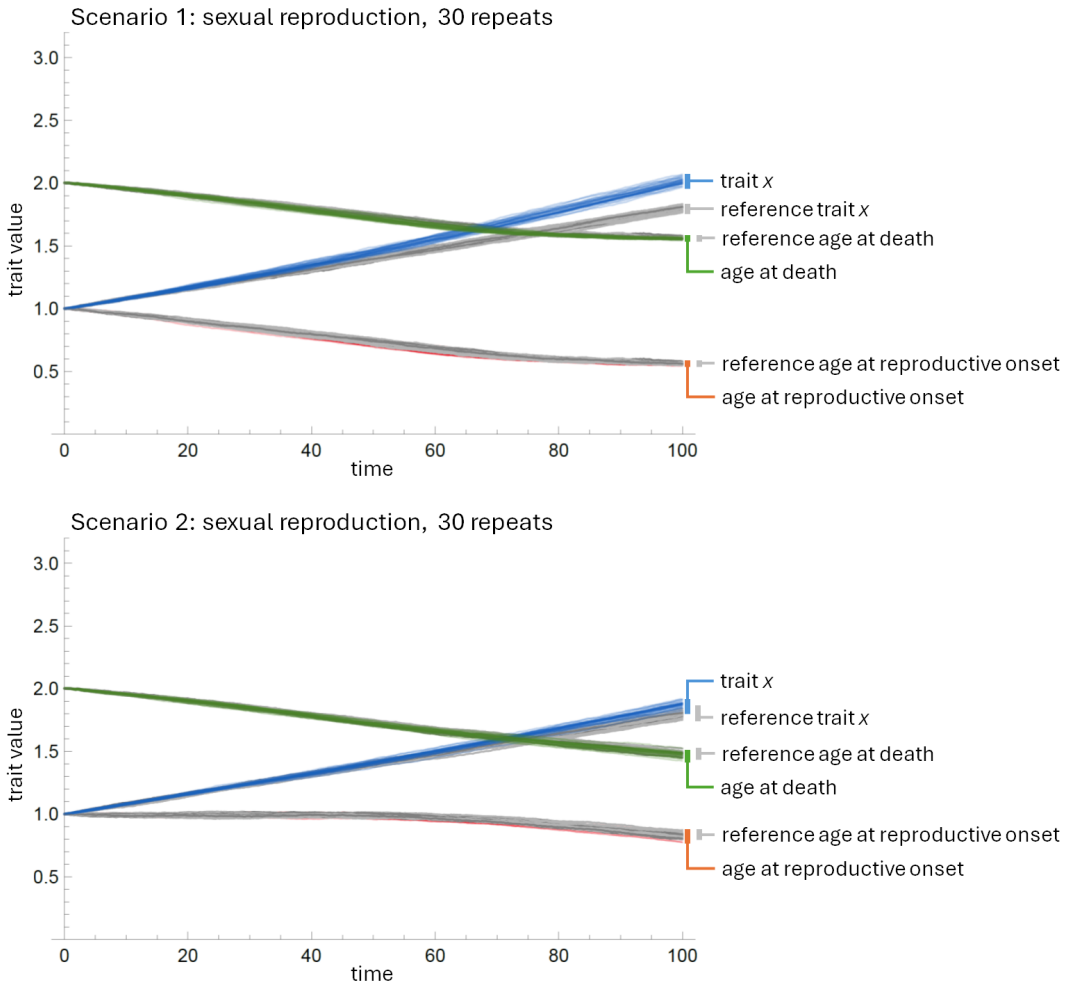

Fig. 9: Trajectories of the selected trait x, age at reproductive onset, and age at death for 30 replicates of the scenarios with strong trade-offs for sexually reproducing populations.

Each plot shows 30 independent simulation replicates for Scenario 1 (top) and Scenario 2 (bottom). Everything is the same as for **Fig. 4**, except that the mutational variances are larger (see **Table 1**).

roughly constant variance in the traits over time. The correlation between the traits was also much lower for the sexually reproducing population. Further, between simulation repeats of a given scenario and set of parameters, the trait trajectories were more similar to one another for sexual reproduction. The trajectories were also more similar there between the main and the reference simulations.

Discussion

The simulation results show us, that there are implementations of life history trade-offs, where an earlier age at death evolves, without any direct selection on the age at death. Across both the asexually and the sexually reproducing populations, the scenarios with strong life history trade-offs through coupling of traits have lead to a decrease in the age at reproductive onset and age at death. Effectively, this is an evolution of a faster life history. The lifespan has been decreased, speeding up the life cycle so that all reproductions occur earlier on average. This decrease in age at reproduction enabled an increase in evolvability, which become apparent when comparing the trajectories of the selected trait x with its reference trajectories. In

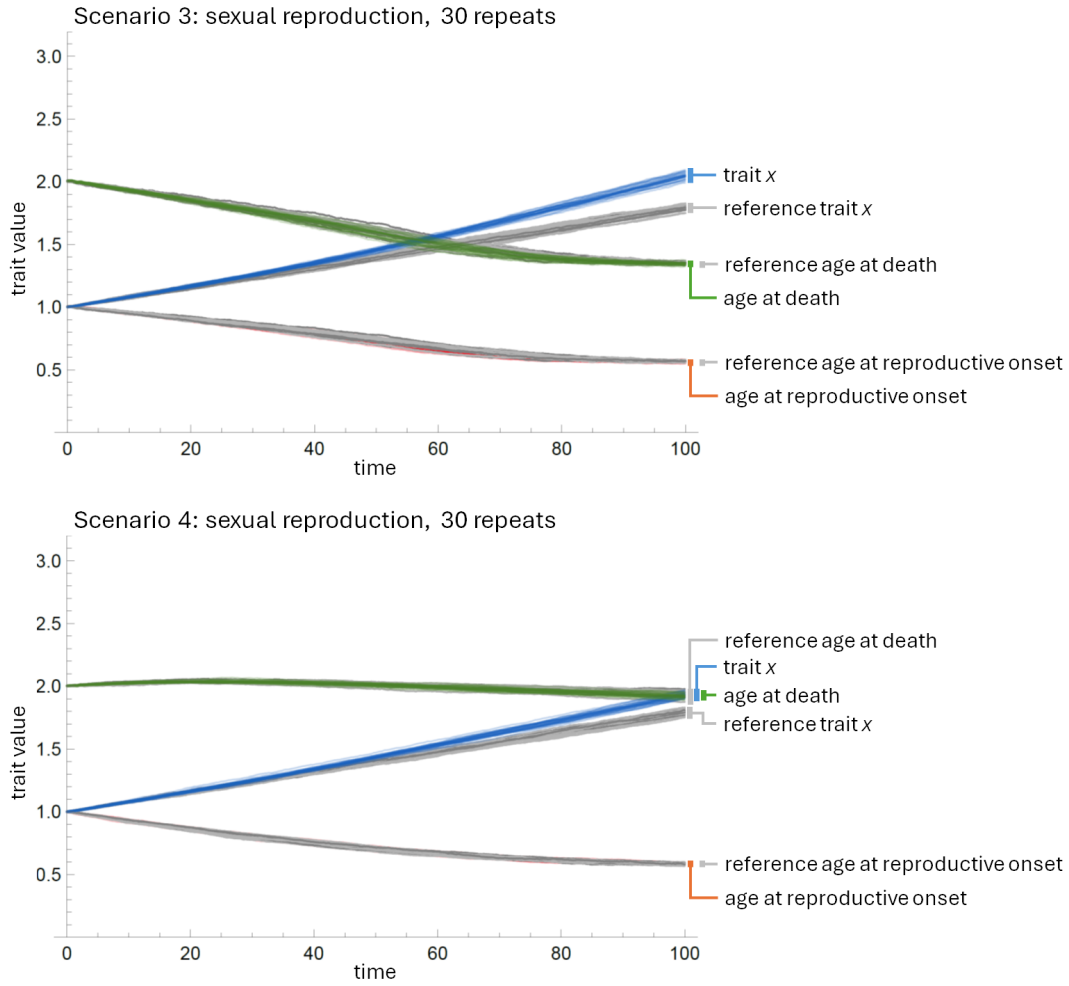

Fig. 10: Trajectories of the selected trait x, age at reproductive onset, and age at death for 30 replicates of the scenarios with intermediate trade-offs for sexually reproducing populations.
Each plot shows 30 independent simulation replicates for Scenario 3 (top) and Scenario 4 (bottom). All else is the same as for **Fig. 9**.

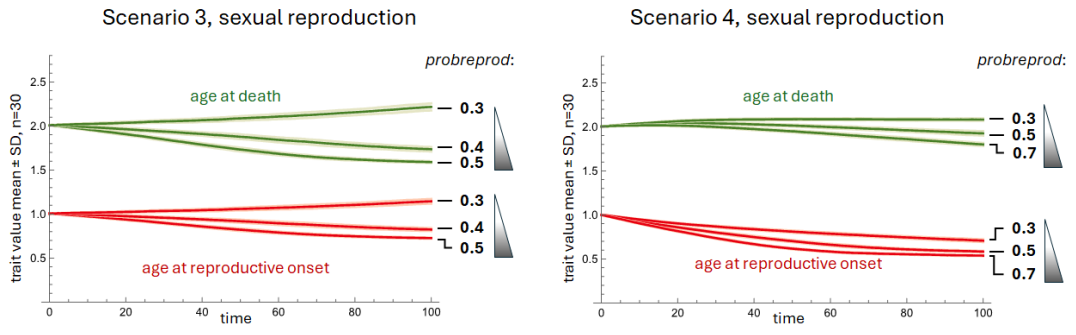

Fig. 11: Age at reproductive onset and death depending on the mean probability of successful reproduction for Scenarios 3 and 4 for sexually reproducing populations. Simulations for Scenarios 3 (left) and 4 (right) were repeated with varying values for *probreprod*: 0.3, 0.4, and 0.5, or 0.3, 0.5, and 0.7, respectively. All else is the same as for **Fig. 6**, except that these are sexually reproducing populations.

the reference, the age at reproductive onset and age at death were kept constant, therefore the life cycle remained the same over generations. Under these conditions, the trait x did not change as strongly, as when the life history traits could change. The rate of change was lower, in other words, the population was less evolvable.

The increased evolvability was a consequence of the shift towards earlier reproductions and an earlier ending of the life cycle, since this meant that the following generations replaced each other faster. Thus, in a given amount of time, the population experienced more variation and could adapt faster.

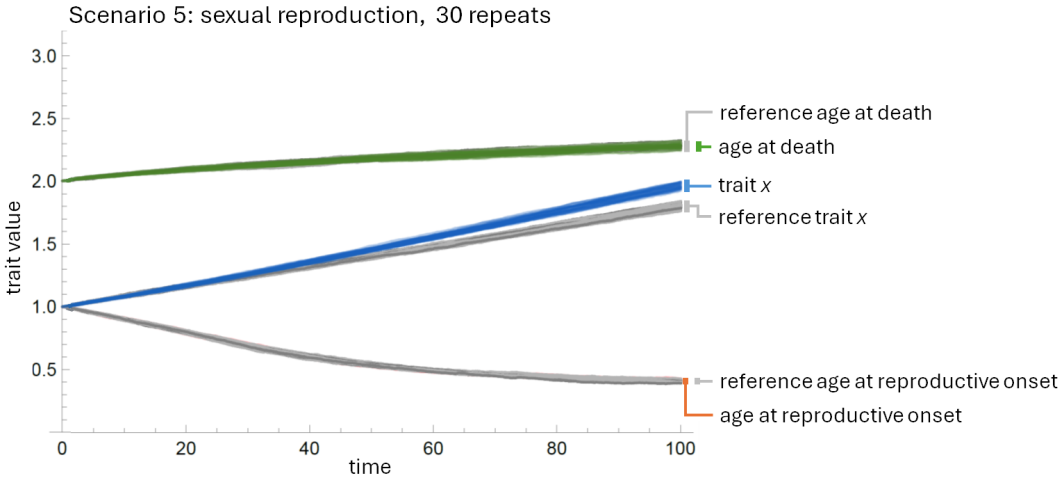

Fig. 12: Trajectories of the selected trait x, age at reproductive onset, and age at death for 30 replicates of the Scenario 5 with weak trade-offs for sexually reproducing populations.
The plot shows 30 independent simulation replicates for the Scenario 5. All else is the same as for **Fig. 9**.

Fig. 13: Average correlation of the selected trait x with the age at death over 30 replicates of Scenario 1 for sexually reproducing populations.
The correlation between trait x and age at death was computed at every time point ($\Delta t = 0.1$) individually using the distributions of the two traits in the population at that time. The correlation coefficients were then averaged over the 30 replicates. Only the mean and standard deviation of the correlation for Scenario 1 (plum and pink) is depicted. The remaining four scenarios are not shown, since they do not visibly differ from the depicted scenario.

This was also the case for the life history trait references. Although the trait x was not under selection, earlier reproduction was by itself beneficial, as it increased the Malthusian fitness. Further, earlier reproduction increased the evolvability for every trait, including the life history traits themselves. Since reproduction ages in the parents and their offspring are correlated due to heritability, early reproduction re-enforces itself and aids its own evolution towards earlier ages (see **Fig. 1**, compare equally-coloured rectangles with different lifespans). In fact, the main driver of the shift towards earlier reproduction was caused by the fitness benefit of earlier reproduction, as could be seen from the strong decrease in reproductive ages from the reference simulations.

Since an earlier reproduction and a beneficial trait value for the trait x are both beneficial individually, they start to co-occur more often, thus becoming statistically coupled. Over time, a negative correlation was built up, where individuals with a higher and more beneficial trait x value also tended to have earlier reproductions. Therefore, when the trait x was under selection, this correlation further drove the change of reproductive onset and death towards earlier ages. This was shown by the differences between the main simulations of the life history traits with selection on the trait x and the references without selection. This additional decrease in age at reproduction, however, was small compared to the decrease seen in the references. The likely reason for this is that also the magnitude of the correlation between the age at reproductive onset or death with the trait x was fairly small, compared to the correlation of the traits with themselves, which is equal to their heritability.

In the sexually reproducing populations, there seems to be no difference at all between the main and the reference simulations for the age at reproductive onset and death. We see here that also the correlation between these traits and the selected trait x is almost zero. This very weak correlation likely occurred due to the averaging and recombination of trait values in the sexually reproducing individuals. Recombination here does not refer to molecular recombination, as chromosomes and alleles were not explicitly modelled in these simulations. However, taking the mean genetic value of the parents and defining that as the pre-mutational genetic value for the offspring corresponds to the result expected of highly polygenic traits after recombination and chromosome segregation (Barton et al., 2017). Thus, recombination here refers to the traits coming together in new ways.

While strong life history trade-offs, such as in the Scenarios 1 and 2, lead to an earlier age at reproductive onset as well as death, in the absence of these life history trade-offs (Scenario 5), the age at death always increased. This strong increase occured because there was the possibility to maximise ones fitness by living longer and producing more offspring without constraints. These late-born offspring would on average not be as well adapted to the changing environment as the offspring of their earlier-born siblings, but they still contributed to the reproductive fitness of their parents. Since the late-born offspring increased the parent's mean age of reproduction, this also contributed to a reduction of evolvability. However, due to the strong and sudden decrease in age at reproductive onset, in total, the evolvability was still higher than in the reference where age at reproductive onset and death were fixed for all generations. Even when age at reproductive onset was fixed but age at death was free to change (simulation results not shown), the age at death increased, showing that at least with the tested parameter set (see **Table 1**), the benefit from more offspring rather than increased evolvability was higher.

Age at death did not increase endlessly though. The trajectories flattened after an initial fast increase, seeming to reach a plateau. This plateau could be caused by (1) selection being so strong that by the time parents reached the plateauing age, they typically were no longer selected to reproduce anyway, or (2) the upper boundary on population size preventing longer life and more offspring from being born. Beyond the plateau point, there would no longer be a benefit of further offspring, as then more individuals would need to die to maintain a constant population size. Most likely, the first explanation is causing the plateau. This is supported by the age at death increasing further when a lower selection strength was used for the simulations (simulation results not shown). On the other hand, doubling the maximal population size (from 5000 to 10000) did not lead to changes in age at death,

excluding the second option.

A unique effect seen in Scenario 5 is that although there was a selection against too early reproduction, the age at reproductive onset could in some cases still decrease below the constraining threshold value, sometimes even below the age of zero (see **Fig. 7**). This was only possible, because unlike for the other scenarios, there was no cost to the number of offspring born after having reached the threshold age. These early reproductions before the threshold age were simply not realised. Therefore, there was effectively no selection pressure for the genetic component of reproductive onset to stay above the threshold or, indeed, above zero. However, this by itself does not explain why the age at reproductive onset continued to decrease so dramatically. The likely reason for this is that while the mean age at reproductive onset has reached the threshold and thus the earliest possible age to reproduce, some individuals might still have been above the threshold and therefore did not use the full potential of their lifespan to reproduce. Therefore, to maximize offspring number in the population, selection kept decreasing the mean age at reproductive onset, until every single individual has reached the threshold age below which it could not reproduce more.

Most interesting, however, are perhaps the scenarios with intermediate life history trade-offs, since they probably most closely resemble the nature of trade-offs in real species. Here, depending on the choice of parameters, age at reproductive onset and age at death could either increase or decrease. The evolvability and therefore the change in the trait x were affected accordingly. An increase in the mean reproductive age would decrease evolvability, which was demonstrated by the trait x increasing slower than its reference in these cases.

The main factor influencing the direction of change was the number of offspring, particularly through the variable *probreprod*, which describes the mean probability of reproductive success of the population. The higher *probreprod* was and thus the more offspring produced, the stronger was the decrease in the life history traits (**Fig. 6** and **Fig. 11**). While a larger population size increases the strength of selection, this could not the case here, since the population size was kept constant. Rather, what might explain this behaviour of the simulations is that with a larger number of offspring, each individual offspring becomes less relevant, contributing to its parent's reproductive fitness to a smaller part. The relative fitness gain per offspring is lower with each additional offspring. Therefore, with high offspring number, gaining one further offspring by living longer might not be worth the reduced evolvability. However, when few offspring are born, then the benefit of one more offspring outweighs the disadvantage of reduced evolvability.

The simulation results show that increasing the pace of life increases evolvability and is thus beneficial. This further accelerates its own evolution. As long as age at death is coupled to other life history traits that contribute to this faster life, such as the age at reproductive onset, it is itself a component of it and evolves towards earlier ages too. These results might explain how senescence evolved and why there are such huge differences in lifespans and the rate of senescence across the tree of life.

In the simulations, different degrees of coupling of life history traits had very different outcomes on the evolution of age at death. When there was no coupling and no trade-off with regard to offspring number, then later senescence evolved, since age at death increased. When there was strong coupling with trade-offs, then earlier senescence appeared as a consequence of a faster life cycle, which increased evolvability. An intermediate degree of trade-offs could lead to very variable outcomes, which also depended on other parameters, most notably, the number of offspring.

Trade-offs between life history traits and constraints on reproduction have been observed in various species (Blomquist, 2009; Flatt, 2011; Lemaître et al., 2015; McKenna-Ell et al., 2023; Pettay et al., 2005). Since different species have opted for differing life history strategies, where distinct trade-offs come to play, this could lead to very diverse responses of life history traits to selection and thus evolution of senescence.

Thus, these results present a valuable addition to the current theories on the evolution of senescence. The theories on adaptive, programmed senescence have most strongly been opposed because no mechanism of evolution has been proposed or because it was mostly suggested to evolve through group selection. The simulations show that an earlier age at death and thus senescence could evolve without group selection but instead as a component of a faster life history, which has beneficial effects on the population level. When looking at senescence alone, age at death does not decrease, because the cost it brings in offspring number is too large and cannot be compensated by increased evolvability. However, with trade-offs between traits, by compressing the reproductive events or shifting them forward, the pace of life is increased with no or a tolerable cost to reproduction. In this case, the age at death can shift forward too. Senescence would thus be a result of the evolution of a faster life under life history trade-offs.

While this work has been on the ultimate cause of aging, it is important to also consider how it could align with the proximal causes, the mechanisms leading to aging in current populations. On a molecular level, aging is often associated with increased mutations and DNA damage, the shortening of telomeres, increased dereg-

ulation of metabolism and inflammation (López-Otín et al., 2023). This could also be described as a decrease in order, or an increase in entropy. To maintain order, energy is required, for example in the form of ATP. The restricted availability of energy could both impose a constraint on life history traits needed for the evolution of senescence, as well as cause the damages associated with senescence. If the available chemical energy is not sufficient to at the same time allow reproduction (e.g., investment of chemical energy in the form of yolk, nutrients, glucose; higher energy expenditure to care for offspring after birth) and accurate maintenance of molecular and cellular structures to occur, damages will start to accumulate. Certain DNA damages might remain unrepaired, telomeres might not be extended, components required for the tight control of cellular processes might not be synthesised sufficiently. Over time, these damages would accumulate in an individual's lifetime. Once they reach a critical point, which depends also on the reproductive activity, their effects might be too severe to allow the organism to function properly. This would also feed back into the reproductive activity, completing the constraint between early and late reproductions. Alternatively, constraints could be imposed by damage directly caused by reproduction. For example, Chatzidaki et al. (2021) have shown that ovulations in mice create strong oxidative stress that damages other oocytes. Thus, the number of viable oocytes is restricted. These are of course only speculations, whether the available energy is indeed restricted in such a way and what the mechanisms behind constraints are remain open questions.

Based on the current evidence for the different theories, it seems that senescence might have evolved as a combination of it being indirectly adaptive by increasing evolvability, and an accumulation of (antagonistic) mutations. There is mixed evidence for both the adaptive and non-adaptive theories, and both only explain parts of the diversity in senescence observed in nature.

Interestingly, similar ideas to the evolution of senescence also exist about the evolution of menopause. Although menopause is often studied separately, it is itself also one aspect of senescence. Theories range from antagonistic pleiotropy, physical constraints on the shelf-life of oocytes, increased inclusive fitness to increased adaptability through shorter generation times (Chatzidaki et al., 2021; Croft et al., 2015; Hawkes et al., 1998; Johnstone & Cant, 2019; Pollycove et al., 2011). Similarly to senescence, no consensus has been found as to how menopause evolved. Our results for senescence could, however, be of relevance here, too, as they support the idea of increased adaptability through shorter generation times.

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